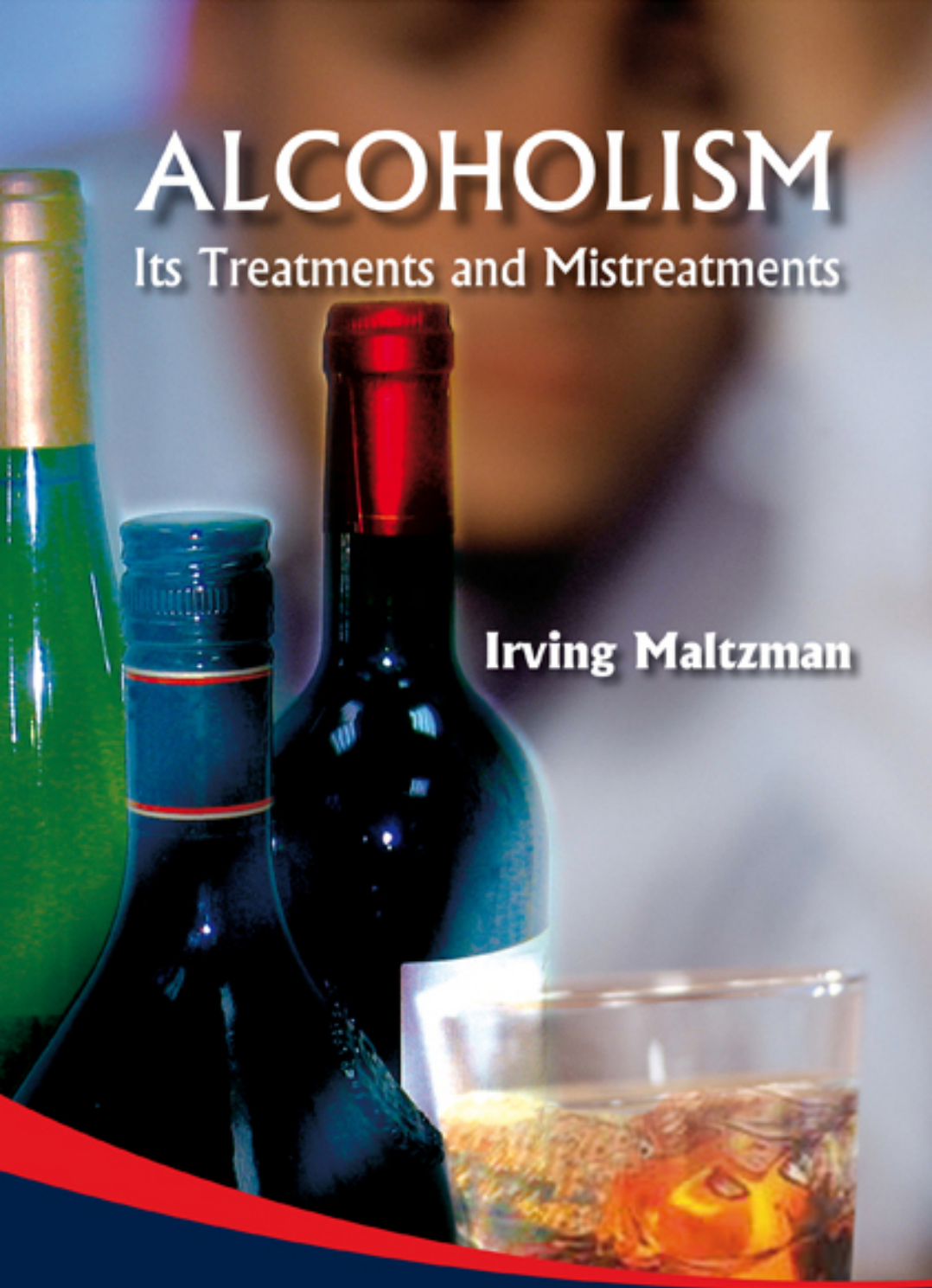
ALCOHOLISM

Its Treatments and Mistreatments



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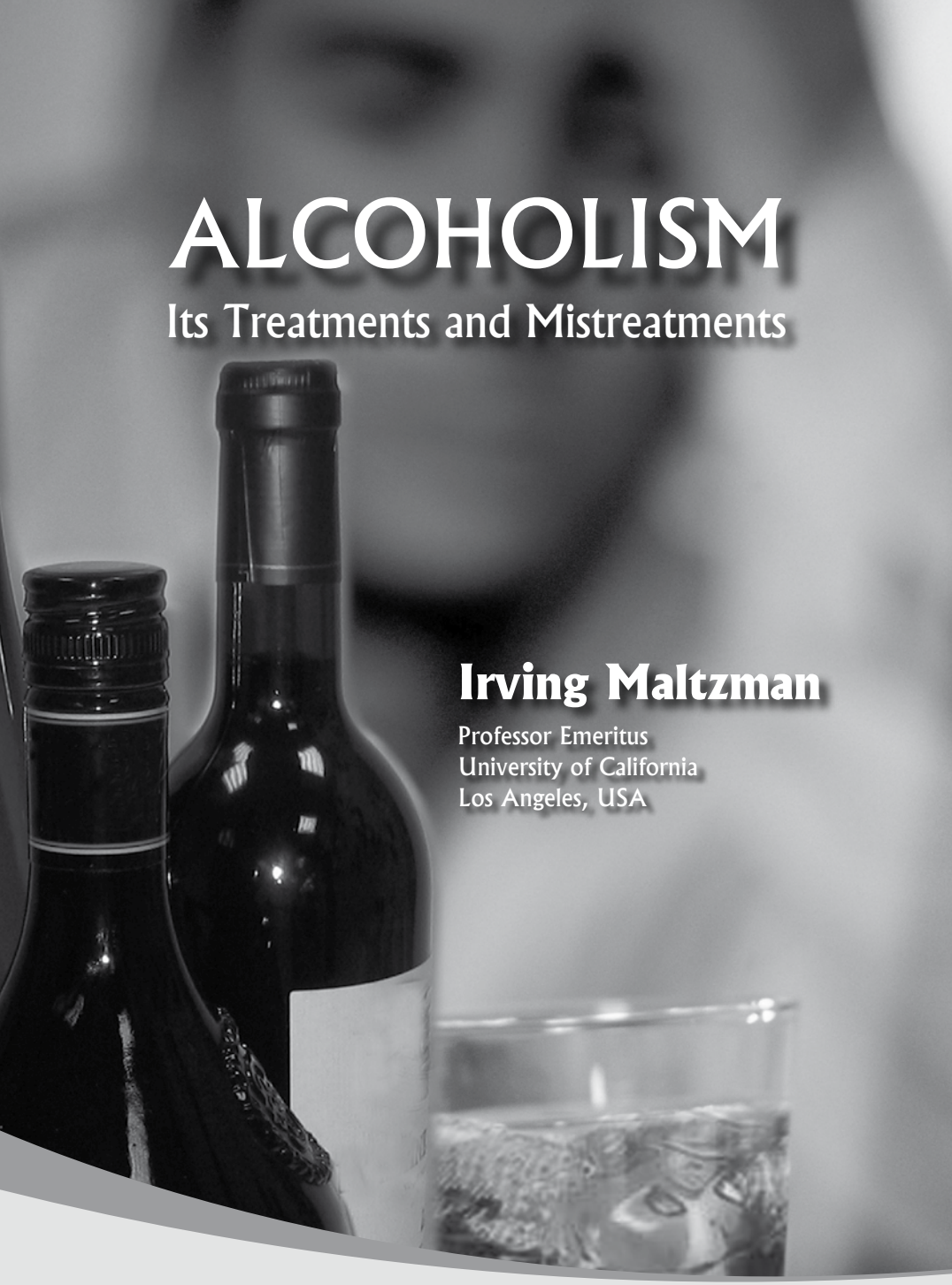

ALCOHOLISM

Its Treatments and Mistreatments

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A Biosociobehavioral Disease Conception of Alcoholism

Miller and Hester (2003), in the introductory chapter of their *Handbook of Alcoholism Treatment Approaches: Effective Alternatives* (3rd ed.), present various "conceptual models" or interpretations regarding the characteristics and etiology of alcohol problems:

> Experts in the field ascribe alcohol problems and alcoholism to a bewildering array of causes: inherent biochemical abnormalities (Blum & Payne, 1991; Milam & Ketcham, 1981), genetic influence (Begleiter & Kissin, 1995), conflictual emotions (Denzin, 1993), irrational cognitions (Ellis & Velten, 1992), social learning processes (Orford, 1985; Peele, 1985), family pathology (Curtis, 1999; Steiner, 1971; Steinglass, Bennett, Wolin, & Reiss, 1987), sociocultural influences (Cahalan, 1987; Edwards *et al*., 1994; Holder, 1998), self-regulation failure (Baumeister, Heatherton, & Tice, 1994), and personal choice (Fingarette, 1988). Given such disagreement about the essence and causes of alcohol problems, it is little wonder that there has been such confusion about how best to treat people who have them. [p. 2]

The above evaluation is confusing and misleading. There is evidence of multiple causes or risk factors in the environment, the host, and their dialectical interactions reported in many studies of alcoholism and its causes, characteristics, and treatments (see Maltzman, 2000). Some

"experts" named by Miller and Hester (2003) are not experts in the field of alcoholism (Baumeister *et al*., 1994), while others are merely anointed fabulists who agree with Miller and Hester (Fingarette, 1988). There is no disagreement among neuroscientists, behavioral neuroscientists, biomedical scientists, or behaviorists concerning the "essence" of alcoholism, just as there is no disagreement about the essence of cancer, malaria, or other diseases. There is no disagreement that it is a scientifically meaningless question — it is a meaningless question, and therefore not asked, because any solution to the question would not be falsifiable. As far as "causes" are concerned, multiple dialectically interacting risk factors are generally involved in the etiology of diseases; and alcoholism is no exception.

The above introduction by Miller and Hester (2003) sets the stage for the presentation of a variety of outdated or misrepresented models, or interpretations, of alcoholism. The models — briefly presented along with their purported implications for treatment and prevention — include moral, temperance, spiritual, dispositional disease, biological, educational, characteriological, conditioning, social learning, general systems, sociocultural, and public health ones. None of these models or interpretations as described by Miller and Hester properly define "disease", nor do they reflect current analyses of a disease conception of alcoholism (Maltzman, 1991, 1994, 1998, 2000). The purpose of the present chapter is to present such an analysis of "disease", i.e. a disease conception of alcoholism and some of its implications.

Two characteristics are common to all diseases: (1) each disease is a syndrome, defined as a lawful pattern of recurring observable signs and symptoms (Igoe,1979). A disease does not cause the observable signs and symptoms, but rather the disease *is* the lawful pattern of observable signs and symptoms; and (2) the syndrome is judged by experts to be a significant deviation from an accepted standard of health. The first characteristic is an empirical matter; while the second is a value judgment and normative in nature, and therefore may vary with the culture and time of the experts' judgment.

A known etiology and pathophysiology are unnecessary for the classification of a condition as a disease (Cohen, 1961); for example, pulmonary tuberculosis and malaria were recognized as diseases for centuries prior to knowledge of their etiology and pathophysiology. The

conception of alcoholism as a disease is not a mere mental construct, an arbitrary decision to label a condition a disease for political or psychological reasons. Alcoholism is a disease entity that exists because the observable symptoms, although differing widely in appearance, are lawfully related. They are a consequence of biological adaptations and damage produced by alcohol and its effects on the nervous, neuroendocrine, immune, and digestive systems. Alcoholism is not defined by the amount of alcohol consumed: although one cannot become an alcoholic without consuming alcohol, the correlation between the amount of alcohol consumed and its negative consequences — alcohol problems — is not high (Drummond, 1990). Lack of a strong relationship may be the consequence of a variety of individual differences in the biology of the host, including changes in metabolism (Mendelson, 1964), brain damage following chronic heavy drinking (e.g. Cala, 1987; Oscar-Berman & Hutner, 1993; Parsons, 1998; Tarter, 1975), and differences in the social environment interacting with behavior and biology (Higley *et al*., 1991). For example, an alcoholic who is independently wealthy and does not work for a living, or a college professor with tenure and teaching and research assistants, will probably manifest fewer negative social consequences and fewer alcohol problems than a cashier at the checkout stand of a supermarket who is closely supervised and whose occupation provides quantitative indices of productivity. Nevertheless, there are profound changes in general health and well-being as well as personality as a consequence of the damage produced by alcohol to the nervous, neuroendocrine, and immune systems. It is a disease of the whole person.

The assertion that alcoholism is on a continuum with normal drinking, and therefore obeys the normal laws of social learning (Marlatt, 1979; Miller, 2001; Goldman *et al*., 1999b), is false. "Laws" of social learning are falsified by critical behavioral studies of alcoholics (Hodgson *et al*., 1979) and by the adoption studies of Goodwin and his colleagues (Goodwin *et al*., 1973, 1974). Hodgson *et al*. (1979) showed that a priming drink of alcohol in the morning produced a satiation effect on alcohol consumption in the afternoon and decreased the alcohol consumption of moderately dependent alcoholics; in contrast, a priming drink produced an appetizer effect in severely dependent alcoholics, increasing their later alcohol consumption. A qualitative difference in priming effects was

found, rather than a continuum as necessarily implied by a social learning continuity interpretation.

A social learning account of the signs and symptoms of alcoholism lacks verisimilitude. Another recurring myth promoted by revisionists (e.g. Barlow & Durand, 1995, 2002, 2005; Fingarette, 1988; Mendelson & Mello, 1985; Peele *et al*., 2000) is that the conception of a lawful symptom progression is based on a study of only 98 members of Alcoholics Anonymous (AA) (Jellinek, 1946). Jellinek's (1952) later study of 2000 members of AA and the more-than-a-dozen replications of the kind of results obtained by Jellinek are ignored by revisionists (see Maltzman, 2000). Replications include both male and female participants who were not AA members (Pokorny & Kanas, 1980), a general population sample (Nelson, Little, Heath, & Kessler, 1996; Piazza & Wise, 1992), and a study of Finnish men (Park & Whitehead, 1973). Core symptoms progress in the same order, regardless of culture, gender, or social status; symptoms may progress at different rates in different cultures and as a function of age and gender, but the order of the core symptoms is essentially the same. Blackouts and denial occur before morning drinks and delirium tremens, whether a person is Finnish or North American, female or male.

Variability in characteristics among alcoholics and in the risks of becoming alcoholic are not peculiar to alcoholism. Variability in etiology is a common characteristic of diseases. Variability probably receives more attention in the case of alcoholism than other diseases because a necessary condition, alcohol consumption, is readily open to study. Necessary causal conditions are not ordinarily as readily apparent in other diseases, requiring far greater intensive research to determine necessary causes. A natural experiment described by Evans (1993) provides an unusual opportunity to examine the variability that occurs in infectious diseases. He reported an incident where the hepatitis B virus contaminated the yellow fever vaccination given in similar fashion to more than 5000 soldiers. Only 20% of the men developed jaundice, a symptom of hepatitis. The appearance of clinical symptoms varied between 60 days and 154 days in soldiers who developed hepatitis. Unknown sources of variability in resistance of the soldiers' immune system were presumably responsible for the enormous variability in the occurrence and appearance of disease symptoms.

If alcoholism meets the criteria for classification as a disease, as evidence and critical analyses show beyond reasonable doubt, then greater effort must be made to study the problem as a disease. Greater research effort is needed to elucidate the basic pathophysiology of the diseased person and the individual's interaction with the social environment. Also essential is the careful study of individual differences as a function of gender, medical history, ethnicity, age, social environment, and culture, as well as of the differences within each larger group manifested in the development of biological dysfunctions and behavioral and social symptoms. A recent international symposium summarized the progress being made in the neurobiology of alcoholism and recovery (Crews *et al*., 2005). A research emphasis on the biosociobehavioral dysfunctions underlying the development of alcoholism and the biosociobehavioral changes accompanying its successful treatment and aftercare would be time, effort, and money far better spent than wasting it on mind-dust research such as the study of expectancy as a cause of alcoholism and its treatment outcome. A verbal report of an expectancy or its definition by responses to a series of questionnaire items is an effect, not a cause, of alcohol consumption and learning. These and related issues concerning expectancy will be discussed in greater detail in Chapter 4.

Revisionists ignore the evidence that alcohol is a powerful drug causing damage to cells, organs, and entire systems when consumed excessively. Much of the damage is unseen and unrecognized. What constitutes excessive consumption will vary both among individuals (in terms of general health, social context, gender, etc.) and for the same individual (at different times, between meals, etc.) (Eckardt *et al*., 1998). For social drinkers generally, 2 drinks/day for men and 1 drink/day for women yield the lowest risk for morbidity and mortality compared to abstinence. This generalization is based on the results of a large prospective interview study of approximately 44 000 participants representative of persons aged 40 years or older at baseline and reassessed approximately 6 years later (Lio *et al*., 2000). It is in accord with the recommendations of the United States Dietary Guideline Committee (US Department of Agriculture, 1995) and other health committees.

Alcoholism must be investigated as the biosociobehavioral disease that it is rather than merely reciting the mantra of "biopsychosocial"

disorder. More than a functional analysis of the discriminated operant of elbow bending is needed. Alcoholics have a higher morbidity, poorer quality of life, and shorter life expectancy than nonalcoholics. They suffer from a higher incidence of liver disease, cardiovascular disease, neuropathy, pancreatitis, cancer, infectious diseases, and structural and functional brain damage (10th Special Report to U.S. Congress on Alcohol and Health, 2000). Important negative social consequences include lost hours of work, disruption of family and social life, and increased medical expenses for the individual and society. Psychopathology is not exempt from the litany of problems; alcoholics as compared to nonalcoholics suffer from a higher incidence of depression, suicide, and anxiety. They do harm not only to themselves, but also to family, friends, and society at large, as the result of automobile crashes and other transportation/industrial accidents. Let us not forget the alcoholism-related 1989 Exxon Valdez oil spill accident, which resulted in an estimated US$2 billion worth of damage to the Alaskan environment. Its costs are still rising due to medical complications produced by the toxic effects of the petroleum to which hundreds of cleanup workers were exposed. A federal judgment more recently awarded US$6.75 billion to the plaintiffs, including US$4.5 billion in punitive damages and approximately US$2.25 billion in interest to the thousands of people who had made their living in the site damaged by the spill (*Los Angeles Times*, January 29, 2004).

Numerous important research problems arise following the adoption of a disease conception of alcoholism. They are foreign to a cognitive social learning theory conception of alcoholism as a bad habit. Damage must be assessed in the biological systems of the human agent: the nervous, neuroendocrine, digestive, and immune systems. Changes within each system produced by alcohol consumption must be studied at different levels, from the subcellular to the behavioral, social, and cultural. Changes in the interactions among systems produced by alcohol consumption must also be examined in detail and in relation to their interaction with behavior and the social environment. We assume there are multiple qualitative as well as quantitative changes within each system and in its interactions, varying from social drinking to chronic alcoholism, cirrhosis of the liver, and Korsakoff's syndrome.

A disease conception of alcoholism implies that the medical history of the mother before, during, and after pregnancy must be examined for alcohol and other drug use, viral and bacterial infections, unusual aspects of the delivery, and diet during the prenatal and postnatal periods. Social support, stress, and conflict within the family must be assessed (Huizink *et al*., 2004; Myslobodsky, 2004), as well as the family history of alcohol and other drug use. Ethnic, racial, gender, and cultural differences must be studied, and not just between large groups such as Asians and European-Americans: examination of specific national, cultural, and ethnic differences is also necessary. Differences among Korean, Chinese, and Japanese men and women must be considered, as well as differences between Mexican-Americans and other residents in the USA originating from South and Central America. In addition, differences within as well as between religious, national, and cultural groups need to be studied; for example, Italian vs. Irish Catholics, Ashkenazi vs. Sephardic vs. Oriental Jews, etc. Effects of cultural traditions, familism, and acculturation of various immigrant groups must be studied (Hillhouse & Fiorentine, 2001; Nielsen, 2001; Straussner, 2001).

There must be greater sensitivity to cultural differences in treatment. Beneficial interactions between counselor and patient may in part be due to a therapeutic alliance that is affected by biological changes produced when a patient is treated by a member of the same ethnic or religious group. For example, Spinrad (1993) found a significant interaction between ethnic group, type of training, and compliance with a disulfiram regime. Neurohumoral states may be changed; serotonin, endorphin, oxytocin, and vasopressin levels may be increased when there is a therapeutic alliance. Basic biological factors are operating that may have important consequences on the course of treatment. Biobehavioral studies need to be conducted during the treatment process as well as before and after treatment.

Alcohol and the Brain

There is increasing brain damage from social drinking to chronic alcoholism, interacting with many variables including age and drinking

pattern. Korsakoff's syndrome involves additional damage (Brokate *et al*., 2003; Crews, 1999; Crews *et al*., 2005; Oscar-Berman *et al*., 2004; Parsons, 1998). Brain damage is not the sudden end result of alcoholism. Alcohol's effects on the brain are insidious, continuous, and destructive, especially on the developing brain (Tapert *et al*., 2002). Other serious consequences are more varied, sometimes depending upon the vulnerability of the host's immune system at the time. There are several different routes to alcohol's damage to the brain. Some are the direct toxic effects of alcohol and its metabolites on brain cells. Other sources of damage are indirect, by way of alcohol's effects on the neuroendocrine system via its effects on the hypothalamic-pituitary-adrenal (HPA) axis and its release of corticosteroids, and on the immune system's release of inflammatory cytokines.

Alcohol's deleterious effects on the nervous system and other biological systems are part of a spectrum of nonobvious negative consequences that include changes in mood, affect, and personality. For ethical reasons, experimental demonstration of these effects, including the increased brain damage produced by withdrawal, have been limited to infrahuman animal models (Paula-Barbosa *et al*., 1993; Phillips & Cragg, 1984). Quantitative analyses by Paula-Barborsa *et al*.

> showed a significant reduction in brain cells in the hippocampus in alcohol fed rats as compared to the matched nonalcohol fed control animals. Magnitude of the effect was related to the length of alcohol treatment. Animals in the withdrawal groups showed significantly greater neuronal loss than the alcohol fed rats who did not suffer withdrawal from alcohol. Earlier research by the same group using the same experimental design examined the medial prefrontal cortex and found a significant loss of brain cells in that region as a function of duration of alcohol treatment with additional loss of cell density following withdrawal. [Maltzman, 2000, p. 63]

Clinical studies of alcoholics leave no doubt concerning the extensive brain damage caused by chronic heavy alcohol consumption (Moselhy *et al*., 2001; Oscar-Berman & Hutner, 1993). Additional damage in alcoholics is caused by repeated withdrawal from alcohol (Glenn *et al*., 1988; Tapert *et al*., 2002).

According to Marlatt (1979),

> All drinking behavior, from social drinking to alcohol abuse, is assumed to be governed by similar principles of learning and reinforcement. As such, it is assumed that there is no crucial difference that distinguishes the social drinker and the problem drinker, other than the amount of alcohol consumed. [p. 324f]

Similar ill-conceived sentiments are expressed by others (Fingarette, 1988; Goldman *et al*., 1999b; Szasz, 1972). Human experimental and clinical research with alcoholic and control populations as well as infrahuman experimental research since the middle of the 20th century have accumulated an extensive body of evidence contradicting Marlatt's and other revisionists' notion that alcoholism is nothing more than a bad habit. Most importantly, Marlatt and other revisionists ignore the dialectical interaction between alcohol, the brain, and behavior. Jellinek (1960), in his classic work on the disease conception of alcoholism, reviewed all relevant theories of alcoholism at the time, including learning and social interpretations. He noted a hypothesis formulated by Lemere (1956) concerning the etiology of loss of control, a pathognomic sign and symptom of alcoholism, in terms of brain pathology. Jellinek's discussion highlights the need for long-term prospective studies differentiating between predisposing risk factors present prior to the initiation of heavy alcohol consumption and the damage produced following its initiation (Schuckit, 1998; Tarter *et al*., 1993).

Grohman and Fals-Stewart (2004) reported the results of a study of a new, relatively brief assessment battery that accurately discriminated neuropsychological deficits in patients receiving treatment for substance abuse. Results showed that more than one third of the patients were suffering from such deficits. It is essential that the administration of such neuropsychological assessments becomes a matter of course in treatment facilities, along with a standardized clinical diagnosis based on the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) categories. Such information is necessary for the individualization of treatment and for the development of shared databases permitting research that evaluates the status of neuropsychological function before, during, and after treatment.

The Saga of Phineas Gage

Advances in neuroscience and technology have provided evidence supporting the conceptualizations of half a century ago. Damage to the prefrontal cortex (PC) caused by alcohol may be a determinant of loss of control, denial, and the socially irresponsible behavior characteristic of many alcoholics. Pre-existing dysfunctions in the PC may predispose a person to alcoholism. Subgroups of individuals characterized by an early or adult onset of alcoholism accompanied by sociopathy may suffer the consequences of structural and/or functional damage in the ventromedial (VM) region of the PC.

A remarkable natural experiment in the mid-19th century contributed to the research and theorizing concerning the kind of decision making influenced by the VMPC (Damasio, 1994; Macmillan, 2002; Stuss *et al*., 1992). In 1848, Phineas Gage — a diligent, hardworking head of a construction gang laying railroad tracks — suffered a terrible accident. An explosion blew a steel tamping bar through his skull. However, it did not kill him. The damage was largely limited to the VMPC. Reports suggested that there was no apparent change in his intellectual ability. His verbal facility and ability to calculate and reason abstractly seemed the same as before.

> On the other hand, he had become irreverent and capricious. His respect for the social conventions by which he once abided had vanished. His abundant profanity offended those around him. Perhaps most troubling, he had taken leave of his sense of responsibility. He could not be trusted to honor his commitments. His employers had deemed him "the most efficient and capable" man in their "employ" but now had to dismiss him. In the words of his physician, "the equilibrium or balance, so to speak, between his intellectual faculty and animal propensities" had been destroyed. [Damasio *et al*., 1994, p. 110]

Gage apparently became a late-onset sociopath, amoral, unreliable, and untrustworthy (see Macmillan, 2002, for a detailed history and its fabulist embroidery).

Research by Damasio (1994) as well as by Bechara and colleagues (Bechara, 2004; Bechara & Damasio, 2002; Bechara *et al*., 1998, 2001,

2002) have provided striking clinical and experimental evidence for the role of the VMPC in regulating social and antisocial behavior, including substance dependence. An experimental gambling task has been devised by Bechara *et al*. (2001) that differentiates between individuals with antisocial personality disorders, those with alcohol and other drug dependencies, and controls.

Participants in the gambling task are provided with a cash allowance to gamble with four decks of cards. Drawing any card from decks A and B results in winning US$1.00; drawing a card from decks C and D wins US$0.50. However, participants lose US$2.50 for every 10 cards drawn from decks A and B, whereas they win US$2.50 for every 10 cards drawn from decks C and D. In addition, half of every 10 cards from decks A and B lose US$1.50–US$3.50 as well as win US$1.00/card. Thus, participants lose US$12.50 and win US$10.00 for every 10 cards played from decks A and B. In contrast, 5 of every 10 cards played from decks C and D lose US$0.25–US$0.75. Participants lose US$2.50 but win US$5.00 for every 10 cards played from decks C and D.

Bechara *et al*. (2001) used the gambling task to study the performance of three groups: individuals with lesions to the VMPC; individuals with a substance dependence (SD), either alcohol, methamphetamine, or cocaine; and a group of normal control subjects. The patient groups, VMPC and SD individuals, were chosen for study because they tend to show similar kinds of behavior:

> (1) they often deny, or they are not aware, that they have a problem, (2) when faced with the choice to pursue a course of action that brings in immediate reward, at the risk of incurring future negative consequences, including the loss of reputation, job, home, and family, they choose the immediate reward and ignore the future consequences. . . . [N]europsychological and functional neuroimaging data suggest that a decision-making impairment linked to a dysfunctional VMPC cortex may be at the core of addiction to substances. [Bechara *et al*., 2001, p. 376]

Additional measures including assessments of psychopathy, depression, and anxiety were obtained to ensure that evidence of decision-making impairment in the SD patients was not confounded with comorbidity.

A number of demographic measures were also used, such as age, gender, education, years of abuse of drug of choice, years of abstinence, cycles of relapse and return to treatment, and years of gainful employment. A prediction index was calculated to estimate the severity of decision-making impairment: "abstinence (in days) divided by the number of years of abuse, times the number of returns to treatment, multiplied by a factor of employment" (Bechara *et al.*, 2001, p. 377). Participants in the SD group met criteria for DSM-IV substance dependence; VMPC group members received neuropsychological and neuroanatomical assessments establishing a bilateral lesion of VMPC cortices. Normal control participants were solicited by newspaper advertisements. A computerized version of the gambling task was employed.

Figure 1.1 shows the results of the gambling task for the three groups: controls, SD, and VMPC lesion patients. It is apparent that

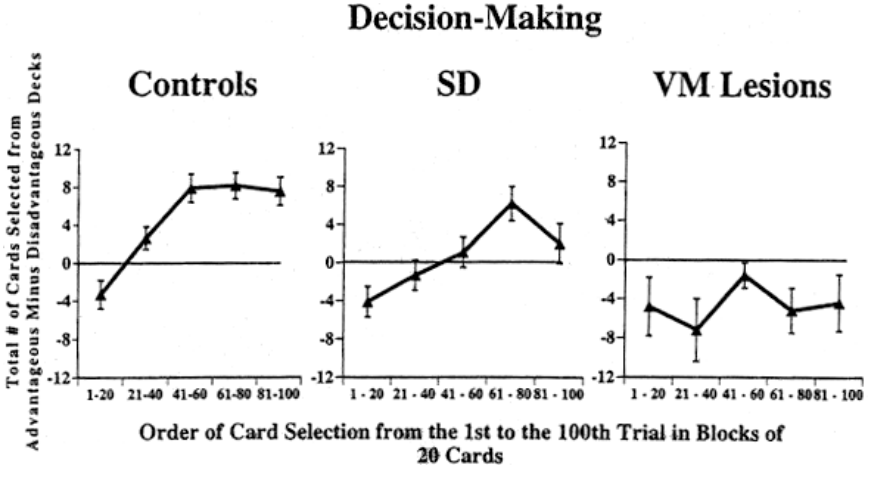

FIGURE 1.1. Relative to normal controls, substance dependents (SD) were impaired in their performance on the gambling task, but the impairment was not as severe as that seen in patients with bilateral lesions of the ventromedial prefrontal cortex (VM Lesions). Scores on the gambling task are presented as the mean + standard error of the mean (SEM) of the difference between the total number of cards chosen from the advantageous decks (C′ + D′) minus the total number chosen from the disadvantageous decks (A′ + B′). The scores are divided into five blocks of trials with 20 trials in each block, i.e. a total of 100 card selections (Bechara *et al.*, 2001, p. 383).

normal control subjects started selecting cards from decks A and B, providing immediate gain but larger future losses; but then quickly shifted to the more advantageous decks C and D, yielding lower immediate gain but larger future gains. Participants with VMPC lesions selected cards from the disadvantageous decks throughout the five blocks of 20 trials each. Performance by the SD group appears to be closer to the VMPC lesion group than to controls. Statistical analyses indicated that the control group differed significantly from the SD and VMPC groups. There was no significant difference between the SD and VMPC groups. For the VMPC group, the difference between the number of cards selected from advantageous and disadvantageous cards was less than 10 cards. Twenty-five (41%) of the 41 SD patients had net scores below 10, within the range of VMPC patients. Thirteen (33%) of the 40 control participants had scores below 10 cards.

Figure 1.2 shows that there was overlap between the groups, but the overlap was greater between VMPC and SD groups than between VMPC and control participants. Disaggregating the three groups revealed a subgroup of SD participants as impaired on the gambling task as the VMPC lesion patients. A smaller subgroup of SD patients was similar to the normal participants in their performance.

Age, gender, and education levels were not significantly correlated with gambling scores in any of the three groups. Analyses of SD participants indicated that net card selection was not significantly related to general neuropsychological measures of executive functions or measures of memory and intelligence. The specific drug of dependence — cocaine, methamphetamine, or alcohol — was not a differential factor. Years of abuse, duration of abstinence, and number of times in treatment were not significantly related to performance on the gambling task. Two demographic variables were significantly related to gambling task performance: holding employment and the previously described prediction index. Spearman correlations with the gambling task for the latter two variables were 0.36 and 0.43, respectively.

Bechara *et al.* (2001) raised an interesting question: did the VMPC dysfunction evident in the gambling task arise as a consequence of the neurotoxic effects of alcohol or other drugs, or was it a pre-existing risk factor for substance abuse? A long-term prospective study is needed to

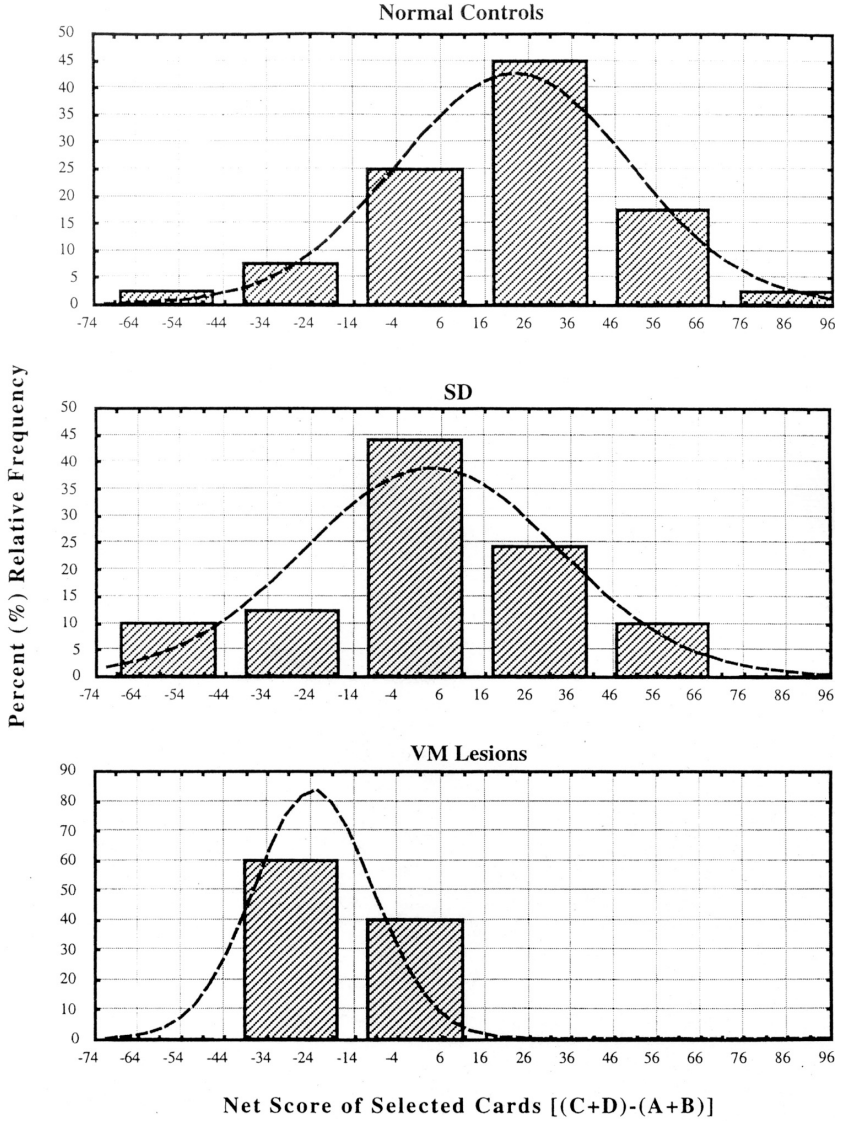

FIGURE 1.2. Normal distribution plots from the three populations of normal controls, substance dependents (SDs), and patients with VM lesions. The figure represents a distribution of the net score of selected cards (the difference between the total number of cards chosen from the advantageous decks [C′ + D′] minus the total number chosen from the disadvantages decks [A′ + B′] and the frequency of occurrence (%) of each score in the sample (Bechara *et al*., 2001, p. 384).

answer this question. Measures of VMPC functioning in children of alcoholics and matched control groups should be obtained and followed over the long term. A number of longitudinal studies that are part of the Collaborative Study on the Genetics of Alcoholism (COGA) project, including the studies by Schuckit (1998) and his collaborators, investigate high and low responsivity to alcohol in young people. Several interesting questions could be addressed if the VMPC measures had been obtained. Is a dysfunctional VMPC related to type 2, early-onset alcoholism as distinguished from type 1, adult-onset alcoholism? It is unfortunate that such information was not available in the Bechara *et al*. (2001) study.

Mazas *et al*. (2000) used a variation of Bechara *et al*.'s (2001) gambling task to study young (18–25 years old) noninstitutionalized participants obtained through media advertisements. Four groups were studied: (1) a group meeting the DSM criteria for antisocial personality (ASP) with no alcohol dependence (AD); (2) a subgroup of ASP subjects who also had AD; (3) a third group meeting the criteria for AD with no ASP; and (4) a control group of participants with neither AD nor ASP. Screening of a large pool of applicants was required using extensive exclusionary criteria before the desired samples were obtained. Participants were paid for their services.

Results showed that ASP participants manifested a similar disadvantageous bias in the gambling task as individuals suffering from VMPC lesions. They favored the initial relatively large reward despite long-term losses over the initial small reward but long-term profit. Noninstitutionalized non-ASP young men diagnosed as AD who were binge drinkers, i.e. drinking more than five drinks/occasion, showed a similar disadvantageous bias as individuals classified as ASP. The relatively small number of participants in the AD group and consequent lack of statistical power may be responsible for the absence of a significant difference between AD non-ASP participants and control subjects. The AD without ASP subgroup showed a disadvantageous bias somewhat midway between the ASP and control participants. A correlational analysis indicated that, over all groups, a higher IQ and drinking smaller quantities of alcohol per occasion (not binge drinking) were associated with a more advantageous decision bias. Frequency of drinking was not significantly correlated with a disadvantageous bias.

A characteristic of the AD and ASP participants distinguishing them from Bechara *et al*.'s (2001) VMPC lesion patients was their tendency to show a learning trend over trial blocks, shifting toward a more advantageous reward bias with trials. Mazas *et al*. (2000) studied a relatively small, noninstitutionalized community sample with limited statistical power, and had no measures of brain structure or function. Nevertheless, their results are striking despite limitations in the size and heterogeneity of their sample.

Fein *et al*. (2004) compared a community sample of alcoholics averaging 6.6 years of abstinence who had been abstinent at least 6 months and a control group of social drinkers. Performance on the gambling task was significantly impaired in the abstinent alcoholics as compared to control participants. A number of interesting problems are posed by the results, assuming that the gambling task impairment reflects the presence of VMPC damage: (1) is there a threshold of damage above which loss of control is inevitable; (2) are there compensatory brain changes that permit effective decision making despite VMPC damage; and (3) how does gambling task performance and VMPC status compare in alcoholics who relapse as compared to those who maintain abstinence? In a more recent study, Fein *et al*. (2006) found that gambling task performance of a group of young adults who were alcohol-dependent but treatment-naive did not differ significantly from control subjects. Their poor decision making was limited to the consumption of alcohol.

Evidence of prefrontal damage in a noninstitutionalized community sample of ASP and SD individuals has been obtained by Raine *et al*. (2000). Reduced prefrontal gray matter volume was found in a community sample of volunteers scoring high on an ASP scale as compared to individuals with substance dependence, including alcoholism. The latter were also higher in ASP as compared to the control group, and displayed diminished volume in prefrontal gray matter compared to the control group. More recently, Laakso *et al*. (2002) found that only the duration of severe alcohol dependence in type 2 alcoholics was associated with volume deficits in the dorsolateral and orbitofrontal cortices of violent ASP men. No significant relationship was found between volume deficits and degree of sociopathy.

Predicting Relapse

Bauer (2001) has provided striking evidence that brain dysfunction is a major proximal cause of alcoholism and relapse from alcoholism treatment. Patients were carefully tracked for 6 months posttreatment with unpredictable visits once or twice a week to assess relapse or continued abstinence. Breathalyzer and urine tests were obtained from patients, as well as verbal reports from patients and their collaterals.

A battery of measures was employed to predict whether individuals remained completely abstinent or relapsed during the 6-month interval. After approximately 3 months of posttreatment abstinence, quantitative measures of electroencephalogram (EEG) power were obtained from 15 different electrode sites. Demographic, medical, alcohol, and drug use data were also obtained, as well as measures of anxiety, depression, and IQ. Subgroups formed on the basis of DSM-IIIR dependence diagnoses of alcohol, cocaine, alcohol and cocaine, or opioid/polydrug dependence were analyzed.

Results showed that 6 months posttreatment, relapsed and nonrelapsed patients did not differ significantly from each other in baseline depression, trait anxiety, or alcohol and drug dependence. Relapsed patients as compared to those who remained abstinent 6 months posttreatment differed significantly on two background measures: conduct disorder and estimated IQ scores. Quantitative analyses of EEG frequency bands of interest showed significantly greater high-frequency beta power in relapsed patients. Its source was localized primarily in the orbitofrontal cortex.

Bauer (2001) also found that fast EEG beta frequency power was a significantly better predictor of relapse than a clinical questionnaire including measures such as alcohol and drug use history, conduct disorder, and a family history of alcoholism. Bauer's study provides further evidence that alcoholism is a disease involving brain dysfunction. It also provides a practical measure, EEG, which is a superior predictor of the risk of alcoholism and relapse from treatment than standard clinical measures.

Saletu-Zyhlarz *et al*. (2004) have replicated Bauer's (2001) study, adding a control group of normal patients. Patients who relapsed showed

greater high-frequency beta power at baseline than patients who abstained and control patients. Relapse-prone patients showed marked hyperarousal of the central nervous system. Quantitative EEG measures may serve as a prognostic measure of this "bad habit".

Denial

Stuss *et al*. (1992) note another important sign induced by frontal lobe damage: denial, a form of anosognosia, i.e. lack of awareness of one's illness. Aspects of denial are discussed in Chapter 5. This important problem has been unduly neglected and subjected to misleading psychodynamic and cognitive folk interpretations, for example, by Edwards *et al*. (2003), and ridicule by some revisionists. According to Peele *et al*. (2000),

> The disease-concept assertion that denial characterizes and is a major symptom of alcoholism is just that: an assertion and a particularly asinine one at that. Everyday experience will show this: a great many alcoholics go to AA and 12-step treatment for help (in itself an admission of a problem), yet many of them continue to drink abusively, often while attending AA and working the steps. Are they in denial? Another problem with the concept of denial is that it is useless as a diagnostic symptom. For, even if everyone who is an alcoholic is in denial about their problem (which, as we've just seen, isn't true), those who are not alcoholic will also deny that they are alcoholic. Thus reliance upon denial as a diagnostic symptom undoubtedly leads to many false positives. It's also worth noting that denial is a Catch-22 accusation: if you admit that you're an alcoholic, you're an alcoholic; if you deny that you're an alcoholic, you're in denial — strong evidence that you are an alcoholic. Either way you lose. Just as many innocent people lost the last time denial of a charge was accepted as evidence of its truth — at witchcraft trials in the Middle Ages. [p. 38]

Miller (2001) asserts,

> It has always been a puzzle to me how we in the United States fell into the particular ideological model that so dominated the treatment of

substance use disorders for much of the latter half of the 20th century. There is, of course, the familiar debate as to whether these behavioral disorders are properly characterized as a "disease," but that is not really the crux of the matter.

Central to most U.S. addiction treatment programs from the 1960s until the 1990s was the notion of *confrontation*. It was assumed, implicitly or explicitly, that people with these disorders were somehow uniquely incapable of comprehending or accepting the nature of their condition, that they were literally unable to see reality. The psychodynamic concept of *denial* as an unconscious ego-defense mechanism was invoked to explain this incapacity, and it was widely accepted that pathological denial was inherent in, and even diagnostic of, alcoholism and drug addiction. Indeed, this innate denial came to be viewed as the principal obstacle to treatment and recovery. . . . [p. x]

It was a house of cards. There is not and never has been scientific evidence to support the belief that people with subsance use disorders show abnormally high levels of primitive defense mechanisms such as denial. Studies instead reveal a heterogeneity of personality that parallels the general population. Prevention and treatment approaches based on education and confrontation have an abysmal track record when it comes to behavior change. Randomized clinical trials show little benefit from increasing the length or intensity of such treatment. If anything, confrontational tactics tend to elicit defensiveness, increase resistance, and decrease the likelihood of retention and constructive behavior change. [p. xi]

Substance use is fundamentally a motivational issue. The pharmacology of drugs of abuse involves motivation. . . . [p. xii]

Miller's comments are a mendacious characterization of current traditional Minnesota Model treatment programs. He ignores the research studies demonstrating that duration and intensity of treatment are significant factors of recovery (McClellan *et al*., 1993), and the research on anosognosia (i.e. denial as a consequence of brain damage). Miller appears to be unfamiliar with the research on denial and with the standardized validated measures of denial for alcoholism and other serious

diseases. The same may be said for Taleff (1997). When it comes to propaganda, "ignorance is strength."

Contrary to the assertions of revisionists such as Peele and Miller, there are standardized tests for the assessment of denial and evidence suggesting that denial is determined at least in part by damage to the prefrontal cortex. It is a form of anosognosia, a lack of awareness of one's illness, a condition by no means peculiar to alcoholism. Denial as a neuropsychological deficit due to damage to the prefrontal cortex has been known for some time (Tarter *et al*., 1984). Its implications for alcoholism treatment have also been considered (Duffy, 1995). A validated standardized form of a denial rating scale is available (Newsome & Ditzler, 1993). Ward and Rothaus (1991) conducted a factor analysis of the responses of 200 male alcoholics, providing a reliable distinction between the lack of awareness of signs and symptoms (denial) vs. the rationalization or justification of excessive drinking. Examples of denial items and their answers are as follows: – "I can never be a moderate drinker", + "I can drink when I want and stop when I want", – "Alcohol controls my life". Rationalization items are as follows: + "I drink to enjoy life", + "I drink because people do me wrong", + "Nagging causes me to drink". The lack of scholarship on the part of Peele *et al*. (2000) and Miller (2001) is inexcusable and cannot be denied.

Rinn *et al*. (2002) have demonstrated that denial of symptoms by alcoholics is significantly related to impairment of their executive functions such as planning and problem solving, as measured by the Wisconsin Card Sorting Test and the Clock Drawing Test. Patients high in denial also showed significantly poorer performance on Logical Memory, Logical Memory–Delayed, and Visual Reproduction subtests of the Wechstar Memory Scale–Revised compared to patients low in denial. Unfortunately, leading textbooks on alcoholism treatment written for the helping professions (Edwards *et al*., 2003; Hester & Miller, 2003) fail to provide the above important information on denial as a form of anosognosia and the availability of standardized measures for its assessment. The problem of denial will be considered further in Chapter 5 in relation to harm reduction and treatment self-selection.

MEOS: A System Affected by Excessive Alcohol Consumption

A progressive dialectical change occurs in many problem drinkers and alcoholics. It is caused by the action of alcohol on the nervous system, exacerbated by increased rapidity of alcohol metabolism due to induction of the microsomal ethanol oxidizing system (MEOS) of the liver. The MEOS "backup" system operates in chronic heavy drinkers when they consume large amounts of alcohol on a given occasion. Induction of the MEOS greatly increases tolerance by increasing the rate at which the liver oxidizes alcohol. A lower blood alcohol level is thereby maintained than would be the case in a social drinker. Induction of the MEOS permits greater consumption of alcohol on a given occasion, leading to increased brain damage and sickness behavior.

Alcoholism, Stress, and the Immune System

Some forms of extreme stress can make you sick, as can excessive alcohol consumption. Nonspecific effects seen after an infection are also observed following some forms of stress. Isolation stress in rats induces an increase in fever and sickness behavior as a result of the activation of macrophages, the first-stage nonspecific immune response (Maier & Watkins, 1998; Maier *et al*., 1994; Segerstrom & Miller, 2004). There is cross-sensitization between some forms of stress and infection for up to 10 days. Such stress may be an antigen in the sense that it activates the immune system. Alcohol is also an antigen that activates the immune system.

The brain and central nervous system (CNS) as a whole is not an isolated system; rather, it is intimately related in an interactive fashion with the neuroendocrine and immune systems, among others. Many of the negative effects of excessive alcohol consumption are the consequence of its effects on the latter systems. Excessive alcohol consumption is a risk factor for infectious diseases, cancer, and noninfectious chronic diseases such as cardiovascular disease. In the "eyes" of the immune system, alcohol is one more antigen. Dai, Thavundayil, and Gianoulakis (2002) demonstrated with college student participants that mild stress induced by a mathematics exam activated the beta-endorphin system. However,

one alcohol drink prior to the stress situation attenuated its effect, although there was an interaction with family history of alcoholism. The attenuating effect was greater in students who had a low risk of alcoholism (i.e. no family history of alcoholism) as compared to students who had a positive history.

Increased morbidity and mortality following chronic excessive alcohol consumption are not merely estimates based on correlational studies. Causal mechanisms at the level of components of the immune system and microbiology are being delineated that explain the relationship between alcoholism and morbidity/mortality. Alcohol directly suppresses natural killer (NK) cells, thereby increasing the risk of malignancies, as shown by Ben-Eliyahu *et al*. (1996). In a striking set of rat experiments, Ben-Eliyahu *et al*. demonstrated that an acute dose of alcohol administered during a critical time window of 24 hours during metastasis of cancer cells suppresses NK cell activity, promoting the development of cancer; chronic heavy drinking would have a comparable effect. Ben-Eliyahu *et al*.'s experiments provide evidence of the biological mechanism whereby alcohol increases the risk of certain cancers and a variety of infectious diseases. Liver disease, bacterial pneumonia, pulmonary tuberculosis, HIV autoimmune disease, cardiovascular disease, and various kinds of cancer are affected by the suppressive effect of alcohol on the nonspecific immune system, particularly NK cells. At the same time, alcohol may induce inflammatory cytokines, contributing to sickness behavior and brain damage. Ben-Eliyahu *et al*. (1991) have also shown that acute intense stress induced in rats by forcing them to swim in a water tank with a weight on their tail decreases NK cytoxicity.

Reichenberg *et al*. (2001), in a double-blind experiment, administered a small dose of endotoxin, an antigen, to normal healthy young men eliciting an immunological response. A neutral vehicle was administered to the same participants in a different session. Every few hours within each session, a variety of physiological measures were obtained as well as ratings of mood, anxiety, sense of well-being, and tests of memory and executive processes. Reichenberg *et al*. found that inflammatory cytokines, the kind normally activated by alcohol, produced an increase in anxiety and depression at different stages following administration of

the endotoxin. There were no reports of sickness behavior. A significant decrease in declarative memory occurred, both verbal and nonverbal, for as long as 10 hours after administration of the endotoxin. Executive functions were not affected by the low-level endotoxin administered. A partial correlation analysis indicated that cytokines and cortisol were independently correlated with depressed mood; however, only cytokines remained significantly correlated with increased anxiety and memory impairment. Since excessive alcohol consumption acts like an antigen and the immune system responds to it by secreting inflammatory cytokines and suppressing NK cells, excessive alcohol consumption leads to similar deleterious consequences, namely, a variety of sickness behaviors and brain damage.

Conclusion

Alcoholism is a noninfectious chronic disease, and must be recognized and treated as such. It is a disease of the whole person as well as the parts, systems, circuits, and cells. Alcohol not only affects behavior, the brain, and the liver; it also activates the immune system, acting much like an antigen inducing inflammatory cytokines that may produce sickness behavior, malaise, anxiety, and depressed mood, while inhibiting NK cells and contributing to brain damage. These are sound reasons for following Sir William Osler's admonition to treat the whole person and not simply features of the disease (Bean & Bean, 1950).

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2

Alcoholism Treatments and Mistreatments

Ignorance Is Strength

George Orwell (1949/1950), in his chilling description of a future authoritarian government, *1984*, describes the Ministry of Propaganda as the Ministry of Truth. Its brainwashing principles include "Truth is falsity", "Ignorance is strength", and "Whoever controls the present controls the past as well as the future."

Miller *et al.* (1995) state,

> [W]hat conclusions seem warranted from the cumulative evidence of these 211 controlled trials? In an earlier review, Miller and Hester (1986) . . . observed that although the scientific literature points to a list of treatment approaches with reasonable evidence of positive benefit, this list overlaps little, if at all, with those components commonly employed in U.S. alcoholism treatment programs. The same pattern is clearly evident in this review. Indeed, one must read halfway down Table 2.4 [not shown here] before encountering the first modality (disulfiram) with anything like common usage as a component of standard practice at least in the United States. Instead, a relatively predictable combination of elements has characterized the generic "Minnesota model" program that continues to dominate American addictions treatment: a milieu advocating a spiritual twelve-step (AA) [Alcoholics Anonymous] philosophy, typically augmented with group

> psychotherapy, educational lectures and films, and relatively unspecific general alcoholism counseling, often of a confrontational nature (Cook, 1988). Some programs have added components such as relaxation training, CENAPS-model relapse counseling, and family therapy. . . . To fill in the complete set of treatment methods with the least evidence of effectiveness . . . one need add only metronidazole, antianxiety medication, videotape self-confrontation, psychedelic medication, hypnosis. . . . [p. 32]

Miller *et al.* (1995) continue:

> The negative correlation between scientific evidence and application in standard practice remains striking, and could hardly be larger if one intentionally constructed treatment programs from those approaches with the *least* evidence of efficacy. Such a gap between science and practice will not be reduced without some disciplined and demanding changes. Clinicians, like scientists, must be willing to test their cherished assumptions against hard data and to relinquish views and practices that do not stand up to the test of evidence. [p. 33]

The above statement epitomizes Orwell's foreboding propaganda tool, "Ignorance is strength", with a heavy lard of intellectual arrogance. Although Miller and other revisionists have PhD degrees and the majority of alcoholism counselors do not, an advanced degree is no guarantee of integrity or scholarship. Lay people forming the AA fellowship, the 12-step approach adopted by the majority of alcoholism counselors, serendipitously hit upon procedures that are in harmony with principles of psychobiology (see Chapter 4); such principles are foreign to cognitive behavior therapy (CBT). A fundamental shortcoming of cognitive behavior therapists' theory and practice approach to alcoholism is that they are only "skin deep": they ignore psychobiology and behavioral neuroscience. It might also be noted that Miller, Hester, Marlatt, and other revisionists have a conflict of interest. They may have private clinics, provide frequent workshops and lectures for a fee, or obtain large federal grants for research institutes, and receive endowed chairs as a consequence. These clinical psychologists profit from their promotion of the

purported advantages of their clinical approach, which is ostensibly founded upon scientific principles developed in the laboratory.

Miller *et al.* (1995) misrepresent the characteristics of Minnesota Model treatment programs, including the nature of confrontations (see Pita, 2004). Miller, in the above quotation as well as in his other literature reviews, misrepresents or fails to report the treatment outcomes for programs in the United States, Australia, and Germany that yield higher treatment outcome rates of abstinence than his favored CBT treatments. It must be remembered that Miller's CBT treatment outcomes are derived from small efficacy studies. Although Miller and associates randomized participants into treatment and control or comparison groups, these studies lack external validity (i.e. generalizability) because of their numerous exclusionary criteria and their selective samples of volunteers who were often procured through advertisements and paid for their services. Miller's studies do not have the characteristics of Sir Bradford Hill's randomized controlled trial (RCT) developed in experimental medicine: external as well as internal validity. Hill's RCT will be described later in this chapter.

We have to put the lie to Miller and other revisionists, and present some of the highly successful outcomes of traditional treatments and the one laboratory-based treatment — Schick Shadel aversion conditioning — with the highest success rate of all (Maltzman, 2000). It is one that has never been reviewed by Miller and other revisionists or is misrepresented (Miller *et al.*, 2001). A description of how an RCT ought to be conducted will be presented, along with the obvious shortcomings in Miller's highly self-rated but fundamentally flawed RCTs. Examples of naturalistic studies of traditional treatment programs with results superior to those of Miller's and other CBT studies are those reported by Harrison *et al.* (1991), Küfner and Feuerlein (1989), Laundergan (1982), and Stinchfield and Owen (1998), in addition to private inpatient and outpatient substance abuse treatment programs studied by McLellan *et al.* (1993). Moreover, there are the laboratory-based treatments conducted in Schick Shadel hospitals using aversion conditioning (Smith & Frawley, 1993; Smith *et al.*, 1991, 1997), studies conducted in Australia by D. I. Smith (1985, 1986) that show highly successful outcomes for a small residential program, and the impact of AA and social resources on long-term

recovery (e.g. Humphreys *et al.*, 1997). The longest follow-up of all, 60 years, shows that two variables best predicted sustained abstinence: prior alcohol dependence and involvement in AA (Vaillant, 2003).

Treatment Programs That Work

Importance of Aftercare

Feuerlein and Küfner (1989) as well as Küfner and Feuerlein (1989) reported a major study of alcoholism treatment programs in Germany. Twenty-one different traditional clinical treatment facilities contributed 1410 participants to the study. Programs were not 12-step–oriented but encouraged aftercare, primarily 12-step group meetings. None of the facilities employed a behavior therapy approach.

Eighty-one percent of the original sample participated in a follow-up interview 48 months after treatment. A 6-month evaluation window was employed. Results showed that 66% were abstinent, 65% of the men and 70% of the women; while 4% engaged in controlled drinking, 4% of the males and 2% of the females. Of the patients who were abstinent during the first 6 months of the follow-up, 71% were continuously abstinent for the full 48 months. Only 3% (two patients) of those classified as controlled drinkers in the first 6-month interval continued controlled drinking for the entire 48-month follow-up, demonstrating that controlled drinking is not a stable state as compared to abstinence.

Abstinence rates in relation to attendance at self-help groups, primarily AA, were 72% for patients who attended meetings regularly, 48% for those with irregular attendance, and 51% for those with no attendance. Of those patients who were abstinent during the entire first 6-month period and who attended self-help meetings on a regular basis during the second 6-month interval, 84% were abstinent in the third 6-month interval. Participants attending irregularly or not at all during the second 6-month interval had an abstinence rate of 75% in the third 6-month period, a significantly lower rate than that of regular attendees. A similar analysis was conducted among patients who relapsed during the first 6-month interval and who did or did not attend self-help meetings regularly during the second 6-month interval; in the third 6-month

interval, 49% of regular attendees were abstinent as compared to 21% of those who attended on an irregular basis or never.

Küfner and Feuerlein's (1989) analysis suggests that there is a causal relationship, not simply a correlation, between self-help meeting attendance and remission. Fiorentine and Hillhouse (2000a, 2000b) suggest that it is the belief of the person dependent on alcohol and other drugs that they are incapable of controlling their drinking that contributes to abstinence, along with regular attendance at AA meetings.

The relatively high treatment outcome success rate reported by Küfner and Feuerlein (1989) is not due to chance. It is a treatment program that sounds much like the traditional Minnesota Model, which treats the whole person rather than just features of the "bad habit":

> The following treatment goals can generally be identified: reduction of the alcohol (drug) related problems; development of psycho-social competence in vocational and leisure behavior and in interpersonal contacts; personal autonomy in life. Abstinence from alcohol and other drugs with high abuse potentials are seen as a precondition in the realization of these goals. In consequence abstinence is generally accepted among German therapists. So-called controlled drinking has not been established as a therapeutic goal.
>
> These treatment goals cannot be realized at once. Six steps have been formulated:
>
> (1) to recognize that things must change
> (2) to accept the need to be helped
> (3) to accept the offered help
> (4) to accept the status of an alcoholic
> (5) to accept the goal of lifelong abstinence
> (6) to accept the goals of a general change in behaviour. [Feuerlein, 1990, p. 355]

German treatment facilities vary in the details of the approaches used to attain their commonly accepted treatment goals. Characteristics of their general clinical approach suggest that treatment facilities in the USA, particularly CBT-oriented programs, have much to learn from the

German facilities. An important characteristic of German treatment facilities is that they provide treatment programs of greater duration — varying from 6 weeks to 6 months — than those in the USA, and obtain more varied and extensive assessments:

> The therapist's approach to assessment varies in different centres, particularly in the first phase of treatment. The scope and purpose of the contact phase may be characterized by identifying the alcohol and individual problems of the patient. What is the present state of the patient and what has to be done first?
>
> The clinical approach involves: intensive, repeated interviews, physical (including neurological) examination, psychological tests, laboratory tests, neurophysiological (EEG, EMG) and neuroradiological examinations. Contacts with family members or other significant persons, with family doctors, counselling facilities and social workers is included. . . . [Feuerlein, 1990, p. 355]

Note the breadth of the assessment. It truly is a biopsychosocial approach to the treatment of the whole alcoholic person and their family.

Comprehensive Assessment and Treatment Outcome Research (CATOR) Registry

Another inexcusable lack of scholarship on the part of revisionists such as Miller and Marlatt, and of textbook writers using them as their primary source, is their failure to consider the Comprehensive Assessment and Treatment Outcome Research (CATOR) registry. It is the largest available database of alcoholism treatment outcome results. The CATOR registry was founded by treatment programs in the Minneapolis–St. Paul area collaborating in the design of data-collection instruments for use in intake, discharge, and study procedures. They collected data on more than 50 000 adults from 80 programs in 29 states. Treatment outcome data from the participating programs were aggregated and analyzed.

> Results from a sample of 9,000 participants from inpatient programs and 1,000 participants from outpatient programs have been presented

> by Harrison *et al.* (1991). Numbers of cases studied, success rates, and sophistication of the detailed analyses of pertinent variables far exceed any reported outcomes for controlled drinking training efficacy or brief intervention studies. Nevertheless, abstinence results reported by CATOR are not cited by Marlatt *et al.* (1993) or Hester and Miller (1995) and other reviewers. . . . These studies are not mentioned much less considered effective ways of producing harm reduction. [Maltzman, 2000, p. 224]

The following are some of the results from the CATOR registry that Harrison *et al.* (1991) reported for a follow-up of approximately 1900 completers of inpatient treatment interviewed at 6 and 12 months following treatment: 72% were abstinent from alcohol and all other drugs for the entire first 6 months, 63% were abstinent for the entire year, 87% were abstinent for at least 6 of the 12 months, and 75% were either totally abstinent or suffered a relatively brief relapse. The outcome varied with polydrug use and the type of drug used: 71% of inpatients with alcohol dependence only were abstinent the entire year, 52% of inpatients with marijuana misuse with or without alcohol misuse were abstinent for the year, and 50% of inpatients with cocaine misuse regardless of other drug misuse were abstinent for the year.

Outpatient results for 900 treatment completers indicated that 83% were abstinent from alcohol and all other drugs at the 6-month follow-up; 75% were abstinent for the entire 12 months. Ninety percent of those abstinent at 6 months were abstinent for all 12 months. Higher outcome abstinence rates among outpatients than among inpatients at 1 year follow-up were predicted from earlier CATOR findings based on the more favorable prognosis of outpatients due to their greater social stability, less polydrug use, and less severe symptomology compared to inpatients.

A consistent finding of the CATOR registry is that continued involvement in support groups such as AA, Cocaine Anonymous (CA), and Narcotics Anonymous (NA) following treatment was significantly related to recovery. Hoffmann and Miller (1993) reported results for a group of inpatient treatment completers. Of those who did not attend support group meetings (n = 519), 51% were abstinent for the entire first year. Of those who stopped attending meetings (n = 186), 41% maintained

remission; when attendance was once a month or less (n = 136), 54% were abstinent; 62% were abstinent for the year when attending several times a month (n = 115); and 76% were abstinent for the entire year when attending support groups on a weekly basis (n = 943). The overall average abstinence rate from all drugs was 63%. Clearly, significant differences in remission rates are related to support group attendance.

The above results extracted from the CATOR database, which were derived primarily from Minnesota Model treatment programs, contradict the assertion by Miller *et al.* (1995) that there is no good evidence supporting traditional alcoholism treatment. The contradiction is an indictment of Miller *et al.*'s lack of scholarship. An even more serious indictment is that similar CATOR results as those above were described by Laundergan (1993) in a chapter in a book entitled *Research on Alcoholics Anonymous,* edited by McCrady and Miller. Either Miller read the chapter in his book written by Laundergan and is suppressing the information on Minnesota Model outcomes, or he did not bother to read a chapter in the book he edited. In either case, the information in the book he coedited calls Miller's scholarly integrity into question.

In his ongoing apologia for "moderate" drinking with no assessment of brain structure and function, Miller *et al.* (2001) ask a seemingly simple question: how effective is alcoholism treatment? He and his collaborators discuss the complexity of the problem, noting that legislators, reporters, and others (including prospective patients) ask this seemingly simple question. Miller *et al.* (2001) provide an answer to this purportedly common question:

> It is a reasonable question, despite the complexities. People with a life-threatening diagnosis often want to know their chance for survival and recovery. What is being asked for is an average, a representative sense of treatment outcomes, rather than the best possible scenario. Though there may be substantial variability across populations, clients, programs, therapists and time, the question asks for a reasonable estimate of typical outcomes. [p. 212]

From the outset, the question is highly ambiguous, thus making the answer necessarily misleading. The term "alcoholism" as used by

Miller *et al.* (2001) purports to correspond to Jellinek's (1960) usage. It does not. As used by Miller *et al.*, "alcoholism" refers to drinking problems; in terms of the *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV), it would include preclinical people who have an occasional problem as well as people suffering from alcohol abuse and alcohol dependence. Miller *et al.* (2001) fail to note that Jellinek distinguishes between different kinds of alcoholics: those who suffer from alcohol dependence with loss of control (i.e. gamma alcoholics), and those with alcohol abuse (i.e. alpha and beta alcoholics). According to Jellinek, gamma alcoholics are the ones suffering from the disease of alcoholism. The percentage of drinkers showing structural and functional brain damage increases in a nonlinear fashion from light drinkers to alcoholics (Cala, 1987). Loss of control is the pathognomic sign differentiating gamma alcoholics from alpha and beta alcoholics, according to Jellinek. Combining the outcome results for all of these groups is misleading because of the qualitative differences among them as emphasized by Jellinek — and ignored by Miller *et al.* (2001). Likewise, Hoffmann and Hoffmann (2003) demonstrate that alcohol dependence as defined by DSM-IV is a distinct and internally consistent diagnostic category; it is differentiable from abuse as well as from preclinical categories. Therefore, "these distinct diagnostic groups should not be mixed together in treatment outcomes research" (Hoffmann & Hoffmann, 2003, p. 301). The following study by McLellan *et al.* (1993) highlights the misleading and potentially harmful nature of Miller's "representative" sense or index of treatment outcome.

Intensity of Treatment Services and Treatment Outcome

McLellan *et al*. (1993) conducted an informative study of 198 patients in four private treatment facilities, two inpatient and two outpatient. Participants were referred for treatment for alcohol and/or cocaine dependence by their employee assistance program (EAP).

> Patient status was measured at both admission to treatment and again at six-months post discharge using the Addiction Severity Index (ASI). . . . The ASI is a one-hour technician administered, structured interview that

> measures the lifetime and recent (past 30 days) severity of problems in seven areas commonly affected among alcohol and drug-dependent individuals. These include medical status, employment and self support, alcohol use, drug use, legal status, family and social relationships and psychiatric symptoms. In each of these areas items measuring the severity of the problem during the previous 30 days are combined through an equally weighted formula, into a composite or factor score.
>
> While the ASI measures the nature and severity of treatment problems presented by the patient at the start of treatment and later at follow-up, we have developed a measure of the treatments received by patients in each of the same seven problem areas covered by the ASI. The Treatment Services Review (TSR) . . . is also a technician administered interview that takes five minutes to complete and is administered on a weekly basis . . . during the course of treatment. This interview can be done in person or over the phone and records the number of professional services (specialized therapy or treatment sessions, medications, etc.) or discussion sessions (group or individual counseling) that the patient receives in each of the seven problem areas during the course of the previous week. The TSR measures both the services that are provided within the program as well as those that are offered through referral. As in the ASI, the total amount of treatment activity received by the patient in each problem area is summarized into a composite score. [p. 246f]

The majority of the patients were employed, insured, African-American males with significant levels of multiple drug use (primarily alcohol and cocaine), family disruptions, and psychiatric problems. Although they were not randomly assigned to treatments, there were no major differences among patients admitted to the four facilities. Treatment facilities employed Minnesota Model programs. However, there were significant differences in the TSR scores across the four facilities. Inpatient treatments offered significantly more alcohol and drug counseling sessions and medical services. The two inpatient services differed significantly from each other in terms of services provided; the two outpatient services also differed from each other. Inpatient treatment and services were for 28 days, whereas outpatient treatment was organized as a daily session for a recommended 6 weeks.

A 6-month follow-up with a 30-day evaluation window showed that dropout rates were higher for outpatients than inpatients. Difference scores in the ASI prior to treatment and 6 months after treatment showed a 74% improvement in alcohol use and 73% improvement in other drug use. There was a significant difference between problems, but not between levels of care: 59% of patients were abstinent from alcohol and 84% were abstinent from all other drug use at the 6-month follow-up. Abstinence from alcohol ranged from 45% to 78% across the four programs; abstinence from all other drugs ranged from 71% to 98%.

According to McLellan *et al.* (1993),

> . . . [T]here has been accumulating evidence that factors such as more severe dependence, poorer social and economic supports, and more severe psychiatric problems are generally related to worse performance during treatment and poorer outcomes following treatment. . . . In the present study, there were no systematic differences in any of these qualities among the patient samples admitted to the four programs. Thus, while these factors were related to outcome generally (averaged across the programs), they cannot explain the between program differences. . . . [T]he programs that provided the most services directed at a particular treatment problem generally showed the best outcomes in that problem area. Comparisons between programs within each of the two levels of care yielded even more specific information. For example, in the case of the two outpatient programs, the one that provided the most services in each problem area showed the best outcome in that area, in nine of eleven criteria measured. Similar results were seen for the two inpatient programs in seven of the eleven areas measured. [pp. 253–254]

Inpatient program #1 yielded an abstinence rate from alcohol of 78%, whereas inpatient program #2 had an abstinence rate of 63%. Outpatient programs #1 and #2 had abstinence rates of 51% and 45%, respectively. The average for all programs was 59%. These figures give the lie to revisionist claims that (1) inpatient and outpatient programs do not differ in effectiveness, and (2) intensity of treatment does not make a difference. We may also conclude from McLellan *et al.* (1993) that,

contrary to Miller *et al.* (2001), relying on aggregate measures of treatment outcome is an inaccurate and misleading practice.

Two Neglected Australian Studies

Another victim of Miller and colleagues' (e.g. Miller *et al.*, 1995) biased presentation of the alcoholism treatment outcome literature is a pair of studies conducted in Australia by D. I. Smith (1985, 1986). These studies have the features that Miller insists are needed in treatment outcome research: a matched comparison group and multiple measures of outcome for alcohol consumption and quality of life, among others. However, Smith's studies have one problem which apparently prevents their inclusion in Miller's exclusive club: they show that traditional 12-step–oriented treatments may be highly effective.

A Study of Women

Both studies by Smith (1985, 1986) used an index treatment group and a matched comparison group, one evaluating the treatment of women and the other of men. In the first study, Smith (1985) compared two groups of women: 43 patients from a small residential 12-step–oriented treatment program, and a matched nontreated comparison group of 35 women who were detoxified in the same center as the index treatment group but did not enter the index treatment program. The women in the two groups were initially matched on a large number of variables. The residential treatment program involved studying 1 of the 12 steps each morning 6 days/week followed by a work program until noon. Afternoons were free of organized activities, with the exception of AA meetings two afternoons/week. Reality therapy was also part of the program. Upon entering treatment, each patient signed a contract stating that she would pay A$42 for room and board in advance and would have to leave immediately if she consumed alcohol. Participants were encouraged to remain in treatment for a minimum of 6 weeks. Unfortunately, the number of noncompleters was not reported, although a footnote indicates that these patients were followed up and compared to the other groups; a report of these analyses was available upon request from the author.

However, in the early 1990s I wrote to Smith at the agency where he worked asking for the results of the additional analyses, but my letter was returned by the postal service. Smith no longer worked at the agency in question and no forwarding address was available.

Follow-up interviews were conducted approximately 15 months after treatment. Ninety percent of the treatment group were interviewed; two participants were deceased. Seventy-three percent of the comparison group were interviewed; one was deceased, and 17 refused to be interviewed. Significantly more women receiving the index treatment than the comparison group reported complete abstinence during the follow-up interval, 79% vs. 3%. Index treatment women in contrast to comparison group participants drank significantly less alcohol during the week prior to the follow-up interview and in a typical week. They had a significantly lower blood alcohol level at the time of the follow-up interview. Women receiving the index treatment were employed for more weeks in the 15-month follow-up period as well as during the 30 days prior to the interview, and had significantly fewer mental and physical problems than comparison women. Index treatment women had no drunk days on the job during the past 30 days in comparison to 3 drunk days for the comparison group.

Thirty-six percent of women in the index group stayed for the recommended 6 weeks. They had a significantly higher abstinence rate than the 32% who stayed for less than 6 weeks. Abstinence rates did not increase significantly for women staying longer than 6 weeks. Only one of the women in the index group who relapsed during the follow-up had stayed in residence for the full 6 weeks; in contrast, only one woman in the comparison group remained abstinent throughout the follow-up period.

A possible criticism of the quasi-experimental design of Smith's study is that women in the index treatment group were more motivated to change than the comparison group. They sought additional treatment following detoxification, whereas the comparison group did not. However, the information gathered by Smith contradicts the motivation hypothesis: 88% of the comparison group had received treatment some time during their drinking career, and 71% of the comparison women participated in at least one other treatment program during the follow-up

period. An additional 17% of the women participated in a treatment program in the year before their detoxification.

A Study of Men

Smith (1986) also reported on residential treatment effectiveness for men and a matched comparison group receiving only detoxification at baseline. There were 137 men in each group at baseline. Seventy-five percent of the men were interviewed approximately 15 months following treatment. Abstinence rates for the entire follow-up interval were 62% and 5% for the index treatment and comparison groups, respectively. As was true of the women, significant differences were found on a variety of quality of life (QoL) measures. Index treatment men were employed for 51 weeks in the follow-up period as compared to 33 weeks for the comparison group; dollars spent on alcohol during the follow-up period, A$6 and A$22 for the index treatment and comparison groups, respectively; and times drunk on the job during the previous month, 0.2 and 3.3 for the index treatment and comparison groups, respectively.

> Smith (1986) emphasizes that the positive results found in his studies of residential treatment do not necessarily generalize to all residential treatment programs. Smith's evaluation of treatment effectiveness for a 12-step oriented treatment program for men and for women as compared to matched comparison groups shows highly significant differences in a wide range of social adjustment variables and abstinence rate in favor of the traditional treatment. It does not follow that all traditional treatment programs are equally as effective. It does show that the program at Serenity Lodge was highly effective, contrary to the myth propogated by Miller, Hester, and others that there is no evidence that traditional treatment works. . . . [W]hy are residential treatment programs of the kind described not more common in view of their apparent effectiveness? Why are not federal funds supporting research on such projects rather than projects based on the shaky research base formed by behavior therapy studies? [Maltzman, 2000, pp. 208–209]

Oxford House

A communal housing setting, Oxford House, has been introduced in the United States by a group of professionals at DePaul University in Chicago, IL, in collaboration with a community-based organization. Oxford House was initiated in 1975. There are now approximately 1200 such democratically operated self-supporting homes in the United States (Jason *et al.*, 2006a, 2006b). Characteristics of the Oxford Houses are (1) they are democratically run; (2) members are responsible for all expenses; (3) members may live in a house as long as they wish, provided that they pay their share of the expenses and do not drink alcohol or use drugs. If they drink or use drugs, they are immediately expelled; (4) any individual may apply for admission and be interviewed; and (5) any group of 6–10 men or 6–10 women may apply for a charter. There are no mixed gender houses.

Jason *et al.* (2006b) conducted an RCT in which 150 individuals were assigned either to Oxford Houses or to usual aftercare, either outpatient treatment or self-help groups. At a 2-year follow-up, Oxford House participants were superior to the comparison group on a variety of measures including lower rates of substance use (31% vs. 64%) and criminality and a higher monthly income.

Undoubtedly, Oxford Houses contribute to the treatment of alcohol and drug abusers at a greatly reduced cost to society. However, much more detailed analyses of the characteristics of the individuals living in such homes are needed. For example, severity of dependence and problems of living need to be obtained with McClellan *et al.*'s (1993) ASI and TSR. The use of these measures would add considerably to the understanding of the effects obtained in residents and the characteristics of Oxford Houses that contribute to the positive effects obtained.

Schick Shadel Aversion Conditioning

Smith *et al.* (1991) matched 249 patients receiving aversion conditioning in a Schick Shadel hospital with patients from the CATOR registry. Aversion conditioning as employed at Schick Shadel hospitals is part of a multimodal treatment that includes, if necessary, detoxification

followed by 10 days of treatment involving alternate days of conditioning (either chemical aversion or faradic shock) and interviews under sodium pentothal. Daily group counseling is provided, as well as individual and family counseling. There is an individualized treatment and continuing aftercare plan, as well as educational programs on alcohol and drug abuse. A biobehavioral disease conception of alcoholism provides the theoretical basis for treatment. Maltzman (2000) describes the conditioning theory underlying the treatment procedure.

Not all participants at Schick Shadel hospitals receive chemical aversion conditioning. Older patients and those with medical conditions precluding nausea and vomiting, an integral part of chemical aversion conditioning, receive faradic aversion conditioning — electric shocks to the wrist — instead. Results obtained with faradic and chemical aversion conditioning were not differentiated in the present study, but they were in a later study (Smith *et al.*, 1997). "Results for faradic aversion conditioning are especially interesting because electric shock in the hands of other investigators (no pun intended) has generally been a failure (Cannon & Baker, 1981; Cannon, Baker & Wehl, 1981)" (Maltzman, 2000, p. 235).

A noteworthy aspect of the Schick Shadel program is that it consists of only 10 days of inpatient treatment, followed by a return after 1 month and again after 3 months for 2-day reinforcement treatments.

Results

Treatment evaluations were conducted by an independent team of investigators, in contrast to studies conducted by Miller and colleagues (1992) in which they designed the experiments and collected the data themselves. Results were continuous abstinence for the entire year by 79% of the Schick Shadel patients and 67% for the comparison group, a statistically significant difference. The high success rates for the aversion conditioning treatment provided by Schick Shadel and for traditional Minnesota Model treatment programs in the private sector are in striking contrast to the results reported by Miller *et al.* (1992) for RCTs. Success rates, defined in terms of abstinence, for Schick Shadel and Minnesota Model programs

were more than twice as high as those reported for Project MATCH (Matching Alcoholism Treatments to Client Heterogeneity) (Project MATCH Research Group, 1997). Results included dropouts as well as treatment completers. Results for completers only were 83% for all drugs and 89% for alcohol alone. Abstinence rates for the completers in the comparison group were 73% and 79% for all drugs and for alcohol only, respectively. Project MATCH results were for completers only.

The Community Reinforcement Approach

Miller and his colleagues (1995) rate the community reinforcement approach (CRA) to alcoholism treatment (Azrin, 1976; Hunt & Azrin, 1973) very high in their evaluation of treatment programs. Miller (1995b) wrote the forword to a book (Meyers & Smith, 1995) coauthored by his colleague Meyers, who had experience with the original studies of the CRA conducted by Azrin. Miller also coauthored several other articles and edited a book with Meyers; and included a chapter by Smith, Meyers, and Milford (2003) in the book on alcoholism treatment approaches edited by Hester and Miller (2003) that Miller dedicated to his colleague and friend, Meyers.

According to Miller (1995b),

> No longer is the CRA an unknown method. Several research teams are now actively studying this clinical approach. Training and research on CRA have spread not only throughout the United States, but as far as New Zealand, Poland, and the Netherlands. Major grant funding for CRA studies has been provided by the National Institute on Alcohol Abuse and Alcoholism, and the National Institute on Drug Abuse. It was included in *Broadening the Base of Treatment for Alcohol Problems*, a 1990 major report from the Institute of Medicine of the National Academy of Sciences. Many of the procedures included in the original CRA — motivational preparation, behavioral marital therapy, communication skills training, and stress management — have subsequently been shown separately in clinical trials to be important and effective elements in treatment. Yet the CRA itself remains an approach unfamiliar to many clinicians in the field. [p. viii]

Note that there are no citations for the research teams around the globe as well as in the USA studying CRA. Inclusion in the *Broadening the Base of Treatment for Alcohol Problems* report (1990) is not surprising, since Miller and Marlatt were involved in the preparation of the volume.

Hunt and Azrin (1973) adopted an operant learning theory approach to alcoholism treatment in their CRA. They first determined the natural deterrents to excessive alcohol consumption. Their theoretical approach suggested that people are deterred from drinking when their drinking interferes with social forms of reinforcement, when alcoholism is a "time out" from social satisfaction. Its model in operant theory is the community mental health approach. Hunt and Azrin assert:

> A realization that mental disorders result from forces operating in and by the community on the individual and suggests that treatment be conducted by rearranging these community influences on the patient in the community rather than a hospital. Examples of community based treatments include the home care program for schizophrenics. . . . [p. 92]

There is some truth to what is assumed by the operant approach. As we shall see in Chapter 4, there is a dialectical interaction between the neurochemical state of the brain, behavior, and the social environment. A reinforcing, supportive community can help the alcoholic. It can change the neurochemical state of their brain and behavior. This, we believe, is an important component in the success of the AA fellowship. However, to assume that a "mental disorder", including alcoholism, is caused by nothing but community influences is nothing but a bad habit, and flies in the face of overwhelming contradictory evidence (Goodwin *et al.*, 1974; Maltzman, 2000).

Hunt and Azrin's (1973) approach is ambitious: arrange a social environment which, in our terms, modifies the brain state of its residents. But, what might be successfully arranged in a sparsely populated rural town like Anna, IL (where the study was originally conducted), or a somewhat larger city such as Albuquerque, NM (where it was replicated), does not seem feasible in large cities such as New York, Los Angeles, Chicago, etc.

Clinical trials of the CRA (Azrin, 1976; Hunt & Azrin, 1973), which are the basis for its high rating by Miller *et al.* (1995), have limited ecological validity and are not readily generalizable to present-day large metropolitan areas. They were conducted in a sparsely populated rural area of Southern Illinois in the 1970s. Financial support was provided by the State of Illinois and the National Institute of Mental Health (NIMH). Such support enabled the research team to rent a tavern on Saturday nights to provide a nondrinking social club environment. An excellent idea, but one that could not be readily implemented by a typical public or private treatment facility, much less an individual alcoholism counselor, unless it received a large grant of some kind. Other components of the CRA are also not feasible (especially in large metropolitan areas); and lack citations and supporting evidence concerning locations, attendance, longevity, cost, etc. Another reason for the CRA's lack of ecological validity is that the traditional program it used as a control does not correspond to the current predominant treatment, the Minnesota Model.

Subjects for the clinical trials were obtained from a larger pool of inpatients at a state hospital, and were randomly assigned to either an experimental group receiving CRA treatment and traditional treatment or a second group of patients matched on seven different variables and receiving only the traditional treatment. The procedure of selecting patients from a larger pool, randomly assigning a group to an experimental condition, and then matching them to patients forming a control condition without considering the larger pool as part of their *N* is similar to the procedure used by Smith *et al.* (1991) in comparing their treatment outcome results from Schick Shadel hospitals with the CATOR registry. Miller *et al.* (2001) criticized the Schick Shadel study for using this procedure, but not the CRA. Miller applies a double standard and finds an excuse to criticize treatment programs with superior results than any of the CBT programs he promotes, including the results from the CRA studies which contain only 8–15 subjects/condition and do not give complete abstinence rates for a 1-year follow-up. Nevertheless, the CRA is among Miller's top ten rated studies (e.g. Miller & Hester, 1986; Miller *et al.*, 2001), whereas Schick Shadel and CATOR programs with far superior treatment outcomes

are unranked and — when mentioned by Miller *et al.* (2001) — misrepresented.

Components of the experimental and traditional program are described by Hunt and Azrin (1973) as follows: "The Community-Reinforcement program was designed to rearrange the vocational, family and social reinforcers of the alcoholic such that time-out from these reinforcers would occur if he began to drink" (p. 93). Components of the program included marital and family counseling, and social counseling designed to improve and increase the interactions with sober individuals; reinforcer-access counseling was another module, probably peculiar to the time and rural location. It helped individuals obtain a telephone or newspaper and transportation, since the rural area had no public transportation facilities. Other than the latter, it sounds like a Minnesota Model program. An add-on design was employed. The experimental group received the above modules as well as the standard hospital treatment available in the area, whereas the control group received only the hospital treatment consisting of 25 one-hour didactic sessions providing a description of the basic workings of AA, statistics on drinking and problems of alcoholics, illustrations of alcoholics' behavior, pathophysiological consequences of alcoholism, sexual disorders produced by alcoholism, and related topics. There were only eight participants in each of the two groups.

The hospital treatment used as a control condition does not represent a standard treatment program such as the Minnesota Model, which dominates the treatment field at present. Its modules are described in some detail elsewhere (Maltzman, 2000). Briefly, a survey of 13 Minnesota Model programs in the Midwest revealed the following modules: release planning, peer confrontations, work therapy, relapse prevention, relaxation training, didactics, individual therapy, AA groups, fourth step, fifth step, exercise, family therapy, group therapy, and assertiveness training.

For the CRA to have an independent identity and external validity (i.e. generalizability), it must be compared with a standard Minnesota Model treatment program and be demonstrated to work significantly more effectively than the Minnesota Model treatment in metropolitan areas as well as small rural areas. However, it would appear to be a waste

of federal funding to support research of the above kind assessing the external validity of the CRA. It is apparent that Minnesota Model programs with AA aftercare provide superior social resources necessary for long-term recovery than the CRA, which by its nature is limited to rural and small metropolitan areas.

Project MATCH: The US$27 Million Dollar 'Slice of Baloney'

There is good reason to be critical of Project MATCH:

1. Project MATCH was unnecessary.

McLellan *et al.* (1997) demonstrated that a systematic approach to matching within the services offered by multimodal Minnesota Model programs is possible. Earlier, McLachlan (1974) demonstrated successful matching of levels of directiveness of therapy with levels of antisocial personality: the lower the level of socialization, the greater the directiveness of therapy needed. Patients were also matched or mismatched with aftercare programs. A 12–16-month follow-up showed highly significant differences in treatment outcomes favoring matching. McLachlan conducted the study at the Donwood Institute in Toronto, Canada, a traditional disease-oriented treatment facility.

Matching is a difficult problem for the individual cognitive behavior therapist in private practice, but not for Minnesota Model programs. Fundamental shortcomings in CBT approaches to the treatment of alcoholism opened the "barn door", Project MATCH, for Miller and fellow ideologs to feed at the trough of public funding.

2. Project MATCH lacks generality (i.e. external validity).

Project MATCH invented a program, Twelve-Step Facilitation (TSF), that does not correspond to any treatment program existing in the community. Project MATCH should have randomly assigned participants to Minnesota Model programs such as Hazelden or to other programs in the CATOR registry, along with motivational enhancement therapy (MET) and CBT. Additional reasons for its lack of external validity are

that it excluded racial minorities, the disadvantaged socioeconomic class, and people with polydrug problems or dual diagnoses.

Given the inappropriate selection of treatment programs, its exclusionary criteria, and use of a single level of treatment intensity, the authors of Project MATCH were catering to managed care and their personal gain, not science. Matching within Minnesota Model programs was employed as needed at the discretion of counselors. However, McLellan and his colleagues demonstrated that matching can be conducted in a consistent fashion within such traditional programs. As previously noted, McLellan *et al.* (1993) found that of four treatment programs, two inpatient and two outpatient, treatment outcomes varied as a function of the intensity and duration of treatment received. A second study (McLellan *et al.*, 1997) employed a new sample of participants. One inpatient and one outpatient program employed a traditional Minnesota Model treatment program, emphasizing the 12 steps to attain sobriety, and provided group and individual counseling as well as AA referral. The other two programs, in addition, provided psychiatric and family therapy services. After placement in one of the programs, each patient was randomly assigned to a standard or a matched service. All patients received ASI scores, but the clinical director of the standard service was asked to treat patients in their usual standard fashion. Clinical directors of the matched services were asked to use the ASI results to match patients to additional services when their ASI results showed a significant problem in the areas of employment, family relations, or psychiatric problems. Patients in the matched group received three additional sessions with an appropriate professional in the area(s) needed. A significant problem requiring additional service was defined as an ASI composite score more than one standard deviation above the norm of their previous study of treatment outcomes from these four programs (McLellan *et al.*, 1993).

McLellan *et al.*'s (1997) findings are as follows:

> [M]atched patients received more services in the targeted areas, stayed in treatment longer, were more likely to complete treatment, and had better 6-month outcomes than did the standard-care patients treated in the same programs. Because an intent-to-treat design was used with random assignment and analyses were not confined only to treatment

> completers, we believe that the experimental intervention was causally associated with the differential outcomes. The findings also were consistent with the *a priori* predictions guiding the matching strategy. Finally, with regard to the importance and robustness of the findings, 2 points are important. First, this was a health services evaluation of treatment effectiveness under *real-world* conditions rather than a clinical trial of treatment efficacy under more controlled but less realistic conditions. It is traditional for clinical trials of efficacy to show substantial reductions in effect size when transferred to field conditions. These data indicate a statistically significant matching effect under field conditions. Second, the control group here was not a 'no treatment' condition. In fact, the control patients were treated in programs that had shown evidence of effectiveness. For these reasons, we believe the findings have potential practical value and impact for the contemporary treatment of addictions. [p. 733]

The authors noted that there are several limitations to the generality of these findings. Patients were all referred to treatment by their EAP; patient characteristics and services may have therefore differed from services provided in public facilities. Nevertheless, the results point the way to a systematic method of matching patients to needed services. Matching can be done *within* a multimodular Minnesota Model traditional treatment program, thereby improving treatment outcomes. Not only did McLellan *et al.* (1997) show that matching services within a traditional program is possible; they also showed that Project MATCH was unnecessary, poorly designed, and a waste of public funds. I believe Project MATCH was based upon the misrepresentation of the treatment literature by Hester and Miller (1989) and by co-opted government employees of the National Institute of Alcohol Abuse and Alcoholism (NIAAA).

3. Project MATCH claims to have internal validity.

Each of the programs followed a manual. However, purported internal validity does not guarantee good treatment. Manuals of the three MATCH treatments did not address sexual and/or physical abuse experienced by

patients — a common problem among people with chemical dependencies as reported, for example, by Maltzman and Schweiger (1991). The Minnesota Model treatment facility in which we conducted our research provided additional individualized treatment for patients who suffered sexual and/or physical abuse. A history of such abuse was discovered during the course of individual therapy with patients. Project MATCH ignored the important problem of physical and sexual abuse as a risk factor for alcoholism and for relapse (Rosen *et al*., 2002) because the manuals used did not provide information necessary for individualized treatment for the problem. They did not provide for matching where it was needed. Since CBT and MET represent treatments available in the community, these treatments and their RCTs lack important treatment services. They do not match treatments to the needs of patients, unlike Minnesota Model treatment programs such as Hazelden (Laundergan, 1982) and others that tailor their programs to the individual needs of patients rather than a manual which does not address the important problems of sexual and physical abuse.

Project MATCH treatments were designed to fit the needs of managed care (i.e. treatments as brief as possible), not to reach for external validity (i.e. duplicate the population and its problems found in most treatment facilities, outpatient and inpatient, and meet the special needs of patients such as those who have suffered sexual and physical abuse). If Project MATCH had been concerned with external validity and the advancement of science-based practice, the Project would have used a 28-day multimodular Minnesota Model treatment program rather than inventing the TSF, which does not correspond to any treatment in existence. A 28-day Minnesota Model–type program should have been compared with the 4 sessions of MET and 12 sessions of CBT used in Project MATCH. The differences in the number of sessions and in the duration of the program should be of no concern to the principal investigators of Project MATCH, since they apparently believe that intensity and duration of treatment do not matter — another one of the myths promoted by Hester and Miller (1995). Evidence contradicting this proposition is available (Küfner & Feuerlein, 1989; Monahan & Finney, 1996; Moos & Moos, 2003).

A major justification for conducting Project MATCH was that "one suit does not fit all." Individual differences in client characteristics must be matched by the treatment provided; traditional treatment, i.e. the Minnesota Model, purportedly does not. However, this premise for undertaking Project MATCH is false, as indicated above. Cognitivists appeal to the prestigious Institute of Medicine (IOM) (1990) report as a justification for Project MATCH. This is one more aspect of the scam: the relevant section on the IOM was probably written by Marlatt and/or Babor, both cognitivists and members of the committee preparing the Report.

Laundergan (1982), a sociologist, has written a book-length description of Hazelden's (one of the founders of the Minnesota Model) treatment program and an examination of outcomes for a sample of patients treated in the 1970s. As was true in my own limited experience with a Minnesota Model treatment program, Laundergan states that the treatment program at Hazelden is tailored to the needs of the individual. He reported treatment outcome results for a sample of approximately 1900 patients; approximately 50% were abstinent from alcohol and other drugs for an entire year following treatment. How does this old study compare to the Project MATCH results? There is no comparison!

Principal Project MATCH findings for outpatients are as follows:

1 year of abstinence: TSF, 24%; CBT, 15%; MET, 14%
Abstinence at 3 years for months 37–39: TSF, 36%; CBT, 24%; MET, 27%

Despite the significant superiority of the TSF as compared to the other two procedures in terms of complete abstinence for 1 year and abstinence at the 3-year mark, the director of the NIAAA stated,

> [P]atients can be confident that in any competently run treatment program they're likely to do equally well. Although the trial had not been designed to test which of the three treatments . . . offered the best outcomes, one of the findings to emerge was that each produced approximately equal results according to the study's principal outcome measures — the percentage of abstinent days and the number of drinks per drinking day. [Gordis, 1997, p. 446]

It is grudgingly admitted,

> In the outpatient groups, however, the 12-step treatment had more favorable results according to several other measures, including (1) continuous abstinence from alcohol. . . . Thus, by all of the measures studied, 12-step clients achieved outcomes at least as pronounced and durable as those of clients in other therapies, and by some measures, 12-step clients achieved better outcomes. [*10th Special Report*, 2000, p. 446]

These passages are in the *10th Special Report to the U.S. Congress on Alcohol and Health* (2000), which informed Congress that the US$27 million boondoggle that they had funded was worth the money. Project MATCH investigators prefer to emphasize quantitative treatment outcome measures such as percentage of abstinent days and number of drinks/day as purportedly being more sensitive to treatment effects than percentage of people attaining abstinence. Revisionists are ignoring the fact that, in reality, their favorite measures are measures of failure. A percentage of days abstinent of less than 100% and a number of drinks/day of more than 0 presuppose that no brain damage is present in the participants and that none will be produced by continued drinking; however, Bates *et al.* (2005) have refuted this assumption.

Some neuropsychological measures (Reitan, 1958; Shipley, 1946) were obtained during a pretreatment assessment and 15 months after treatment. Bates *et al.* (2005) found significantly greater improvement in executive functions, abstract reasoning, and planning in TSF than in CBT and MET. Further research is needed to determine the bases for the greater neuropsychological improvement in TSF than in CBT and MET. As Bates *et al.* suggest, it may be due to the greater percentage of participants attaining abstinence in TSF than in the other two treatments. Further research is needed, for example, using the Iowa Gambling Task in order to determine whether or not decision making improves with abstinence. Ideally, in addition to neuropsychological measures, neuroimaging assessments of brain structure and function that track changes in brain structure and function before, during, and after treatment need to be obtained (Oscar-Berman & Marinkovic, 2003). Giving patients information about their brain state as compared

to comparable-age social drinkers would provide an enormous incentive for them to reach sobriety. Unfortunately, this sort of research is not likely under the aegis of the skin-deep cognitive behavior therapists who designed Project MATCH (Babor & Del Boca, 2003).

The Randomized United Kingdom Alcohol Treatment Trial (UKATT): Harm Reduction or Induction?

A group of distinguished cognitive-oriented clinical psychologists in the United Kingdom organized a multisite study of alcoholism treatments that successfully competes with Project MATCH for its occult value and potential maleficence. The United Kingdom Alcohol Treatment Trial (UKATT) is an example of harm reduction as harm induction. Treatments studied were MET and a treatment approach developed in the UK, social behavior and network therapy (SBNT).

The UKATT Research Team cited Project MATCH as the only previous large multisite randomized trial: "Motivational enhancement therapy achieved outcomes essentially similar to those of the two more intensive treatments" (UKATT Research Team, 2005b, p. 541). As previously noted, this interpretation is incorrect: TSF was significantly superior to MET and CBT in the critical measure of percentage of patients achieving abstinence. The UKATT Research Team continued:

> This evidence and the increasing popularity of motivational enhancement therapy led to the proposal that this therapy should act as standard treatment in research on the effectiveness of treatment for alcohol problems. We proposed that motivational enhancement therapy should act as reference treatment within our trial. . . . We compared a novel social treatment with a strong theoretical and empirical basis with an established but briefer motivational treatment of proved effectiveness. [p. 541]

Seven different sites where chosen for the trial. Therapists were recruited and trained in each trial site. Patients were randomly assigned either to eight 50-minute treatment sessions spread over 8–12 weeks devoted to SBNT or to three sessions of MET. A number of standards were employed in patient selection: participants had to be at least 16 years old, were literate, could provide a contact person, planned to

stay in the area, were not psychotic or severely impaired cognitively, had alcohol as their principal drug problem, and were not in an alcoholism treatment program. Assessments were obtained at baseline prior to the start of treatment and 3 and 12 months after the completion of treatment. A total of 742 individuals participated in the treatment; 689 (28%) were interviewed after 3 months and 617 (83%) after 12 months.

The treatment groups did not differ in any of the major dependent variables. Patients were abstinent 29.5% days at baseline, 42.7% days at 3 months, and 46% days at 12 months. Drinks per drinking day were 26.8 at baseline, 17.9 at 3 months, and 19.2 at 12 months. Size of the evaluation window was 3 months. Percentage of individuals who were abstinent, if any, was not reported. Significant improvements in measures of quality of life and alcoholism problems were reported: "Both therapy groups reported substantial reductions in drinking and associated problems and improved mental health" (UKATT Research Team, 2005b, p. 543). High levels of gamma-glutamyl transferase (GGT) measured at baseline and showing no change at 3 and 12 months after treatment were considered of no consequence by the UKATT Research Team, despite GGT showing no improvement of liver function as a consequence of treatment. No neuropsychological or radiological measures of brain structure and function were obtained.

Of course, a reduction in drinking from an average of 26.8 drinks per day to 19.2 drinks per day and an increase in percent days abstinent from 29.5 days to 46.0 days are an "improvement". But, drinking an average of 19 drinks approximately every other day is not remission; rather, it will exacerbate and cause further brain and liver damage. Nineteen drinks per day is neither social nor moderate drinking; it is a highly likely cause of brain damage (Cala, 1987; Crews *et al.*, 2005; Eckardt *et al.*, 1998). The fact that GGT did not decrease over the treatment period, showing continued symptoms of liver disease, was downplayed by the UKATT Research Team. Instead, they emphasized the cost-effectiveness of the two treatments: "Both therapies saved about five times as much in expenditure on health, social, and criminal justice services as they cost. Neither net savings nor cost effectiveness differed significantly between the therapies" (UKATT Research Team, 2005a, p. 544).

The real problem is this: what will the cost be to the individual alcoholic's health, their family, and society at large in the long run when drinking 19 drinks every other day? Cognitive behaviorists in the UK as well as in the USA seem oblivious to the brain damage caused by the levels of alcohol consumption they feel is acceptable, moderate, an improvement — "Ignorance is strength."

Not all investigators of alcoholism treatment outcomes in the UK, fortunately, are oblivious to the brain damage caused by excessive drinking. Davies *et al.* (2005) reported that apparently clinically "healthy" abstinent alcoholics meeting DSM-IV standards of alcohol dependence and of above-average intelligence showed significant impairment on neuropsychological assessments of frontal lobe functioning. Davies *et al.*'s results, once more, support the necessity of obtaining assessments of brain structure and function prior to, during, and after treatment for alcohol dependence. Given Cala's (1987) results, we would add that such assessments are needed prior to the treatment of all alcohol problems.

Randomized Control Trials (RCTs)

Miller and other revisionists have had much to say about the efficacy of their small RCT studies of CBT, but little has been reported on the long-term treatment outcomes of such approaches. There is an exception: Miller's long-term follow-up of his highly rated studies with Graber (1988), Harris (1990), and others (Miller *et al.*, 1986, 1992). We read little about the long-term follow-up of these RCTs because of their dismal results. Despite exaggerated claims by Hester (1995), Miller's colleague, 3.5 to 7/8 years following the index intervention, participants treated in the Graber and Miller (1988), Harris and Miller (1990), and other RCTs who engaged in "asymptomatic" controlled drinking were doing no better (14%) than the natural remission rate for such "problem drinkers" (19%) (Miller & Hester, 1980). The evaluation window was the previous 12 months.

Miller *et al.* (1992) reported administering a battery of neuropsychological tests including Trail-Making, Finger Oscillation, and Tactual Performance, as well as the Digit/Symbol and Block Design subtests of the Wechsler Adult Intelligence Scale (WAIS). An aggregate measure of

the neuropsychological test results correlated significantly with outcome measures of consumption and abnormal serum chemistry. Unremitted cases correlated significantly with abnormal scores on the Digit/Symbol subscale of the WAIS. These results indicated that participants who drank "moderately" as well as excessively suffered from brain dysfunction, which may be exacerbated by their continued drinking (even "asymptomatic" drinking). The implications of these results were not considered by Miller *et al.* They also failed to provide information on the quality of life of the participants, whether they were abstinent, drinking "asymptomatically", or unimproved.

Heather (1989) has criticized brief intervention treatments such as Miller's for "proving the null hypothesis" and for its lack of statistical power. These criticisms have not deterred Miller and his colleagues from rating his studies among the methodologically best RCTs in the field (Miller & Wilbourne, 2002). Miller's claim for the efficacy of BSCT based on improvement from baseline (Stimmel *et al.*, 1983) is falsified by available data ignored by Miller. Stimmel *et al.* have not been accurately cited by Miller, Hester, and other revisionists. When Stimmel *et al.* have been cited, the absence of a difference in improvement from baseline between cognitive and traditional treatments is described. Both groups show significant improvement from baseline; this is taken as evidence that the two treatments, as provided, are equally effective. But, the fact that the two treatments did not differ from a nontreated control group, which also showed significant improvement from baseline, is not mentioned by Miller and his colleagues (Miller, 1990; Miller *et al.*, 1992). The fact of the matter is that Stimmel *et al.* demonstrated that the two treatments are equally ineffective.

The dismal long-term outcomes found by Miller *et al.* (1992) and their distorted interpretations are not the only long-term treatment effects in the literature. Like so much else of interest and importance concerning the evaluation of treatment outcome, Miller, Hester, and other revisionists ignore the important work of Moos and his collaborators (Moos *et al.*, 1999, 2003, 2004, 2005) on the effects of social and community resources and treatments on long-term alcoholism remission. Before turning to a discussion on naturalistic longitudinal studies, we need to review the origin and nature of the RCT and how

it should be conducted, in contrast to how it is conducted by Miller and his colleagues.

Miller and other revisionists consider the RCT as the gold standard of clinical trials. On these methodological grounds, Miller and colleagues (Miller & Hester, 1986; Miller *et al.*, 2001) rate their own RCTs very highly. However, an examination of the characteristics and results of the original RCT puts the lie to revisionists' claims concerning the methodological superiority of their research. It also illuminates another claim of fabulists: their interpretation of natural remission as a consequence of an appraisal of alternatives and a rational decision to reduce drinking (Sobell *et al.*, 1993).

Pulmonary tuberculosis (TB) is the deadliest disease in recorded history. It is estimated that two million people/year are still dying from this disease, primarily in developing countries. Streptomycin, a vaccine for the disease, was discovered in the United States by S. A. Waksman in 1944. He was awarded the Nobel Prize for his discovery in 1956.

In 1946, following the end of World War II, Great Britain had very little money and could afford only a small supply of streptomycin. Would it be worth the investment to determine if streptomycin was an effective treatment for TB? Austin Bradford Hill, a medical statistician, convinced the British Medical Research Council that it was ethically proper to proceed with a controlled trial, one which for the first time randomized patient assignment (Hill, 1990). Hill was eventually knighted for his accomplishment. He randomly assigned hospitalized TB patients to a control group receiving the standard treatment (i.e. bed rest) or to an experimental group receiving streptomycin plus bed rest. A number of restrictions on the type of patient were employed:

> A first prerequisite was that all patients in the trial should have a similar type of disease. . . . To avoid having to make allowances for the effect of forms of therapy other than bed-rest, the type of disease was to be one not suitable for other forms of therapy. The estimated chances of spontaneous regression must be small. On the other hand, the type of lesion should be such as to offer some prospect of action by an effective chemotherapeutic agent; for this reason old-standing disease, and disease with thick-walled cavities, should be excluded.

> Finally the age group must be reasonably limited, since the total number of patients in the trial could not be large. . . . For these several reasons the type of case to be investigated was defined as follows: acute progressive bilateral pulmonary tuberculosis of presumably recent origin, bacteriologically proved, unsuitable for collapse therapy, age group 15 to 25 (later extended to 30). [Streptomycin in Tuberculosis Trials Committee, 1948, p. 769f]

Detailed biomedical assessments were conducted during the year following the treatment. Mortality results 12 months after the treatment were as follows (Streptomycin in Tuberculosis Trials Committee, 1948, p. 782):

> Streptomycin (streptomycin plus bed rest): 22% (12/55) died
> Control (bed rest): 46% (24/52) died

Twice as many died in the control group as in the group receiving streptomycin, 46% vs. 22%, respectively. Note, however, that despite this deadly disease, more than half (54%) showed natural remission in the control group, recovering from TB with nothing more than bed rest. Does this mean that we should not have vaccines and antibiotics because more than half of the people recovered from the deadliest disease in recorded history through natural remission (as I did as a child)? Of course not. Natural remission from the disease of alcoholism is characteristic of other diseases as we have seen above, including cancer (Shilder, 1993). No one would probably be alive today if it were any different, if our ancestors afflicted with diseases could only survive as a result of the administration of vaccines or antibiotics.

Sir Bradford Hill's (1990) remarks are worth noting:

> At that time [1946] we were handicapped — we could have enough of the new drug to use on only about 50 patients, and there was said to be no dearth of patients. In point of fact, the planners laid down so many rules there did appear at times to be a bit of a dearth; but when we persisted there were plenty. We were limited to this number, about 50 in the streptomycin-treated arm of the trial, and I thought that was probably enough to get a reliable answer so long as it was strictly controlled and if streptomycin was really effective. And so it proved.

> I think there was no doubt it was the first strictly controlled trial—it ushered in the new era of medicine. As I have stressed, the shortage of streptomycin was the dominating feature of the situation in Britain when the trial was under consideration. . . .
>
> Of course, there were no ethical problems in those days: we did not ask the patient's permission or anybody's permission. We did not tell them they were in a trial — we just did it. To tell the truth, all of the discussion today about the *patient's* informed consent still strikes me as absolute rubbish. Personally, I would like to see an ethical committee overlooking the experimenting doctors: Is the clinical question worth asking? Is it reasonable to ask patients to enroll? Is the question asked in a way (numbers, duration, drafting of questions, and so on) that will give a valid answer? The patients should be told of the ethical committee's decision and asked whether they will agree. . . .
>
> I think it is wrong to shift the entire consent-giving responsibility onto the shoulders of patients who cannot really be informed or know what weight relatively to put upon the technical information provided concerning risks and benefits. The doctors, it seems to me, must weight all this in the light of their medical training. It is my personal opinion that the responsibility rests with them and their sense of morality.
>
> The reader must realize that I am now into my 93rd year, and all this happened some 44 years ago — but I remember it all very clearly. I do hope this personal account of what went on is of some interest to the readers of *Controlled Clinical Trials*. [pp. 78–79]

The problem Sir Bradford Hill raises concerning the ethical oversight of the "experimenting doctors" is still with us, as evident in our discussion of treatment self-selection in Chapter 6. This oversight responsibility should now rest with internal review boards (IRBs), which must approve a research proposal before a research institution can submit it to a funding agency. A basic problem is that there must be members of the IRB who have expertise in the research area under consideration and who are sufficiently knowledgable to know the risks and benefits involved in the proposed research. I suspect that there are very few IRBs containing such knowledgable members in the field of alcoholism treatment research. This lack and the lack of a "sense

of morality" on the part of principal investigators promotes the current unfortunate situation facing alcoholism treatment studies and the commonweal.

Naturalistic Longitudinal Studies

Although the RCT when properly conducted is the gold standard of clinical research, the great majority of RCTs in the field of alcoholism studies unfortunately do not approximate this ideal because they lack the external validity present in Sir Bradford Hill's classic RCT. Remember, he used patients already in hospitals receiving the standard treatment; his subjects therefore represented the kind of people to which the RCT outcome is generalizable. This is not true of the typical RCT in alcoholism studies, such as the ones conducted by Miller and his colleagues, the ones receiving their bloated methodological ratings. Miller and colleagues advertised for volunteers for their RCTs and, in some cases, paid them. They employed numerous exclusionary criteria: no polydrug use, no comorbidity, no lower socioeconomic class, etc. The closest approximation to the ideal RCT with alcoholics are the studies conducted by Foy *et al.* (1984) and Rychtarik *et al.* (1987); but even their studies have a limitation in that their hospitalized patients did not receive traditional abstinence treatment, the Minnesota Model. Nevertheless, in the short term, patients randomly assigned to controlled drinking plus abstinence training were significantly worse off than the randomly assigned abstinence-only patients. In the long term, controlled drinking training had no statistically significant effect: controlled drinking plus abstinence training did not differ from the abstinence-only group. These studies have been repeatedly misinterpreted by revisionists as showing that controlled drinking and abstinence are equally effective in the long run (see Maltzman, 2000).

An alternative to the typical RCT of alcohol treatment lacking external validity is a carefully conducted naturalistic longitudinal study of outcomes for people receiving alcoholism treatment in the community. We have previously discussed such studies of the CATOR registry (Harrison *et al.*, 1991) and Laundergan (1982), among others. We shall now turn to longer-term follow-up investigations. One such study was reported by Humphreys *et al*. (1997).

The primary measure used by Humphreys *et al.* (1997) was remission, defined as "no dependence symptoms and no drinking-related problems in the previous 6-months" (p. 234). They followed an approximately equal number of previously untreated alcoholic men and women, 395, for 8 years. An aim of the research project was to avoid possible confounding of earlier treatment effects and subsequent effects of social and community resources and interventions. At the pretreatment baseline period and 1, 3, and 8 years later, participants were asked to complete an extensive self-administered inventory. Information was obtained for the intervening period concerning the frequency and amount of treatment received, the amount and kind of participation in AA, and the amount and quality of social and familial relationships.

Humphreys *et al.* (1997) reached several conclusions from their research project:

1. Psychosocial contexts such as the quality of friendships, spouse/partner relationships, and particularly family relationships provide better prognostic indicators of long-term remission than problem severity.
2. Women are more likely to be remitted at 8 years than men. They also have better-quality friendships and family relationships.
3. The amount of inpatient treatment does not predict remission at 8 years, whereas outpatient treatment does predict remission. A probably important variable is that the outpatient treatments were of longer duration than inpatient treatments. Outpatient care may therefore provide a greater opportunity for patients to encounter and discuss everyday problems with a counselor.
4. Of all the demographic, psychosocial, and treatment variables studied, participation in AA has the broadest and most long-lasting positive effect on the quality of relationships. It best predicts remission 8 years following baseline treatment.

Further analyses indicated that the advantage of AA was not a self-selection artifact, i.e. the more social and less severely dependent people attended AA more often and thus showed better remission results. Humphreys *et al.* suggested that the overall advantage of AA may be due to the fact that it is readily available, it can be attended indefinitely and without cost, and it provides a network of sober supportive friendships.

Humphreys *et al.* (1997) made the following observations:

> Motivation is not a static, acontextual trait that alcoholic individuals carry with them. As has been demonstrated in both clinical and experimental settings . . . individuals' motivation to change is shaped by the interactions they have with those who are trying to help them. . . . Naturalistic longitudinal studies are the only way to capture these dynamic processes and to thereby shed light on how alcoholism is resolved or continues to cause suffering in the real world, where change continues long after the typical clinical trial follow-up study is complete. [p. 237]

This conclusion calls into question Miller's and other revisionists' reliance on short-term (e.g. 12-month) follow-ups of RCTs lacking external validity, largely restricting themselves to measures of alcohol consumption that ignore quality-of-life changes. Moos and Moos (2003) examined in some detail the long-term influence of treatment duration in the sample studied by Humphreys *et al.* (1997). Duration, defined as the number of weeks or months of treatment, must be differentiated from intensity, the number of treatment sessions per week or month. Duration in the above sense has a greater impact on the long-term outcome than intensity of treatment. Similarly, Moos and Moos (2004) reported that the duration and frequency of participation in AA, particularly duration, were related to better outcomes at the 8-year follow-up.

Continuing their long-term study of the effectiveness of alcoholism treatment and participation in AA, Moos and Moos (2005) compared three different groups of individuals with alcohol problems: (1) those who participated in AA and did not receive professional treatment during their first year of help seeking, (2) those who received professional alcoholism treatment but did not join AA during their first year of help seeking, and (3) those who received both professional alcoholism treatment and participated in AA during their first year of help seeking. A total of 362 participants were interviewed at baseline and 1, 3, 8, and 16 years after receiving treatment and/or initiating AA participation. Results indicated that individuals who received both professional treatment and participated in AA during their first year of help seeking had a higher

rate of remission in the long term than the other two groups of individuals who did not utilize both resources during their first year of help seeking. In general, the duration of AA participation predicted remission in subsequent years such that "the duration of participation in AA in years 4–8 independently predicted 8-year remission; the duration of participation in AA in the first year and in years 2 and 3 independently predicted 16-year remission. Controlling for the duration and frequency of treatment did not affect these findings" (Moos & Moos, 2005, p. 1864).

Additional analytical studies of the impact of social relations on remission have been undertaken. A study of social networks contributing to favorable treatment outcomes in a representative group of people treated in public and private facilities in Northern California was conducted by Bond *et al*. (2003). Abstinence at follow-up 1 and 3 years after baseline treatment was the dependent variable, using a 90-day evaluation window. Major determinants of abstinence were being involved in AA, attending meetings, having a sponsor, and having social networks with proportionately fewer heavy drinkers and proportionately more people who encourage reduced drinking. Increased participation in AA in later years further increased the likelihood of abstinence in the long term.

Results of an exceptional 10-year follow-up of 200 randomly selected patients from a single private inpatient facility in Georgia have been described (Cross *et al.*, 1990). The treatment duration was 4–6 weeks. Following detoxification, there were daily meetings studying the 12 steps of AA, counseling, and didactic sessions. In the evenings, there were AA speaker meetings. An additional task was the completion of a written life history subsequently read to a counselor and then destroyed. The hospital hit upon a procedure developed by Pennebaker (1997) and found it to have significant therapeutic value. This and related procedures are further considered in Chapter 4. Family members were involved in the treatment program; for example, the spouse was encouraged to stay with the patient during the last week of treatment. Aftercare was emphasized, and an AA contact in their local community was obtained for each patient. Former patients were also contacted periodically for various activities and an annual "homecoming" affair.

Dependent variables used at the follow-up assessed drinking and social status. Questionnaire items assessed the degree of involvement in

AA following treatment, for example, frequency of attendance at meetings during the first year and the last 2 years of the follow-up period, an AA sponsor, a "home" AA group, and whether the patient sponsored an AA member. Information was also obtained on other organizations that may have helped in maintaining sobriety, such as church, civic, professional, and social groups. A random validity check of patients who reported stable long-term abstinence was obtained by contacting spouses for confirmation.

Results showed that involvement in AA was significantly related to remission and psychosocial status. The greater the involvement, the longer the length of remission: "active involvement in a local AA group, especially when it includes continual sponsorship, is the best insurance against relapse" (Cross *et al.*, 1990, p. 172). About 61% of patients were reported in complete or stable remission for at least 3 years at follow-up, and 84% were in stable psychosocial condition. Quality of life was normalized, including social relationships and employment. Compare these long-term results for a traditional treatment program with Miller *et al.*'s (1992) abysmal long-term BSCT outcomes. Cross *et al.*'s important longitudinal study was not cited by Hester and Miller (2003).

An obvious gap remains in the typical naturalistic longitudinal study of treatment effects. A comparison group of alcohol-dependent people is needed to determine the percentage of individuals in remission due to treatment above and beyond changes occurring as a consequence of natural remission. Weisner *et al.* (2003) have provided such a study. Using a probability sample of the population in Northern California, they identified a control sample of alcohol-dependent people not in treatment. A survey of consecutive admissions to public and private treatment facilities in the same region provided a treatment group. Interviews at baseline and 1 year later showed that 57% of the treatment group were abstinent; 12% in the nontreated control group were abstinent as a result of natural remission.

It is interesting to compare these results with those reported by Sir Bradford Hill in his classic RCT of treatment for TB. A smaller percentage of people showed natural remission from alcoholism than from TB, the deadliest human disease in recorded history. The point here is that some people show natural remission (i.e. recovery) from deadly diseases

without formal treatment. This fact does not justify ceasing to search for treatments that increase recovery above and beyond natural remission; nevertheless, there is the suggestion by some investigators of natural remission from alcoholism that this is a whole new approach, "thinking outside the box", and that its investigation is worthy of extensive research support to study the process involved (i.e. cognitive appraisal) (Sobell *et al.*, 1993).

Causes of natural remission cannot be discovered as the result of cognitive folk psychology, but through studying the neurochemistry of the brain and neuroendocrine system interacting with behavior and the social environment. Tucker's (2003) extensive review of natural remission is primarily concerned with remission in people who are not suffering from severe alcohol dependence. Her promotion of "harm reduction" for people with alcohol abuse or alcohol problems is unethical in the absence of measures of brain structure and function indicating that alcohol consumption would not exacerbate or prevent recovery from existing brain damage.

Rehabilitation of the Rand Report

As McLellan *et al.* (1993) have demonstrated, given patients with comparable prognostic indicators, treatment outcomes may vary significantly between different inpatient treatment facilities, between different outpatient facilities, and between inpatient and outpatient facilities, depending upon the intensity and number of different services, other things being equal. A knowledgable patient needs to know these outcome rates as they pertain to his or her particular needs, for example, suffering from alcohol dependence, polydrug dependence, comorbidity, psychiatric and family problems, etc. The utilization of McLellan's ASI and TSR measures would improve the matching of patients' needs to treatments and provide caregivers with information that could be used to improve treatment programs (McLellan *et al.*, 1997).

It is noteworthy that Miller *et al.* (2001) ignored McLellan *et al.*'s (1993) important instruments and report of treatment outcomes. Instead, they used selected rather than all multisite studies to buttress their argument, avoiding private facilities. Many of their evaluated studies

employed multiple Veterans Affairs (VA) facilities. Do these facilities represent the kind and quality of treatment a nonveteran or a veteran would obtain in a private facility? Most likely, they do not. Private facilities would probably provide more and better services and treatments because they generally pay higher salaries and have a more skilled and larger staff; as a consequence, they may provide a better treatment program than public facilities. Furthermore, the socioeconomic status and social support systems of the patients must be considered in evaluating treatment outcomes. Patients in VA hospitals are probably handicapped by a lower socioeconomic status and lack of social support systems. Within the VA system, the prospective patient would want to know whether the treatment facility provides traditional treatment or BSCT. They would want to choose among the former rather than the latter facilities, given comparative treatment outcomes for treatment completers that are superior in the traditional 12-step–oriented programs than BSCT programs (Ouimette *et al.*, 1997).

In addition to VA facilities, multisite studies included in the Miller *et al.* (2001) analyses were the Project MATCH, previously considered, and the Rand Reports. Miller *et al.* described the latter multisite study in the following revealing manner: "One of the earliest studies of alcohol treatment outcomes was reported by the Rand Corporation (Armor *et al.*, 1978; Polich *et al.*, 1981). Although these reports stirred public controversy over 'controlled drinking,' the study was large and well conducted, encompassing 1,340 clients from eight programs" (p. 216).

The above comment is a striking example of the rewriting of the history of alcoholism treatment outcome studies. It is revisionism in the extreme, relying on the likelihood that new generations of readers will not make the effort to study the Rand Reports themselves, but rely upon the judgment of a current so-called expert. There was "public controversy" over the Rand reporting of "controlled drinking" for good reason. Polich *et al.* (1980, p. 81) reported that 28% of patients were abstinent at a 4-year follow-up, and an astonishing 18% were drinking moderately and functioning without major dependency symptoms. Scholarly analyses have demonstrated the major methodological shortcomings of the Rand Report (Wallace, 1989a, 1989b, 1990). First, we must remember

that the Rand criterion for controlled drinking was "an average of 6-drinks/day with not more than 10 on a typical day with a restriction that no more than 3 dependency symptoms occur (Armor, Polich & Stambul, 1976, 1978)" (Maltzman, 2000, p. 202). No neuropsychological or electroencephalography (EEG) measures assessing brain structure and function were considered in the Rand Report. Evidence previously cited (Cala, 1987; Eckardt *et al.*, 1980) indicated that an average of 5 drinks/day may lead to brain dysfunction.

The following is a quotation from Wallace (1989a), with reference to Rand Report I, that I have used before (Maltzman, 2000). It is worth repeating, given Miller *et al.*'s (2001) revisionist evaluation of the Rand Report:

> [T]he original sample at the time of intake was 11,500 clients. The six-month study was conducted upon only 2,371 subjects. An astonishing 9,129 subjects were lost during the course of treatment. . . . One of the more egregious errors was to leave the reader with the impression that Armor's major analyses in the 18-month study were conducted upon 1,340 subjects. Actually, the facts of 'sampling' in this study are complex, incompletely described, and, in some critical particulars, elusive and controversial. A reasonably informed source . . . has stated that from a group of 3,243 eligible clients at the ATC's [alcoholism treatment centers] involved in the 18-month study, 2,320 subjects were somehow assembled. The sampling was not random. While the Rand authors assert that the sampling was 'representative', . . . it was 'convenience sampling' based largely upon the numbers each treatment center could contribute. Of these 2,320 clients, 1,340 were located and interviewed at follow-up. From these 1,340 interviewed clients, 600 clients were non-randomly assembled (all women and driving-while-intoxicated clients were systematically eliminated) and served as the principal sample for the major analyses. Further non-random and systematic reductions resulted in analyses conducted upon still fewer clients. . . . [T]he very important analyses conducted upon relapse rates comparing abstinent clients to normal drinking clients were conducted upon 220 clients in one analysis and 161 clients in another. [Wallace, 1989a, p. 268f]

Wallace (1990) continues,

> Aside from the issue of bias on outcome due to the loss of large numbers of patients on follow-up, the Rand studies used a very short follow-up window. While the 1976 studies consisted of a six-month study and an 18-month study, the actual window on moderate drinking behavior in both studies was only 30 days or one month. Even in the so-called four-year study (Polich, Armor & Braiker, 1981), the actual window on drinking behavior varied from one to six months, with the bulk of observations on 'nonproblem drinking' centering around one to three months. [p. 271]

Concerning Rand Report II, Wallace (1989b) states,

> When one asks how many of the Rand subjects were capable of long-term controlled or nonproblem drinking, then quite a different answer emerges (than the one given by Peele). The Rand study reported that only seven percent of their nonproblem drinking subjects showed consistency of drinking status from admission to four-year follow-up (Polich *et al.*, 1980). But even this seven percent long-term nonproblem drinking estimate is questionable in the Rand Report. The fact that subjects were drinking nonproblematically at these two points does not ensure that they were drinking in this fashion throughout the entire follow-up period. Moreover, the authors of the Rand study readily admitted that their quantity–frequency measure was invalid in the sense that actual consumption was underreported by roughly 25 percent. Adjusting their long-term nonproblem drinking rate for substantial underreporting of consumption and for other factors leads one to a long-term nonproblem drinking estimate of three percent, exactly what Pettinati and colleagues (1982) found in their four-year follow-up study. [p. 265]

Some background context is necessary to appreciate the magnitude of Miller *et al.*'s (2001) rewriting of history. The Rand Corporation, a think tank located in Santa Monica, CA, received a contract from the

NIAAA to complete the data analyses and write the final report for a study of 45 treatment facilities receiving funding from the federal government. The Stanford Research Institute (SRI), another think tank in Palo Alto, CA, received an earlier grant to establish the intake and follow-up procedures including questionnaires, and collect the intake interviews from the alcoholism treatment centers (ATCs). Procedures established for the study as well as intake data from each of the centers are described in two SRI reports (Ruggles *et al.*, 1977; Wilson *et al.*, 1971). The second report is particularly interesting because it provides statistical analyses of the results of the intake interviews of the patients who were followed up and of those lost to follow-up interviews at 6 months and 18 months. These statistical analyses were not described in the Rand Reports or discussed by Miller and other revisionists. Comparisons of the characteristics of the patients entering and completing treatment who were followed up and interviewed as compared to those who were lost to the follow-up and not interviewed are critical to the determination of possible bias due to attrition. It must be remembered that, because of the very high attrition rate at the 6-month follow-up, the follow-ups at 18 months and at 4 years reported in Rand Report II were conducted on a convenience sample of only 8 of the original 45 ATCs.

Ruggles *et al.* (1977) reported a consistent finding: patients lost to the follow-up interviews were significantly different from those interviewed at follow-up. The differences were all in one direction. Those lost to the follow-up had a poorer prognosis. According to Ruggles *et al.* (1977),

> In particular, the noninterviewed clients appear to be more unstable than the interviewed clients, either recovered or nonrecovered. For example, for non-DWI [non–driving-while-intoxicated] males, the noninterviewed clients are less likely to be married, have more addresses over the past five years, are less likely to own their own homes, are much less likely to be living with others, are more likely to be unemployed, are much less likely to have a yearly income of $5000 or more, and have higher impairment and Q-F [quantity–frequency of alcohol consumption] scores than the interviewed clients. [p. 108]

Overall, the extensive attrition rate appears to be highly biased. People with more severe alcohol problems and a lack of social resources were more likely to have been lost to follow-up. Investigators at the SRI also detailed the unreliable and careless manner in which patients were interviewed and data collected. Gross inadequacies in the methodology of the Rand study were apparent when the report first appeared. The following comments from professionals appeared in *The Alcoholism Report* at the time:

> Dr. John Wallace, Professor of Psychology, State University of New York at Purchase, said he would flunk any student in a freshman course in research design who handed in a paper like the Rand report.
>
> Dr. Samuel Greenhouse, Chairman and Professor of Statistics, George Washington University . . . not associated with the field of alcoholism, called the Rand study a 'bad design,' and a 'shoddy report from the point of view of data analysis.' . . . He noted that there was no attempt to validate the statements on alcohol consumption made by any of the alcoholics, including those who were reported to have returned to normal drinking. [*The Alcoholism Report* **4**(18): 3, 1976]

Continuing in their rewriting of the alcoholism treatment outcome literature, Miller *et al.* (2001) state,

> Other multisite studies were considered but did not meet review criteria. . . . A Schick study (Smith *et al.*, 1991) reported an 83% follow-up rate at 12 months for Schick clients and 82% follow-up with a multi-site comparison sample. The number actually interviewed, however, comprised only 27% and 2% of the source samples from which they were drawn. Even when the number of clients contacted at 6-month follow-up is used as the denominator only 45% and 3% were completed at 12 months. [p. 216]

The difference in Miller *et al.*'s (2001) treatment of the Schick study and the Rand Report is striking. An inappropriate attrition rate was invented for the Schick study, whereas the inadequacies in the Rand Reports (including their large attrition rate) were ignored.

Miller *et al.* (2001) ridiculed the crude measure of abstinence. However, it is the only valid measure of a successful treatment outcome, particularly when Miller and other skin-deep cognitivists failed to obtain measures of brain structure and function from participants in their treatment programs and subjects in their small RCT efficacy studies and/or failed to interpret and report them properly. They failed to inform their patients/participants concerning the status of their brain structure and function. In the absence of such material information provided to their patients/participants, they failed to obtain a proper informed consent for treatment/research.

Finally, Miller *et al.* (2001) failed to utilize the results from the CATOR registry and the McLellan *et al.* (1993) study of private facilities that have far superior outcomes than Project MATCH treatments. An excuse was Miller *et al.*'s requirement that only studies with at least a 1-year follow-up were to be included. However, results from 1 year follow-ups reported in the CATOR registry were not considered by Miller *et al.* McLellan *et al.* argued that in their experience the great majority of relapses occur within 6 months of discharge; a longer follow-up period may be contaminated by subsequent treatments following a relapse, leading McLellan *et al.* to use a follow-up of 6 months. Vaillant (2003), on the other hand, argued that in keeping with the disease nature of alcoholism the follow-up period should be at least 5 years.

> It has become reasonably clear that one should consider at least two dimensions of drinking outcomes: frequency and intensity (Project MATCH Research Group, 1993). Although more emphasis is often given to frequency (e.g., percent days abstinent), *how often* a person drinks is not the whole story, even with alcohol dependent clients. . . . Frequency of drinking is usually discussed as its inverse, abstinence. One of the crudest measures of outcome is the proportion of cases maintaining perfect continuous abstinence from alcohol during a specified period. An obvious shortcoming of this metric is that recurrence of addictive behaviors is exceedingly common . . . even among those who ultimately maintain stable abstinence. Definitions of abstinence differ, allowing for various levels of slippage before incurring a judgment of relapse or treatment failure. The definition, method and care

> taken in ascertaining abstinence are sometimes left unspecified. . . . [Miller *et al.*, 2001]

It is noteworthy in the quote above that Miller *et al.* (2001) failed to cite the specific studies using crude measures of abstinence, instances of slippage, etc., such as the abstinence measures used in the CATOR registry (Harrison *et al.*, 1991), the Schick Shadel program (Smith & Frawley, 1993), and the McLellan *et al.* (1993) studies — all of which provide superior treatment outcomes than any of the CBT treatment studies of people with alcohol problems varying from preclinical to DSM-IV dependence, or the biggest boondoggle of all, Project MATCH. Instead, resorting to sophistry, Miller *et al.* (2001) conclude,

> Drawing together the outcomes for more than 8,000 people treated for alcoholism, we offer the following narrative summary. After a single treatment episode, roughly one client in four will abstain from alcohol throughout the first year, which is the period of highest risk for return to drinking. In addition, about 1 in 10 will moderate the quantity and frequency of their drinking to remain free of alcohol-related problems in this same 1-year period. In combination, these unambiguously positive outcomes account for about one third of treated cases. . . . The remaining two thirds of treated clients continue to have at least some periods of heavy drinking in the first year, but outcome data reflect substantial improvement, a fact often overlooked. After treatment, even those who do drink are abstinent on 3 days out of 4. . . . [T]hey go from drinking on 2 days out of 3 on average before treatment, to 1 day out of 4 afterward. On days when they do drink, the average amount of alcohol they consume is less than half what it was before treatment, albeit still heavy. The combined effect of these reductions in frequency and quantity is substantial. Even for those who continue to drink, alcohol consumption drops by more than 87% on average in the year after treatment (from an average of 77 standard drinks per week to 10). Clearly, this is enough to result in substantial reduction of health and social problems related to drinking. Alcohol-related problems are also reduced by 60% after treatment. This substantial improvement in clients who do not maintain perfect abstinence or moderation is

> obscured by any simplistic classification of cases as 'successful' versus 'relapsed'. [p. 218]

There are no references to the studies using the "simplistic" classification of 'successful' or 'relapsed'. More importantly, there are major shortcomings in Miller *et al.*'s (2001) classification. What is the percentage of subjects in the abstinent and moderate drinking classes? What percentage in each class is diagnosed as suffering from alcohol abuse or alcohol dependence, or is preclinical according to DSM-IV? There is no indication of the duration of the "substantial improvement". Ten years, perhaps? I doubt it. In none of this discussion of improvement and moderate drinking is there any mention of the fundamental problem of brain damage. Why is there no report of neuropsychological, EEG, event-related potential (ERP), or radiological assessments of brain structure and function? Assessments of "improvement" and "moderation" in drinking without accompanying assessments of brain structure and function demonstrating that brain damage is not exacerbated or recovery prevented is unacceptable, given current knowledge of the consequences of alcohol consumption on the brain (Bates *et al.*, 2005; Cala, 1987; Crews, 1999; Eckardt *et al.*, 1980, 1998; Hunt & Nixon, 1993; Nelman, 1998; Oscar-Berman & Hutner, 1993; Parsons, 1998).

Furthermore, 10 drinks/week do not break down to a modest little more than 1 drink/day. Where is the intensity of drinking measure that Miller *et al.* (2001) said is so important? For some participants, the 10 drinks/week could all be consumed in 1 day. Aggregate measures (i.e. averages) such as these are therefore misleading. More than 2 drinks/day for men and 1/day for women affect morbidity and mortality in nonalcoholics (Liao *et al.*, 2000). Once more, the strength of Miller *et al.*'s (2001) argument is based on their ignorance of what is happening to the brain. As noted in Chapter 5, Wilkinson and Sanchez-Craig (1981) reported that an average of less than one drink/day or abstinence is necessary for reversal of a neuropsychological deficit. None of the multisite studies rated highly by Miller *et al.* (2001) reported initial and follow-up assessments for brain damage in patients, damage that would be exacerbated by continued drinking. Why settle for less than success? Why consider an 87% decrease in consumption a success when there are treatments, reviewed earlier, that

produce a 100% decrease in a larger number of their alcohol-dependent participants? Such treatments avoid the danger of exacerbating existing brain damage or inducing such damage by continuing to drink.

Controversial Results of a Large Epidemiological Survey

Dawson *et al.* (2005) reported the results of an analysis of the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). It involved a sample of approximately 4400 adults 18 years of age and older who were personally interviewed and classified according to DSM-IV criteria as suffering from alcohol dependence prior to the past year (PPY). Participants were drawn from a sample of approximately 43 000 adults. The drinking status of participants with a history of alcohol dependence was evaluated for "past year dependence, partial remission, full remission, asymptomatic risk drinking, abstinent recovery (AR) and non-abstinent recovery (NR)" (p. 281). Classifications were defined as follows:

1. Dependence — To be classified with PPY alcohol dependence, participants had to report one or more of at least three of the seven DSM-IV criteria for dependence, such as withdrawal, tolerance, repeated failed attempts to reduce drinking or abstain, etc.
2. Abuse — Participants had to report at least one of the four criteria for abuse as defined by DSM-IV.

Categories of past-year status and the percentage of participants in each category were as follows:

1. Still dependent — Participants reported three or more symptoms of alcohol dependence during the past year; 25%.
2. Partial remission — Participants did not meet the criteria for dependence, but reported one or more symptoms of either dependence or abuse; 27.3%.
3. Asymptomatic risk drinker — Participants reported no symptoms of abuse or dependence during the past year, but were consuming

alcohol. A past-year risk drinker in partial remission with no symptoms of abuse or dependence; 11.8%.

4. Low-risk drinker — Participants reported consuming alcohol, but were never past-year high-risk drinkers or dependent on alcohol; 17.7%.
5. Abstainer— Participants reported consuming no alcohol during the past year; 18.2%.

Participants who were PPY alcohol-dependent in categories 3–5 were classified as being in full remission. Participants categorized as 4 were in nonabstinent recovery (NR), whereas those in category 5 were in abstinent recovery (AR).

Dawson *et al.*'s (2005) report is followed by four commentaries solicited by the editor of *Addiction*. One, by Sobell and Sobell (2005), is titled "Time to tear down the wall", referring to the purported wall between the results of scientific research such as those reported by Dawson *et al.* and what is practiced by alcoholism counselors and other helping professionals in the alcoholism treatment field: attaining abstinence as the goal of treatment. According to the Sobells, controlled drinking must be provided for patients seeking help, especially those who are diagnosed with alcohol abuse and are preclinical, as well as people with less severe alcohol dependence. Since more than half of the participants in Dawson *et al.*'s study were diagnosed in these DSM categories and were found to be drinking without DSM symptoms, moderation (i.e. controlled drinking) has been vindicated as a reasonable alternative to abstinence as the alcoholism treatment goal for individuals suffering from alcohol dependence as well as alcohol abuse and preclinical problems.

Rather than the recommended "tearing down the walls", the bugle call should sound, "Open your eyes". A major shortcoming of the Dawson *et al.* (2005) study is a consequence of the inadequacy of DSM-IV and previous editions of DSM and the *International Classification of Diseases* (ICD): they fail to include measures of brain structure and function. Dawson *et al.* did not obtain neuropsychological assessments because none are provided or required by DSM-IV diagnoses of abuse or dependence. DSM is the acronym for *Diagnostic and Statistical*

Manual of Mental Disorders (1994), which says it all. Alcoholism is nothing but a mental disorder with observable manifestations such as the "need for markedly increased amounts of the substance to achieve intoxication or desired effect", etc. (p. 1181); no measures of brain structure and function are part of the diagnostic scheme. As a consequence, Dawson *et al.* failed to use such measures, thereby opening the door for the Sobells' recommendations of controlled drinking with no concern for its effects upon brain structure and function as well as the consequences for social decision making and adaptation to the social environment (see Chapter 1).

The Sobells are trying to tear down the wrong wall, so to speak. They need to open their eyes and tear down the wall built by their mentalistic cognitive folk psychology and see the neurobiological damage that alcohol dependence, alcohol abuse, and even preclinical heavy drinking may cause (Cala, 1987). With abstinence, such damage is reversible in varying degrees. As I have repeatedly noted, Wilkinson and Sanchez-Craig (1981) demonstrated that as little as one drink/day may prevent the recovery of a neuropsychological deficit.

Relying solely on the behavioral measures of dependence and abuse closes the eyes to what lies behind: what is causing the maladaptive behavior and thinking? Dawson *et al.* (2005) are misguided in labeling their categories AR and NR in the absence of evidence, particularly in the latter case, that the participants' brain structure and function have recovered. Dawson *et al.*'s conclusions are dangerously misleading, as evident in the commentary by Sobell and Sobell (2005) offering various programs of controlled drinking, i.e. avenues for exacerbating existing brain damage and preventing recovery from such damage.

Dawson *et al.* (2005) indicated that they would follow Hasin's (2005) suggestion and attempt to obtain biological measures in their follow-up study. They also need to obtain measures of brain structure and function, such as MRI, fMRI, ERP, and neuropsychological measures of brain function in their follow-up study.

Vaillant's (2005) commentary on the Dawson *et al.* (2005) study calls attention to an important problem concerning the validity of verbal reports involving highly personal "secrets" such as descriptions of excessive drinking and related behavior and misbehavior. In order to ensure

the validity of such verbal reports, a more intensive study of a smaller sample of participants and collaterals may be needed rather than a large representative sample such as the one studied by Dawson *et al.* Unfortunately, Vaillant (2005) misses the mark in his subsequent discussion of the problem of the validity of verbal reports:

> A study reported by two reputable investigators, the Sobells, of treated alcoholics returning 'successfully' to controlled drinking illustrates my point. At the 2-year mark Sobell & Sobell (1976) claimed success, but at the 10-year mark Pendery *et al.* (1982), using time and a less gullible approach to their subjects' assertions, revealed that the Sobells' earlier reports of asymptomatic drinking were an illusion. In my own longitudinal studies, when men in my study assert for 5 years of continuous follow-up that they have been out of control in their use of alcohol or that they have been abstinent, their relatives and the next decade uphold their story. However, when alcoholics tell me, even over a couple of years, that they have been regularly using alcohol without problems, continued follow-up often reveals self-deception. [p. 294]

Vaillant misses the mark because he failed to note that Pendery *et al.* (1982) did not rely solely on verbal reports of alcoholics and their collaterals. Repeated and extensive interviews were obtained, enabling us to track down hospital records and thereby independently document the alcoholics' status.

Table 2 in Pendery *et al.* (1982, p. 172) lists excerpts from medical records reviewed at Patton State Hospital, where the original treatment was conducted, as well as other hospitals in Southern California. It shows that 65% of Sobells' patients receiving controlled drinking training had relapsed and required hospital care within approximately the first year following treatment, yet Sobell and Sobell (1976, p. 127) asserted that these patients functioned well (i.e. were abstinent or controlled their drinking) 70.5% of the days in the year following treatment. Evidence showing I believe beyond reasonable doubt that the Sobells fabricated their results and intentionally misrepresented their procedures is presented in Maltzman (2000, Chapters 4 & 5). Investigations purporting to exonerate the Sobells of wrongdoing are shown to be fundamentally

flawed and do nothing of the sort. A purported congressional investigation never occurred (Maltzman, 2000, p. 168f). Inviting investigators with an evident lack of integrity of the Sobells to comment upon Dawson *et al*. (2005) says much about the integrity — or lack thereof — of the journal editors. The problem of who should watch over these watchdogs is discussed in Chapter 7.

3

What Makes Alcoholics Anonymous Work

No single variable or basic process can explain complex behavior such as the long-term remission of alcoholism following participation in Alcoholics Anonymous (AA). I will, however, consider what I hypothesize to be bases for the effectiveness of AA and other forms of self-help and support groups, recognizing that a problem of this complexity has no simple answer. Infrahuman research on attachment and affiliation will be reviewed, including research on the importance of beta-endorphins, serotonin, oxytocin, vasopressin, and prolactin as mediators of social attachments. It is the kind of biosociobehavioral research that I believe is necessary to reveal the fundamental biological mechanisms underlying the success of AA in promoting remission from alcoholism.

Attachments and Social Affiliations

Like so much else of importance in psychology, the study of social attachment and affiliation has its origin in the research of behaviorists. Such research was initiated by the greatest behaviorist of all and one of the greatest physiologists of the 20th century, Ivan Pavlov. He had already won the Nobel Prize for his research on the digestive system when he turned his attention to conditioning. In the course of these latter studies, he reported his observation of what he called the "social reflex" (Pavlov, 1928). For example, the presence of a familiar person had

a calming, positive effect upon the emotional lability of a dog in the laboratory. Research on the effect of the presence of another was subsequently expanded by the two Americans, Gantt and Liddell, who served their apprenticeships in Pavlov's laboratory in the 1920s. In addition to the effect of a familiar human, they examined how members of the same species such as the herd as well as maternal attachment reduced fear, enhanced stability, and shielded against the establishment of "experimental neuroses" (Gantt, 1944; Gantt *et al.*, 1966; Liddell, 1956). Harry Harlow (1958), another brilliant behaviorist psychologist, targeted the role of touch in attachments in a series of original experiments.

Following the development of behavioral neuroscience, investigators were no longer limited to behavioral studies and peripheral physiological measures such as skin conductance, heart rate, and respiratory rate. They could relate the target behavior to biological mechanisms. I will briefly describe some of the early research on separation stress and its long-term effects, followed by Harlow's classic research demonstrating the importance of touch in maintaining well-being, and more recent studies of separation stress examining neurochemical factors mediating such stress and its consequences.

Early Studies of Separation Stress and Attachment

Liddell (1956) described an experiment conducted with a mother goat and her 10-day-old twins. The twins were simultaneously subjected to classical conditioning, in which dimming of the room lights was followed by an unavoidable electric shock to a foreleg. One twin was conditioned by itself in a room, while the other twin was conditioned in a different room occupied by their mother. After 20 such conditioning trials, there was a 45-minute test period in which the experimenter entered each of the experimental rooms every 10 minutes and loudly clapped his hands, eliciting quite different behavior from the twins. The infant lying on the floor next to its mother lifted its head and perked up its ears, orienting in the direction of the sound; whereas its twin, subjected to the same noxious conditioning while separated from its mother, made no observable response to the hand claps and continued lying quietly on the floor without moving until the end of the 45-minute period.

A similar study was conducted with four pairs of twin goats. One of each pair was raised with its mother and the other in an "orphanage". At maturity, 2 years of age, a more extreme form of the electric shock conditioning experiment was conducted. Each of the goats was exposed to 10 seconds of darkness (i.e. the conditioned stimulus [CS]) followed by electric shock (i.e. the unconditioned stimulus [UCS]) every 6 minutes for 2 hours, 7 days/week, for 24 days. The behavior of the twins was strikingly different at the end of the experiment. Twins who had been conditioned as infants in the presence of their mother missed 25% of their conditioned foreleg response on the 1st and the 24th days of conditioning; whereas twins who were orphaned and conditioned as an infant in the absence of their mother missed 25% of their trials on the 1st day of the experiment in adulthood, increasing to 45% by the last day of the experiment. The "orphans" failed to respond to the CS in the experiment, and also showed extreme lethargy to their general surroundings. Today, this behavior would probably be described as "learned helplessness".

We will now turn to a more intimate aspect of attachment: the role of touch.

Harlow on Love

It is unlikely that a cognitive psychologist using notions such as expectancy would conceive and conduct the groundbreaking experiments on love devised by Harlow (1958). He and his colleague demonstrated the importance of touch and affiliation in bonding and in love, the devastating effects of their deprivation on development, and the successful treatment of the effects of social deprivation (Novak & Harlow, 1975).

Harlow's (1958) research contradicts the hypothesis that I learned many years ago in my undergraduate psychology classes: We learn to love our mothers because they are associated with our relief from hunger and pain. Affection or love is a secondary, learned drive in contrast to the primary drives of hunger, thirst, elimination, pain, and sex. Harlow notes that Watson differed from this classical stimulus–response (S-R) learning view. Watson (1924, p. 221) believed that a mother's love was a primary drive elicited by cutaneous stimulation of the infant's erogenous

zones. Harlow, studying monkeys, demonstrated that stimulation of the infant's skin, not only the erogenous zones, is rewarding and bonding.

Harlow (1958) found that infant rhesus monkeys raised on a bare wire-mesh cage floor survived the first 5 days of life with difficulty; in contrast, infant monkeys flourished if a cone covered with terrycloth was placed in the cage. These and other observations suggested that contact stimulation may be an important variable in the healthy development of an infant and its affection for the mother. They led to Harlow's classic experiments on the effects of mother surrogates on the physical and emotional growth of baby monkeys and on their love and attachment to their mother.

The first experiment was designed to determine the importance of contact vs. nursing comfort. A model of a wire mother and one with the wire covered by terrycloth were placed in different cubicles attached to the infant monkeys' living quarters. Four newborn monkeys were randomly assigned to quarters, where the cloth mother provided all of its milk and the wire mother did not. The lactating and dry mothers were switched among the four different newborn monkeys.

Results showed that contact comfort, i.e. time spent with the cloth mother whether or not she provided milk, was far more important in the development of affection responses than nursing. Infant monkeys spent increasing amounts of time with the nonlactating cloth mother rather than with the lactating wire mother. These differences persisted for the more than 5 months of consecutive days of testing. The results contradict the traditional conditioning notion that affection, bonding, and love for the infant's mother is a learned response as a result of the mother (an initially neutral cue) accompanying the reduction of hunger, thirst, and pain. The disparity between the preference for contact comfort over nursing "is so great as to suggest that the primary function of nursing as an affectional variable is that of insuring frequent and intimate body contact of the infant with the mother" (Harlow, 1958, p. 678).

Experiments using an open field test to induce stress provided an additional measure of the strength of the bond produced by contact comfort of the terrycloth mother. Young monkeys were placed in a 6 ft × 6 ft open space containing a variety of different stimuli

twice a week for 8 weeks. They were placed there alone or with the surrogate mother on alternate weeks. When the terrycloth mother was present, the infant monkeys always went to her for a body rub, explored, and then returned for a rub or to be near. When a wire monkey providing milk was present, the infant monkey would go to her for a drink but return to the terrycloth mother for rubs or to be near her as before.

Later studies by Harlow and his colleagues (Meyer *et al.*, 1974) investigated the behavioral and hormonal effects of separation from mother-reared (MR) monkeys as compared to surrogate-peer–reared (SPR) infant monkeys. Six-month-old MR monkeys and SPR monkeys were separated from their mothers and peers, respectively, for 9 weeks. Physiological reactivity to stress was measured by assaying plasma cortisol. The subjects were eight 6-month-old rhesus monkeys, four males and four females. Half were reared with their biological mothers in individual housing units, and the other half (two males and two females) were separated from their mothers at birth and reared in the laboratory nursery for 15 days. At 10 days of age, each monkey was provided with a terrycloth-covered surrogate wooden mother. Both groups had limited visual and physical contact with their mothers, biological or surrogate. At 6 months of age, they were all separated from their mothers. Animals were stressed once/week by being placed in a cage containing an electrically operated "monster" at each end that flapped its arms, emitted loud sounds, and had lights flashing from its eyes. Blood samples were obtained immediately before being placed in the cage with the "monster" and 15 minutes after their return to their home cage. Behavioral measures such as stereotypical behavior as well as cortisol levels were higher in MR than in SPR animals following separation and following exposure to the frightening "monster". These results suggested that monkeys removed from their biological mothers show greater stress and, hence, attachment to their mothers than monkeys reared by surrogate mothers, although both groups were agitated by separation from their attachment objects. Since Harlow's groundbreaking research, many studies have confirmed the fundamental importance of cutaneous stimulation for the growth, well-being, and love of the human infant (Field, 2001; Sapolsky, 1994).

Beta-endorphins

Panksepp and colleagues (Herman & Panksepp, 1978; Panksepp *et al.*, 1978a, 1978b) integrated apparently disparate observations to arrive at an interesting hypothesis: distress vocalization (i.e. crying) of an infant separated from its mother resembles the distress of an addict in withdrawal from opiates. A similar biological basis underlies both phenomena: a decrease in the level of circulating endorphins, the body's endogenous morphine.

If endorphins mediate bonding and social attachments, then distressful crying produced by separation from a mother should be manipulable by varying the levels of endorphins. Herman and Panksepp (1978) randomly assigned infant guinea pigs to two different separation conditions. In each condition, animals received one of three different treatments on each of three different days. The conditions were (1) infants separated physically from the visible mother by a wire-mesh cage, and (2) infants isolated physically from the mother who was not visible. On different days, the infants were injected with either a placebo, morphine, or naloxone (a morphine antagonist). Distress vocalizations were evoked under both separation conditions, but were greater when the infant was socially isolated. Distress vocalizations were significantly decreased by morphine as compared to the placebo control treatment under both conditions of separation, with the greater effect in the mother-absent group. Naloxone increased distress vocalizations compared to the placebo control in both kinds of separations. Infants in the mother-present group given a placebo emitted significantly less distress vocalizations than in the mother-absent group. The sight of the mother, despite the absence of physical contact, resulted in a significant decrease in distress. The generality of the experimental effect of separation of infrahuman infants from their mothers is supported by additional experiments with chicks (Panksepp *et al.*, 1978b) and puppies (Panksepp *et al.*, 1978a).

Separation stress explained in terms of a reduction in the level of endorphins is based on an inference from the opposite effects of naloxone and morphine on distress vocalizations. An experiment with chicks provides more direct evidence of the role of opiate-like peptides in separation stress as well as the interaction between behavior, brain chemistry,

and social attachment (Panksepp *et al.*, 1978b). Distress vocalizations in chicks isolated from their mothers were significantly reduced by peripheral and central injections of several different enkephalins and endorphins as well as morphine.

A different approach was adopted by Keverne *et al.* (1989). They assayed the cerebrospinal fluid (CSF) for endorphins in relation to sociality, attachment, and bonding between same-sex adult monkeys, broadening the investigation of the role of endorphins to social attachments beyond maternal–infant bonding. Monkeys living in isolation cages were permitted social interactions with a same-sex neighbor on alternate days. Grooming of the neighbor, behavioral overtures soliciting grooming, and occasional aggressive behavior were the kind of interactions taped by TV cameras and analyzed by observers blind to the experimental treatments. Significant increases in CSF endorphins occurred following grooming and interactions with another same-sex monkey following isolation.

A second experiment studied pairs of same-sex monkeys living together. The frequency of grooming was low. Naltrexone, a morphine antagonist, was given to half the partners; while the other half were given a placebo. Grooming invitations and time spent grooming their cagemate increased significantly in animals receiving naltrexone. Administration of an opiate antagonist produced behavior comparable to isolation. In both cases, the experimental treatment lowered endorphin levels which were significantly increased following grooming, suggesting that animals living in groups maintain high levels of endorphins as a result of their social interactions.

In a third experiment, a low dose of morphine was administered to half the cagemates following isolation; a placebo was given to the other cagemates. Morphine resulted in a significant decrease in overtures for grooming and in the amount of time spent on grooming. Social behavior in the form of grooming, touching, and physical contact increased markedly following social isolation. Social affiliation changed the neurochemical brain state of these animals, significantly increasing the concentration of endorphins and positive affect obtained from social contacts. The administration of morphine significantly decreased the grooming of cagemates and the sociality that it represents; loss of interest in

social affiliations also characterizes the behavior of addicts high on exogenous opiates.

A fundamental dialectical relationship is apparent between behavior, social affiliation, and the neurochemical state of the animal. Grooming, touching, and close physical contact raise the level of endorphins and are reinforcing, thus bonding social relationships. A high level of sociality maintains a high level of endorphins. The neurochemical brain state in part determines behavior, which reciprocally modifies the brain state along with social behavior. These results and their interpretation have obvious implications for risk factors contributing to the development of alcoholism, and for its treatment and prevention.

Separation Stress and Alcohol Consumption

Using rhesus monkeys, Higley *et al.* (1991) studied the effects of two kinds of separation stress on alcohol consumption and social interactions: (1) separation from the mother in infancy, and (2) separation from peers in adulthood. In the former condition, monkeys were randomly assigned at birth to a mother-reared (MR) or a peer-reared (PR) condition. In the MR condition, infants were raised by their mothers for approximately 7 months at which time they were weaned; they were then placed in a peer group and raised with other monkeys. PR monkeys were separated from their mothers at birth, raised with other monkeys, and fed Similac by human caretakers until the age of weaning. From the age of 7 months, the two groups were treated in an identical fashion.

In adulthood, 50 months, monkeys in both groups were given access to a red-colored sweetened solution of water containing 7% alcohol for 1 hour a day, 4 days a week, for 8 consecutive weeks. The first 2 weeks served as a baseline control phase; the final 2 weeks served as a postseparation phase. During the intervening social separation phase, monkeys were housed in individual plexiglass cages where they could hear but not see their cagemates. Sweetened green-colored water as well as the red-colored sweetened water–ethanol solution were available during the preseparation, separation, and postseparation phases.

PR monkeys consumed significantly more alcohol than MR animals during the preseparation baseline and postseparation recovery phases.

MR animals increased their alcohol consumption significantly during the social separation phase of the experiment occurring in adulthood, matching the PR animals during this phase. Mother rearing did not provide protection when these offspring encountered a different and very stressful separation from peers in adulthood.

Some monkeys consumed enough alcohol to fall, vomit, and lapse into unconsciousness despite having a choice of sweetened water. In addition to consuming more alcohol during the baseline periods, PR monkeys exhibited more behavioral and physiological signs of anxiety and fear during their home cage interactions. Average peak plasma cortisol concentration was positively correlated with average alcohol consumption. PR as compared to MR monkeys showed significantly higher plasma cortisol and corticotropin concentrations, indicating greater activation of the hypothalamic-pituitary-adrenal (HPA) axis and stress in PR monkeys.

A significant negative correlation between time in social affiliation and alcohol consumption was also found. The greater the amount of time engaged in affiliative behavior, the lower the alcohol consumption. Serotonin deficits were correlated with increased alcohol consumption during separation stress. Norepinephrine deficits were correlated with high levels of alcohol consumption during nonstress as well as separation stress periods.

Stable individual differences in alcohol consumption were apparent independent of rearing conditions and social separation. Infant monkeys having no contact with their father and nurtured by foster mothers showed significant correlations with parents in terms of metabolites of norepinephrine, dopamine, and serotonin as well as the stress hormones cortisol and corticotropin. Although serotonin levels appear sensitive to stress from infancy to adulthood, as the animals matured they were less disturbed by separation, as indicated by the decreased effect on dopamine and norepinephrine levels.

Animals with low levels of serotonin were at risk for impulsive aggressiveness and lack of social competence. PR as compared to MR animals engaged in more violent aggressive behavior and more infant-like ventral clinging. Serotonin level was positively correlated with social competence and negatively correlated with excessive

aggression, a relationship also obtained in human studies (Virkkunen *et al*., 1994).

Separation stress, from mother or peers, may be followed by heightened alcohol consumption and lower levels of serotonin. However, it cannot be concluded that low levels of serotonin caused the increase in drinking; other factors may also be at work. Separation stress activates the HPA axis, producing a momentary increase in beta-endorphin levels, the release of stress hormones, and a heightened level of orienting. Persistent stress results in lowered levels of endorphins and serotonin, which interact with and modulate dopamine in reaction to alcohol ingestion (Olausson *et al*., 1998). Consumption of alcohol under these circumstances may produce a disproportionately greater reinforcement from endorphins than normal. A positive family history of alcoholism may produce a similar effect (Gianoulakis *et al*., 1989, 1996).

Serotonin

A brief report by Knutson *et al*. (1997) shows that increasing levels of serotonin by administering a serotonin uptake blocker significantly increased affiliative behavior in a collaborative puzzle-solving task involving young adults. Pairs of participants worked on a collaborative puzzle where a placebo or serotonin uptake blocker was administered in a randomized double-blind fashion for repeated sessions over a 4-week period. Videotaping of the puzzle-solving behavior was rated for affiliative behavior in a double-blind manner. Significantly greater affiliative behavior occurred in the experimental member of the pair receiving the serotonin uptake blocker.

Oxytocin and Vasopressin

According to Panksepp *et al*. (1997), "all prosocial behaviors such as sexuality, maternal nurturance, separation-distress, gregarious-friendliness, social bonding, play, and social-memory systems share neurochemical controls, including prominently oxytocin and endogenous opioids" (p. 80). I would add serotonin, vasopressin, dopamine, and prolactin to this latter list.

Social comfort, support, and social attachments are mediated in part by the same neuropeptide: oxytocin, which is involved in giving birth, lactation, and sexual behavior. Oxytocin and beta-endorphins decrease aggression and infanticide in rats as well as serve as inhibitors of separation stress. Infusion of oxytocin facilitates nonsexual social contacts in both male and female infrahuman animals (Witt, 1997). In contrast to the effects of activation of the sympathoadrenal nervous system hormones energizing "fight or flight" behavior, oxytocin and vasopressin have a calming effect, reduce stress, and promote social bonding, further stimulating their release in a continuous dialectical interaction. Socializing effects and stress reduction are also mediated by prolactin and serotonin.

Approximately 3% of mammalian species are monogamous. Evidence suggests that bonding among all of these monogamous species may be mediated by a similar neurochemistry (Curtis & Wang, 2003). Research on two species of small rodents, monogamous prairie voles showing pair bonding and paternal care of offspring in contrast to promiscuous montane voles showing a lack of pair bonding and social attachments, has been an important source of information concerning the neurochemistry of social bonding.

Vasopressin is one of the neurochemicals involved in pair bonding, and is released in the septum region of the brain of a male prairie vole when exposed to a female vole. Vasopressin is released in neither the female vole nor the promiscuous montane vole. Further evidence that vasopressin mediates bonding in male prairie voles stems from research demonstrating that infusion of vasopressin into the brain of the prairie vole induces bonding in the absence of mating, whereas infusion of a vasopressin antagonist inhibits pair bonding (Bamshad *et al.*, 1993). Similar effects are not obtained in montane voles.

Oxytocin plays a major role in mediating parturition and lactation in all mammals. However, oxytocin is not the exclusive bonding neuropeptide in females, nor is vasopressin the social "glue" only in males. Injection of high doses of either neuropeptide can induce pair bonding in both sexes, and antagonists of each neuropeptide can block bonding in both sexes (Cho *et al.*, 1999). Oxytocin injected into the brain of male prairie voles can induce pair bonding just as readily as an injection of vasopressin. There may be, however, a sex difference in the amount of

neuropeptide needed to produce a bonding effect. The administration of dopamine may also facilitate pair bonding, and a dopamine antagonist may block pair bonding and mating (Wang *et al*., 1999).

A variety of experiments illustrate the kind of conditions inducing the release of oxytocin and its effects, aside from parturition and lactation in females. A rat experiment demonstrated that increased oxytocin levels induced by three different kinds of innocuous stimulation — electroacupuncture, warm temperature, and vibrations of the skin — significantly raised the threshold for pain as measured by tail-flick latency (Uvnas-Moberg *et al.*, 1993). Altemus *et al*. (1995) provide indirect evidence that oxytocin reduces exercise stress in postpartum breast-feeding women as compared to women bottle-feeding their infants. A quantifiable treadmill exercise was used as the stressor. Previous research has shown that such a stressor produces significant increases in plasma adrenocorticotropic hormone (ACTH), cortisol, vasopressin, norephinephrine, epinephrine, and prolactin. Results of the experiment by Altemus *et al*. showed that, prior to the stress-induced exercise, lactating women showed normal levels of ACTH and cortisol. However, as compared to bottle-feeding women, breast-feeding women showed lower levels of stress hormones following the stressful treadmill exercise, presumably because of their heightened levels of oxytocin mediating lactation.

A study by Heinrichs *et al.* (2003) using healthy young men demonstrated that the application of an intranasal spray of oxytocin as well as social support decreased salivary cortisol and verbal reports of stress following public speaking and a mental arithmetic task. Zak *et al.* (2004) found that trust in an economic exchange was related to increased blood serum levels of oxytocin; eight other hormones assayed failed to show any relation to trust or trustworthy behavior. Kosfeld *et al.* (2005) found that the application of an intranasal spray significantly enhanced trust in a variety of interpersonal monetary exchanges.

Prolactin and Touch

Wilson (2001) showed that prolactin mediates the effects of touch and stress reduction. She randomly assigned three groups of 70 young male

and female rats to a stressful situation, an open field. Group A animals were alone; animals in group S were in the open field separated by a clear plexiglass partition through which they could see and smell a same-sex conspecific; and group T animals were in the open field with a same-sex conspecific that they could touch. Following a period in which the animals were habituated to the open field, they were distributed to their randomly assigned conditions and a camera recorded their behavior for 10 minutes. Animals were then removed, decapitated, and blood obtained for prolactin assay. Results showed that animals allowed to touch had significantly lower levels of prolactin than the alone group and the group separated by a partition, thus allowing them to see and smell a conspecific; the latter two groups did not differ significantly in prolactin levels. Analyses of camera-recorded behavior showed that animals allowed to touch came together significantly more often and spent significantly more time together than animals separated by a partition who could see and smell each other.

We will now turn to an examination of the positive influence of a variety of different support groups. Research reviewed above suggests that their positive influence is due to increased levels of various neuropeptides interacting with behavior and the social environment.

Effects of Support Groups and Self-disclosure

Pain can kill (Liebeskind, 1991). As noted in Chapter 2, severe stress may facilitate the metastases of cancer cells. Reductions in stress and in excessive alcohol consumption may have widespread health benefits including slowing the metastases of cancer (Ben-Eliyahu *et al*., 1991, 1996). A groundbreaking study by David Spiegel and his colleagues (Spiegel *et al.*, 1981) introduced a social treatment program demonstrating the power of the group to significantly affect the quality of life, emotional state, and length of survival of women suffering from metatastic breast cancer.

Spiegel *et al*. (1981) randomly assigned 86 women with diagnosed metastatic breast cancer to an experimental group or a control group for a 12-month prospective study. Following informed consent, a battery of psychological scales was administered to each group and at

4-month intervals for 1 year. Both groups received standard cancer treatment. Women in the intervention condition, in addition, met in groups of 7–10 for 90 minutes on a weekly basis for 1 year. Each group had two leaders, a psychiatrist and a social worker or counselor who had breast cancer that was in remission. Surviving members maintained supportive informal social networks after the formal group meetings ended following completion of the data collection phase after 12 months.

> The groups were designed primarily to be supportive. There was a high degree of cohesion and relatively little confrontation and here-and-now interpersonal exploration. Interaction in the group often contained a considerable amount of self-disclosure and sharing of mutual fears and concerns. This atmosphere fostered frequent phone calls among members between meetings and cards and visits to members when they were hospitalized. Unlike a therapy group, there were few process interpretations; the focus was more on content, which included discussions of death and dying, related family problems, difficulties in obtaining treatment, issues of communication with physicians, and living as richly as possible in the face of a terminal illness. [Spiegel *et al*., 1981, p. 529]

The questionnaire battery administered by Spiegel *et al*. (1981) included self-report assessments of health; internal or external locus of control; and the Profile of Mood States (POMS) obtaining measures of anxiety, depression, anger, vigor, fatigue, confusion, and a total mood disturbance score. Scales also assessed self-esteem as well as maladaptive coping responses to manage stress such as eating too much, drinking too much, and smoking. An inventory of phobias and an assessment of denial were also administered.

Results for the subgroup of participants who completed all four administrations of the questionnaire battery were as follows: members of the intervention group as compared to control women had significantly less tension, depression, fatigue, confusion, maladjusted coping responses, and phobias, and more vigor. Only 52% of the original sample were able to complete the four administrations. A second analysis

was therefore conducted on each measure for each participant who completed at least two administrations of the questionnaire battery; 74% of the women participated. Intervention as compared to control group women were significantly less anxious, fatigued, phobic, and confused; used fewer maladaptive coping responses; had significantly lower overall POMS scores; and had greater vigor. Contrary to predictions, no differences between the two groups were found on measures of self-esteem, denial, or health locus of control.

Spiegel *et al.* (1981) suggest that the group experience helped participants focus and clarify the overwhelming problems they faced. It also avoided the isolation that may accompany the process of dying:

> . . . [T]he evidence from this study confirms the clinical observation of recent years that direct group discussion of terminal illness, doctor/patient relationships, the process of dying, and related family problems provides comfort and psychological support to patients and is not psychologically demoralizing or destructive. Objective as well as phenomenological measures demonstrate that patients given the opportunity of working together in facing common problems become less anxious, confused, fatigued, and fearful. [Spiegel *et al.*, 1981, pp. 532–533]
>
> Average survival rates following occurrence of the intervention phase differed significantly, 37 months vs. 19 months for the intervention and control groups, respectively. [Spiegel *et al.*, 1989]

Two basic variables are apparent in Spiegel *et al.*'s (1981) description of the groups at work: (1) verbal expression of emotion before a group of people with similar problems, and (2) social support and affiliation provided by the group. These factors appear to be responsible for the powerful nonspecific effects upon the biological state and sense of comfort of its members. It is expressed as a higher power, a power greater than themselves — it is the power of the neurobiological effect induced by affiliation. In 1935, Bill W (1994) succeeded in developing a powerful social force, the AA fellowship and its 12 Steps, based unknowingly upon the same fundamental biosociobehavioral processes manifest in Spiegel *et al.*'s (1981) groups — social affiliation, the overt expression of emotions,

and their neurobiological effects including increases in serotonin, endorphins, and oxytocin.

Some caution must be practiced in generalizing Spiegel *et al*.'s striking results. A relatively small sample of Caucasian women was employed; additional research is needed to assess the effects of similar processes varying in ethnicity, gender, age, and disease. Research repeatedly confirms the stress-reducing and emotional benefits derived from social affiliation and the expression of emotions, but less support is obtained for the results enhancing cancer survival rates.

Goodwin *et al*. (2001) studied a large multisite sample of women with metastatic breast cancer. Participants in the intervention group had significantly greater improvement in reported stress and pain, especially those women who reported the greatest stress at baseline. The survival rate, however, was not affected.

In commenting upon the Goodwin *et al*. (2001) study, Spiegel (2001) notes that there may now be a ceiling effect. Medical treatment of breast cancer has improved in the two decades since he initiated his study and Goodwin's more recent clinical trial. There are now many more support groups, so that women are less likely to suffer from the stressful effects of isolation and stigmatization; as a result, it is more difficult to show an effect of social support on the survival rate. Social support, however, continues to show beneficial effects on reports of emotional well-being.

Spiegel (2001) concludes,

> . . . [I]n group therapy a direct confrontation with the possibility of dying from metastatic breast cancer is emotionally helpful and not physically harmful. It is now well documented that the secrecy that surrounded cancer in the medical practice of yesteryear undermined rather than enhanced the patient's well-being. Facing the realities of metastatic breast cancer does not cause physical deterioration or hasten death. Indeed, the study by Goodwin *et al*. confirms that bearing and sharing all the emotions associated with advancing cancer in a supportive setting reduces distress and pain. The literature is divided on the question of a survival benefit, and more trials are being conducted, both in the United States and overseas. In the meantime, group therapy

> for patients with cancer can be prescribed for its psychological benefit, if not necessarily for any prolongation of survival. [p. 1768]

Research has also investigated the effect of the expression of stressful emotions on measures of the immune system and general health as well as self-reports of emotional well-being (Pennebaker *et al.*, 1988). Fifty healthy undergraduate college students were randomly assigned to a control or an experimental group. Control participants were asked to write about superficial experiences for 4 consecutive days. Experimental participants were asked to describe their deepest thoughts and feelings about some traumatic experience, ideally one that had not been discussed in detail with other people. Blood samples were obtained the day before the first writing day, the last of the 4 days, and 6 weeks after the last writing day. Psychophysiological and self-report measures were also obtained.

Assays showed that writing about traumatic experiences significantly enhanced cellular immune system function and reduced visits to the student health center because of illness. Two subgroups of experimental subjects were formed by categorizing students into high and low disclosers, depending upon whether or not they had written about topics that had not been previously disclosed to another person. It was found that high disclosers had a significantly higher antigen response than low disclosers and control subjects. During the course of the experiment, high disclosers also showed a significantly greater decline in systolic and diastolic blood pressure than the other two groups. Overall, the findings indicated that disclosure of traumatic experiences may prevent health problems by reducing stress and enhancing the immune system. Results showing a decline in blood pressure with disclosure are in accord with Lynch's (2000) extensive studies of communication and cardiovascular health and disease.

Esterling *et al.* (1994) adopted a somewhat different approach to the study of the effects of emotional disclosure. Instead of examining the effects of disclosure on nonspecific immune system responses, they investigated the effects of disclosure on the immunological response to a specific virus, a relatively common latent viral pathogen, the Epstein–Barr virus (EBV). It is a form of herpes virus that may lead to

infectious mononucleosis. Antibody responses to the EBV antigens reflect the effectiveness of the immune system's control of this virus.

A total of 72 college students met various inclusionary and exclusionary criteria for participation in the study; of these, 57 were seropositive for EBV and participated in all phases of the experiment. They were randomly assigned to one of three conditions: (1) provide a written description of stressful events, (2) provide a written description of a trivial event such as the contents of their bedroom closet, or (3) provide a verbal description of stressful events that was tape-recorded. There were three-weekly 20-minute sessions of the same kind. Participants in the written and verbal stressful conditions could describe either the same stressful experience or a different one in each session, as though it was to be presented to some one they could trust. Participants in the trivial written session were assigned a different topic each session. Blood for immunological assays was collected 1 week before the first of the three weekly sessions and 1 week after the final session. Prior to the first session, they were given a personality assessment designed to permit classification of the students into coping styles. Three groups were formed: repressor, sensitizer, and neither-personality styles. Sensitizers were students who met the criterion on any one or more of the scales designated as inhibited, forceful, or sensitive. Students classified as repressors met the criterion for one or more of the scales designated as introversive, cooperative, or respectful. Students who were neither repressors nor sensitizers demonstrated elevated scores on either or both scales designated as sociable or confident.

Results showed that the three most stressful personal experiences were described equally often by participants in the verbal and written stress conditions: death of a relative or friend, divorce of their parents, or termination of a romantic relationship. Conditions differed on a variety of self-report measures such as increased self-esteem, with greater increases in the verbal stress condition than written and control conditions, with no difference between the latter two conditions. Behavioral measures such as word count and judged emotionality of words showed that the verbal stress condition induced greater responsivity than the other two conditions. Measures of the immune system indicated significant differences in antibodies produced by the three personality styles.

Repressors produced significantly higher levels of antibodies than the sensitizer and neither-personality styles. Baseline levels of antibodies did not differ among participants assigned to the three conditions. There was no change in antibodies in the control group during the course of the study; whereas there was a significant decrease in antibodies in the two stress conditions, showing better cellular immune control. Following completion of the interventions, the written trivial condition participants showed significantly higher levels of antibodies than the two stress condition participants. Verbal stress participants showed better immune control of the EBV than participants in the written stress condition.

Although this is an interesting experiment showing that the strength of the immune system can be enhanced, it needs another control group: a trivial verbal expression condition. At present, the best intervention, verbal expression of stress, confounds verbalization *per se* with the expression of stress. A second control condition corresponding to the written trivial condition is needed: a verbal trivial condition. Would it affect the immune system? Probably not, if we can generalize from Lynch's (2000) data on blood pressure and communication, but it is a needed control condition.

Pennebaker and others have gone on to conduct a relatively large number of studies on self-disclosure effects (Pennebaker, 1997). They all have the same general characteristics: writing about emotional experiences that will remain confidential, and using nonclinical samples. Outcome results are relatively consistently positive. Bases for the effects remain a matter of conjecture, although Pennebaker offers a number of theoretical interpretations as have others (Bootzin, 1997). Whether confidential written statements about one's dependence or abuse of alcohol and other drugs would be affected by the procedures employed by Pennebaker is not known. Although Cross *et al*. (1990) obtained a confidential written statement from their patients, they did not randomly assign the task to some patients and not others, therefore not permitting an assessment of the effect of writing about emotional experiences on alcoholism treatment outcome. Controlled research on this problem is needed. Until that time, individuals making use of written self-disclosure in addition to formal treatment and/or participation in AA group meetings

would not hurt themselves and it could help. Frattaroli (2006) has provided an extensive review of studies of disclosure, its effectiveness, and possible moderators.

An interesting series of experiments was conducted by Fawzy *et al*. (1990a, 1990b, 1993) using a somewhat different intervention than the studies previously described. A 6-week psychiatric intervention provided multimodal behavioral treatments to individuals suffering from malignant melanomas. An experimental group received a psychiatric intervention in addition to the standard treatment received by a control group. Participants in the intervention met for six weekly sessions in groups of 7–10 people. Participants in the control group received only standard individual psychological and immunological assessments following surgery. A 6-month follow-up showed significant superiority of the experimental group in their affective state and elements of their immune system. A 5–6-year follow-up found a significant difference in survival rates: 3 of 34 participants in the experimental group had died, compared to 10 of 34 in the control group. Since a variety of different treatment modalities were employed in the intervention along with the development of social affiliations and support among the members of the small groups in the experimental condition, which element(s) of the intervention were responsible for the behavioral and immunological improvements cannot be discerned. The study does demonstrate that nonmedical behavioral treatments can improve the emotional and immunological states of individuals suffering from malignant melanoma and can prolong survival.

Cohen *et al*. (2003) collected data from 159 men and 175 women who were recruited for their study via newspaper advertisements, passed a medical examination, and were paid US$800 for their participation. Subjects were individually quarantined and exposed to a cold virus. For 5 subsequent days, they reported any cold symptoms. Virus cultures were also examined. Four weeks after exposure to the virus, a blood sample was obtained to test for an increase in viral antibodies or signs of viral infection.

Prior to exposure to the virus, scales of extraversion, agreeableness, and positive relationship style were administered providing an overall index of sociability. During the month prior to quarantine, social

interactions were assessed by telephone interviews three evenings a week. Social networks and social support were determined by means of standardized questionnaires. Hormone samples were obtained on the first day of quarantine. Antibodies specific to the viral infection were collected from nasal secretion samples obtained before and 28 days after exposure to the virus. Signs and symptoms of a cold were a significant increase in antibodies and reported symptoms of a cold and running nose.

Analyses of the results showed a significant linear relationship between increases in measures of sociability and decreases in rate of illness. Bases for the association between sociability and resistance against colds remain to be fully determined, but there is little doubt that the relationship is reliable and socially significant. There is also little doubt that important problems such as this one as well as many others in the domains of health, religion, affiliation, and alcohol misuse will not be explained by cognitive folk psychology using such notions as expectancy. In contrast, a biosociobehavioral disease conception of alcoholism opens the door to the study of these problems in relation to the etiology, characteristics, treatment, and prevention of alcoholism.

Alcoholics Anonymous and Social Affiliation

"Our hypothesis assumes that a type of endogenous endorphin addiction underlies social cohesiveness" (Herman & Panksepp, 1978, p. 213). Evidence now indicates (e.g. Panksepp, 1998) that social attachments and stress reduction are also mediated by oxytocin, prolactin, serotonin, and vasopressin. Research described earlier in this chapter also demonstrated that affiliation, the effect of other persons, and especially touch, physical contact, and meaningful face-to-face communications produce bodily states conducive to bonding and well-being (Bowlby, 1982; Gantt *et al.*, 1966; Harlow, 1958; Heinrichs *et al.*, 2003; Kosfeld *et al.*, 2005; Liddell, 1956; Lynch, 2000; Panksepp, 1998; Pavlov, 1928; Wilson, 2001; Zak *et al.*, 2004). My hypothesis is that these same behavioral and neurobiological processes underlie active participation in 12-step groups.

Pagano *et al.* (2004) utilized the Project MATCH (Matching Alcoholism Treatments to Client Heterogeneity) database to conduct a

prospective study of helping, as reflected by serving as a sponsor to another AA member. Controlling for the number of AA meetings attended, Pagano *et al*. reported that 9% of the participants in AA served as sponsors for another member by the end of the treatment phase. Such sponsors were significantly less likely to have relapsed 1 year later than nonhelping participants. Helping someone else appears to help oneself. I hypothesize that the positive helping effect is a consequence of increased levels of neuropeptides such as endorphins, serotonin, oxytocin, and prolactin.

AA works in part because it provides a platform for the expression of stressful emotions, in addition to promoting social affiliation. Similar to Spiegel's (1981) groups, in an AA meeting a person stands before a group of sympathetic people bonded by common problems and describes their most painful experiences. Audience members respond with support, hugs, touching, and a demonstration of fellowship. Physiological states induced by affiliation are incompatible with pain, fear, and stress. Increases in serotonin, oxytocin, vasopressin, and endorphins occur with a concomitant decrease in cortisol, anxiety, and blood pressure induced by stressful situations or by thinking about such situations. As a consequence of involvement in AA groups, the serotonin-limbic-HPA axis as well as cardiovascular and immune systems are reset and normalized. Abstinence may also provide time for recovery of brain function, including executive functions, varying as always with individual differences in the host and social environment.

These notions are testable. Studies employing ambulatory recordings of heart rate, blood pressure, and electroencephalography (EEG) as well as measures of salivary cortisol and assays of serotonin, norepinephrine, endorphins, and oxytocin need to be obtained before and after an individual attends and speaks at an AA meeting, obtains a sponsor, or becomes a sponsor. Similar assessments should be obtained from comparison groups consisting of randomly selected individuals matched for age, gender, ethnicity, and socioeconomic class before and after attending a comedy, action, or dramatic play. Ideally, a prospective study is needed. Physiological recordings and assays must be obtained prior to an individual's participation in AA meetings, after the first meeting, and after repeated meetings over the long term, comparing individuals who continue participating in AA

and those who drop out. Assessments of alcohol and other drug use along with quality-of-life measures should be obtained throughout the study.

Similarly, studies obtaining ambulatory recordings and assays of neurotransmitters before, during, and after attending religious services of different religious groups would be of interest. Laboratory experiments need to be conducted analogous to Pennebaker's (1997) procedure in which an individual gives a speech presenting his or her most painful experiences, knowing that no one will hear the speech except an experimenter who is blind to the speaker's identity. The speaker must first be habituated to the laboratory surroundings and forewarned that at future sessions they will be asked to speak publicly. Ambulatory recordings should be obtained from the participant before, during, and after the presentation, as should blood samples for assaying levels of endorphins, oxytocin, prolactin, vasopressin, etc.

An add-on design experiment needs to be conducted using Pennebaker's method of confidential writing about stressful emotional experiences along with AA participation. Such a study could employ two randomly assigned groups: (1) Pennebaker method plus AA participation, and (2) AA participation alone. A similar design needs to be employed with AA and attendance at religious services; in both cases, measures of involvement in AA and religiosity/spirituality need to be obtained along with measures of frequency of attendance at meetings and services.

Much of the research on bonding and attachments suggest the importance of touch and cutaneous contact in the normal development of infrahuman animals and humans. Studies of biological processes; separation stress; the activation of endorphins, growth hormones, oxytocin, vasopressin, and prolactin; and decreases in cortisol and other stress hormones are primarily studied in infrahuman animals for obvious ethical reasons. Much can be done, however, by way of studying the biological processes in adult human affiliation to confirm the hypothesis that similar biological processes underlie adult human affiliations and attachments found in infrahuman animals. Fortunately, a start has been made (e.g. Heinrichs *et al.*, 2003; Knutson *et al.*, 1997; Zak *et al.*, 2004).

Cue Exposure and Response Prevention

Cue exposure and response prevention is a procedure used with some success in the treatment of phobias. As is true of so many other methods employed by behavior therapists and cognitive behavior therapists, it was first described many years ago by Guthrie (1935). It is a method for extinguishing an undesirable response. As is true of so much of current cognitive psychology, it repeatedly rediscovers fire, or the wheel, and renames the "discovery".

In the classic procedure for extinguishing a dirt phobia, reflected by repeated hand washing, the patient may be shown a dirty rag and rubs their hands with it, but is not allowed to wash their hands. Exposure to the cue and prevention of the undesirable response eventually results in extinction of the response of hand washing and, presumably, of the underlying fear of dirt. Cue exposure and response prevention when applied to alcohol and alcoholics has had mixed results, in part because of poor experimental methodology (Maltzman & Marinkovic, 1996); nevertheless, it has been extensively promoted by cognitive therapists in the United Kingdom who were among the first to employ the procedure with alcoholics. Because of the limited success of the method, it has more recently been proposed that cue exposure and response prevention should be employed in actual drinking situations to enhance its external validity (Hodgson, 2001). Variations in the procedure and its use as an adjunct to other cognitive behavior therapy modules are described at length by Monti and Rohsenow (2003). Cognitive behavior therapists overlook research demonstrating that desensitization of a phobia involves activation of endorphins, a change in neurochemistry (Egan *et al.*, 1988).

Cognitive behavior therapists also fail to recognize that cue exposure and response prevention is not the most effective extinction method available. A more effective extinction procedure is conditioning an explicit competing and stronger response than the undesirable response evoked by the CS, a distinction made years ago by Guthrie (1935). One such competing response extinction procedure is the highly successful Schick Shadel method of aversion conditioning described in Chapter 2. Another competing response extinction method is adapted to humans capable of

speech, semantic conditioning, and generalization of response acquisition and extinction (Maltzman, 1968; Razran, 1961); Soviet psychologists initiated the use of this method in the 1920s. Unfortunately, the "cognitive revolution" ignores this important area of research.

Since its inception in 1935, the AA fellowship has unknowingly employed semantic conditioning and generalization of extinction in their use of semantic cue exposure and response inhibition. It was used unknowingly in Spiegel *et al.*'s (1981, 1989) pioneering studies of support groups. In AA meetings, an individual — in the presence of strong social support — talks about their most difficult, embarrassing experiences when under the influence. The HPA axis stress response ordinarily induced by such semantic cues is inhibited by the heightened levels of endorphins, serotonin, and oxytocin induced by the presence of the support group. There is no need to show an individual a glass of their favorite alcohol beverage, to have them smell, taste, or sip but not continue to drink it, or to produce nausea in the presence of these cues in order to extinguish drinking. Humans can talk about the experiences they had under the influence of alcohol, and imagine those experiences in the presence of competing neurochemical states and behavior induced by caring, sympathetic people who have suffered through the same experiences. These competing neurochemical states inhibit the undesirable behavior (i.e. alcohol consuming and thinking about drinking) by modifying the dysfunctional neurochemistry producing the undesirable behavior. The establishment of lifelong friendships and healthy patterns of behavior sustain the normalized neurobiological states.

Critics assert that a large percentage of people do not continue in AA because they object to references to a higher power, God, and the religiosity/spirituality of meetings. It is possible that such people can "shop around" and find AA groups more in accord with their beliefs and attitudes. It is also possible that alternative interpretations may be constructed more in keeping with their beliefs. One is that "higher power" may be interpreted as a verb, a way of behaving, performing good deeds, helping others, rather than a noun that is reified. A related interpretation in keeping with our discussion of attachment and its biological correlates is B. F. Skinner's (1987) view presented in Fig. 3.1, emphasizing the group as the higher power.

A Humanist Alternative to A.A.'s Twelve Steps

A human-centered approach to conquering alcoholism

By B. F. Skinner

Several people have told me that they turned to Alcoholics Anonymous for help but have been offended by its heavily religious character. In view of this, I have proposed a humanistic alternative to A.A.'s "The Twelve Steps." I sent this version to Alcoholics Anonymous, suggesting that they offer it as an alternative for nonreligious members. I was not suggesting that they abandon their own twelve steps. I was told, however, that it would be impossible to change their practices without a majority vote of all Alcoholics Anonymous and was assured that many atheists and agnostics have found the original twelve steps helpful. Humanist counselors may, nevertheless, find an alternative version useful. Below are listed both "The Twelve Steps" of Alcoholics Anonymous and my suggested alternative:

THE TWELVE STEPS	THE HUMANIST ALTERNATIVE
1. We admitted we were powerless over alcohol . . . that our lives had become unmanageable.	1. We accept the fact that all our efforts to stop drinking have failed.
2. Came to believe that a Power greater than ourselves could restore us to sanity.	2. We believe that we must turn elsewhere for help.
3. Made a decision to turn our will and our lives over to the care of God *as we understood Him.*	3. We turn to our fellow men and women, particularly those who have struggled with the same problem.
4. Made a searching and fearless moral inventory of ourselves.	4. We have made a list of the situations in which we are most likely to drink.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.	5. We ask our friends to help us avoid those situations.
6. Were entirely ready to have God remove all these defects of character.	6. We are ready to accept the help they give us.
7. Humbly asked Him to remove our shortcomings.	7. We earnestly hope that they will help.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.	8. We have made a list of the persons we have harmed and to whom we hope to make amends.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.	9. We shall do all we can to make amends, in any way that will not cause further harm.
10. Continued to take personal inventory and when we were wrong promptly admitted it.	10. We will continue to make such lists and revise them as needed.
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.	11. We appreciate what our friends have done and are doing to help us.
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.	12. We, in turn, are ready to help others who may come to us in the same way.

B. F. Skinner, 1972 Humanist of the Year, continues his research and writing at Harvard University.

FIGURE 3.1. B. F. Skinner's "humanistic" interpretation of AA's 12 Steps.

It must be recognized that no good data have been provided demonstrating that the purported high dropout rate from AA is due to dissatisfaction with its emphasis on religiosity/spirituality and a higher power. Laudet (2003) surveyed clinicians and patients in five different outpatient 12-step–oriented treatment programs in New York City. Her findings indicated that the principal obstacles to participation in 12-step groups are not related to the nature or content of the meetings, but to problems of motivation, convenience, and scheduling problems. These are obstacles that would hinder participation and recovery in any outpatient treatment program or support group.

Religiosity/Spirituality

Affiliation, Pavlov's presence of the person (Gantt *et al*., 1966; Pavlov, 1928), has a significant impact upon health. Our interpretation of AA is that it promotes sobriety and well-being through social relationships that influence the biological state of the individual, which in turn promotes social relations. There is a dialectical interaction between biology, behavior, and social relations. A relatively large literature is now available showing that social relationships promote health (Ryff & Singer, 2001). A related literature indicates that religiosity/spirituality also promotes health, reducing the rate of morbidity and mortality (Fontana, 2003; Kark *et al*., 1996; Koenig, 1997; Plante & Sherman, 2001). Finally, research indicates that religiosity promotes a clean and sober lifestyle (Pardini *et al.*, 2000; Wills *et al.*, 2003). A prospective study of older adults over a period of 12 years found that attending religious services more than once a week predicted lower mortality (Lutgendorf *et al.*, 2004). Blood samples provided assays of a complex cytokine (interleukin-6) produced by the immune system that is elevated in various kinds of stresses and illnesses. Correlational analyses conducted by Lutgendorf *et al*. suggested that cytokines may mediate the relationship between religious participation and mortality. Whether the critical factor is religiosity *per se* or the participation in a group activity remains an open question.

Kendler *et al*. (2003) found seven factors in their analysis of the results of a questionnaire on religiosity obtained from 2600 male and female twins. Five of these factors are associated with a reduced risk for alcoholism. How do such paper-and-pencil measures of "general religiosity"

and "thankfulness" come to be negatively correlated with paper-and-pencil measures of alcohol dependence? Our interpretation is that attendance at religious services and related religious activities promotes social affiliation, attachments, and the related positive biological states. Whether or not more is involved is difficult to determine because of the complexity of the notions of religiosity/spirituality and the possible influence of multiple correlated factors. Adequate explanations for the relationship remain to be determined. As previously noted, studies obtaining ambulatory recordings of EEG and autonomic measures before, during, and after attending religious services from different religious groups are needed as well as measures of neurotransmitters, moderators, and the immune system. This is an interesting and important area that may be studied by a variety of methods, requiring a multidisciplinary approach.

Kaskutas *et al.* (2003) studied religiosity and spirituality in relation to AA involvement. Their aim was to determine what influence these have on long-term sobriety. They recruited a representative sample of 587 men and women from public, health maintenance, and private treatment programs in a heterogeneous Northern California county. Participants were interviewed at the start of treatment (T1), and again 1 (T2) and 3 (T3) years later. The aim of the project was to determine whether individuals who are neither religious nor spiritual can become involved in AA, maintain their involvement, and achieve long-term sobriety as compared to those who profess greater religiosity and spirituality. Briefly, the answer is yes, they can.

Kaskutas *et al*. (2003) distinguished between religiosity and spirituality by categorizing participants as follows: (1) atheists, who do not believe in the existence of God; (2) agnostics, who believe that it is impossible to determine whether or not God exists; (3) the spiritual, who believe in God but do not engage in religious practices; (4) the religious, who believe in God and engage in religious practices; and (5) the unsure, who are uncertain as to what to believe about God's existence. Because of the small number of participants, the atheist and agnostic groups were combined into a single secular group. Based on the following question, a measure of spirituality was obtained at baseline and at T2 and T3 follow-up interviews: "Have you had a spiritual wakening as a result of your involvement in AA?" (p. 5)

The severity of alcohol problems was assessed by the Addiction Severity Index, categorizing participants on the basis of a composite alcoholism score. Involvement in AA was measured by items from the AA Affiliation Scale, such as the number of AA meetings attended in the 12 months prior to T1, T2, and T3; currently having a sponsor or being a sponsor; and having read any AA literature during the period in question. The basic measure of remission was total sobriety for the previous 12 months at T3. Using this criterion, 35% of the sample had been sober for at least 12 months.

Results assessing religiosity indicated that 38% were religious, 44% spiritual, 9% secular, and 9% unsure. Secular participants showed significantly higher alcohol problem severity at T3 than spiritual participants. At T1, more of the spiritual and religious participants reported a spiritual awakening than secular and unsure individuals. At T1, approximately half of each of the groups categorized by religiosity attended AA meetings during the 12 months prior to the start of treatment. Attendance at meetings declined in all groups over the 3-year period. It is noteworthy that the spiritual approach of AA did not deter "nonbelievers" from attending meetings. By the end of T3, the average number of meetings per month, two, by the atheistic/agnostic group was the same as that of the religious group; the unsure group approximated the spiritual group, with an average of three meetings per month.

The increased number of AA meetings attended between T1 and T2, but not between T2 and T3, predicted sobriety at T3. Having experienced a spiritual awakening at T3 was also a significant predictor of sobriety at T3. Twice the percentage of people reporting a spiritual awakening at T3 (40%) were sober for the previous 12 months than those who did not have a spiritual awakening (20%). Finding that increases in AA attendance and involvement between T1 and T2 promoted sobriety at T3 suggests "the primacy of AA involvement immediately following treatment entry, and highlights the value of treatment programs getting patients involved in AA as part of the treatment experience" (Kaskutas *et al*., 2003, p. 14). Results reported by Kaskutas *et al*. concerning the relationship between spirituality and sobriety certainly warrant further research from a variety of perspectives.

Zemore and Kaskutas (2004) expanded the scope of analysis of the relationships among spirituality, AA participation, helping, and sobriety by examining the changes among these variables over an extended period of sobriety. They found that long-term sobriety involved a greater amount of time spent in helping in community activities and less in recovery-oriented helping. Over time, the nature of the spirituality and the kind of helping change along with the kind of involvement in AA: helping, spirituality, and involvement in AA are not static states, but changing processes that are in continuous interaction. Zemore and Kaskutas recognized that a shortcoming of this interesting study is that it was a panel study; different groups were studied at different stages of sobriety. A longitudinal study is needed. Hopefully, this team will conduct such a long-term study, and when they do they will add pharmacological assessments in order to complete the biosociobehavioral loop.

Members of AA believe that a spiritual awakening characterizes long-term sobriety. Our prior discussion suggests that increases in serotonin, endorphins, oxytocin, and normalization of the HPA axis may be necessary conditions for an experience of a spiritual awakening. Much research needs to be done on the association between biosociobehavioral changes and long-term sobriety as experienced by active participants in AA, other successful treatment approaches, and spontaneous remission. Neurohumoral changes may be necessary for the experience of spirituality and a spiritual awakening, but they may not be sufficient. Spirituality may be the consequence of changes in the cerebral cortex, primarily the temporal lobes. Just as the brain is the victim of damage produced by excessive alcohol consumption, it may also be the source of the spiritual awakening signaling the initiation of recovery. Neurohumoral changes and spirituality may be a top-down effect (Ramachandran & Blakeslee, 1999).

Conclusion

The lesson implied by all of the above is embodied in part by Sir William Osler's aphorism (Bean & Bean, 1950), which may be paraphrased as, "You must treat the person rather than the disease." Cognitive behavior therapists have yet to learn this lesson.

4

Expectancy Theory and Research: Balderdash!

> [T]he day may come when the social community of scientists will consist mainly or exclusively of scientists who uncritically accept a ruling dogma. They will normally be swayed by fashions. And they will accept a theory because it is the latest cry, and because they fear to be regarded as laggards.
>
> I assert, however, that this will be the end of science as we know it. . . . As long as science is the search for truth, it will be the rational critical discussion between competing theories. . . . [Popper, 1962, p. 57f]

The Balanced Placebo Experiment: Dogma Rules

Unfortunately, the day in question is here in the alcoholism treatment field as a practice and research specialty within psychology. Uncritical acceptance of the ruling dogma of "expectancy theory" by cognitive behavior therapists, probably the dominant approach within academic clinical psychology, has made this unfortunate state of affairs possible. Evidence supporting our assertion follows.

In 1995, *Alcohol Health & Research World*, the magazine published by the National Institute on Alcohol Abuse and Alcoholism (NIAAA), printed an issue celebrating the 25th anniversary of the founding of the institute. It included a section titled, "Seminal articles in alcohol research": "This special section features commentaries from experts in

the alcohol field on original articles that have described some of the most important alcohol research of the past four decades . . . " (*Alcohol Health & Research World*, 1995, p. 3). One of the 16 honored articles was the study by Marlatt *et al*. (1973); it is represented by Miller's (1995a) commentary extolling its originality and impact on subsequent alcoholism research and theory.

Miller's (1995a) approximately 1½-page commentary contains numerous errors of omission and commission. All of them are in one direction, praising the fundamentally flawed study by Marlatt *et al*. (1973), misinterpreting follow-up experiments, and omitting studies with contradictory results and alternative interpretations. Miller's commentary promotes the notion that alcoholism is a form of learned behavior no different than any other kind of learning, except for the intrapersonal and interpersonal damage produced as a consequence of this bad habit. Marlatt *et al*.'s research is recognized as a major force in overthrowing the dominance of the so-called medical model disease conception of alcohol.

According to Miller (1995a),

> When this landmark article was published in 1973 the dominant model of alcoholism considered it a dispositional disease the cardinal symptom of which is the inevitable loss of control whenever alcohol is consumed. . . . A few controversial voices in the alcohol field, including Jellinek . . . had questioned the scientific accuracy of the disease model as a universal description of alcoholism. [p. 36]

Note first that Miller (1995a) does not define alcoholism. Second, Jellinek (1960) said that there were several kinds of alcoholics, including alpha and beta alcoholics who today would be classifiable in DSM-IV as alcohol abusers; for simplicity, we call them problem drinkers. Jellinek also stated that gamma alcoholics were the primary kind of alcoholics found in North America who would qualify as victims of alcoholism disease; they would be classifiable according to DSM-IV as alcohol-dependent, with withdrawal experiences and a high degree of tolerance. The pathognomic sign and symptom that differentiates gamma alcoholics from alcohol abusers is loss of control over drinking. Jellinek (1960) took

pains to clarify his disease conception: loss of control was not inevitable after a single drink. He stated that learning and social factors were important in the development of problem drinkers and gamma alcoholics. Jellinek also suggested that loss of control might be the consequence of alcohol damage to the frontal lobes, as described by Lemere (1956) based on autopsy studies. Jellinek, incidentally, was not a doctor of medicine (MD), but a biostatistician by training.

Miller (1995a) goes on to state that, at the time of Marlatt *et al.*'s (1973) landmark study, the essence of alcoholism was a "biomedical abnormality inexorably rooted in the alcoholic's constitution" (p. 36). This is a confabulation contradicted by Jellinek's (1960) pre-eminence in alcoholism studies at the time. It was Jellinek's formulation of loss of control as the pathognomic sign of the disease of gamma alcoholism that Marlatt *et al*. were attempting to test in their balanced placebo experiment.

Miller (1995a) further states,

> This innovative experiment by Marlatt and his colleagues put this assumption about alcoholism to the test by studying whether behavior changes resulted from the actual *presence* of alcohol or from the *belief* that alcohol was present. The authors introduced two novel research methods . . . both of which inspired many subsequent studies. The first of these was the taste-rating task, in which subjects made up of both alcoholics and social drinkers, were asked to taste and compare three ostensibly different beverages. . . . The actual purpose of the task was to study the amount and manner of drinking the subjects did without making them self-conscious that their drinking was being monitored. Later research has shown that this clever procedure does, in fact, mirror a person's real-life drinking habits. It also is clear from two decades of subsequent studies that this unobtrusive measure is useful in gauging how a person's drinking is affected by social and environmental factors. [p. 36]

There are no citations to support the above assertions concerning the common use of the taste-rating procedure in experimental studies of alcohol effects. None of the major experimental studies attempting to replicate Marlatt *et al.*'s (1973) results used the taste-rating procedure

(Berg *et al*., 1981; Korytnyk & Perkins, 1983; Laberg, 1986; Stockwell *et al*., 1982; Wigmore & Hinson, 1991).

> The second innovative method introduced in this study was the balanced placebo design. . . . This study's central finding was that the subject's *belief* that he was drinking alcohol, rather than its actual presence, determined the amount he consumed on the taste-rating task. This effect was found for both alcoholics and social drinkers, although the difference was greater for alcoholics. As a result of Marlatt and colleagues' demonstration, the balanced placebo design became a common research tool in the alcohol field. Dozens of subsequent studies have shown that it is the subject's expectation that alcohol is present rather than the actual presence of alcohol that influences a broad range of social 'effects' of drinking, including aggression, humor, sexual arousal, and anxiety. Such studies also have shown that it is mostly the amount, rather than the expectation, of alcohol that causes impairment on motor and memory tasks. *In a direct replication of Marlatt and colleagues' classic study, Stockwell and colleagues (1982) reproduced the findings in their study of dependent drinkers* [emphasis added]. However, Stockwell and colleagues reported that the presence of alcohol became a significant predictor of craving for severely dependent drinkers. [Miller, 1995a, p. 36f]
>
> Marlatt and colleagues classic study questioned . . . the adequacy of this disease view of alcoholism. Perhaps the puzzling drinking behavior of alcoholics should be understood not as the inescapable product of a mysterious physical defect but rather as a modifiable behavior responsive to the same, learning, cognitive, and psychosocial principles to which all behavior is responsive. . . .
>
> Thus what was viewed in 1973 as an [*sic*] unitary medical disease is now understood in a broader and more complex context. This study opened one important door toward this integration of psychosocial and biomedical factors. [Miller, 1995a, p. 37]

It also opened an important door for controlled drinking. An implication of loss of control in alcoholics is that abstinence must be the treatment of choice. If, however, alcoholism is nothing but a form of social learning, the implication follows that control can be learned and that

controlled drinking is a reasonable goal (Cummings *et al*., 1980), unless serious medical conditions prohibit alcohol consumption.

We have to examine what Marlatt *et al*. (1973) did in their experiment, note its fundamental shortcomings, and review the studies attempting to replicate the experimental results. We will find that subsequent experiments failed to reproduce Marlatt *et al*.'s results, including Stockwell *et al.*'s (1982) study which is inaccurately described by Miller (1995a). We must also consider a study by Korytnyk & Perkins (1983), which is never cited by cognitive behavior therapists and other revisionists; it supports an alternative interpretation of Marlatt *et al*.'s results, i.e. the demand characteristics of the experimental situation (Orne, 1962). Both studies (Korytnyk & Perkins, 1983; Stockwell *et al*., 1982) are clear discomfirmations of the expectancy hypothesis. The fundamental assumption, or rather dogma, of Marlatt, Miller, and other ideologs (e.g. Fingarette, 1988) is false. Social drinking and alcoholism are not on a continuum differing only in terms of the amount of alcohol consumed or the number of alcohol-related problems. This "social learning interpretation" has been repeatedly contradicted by behavioral studies conducted by British psychologists, including the study by Stockwell *et al*. (1982) misrepresented by Miller (1995a).

A neurological basis for alcoholism suggesting that the pathognomic symptom of loss of control is due to damage to the frontal lobes (Lemere, 1956) was available at the time Marlatt *et al*. (1973) published their study. Subsequent extensive infrahuman and human research support the hypothesis that the behavioral symptoms of alcohol abuse and alcohol dependence are at least in part a consequence of structural and functional brain damage (e.g. Crews, 1999; Moselhy *et al*., 2001).

It must also be noted that, contrary to Miller (1995a), Marlatt *et al*. (1973) did not devise the balanced placebo design. It was first employed by Ross *et al*. (1962) in a study of amphetamines. Marlatt *et al*. were the first to use the design with alcohol.

A Critical Examination of Marlatt et al. *(1973)*

Marlatt *et al*. (1973) designed their experiment to test Jellinek's (1960) formulation that loss of control over the amount of alcohol consumed is

the pathognomic or defining sign and symptom of gamma alcoholism, yet they made no attempt to obtain participants who met the criteria of gamma alcoholism. To qualify as an alcoholic in the Marlatt *et al*. study, an individual had to meet one or more of three criteria: (1) patient in an alcoholism treatment program, (2) five or more arrests for "drunk and disorderly conduct", and (3) participation in Alcoholics Anonymous (AA) or in the local Vocational Rehabilitation for Alcoholics Program. Seventy-one percent of participants in the alcoholic group qualified as alcoholics for the experiment on the basis of two or more of these criteria.

If an individual met any one of the above criteria and was not abstinent, had consumed alcohol within the previous 2 weeks, and had no intention of attempting abstinence, they were accepted for inclusion in the alcoholics group. None of the alcoholic participants need have experienced withdrawal symptoms or extreme tolerance or evidenced loss of control, the necessary criteria for gamma alcoholism. No detailed reports were obtained concerning the frequency and quantity of alcohol consumed or the amount consumed on a typical drinking day within the recent past. No assessment of negative consequences was obtained, for example within the past 6 months, as an independent means of determining whether participants met the criteria for alcoholism as defined by Jellinek, whose hypothesis they were attempting to test.

Social drinkers were defined as any one who did not abstain and who did not meet any of the criteria for an alcoholic as defined above. Social drinkers were excluded from the study if they reported heavy drinking or considered their drinking a problem.

The experiment was introduced with a primer of 1 oz of vodka mixed with tonic; it was followed in 15–20 minutes by the taste rating of different drinks of vodka and tonic or tonic alone. Alcoholics who were told alcohol/given alcohol consumed 22.13 oz, as compared to 23.87 oz for alcoholics who were told alcohol/given tonic. In contrast, alcoholics who were given alcohol/told tonic consumed 10.25 oz, as compared to 10.94 oz for the alcoholics who were told tonic/given tonic. The overall mean was 23.00 oz for the told-alcohol groups vs. 10.60 oz for the told-tonic groups. It is apparent that what alcoholics were told had a significant differential effect, whereas what they were given did not have a differential effect. Marlatt *et al*. interpreted their results as demonstrating

that what an individual expects, not the pharmacology of alcohol, determines their drinking behavior.

The results were presented as a contradiction of Jellinek's formulation of loss of control as the pathognomic sign and symptom of alcoholism and as the initiation of the chronic phase of alcoholism. It must be kept in mind that the experimental variables in the study were the type of instructions and beverage. Expectancy is an interpretation of the effects of instructions, a hypothesis.

Demand Characteristics: An Alternative Interpretation

There is an alternative interpretation of the effects of instructions that Marlatt, Miller, and other revisionists never take into account: demand characteristics. Orne (1962) describes the psychological experiment using human participants as a social situation in which there is a difference in social status (i.e. power) between the experimenter and the participant. In such a situation, the participant will attempt to please the experimenter by generating the results that the participant believes are desired by the experimenter. A fundamental difference between the expectancy and demand characteristics interpretations is that according to the demand characteristics interpretation, the instruction effect is peculiar to the social situation of the usual laboratory experiment with its difference in power and status between the investigator and the participant; the conception of expectancy, in contrast, is generalizable to behavior outside the laboratory.

Evidence in support of a demand characteristic interpretation of the effects of instructions in the balanced placebo experiment, contradicting the expectancy interpretation, has been provided by Korytnyk and Perkins (1983). A balanced placebo design was used with male college students as participants. Students were given the impression that the experiment in which they were participating on the effects of alcohol on problem solving was interrupted by an important telephone call their instructor had to answer in a different room. In the absence of the experimenter and therefore in the absence of demand induced in the experimental situation, students scribbled graffiti on the classroom walls already littered with such trash. More graffiti was produced by participants

administered with alcohol than with tonic, regardless of the instructions concerning drink content. Korytnyk and Perkins' experiment needs to be replicated using alcohol consumption as the dependent variable; nevertheless, the results clearly contradict the implications of an expectancy hypothesis.

Wigmore and Hinson (1991) employed a $2 \times 2 \times 2$ balanced placebo design that included a dimension of context, thus approximating the necessary extension of Korytnyk and Perkins (1983). Wigmore and Hinson conducted their balanced placebo experiment either in a laboratory characteristic of most balanced placebo studies or in an actual bar room. Results obtained in the laboratory replicated the usual general finding: an instruction effect was obtained whereby participants consumed more beverage when they were told that it contained alcohol than when they were instructed that the drink did not contain alcohol, regardless of actual alcohol content. "Consumption in the bar-room setting was higher than in the laboratory setting, and most importantly, consumption was not differentially affected by instructions about alcohol content or actual alcohol content" (Wigmore & Hinson, 1991, p. 205). In other words, the so-called expectancy effect was not obtained when demand characteristics were absent. An instruction-induced expectancy effect does not generalize to the world outside the demand characteristics of the laboratory.

Stockwell *et al*. (1982) employed a $2 \times 2 \times 2$ within-subject balanced placebo design to test the expectancy hypothesis. Half of the participants on a given day received alcohol and tonic, while the other half received tonic, as their priming dose. Half of each group was told that the drink was vodka and tonic, and half was told that it was tonic only. Each of the participants served in each of the subgroup cells on different days. Half of the participants in the experiment were severely dependent alcoholics, and half were moderately dependent. All were hospitalized for treatment of alcoholism, had completed their withdrawal period, and volunteered for the experiment. They had been screened for heart and liver conditions, brain damage, and psychosis. Independent psychiatric assessments determined the levels of dependence severity as in the study by Hodgson *et al*. (1979).

A variety of self-rating, psychophysiological, and behavioral measures were obtained before and after the priming dose. Approximately

1 hour after the priming dose, participants were given two glasses of vodka and tonic. The speed of consuming the first vodka and tonic was the principal dependent variable.

Results showed that instructions did not have a priming effect on severely dependent alcoholics. They consumed the test vodka and tonic faster when they were previously given alcohol as compared to tonic alone, regardless of what they were told about the contents of the drink. Results for the severely dependent participants contradicted the expectancy interpretation of instructions, and failed to replicate the results obtained by Marlatt *et al*. (1973) with their purported alcoholics. These results contradict Miller's (1995a) comment quoted earlier, asserting that Stockwell *et al*. (1982) "reproduced the findings in their study of dependent drinkers". Moderately dependent participants (i.e. problem drinkers), in contrast, showed an instruction effect: what these subjects were told about the priming drink had a significant effect on the speed of consuming the test vodka and tonic as compared to what they were given, replicating the kind of results obtained by Marlatt *et al*. (1973).

It should be noted that this carefully controlled study by Stockwell *et al*. (1982), with extensive measures and independent assessments of severity of dependence, used a simple, previously validated behavioral measure — speed of consumption of the first drink in the test situation — as their primary dependent variable. They did not use the "innovative" taste-testing procedure that Miller (1995a) gratuitously credits with widespread adoption. Miller also misrepresented the results of Stockwell *et al*. that contradict the findings of Marlatt *et al*., disconfirming the expectancy interpretation of the balanced placebo experiment. Alcohol consumption by severely dependent alcoholics was influenced by what they were given, not by what they were told. Why did Miller fail to recognize this critical result in his commentary? How could he have misinterpreted the results obtained by Stockwell *et al*. (1982)? How could NIAAA officials be unaware of the results obtained by Stockwell *et al*.?

Additional Contradictions of the Expectancy Hypothesis

Miller *et al*. (1978) reported two experiments investigating the learning and retention of verbal material as a function of alcohol consumption

and expectancy in heavy social drinkers. Participants were randomly selected from a pool of male college students 18 years or older who had previously been assessed as heavy social drinkers based upon a drinking practices questionnaire distributed to students in introductory psychology classes. For experiment 1, students solicited as participants were offered incentives of either US$8.00 or course credit. They were asked not to consume alcohol for 24 hours and not to eat for 4 hours prior to the experiment. A balanced placebo design was employed. Half of the participants were given vodka and tonic in an amount that produced a blood alcohol level (BAL) of approximately 0.06, and half were given tonic. Half of each of these groups were told that they were given alcohol, and half were told that they were given tonic. After consumption, students were shown lists of high-frequency common words at a rate of every 3 seconds. Five lists of 16 words each were presented. Following the presentation of each list, students were asked to write down as many of the words as they could remember. After the completion of all five lists, students were asked to recall as many words as possible from all of the lists.

Results showed that alcohol consumption had a detrimental effect on immediate and delayed recall. Students were asked to return 2 days later, when they were asked to recall as many words as possible from all of the lists. They were then presented with the lists for relearning. No beverages were provided. The purpose of this delayed recall and relearning was to determine whether or not there was state-dependent learning. No such effect was obtained. Likewise, no instruction-induced expectancy effect occurred.

In experiment 2, low-imagery words were employed in an effort to increase the difficulty of the task. Modifications were also made to the design in an effort to further test the state-dependent learning hypothesis. Once more, instructions about drink content had no significant effect. Results obtained by Miller *et al*. (1978) disconfirming the expectancy hypothesis are characteristic of a large body of human learning and performance studies (Hull & Bond, 1986). Miller *et al*. (1978) offer a demand characteristics account of their failure to obtain an expectancy effect:

> Perhaps preconceptions of how one is expected to behave while intoxicated are developed primarily with regard to social behaviors. Subjects

> are not likely to have had much drinking experience in situations in which free recall of lists of words is the relevant behavior, nor is it likely that there is much folklore dealing with such situations. [p. 249]

No expectancy effect is obtained in learning and performance situations because participants cannot readily surmise the investigators' desired outcome.

Instruction-induced expectancy effects also fail to occur in situations where participants have no verbal control or awareness of ongoing physiological responses such as event-related potentials (ERPs) and skin conductance responses (SCRs) (Lyvers & Maltzman, 1991; Marinkovic *et al*. 2001).

British and Scandinavian Research Contradicting the Social Learning Theory of Alcohol Dependence

Stockwell *et al*. (1979) developed a questionnaire based on the alcohol dependency syndrome formulated by Edwards and Gross (1976), assessing symptoms such as frequency and severity of withdrawal experiences. An experimental study by Stockwell *et al*. led to the development of a behavioral measure of craving (Rankin *et al*., 1979). Outpatient alcoholics assessed as severely dependent volunteered for the experiment. They were required to live at home, drink all day at home, and permit the experimenter to visit them in their home. Each participant served in high- and low-craving conditions on successive days.

Upon arrival in the home, the experimenter gave the participant two glasses of vodka and tonic and two more again 3 hours later, at the end of the session. Participants were instructed to consume the drinks at their own rate. They were told that the purpose of the experiment was to determine how pleasurable they judged the drinks. Determining the speed of consuming the first of the two drinks at the end of the session was the real purpose. Self-ratings were obtained of anxiety, desire for a drink, and difficulty in resisting alcohol at the start and end of the session. Body temperature, pulse, BAL, and finger tremor were also recorded.

Two craving conditions were studied. In the high-craving condition, participants were told to drink normally before the experimenter arrived

at the home. They were given two drinks and instructed not to consume any more alcohol for 3 hours. In the low-craving condition, participants were also asked to drink normally prior to the arrival of the experimenter. Participants, however, were allowed to continue drinking for 2½ hours and were instructed to refrain from drinking for only half an hour prior to the final two drinks ending the session.

Results showed that under the high-craving condition, i.e. the 3-hour delay, there was a significant increase in participants' speed of consuming the drink at the end of the session as compared to the low-craving condition. Increases in finger tremor, self-rated craving, and inability to resist a drink were also observed in the high-craving, but not the low-craving, condition.

The validated behavioral measure of craving was then employed in a study of priming (Hodgson *et al*., 1979). Hospitalized alcoholics volunteered for the experiment. Following a period of at least 10 days of abstinence, participants were given either a high, a low, or no priming dose of alcohol in the morning and asked to consume the drink within 45 minutes. It was either a mix of tonic and alcohol or only tonic. A within-subject design was used where participants served in each of the three different conditions, with at least 1 day between each condition. In the afternoon of the priming day, participants were presented with five drinks; to provide a measure of the speed of consumption of the first drink, participants were asked to consume at least one of the drinks.

Results showed a significant interaction between the severity of dependency and the speed of consumption of the first drink in the afternoon, the behavioral measure of craving. Severely dependent alcoholics consumed the first drink more quickly, reported a stronger desire to drink, and consumed significantly more alcohol during the test than the moderately dependent alcoholics. The larger the priming dose, the faster the severely dependent alcoholics consumed the first drink. Moderately dependent alcoholics showed the opposite results: the larger the priming dose, the slower their consumption. A significant interaction was obtained between size of priming dose and severity of dependence. Severely dependent alcoholics showed an appetizer effect: the larger the priming dose in the morning, the faster they consumed the first drink in the afternoon. Moderately dependent alcoholics showed a satiation effect: the

larger the priming dose consumed in the morning, the slower they drank their first drink in the afternoon. If the two groups differing in severity of dependence are averaged, ignoring severity of dependence, alcohol appears to have no priming effect as compared to the tonic-only condition. These are the kind of results also obtained by Marlatt *et al*. (1973).

Hodgson *et al*.'s (1979) results showing an interaction between severity of dependence and speed of consuming the first drink as well as total amount consumed contradict the hypothesis of cognitive behavior therapists: there is only a quantitative difference between social drinkers and alcoholics in terms of the amount of alcohol consumed. Hodgson *et al*.'s results support the disease conception of alcoholism, which posits a qualitative difference between problem drinking and gamma alcoholism (i.e. alcohol dependence with withdrawal symptoms and high tolerance). Gamma alcoholism is a disease condition involving brain dysfunction in the prefrontal cortex, leading to the pathognomic sign and symptom of loss of control. Hodgson *et al*., however, did not employ a balanced placebo design. All participants obtained accurate information concerning the nature of their drinks; instructions were not a variable.

Studies employing a balanced placebo design conducted by a group of Scandinavian psychologists failed to replicate the results of Marlatt *et al*. (1973). Berg *et al*. (1981) studied male social drinkers and patients, severity of dependence unspecified, although all were "pharmacologically dependent", in an inpatient alcoholism treatment center. A within-subject double-balanced placebo experiment was conducted. The control group of social drinkers were apparently friends and associates of the investigators. Triads of subjects from each of the two groups who knew each other participated in a social drinking situation in the hospital lounge where they watched a televised soccer match. Each had their own decanter and glass, and was told that it was up to them how much they wished to drink. Significant interactions were obtained between groups × alcohol and groups × instructions. What they were told about the contents of the beverage affected the amount of alcohol consumed by the patients, but not by the social drinkers. The type of beverage tended to have an effect upon the control group, but not the patients. No adequate explanation for the results obtained in the two groups as well as for the difference in results obtained in this study and in

Marlatt *et al.*'s (1973) study was provided. We suggest that the similar results obtained with alcoholics in the two experiments reflect the similar influence of demand characteristics. Demand characteristics would be present in the Marlatt *et al.* experiment for the social drinkers in a laboratory setting who were not associates of the investigators as well as for the alcoholics. Demand characteristics of an experiment and the status differences between experimenter and subject were absent for the control group in the Berg *et al.* (1981) study because they were friends or associates of the investigators. Patients were not friends of the investigators. An experiment is needed in which demand characteristics are varied systematically for patients classified as alcohol-dependent, a second group classified as alcohol abusers according to DSM-IV criteria, and a third group of social drinkers.

Laberg (1986) conducted a study meeting some of the above conditions. Severely dependent, moderately dependent, and social drinkers were studied in a balanced placebo within-subject design. Laberg used as his dependent variables the time to the first sip and the time engaged in drinking as well as several psychophysiological measures and self-reports of craving. Laberg failed to find a priming effect on the critical measures of alcohol consumption either as a function of beverage given or instructions in any of the three groups. SCRs — a measure of the orienting reflex index of arousal — was greatest in the severely dependent group, as predictable from classical conditioning studies of semantic conditioning and generalization (Maltzman, 1979a, 1979b).

As previously indicated, a major problem with Miller's (1995a) revisionist discussion of Marlatt *et al.* (1973) and its interpretation in terms of expectancy is his ignoring the existence of an alternative testable theoretical interpretation of the results obtained by Marlatt *et al.*: the demand characteristics of the experimental situation (Knight *et al.*, 1986; Korytnyk & Perkins, 1983; Orne, 1962). The difference in status between experimenter and subject in the experimental situation induces a tendency in the participant to please the experimenter. The subject in an experiment tries to produce the results desired by the experimenter. This interpretation implies that the effects of instructions are limited to the social psychology of the laboratory situation. Effects induced by instructions do not generalize beyond the laboratory.

Research results obtained by the above British and Scandinavian psychologists as well as American investigators contradicting the expectancy interpretation of the balanced placebo design have been a well-kept secret. Miller (1995a) does not cite them in his laudatory comments celebrating Marlatt *et al*. (1973), other than to misrepresent the results of Stockwell *et al*. (1982). Marlatt and his students and former student collaborators never cite these studies (Cummings *et al*., 1980; Marlatt, 1983; Marlatt *et al*., 1993), nor do others who acclaim the Marlatt *et al*. (1973) study (Fingarette, 1988; Searles, 1993; Thombs, 1999). I have cited these contradictory results before to no avail (Maltzman, 1987, 1991, 1994, 2000).

How could the Marlatt *et al*. (1973) study be acclaimed as one of the most important studies in the first 25 years of the NIAAA's existence given the above evidence of its lack of verisimilitude? Dogma, politics, and power, including co-opting officials with little knowledge of the field in which they oversee research funding — that is how. Lack of scholarship, fear of alienating powerful figures in the field of specialization, people who may be sitting on the committees reviewing one's grant applications, and the binding of a common ideology contribute to this destruction of science in alcohol studies.

Disregard for the truth discussed in relation to Marlatt *et al*. (1973) is not an exception. It is one more expression of the decline in the integrity of professionals in the alcoholism studies field. The problem has been encountered before (Maltzman, 2000, Chapters 4 & 5), including investigations into charges of the Sobells' scientific misconduct. We now turn to another research area dominated by Marlatt and his associates stained by the lack of integrity.

Expectancy and Behavioral Skills Training as Secondary Prevention for At-Risk College Students

Marlatt and his students have published a number of studies on the secondary prevention of college students' heavy drinking. We touch upon this issue in Chapter 5 in relation to the difficulties in Marlatt's self-selection approach to alcohol problems and the superiority of primary as compared to secondary prevention as applied to college

students' excessive alcohol consumption. Chapter 5, however, does not address the present concerns: the cover-up of results contradicting the hypothesis that expectancy is a determinant of excessive college student drinking and alcohol consumption in general, and the inadequacy of behavioral skills training (BST) as a secondary prevention method.

Marlatt and associates' (Fromme *et al*., 1986) initial study employed three different conditions in which students reported their daily self-efficacy, drinking outcome expectancies, and alcohol consumption at baseline, during a subsequent 8-week training period, and at a 4-month follow-up. Group 1 received BST in addition to their assessments of expectancies and drinking; group 2 received didactic alcoholic information (AI) concerning the negative consequences of heavy drinking in addition to the assessments of expectancies and drinking; and group 3 received neither BST nor didactic information, instead recording only their daily drinking assessments and expectancies at baseline, during the 8-week training period, and at the 4-month follow-up. Participants were recruited from the University of Washington campus through newspaper advertisements and class announcements describing an 8-week program that would provide information on drinking behavior and how to change such behavior. Students evidencing moderate-to-severe alcohol dependence were screened out and encouraged to seek abstinence treatment for alcoholism or participate in AA. Students received US$50.00 if they participated in the training program and follow-ups over a 1-year period. Forty-five students agreed to participate.

Results showed that the group × time interaction of drinking during baseline, training, and follow-up was not significant. All groups showed a significant decline in alcohol consumption with time, but the BST group (group 1) did not differ significantly from the group receiving didactic information (group 2) or the group that only monitored alcohol consumption and expectancies (group 3). Self-efficacy and drinking outcome expectancies did not change, despite the significant decline in alcohol consumption in all conditions. This absence of expectancy changes was rationalized by Fromme *et al*. (1986) as due to "crystalization" of the expectancies. What the results really mean is that, once more, the expectancy hypothesis was contradicted by experimental

results. Unfortunately, this does not end the mythology that expectancy is a cause of excessive drinking.

Kivlahan *et al*. (1990) published results of a 4-, 8-, and 12-month follow-up of the students studied by Fromme *et al*. (1986). Kivlahan *et al*. do not cite the earlier Fromme *et al*. study of the same participants, much less the negative results obtained with the extensive expectancy measures and BST as a secondary prevention method. The group × time interaction reported by Kivlahan *et al*. was not statistically significant, and thus failed to show significant group differences in drinking. Once more, BST was not a significantly better method of secondary prevention than the traditional alcohol information (AI) treatment emphasizing the negative consequences of excessive drinking. Kivlahan *et al*. do not mention the expectancy results obtained by Fromme *et al*. (1986), and do not report using their expectancy measures to predict drinking at 8 and 12 months. Did Kivlahan *et al*. again fail to obtain significant relationships between expectancy and alcohol consumption? Or, did they not bother to relate their expectancy measures to alcohol consumption over 12 months because expectancies were "crystalized" at 4 months (Fromme *et al*., 1986)?

Marlatt *et al*. (1998, p. 604) continue the cover-up, citing Kivlahan *et al*. (1990) as the first secondary prevention study, but never citing Fromme *et al*. (1986). Larimer and Cronce (2002) engage in the cover-up of the inadequacies of Marlatt's secondary prevention methods, never mentioning the results obtained by Fromme *et al*. (1986) and misrepresenting BST as a superior secondary prevention method despite its lack of external validity and statistical significance as compared to AI. Chapter 5 provides further information illuminating the lack of external validity of Marlatt's secondary prevention methods and the need for primary prevention in dealing with excessive alcohol consumption on college campuses.

Goldman's Interpretation of Expectancy

We shall now turn to another ambitious program embracing expectancy and alcohol consumption (Goldman *et al*., 1999b). It is based upon survey questionnaire studies of expectancy and experiments purporting to

study the causal role of expectancies mediating alcohol consumption. The research program claims to have important implications for alcoholism treatment and prevention. It is productive and successful in the sense that it receives considerable financial support from government institutes and the drinks industry, and has enabled Goldman to obtain the influential position of associate director of the NIAAA. However, the program is based on word magic. It involves a fundamental misunderstanding of the nature of science.

For example, in a chapter exploring the limitless boundaries of expectancy notions titled, "Alcohol expectancy theory: the application of cognitive neuroscience", Goldman *et al*. (1999b) state,

> [M]uch of this [neurobiological] research falls short of full explanation, in part because of what we do not yet know, but also because, in many cases, it primarily has served only to identify and describe variables reliably associated with the target conditions. No matter how elaborate, description alone does not constitute explanation. Explanation requires specification of the theoretical processes by which variables influence one another. And even when the aforementioned research does emphasize neurobiological mechanisms and processes, these mechanisms are often variations of the normal substrate for learning, memory, motivation and emotion, rather than some independent and specialized pathway(s) of drug abuse. [p. 204]

Goldman *et al*. (1999) believe they are explaining why people drink and can become alcoholic because of the activation of hypothetical association pathways of "if-then" sequences of expectancies, but these are all reified hypothetical entities or processes lacking independent measures. They have the same difficulty as the stimulus–response (S-R) theories of half a century ago that can just as readily account for the same phenomenon (Maltzman, 1955). An S-R theory can predict the same effects as those claimed by Goldman and his students in priming experiments, but so what? Expectancies do not afford genuine explanations because they do not "point to the mechanism — causal, probabilistic or mixed — by virtue of which the facts to be explained occur" (Mahner & Bunge, 2001). "Expectancies" are reified hypothetical entities

that, when activated, cause people to drink — this is word magic, not science. A real explanation in biological and psychological science uncovers the biological mechanisms, the brain structures and functions, by virtue of whose activation the fact to be explained — e.g. consumption of alcohol — occurs.

Water is constituted as H_2O. It does not explain wetness, which is an "emergent", the result of an interaction between H_2O and receptors in the skin. Wetness is reducible to, or explainable by, neither molecular chemistry nor biology. We learn the usage and meaning of the word "wet" as an infant or child as the result of experience and verbal learning. Mom says, "It's raining; put on your coat and hat or you will get wet." In the future, when we say we expect to get wet if we do not put on a raincoat when it is raining, does that mean the expectancy causes "wetness"? Of course not. We are able to predict or estimate what may happen, and call it some form of expecting. It is the same with expecting positive effects from drinking alcohol, etc. We predict that if we consume alcohol, we will experience a positive effect. There is no unique causal process corresponding to the term "expect", no more than there are mermaids because there is a word for them. Expectancy in the hands of Goldman and others as a cause of alcohol consumption is simply a misuse of language or word magic: Balderdash.

Furthermore, Goldman, as do the other revisionists, ignores the large body of research showing that there is structural and functional brain damage which increases with the amount of alcohol consumed, although the increase is characterized by individual differences (Cala, 1987; Muuronen *et al*., 1989; Tarter, 1975; Volkow *et al*., 1992). Extensive infrahuman animal research carefully controlling diet demonstrates the neurotoxic effects of alcohol on brain cells, and the further damage produced by withdrawal that we have mentioned in Chapter 2 and discussed in some detail elsewhere (Maltzman, 2000). This lack of concern for the brain damage caused by alcohol is particularly surprising in the case of Goldman because he began his career in the field of alcoholism studying that very problem: the neuropsychological damage caused by alcohol consumption (Goldman, 1983).

Many qualitative differences in biological systems are involved in the development of alcoholism. Their specifications, which Goldman and

other cognitive behavior therapists ignore, predict far better the risk for excessive alcohol use and provide methods of treatment beyond the ken of expectancy theories. For example, the use of naltrexone, an opioid antagonist, reduces the risk of relapse (O'Malley *et al*., 1996). Participant alcoholics receiving naltrexone say that if they do drink, they do not get their high, their usual reinforcement. The reason is that alcohol consumption activates beta-endorphins, the body's endogenous opioid, an important source of reinforcement from alcohol that is blocked by naltrexone. There is no way cognitive expectancies can predict or explain such treatment results, remaining — as expectancies necessarily are — only skin deep.

Whatever happens between input and output — the proximate causes of excessive drinking, relapse, and remission that occur in the brain — is increasingly capable of being measured both directly and indirectly. Cognitive interpretations depending upon word magic, ignoring the old insight that correlation is not causation, can no longer suffice. The cognitive "revolution" is over because increasingly sophisticated techniques permit neuroimaging of brain structure and function. Repeated within-subject neuroimaging is now possible with magnetic resonance imaging (MRI) and functional MRI (fMRI), which do not expose participants to radioactivity. Other technologies such as biological measurements from infrahuman animals during performance, as well as molecular biology and behavioral genetics, are leaving social learning and expectancy theories in the dustbin of history.

Priming Alcohol Consumption by Positive Expectancies

Goldman and his students (Goldman, 1999a, 1999b; Goldman *et al*., 1999b) have published a number of experiments and questionnaire studies on alcohol use purporting to show the profound importance of expectancy. Expectancy theory is assumed to provide an explanation for many alcohol-related behaviors, and forms a basis for alcoholism treatment and prevention.

A scholarly review and critique of the theorizing and research of expectancy theorists has been published by Jones *et al*. (2001). They

describe the numerous inconsistent and contradictory results involving-expectancy-related research, particularly in relation to prevention and treatment. Results reported at a symposium (Wiers *et al*., 2003) reaffirm the inconsistency in expectancy research as it may pertain to alcoholism treatment and prevention.

However, according to Goldman *et al*. (1999b),

> The most persuasive evidence for [causal] mediation comes from true experiments with random assignment of participants, manipulation of the hypothesized mediator, and inclusion of appropriate control groups to rule out the influence of alternative variables on the dependent variable. Several studies support the inference that expectancies influence drinking, but in these studies the operational definition of expectancy must necessarily expand beyond psychometrically developed questionnaire responses. Manipulation of expectancies instead must be inferred from changes in observable variables, presumably, linked to expectancies. . . .
>
> Six experiments conducted in our laboratory have directly manipulated expectancies and shown effects on self-reported and observed drinking. . . . [pp. 222–223]

Experimental studies by Goldman and his colleagues purport to show that expectancy has a causal role in the determination of alcohol-related behavior. If the results of these studies are confirmed, expectancy theory is validated and cannot be ignored in the field of alcohol studies (i.e. the causes, treatment, and prevention of alcoholism).

Roehrich and Goldman's (1995) study is, perhaps, the most important experimental one by the Goldman group. It appears to demonstrate that the experimental manipulation of expectancies causes an increase in alcohol consumption in college students. Subjects in the study participated in two ostensibly independent experiments. The first was presented as a consumer survey of beers. Participants watched segments from two popular TV comedy programs: one was "Cheers", occurring in a Boston bar, providing cues for alcohol consumption; the other was "Newhart", occurring in the dining room of a Vermont inn, where no alcohol cues were present. Following the observation of the

TV program segments and while waiting for a recall test of events in the program, students were asked to participate in a different experiment and earn additional course credit. It involved a Stroop test, presenting combined pairings of colored cards with either a neutral or an alcohol-associated word printed on its face; participants were subsequently asked to recognize the words that had appeared — a form of incidental learning test. Following the memory test, students received a taste-rating survey asking them to assess three different nonalcoholic beers. They were informed that the beverages were alcoholic beers and were asked to provide their preferences, sampling each beer as much and as often as they felt necessary. A 2×2 factorial design was employed, where half of the students received the "Cheers" and the other half viewed the "Newhart" video segment. Half of the participants in each group received alcohol-associated adjectives in the Stroop test and half received neutral unrelated nouns. All participants in the study were women.

Roehrich and Goldman (1995) found that both kinds of priming effects, watching an alcohol-related TV program and viewing alcohol-associated words, significantly increased alcohol consumption as compared to the control, nonalcohol-related video segments and neutral words. Effects of the alcohol primes were additive.

Sumarta (2000), in a dissertation conducted in my laboratory, replicated the Roehrich and Goldman (1995) study with several important additions. An independent condition was added in which beverage taste ratings were of sparkling waters as well as beers, thereby testing the specificity of the priming effect. Roehrich and Goldman (1995) did not determine whether the experimental prime was specific to alcoholic beverages or not; they simply assumed that it was.

Using the same segments of "Cheers" and "Newhart" and the same Stroop test employed by Roehrich and Goldman (1995), Sumarta (2000) failed to find a significant priming effect in either test situation. She also found that participants, both men and women, drank as much sparkling water as beer. The established drinking style, i.e. the amount consumed during the previous month, predicted the amount of beer and sparkling water consumed in the experiment. Overall, Sumarta's results contradict the hypotheses that (1) consumption of alcohol can

TABLE 4.1. Sumarta's (2000, p. 205) failure to find a priming effect.

Mean Amount of Nonalcoholic Beer Consumed by Men and Women as a Function of Priming Group

	Men		Women	
Priming Group (n = 12)	M	(SD)	M	(SD)
Cheers/AE	2.17	(0.30)	1.86	(0.38)
Cheers/Control	2.11	(0.19)	2.04	(0.40)
Newhart/AE	2.26	(0.17)	1.97	(0.49)
Newhart/Control	2.19	(0.24)	2.22	(0.32)
Overall	2.18	(0.22)	2.02	(0.40)

Note: Logarithmic transformed amount of beverage consumed. M = mean; SD = standard deviation; AE = alcohol-expectancy words; Control = control words.

be primed by the "Cheers" TV program as compared to the "Newhart" program, and (2) alcohol consumption can be primed by words which are associated with alcohol consumption — so-called expectancy words — as compared to neutral words. Sumarta's results are shown in Table 4.1.

Additional evidence is available falsifying Roehrich and Goldman's (1995) hypothesis that a hypothetical expectancy process is a causal factor determining alcohol consumption. Two independent studies conducted by Aarons (1996) testing the priming effect of alcohol-related words failed to replicate Roehrich and Goldman's (1995) results.

In the first study, Aarons (1996) used a 2×2 factorial design varying in the type of prime either by the type of word or by the type of beverage. He employed Roehrich and Goldman's (1995) procedure of conducting two seemingly independent experiments: (1) an apparent language and memory experiment, and (2) a market survey of beverages requiring taste ratings. In the first experiment, priming consisted of alcohol-expectancy words or neutral words presented as stimuli on a computer monitor. The participants' task was to find a synonym for each word presented. They then received a Stroop color-naming test in which either the previously seen alcohol-related words or neutral words were presented along with a set of control words appearing on various colored

backgrounds in randomized orders. Participants were instructed to name the colors as quickly as possible.

The second, seemingly unrelated, task was presented as a market survey where participants drank either alcoholic or nonalcoholic beverages, fruit juice and vodka or fruit juice. No significant priming effect on the consumption of alcoholic beverages was obtained. A group of subjects, half receiving a prime and half a neutral word, provided taste ratings with fruit juice to test the specificity of the alcohol prime.

A second study was conducted attempting to correct for possible flaws in the design of the above experiment. College students' favorite alcoholic beverage by a wide margin is beer, not vodka and fruit juice; perhaps the use of an inappropriate taste-testing beverage was responsible for the failure to obtain a priming effect. Alcoholic beer was therefore employed in the second experiment for half of the students and nonalcoholic beer for the remaining subjects. In the Stroop test, no significant difference occurred in response latency for color naming as a function of the word, alcohol-related or neutral. Negative results were again obtained in the critical consumer market survey, this time with alcoholic or nonalcoholic beer taste ratings. The amount of beer consumed, either alcoholic or nonalcoholic, did not vary significantly as a function of the prime, alcoholic or nonalcoholic.

Goldman and his colleagues (Goldman, 1999a, 1999b; Goldman *et al*., 1999a, 1999b) fail to cite or describe Aarons' (1996) studies contradicting Goldman's expectancy hypothesis. It is noteworthy that Aarons' experiments were conducted in Goldman's laboratory as a doctoral dissertation under his supervision.

Three carefully conducted experiments, two by Aarons (1996) and one by Sumarta (2000), have falsified the hypothesis that expectancy is a determinant of alcohol consumption. It is time to reject this blind alley and stop wasting public funds supporting research on a poorly considered hypothesis that has been shown to lack verisimilitude. It has been repeatedly falsified in the form of the purported seminal balanced placebo experiment (Marlatt *et al*., 1973) and expectancy priming (Goldman *et al*., 1999a, 1999b). It is time to recognize what the expectancy hypothesis is: Balderdash.

Expectancy and Secondary Prevention: A Double Whammy

An early study conducted by Marlatt and associates (Fromme *et al*., 1986) is especially pertinent because it obtained multiple assessments of expectancies and alcohol consumption over an extended time period. Three different conditions were studied in which all students reported their daily expectancies and alcohol consumption at baseline, during an 8-week training period, and at a 4-month follow-up. Group 1 received BST as secondary prevention in addition to the assessments of expectancies and drinking; group 2 received didactic information concerning the negative consequences of heavy drinking along with the assessments; and group 3 received neither BST nor didactic information, instead recording only their daily drinking and expectancies at baseline, during the 8-week training period, and at the 4-month follow-up.

Results showed that alcohol expectancies did not change with decreases in alcohol consumption, contradicting the hypothesis that expectancies are a causal factor determining alcohol consumption. Furthermore, Marlatt's method of secondary prevention, BST, was not significantly superior to a didactic program of information emphasizing the negative consequences of drinking. Group 3, only monitoring its expectancies and alcohol consumption, showed a significant decrease in alcohol consumption no different than the other two groups. The failure of expectancies to change with changes in alcohol consumption was rationalized by Fromme *et al*. (1986) as due to "crystallization" of the expectancies!

Kivlahan *et al*. (1990) published the results of a 12-month follow-up of the students studied by Fromme *et al*. (1986), contradicting the expectancy theory and showing that BST was not a superior form of secondary prevention for college students at risk as compared to didactic information. Kivlahan *et al*. do not cite the earlier Fromme *et al*. (1986) study of the same participants, much less the negative results obtained with their extensive expectancy measures. The groups × time interaction reported by Kivlahan *et al*. failed to attain statistical significance for measures of drinking, indicating that BST was not a significantly better method of secondary prevention than the traditional treatment which emphasized the negative consequences of extensive drinking. Kivlahan

et al. do not describe the expectancy results obtained by Fromme *et al*. (1986), and do not use the expectancy measures to predict drinking at 12 months. Once more, negative results contradicting the expectancy theory are hidden. Did they again fail to obtain significant relationships between expectancy and alcohol consumption? Or, did they not bother to relate expectancy measures to alcohol consumption because expectancies were purportedly "crystallized" (Fromme *et al*., 1986)? Once more, Marlatt and his associates covered up the results contradicting the expectancy theory.

One wonders what the effect of a didactic treatment that shows neuroimages of the brains of college students who drink heavily and binge frequently as compared to those who do not would be. Such a study is not likely to be conducted by skin-deep cognitivists such as Marlatt and his students. We demonstrate in Chapter 5 that BST as secondary prevention for heavy-drinking college students has inherent deficiencies as compared to primary prevention. Research shows that the expectancy hypothesis lacks verisimilitude and that primary prevention promoting abstinence must be adopted to avoid the brain damage produced by excessive drinking. Revisionists argue that clinical practice should be based upon scientific research. It is time they practice what they preach.

Disclaimer

My above comments concerning the lack of verisimilitude of the expectancy hypothesis are directed specifically at the use of the concept of expectancy as a causal factor in alcohol studies, especially the balanced placebo experiment, longitudinal questionnaire studies, and priming experiments. These negative evaluations do not include all expectancy research, especially earlier research by Robert Rosenthal (1976). His important research and analysis of such phenomena as experimenter bias and the Pygmalion effect are lasting contributions.

Conclusion: A Glimpse of the Past and the Future

Franz Brentano (1874/1973), one of the founders of modern psychology and the founder of modern cognitive psychology, asserted that intentional

terms such as "expectancy" are purely descriptive. Explanations await advances in physiological psychology; in contrast, contemporary cognitive behavior therapists think they are explaining a phenomenon by labeling it an expectancy. They assert that they are inferring an expectancy which causes the behavior in question. For example, an expectancy inferred from a subject's behavior purportedly induced by the instructions they received causes their alcohol consumption (Marlatt *et al*., 1973), i.e. causes the very same behavior from which the intention or expectancy was "inferred" in the first place. Brentano never used such word magic: "X drinks excessively because he or she has a positive expectancy." How do you know they have a positive expectancy? Because they drink excessively. What was true in Brentano's time remains true today. Proximal causes of behavior are uncovered by tracing the physiological changes occurring in the brain that interacts dialectically with the environment; this will lead to explanations of behavior now said to be caused by cognitions, expectancies, representations, mental processes, etc. The problem with much of current cognitive psychology, including cognitive behavior therapy, is that its theoretical terms are derived from the very same behaviors the terms are used to explain. To make matters worse, the representations such as expectancies are reified, hypostatized, transformed into a real thing or process that causes the very behavior taken as their marker. What they really are is bad science.

Conceptions such as nodes, templates, and others (Goldman *et al*., 1999b) do not provide an account of the biological mechanisms by virtue of which the facts to be explained occur. Questionnaire studies of self-reported expectancies in relation to alcohol consumption are as readily interpreted as a consequence of prior learning as they are a cause of future behavior. It must be noted that the original questions used in expectancy questionnaires were questions first used in Mulford and Miller's (1960) study, which asked participants to provide a definition of drinking. Adding the word "expect" or "expectancy" to a questionnaire does not provide a causal explanation of subsequent answers on the questionnaire or of actual drinking behavior. Correlation is not causation.

It is now increasingly possible to measure what happens in the brain and body, between input and output, the proximate causes of drinking,

relapse, etc. Functional interpretations which depend upon word magic, ignoring the old insight that correlation is not causation, can no longer suffice. The "cognitive revolution" is over because increasingly sophisticated techniques permit neuroimaging of brain function and quantitative electroencephalographic brain mapping. Repeated within-subject neuroimaging is now possible with MRI and fMRI, which do not expose participants to radioactivity or other invasive procedures. Technologies such as biological measurements from infrahuman animals during performance, as well as molecular biology and behavioral genetics, are leaving social learning and expectancy theories in the dustbin of history.

Ernst Mayr (1994), a distinguished systematic biologist, asserts that a theory may be invalid for (1) the generally accepted principle of contradictory evidence or (2) the use of equivocal, ambiguous terms. Expectancy theory in the field of alcohol studies is invalid on both counts. It is Balderdash.

5

Self-selection of Alcoholism Treatment Goals: Harm Reduction or Induction

The purpose of the present chapter is to examine the ethical and research implications of the radical shift in approach to the caregiver/investigator–patient/participant relationship proposed by cognitive behavior therapists engaged in alcoholism treatment and treatment research. Revisionists argue that patients/participants ought to be autonomous and self-select their treatment goal. This represents a fundamental change from paternalism, the dominant practice in alcoholism treatment in the United States, where the caregiver rather than the prospective patient determines the treatment goal.

After presenting the argument for self-selection, we will review ethical principles and their applications guiding the research and treatment of alcoholism and other disorders. Federal regulations governing human subject protection are based on these ethical principles and tort law. The ethical principles are described in the Belmont Report, a synopsis of the extended deliberations of the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research (National Commission, 1978) appointed by then-President Jimmy Carter. The Belmont Report also provides the norms for institutional review boards (IRBs), which institutions receiving federal funds for research or teaching are required by law to establish. We will then determine whether or

not the principle of treatment self-selection, as described by cognitive behavior therapists and as apparently used in their alcoholism treatment research and practice, conforms to federal regulations and the standards of ethical research and treatment (Basic DHHS Policy for Protection of Human Research Subjects, Title 45, Part 46, 1991).

Self-selection of Alcoholism Treatments

> In terms of addictive behaviors such as alcohol *dependence* [emphasis added] it may be more productive to view individuals who seek or are referred for treatment as 'consumers' who are capable of selecting among viable treatment options, rather than as 'patients' who are assigned to a particular treatment by a professional utilizing treatment-matching criteria. . . . [I]n the consumer choice paradigm, prospective consumers of treatment services are allowed to 'shop around' and select a treatment or self-help group of their own choosing. [Marlatt, 1999, pp. 45 & 60]

> In the field of alcohol treatment there has been reluctance in acceptance of the client as a responsible participant in the treatment process. Therapists have often been concerned about what kind of clients should be allowed to choose moderation, and under what circumstances control training would be appropriate. With few exceptions, the drinkers' own wishes and attitudes are not considered or taken very seriously. Also, the definitions of controlled drinking have been made in accordance with what therapists have considered 'decent' or acceptable drinking according to the therapists' subjective norms. [Duckert, 1995, p. 1168]

> In my opinion the real paradigm shift is not so much the choice of different treatment methods as in the choice of model for understanding human nature. Is the problem drinker to be met as an ally or as an untrustful adversary? Shall we trust the person's ability to evaluate his/her own situation and needs and his/her capacity to make adequate choices about future relationship to alcohol — or shall we as therapists be the ones who decide what is the best alternative for him/her. [Duckert, 1995, p. 1169]

> Harm reduction focuses on helping the individual move toward problem resolution, even if that resolution does not occur entirely or all at once. Moderation or abstinence, is a personal decision, and abstinence is not a rigid requirement. The approach incorporates the ideas that "something is better than nothing," and that a small step is often easier than, and sets the stage for, a large one. Harm reduction is a practical approach that acknowledges the differences between individuals and respects their capacity to shape their own lives. [Marlatt, 1998, Foreword]

> My colleagues and I have been reviewing, summarizing, and writing about addiction treatment research for two decades now. What we have observed is both encouraging and disturbing. The research clearly shows that there is no single approach which is superior to all others. Rather, there are a number of alternatives that are consistently supported by scientific research. These treatments have been developed and tested primarily in the last two decades and are the best that science has to offer for those seeking to overcome alcohol problems. That's the good news. The not so good news is that alcohol treatment programs in the U.S. have only slowly begun to offer these treatments. Also, the average person, not inclined to spend hours in medical libraries, would have difficulty learning about these approaches or even learning that they exist. In a country that prides itself on technological and scientific sophistication, the best that science has to offer for overcoming addiction is not widely known.
>
> If you are considering treatment for yourself or another, you need to be aware of these alternatives. Fortunately, some psychologists and other addiction professionals offer scientifically based alternatives. This workbook is a good place to learn about them. [Hester, 1998, Foreword]

Ethical Principles Guiding Treatment Research and Practice

The Belmont Report (National Commission, 1978), which serves as the basis for current ethical guidelines and federal regulations governing research and practice, is a part of the history of research codes and guidelines. It follows the Nuremberg Code (1949) prompted by the Nuremberg Trials; the Helsinki Declaration (World Medical Association

Declaration of Helsinki, 1964); and the concerns in the United States following the exposure of the mistreatment of patients in the Tuskegee study, the Jewish Hospital study, the Willowbrook study, and others (Levine, 1988). Although the principles discussed in the Belmont Report are designed primarily to guide research, the distinction between human research and treatment is difficult to maintain (Levine, 1988). Both research and treatment are guided by essentially the same three basic ethical principles delineated in the Belmont Report: (1) respect for persons, (2) beneficence, and (3) justice.

Respect for Persons

This is also known as the principle of autonomy.

> It incorporates at least two basic ethical convictions: first, that individuals should be treated as autonomous agents, and second, that persons with diminished autonomy are entitled to protection. The principle of respect for persons thus divides into two separate moral requirements: the requirement to acknowledge autonomy and the requirement to protect those with diminished autonomy. . . . Respecting a person's autonomy means giving weight to their reasoned choices provided that they are not clearly harmful to others. To show a lack of respect for an autonomous agent is to repudiate that person's considered judgments, to deny an individual the freedom to act on those considered judgments, or to withhold information necessary to make a considered judgment, when there are no compelling reasons to do so. [National Commission, 1978, p. 4f]

The Belmont Report (National Commission, 1978) notes that not everyone is capable of autonomy:

> Children and some individuals lose this capacity wholly or in part because of illness, mental disability, or circumstances that severely restrict liberty. Respect for the immature and the incapacitated may require protecting them as they mature or while they are incapacitated. . . . The extent of protection afforded should depend upon the risk of harm

> and the likelihood of benefit. The judgment that any individual lacks autonomy should be periodically reevaluated and will vary in different situations. [p. 5f]

Beneficence

An obligation of the caregiver and research investigator is mandated by the ethical principle of beneficence.

> Two general rules have been formulated as complementary expressions of beneficent actions in this sense (1) do not harm, and (2) maximize possible benefits and minimize possible harms.
>
> The Hippocratic maxim 'do no harm' has long been a fundamental principle of medical ethics. Claude Bernard extended it to the realm of research, saying that one should not injure one person regardless of the benefits that might come to others. However, even avoiding harm requires learning what is harmful. [National Commission, 1978, p. 6f]

Justice

This principle is complex, but generally means "fairness of distribution" of the benefits and harms of research. Certain groups such as prisoners, the poor, ethnic minorities, and the uneducated should not be disproportionately involved in research that may harm the individual. Research that may benefit participants should not exclude people because they are poor, uneducated, ethnic minorities, etc.

Applications of the above ethical principles must meet the standards of three essential requirements for the protection of human subjects: "informed consent, risk/benefit assessment, and the selection of subjects of research" (National Commission, 1978, p. 10).

Informed Consent

There are three essential components to a valid consent: (1) information provided by the investigator or caregiver, (2) comprehension of the material

by the patient/participant, and (3) voluntary participation on the part of the patient/participant. Each component in turn is complex.

Information

Prior to participating in treatment/research, the patient/participant must be informed of the purpose of the treatment/research, the procedure involved, possible harms and benefits stemming from the treatment/procedure, and alternative procedures or treatments and their possible harms and benefits. Magnitude and duration estimates of the possible harms and benefits also need to be described. The patient/participant is informed that they may ask questions or withdraw from the experiment or treatment at any time.

The criterion for what information ought to be provided by the caregiver and the investigator is that it is material to a reasonable person's decision. For example, information that continued consumption of alcohol would exacerbate existing brain dysfunction or prevent its reversal whereas abstinence may result in reversal of the dysfunction is the kind of information that reasonable people would want to consider in reaching a decision whether to select abstinence or controlled drinking as their treatment goal. Furthermore, "information about risks should never be withheld for the purpose of eliciting the cooperation of subjects, and truthful answers should always be given to direct questions about the research" (National Commission, 1978, p. 12).

Comprehension

> Investigators are responsible for ascertaining that the subject has comprehended the information. While there is always an obligation to ascertain that the information about risk to subjects is complete and adequately comprehended, when the risks are more serious, that obligation increases. [National Commission, 1978, p. 13]

More specifically, comprehension is a complex process involving understanding the information provided, appreciating the relevance of the information to one's self, and retaining the information and its

integration in a manner permitting a reasoned decision. It also requires the reliable communication of the decision to the caregiver/investigator (Grisso & Appelbaum, 1998).

Voluntariness

Participation must be free of coercion, undue influence, pressure, or excessive or inappropriate incentives.

Assessments of Risks and Benefits

> 'S]o-called risk/benefit' assessments are concerned with the probabilities and magnitudes of possible harms and anticipated benefits. Many kinds of possible harms and benefits need be taken into account . . . [such as] risks of psychological harm, physical harm, legal harm, social harm and economic harm and corresponding benefits.
>
> Risks and benefits of research may affect the individual subjects, the families of the individual subjects, and society at large. The idea of systematic, nonarbitrary analysis of risks and benefits should be emulated insofar as possible. *This ideal requires those making decisions about the justifiability of research to be thorough in the accumulation and assessment of information about all aspects of the research and to consider alternatives systematically* [emphasis added]. This procedure renders the assessment of research more rigorous and precise, while making communication between review board members and investigators less subject to misinterpretation, misinformation and conflicting judgments. . . . It should be determined whether an investigator's estimates of the probability of harm or benefits are reasonable, as judged by known facts or other available studies. [National Commission, 1978, pp. 16–17]

Justice

"The principle of justice gives rise to moral requirements that there be fair procedures and outcomes in the selection of research subject" (National Commission, 1978, p. 18). Potentially beneficial research should not be limited to certain kinds of subjects such as the upper

socioeconomic classes, or be influenced by ethnic or gender bias. The burden of risky or potentially harmful research should not be restricted to classes of subjects who are vulnerable, such as prisoners, the poor, or ethnic minorities.

Evidence Purporting to Support Self-selection

Revisionists claim that self-selection of treatment builds self-efficacy, thereby increasing the probability of a successful treatment outcome. A number of different studies are cited that apparently lend empirical and moral support to the self-selection position. However, such support is more apparent than real.

Rotgers (1996) argues that if treatment goal options are available, patients who need abstinence will choose it; furthermore, a treatment goal prescribed for a patient produces poorer results than one freely chosen. A study by Booth *et al*. (1984) purports to support these generalizations. It does not. Booth *et al*. formed three treatment goal groups: (1) a prescribed abstinence (PA) group, where all 10 patients had abnormal liver function; (2) a group that chose abstinence (CA), where 7 out of 15 patients had abnormal liver function; and (3) a group that chose controlled drinking (CD), where 4 out of 12 patients had abnormal liver function. Results in terms of positive outcomes (abstinence plus controlled drinking) were PA = 20%, CA = 27%, and CD = 42%. These results were not statistically analyzed, and are unreliable due to the small numbers of participants involved such that a shift in the opposite direction of only two subjects would erase the apparent percentage differences. The PA group suffered the most from the biomedical consequences of alcoholism. Is it surprising that they appeared to do worse than the two groups self-selecting their treatment goals? This poorly designed and conducted efficacy study, with an unreliable outcome confounding the severity of alcoholism with experimental conditions, is no basis for treatment program innovations.

The other study Rotgers (1996) cites in support of patient autonomy, especially the free choice of moderate drinking (Sanchez-Craig & Lei, 1986), used participants from an earlier efficacy study (Sanchez-Craig, 1980) which explicitly excluded participants who believed in the disease

concept or who participated in Alcoholics Anonymous (AA). The results from such a biased sample are not generalizable to the population of people seeking help for problems of alcohol misuse or to the ethical conflict between autonomy and beneficence.

Rotgers (1996) fails to cite the most extensive experiment available comparing choice vs. prescribed treatments (Walsh *et al*., 1991). A total of 227 participants in an employee assistance program (EAP) as a result of drinking problems were randomly assigned to a traditional inpatient treatment program, mandatory AA meetings, or a choice group. The latter group was free to choose either of the other two treatments, a different treatment of their own choice, or no treatment as long as they were not drunk on the job. At the 2-year follow-up, the prescribed inpatient treatment group had a significantly higher continuous abstinence rate and a significantly lower percentage of heavy drinking days than the choice group, which did not differ significantly from the prescribed AA group. Miller and Hester (2003), Rotgers (1996), and other revisionists still claim that there is no evidence for the effectiveness of traditional treatments and promote the vague notion of self-efficacy, which is purportedly enhanced by self-determination (i.e. choice) of treatment goal.

Evaluation of Treatment Self-selection

We will now consider how alcoholism treatment self-selection, as advocated and apparently practiced by revisionists, upholds or violates the ethical principles previously described and the federal regulations based on them. Treatment self-selection as advocated by revisionists falls under the ethical principle of respect for persons: individuals should be treated as autonomous agents. Allowing patients to select their own treatment gives them the power to control what is done with their bodies. However, application of the principle of autonomy entails certain requirements.

First, there is another part of the principle of respect for persons: "persons with diminished autonomy are entitled to protection" (National Commission, 1978). This part of the principle of respect for persons is ignored by advocates of self-selection. They do not attempt to determine whether or not the patient/participant has diminished capacity (i.e. diminished autonomy). Diminished capacity must be distinguished from

competence, a legal concept. The latter is decided by a court and does not necessarily mean that an individual cannot provide a proper informed consent under given circumstances. A developmentally disabled adult may be declared incompetent to manage their financial affairs and live independently; however, they may have the capacity to decide on a particular treatment for a serious disorder, for example a cancer treatment. Diminished capacity, on the other hand, is an empirical problem that varies with the situation. An alcoholic may be legally competent, but have diminished capacity to provide a fully informed consent; when under the influence, information presented may not be comprehended because the person may not remember the information or be capable of reasoning, integrating the information, or reliably communicating their decision to the caregiver/investigator. A major shortcoming of the revisionist position is that it fails to consider that, even when sober, an alcoholic, a problem drinker, or even a heavy social drinker may have diminished capacity as a result of brain damage caused by alcohol and its metabolites, head injury, comorbid depression, panic, or schizophrenia (Bechara *et al*., 2001; Cala, 1987; Parsons, 1998; Ryback, 1971). They may not have the capacity to comprehend the informed consent even when sober.

Second, the informed consent must contain information material to the decision to select one treatment goal or another. Heavy drinkers as well as alcoholics are at risk for alcohol-induced neuropsychological dysfunction (Cala, 1987; Parsons, 1998; Ryback, 1971). The caregiver/investigator is obligated to assess brain structure and function, and provide the results of these assessments to the participant/patient. Material information that must be provided is that alcohol consumption may exacerbate and/or prevent reversal of an existing dysfunction.

Reading the descriptions of cognitive behavior therapy (CBT) procedures designed to produce harm reduction in problem drinkers is revealing (Hester, 2003; Hester & Miller, 1995). It appears that a valid informed consent was not obtained from participants in the treatment efficacy studies purporting to show the success of behavior therapy treatments for controlled drinking or harm reduction. Miller, Hester, and other cognitive behavior therapists do not, as a matter of course, first obtain measures of brain structure and function, assess these in

relation to age-corrected norms for social drinkers or abstainers, and provide participants with this material information. They also fail to determine if participants have the capacity to comprehend the informed consent.

Cognitive behavior therapists do not inform their patients that continued "moderate" controlled drinking may lead to further brain dysfunction or prevent reversal of the dysfunction (Cala, 1987; Eckardt *et al*., 1980, 1995, 1998; Gual *et al*., 1999; Muuronen *et al*., 1989; Parsons, 1998; Wilkinson & Sanchez-Craig, 1981). Patients' significant others, including family members, are not provided material information concerning the patient's neuropsychological function. Potential harm produced by patients' continued drinking at reduced levels, the particular level to be decided by the patient (Duckert, 1995), is not estimated.

Harm Reduction versus Harm Induction: Brain Damage, Quality of Life, and Alcohol Consumption

Cala (1987) reports that 95% of the alcoholic participants she studied showed evidence of brain atrophy in their computerized tomography (CT) scans; they had been consuming approximately 10 drinks/day or more. She also found that 65%–85% of light and moderate drinkers showed brain atrophy; they had been consuming approximately one to five drinks/day. Physiological and anatomical changes were apparent before the stage of alcoholism was reached. Cala's results showed that brain dysfunction may develop in the majority of light to moderate drinkers over time; however, individual differences were evident. Some people who were heavy drinkers showed less brain atrophy than light drinkers. Some heavy drinkers showed no atrophy. Some light drinkers showed greater atrophy than many heavy drinkers. Sixty-seven percent of light and moderate drinkers who abstained for 6 months showed a significant reversal of brain shrinkage and an improvement in neuropsychological test scores, but reversal of atrophy was not complete in all people.

Variables responsible for individual differences in atrophy, and how and what variables facilitate or retard the reversal of atrophy with abstinence, are not known. It is known that continued drinking leads to the persistence of dysfunction and its exacerbation, whereas abstinence may

result in the reversal of deficits (Beatty *et al*., 2000; Cala, 1987; Eckardt *et al*., 1980, 1998; Ganzler *et al*., 2000; Mann *et al*., 1999; Muuronen *et al*., 1989; Shear *et al*., 1994; Wilkinson & Sanchez-Craig, 1981). Brain dysfunction is not merely an end result sometimes found in alcoholics. Brain dysfunction in a majority of individuals can be produced by as little as five drinks a day or less (Cala, 1987), a level below that which the Rand Report (Armor *et al*., 1976) and the Sobells (Sobell & Sobell, 1978) considered a return to controlled or moderate drinking.

Wilkinson and Sanchez-Craig (1981) found that an average of less than one drink/day or abstinence following treatment is necessary to permit the reversal of a neuropsychological deficit. An average of four drinks/day prevented reversal of the deficit. However, Sanchez-Craig *et al*. (1995) recommend not more than four drinks/day for men and not more than three drinks/day for women. Symptom progression with higher levels of alcohol consumption was the basis for the recommended limits of controlled drinking following treatment; while ethical and iatrogenic implications of their own findings of continued neuropsychological deficits with four drinks/day were ignored (Wilkinson & Sanchez-Craig, 1981), other than considering such deficits as a possible handicap in acquiring coping skills. The criterion for controlled drinking in terms of the number of drinks is a questionable criterion for safe drinking: they fail to consider within- and between-subject variability in the effects of consuming alcohol, individual differences in lean body mass and drinking style, alcohol consumption before or after meals, ethnic differences in alcohol effects, and biological vulnerabilities due to family history (Eckardt *et al*., 1998). Parsons' (1998) conclusion following an extensive review of neuropsychological research from his laboratory and others' over a period of more than two decades is that there is strong support for the theory proposed by Ryback (1971): there is continuity of brain damage from social drinking to chronic alcoholism.

It is apparent that brain damage and neuropsychological deficits are common consequences of heavy drinking and that, for many individuals, reversal of these deficits may occur in the absence of further drinking. Failure to provide such information invalidates informed consent. It is the failure to provide material information that may influence a reasonable person's decision to select one form of alcoholism treatment or

another. If further brain damage or lack of reversal of prior damage is obtained as a consequence of controlled drinking in the absence of a valid informed consent, the patient/participant has grounds for a malpractice lawsuit (Levine, 1988).

Gual *et al*. (1999) conducted a prospective multisite study of treatment outcomes for 850 patients meeting the DSM-III criteria for alcohol dependence receiving 2 years of individualized outpatient treatment. Five years after treatment, drinking status and measures of quality of life (QoL) — such as morbidity, mortality, alcohol-related problems, and psychological and social functioning — were assessed. A 3-year evaluation window, the status during years 3–5, showed that 44% were abstinent, 38% were heavy drinkers, and 7% were controlled drinkers. Abstinent patients manifested significantly better outcomes than controlled and heavy drinkers on most QoL measures (medical, socioeconomic, legal, and psychological). Approximately 42% of patients suffered some clinical pathology, with controlled and heavy drinkers displaying significantly higher morbidity than abstainers. Heavy and controlled drinkers showed a significantly higher accident rate than abstainers. Emergency room use was also significantly greater in heavy drinkers (31%) and controlled drinkers (24%) than in abstainers (14%).

A criticism of the study might be that the abstinence classification Gual *et al*. (1999) employed included individuals who did occasionally drink. Classifying these individuals as controlled rather than abstinent drinkers would increase the percentage of controlled drinkers. More importantly, such reclassification would also increase the advantage of abstinence over any form of drinking in terms of most QoL measures employed by Gual *et al*. It is clear that more treatment evaluation studies should employ a variety of objective QoL measures, particularly studies advocating harm reduction or controlled drinking. It is ironic that advocates of harm reduction have generally failed to use such measures, limiting themselves to alcohol consumption measures and ignoring the biological, behavioral, and social consequences of alcohol consumption, even reduced consumption. Evaluations of alcoholism treatment studies have an advantage over psychotherapy treatment studies in that the former have many more objective QoL measures available than the latter (Foster *et al*., 1999; Gladis *et al*., 1999).

Hester (1995) advises alcoholism counselors,

> [R]esearch has noted relatively little success in teaching moderation to severely dependent alcoholics. You may find it helpful to distinguish, in your own mind, between problem drinkers and alcoholics, viewing abstinence as the only ultimately feasible goal for the latter. This distinction, though an oversimplification is generally consistent with the matching data. [p. 149]

This is the "method" suggested to alcoholism counselors for distinguishing between a problem drinker who will remain a problem drinker if they continue to drink and one who may become an alcoholic. Imagine the difference in your own mind! Fals-Stewart (1997) has demonstrated that in the absence of results from a neuropsychological test battery, substance abuse counselors cannot discriminate between impaired and unimpaired patients.

Hester (1995) continues,

> In those cases where we disagree with a client's desire to pursue a goal of moderation we negotiate a contract. We agree to work with the client for six to eight weeks, providing them training in BSCT [behavioral self-control training]. We agree, however, that if at the end of that time the client is still having difficulty drinking moderately, he or she will consider a goal of abstinence. [p. 149]

Hester assumes that he is dealing with a rational consumer similar to one considering whether to buy a four-door or a two-door car, with or without cruise control or turbocharger, etc. There is no apparent concern that the individual may be suffering from diminished capacity as a result of alcohol-induced brain dysfunction (Bechara, 2004), depression, panic, polydrug use, etc. How many of Hester's participants chose abstinence and how many insisted on controlled drinking? What limits were set as the cut-off for controlled drinking? What follow-ups have been done? Is there any evidence that this potentially iatrogenic treatment lacking a valid consent form is effective? There is no information or apparent concern to carefully monitor the behavior of the patients and repeatedly

contact collaterals, employers, and family members, much less obtain assessments of brain function and neuropsychological testing before, during, and after the 6–8 week trial period. Likewise, Sobell and Sobell (1993, 1995), in advocating their stepped-care variation of harm reduction, fail to address the critical issues of a valid informed consent and probable iatrogenic effects of continued drinking on brain structure and function already showing signs of damage.

Harm Reduction as Secondary Prevention

Effective prevention methods must be developed to adequately address the problem of alcoholism. Harm reduction has been presented as such an approach. It has been applied to heavy drinking on college campuses by Marlatt and his students (Dimeff *et al*., 1999; Marlatt *et al*., 1998). A manual implementing the program in question has been published (Dimeff *et al*., 1999).

In addition to its lack of verisimilitude as discussed in Chapter 4, Marlatt's secondary prevention of college students' alcohol problems lacks ecological validity, i.e. it has no value as a means of dealing with problems of excessive drinking on college campuses. A program such as the one in question requiring the assessment of incoming students who volunteer for the service cannot meet the needs of all heavy drinkers matriculating in a college. Offering a monetary reward for participating in the harm reduction program, Marlatt *et al*. (1998) were able to recruit approximately 75% of the 25% of matriculating students with the highest levels of alcohol consumption and problems. Criteria for the classification of the latter group as binge drinkers were that they consumed at least five or six drinks on one occasion in the past month or experienced at least three alcohol-related problems in the past 3 years. Participants were randomly assigned to a brief intervention or a control group. A representative comparison group was also assessed. None of the participants were over 19 years of age.

A follow-up 2 years after the intervention indicated that the treated students reported 3.3 alcohol-related problems in the previous 6 months, as compared to 4.7 problems for the untreated control group and 2.4 problems for the normative comparison group. The difference

between the experimental and control groups was statistically significant. Both groups showed a significant decline in problems since their college matriculation. Average drinking declined to 3.6 drinks/occasion by the treatment group as compared to 4 drinks/occasion by the nontreated control group; 70% of the high-risk binge drinking students receiving the intervention treatment reported drinking as much as five or six drinks on at least one occasion during the previous month as compared to 78% in the nontreated control group. In comparison, 42% of a normative representative cohort were binging. Although the results were statistically significant, the authors admit that the effect sizes were small, (i.e. 0.14–0.20). Results for the intervention and control participants were for students captured for the study as paid volunteers, less than half of the entering freshman class. Neuropsychological assessments were not obtained at baseline or follow-up.

A number of serious problems are posed by the study in addition to its lack of external validity. Evidence suggests that neurobiological brain function matures until at least the age of 21 years, and that there is an important spurt in maturation of the frontal lobe function between years 17–21 (Davies & Rose, 1999; Hudspeth & Pribram, 1992). Acheson, Stein, and Swartzwelder (1998) show that the consumption of alcohol producing a blood alcohol level (BAL) below 0.08 significantly impaired semantic and figural memory to a greater extent in men aged 21–24 than 25–29 years old. Animal studies demonstrate that binge drinking young rodents showed significantly greater brain damage than binge drinking adults (Crews, Braun, Hoplight, Switzer III & Knapp, 2000). What effect does drinking an average of more than 3 drinks/occasion and 5–6 or more at least once/month, the drinking rate of the treated students, have on a young person whose brain function is still maturing? Marlatt's harm reduction for college binge drinkers is, in all likelihood, a form of harm induction. It must be considered as such until it can be demonstrated that the level of drinking attained after "harm reduction" treatment does not increase brain dysfunction, which may or may not be reversible.

Sher *et al*. (1997) obtained significant correlations between neuropsychological tests and alcohol use disorders in heavy-drinking college freshmen. The greater the evidence of alcohol use disorders, the

greater the neuropsychological deficits, especially in visual-spatial functions. Such results indicate that Marlatt's harm reduction approach to binge drinking by college freshmen may be iatrogenic, placing these young people at risk for further brain damage as compared to the alternative of abstinence. Revisionists may argue that harm reduction/controlled drinking is used because it is difficult to convince underage college students to abstain; however, this paternalistic view is contrary to the principle of autonomy espoused by revisionists in their support of alcoholism treatment self-selection. Apparently, self-selection is needed only when abstinence treatment is offered paternalistically. Revisionists fail to consider the impact of material information on treatment self-selection when students are shown their neuropsychological and neuroimaging deficits in comparison to their abstinent cohort.

Another difficulty with Marlatt *et al.*'s (1998) study is that more than half of the high-risk drinkers were women, yet there was no indication that they were warned about fetal alcohol effects of drinking even moderate amounts of alcohol. They were not required to provide proof that they were not pregnant before engaging in controlled drinking training.

Marlatt *et al*. (1998) ignore the ethical and legal problems entailed by training minors to drink "moderately" rather than abstaining. Their harm reduction program apparently does not inform the student about the possible damage of brain function if they continue to drink. It condones the students' breaking the law with no attempt to determine the effects of the program on the attitudes of participants. "Harm reduction" for minors may produce far more serious psychological and neuropsychological problems than it reduces. It may produce moral harm which

> is inflicted on someone when some course of action produces in that person a greater propensity to commit wrongs. If a course of action will predictably inflict moral harm, will predictably make some person or persons morally worse, then even if the action which produces the moral harm is not itself the infliction of a wrong, it is prohibited for reasons that arise clearly from the wrongs that will be produced.

> Inducing others to look for the quick and undeserved reward and teaching others to behave in ways that will produce cynicism are clearly examples of the infliction of moral harm. Thus *moral harm* is incommensurable with benefit in just the way that wrong is. [MacIntyre, 1982, p. 178]

There are further empirical and ethical problems of misusing limited resources when harm reduction/controlled drinking is used as secondary prevention. If we generalize the number of alcohol-related problems produced by the treated binge drinking students as compared to their normative cohort, we find that the former averaged approximately 3300 problems overall as compared to 7200 produced by their larger normative cohort. Individual treatment of a minority of the students who are heavy binge drinkers while neglecting the majority of students who drink on a college campus will not solve the problem of harm produced by college drinking.

The most effective approach to reducing the problem of heavy drinking on college campuses is primary, not secondary, prevention. A host of community-oriented models can be applied to the campus drinking problem. Above all, effective prevention must involve the entire community — students, student leaders, fraternity and sorority leaders and members, faculty, staff, administration, and the surrounding community. Norms of acceptable behavior must be changed, alternative activities must be encouraged, and policies limiting the access and exposure to alcohol and the social reinforcement for heavy drinking must be changed. These approaches work in broader communities (Williams *et al*., 1999). They must be employed in the college community. Encouraging results suggest that an effective component of the primary prevention approach is the use of "social capital" (Weitzman & Kawachi, 2000). Examination of a national representative college sample of more than 17 000 college students showed that students on campuses with a higher-than-average rate of volunteering had a 26% lower rate for binge drinking than their cohort on other campuses. Promoting participation in volunteer activities not only promotes the social good, but also reduces individual harm.

Advantages of primary prevention as compared to the secondary prevention approach advocated by Marlatt are reviewed by Wagenaar and Perry (1994):

> Prevention efforts and public action strategies must be based on an understanding of the factors that affect patterns of alcohol use across the whole population of young people rather than just the smaller sample of those identified as problem drinkers. Predicting with great sensitivity and selectivity why a given individual will drink and understanding the nature of the consequences of individual drinking may be important in a clinical setting in which a small set of individuals with alcohol-related problems is the focus of attention. Applying such an individual focus to the development of population-wide prevention efforts is, however, not fruitful.
>
> A population-wide focus on how the social environment fosters youth drinking is necessary for several reasons. . . . First, the magnitude of the problem is such that even if we had a perfect "cure" we most likely would not have the resources to apply it to all who are at risk. Second, because more than half of high-school seniors drink and one third become intoxicated at least once in 2 weeks . . . it is evident that many youths are at significant risk for car crash, assault, rape, injury, or another serious problem associated with drinking. Third, there is constant turnover in the high-risk population. . . . Thus, even if we cure the current incumbents of the high-risk pool, in a short period of time turnover is such that little long-term benefit is achieved. The fourth reason a population-wide focus is necessary is that the majority of alcohol-related death and disability is attributable to moderate drinkers, not those who are alcohol dependent. The heaviest drinkers are clearly at highest risk of problems, but there are so many more people in the lower risk 'moderate' drinking group that a lower individual risk still results in a larger aggregate burden to society. . . . This is the classic *population attributable* risk concept in epidemiology . . . which is so frequently overlooked when prevention problems are designed solely focusing on 'high-risk' youth. [pp. 197–198]

Fortunately, there is a population-wide primary prevention method available for college drinking that is supported by extensive evidence on different college campuses (Perkins, 2003). It is called the social norms marketing (SNM) campaign. Further evidence supporting the effectiveness of SNM as a primary prevention method has been provided by a striking randomized controlled trial by DeJong and his colleagues (DeJong *et al*., 2006).

Underlying the SNM approach is the hypothesis that college students who drink excessively believe that the average college drinking, the norm, is much higher than it actually is. They aim to match the mythical norm. Providing accurate information on the actual levels of drinking at students' specific schools will result in lowered levels of alcohol consumption and its negative consequences. Eighteen institutions of higher learning varying in size were selected from four different geographic regions. They were assigned to pairs matched on the basis of a variety of predictors, including student demographics such as gender, ethnicity, and membership in fraternities/sororities. One school from each pair was randomly chosen to be part of the experimental condition receiving SNM campaigns. No SNM campaign occurred in the schools randomly assigned to the control condition. More than 5000 students participated in the study, which was conducted over a period of 3 academic years. An enormous amount of detailed results was obtained. For our purposes, it is sufficient to report that students receiving SNM campaigns were consuming significantly less alcohol than the control group at the end of 3 years and suffered significantly fewer negative consequences.

Generality of Problem of Adherence to Ethical Principles

A mail survey conducted by McCrady and Bux (1999) confirmed that federally funded investigators of alcohol and drug abuse problems are lax in their adherence to the ethical principles of the Belmont Report (National Commission, 1978) and the federal regulations based on them (Basic DHHS Policy for Protection of Human Research Subjects, 1991). However, their survey failed to consider the obligation of the caregiver/investigator to provide material information on brain function, of

particular concern when continued controlled drinking is a treatment option. McCrady and Bux, as is true of other cognitive behavior therapists, failed to consider the iatrogenic consequences of continued drinking in the presence of existing brain damage.

Evaluating Diminished Intellectual Capacity

To make a valid autonomous selection among alternative treatment goals, the prospective patient or research participant must be informed of the alternative treatments available and the possible positive and negative consequences of each. Patients must receive information on the long-term recovery rate for their condition, abuse or dependence, polydrug use, comorbidity, etc. Patients must have the capacity to understand the information concerning consequences of the treatments and their relevance to their own condition. They must be capable of evaluating and integrating the different kinds of information for each treatment alternative, and transmitting their selection to the therapist in an understandable fashion (Beauchamp & Walters, 1982; Faden & Beauchamp, 1986; Grisso & Appelbaum, 1998; Levine, 1988; Pincus *et al*., 1999; Schneider, 1998).

Understanding Information

Following many others (Grisso & Appelbaum, 1998; Levine, 1988), Mann (1994) has conducted an experimental study of informed consent that is particularly relevant to the problem at hand. Employing normal college students as participants rather than schizophrenics or people suffering from life-threatening illnesses, she investigated variables possibly affecting participants' understanding of the consent form material they read prior to entering an experimental study. Eighty-three Stanford University undergraduate students were prepared to participate in an experiment exposing them to magnetic resonance imaging (MRI), a brain scan designed to study the localization of memory functions. Three different groups of randomly assigned students received an adaptation of a standard consent form used in MRI studies. They received either a long consent form, a short form matched for readability with the long

form, or an information sheet comparable to the long consent but without a space requiring the student's signature. Less detail and redundancy distinguished the short form from the long form. The information sheet differed from the long consent form only in the absence of the "consent form" title, space for a signature, and information pertaining to the meaning of a consent form. Reading difficulty of the three forms was at the 12th-grade level.

Results showed that participants receiving the short form correctly answered more of the specific questions concerning the experiment than those who received the long consent form. They did not differ in response to the general questions. Some of the questions were answered incorrectly by a majority of participants, regardless of length of consent form. A sample of such questions follows, with the percent correct in parentheses:

> "Are there risks to this procedure?" (48%)
> "What can you do if the sound of the machine bothers you?" (45%)
> "What will the researchers do for you if you get hurt?" (45%)
> "Name two of the four things your signature on the consent form means." (20%)

The results obtained by Mann (1994) with Stanford undergraduates gives pause about how truly informed an individual is when making a choice of alcoholism treatment. Stanford students correctly answered only 60% of the specific questions dealing with the risks, procedures, and benefits of the experiment — information essential for a truly informed consent. Only 1 of 53 students could state the procedure for registering a complaint against an experiment. Two thirds thought that they gave up their rights to sue for negligence when they signed the consent form, despite the fact that the consent form "stated explicitly that subjects who signed the forms were not giving up any rights to sue" (Mann, 1994, p. 142). If an intellectually superior group of young people are so poorly informed after reading an informed consent providing information about the experiment as well as their rights, what could one expect from the average problem drinker or alcoholic?

A selection or decision among alternative treatment modalities is difficult enough in terms of remembering, integrating, and evaluating diverse kinds of information. The difficulty is compounded by individual differences among problem drinkers and alcoholics produced by such coexisting problems as alcohol-induced brain dysfunction, depression, anxiety, panic, and polydrug use. Alcoholics and problem drinkers poised for treatment are usually there because of an acute situation, a crisis of some kind. It is not the best of conditions for making judicious decisions; reasoning; and balancing positive and negative aspects of alternative treatments as well as conflicting advice from different counselors, friends, family, and what has been read in popular magazines and found on the Internet.

Techniques are available for assessing patients' capacity to provide an informed consent and self-select treatments (Grisso & Appelbaum, 1998). They involve the direct observation of patients' capacity to understand the meaning of information provided, appreciate its relevance, and express a choice that can be sustained. Procedures for assessing the necessary capacity to provide an informed consent are contained in a structured interview and rating procedure developed by Grisso and Appelbaum, the MacArthur Competence Assessment Tool for Treatment (MacCAT-T). The MacCAT-T assesses the four abilities judged as necessary for an informed consent that is ethically and legally valid. It requires approximately 20 minutes to administer and score. Quantification of the interview responses permits the development of norms to which individual cases can be compared. Grisso and Appelbaum describe the results of a number of studies they and other investigators have conducted assessing the capacity of mental patients to provide informed consent. I know of no such studies assessing the capacity of alcoholics, problem drinkers, and heavy drinkers necessary for a valid informed consent. Such research is essential before harm reduction can be offered as a treatment alternative.

When the principles of autonomy and beneficence are in conflict, for example when problem drinkers and alcoholics are faced with the decision to select either an abstention or a harm reduction treatment goal, paternalism resolves the conflict. A decision is made by the caregiver to override the autonomous decision of the individual when he or

she selects a treatment goal of controlled drinking that increases the risk of causing harm to themselves and others. Given the neurological and neuropsychological evidence that the majority of individuals who are classifiable as problem drinkers and alcoholics have brain dysfunction, caregivers and investigators who advocate self-selection and "harm reduction" are providing an iatrogenic treatment that may exacerbate existing brain damage.

Denial

The assertion that all patients entering treatment want autonomy and wish to have self-selection of their treatment is not self-evident. Schneider (1998) has shown that a large number of patients entering medical treatment for serious illnesses do not wish to have self-selection of their treatment. They wish someone else to make the decision for them; this is often the health provider. Schneider also reports that there is considerable denial in seriously ill people such as patients in need of a liver transplant. They deny that they are seriously ill and may therefore select a less severe treatment. Standardized procedures for assessing the denial of physical illnesses are available (Levine *et al*., 1994). Denial or diminished capacity to appreciate that one is ill has also been reported as a diagnostic feature of schizophrenia (Carpenter *et al*., 1976, 1978).

Denial of illness or its symptoms, a person's lack of capacity to appreciate the nature or severity of their disorder, is common to a variety of disorders. It is a form of self-deception (Myslobodsky, 1997). Two general interpretations of denial have been offered. One is motivational: denial is a form of avoidance serving as a means of coping with anxiety. Another interpretation is that the lack of insight into the severity and characteristics of the disorder typical of denial is a consequence of neuropsychological deficits. The latter theory is supported by evidence from anosognosia, the lack of awareness of deficits due to a specific brain lesion, damage to the frontal lobes (Goldberg, 2001). Our hypothesis is that denial or lack of insight by alcoholics is at least in part the result of brain damage, especially in the frontal lobes responsible for executive functions: in some alcoholics, denial is a form of anosognosia.

Confirmation of this hypothesis requires assessments of frontal lobe structure and function.

Mohamed *et al.*'s (1999) study of denial in schizophrenics provides evidence supporting the neuropsychological deficit hypothesis. They obtained significant correlations between performance on measures of frontal lobe executive functions (Wisconsin Card Sorting Test and fluency) and measures of symptom awareness. Their results are noteworthy because the neuropsychological correlation with symptom awareness was corrected for intelligence. Given the extensive evidence — some of which has been mentioned above — that excessive alcohol consumption produces significant deficits in brain function, including deficits in frontal lobe function, the implication is that denial or lack of awareness of alcoholism symptomology is due at least in part to frontal lobe deficits. As noted in Chapter 2, Rinn *et al.* (2002) demonstrated that the denial of symptoms of alcohol misuse is significantly related to validated neuropsychological tests of frontal lobe functions.

Denial is a major treatment problem that is largely ignored by cognitive behavior therapists, other than to denigrate it or assume that it occurs because people wish to avoid the stigma of being labeled an alcoholic (Tucker & King, 1999). The problem is to overcome denial in people who have anosognosia and differentiate them from people who rationalize their drinking behavior.

The alcoholic in true denial must be differentiated from the person with a drinking problem who desires harm reduction rather than abstinence, is not in denial, and shows no evidence of structural or functional brain damage. Making this distinction requires standardized assessment procedures and a reliable research-derived database. Instruments are available for assessing denial in alcoholics (Goldsmith & Green, 1988; Newsome & Ditzler, 1993; Ward & Rothaus, 1991). There clearly is a need to design and conduct studies investigating the implications of denial for treatment self-selection. Harm reduction self-selection advocates have failed to conduct this essential research, just as they have failed to conduct essential neuropsychological research and research on the capacity of patients to provide a valid informed consent.

Reasons for Revisionists' Failure to Accept Paternalism: Hyperrationalism, Demonization, and Conflict of Interest

There are several apparent reasons for the failure of revisionists to recognize the need for paternalism in the conflict between autonomy and beneficence in the treatment of alcoholics and problem drinkers: (1) hyperrationalism, (2) demonization of the disease conception of alcoholism, and (3) a conflict of interest between the goals of science and ambition.

Hyperrationalism

According to Schneider (1998), hyperrationalism is

> . . . the substitution of reason for information and analysis. It has two components . . . [:] the belief that reason can reliably be used to infer facts where evidence is unavailable or incomplete and . . . the practice of interpreting facts through a set of artificial analytic categories. . . . [I]t tempts us to believe we can understand how people think and act merely by reasoning and without investigating. It lures us to discuss human behavior without studying how people actually behave. It is the conceptualist's revenge for the world's complexity. . . . [I]n bioethics as in other areas it sees people as acting in remarkably rational ways. [p. xv]

Advocates of self-selection paint a hyperrationalist picture of individuals presenting for alcoholism treatment. According to revisionists, the individual in need of treatment for alcoholism is a consumer, similar to someone shopping for a car. They ought to compare all of the models and features, prices, discounts, etc., and select the best buy meeting their needs. Autonomists assume that the prospective patient has normal memory, reasoning, and planning capacity. Patients are partners in decision making concerning treatment because they are reasonable, rational people in search of the means of changing their bad habit of drinking excessively. This is in the face of an extensive literature indicating that alcoholics are often comorbid, suffering from anxiety, panic, or depression, and/or other drug misuse quite aside from probable brain damage

due to the direct effects of alcohol and its metabolites as well as a relatively high incidence of head injuries (Chandler *et al*., 1975; Glatt, 1995; Hore, 1995; Parsons, 1998).

Demonization

A second reason for revisionists' failure to accept paternalism is their demonization of the disease conception of alcoholism. Alcoholism for them is nothing but a bad habit (Fingarette, 1988; Marlatt, 1983). Consequences of the bad habit may be brain dysfunction, liver disease, legal and interpersonal problems, etc. Disease is the consequence, not the cause, of bad habits and inappropriate expectancies. According to revisionists, the current notion that alcoholism is a disease is the myth of lay people in AA. Anyone who adopts an alternative, more scientific, view such as controlled drinking based on scientific research is vilified. Supposedly, "The disease model of addiction has shown impressive tenacity in the face of overwhelming contrary scientific evidence (e.g., Fingarette, 1988; Sobell & Sobell, 1984)" (Vuchinich, 1996, p. 598), but nothing could be further from the truth. Critical analyses of Fingarette (1988) and of Sobell and Sobell (1984) delineating their many shortcomings may be found in Maltzman (2000).

An extraordinary characterization of the disease conception is the following:

> The treatment of alcohol abuse is now in a state of crisis and those involved in this treatment have a crucial choice to make. The choice is not between total abstinence and moderation, or between Alcoholics Anonymous and behavioural psychology. It is simply between dogma, prejudice and arbitrary authority, on the one hand, and openness, reason and a belief in the scientific method, on the other. [Heather, 1980, p. 258]

Reality

The "physical disease" treatment provided by the most widely used approach to alcoholism treatment in this country, the Minnesota Model,

began its development in the 1950s. It was initiated, primarily, by a clinical psychologist, Daniel J. Anderson (Anderson *et al*., 1999). Its purpose was to combine the latest in clinical approaches with principles of the 12 steps of AA. Elsewhere (Maltzman, 2000, p. 204), I present the average time devoted to different treatment modalities in 13 Minnesota Model treatment facilities in the Midwest. They vary considerably; there is no "one suit fits all". Aside from the discussion of the 12 steps of AA, there would be little difference from an eclectic clinical treatment program, except perhaps for a greater emphasis on family counseling and education in the Minnesota Model programs. An evaluation of some 9000 patients from primarily Minnesota Model programs by Harrison *et al*. (1991) yielded treatment outcomes far superior to results obtained with CBT such as those reported by Hester and Miller (1995) and by Miller *et al*. (1992).

Differential aversion conditioning combined with interviews under sodium pentothal and individual and family counseling in a 10-day treatment program, as practiced by the Schick Shadel hospital, is also based on the disease conception of alcoholism. Its success rate is higher than any other treatment program that has had thorough treatment evaluations, yet it is ignored and misinterpreted by cognitive behavior therapists (see Maltzman, 2000). Revisionists also fail to consider the voluminous biomedical literature as well as conceptual analyses demonstrating that alcoholism meets the criteria for classification as a disease, as discussed in Chapter 1 and elsewhere (Maltzman, 1994, 2000).

A Conflict of Interest

There is a third factor determining revisionists' failure to use a valid informed consent, to provide assessments that identify the harms and benefits of harm reduction (material information for a consent form), and to ensure the competence of patients they ask to self-select treatment: a conflict of interest between the goals of science and their practice. The goal of science is to approximate the truth (Popper, 1962). The goal of cognitive behavior therapists' clinical practice is to enlarge market share vs. traditional Minnesota Model treatment (McCrady, 1986; Miller, 1987). There is an inherent conflict of interest when a scientist

advocates a procedure lacking verisimilitude (Maltzman, 2000; Popper, 1962) and when the procedure benefits the scientist/practitioner financially. Harm reduction advocacy will produce monetary gain through enhanced market share, grants, workshop fees, etc.

Exemplary treatment evaluations separate the investigator from the caregiver; independent teams treat the patients and conduct the research. In contrast, in most efficacy studies conducted by cognitive behavior therapists, they or their students and colleagues treat the patients as well as conduct the research and prepare the results for publication. There is a vested interest, i.e. monetary gain, involved in obtaining results of a particular kind. Such a conflict of interest leads to doubts concerning the reliability and validity of the research, and the procurement of valid informed consent for harm reduction research and treatment. Such conflict of interest contributes to poor research and a lack of criticism from fellow revisionists. The problem is exacerbated by the effort to silence criticism from other sources. Science, practice, and the public they serve suffers as a consequence (Maltzman, 2000).

Conclusions

Scientist-practitioners in clinical psychology have much to learn from biomedical science. Foremost is that alcoholism is more than merely a bad habit obeying the ordinary principles of learning. It is a disease in which the behavioral symptoms are a consequence of structural and functional brain damage produced as a consequence of alcohol and interactions with the social environment. Standardized methods for determining the capacity of patients to provide a valid consent to participate in research and treatment have been developed. There is a vital need to conduct research with these procedures on individuals entering treatment for alcohol misuse, abuse, and dependence.

Standardized scales have also been developed for the assessment of denial of alcoholism signs and symptoms. They should be used in systematic studies of heavy drinkers as well as individuals diagnosed as suffering from alcohol abuse and dependence. Denial, a form of lack of self-awareness caused by brain dysfunction, must be differentiated from rationalization, caused by fear and by a true absence of symptoms.

Administration of a neuropsychological test battery should be standard practice as part of an assessment that must be conducted to provide information necessary for a valid consent, a consent that contains all material information. The use of such assessments as well as QoL measures is essential. Research using neuroimaging and electrophysiological methods must determine the state of brain structure and function over the entire range of alcohol use and misuse disorders. It is time that cognitive behavior therapists and others recognize the overwhelming evidence that alcoholism is more than merely a bad habit or a particular lifestyle chosen by an individual and, because of its deviance, stigmatized by a straight majority.

Medicine recognizes the serious problem of iatrogenic harm, damage caused by a practitioner's own treatments (Sharpe & Faden, 1998). Psychology must recognize that it faces a similar problem with the promotion of "harm reduction" as a method of dealing with alcohol misuse disorders in the absence of relevant patient assessments and ongoing basic research on the brain, behavior, and their interaction with the social environment.

6

Little Albert Redux II: Bias and Lack of Scholarship in Textbooks

According to the *APA Ethics Code 6.3. Fairness in Teaching*, "When engaged in teaching or training psychologists present psychological information accurately and with a reasonable degree of objectivity" (APA, 2002, p. 1068). Unfortunately, all too often we have found cognitive behavior therapists in the alcoholism field following a different rule: "If you tell a big enough lie and tell it frequently enough, it will be believed" (Joseph Goebbels).

The present chapter is an update of Chapter 9, "Little Albert Redux", in Maltzman (2000). To me, an inveterate behaviorist, the story of Little Albert is a classic. I read the original report by Watson and Rayner (1920) when I was an undergraduate more than half a century ago. I will summarize it briefly for readers unfamiliar with the tale and its aftermath. It is the latter phenomenon which is of primary interest here.

Little Albert was the subject in the first experiment demonstrating the classical conditioning of fear in a human — a 9-month-old infant. Watson and Rayner (1920) first demonstrated that 9-month-old Albert displayed no fear in the presence of live animals (e.g. a rat, rabbit, or dog) or inanimate objects (e.g. cotton, a human mask, a burning newspaper).

Watson had earlier established that two conditions evoke unconditioned fear in an infant: a sudden loud noise, and loss of support. Watson and Rayner used the former as their unconditioned stimulus. Albert showed fear, manifested by crying and avoidance, when a loud noise was suddenly produced behind him by striking a steel bar with a hammer. Fear was conditioned to a rat by pairing the loud noise with Albert touching the rat. Generalization test trials were later introduced by presenting a rabbit, dog, sealskin coat, cotton, etc. Albert displayed generalized fear to the rabbit, dog, and sealskin coat, but not many other objects. Watson and Rayner did not determine the persistence of the fear, and did not attempt to extinguish the crying and avoidance elicited by the rat and generalization stimuli.

With the passage of time, accounts of this classic experiment drifted from the original in a variety of details. Was the feared subject a rat or a cat? Did Watson and Rayner (1920) extinguish the fear in Little Al or allow him to remain fearful of small furry animals for the remainder of his life? Failing to read the original study by Watson and Rayner led later writers to invent a number of variations in the nature of the experiment, as described by Cornwell and Hobbs (1976) and Harris (1979).

Maltzman (2000) describes in some detail the Little Al phenomenon as it played out in earlier efforts to publish the evidential basis of my allegations that the Sobells committed fraud, and to show the misrepresentations of related events such as the nature and outcome of the fundamentally flawed investigations of my allegations. A singular bias in dealing with this problem of integrity in science and scholarship is still apparent in the psychology literature (Maltzman, 2000). However, our present effort in Little Al Redux II is not limited to the textbook treatment of the alleged Sobell fraud. It is concerned with a broader discussion of alcoholism: its etiology, characteristics, and treatment — or more accurately, mistreatment — in abnormal psychology textbooks. Since the Sobell controversy is still often misrepresented as an attack by an outdated ideology (i.e. the disease conception) on a scientific approach to alcoholism, a brief presentation of some of the evidence substantiating the charge of fraud will be presented during the course of this review. A more extensive account of the case as well as the evidence supporting the allegation of fraud

and the failure to adequately evaluate the problem may be found in Maltzman (2000).

Textbooks were not randomly selected for study from among all available abnormal psychology textbooks. The textbooks examined were available in my Psychology Department office because they were used in abnormal psychology courses taught by the University of California, Los Angeles (UCLA) Department of Psychology faculty, they were submitted to the Department by the publisher for consideration, I found them in the UCLA Bookstore, or a colleague gave them to me. Books examined here should be representative of the kind of textbooks used in large state universities, and probably many other universities and colleges. This generalization seems reasonable because of the name recognition of the majority of textbook authors and the high repute with which they are held in the field of clinical psychology. The textbooks will be reviewed in alphabetical order by author or senior author, followed by an examination of two popular textbooks written for alcoholism counselors and other helping professionals in the alcoholism treatment field. My comments are limited to the topic of alcoholism in the abnormal psychology textbooks reviewed.

Textbooks reviewed cover the DSM-IV classification of alcoholism as one of several forms of drug dependence. However, during their discussion of alcohol use, they do not always distinguish between abuse and dependence, and may use the terms "addiction" or "problem drinking" without clearly defining them.

The etiology of alcohol use and alcoholism is discussed in textbooks, with an emphasis on the role of expectancies. Treatment discussions center around cognitive behavioral treatments and Alcoholics Anonymous (AA). The most widely used treatment program, the Minnesota Model, the treatment for which there is more treatment outcome data available than any other by far (Harrison *et al*., 1991), is not mentioned — much less discussed at length — in the textbooks reviewed. The textbooks fail to present any of the experimental research discussed in Chapter 5 contradicting the expectancy formulation favored by textbook authors. Research on animal models of alcoholism is also ignored.

If the disease conception of alcoholism is mentioned, it is the usual straw man invention concocted by cognitivist opponents misrepresenting

Jellinek's (1946, 1952, 1960) classic work on the disease conception of alcoholism and its origins. Finally, and inexcusably, not one textbook discusses the fundamental problem of structural and functional brain damage caused by excessive alcohol consumption, the possibility of reversing the damage following abstinence, and the likelihood of exacerbating existing brain damage by continued (even moderate) drinking. Before turning to the specific textbooks for a more detailed examination of their discussion of alcoholism and its etiology, characteristics, and treatment, I will digress to provide some background and evidence for my allegations that the Sobells fabricated their results and misrepresented their procedures as well as some background on how I became involved in the controversy.

Background to My Entering the Field of Alcoholism Studies

It was a result of my responsibilities as Chair of the Psychology Department at UCLA that I became enmeshed in the aftermath of the Little Albert affair and that my research focus on human psychophysiology and the orienting reflex (OR) shifted to alcohol studies. I received a grant proposal from a young assistant professor in my Department that I was required to review before submitting it to the university administration, who in turn would submit it to the appropriate national funding agency. The research grant proposed establishing a program in which undergraduate psychology students in a fieldwork course would train alcoholics treated at a Veterans Administration (VA) hospital located near the university campus to control their drinking. A student "behavior modifier" would escort a veteran to a local bar and train the veteran to control his drinking according to the principles laid down by Mark Sobell and Linda Sobell (1972) in a monograph published by the California Department of Mental Hygiene. It did not make sense to me because I did not think that young undergraduate students could effectively assume such a responsibility.

At the time, I knew nothing about the disease conception of alcoholism, AA, or abstinence as a treatment goal. However, a former graduate student of mine who conducted her PhD dissertation on the psychophysiology of the OR was married to a resident in psychiatry at

UCLA. Following the receipt of his MD, he became Chairman of the Psychiatry Department at the new University of California campus in Irvine, CA. A few years later, he moved south to take on the role of the Chair of Psychiatry at the new University of California campus in San Diego. Mary Pendery, my former student, realized that she could not obtain academic positions in psychology with the frequent moves of her husband, Arnold, from Chair to Chair in psychiatry. She received training in clinical psychology, and in a few years was licensed as a clinical psychologist. When Arnold was hired as Chair of the Department of Psychiatry at the new UC San Diego, Mary obtained a position as head of the alcoholism treatment unit at the new San Diego VA Hospital.

I called Mary and asked her what she thought of the Sobells' controlled drinking approach to alcoholism treatment. She said she was not familiar with the monograph and had never seen an alcoholic learn to drink in a controlled fashion. She suggested we visit Patton State Hospital and speak to the Sobells about their new kind of treatment. I called Jack Fox, a former PhD student of mine who had become the chief clinical psychologist at Patton, and asked him if he could arrange a meeting with the Sobells. Jack told me that the Sobells had moved to a facility in Orange County, but that Mary and I could visit Patton and see the alcoholism treatment facilities (including a bar) used by the Sobells in their treatment. When we came to Patton, Jack and other members of the clinical psychology staff, who also happened to be former PhD students of mine, urged us to follow up on the patients. Jack had been receiving telephone calls from all over the country asking about the controlled drinking study conducted at Patton. However, Jack stated that he saw many of the patients returning to the hospital for treatment following relapse. We eventually did follow up on the patients. Further details describing the background to our follow up of the patients in the Sobell study are provided in Maltzman (2000, Chapters 4 & 5).

I did not attack the Sobells' study and accuse them of fraud because of my belief in the disease conception of alcoholism. I subsequently learned about the disease conception of alcoholism, the Minnesota Model of treatment, and AA because of the Sobells' attacking me for my assertion to the press that I believe they faked their data and misrepresented their procedures. My allegation was in response to their inappropriate

assertions to the media following the publication of the Pendery *et al*. (1982) study. I accused the Sobells of fraud because the majority of patients we interviewed informed us that they were not interviewed every 3–4 weeks for 2 full years. My accusation of fraud was not based solely upon patients' verbal reports. A study of the medical records maintained by the Patton State Hospital revealed that, contrary to the Sobells' public pronouncements and publications, the majority of patients had relapsed within a year following their treatment.

Maltzman (2000, p. 144) provides a photocopy of a page from the Dickens Committee Report (Dickens *et al*., 1982) showing the number of telephone interview contacts the Sobells purportedly obtained. Participants obviously were not interviewed every 3–4 weeks, a total of some 24 times a year. Neither the Dickens Committee nor any other investigative body took the first essential step in an investigation of fraud in science: obtain the raw data — in the Sobells' case, the timeline follow-back answer sheets — and determine whether or not they can reconstruct the published data. The Dickens Committee never obtained the timeline follow-back interview forms from each of the patients. The following extract from my book (Maltzman, 2000) provides some of the evidence that I believe incontrovertibly shows that the Sobells intentionally misrepresented their experimental procedures and invented much of their data:

> The Sobells concede that they were careless in stating the number of contacts (Sobell & Sobell, 1989). An important point made by the Dickens Report is that the Sobells were visibly surprised when the deficient number of contacts was revealed to them. The implication is that if they had not been surprised, then they would have had prior knowledge that the number of contacts was less than stated. If this were true, they would be guilty of intentionally misrepresenting the frequency of interviews. Furthermore, if the interviews were less frequent than stated, the data obtained from these interviews, the daily drinking dispositions, might also be missing, therefore summary results based upon them fabricated.
>
> Contrary to the assertion of the Dickens Report and the Sobells' current position, there is evidence that the Sobells knew from the

TABLE 5.2 Contact Dates for Experimental Subject #14 Discharged from Patton State Hospital, October 9, 1970 (Dickens *et al*., 1982, p. 81)

Year 1 subject contacts	Year 2 subject contacts
November 8, 1970	March 23, 1972
January 15, 1971	May 24, 1972
January 25, 1971	July 3, 1972
February 24, 1971	August 3, 1972
March 15, 1971	August 31, 1972
April 13, 1971	September 27, 1972
June 2, 1971	
June 17, 1971	
September 10, 1971	
October 9, 1971	

outset that the frequency of interviews was less than every 3–4 weeks. Furthermore, the Dickens Committee had to know the Sobells possessed this knowledge. These conclusions follow from (a) the nature of the time-line follow-back method, which the Sobells insist they used, and (b) mine and Pendery's affidavits that are listed in the data base of the Dickens Report (p. 40). Comments on these affidavits indicate that the Committee had read my allegations. Table 5.2 [as designated in original text] shows the results for one of the four experimental patients randomly selected by the Dickens Committee (p. 81) to illustrate frequency of patient interviews.

He is experimental Subject #14, HC. Note the gap of approximately 5.5 months between the last contact in October 9 of the first year, 1971, and March 23, 1972, the first contact in the second year. There were only 10 days between the second and third contacts, January 15 and January 25, 1971. Contacts were not evenly distributed. In the case of the 5.5-month interval, as in every other time interval, the daily drinking disposition is reported as days abstinent, controlled drinking, drunk days, days incarcerated in hospital or jail. The first three are reconstructed from the interview, dependent entirely upon the daily drinking disposition for every day of the previous 5.5 months. Since such data are reported for this subject, how did the Sobells know that they had to reconstruct the drinking dispositions for 5.5 months?

> Since they reported data for two years for this subject, they had to know that they were not interviewing him on a monthly basis, otherwise they could not know when to stop their time-line follow-back procedure — how far back to reconstruct to have complete information for every day of the previous 5.5 months.
>
> Daily drinking dispositions for every day of the year obtained by the time-line follow-back method necessarily requires knowing when a subject was last interviewed, and therefore how frequently he was interviewed. If the Sobells did not fabricate their results for experimental Subject #14, and in fact used the time-line follow-back method to reconstruct his daily drinking disposition, they had to reconstruct 5.5 months of daily drinking dispositions; to do so, they had to know they did not interview him every 3–4 weeks. This could be true of all their subjects. As Table 5.1 [not shown here] indicates, the majority did not receive a minimum of 26–34 contacts, but they have published summaries of purported daily drinking dispositions for every day of these two years. One cannot have the latter drinking dispositions using the time-line follow-back method, and *not know* that interviews are not occurring every 3–4 weeks. [Maltzman, 2000, p. 147]

We will turn now to the review of the topic of alcoholism found in my sample of abnormal psychology textbooks.

Barlow and Durand (2005)

Barlow and Durand (2005) have written a popular undergraduate abnormal psychology textbook, now in its fourth edition. It repeats verbatim errors of fact and misrepresentations found in the first edition (Barlow & Durand, 1995) examined earlier (Maltzman, 2000, Chapter 9).

Phaseology

Barlow and Durand (2005) state,

> It used to be thought that once problems arose with drinking, they would become steadily worse, following a predictable downward

pattern so long as the person kept drinking (Sobell & Sobell, 1993). In other words, like a disease that isn't treated properly, alcoholism will get progressively worse if left unchecked. First championed by Jellinek more than 50 years ago, this view continues to influence the way people view and treat the disorder (Jellinek, 1946, 1952, 1960). Unfortunately, Jellinek based his model of *the progression of alcohol use* [emphasis added] on a now famous but faulty study (Jellinek, 1946), which we briefly review.

In 1945 the newly formed self-help organization Alcoholics Anonymous (AA) sent out some 1,600 surveys to its members *asking them to describe symptoms related to drinking* [emphasis added], such as feelings of guilt or remorse, and rationalizations about their actions, and to note when these reactions first occurred. Only 98 of the almost 1,600 surveys were returned, however. As you know, such a small response could seriously affect data interpretation. Obviously, a group of 98 may be very different from the group as a whole, so they may not represent the typical person with alcohol problems. Also, because the responses were retrospective (participants were recalling past events), their responses may be inaccurate. Despite these and other problems, Jellinek agreed to analyze the data, and he developed a four-stage model for the progression of alcoholism based on this limited information (Jellinek, 1952). According to his model, individuals go through a *prealcoholic stage* (drinking occasionally with few serious consequences), a *prodromal stage* (drinking heavily but with few outward signs of a problem), a *crucial stage* (loss of control, with occasional binges), and a *chronic stage* (the primary daily activities involve getting and drinking alcohol). A number of attempts by other researchers to confirm this progress of stages has not been successful (Schuckit *et al*., 1993). . . . Among these chronically alcohol-dependent men, a general progression of alcohol-related problems did emerge, although not in the specific pattern proposed by Jellinek. . . . This study suggests a common pattern among people with chronic alcohol abuse and dependence, one with increasingly severe consequences. This progressive pattern is not inevitable for everyone who abuses alcohol, although we do not as yet understand what distinguishes those who are and those who are not susceptible. . . . [p. 388f]

Numerous errors are present in the above quotation from Barlow and Durand (2005). Criticisms of Jellinek's (1946) study are appropriate; they were criticisms that Jellinek (1946) himself made. However, Barlow and Durand fail to report that Jellinek modified and added questions to the original questionnaire and then obtained results from a new sample of more than 2000 AA members (Jellinek, 1952). His formulation of a temporal progression is based on the latter report, not the 1946 study of 98 selected questionnaires from AA members.

Barlow and Durand (2005) erroneously assert that the questions on the original survey in 1946 elicited descriptions of the symptoms from AA members. This is an invention. The questions were actually formulated by the authors of the *Grapevine*, the AA newsletter. Jellinek was a biostatistician and was fully aware of the shortcomings of this original survey. He treated it as an exploratory study, revised the original questions, and added new questions. Jellinek used this new questionnaire to obtain data from 2000 male alcoholics providing the basis for the phaseology (Jellinek, 1952). Barlow and Durand (2005) cite the Jellinek (1952) study above, but apparently did not read it. If they had, they could not have made the false assertion that the phaseology is based on 98 subjects. Jellinek (1952, p. 676) explicitly states that the phaseology is based upon the analysis of results obtained from 2000 men. Jellinek is also explicit in stating that the phaseology describes the progression of symptoms in individuals who develop *gamma alcoholism*, not alcohol use or abuse. In modern terms, the progression describes individuals who are diagnosed as suffering from physiological dependence on alcohol; preclinical heavy drinkers or people diagnosed as suffering from alcohol abuse do not show the progression, unless they subsequently develop alcohol dependence.

Barlow and Durand's (2005) assertions concerning Jellinek's phaseology are further contradicted by the extensive body of subsequent research supporting the phaseology. This research is described at some length in Maltzman (2000). Briefly, early studies of the phaseology [e.g. Chick and Duffy (1979)] did not use all of the questions devised by Jellinek and analyzed their results using different methods; nevertheless, they agree that their results generally support Jellinek's phaseology, even if not the order of specific symptoms. The important finding in

harmony with Jellinek is that symptom clusters are not random in their appearance. Pokorny and Kanas (1980) administered all of Jellinek's questions as well as additional questions to patients at a VA hospital for alcoholism treatment rather than AA members; a comparison group of nonalcoholic VA patients were also given the questionnaire. The rank order at age of onset of symptoms and the rank order of Jellinek's symptoms were correlated at 0.72. Many of the same psychosocial signs and symptoms were shown by the control group, but not signs of physical dependence.

Schuckit *et al*. (1993) studied the progression of symptoms in 636 VA patients using a structured personal interview to obtain objective symptom information and collateral confirmation of symptoms. Contrary to Barlow and Durand (2005), Schuckit *et al*. concluded that their results are in accord with earlier studies showing a lawful progression in general agreement with Jellinek's results, although differing in the details of specific symptoms.

An extensive study supporting the conception of alcoholism phaseology as a manifestation of a progressive disease stems from a general population survey of more than 8000 people in a national stratified probability sample of noninstitutionalized adults in the continental United States (Nelson, Little, Heath, & Kessler, 1996). Previous studies usually employed hospital patients, AA members, or other convenience samples. Nelson *et al*. studied a national representative sample in an attempt to determine possible differences in the rate of progression, symptoms, and onset of dependence as a function of variables such as comorbidity. Criteria for dependence were based on the DSM-III-R. Nelson *et al*. report that three clusters of symptoms were isolated. Cluster A consists of symptoms of abuse, such as use in hazardous situations and use despite knowledge of problems. Cluster B is characterized by signs of tolerance and loss of control, unsuccessful attempts to reduce drinking, and increasing time spent drinking. Cluster C is characterized by withdrawal symptoms, drinking to avoid withdrawal, and restriction of activities due to drinking. An analysis of lifetime symptom profiles suggests that most people who have experienced alcohol dependence start with cluster A and then progress to cluster B and cluster C. Nelson *et al*. extended the generality of phaseology results obtained in prior

research on clinical samples by finding a characteristic temporal progression of stages in alcohol dependence in the noninstitutionalized general population. It must be recognized that, as in all diseases, infectious as well as chronic noninfectious, there is variability in the agent or pattern of alcohol consumption and in the host (i.e. the individual alcoholic.)

Controlled Drinking

Barlow and Durand (2005) continue their error-ridden discussion of alcoholism in their examination of controlled drinking:

> In the alcoholism treatment field, the notion of teaching people *controlled drinking* is extremely controversial, in part because of a study showing partial success in teaching severe abusers to drink in a limited way (Sobell & Sobell, 1978). The subjects were 40 male alcoholics in an alcoholism treatment program. . . . The men were assigned either to a program that taught them how to drink in moderation (experimental group) or a group that was abstinence oriented (control group). The researchers, Mark and Linda Sobell, followed the men for over 2 years, maintaining contact with 98% of them. During the second year after treatment, those who participated in the controlled drinking group were functioning well 85% of the time, whereas the men in the abstinence group were reported to be doing well only 42% of the time. . . . The results of this study suggest that controlled drinking may be a viable alternative to abstinence for some alcohol abusers, although it clearly isn't a cure.
>
> The controversy over the study began with a paper published in the prestigious journal *Science* (Pendery, Maltzman, & West, 1982). *The authors reported they had contacted the men in the Sobell study after 10 years* [emphasis added] and found that only 1 of the 20 men in the experimental group maintained a pattern of controlled drinking. [p. 380]

The emphasized phrase is an invention by Barlow and Durand (2005) — unfortunately, a not uncommon invention. We did *not* report

that we had "contacted the men in the Sobell study after 10 years". The Pendery *et al*. paper was published in 1982, 10 years after the Sobell study; however, the examination of hospital records and interviews with patients was initiated 3 years after treatment was completed. Critical data from hospital records were reported for 1-year posttreatment and interviews at a 3-year follow-up, the same time at which a purported follow-up by Caddy *et al*. (1978) was conducted with the cooperation of the Sobells. Details of the Caddy *et al*. affair — which I believe to be another extraordinary case of fraud — are provided elsewhere, along with the reasons for the delay in our follow-up (Maltzman, 2000).

Barlow and Durand's (2005) defense of the Sobells is essentially a duplication of their discussion in the first (Barlow & Durand, 1995) and subsequent editions of their textbook. They assert that the Pendery *et al*. (1982) paper had a number of flaws as revealed by Marlatt *et al*. (1993):

> Most serious was the lack of data on the abstinence group over the same 10-year follow-up period. Because no treatment study on substance abuse pretends to help everyone who participates, control groups are added to compare progress. In this case, we obviously need to know how well the controlled drinking group fared compared to the abstinence group. [p. 410f]

Barlow and Durand (2005) rely on secondary sources such as Marlatt *et al*. (1993) — fellow cognitive behavior therapy (BT) ideologs, hardly disinterested or careful observers — for a description of the Sobell affair. Barlow and Durand assume that Marlatt can be trusted. Unfortunately for science and the common good, he cannot. Chapter 8 in Maltzman (2000) is devoted to a critical analysis of the numerous errors of omission and commission in the Marlatt *et al*. (1993) paper.

If Barlow and Durand (2005) had read Pendery *et al*. (1982), they would have seen Table 2 (p. 172), which provides excerpts from hospital records showing that the majority of patients receiving controlled drinking training were rehospitalized for alcoholism treatment within approximately 1 year following treatment by the Sobells. These documented findings contradict the Sobells' published results obtained from purported interview follow-ups for that year.

Reading my book on alcoholism (Maltzman, 2000) or the original paper in question (Pendery *et al*., 1982) would have informed Barlow and Durand (2005) that many of the patients were interviewed 3 years after treatment. Table 3 in Pendery *et al*. (1982, p. 173) has the following heading: "Current findings regarding third-year treatment outcomes of the six subjects ranked highest by Caddy *et al*. [1978] all of whom they reported as functioning well 100 percent of the days in that year".

Justification for not comparing the treatment outcomes of the experimental and control groups is provided by Pendery *et al*. (1982). Not one critic has commented upon the justification or refuted the reasons provided for not comparing the outcomes between the two groups:

(1) There was a statistically significant order effect between the groups. The great majority of patients in the experimental group were treated and released prior to treatment of the control group, which was given an abstinence treatment goal.
(2) An additional bias was present in that control group patients had volunteered for the experiment believing that they would be trained to control their drinking. Instead, they were given an abstinence treatment goal. Because of the treatment order effect, patients in the control group were aware of the considerable publicity the experimental group was receiving in the media extolling the success of the new treatment for alcoholics permitting controlled drinking, which had been denied to them.

All of this information and more is available elsewhere (Maltzman, 2000).

Another fabulist assertion is the following:

> Despite opposition, research on this approach has been conducted in the ensuing years (Marlatt *et al*., 1993), and the results seem to show that controlled drinking is at least as effective as abstinence, but that neither treatment is successful for 70% to 80% of patients over the long term — a rather bleak outlook for people with alcohol dependence problems. [p. 411]

It is noteworthy that there are no citations for this discouraging — and false — statement. See, for example, the results reported by Feuerlein

and Küfner (1989), Smith (1985, 1986), and the CATOR registry (Harrison *et al*., 1991) described in Chapter 3 and at length in Maltzman (2000). It is also possible to obtain successful treatment outcomes over the long term with aftercare (Humphreys *et al*., 1997).

Barlow and Durand (2005) repeat the misinformation found in the first (1995) and subsequent editions of their textbook. They cannot be excused for their lack of scholarship in treating an important problem such as alcoholism. This serious social problem may affect many of the undergraduate readers of this encyclopedic text who will believe the inaccurate information found therein. If Barlow and Durand are writing about important problems such as alcoholism and its treatment, it is their ethical responsibility to be current and accurate. They have failed in their responsibility. They are also guilty of the eighth deadly sin: they cite studies [e.g. Pendery *et al*. (1982)] that they did not read or, if they did read it, intentionally misrepresent.

Barlow and Durand (2005) never mention the paper by Maltzman (1989), which presents evidence that the results reported by the Sobells are fraudulent. Further evidence showing, I believe beyond reasonable doubt, that Sobell and Sobell (1978) knowingly fabricated their results and misrepresented their procedures and other aspects of their study are presented in Maltzman (2000), as well as a deconstruction of the two inept investigations and the nonexistence of the third — a congressional investigation. Barlow and Durand continue to perpetuate the big lie that the Sobells' (1972, 1973a, 1973b, 1976, 1978) results demonstrating the effectiveness of controlled drinking training of alcoholics are valid, despite overwhelming evidence to the contrary.

Barlow and Durand (2005) continue, "It seems that about 20% of people with severe alcohol dependence have a spontaneous remission and do not reexperience problems with drinking (Ludwig, 1985; Vaillant, 1983)" (p. 388). Citing Ludwig (1985) in support of the latter assertion is inappropriate. Ludwig advertised for people who had been able to quit, and interviewed them to determine the "cognitive processes" involved in such an experience. There was no comparison group to determine a percent spontaneous recovery. Vaillant (1983) provides many different analyses. It is difficult to determine to which the citation refers and the severity of dependence in people displaying spontaneous

remission. What is apparent is that spontaneous remission is common to infectious and noninfectious diseases, is not peculiar to alcohol dependence, and is not contrary to the categorization of alcoholism as a disease. Weisner *et al*. (2003) report the results of a general population probability sample of 111 untreated alcohol-dependent people living in Northern California and a comparison group of 371 alcohol-dependent individuals living in the same region who had received treatment. One year later, 12% of the nontreated group showed spontaneous remission in comparison to 57% who were in remission following treatment in public and private facilities. Treatment does work and may produce significantly greater remissions than a nontreated group. A percentage of people do show natural remission from alcoholism, as is true of tuberculosis, cancer, and other diseases, as discussed in Chapter 1.

Causes of Alcoholism

Cognitive factors are considered at length in the section on causes of alcoholism and other drugs. According to Barlow and Durand (2005),

> What people expect to experience when they use drugs influences how they react to them. . . . A person who expects to be less inhibited when she drinks alcohol will act less inhibited whether she actually drinks alcohol or a placebo that she thinks is alcohol (M.L. Cooper, Russell, Skinner, Frone, & Mudar, 1992; Wilson, 1987). This observation about the influence of how we think about drug use has been labeled an *expectancy effect* and has received considerable research attention. [p. 404]

Cooper *et al*. (1992) is an inappropriate citation in the above quotation since they conducted a panel population study, not an experiment providing alcohol or a placebo. Barlow and Durand (2005) give a cognitive interpretation of urges — craving is created via powerful expectations. Expectancy is a causal factor. They never stop to wonder what causes an expectancy. Answers on paper-and-pencil questionnaires have been reified. Shortcomings and contradictions evident in the expectancy formulation are discussed at length in Chapter 4; they will not be repeated here.

I will ask one obvious question of the reader who is an adherent of expectancy formulations: how does an opioid antagonistic drug such as naltrexone reduce craving if it is all an expectancy in the "mind"? Barlow and Durand's discussion of "alcohol myopia" is similarly shortsighted (forgive the pun) in that they simply relabel a phenomenon and write as though it is now explained. An explanation requires the deduction of the phenomenon from more general principles. Such an explanation is available and testable: "alcohol myopia" is a function of damage to the ventromedial prefrontal cortex as demonstrated by Bechara and Damasio (2002) as well as other colleagues (2001, 2002), and as described in Chapter 1.

Treatment

In keeping with their selective use of untrustworthy secondary sources, Barlow and Durand (2005) state,

> Inpatient care can be extremely expensive. . . . The question arises, then, as to how effective this type of care is compared to outpatient therapy that can cost 90% less. Research suggests that there may be no difference between intensive residential setting programs and quality outpatient care in the outcomes for alcoholic patients (W. R. Miller & Hester, 1986). . . . Although some people do improve as inpatients, they may not need this expensive care. [p. 409]

The above conclusion assumes that inpatients and outpatients are equal in all respects, and that the treatment facilities do not differ except for inpatient or outpatient service. These assumptions are false, as demonstrated by Harrison *et al*. (1991) and as described in Maltzman (2000). Inpatients usually have a poorer prognosis than outpatients; have greater severity of dependence, less social support, greater polydrug use and comorbidity; are suicidal; etc. Inpatient and outpatient care thus cannot be directly compared in terms of cost-effectiveness or outcome when the participants differ so markedly. Whether an individual needs inpatient or outpatient treatment should be determined on the basis of need, accessibility, and safety of the individual in question and their

families. McLellan *et al*. (1993) describe how the effectiveness of inpatient and outpatient facilities varies as a function of the intensity and duration of services provided. The complexity of the problem and efforts to determine the cost-effectiveness of inpatient and outpatient treatment are far greater than Barlow and Durand appear to imagine (Walsh *et al*., 1991, 1992). Their simplistic approach to the problem does not inform students and does not stimulate critical thinking.

Barlow and Durand (2005) also address the effectiveness of AA:

> Without question, the most popular model for the treatment of substance abuse is a variation of the Twelve-Step program first developed by Alcoholics Anonymous (AA). . . . [T]he foundation of AA is the notion that alcoholism is a disease and that alcoholics must acknowledge their addiction to alcohol and its destructive power over them. . . . An important component is the social support it provides through group meetings. . . . Because participants attend meetings anonymously and only when they feel the need to, conducting systematic research on its effectiveness has been unusually difficult. . . . There have been numerous attempts, however, to evaluate AA's effect on alcoholism. . . . Although there are not enough data to show what percentage of people abstain from using alcohol as a result of participating in AA, Emrick and his colleagues found that those people who regularly participate in AA activities and follow its guidelines carefully are more likely to have a positive outcome. Other more recent studies suggest that persons who fully participate in AA do as well as those receiving cognitive-behavioral treatments (Ouimette, Finney, & Moos, 1997). On the other hand, a very large number of people who initially contact AA for their drinking problems seem to drop out, 50% after 4 months, and 75% after 12 months (Alcoholics Anonymous, 1990). AA is clearly an effective treatment for *some* people with alcohol dependence. We do not yet know, however, who is likely to succeed and who is likely to fail in AA. Other treatments are needed for the large numbers of people who do not respond to AA's approach. [p. 411f]

The above comments contain a number of misinterpretations and errors of fact. AA does not consider itself a treatment and is not founded

on the disease conception of alcoholism. According to Kurtz (2002), a student of the history of AA and the disease concept, "Contrary to common opinion, Alcoholics Anonymous neither originated nor promulgated what has come to be called the disease concept of alcoholism" (p. 6). Barlow and Durand (2005) also misrepresent the results of the study of patients in VA hospitals (Ouimette *et al*., 1997). Patients in VA hospitals receiving a 12-step–oriented treatment were significantly better on the most important measure of alcoholism treatment outcomes, abstinence. Patients treated in 12-step–oriented VA programs had a significantly better outcome at a 1-year follow-up in terms of abstinence from alcohol and all other drugs, 45%, versus 36% for cognitive behavior therapy (CBT) treatments and 40% for an eclectic blend of the two treatments (Moos *et al*., 1999). Moos *et al*. did not compare AA participation with CBT, but rather compared VA programs that did or did not integrate treatment and 12-step principles; continued participation in AA as a form of aftercare was not examined.

Contrary to Barlow and Durand's remarks concerning dropout rates from AA, Laudet's (2003) research indicates that attrition and decreases in participation rates are not peculiar to AA. They are related to mundane problems such as motivation or readiness to change, convenience of facilities, and problems of scheduling — problems common to participation in all treatment programs. Finally, Barlow and Durand's (2005) assertion that "the foundation of AA is the notion that alcoholism is a disease" is clearly falsified by a scholarly reading of the AA literature (Kurtz, 2002). Kelley (2003) provides a review of the extensive research on self-help groups, including the many subpopulations helped by participation in AA and other self-help groups.

Project MATCH

Barlow and Durand (2005) provide a perfunctory and misleading presentation of Project MATCH (Matching Alcoholism Treatments to Client Heterogeneity), its origins, and its results. They state,

> This type of treatment matching has received increased attention from workers in the area of substance abuse. For example, the National

> Institute on Alcohol Abuse and Alcoholism initiated Project MATCH (Matching Alcoholism Treatment to Client Heterogeneity) to assess whether people with different characteristics (having little hope for improvement versus searching for spiritual meaning) would respond better or worse to different treatments. . . . Initial reports suggest that well-run programs of various types can be effective with a range of people with substance use problems (Project MATCH Research Group, 1997). Although no exact matches are yet recommended, research is ongoing to help clinicians tailor their treatments to the particular needs for their clients. . . . By identifying the factors that support a person's substance abuse and treating them in an integrated fashion, clinicians may improve the success rates of the various approaches we have discussed. [p. 411]

Neither the Minnesota Model, the most widely used treatment approach, nor the Schick Shadel unique kind of aversion conditioning program are mentioned, despite their successful treatment outcomes described in Chapter 2. Barlow and Durand (2005) do mention covert desensitization treatment, but no evaluations of its treatment outcomes are provided. Barlow and Durand also describe relapse prevention and the abstinence violation effect (AVE) hypothesis underlying relapse prevention, as espoused by Marlatt and colleagues (Cummings *et al*., 1980). They fail to cite the results of studies contradicting Marlatt's formulation (e.g. Hall *et al*., 1990, 1991).

As stated in the *APA Ethics Code 6.3. Fairness in Teaching*, "When engaged in teaching or training psychologists present psychological information accurately and with a reasonable degree of objectivity" (APA, 2002, p. 1068). Barlow and Durand (2005) fail to satisfy this ethical principle in their discussion of alcoholism and its etiology, characteristics, and treatment.

Butcher, Mineka, and Hooley (2004)

Butcher, Mineka, and Hooley's (2004) book is the 12th edition of an abnormal psychology textbook. After a thoughtful survey of drinking problems, including binge drinking on college campuses, Butcher *et al*.

discuss risk factors and treatments including medications, Antabuse, and a section on "Psychological Treatment Approaches". Characteristics of AA are reviewed, followed by group therapy, environmental interventions, and a lengthy section on "Behavioral and Cognitive Behavioral Therapy".

There are several kinds of behavioral therapies, including aversive conditioning designed to suppress drinking. Butcher *et al*. (2004) write,

> For example, the ingestion of alcohol might be paired with an electric shock or a drug that produces nausea. A variety of pharmacological and other deterrent measures can be used in behavioral therapy after detoxification. One approach involves an intramuscular injection of emetine hydrochloride, an emetic. Before experiencing the nausea that results from the injection, a patient is given alcohol, so that the sight, smell, and taste of the beverage become associated with severe retching and vomiting. That is, a conditioned aversion to the taste and smell of alcohol develops. With repetition, this classical conditioning procedure acts as a strong deterrent to further drinking — probably in part because it adds an immediate and unpleasant physiological consequence to the more general socially aversive consequences of excessive drinking. [p. 399]

The above is a misleading presentation of the Schick Shadel hospital treatment procedure. It uses emetine for chemical aversion conditioning or electric shock for older patients or those with medical complications. Its faradic shock treatment is not simple classical conditioning, but a choice situation in which an array of beverages are available and the patient may avoid the noxious stimulus by choosing a nonalcoholic beverage from among the array. In addition, patients receive standard forms of counseling: family and group therapy as well pentothal therapy. It is a 10-day program with two follow-up sessions, and is based upon a disease conception of alcoholism. It has the highest success rate in the alcoholism treatment field (see Maltzman, 2000), but its results are never cited by cognitive behavior therapists. Is it because Shick Shadel does not employ CBT?

Butcher *et al*. (2004) as well as other textbook writers fail to distinguish between established treatment programs available in the

community and small efficacy experiments using volunteer participants, some paid for their services, who are selected after applying numerous exclusionary criteria. Such efficacy studies lack external validity. Butcher *et al*. state,

> One of the most effective contemporary procedures for treating alcohol abusers has been the cognitive behavioral approach recommended by Alan Marlatt (1985) and Marlatt, Baer, and colleagues (1998). This approach combines cognitive-behavioral strategies of intervention with social-learning theory and modeling of behavior. The approach, often referred to as a skills training procedure, is usually aimed at younger problem drinkers. . . . Self-control training techniques (Miller, Brown, *et al*., 1995), in which the goal of therapy is to get alcoholics to reduce alcohol intake without necessarily abstaining altogether, have a great deal of appeal for some drinkers. . . . [p. 400]

Obviously, the fact that an approach appeals to drinkers does not mean that it is effective. This segues into the next controversial topic: "Controlled Drinking versus Abstinence". According to some investigators,

> [S]ome problem drinkers need not give up drinking altogether but, rather, can learn to drink moderately, (Miller, Walters, & Bennett, 2001; Sobell & Sobell, 1995). Several approaches to learning controlled drinking have been attempted . . . and research has suggested that some alcoholics can learn to control their alcohol intake. . . . Miller and colleagues (1986) evaluated the results of four long-term follow-up studies of controlled drinking treatment programs. Although they found a clear trend of increased numbers of abstainers and relapsed cases at long-term follow-up, they also found that a consistent percentage (15 percent) of subjects across the four studies controlled their drinking. The researchers concluded that controlled drinking was more likely to be successsful in persons with less severe alcohol problems. The finding that some individuals are able to maintain some control over their drinking after treatment (without remaining totally abstinent) was also reported by Polich, Armor, and Braiker (1981).

> These researchers found that 18 percent of the alcoholics they studied had reportedly been able to drink socially without problems during the 6-month follow-up of treatment. [Butcher *et al*., 2004, p. 400]

The above passage has several errors of omission and commission all in one direction: supporting the skin-deep cognitivist view of the alcoholism treatment field. The Miller and colleagues study referred to above is the Miller *et al*. (1992) long-term follow-up of participants in the series of small efficacy studies conducted by Miller and his colleagues, as described in Chapter 3. The rate of 15% of participants who controlled their drinking is not significantly different from the spontaneous remission rate for such people. It must also be reiterated that participants were initially selected as problem drinkers not meeting the criteria for alcohol dependence; at long-term follow-up, an appreciable number had become dependent. Would they have done so if they had received traditional abstinence-oriented treatment? Butcher *et al*. (2004) neither ask such questions themselves nor stimulate their student readers to ask such questions.

The resurrection of the RAND Report (Polich *et al*., 1981) is another embarrassing and harmful lapse of scholarship. Butcher *et al*. (2004) fail to mention the bias produced by the large attrition rate and the criterion for "social drinking" used in the RAND Report: not more than an average of 6 drinks/day nor more than 10 on a typical day with no more than three symptoms of dependency. This sort of drinking would prevent recovery from brain damage and exacerbate existing damage in the majority of alcoholics. Butcher *et al*. ought to read the RAND Report before describing its results. In the meantime, readers of their textbook must suffer the unfortunate consequences of being misinformed about this important area of alcohol treatment research.

Comer (2004)

Comer (2004) is the fifth edition of an abnormal psychology textbook. Do important new research and theories appear so often in psychology that a new edition must be published every 3 years? Of course not. A new edition appearing every 3 years ensures continuing profits to the

publisher and royalties to the author. Adding insult to injury for the money-strapped undergraduate student, the price of the book is in the US$90 range, inflated by unnecessary artwork on the cover and expensive colored pictures and designs throughout the text, as is characteristic of the other textbooks reviewed.

Quality coverage of alcoholism and other drug use does not compensate for the price. On the contrary, the discussion of alcoholism treatment contains ideologically driven inventions and reliance on selected biased secondary sources that misrepresent the literature and misinform readers. Comer (2004) confuses small efficacy studies with standing treatment programs available to people in need. As is true of the other texts reviewed, there is no consideration of the effects of alcohol on the brain, that drinking even as little as one drink/day may prevent recovery of a neuropsychological deficit (Wilkinson & Sanchez-Craig, 1981). There is no mention of Minnesota Model treatment programs, much less outcome evaluations from the Comprehensive Assessment and Treatment Outcome Research (CATOR) registry.

In a section labeled "Behavioral Therapies", Comer (2004) states,

> A widely used behavioral treatment for substance-related disorders is aversion therapy, an approach based on the principles of classical conditioning. . . . In one version of this therapy, drinking behavior is paired with drug-induced nausea and vomiting (Owen-Howard, 2001; Welsh & Liberto, 2001). [p. 388]

The latter two citations are missing from Comer's References. The assertion that aversion therapy is widely used is false. Simple electric shock aversive conditioning was discarded by behavior therapists years ago because of its lack of efficacy (Hester & Miller, 1995; Wilson, 1978). It was used in a variety of small experiments not in standing treatment facilities. In contrast, discriminative aversive conditioning is used in a highly successful manner at Schick Shadel hospitals. As previously mentioned, it involves a 10-day inpatient treatment program with family counseling, interviews under sodium penthothal, follow-up treatments, and aftercare; and is described in Chapter 2 and Maltzman (2000). Use of these kinds of conditioning is limited to Schick Shadel hospitals. Their

treatment program for alcoholics has the highest abstinence rate of any treatment program with which I am familiar; however, it is not discussed by Comer or any other textbook of abnormal psychology that I have reviewed.

Cognitive behavioral therapy (CBT) is briefly and favorably reviewed by Comer (2004): "Approximately 70 percent of the people who complete this training apparently show some improvement, particularly those who are young and not physically dependent on alcohol (Walters, 2000; Hester, 1995)" (p. 389). These inflated figures refer to small efficacy studies lacking external validity, not standing treatment programs. Once again, the reader is being misled. A section titled "Controlled Drug Use vs. Abstinence" follows:

> Some cognitive-behavioral theorists believe that people can continue to drink in moderation if they learn to set appropriate drinking limits. They argue that demanding strict abstinence of people may in fact cause them to lose self-control entirely if they have a single drink (Marlatt *et al*., 1982; Peele, 1989; Heather *et al*., 1982). In contrast, those who view alcoholism as a disease take the AA position of 'Once an alcoholic, always an alcoholic,' and argue that people with alcoholism are in fact more likely to relapse when they believe that they can safely take one drink (Pendery *et al*., 1982). This misguided belief, they hold, will sooner or later open the door to alcohol once again and lead back to uncontrollable drinking.
>
> Feelings run so strongly that the people on one side have at times challenged the motives and honesty of those on the other (Sobell & Sobell, 1984, 1973; Pendery *et al*., 1982). Research, indicates, however, that both controlled drinking and abstinence may be useful treatment goals depending on the individual's personality and on the nature of the particular drinking problem. . . . [Comer, p. 390]

The passages above have several serious errors of omission and commission. The generalization that there is an improvement rate of 70% rests on citations to Walters (2000) and Hester (1995). Walters conducted a meta-analysis of alcoholics and problem drinkers. I had written a reply to Walters' paper showing that it is "garbage in, garbage

out". His meta-analysis is invalid because the studies analyzed were poorly conducted or, in the case of Sobell and Sobell (1973a, 1973b) and Caddy *et al*. (1978), do not belong in the science canon. Other studies such as Foy *et al*. (1984) and Rychtarik *et al*. (1987) were misinterpreted, as was the study by Stimmel *et al*. (1983). I presented the evidence for the above statements and more in a reply to Walters. My manuscript was rejected. I was instructed to conduct my own meta-analysis, a suggestion that misses the point: there is an insufficient number of carefully conducted studies with external validity comparing treatments for abstinence vs. controlled drinking goals to permit an adequate meta-analysis.

Citing Pendery *et al*. (1982) in connection with the above statement, "those who view alcoholism as a disease . . . and argue that people with alcoholism are in fact more likely to relapse when they believe that they can safely take one drink" is a gross misrepresentation. Pendery *et al*. make no such statements or allusions. Their paper is a descriptive study presenting the results of follow-up interviews of the Sobells' patients and excerpts of their medical records, which describe rehospitalizations for alcoholism within 1 year after their purportedly successful treatment. Pendery *et al*. make no reference to the disease conception of alcoholism or loss of control.

Another mendacious assertion follows: "Feelings run so strongly that the people on one side have at times challenged the motives and honesty of those on the other (Sobell & Sobell, 1984, 1973; Pendery *et al*., 1982)." Pendery *et al*. (1982) make no statement about the motives or honesty of the Sobells. Our manuscript probably received the most careful review of any ever accepted by *Science*. The process is described in detail in Maltzman (2000), together with the evidence supporting my charge that the Sobells fabricated their results and intentionally misrepresented their procedures. Some of the evidence is also presented in Maltzman (1989). Comer's (2004) insinuation concerning my motives for attacking the Sobells is demeaning and false. I became involved in the Sobell controversy because of my investigation of possible fraud; my knowledge of alcoholism and the disease conception of alcoholism came later.

My reply to the Sobells' defense of their 1973 papers (Sobell & Sobell, 1984) was rejected by S. J. Rachman, editor of *Behaviour Research and Therapy*, as libelous. Before submitting my manuscript to Rachman, it was reviewed by an attorney for the University of California who was familiar with the issues. It must be remembered that, in the United States, the defense against libel is the truth. I have never been sued for libel as a consequence of my statements concerning fraud.

Davison, Neale, and Kring (2004)

Alcoholism — its etiology, description, and treatment — receives a relatively brief treatment in Davison *et al*. (2004), with a variety of errors of omission and commission, all in one direction: supporting a brainless cognitivism.

Therapy for Alcohol Abuse and Dependence

Traditional hospital treatment is mentioned: "Public and private hospitals worldwide have for many years provided retreats for alcohol abusers, sanctums where individuals can dry out and avail themselves of a variety of individual and group therapies" (Davison *et al*., 2004, p. 389). However, details of the treatment, the nature of the Minnesota Model program, as well as treatment outcomes from CATOR, Hazelden, and McLellan *et al*.'s (1993) studies are not discussed.

Davison *et al*. (2004) assert,

> The number of for-profit hospitals treating alcohol abuse has increased dramatically over the past thirty years, in part because such treatment is covered in large measure by both private insurance companies and the federal government. . . . Annual costs run in the billions. Because inpatient treatment is much more expensive than outpatient treatment, its cost-effectiveness has been questioned. Is it worth the expense? Apparently not, at least in many cases. The therapeutic results of hospital treatment are not superior to those of outpatient treatment. . . . [p. 289]

Citations purporting to support this assertion are not in the Davison *et al*. (2004) References or readily available. We have previously noted in our discussion of Barlow and Durand (2005) that, to compare inpatient and outpatient treatments, the patients must be similar and the duration and intensity of the programs must be equal. Davison *et al*. concede,

> However, an analysis of treatment for alcohol dependence concludes that an inpatient approach is probably necessary for people with few sources of social support who are living in environments that encourage the abuse of alcohol, especially individuals with serious psychological problems in addition to their substance abuse (Finney & Moos, 1998). [p. 390]

Cognitive and Behavioral Treatments

Davison *et al*. (2004) turn to what they report are the most effective psychological treatments. They first consider aversion therapy, including covert sensitization, but no evidence for the effectiveness of the latter is provided. An extraordinary statement follows:

> Despite some evidence that aversion therapy may slightly enhance the effectiveness of inpatient treatment (Smith, Frawley & Polissar, 1991), some well-known behavior therapists discourage its use because it lacks empirical support and causes great discomfort (e.g., Wilson, 1991). Aversion therapy, if used at all, seems best implemented in the context of broadly based programs that attend to the patient's particular life circumstances, for example, marital conflict, social fears, and other factors often associated with problem drinking. . . . [Davison, *et al*., 2004, p. 393]

This is an extraordinary statement because Schick Shadel hospitals (Smith *et al*., 1991, 1997) have the best treatment outcomes that I have seen. Their results are described in Chapter 2. Aversion conditioning in a Schick Shadel hospital is conducted in a program including family and

individual counseling, etc. Furthermore, as previously noted, it is not simple classical conditioning, the kind of conditioning criticized by Wilson (1991). Davison *et al*. (2004) also fail to note that the CATOR database of Minnesota Model treatment programs with which Schick Shadel is compared has significantly superior average outcomes than the small efficacy studies lacking external validity promoted by Hester and Miller (2003). Results from Schick Shadel hospitals are significantly superior to the CATOR registry. How can Davison *et al*. conclude that Schick Shadel aversion therapy "may slightly enhance the effectiveness of inpatient treatment"?

Moderation in Drinking

Further ideologically driven misinformation is provided:

> Until recently it was generally believed that alcohol abusers had to abstain completely if they were to be cured, for they were assumed to have no control over imbibing once they had taken that first drink. Although this continues to be the belief of Alcoholics Anonymous, research mentioned earlier, indicating that drinkers' beliefs about themselves and alcohol may be as important as the physiological addiction to the drug itself . . ., has called this assumption into question. Considering the difficulty in society of avoiding alcohol altogether, it may even be preferable to teach the problem drinker, at least the person who does not abuse alcohol in an extreme fashion, to imbibe with moderation. A drinker's self-esteem will certainly benefit from being able to control a problem and from feeling in charge of his or her life.
>
> The term *controlled drinking* was introduced into the domain of alcohol treatment by the Sobells (Sobell & Sobell, 1993). It refers to a pattern of alcohol consumption that is moderate, avoiding the extremes of total abstinence and inebriation. Findings of one well-known treatment program suggested that at least some alcohol abusers can learn to control their drinking and improve other aspects of their lives as well (Sobell & Sobell, 1976). [Davison *et al*., 2004, p. 393f]

Photographic portraits of Mark Sobell and Linda Sobell adorn the border of the page. Once more, an invalid efficacy experiment is promoted as a program. After a discussion of Marlatt and Gordon's (1985) conception of relapse prevention, absent the contradictory evidence, the Sobells' notion of guided self-change is presented. Davison *et al*. (2004) conclude,

> Whether abstinence or controlled drinking should be the goal of treatment is controversial. This issue pits influential forces, such as AA, that uphold abstinence as the only proper goal for problem drinkers against more recent researchers, such as the Sobells and those adopting their general approach, who have shown that moderation can work for many patients, including those with severe drinking problems. If the therapeutic means of achieving the goal of moderate drinking are available — and research strongly suggests that they are — then controlled drinking may be a more realistic goal even for an *addicted* [emphasis added] person. Controlled drinking is currently much more widely accepted in Canada and Europe than it is in the United States. [p. 395]

There is no mention of fraud charges made against the Sobells (1973a, 1973b) and Caddy *et al*. (1978). There is no reference to the evidence showing that the Sobells' rise to fame, the research that is the cornerstone of controlled drinking promoted by the authors, is built on quicksand. As is true of the other textbooks, there is no reference to brain damage caused by excessive alcohol consumption long before the appearance of liver disease and the Korsakoff syndrome. There is no definition of "problem drinking" or "addicted" person, and how these terms are related to the DSM-IV. This is thus not teaching students to think clearly. Finally, there is a confused and biased presentation of Project MATCH and its initial findings, omitting the results for abstinence.

Kendall and Hammen (1998)

Another popular abnormal psychology textbook has been written by Kendall and Hammen (1998), two well-known clinical psychologists, one

a colleague of mine at UCLA. Concerning the disease conception of alcoholism, they write,

> Although a high percentage of Americans think alcoholism is a disease, there is no universally accepted 'single-cause' for alcoholism (or any of the substance-use disorders) among scholars and scientists. A more modern perspective is the unitary disease model of addiction. This model holds that alcoholics differ from normal persons in terms of psychological disposition an "allergic" sensitivity to alcohol. . . . It maintains that these differences cause alcoholics during their drinking careers to experience psychological changes (or both) that are progressive and irreversible and that leads to (1) craving and (2) loss of control regarding alcohol. According to the model, alcoholism and other drug-use disorders can be remedied only by lifelong abstinence. This notion is widely used in the field and was perhaps best expressed in an influential book, *The Disease Concept of Alcoholism* (Jellinek, 1960). [p. 346]

Misconceptions, misrepresentations, and pure invention are expressed in the above passage. There is no universally accepted single cause for cancer or cardiovascular disease; does this mean that they are not diseases? Knowledge of etiology is not a prerequisite for classifying a condition as a disease (Cohen, 1961; Maltzman, 1994, 2000). There is no indication to the reader that there has been considerable discussion of the concept of disease generally and the disease concept of alcoholism by a variety of scholars and scientists including psychologists, medical doctors and philosophers, and historians of science, some of whom wear more than one hat (Cohen, 1961; Flavin & Morse, 1991; Fulford, 1989; Maltzman, 1989, 1994, 2000; Reznek, 1987). Instead of considering these sources, Kendall and Hammen (1998) fall back on the myth propagated by Marlatt (1979), Fingarette (1988), and others. They present an inaccurate description of the contents of Jellinek's book (1960). Jellinek (1960, p. 87) rejects the notion of alcoholism as an allergy, a notion apparently introduced by Bill Wilson, one of the founders of AA. Jellinek describes research contradicting the allergy hypothesis. He does

not argue that all alcoholics show progressive and irreversible damage; only certain ones, gamma alcoholics, show progressive changes.

Textbook writers attempting to present a rounded view of alcoholism or any other behavior problem must recognize and distinguish between what is lay knowledge, the teachings of AA, and the writings of scholars and scientists in the field of alcoholism studies. But first, textbook writers must acquire that knowledge themselves. It will not come from ideologically biased and inaccurate secondary sources (e.g. Fingarette, 1988; Hester & Miller; 1995, 2003; Marlatt, 1983; Marlatt *et al*., 1993; Miller & Caddy, 1977; Miller & Hester, 1986) or from sources that do not belong in the science canon (Sobell & Sobell, 1978, 1984, 1995). Nevertheless, this is the literature that these and other textbook writers of a cognitive behavioral persuasion seem to rely upon.

Kendall and Hammen (1998) continue:

> Is the unitary disease model of any worth? By providing a palatable explanation of addiction the model improved public awareness of the problem and cultivated optimism about its treatability. For example, it was during Jellinek's heyday that alcoholism was recognized as a medical condition open to treatment. Further, clinicians could argue that alcoholism is a disease because there is a fairly uniform and predictable set of features, course, prognosis and treatment. [p. 346]

The authors are confusing the scientific classification with its practical effects. The public awareness of alcoholism as a disease that is treatable was advanced primarily by Marty Mann (1950), the founder of the National Council on Alcoholism. She promoted the public health approach that alcoholics are sick, not sinners, and that there is hope for such people: Alcoholics Anonymous (AA).

Kendall and Hammen (1998) continue:

> The disease model of alcoholism has been criticized on many grounds, however (e.g., Caddy, 1978; Fingarette, 1988; Mello, 1975; Pattison, 1976). First, there are limited scientific foundations for many of the predicates of the disease model, such as the idea that alcohol *or other drug problems* [emphasis added] are progressive diseases (developing

> from the presymptomatic to the prodromal, the crucial, and the chronic phases). [p. 346]

I have dealt with this and other criticisms that follow at length elsewhere (Maltzman, 1994, 2000), in Chapter 2, and in the preceding examination of Barlow and Durand (2005). The criticisms are without foundation, based upon a lack of knowledge of the extensive relevant research literature and reliance on an untrustworthy secondary source such as Fingarette (1988). The numerous errors of commission and omission in the latter work have been discussed in Maltzman (2000). Phases in the development of alcohol dependence have been repeatedly confirmed. Furthermore, Jellinek and other investigators of alcoholism phases have studied phases in the development of alcohol dependence, not phases in other forms of drug dependence.

"Second, the model reduces the addict's accountability for the damage he or she causes and could subvert the addict's autonomy and will to change" (Kendall & Hammen, 1998, p. 346). No empirical evidence is provided to support the above assertions contradicted by the 12 steps of AA, an integral part of the Minnesota Model of alcoholism treatment, and 12-step facilitation treatment programs generally. Recognizing that one is suffering from the disease of alcoholism should no more subvert a person's autonomy and will to change than recognizing that one has the disease of diabetes or cancer.

Kendall and Hammen (1998) continue: "Third, some of the postulates of the model (such as that one drink leads inevitably to a full-blown relapse) function as self-fulfilling prophesies (if you believe that one drink will lead to a full relapse, then, after one drink, the prediction of a relapse is likely to be fulfilled)" (p. 346). This assertion echoes secondary, inaccurate sources promoting an ideological framework in agreement with the authors' cognitive behavior therapy (CBT) view. The following is what Jellinek (1960) has to say concerning the loss of control, a problem on which he has been repeatedly misrepresented (e.g. Marlatt *et al.*, 1993, 1973): "It should be mentioned at this time, however, that the loss of control does not emerge suddenly but rather progressively and that it does not occur inevitably as often as the gamma alcoholic takes a drink" (Jellinek, 1960, p. 42).

Kendall and Hammen (1998) continue:

> Critics further argue that the concept of addiction communicated to the public may prevent many drinkers and drug users who do not fit this stereotype from identifying their use patterns as problematic and thereby entering treatment. Finally, the disease model is inconsistent with the data that have suggested that controlled use is a viable goal for the treatment of some alcohol and drug users and that some addicts "recover" by natural processes or after minimal intervention. [p. 346]

A new myth promoted by CBT revisionists is that natural or spontaneous recovery from alcoholism indicates that it is not a disease. We have already addressed this problem and the claim that controlled drinking is a viable treatment goal for some alcoholics, but will repeat the gist of our comments. The assertion that natural remission from alcoholism means that it is not a disease is contradicted by the survival of the human race. Diptheria, pneumonia, influenza, scarlet fever, measles, mumps, and pulmonary tuberculosis (TB) are deadly diseases. As a child growing up before the development of antibiotics and vaccines, I and millions of others had natural recovery from these and other diseases. Pulmonary TB is the deadliest disease in human history. An estimated two million people, primarily in developing countries, are still dying from it each year. There are natural recoveries — spontaneous remissions — from all of the above diseases and cancers; however, this does not result in their removal from the list of life-threatening diseases or lead to the conclusion that vaccines and antibiotics need not be developed for their treatment and prevention.

Kendall and Hammen (1998) continue:

> What, then, is the status of the unitary disease model? It is prudent to acknowledge that a group of patients with alcohol- and other drug-use disorders can be described by the unitary disease model. Similarly, although abstinence is *clearly* [emphasis added] not a treatment goal for all patients, it is indicated sometimes and can be recommended for some persons. However, it is important to note that alcohol- and other drug-use disorders constitute a heterogeneous class of problems in

> living for which the unitary disease model is perhaps metaphorical: Although not literally a disease, substance-use disorders do have serious negative physical effects. [p. 346]

Nowhere in this text designed to teach students critical thinking do Kendall and Hammen (1998) explicate the meaning of the concept of disease, which they claim alcoholism is not. Once more, the authors reveal their lack of scholarship as well as ignorance of the extensive discussions of the nature of disease and the conditions needed to meet the criteria for classification of a condition as a disease. The question of what constitutes a disease, the conditions for classifying a condition as a disease, and whether or not alcohol dependence meets those conditions have been discussed in Chapter 2, along with rebuttals of the criticisms made by critics of the disease concept (Maltzman, 2000, Chapters 1 & 2).

Kendall and Hammen (1998) fail to distinguish between dependence and abuse in their discussion of alcohol use disorders. The disease conception as formulated by Jellinek was not made to cover all alcohol and drug-use disorders. Jellinek (1960) was explicit in his distinctions among different kinds of alcoholics. Failure to acknowledge his distinctions is inexcusable in a textbook or any other scholarly work attempting to review the alcoholism literature and inform its readers about its characteristics, etiology, treatment, and the contribution of Jellinek to the promotion of research on alcoholism.

Additional major failures of scholarship are that the authors do not consider the evidence that there is a continuum of neuropsychological deficits, ranging from social drinking to chronic alcoholism (Parsons, 1998; Tarter, 1975). Evidence suggests that the prefrontal cortex may be differentially sensitive to the continuity of damage (Brokate *et al*., 2003). Kendall and Hammen never mention the charge, much less the evidence, that Sobell and Sobell's (1978) study is fraudulent. Evidence is overwhelming that controlled drinking or moderation is not a viable treatment goal for individuals diagnosed as suffering from alcohol dependence (e.g. Vaillant, 2003). As we have noted, evidence of structural and functional brain damage along with neuropsychological deficits are highly probable in alcohol-dependent individuals. In the

absence of neuropsychological and/or neuroimaging evidence that the patient is not suffering from brain damage, it is unethical for Kendall and Hammen (1998) and the authors of other textbooks reviewed to promote controlled drinking as a treatment goal.

Nolen-Hoeksema (2004)

Nolen-Hoeksema (2004) has written another abnormal psychology textbook from a CBT orientation replete with errors of omission and commission. As is true of other textbooks considered in this chapter, Nolen-Hoeksema misrepresents Jellinek's statement of the disease conception of alcoholism, asserting that the disease model of alcoholism views alcoholism as an incurable disease like epilepsy. Although she grants that there is some evidence of genetic and other biological influences, she asserts that social and psychological forces clearly operate as risk factors for alcoholism. This straw man caricature ignores Jellinek's (1960) discussion concerning the risk factors for alcoholism. An entire chapter (II) is devoted to "Social, Cultural, and Economic Factors" influencing the development of alcoholism. It is obvious that Nolen-Hoeksema is guilty of the eighth deadly sin of scholarship: referencing material that she did not read.

Nolen-Hoeksema's (2004) theoretical interpretation of the origins of alcoholism is based upon the notion of expectancy and cognitive folk psychology. She states,

> Children and adolescents learn alcohol-related behaviors from the modeling of their parents and important others in their culture. . . . The children of parents who abuse alcohol by frequently getting drunk or driving while intoxicated learn that these are acceptable behaviors and are thus more likely to engage in them as well. . . . Thus, maladaptive patterns of alcohol use may be passed down through the males in a family through modeling. [p. 626]

This modeling formulation is contradicted by the classic adoption study by Goodwin and his colleagues in Denmark (Goodwin *et al.*,

1974). Siblings from families with an alcoholic father were separated within the first 6 weeks of birth. One child was adopted by a nonalcoholic nonblood relative, and the other was raised by the biological parents where the father was an alcoholic. Thus, one sibling had an alcoholic father as a role model, whereas the adopted sibling did not. Contrary to Nolen-Hoeksema's (2004) cognitive modeling theory, there was no significant difference in the adult alcoholism rate between siblings adopted by nonblood relative nonalcoholic families and those who stayed in the alcoholic family. Both had higher alcoholism rates than adoptees from nonalcoholic families.

Alcoholism Treatment

Nolen-Hoeksema (2004) is dismissive of AA. She incorrectly states that it is based on the disease conception of alcoholism, which claims that if the alcoholic takes one drink they will lose all control over alcohol. She incorrectly states that there are 23 000 AA chapters around the world. The 1999 AA membership survey reports that there are approximately 100 000 chapters around the world with a membership of more than two million.

There is no mention of the widely available Minnesota Model traditional treatment program combining current clinical treatment modules with an emphasis on the 12 steps, let alone a discussion of their treatment outcome rates in the CATOR registry (Harrison *et al*., 1991). There is neither any reference to the long-term follow-up studies demonstrating the effectiveness of AA as aftercare (Humphreys *et al*., 1997), nor a discussion of the brain damage that may accompany heavy drinking even before reaching the stage of alcohol dependence (particularly in young people such as college binge drinkers). These are major omissions contributing to a highly inaccurate view of alcoholism and its etiology, characteristics, and treatment.

Behavioral and cognitive treatments receive considerable but noncritical coverage, with repeated errors of omission and commission. Aversive classical conditioning is described under the heading of "Behavioral Treatments", and is inexcusably confused with the use of

disulfiram (Antabuse). Schuckit (2000) accurately describes the two procedures as follows:

> Motivating the patient toward abstinence might sometimes be enhanced through the use of drugs that make it difficult for him to return to drinking on the spur of the moment (e.g., disulfiram, as described in section . . .). Motivation is also helped through establishing a conditioned reflex that causes the smell or taste of an alcoholic beverage to precipitate nausea or vomiting (as described in section . . .). [p. 315]

Schuckit (2000) continues:

> Disulfiram is a traditional drug for the treatment of alcoholism, and it is usually given at a daily oral dose of 250 mg over an extended period of time, perhaps up to 1 year. . . . Although disulfiram does not decrease the 'drive' to drink, the hope is that the patient's knowledge of a possible severe physical reaction following drinking while on disulfiram will be associated with an improved recovery rate. . . . In the midst of a reaction [to disulfiram] the most frequent symptoms include facial flushing, palpitations and a rapid heart rate, difficulty breathing, a possibly serious drop in blood pressure, and nausea and vomiting. . . . [p. 319]

Schuckit (2000) describes aversion conditioning as follows:

> Behavioral approaches can also form a core resource in the treatment of alcoholism. The behavioral modification procedures are usually added to the regular education and counseling. . . . Most often, this treatment involves attempts to 'teach' the patient *not* to drink by coupling the sight, scent, or taste of alcohol with an unpleasant event, such as vomiting or receiving a mild electric shock to the skin. Chemical aversion treatments, aimed at inducing vomiting in the presence of alcohol, usually utilize such substances as emetine or apomorphine and are generally felt to be more effective than electrical aversion. These treatments are usually offered in hospitals that have special experience with them. . . . [p. 324]

Nolen-Hoeksema (2004) suggests that covert sensitization therapy is an alternative treatment, but gives no evidence to support its use. The cue exposure and response prevention approach is also described, but no treatment outcome data are provided. The naive reader is left with the erroneous impression that there are a variety of cognitive treatment programs available to the public as alternatives to 12-step–oriented treatment programs. The amount of misinformation in this section of Nolen-Hoeksema's textbook is stunning. There is more still to come.

Cognitive-Oriented Treatments

> Interventions based on the cognitive models of alcohol abuse and *dependency* [emphasis added] help clients identify those situations in which they are most likely to drink and lose control over their drinking and their expectations that alcohol will help them cope better with those situations (Marlatt *et al*., 1998). Therapists then work with clients to challenge these expectations by reviewing the negative effects of alcohol on their behavior. For example, a therapist may focus on a recent party at which a client was feeling anxious and thus began to drink heavily. The therapist might have the client recount the embarrassing and socially inappropriate behaviors he engaged in while intoxicated, to challenge the notion that the alcohol helped him cope effectively with his party anxiety. . . . [Nolen-Hoeksema, 2004, p. 631]

Throughout the discussion, patients (including those diagnosed as suffering from alcohol dependence) are treated as rational people who simply have to be taught that their thinking is inappropriate, shortsighted, and maladaptive. This is the typical hyperrational cognitive approach to alcoholism treatment, even for people suffering from alcohol dependence. There is no recognition of the evidence indicating that people suffering from alcohol abuse and dependence are also probably suffering from structural and functional brain damage, which in many cases is the basis for their inability to plan or inhibit impulsive behavior including drinking and for their denial of a problem. Their neurobiological state must first be changed in order to produce a fundamental change in their

way of thinking. No evidence is provided by Nolen-Hoeksema or any other cognitive behavior therapist that an individual therapist rationally conversing with an alcoholic will bring about lasting fundamental changes in their way of thinking, social behavior, brain state, and alcohol consumption. Cognitive treatments are presented to the student in an uncritical fashion. No treatment outcome results are provided to support the procedures discussed as the preferred treatments for people suffering from alcohol dependence as well as preclinical heavy drinkers.

Nolen-Hoeksema (2004) next provides a stunning example of cognitive therapy with a severely dependent alcoholic. She states,

> The following is an excerpt from a discussion between a therapist and a client with alcohol-related problems in which the therapist is helping the client generate strategies for coping with the stress of a possible job promotion. The therapist encourages the client to brainstorm coping strategies, without evaluating them for the moment, so that the client feels free to generate as many possible strategies as he can. . . . (adapted from Sobell & Sobell, 1978, pp. 97–98). [An almost page-long purported exchange between patient and therapist then follows.]
>
> The therapist then helps the client evaluate the potential effectiveness of each option and anticipates any potential negative consequences of each action. . . . [p. 632]

I describe this example as stunning because the exchange is presented by Nolen-Hoeksema (2004) as authentic, when in fact the Sobells (1978) present it as a hypothetical exchange between therapist and patient. The Sobells present the hypothetical exchange to illustrate the manner in which a cognitive therapist functions at a state hospital treating gamma alcoholics.

Nolen-Hoeksema's (2004) fabulist presentation continues:

> In most cases, therapists using these cognitive-behavioral approaches encourage clients to abstain from alcohol, especially when clients have histories of frequent relapses into alcohol abuse. When clients' goals are to learn to drink socially and therapists believe clients have the capability to achieve these goals, then therapists may focus on teaching clients to engage in social or controlled drinking. [p. 632]

Nowhere does Nolen-Hoeksema (2004) indicate what the therapists' grounds are for believing that a client can control their drinking and so teach the patient these skills. Nowhere is there any mention of studies conducted on this question and of the ethical problems involved in allowing patients, who may have brain damage not assessed by the therapist, to make decisions about whether or not they may continue drinking.

Nolen-Hoeksema (2004) continues her misguided discourse in a section titled "The Controlled Drinking Controversy":

> The notion that some alcoholics can learn to engage in controlled, social drinking directly clashes with the idea that alcoholism is a biological disease and that, if an alcoholic takes even one sip of alcohol, he or she will lose all control and plunge back into full alcoholism. In 1973, researchers Mark and Linda Sobell published one of the first studies showing that a cognitive-behaviorally oriented controlled drinking program can work for alcoholics perhaps even better than a traditional abstinence program. They found that the alcoholics who had had their controlled drinking intervention were significantly less likely than alcoholics in the abstinence program to relapse into severe drinking, and they were significantly more likely to be functioning well over the two years following treatment.
>
> These findings were assailed by proponents of the alcohol-as-a-disease model. For example, Pendery, Maltzman, and West (1982) published a 10-year follow-up of the alcoholics in the Sobells' controlled drinking group in the journal *Science*, based on interviews with these alcoholics, their family members, and investigations of public records. Pendery and colleagues reported that 10 years after the Sobells' study 40 percent of the men in the controlled drinking treatment group were drinking excessively, 20 percent were dead from alcohol-related causes, 30 percent had given up attempts at controlled drinking in favor of becoming abstinent, and only 5 percent were engaging in controlled drinking. . . . [p. 632]

As previously discussed, Pendery *et al*. (1982) do not present alcoholism as a biological disease and do not refer to loss of control following "even one sip". Pendery *et al*. did not initiate their study 10 years after the Sobells' report; it was initiated 3 years later, but was delayed by

a lawsuit brought by the Sobells in an effort to prevent our independent follow-up (see Maltzman, 2000). As previously indicated, I knew nothing about the disease conception of alcoholism at the time of the Sobell's publication; I began to study alcoholism and the conception of disease in the history and philosophy of medicine *after* being attacked for promoting this conception with which I had no familiarity. When we began the study of the patients treated by the Sobells, I was still immersed in my psychophysiological research on the orienting reflex (e.g. Maltzman & Langdon, 1969; Maltzman & Mandell, 1968; Wingard & Maltzman, 1980).

Pendery *et al.*'s (1982) treatment outcome results are in stark contrast to the results reported after 2 years by the Sobells and the 3-year follow-up results by Caddy *et al.* (1978). Pendery *et al.* (1982) provide objective evidence, i.e. excerpts from hospital records, showing that the majority of patients trained to control their drinking relapsed and were rehospitalized for alcoholism within the first year after treatment (Table 2, p. 172). They could not have been functioning well 71% of the days covered by these hospital records. How could there be such a discrepancy between what the Sobells report as results of patient follow-up interviews and objective, independent medical records showing patient rehospitalizations for alcoholism treatment at Patton State Hospital (where the Sobell study was conducted), other state hospitals, and a Veterans Administration (VA) hospital? The number of hospitalizations is not at issue; rather, the reason for the hospitalizations is the issue prevaricated by the Sobells. Evidence was available as early as 1989 (Maltzman, 1989) that the treatment outcomes reported by the Sobells and by Caddy *et al.* (1978) were fraudulent. Nolen-Hoeksema (2004) joins the group of cognitivists guilty of the eighth deadly sin, citing a reference she did not read (Pendery *et al.*, 1982), and of not reading and citing later pertinent publications (Maltzman, 1989, 2000).

Nolen-Hoeksema (2004) states that there were multiple investigations of fraud charges, "including one by the U. S. Congress, interrupting their [the Sobells'] research for years. They were eventually cleared of any wrongdoing" (p. 632). Once again, Nolen-Hoeksema is in keeping with the ideological fashion sculpted by Marlatt and the Sobells. On the contrary, a photocopy of a letter from then-Senator Gore who

was Chair of the Senate Subcommittee on Investigations and Oversight, the committee that would conduct a congressional investigation of fraud if one had been undertaken, states that no such investigation occurred (Maltzman, 2000, p. 173). Investigations by the Dickens and Trachtenberg committees clearing the Sobells of wrongdoing are fundamentally flawed: both investigations uncovered evidence of fraud, but failed to appreciate the significance of their findings (Maltzman, 2000).

Nolen-Hoeksema (2004) concludes her chapter on the treatment of substance-related disorders with a lengthy discussion of Marlatt's method of secondary prevention for dealing with excessive drinking by college students. She provides no information on primary prevention as an approach to drinking on college campuses. These issues are addressed in Chapter 5, including the fundamental shortcomings of Marlatt's method of secondary prevention. It comes as no surprise that Nolen-Hoeksema (2004) acknowledges, among others, the assistance of Alan Marlatt (Preface, p. xix) in the preparation of her textbook.

Oltmanns and Emery (2004)

Oltmanns and Emery's (2004) book is the fourth edition of a textbook, a new edition appearing every 3 years. It does not mention allegations of fraud and the Sobell affair, but does indicate that abstinence vs. controlled drinking is a controversial problem.

Oltmanns and Emery (2004) devote considerable space to etiological risk factors, reviewing neurobiological and psychological risk factors along with an extensive and uncritical coverage of expectancy. The "Treatments" section describes AA, CBT, coping skills, and motivational interviewing. A sidebar contains a sketch of Marlatt highlighting his purportedly important contributions to the field. No negative experimental results or alternative interpretations of the balanced placebo experiment are provided, and none of the negative results found in studies testing Marlatt's relapse prevention model are cited. Minnesota Model treatment programs are not mentioned. Neither Project MATCH nor the comparative VA study (Ouimette *et al.*, 1997) reporting superior abstinence treatment outcome results for traditional Minnesota Model,

12-step–oriented programs as compared to behavioral self-control training (BSCT) programs is discussed.

Instead, a fabulist summary of treatment outcome results is offered attributed to Project MATCH (1997): "There is little if any evidence to suggest that one form of treatment (inpatient or outpatient, professional or self-help, individual or group) is more effective than another" (p. 421). Project MATCH could not compare outpatient and inpatient results because patients were not randomly assigned to these two arms. There were no comparisons of professional vs. self-help results because patients were not randomly assigned to one or the other such groups; the same holds for individual vs. group treatment. The purpose of Project MATCH was to study the matching of persons' characteristics to three different forms of treatment, not to compare different forms of treatment.

Raulin (2003)

Raulin (2003) provides an extensive coverage of alcoholism, emphasizing the importance of expectancy. A detailed description of the Marlatt *et al*. (1973) balanced placebo experiment and its results are provided. No reference is made to the studies failing to replicate the experimental results of Marlatt *et al*. or to the alternative demand interpretation of balanced placebo studies reviewed in Chapter 4.

The section on "Psychological Treatments" emphasizes behavioral treatments and "an approach unique to the treatment of alcohol abuse — controlled drinking" (Raulin, 2003, p. 464). The topic is introduced by an incomplete and inaccurate report of the classic study by Davies (1962). It is followed by the controlled drinking controversy and the purported baseless attack on the integrity of the Sobells. Raulin (2003) asserts,

> For decades, the popular wisdom among treatment professionals, especially those committed to the AA approach, was that the only reasonable goal for alcoholics was abstinence, because alcoholics could not control their drinking. Like many strongly held beliefs, this one seemed to be impervious to the influence of data. For example, Davies (1962) found that 7 out of 93 alcoholics in a long-term follow-up study

> showed a pattern of normal drinking. Granted, that is only 8% of his sample, but it is more than enough to refute the general proposition that *no* alcoholic is capable of controlled drinking. How well does controlled drinking work? [p. 465]

This question leads to an extensive discussion of controlled drinking that contains numerous errors of omission and commission. Before turning to this material, a correction to the evaluation of the Davies study is necessary. Raulin (2003) fails to report that Davies' patients were followed up by Griffith Edwards (1985), who found that at best one or two engaged in controlled drinking as evidenced by independent sources including hospital records. Patients misled Davies in describing their condition. Furthermore, examination of the patient records indicates that only one of the patients had experienced withdrawal symptoms, thus meeting the current criteria for alcohol dependence. Historically, the inadequacies of the Davies study were a signal that follow-up studies must obtain collateral information for each participant. None of these problems are addressed. Raulin continues:

> The controlled-drinking controversy came to a head following the publication of several articles describing a controlled-drinking treatment program developed by Mark and Linda Sobell . . . [it was not a program — it was a small efficacy experiment, something very different than a program]. Treating alcoholics to control their drinking was at least as effective, and in some ways more effective, than teaching them abstinence. Pendery *et al*. (1982) interviewed some of the participants in the Sobells' study several years later and came to startlingly different conclusions. They found that most participants in the controlled-drinking group of the Sobells' study 'failed from the outset to drink safely. The majority were rehospitalized for alcoholism treatment within a year after their discharge from the research project' (p.169). However, Pendery *et al*. did not interview the participants in the abstinence group, who also showed high rates of relapse. Nevertheless, these investigators went so far as to accuse the Sobells of scientific fraud (*New York Times*, June 28, 1982). Accusing a scientist of scientific fraud is *the* most serious charge

> that one can make, and it triggered a series of investigations by an independent blue-ribbon panel convened by the Addiction Research Foundation, where the research was conducted; the United States National Institutes of Health, which funded the research; and the United States Congress. These investigations completely exonerated the Sobells of scientific misconduct, noting that their conclusions were supported by their data and that their data were collected in a manner consistent with the best scientific methodology (Marlatt, 1983). . . . [p. 466]

Raulin (2003) has not read the primary sources; relies upon an untrustworthy secondary source, Marlatt (1983); and as a consequence commits numerous errors. The Sobells reported following up on the patients at Patton State Hospital, Riverside, CA, while living in the area. They subsequently obtained a position at the Addiction Research Foundation in Toronto, Canada, as a result of their widely publicized groundbreaking study challenging the received wisdom. The reported follow-up was conducted while Mark Sobell was a graduate student and Linda Sobell an undergraduate (see Maltzman, 2000). Pendery *et al*. (1982) did not accuse the Sobells of fraud in the *New York Times* article; I was the sole source of the statement. The institutional investigations conducted were fundamentally flawed; there was never a congressional investigation as discussed in Maltzman (1989) and in greater detail in Maltzman (2000), where I show the inadequacies of each of the investigations.

None of the investigations took the first essential step in an investigation of fraud: obtain all of the raw data and determine whether these data permit reconstruction of the results in the published report. Investigating committees did not report that the Sobells' conclusions were supported by their data and that "their data were collected in a manner consistent with the best scientific methodology." This is pure invention on the part of Marlatt (1983). A major failure of the investigations was that they did not examine the raw data in detail. The Sobells were criticized by the Dickens Committee for being careless in their procedure, failing to interview as frequently as they reported in their publications. Maltzman (2000, Chapters 4 & 5) provides a detailed examination

of the investigations, showing their gross inadequacies. As is true of the other textbook writers reviewed, Raulin is content with depending upon fellow ideologs such as Marlatt for a mendacious account of the Sobell affair.

Discussion

The textbooks reviewed here, and in all likelihood other cognitive-oriented abnormal psychology textbooks, demonize the disease conception of alcoholism, if they discuss it at all. Some of them misrepresent AA teachings as though they were dependent upon the disease conception of alcoholism, and misrepresent Jellinek (1960). The Pendery *et al*. (1982) study is said to have been conducted 10 years after the Sobells' treatment study; if authors had read Pendery *et al*., they would have seen that it had been conducted 3 years after treatment. In addition to the errors of commission noted in each of the textbooks, there are three inexcusable errors of omission:

1. None of the textbooks discuss the large and incontrovertible research literature demonstrating that excessive alcohol consumption may cause structural and functional brain damage long before liver disease and the Korsakoff syndrome (Oscar-Berman, 2000; Sullivan, 2000). Gender, age, nutrition, ethnicity, comorbidity, etc. may affect the risk of structural and functional brain damage. None of these important issues are considered.
2. Another serious error of omission related to the first is that none of the textbooks reviewed discusses the evidence clearly suggesting that controlled drinking may exacerbate or prevent recovery from existing brain damage and neuropsychological deficits caused by heavy drinking (Cala, 1987; Wilkinson & Sanchez-Craig, 1981). They all ignore human biobehavioral or behavioral neuroscience and neuroimaging research (e.g. Cala, 1987; Moselhy *et al*., 2001; Schuckit, 1998); research with infrahuman subjects (e.g. Higley *et al*., 1991, 1996a, 1996b; Wolfgramm & Heyne, 1995); and clinical and laboratory research showing that excessive alcohol consumption and withdrawal from alcohol cause

brain damage in the frontal lobes, hippocampus, and cerebellum (Glenn *et al*., 1988; Oscar-Berman, 2000; Paula-Barbosa *et al*., 1993; Sullivan, 2000).

3. None of the textbooks considered discusses the most common treatment program in the United States, the one in the community that a person in need of help may go to, aside from or in addition to the AA fellowship: the Minnesota Model of alcoholism treatment, which combines what is current in clinical psychology with an emphasis on the 12-step principles and AA meetings as aftercare. A well-known example is Hazelden, one of the origins of the Minnesota Model. There is no mention of the treatment outcome results found in CATOR (Harrison *et al*., 1991) or Hazelden (Stinchfield & Owen, 1998). Instead, textbook writers often call the small efficacy experiments they cite such as the Sobell and Sobell (1973) controlled drinking study, simple aversion conditioning, and behavioral self-control as "programs", falsely implying that there are established facilities in the community providing such treatments.

The textbooks reviewed above, and probably other textbooks of a CBT persuasion, are not educating their readers about alcoholism. They are misleading students, evading the truth, and failing to teach them to think critically. These textbook authors are endangering the public good. Their lack of scholarship is unethical, contrary to the canons of academic scholarship and the American Psychological Association's (APA) Ethical Principles. Instead, they follow a different rule: "If you tell a big enough lie and tell it frequently enough, it will be believed."

Textbooks for Alcoholism Counselors and Other Helping Professionals

Hester and Miller (2003)

The ethical requirement of scholarship is obvious in the case of textbooks written for counselors and other helping professionals in the alcoholism treatment field. Counselors who are directly responsible for the

well-being of their patients need accurate and current information, not ideology. A leading textbook in the field in the USA by Hester and Miller (2003) fails to reach this obvious ethical standard. Hester (2003) states,

> Mark and Linda Sobell conducted what was to become the most publicized evaluation of self-control training procedures (Sobell & Sobell, 1973). In a controlled evaluation with inpatient gamma alcoholics, they reported greater improvement in an experimental group receiving moderation training than in three comparison groups in abstinence-focused treatment. Pendery, Maltzman, and West (1982) questioned the successfulness of this treatment, reporting an independent review of the experimental cases. The controversy surrounding this study is complex (Marlatt, Larimer, Baer, & Quigley, 1993; Sobell & Sobell, 1984). A fair conclusion is that few of the alcoholics receiving experimental treatment sustained moderate drinking over an extended period, and they fared better than those receiving standard abstinence-oriented treatment. At the same time, both groups had rather poor outcomes overall. This conclusion is consistent with the findings of a subsequent study with similar inpatient population (Foy *et al*., 1984). [p. 159]

The above comments by Hester (2003) contain errors of omission and commission discussed in our review of abnormal psychology textbooks. No, it is not a fair statement of the Sobells' outcomes. It cannot be fair when the evidence presented elsewhere (Maltzman, 1989, 2000) shows, I believe beyond reasonable doubt, that the Sobells intentionally misrepresented their procedures and fabricated much of their results.

Foy *et al*. (1984) did not replicate the Sobells' purported results. Foy *et al*. conducted an add-on randomized controlled trial (RCT) with random assignment to an abstinence treatment in one condition and abstinence plus controlled drinking in the second condition. Five months following treatment, the abstinence plus controlled drinking group was significantly worse than the abstinence-only group. After 5–6 years, the two groups did not differ (Rychtarik *et al*., 1987). Since it was an add-on design, the results show that controlled drinking training had no effect in the long run. This inaccurate description of the Sobell and Sobell

(1973a) study and of the studies by Foy *et al*. (1984) and by Rychtarik *et al*. (1987) is not an anomaly in Hester and Miller (2003). It epitomizes the lack of scholarship and ideological bias found in their chapters in their textbook for alcoholism counselors.

Edwards et al. *(2003)*

A popular textbook for helping professionals in the United Kingdom written by Edwards *et al*. (2003) suffers from a variety of deficiencies reflecting the cognitive ideology adopted by its authors. The back cover of the book by Edwards *et al*. proclaims, "*The Treatment of Drinking Problems* has become . . . the definitive text in its field. Internationally acclaimed and translated into six languages, it is the most authoritative source book for the treatment of alcohol problems for all professionals who encounter them." It is a sorry state of affairs when such an acclaimed text promotes the brainless cognitivist approach to alcoholism treatment, adopts a cognitive self-selection (i.e. stepped-care) approach to treatment goals, and fails to consider brain dysfunction as a problem and abstinence as a recommended treatment goal long before the patient reaches the criterion of severe dependence (i.e. comorbidity, polydrug use, or liver disease).

According to Edwards *et al*. (2003), the caregiver and the patient should agree upon a drinking goal at the outset of treatment. Edwards *et al*. suggest 2 drinks/session with not more than 10 drinks/week. This is above the limit that Wilkinson and Sanchez-Craig (1981) report will prevent recovery from a neuropsychological deficit. Nowhere do Edwards *et al*. indicate that brain dysfunction is common in alcoholics long before Korsakoff's syndrome and cirrhosis of the liver are apparent. Nowhere in this text for the helping professions do the authors suggest that brain damage is likely to be present in patients who have not reached the stage of alcohol dependence. Nowhere do they mention the possibility that (1) the patient, because of brain damage, may not be capable of providing a proper informed consent; (2) detailed CBT requiring the planning and learning of new problem-solving skills may be seriously impaired; and (3) even moderate drinking may exacerbate

existing brain dysfunction and prevent recovery from neuropsychological deficits.

Edwards *et al*. (2003) describe criteria for whom controlled drinking is an inappropriate goal: patients with fully developed dependence, comorbidity, polydrug use, or organ damage (usually of the liver). These rules fail to recognize that brain damage does not suddenly appear when the individual meets the criteria for alcohol dependence. Brain damage is a gradual and continuously developing condition; brain damage accompanied by neuropsychological deficits are present before liver disease appears (Cala, 1987). There are individual differences in the susceptibility to damage and in the extent of recovery with abstinence. Only careful assessments of the brain structure and function of each patient can provide adequate information on whether or not the caregiver is inducing or reducing harm. This leading text in the United Kingdom by Edwards *et al*. (2003) as well as a leading text in the United States (Hester & Miller, 2003) are fundamentally flawed. They fail to meet a basic standard of caregiving and education: "Do no harm." The cognitivist authors of these texts have to get it through their heads: "It's the brain, stupid."

Conclusion

> When a community is directed to a single objective, its code of conduct becomes simpler and more severe than the easy-going code of compromises which does duty for most people. In daily life few of us are entirely rigid about white lies and tax-deductible expenses and other small evasions. We accept, even if we do not condone, such venial sins. But the professional morality of scientists allows no compromises. It tells each man that he must report what he believes to be true, exactly and without suppression or editing. Nowhere in a research journal is a scientist allowed to minimize an awkward discrepancy or to stress a comforting confirmation. Nowhere is he allowed to put what seems expedient in place of an unpalatable truth. A scientist takes it for granted that when another scientist reports a finding, he can be believed absolutely — by which we mean, that we can be certain that what the man reported is exactly what he thought he saw or heard, no less and no more.

> This absolute trust of each man in the word of every other man is remarkable in the society of scientists. Yet it is not by itself the whole of scientific morality. For a morality embraces not only the individual and his trust, but a whole community, and it therefore has to provide for all the subtle relations between the members of the community. The morality of science is subtle in this way, but it has grown from a simple principle — the principle that the community of scientists shall be so organized that nothing shall stand in the way of the emergence of the truth. . . . [Bronowski, 1977, pp. 199–200]

My conclusion is that the textbook writers I have reviewed and certain secondary sources they rely on are, according to the standard described by Bronowski (1977), immoral and have violated the *APA Ethics Code 6.3. Fairness in Teaching* (APA, 2002, p. 1068).

7

Sociology of Science and Alcoholism Studies

Bronowski (1972), Shapin (1994), and others have observed that science and the public good require trust and the truth upon which trust is based. Unfortunately, a lack of trust is warranted in the specialty field of alcohol studies due to mendacity on the part of leading cognitive behavior therapists in this area. Examples are Sobell and Sobell's (1973a, 1973b, 1976, 1978) falsification of the results and procedures of their study purporting to demonstrate controlled drinking by alcoholics; Miller's (1995a) hagiographic description of Marlatt *et al.*'s (1973) balanced placebo experiment omitting and/or misrepresenting contradictory evidence; Goldman *et al.*'s (1996b) failing to cite or describe Aarons' (1996) doctoral dissertation studies conducted in Goldman's laboratory under his supervision contradicting his expectancy hypothesis; and Marlatt *et al.*'s (1993) repeated errors of omission, commission, and misrepresentation in the controversy over controlled drinking as a treatment goal for alcoholics (Maltzman, 2000, Chapter 8).

What drives this disregard for the truth? Ideology, the desire for hegemony, greed, and hatred of a common foe — the disease conception of alcoholism — are responsible for revisionists' contempt for the truth. A lack of scruples is the added topping to this witches' brew. Revisionists' ideology is a skin-deep cognitivism leading to the root falsehood, "Alcoholism is nothing but a bad habit." Revisionists' cognitive approach to alcoholism is an ideology fueled by the bottom line — the

same kind of motivation that drove the fraud committed by unscrupulous executives at Enron as well as their banks and accounting firms, scientists at the National Institutes of Health (NIH) with stock options and fees from drug companies whose products they evaluate, and the falsification of stem cell lines. Science is big business (Greenberg, 2001). It involves the work of entrepreneurs, large and small; research institutes; science societies; and individual investigators who have well-funded grants supporting graduate students, postdoctoral fellows, and research assistants. A few even have government-supported research centers. Some are driven to a great extent by the need for success, status, power, influence, and greed, as in any other business or profession.

Ideology driven by greed and the lust for power varies among sciences and in specialized fields within a particular science. From personal experience, I am best prepared to write about psychological science. Alcohol studies as a specialization within clinical psychology is the area containing the investigators that I am characterizing.

The Eighth Deadly Sin

Literary folklore suggests that there are seven deadly sins: lust, avarice, gluttony, pride, anger, idleness, and envy. I submit an eighth, the sin in academia: lack of scholarship. It is manifest by revisionists in the field of alcoholism, for example, (1) citing references that have not been read (see e.g. Chapter 6); (2) promoting generalizations that have been falsified or contradicted by a body of evidence (e.g. the assertion that balanced placebo experiments demonstrate the effect of expectancy, or that what you think is more important than what you drink); (3) arguing that alcoholism is nothing more than a bad habit which obeys the same laws of learning as any other form of learning, and that alcoholism as a bad habit is nothing more than a lifestyle choice; and (4) claiming that Pendery *et al*. (1982) initiated their follow-up 10 years after the Sobells treated their patients, or that Pendery *et al*. did not compare the experimental and control groups.

The academic sin of failed scholarship is a derivative, making its appearance as a consequence of one or more of the first seven sins. The first seven deadly sins are primitives; each is irreducible to another. They

are not visible in scholarly writings. Manifestations of the eighth sin include lacking citations for assertions, avoiding contradictory evidence, and relying on the Orwellian principle that "ignorance is strength." Avarice, greed for money and power, anger at nonprofessionals and Alcoholics Anonymous (AA), envy of their wide acceptance, laziness, and not bothering to study the pertinent literature may all be undercurrents giving rise to the tidal wave of misinformation — the sin so evident in Miller and Hester's (2003) ostensibly disinterested reviews of the treatment outcome literature; Peele *et al.*'s (2000) failure to recognize that Davies' work is no longer recognized as valid because his interviewees had lied to him; Marlatt and colleagues' (1983, 1993) reviews replete with errors of omission, commission, and misrepresentation; and, finally, scientific misconduct in alcoholism studies (Sobell & Sobell, 1973a, 1973b, 1976, 1978) and the failure to adequately investigate it (Maltzman, 2000, Chapters 5–7).

In criticizing cognitive behavior therapists and other revisionists, I wish, again, to make clear that I am not opposed to cognitive behavior therapy (CBT) in all forms and applications. I do not believe that all cognitivists lack scientific integrity and cannot be trusted. I certainly am not including all psychologists in alcoholism studies in the same class as the revisionists. Psychologists have made important contributions to the field of alcoholism studies, for example, Oscar-Berman (Oscar-Berman *et al.*, 1991), Parsons (1998), and Tarter (1975). They happen to have made their contributions in the field of neuropsychology, the very field that revisionists ignore for ideological reasons. Other important contributions have been made by Moos and his colleagues (Humphreys *et al.*, 1997; Moos & Moos, 2003, 2004, 2005) as well as by nonpsychologists such as Bechara and Damasio (2002), Schuckit (1998), and in years gone by Begleiter and his colleagues (1984), Goodwin and his colleagues (1973), and Cloninger and his colleagues (1981) in their studies of heredity as a risk factor for alcoholism.

Who Is Watching over the Watchdogs?

One of the most serious obstacles to the pursuit of truth in the field of alcohol studies is posed by individuals with a special trust and

responsibility: the gatekeepers of science and scholarly journals, namely, editors and members of editorial boards as well as specialists within the subspecialty of alcoholism studies served by journals.

The editor, associate editors, members of the editorial board, and reviewers are all assumed to be capable of objective, scholarly reviews. They play a major role in determining what will appear in print and be read by their professional peers, students, and scholars in related areas and specialties. They help shape the direction of future research, training, and funding supporting these essential activities. It is therefore sobering to experience not scholarly virtues, but hostility, *ad hominem* attacks, obvious bias, and a lack of scholarship in reviews of my manuscripts. Lack of integrity on the part of reviewers and editors is not obviously driven by greed, power, or the bottom line. Except, perhaps, for editors of private, commercial journals, editors are not paid for their services; they take the time to provide reviews because of dedication to their profession and, as it appears in the case of alcoholism studies, to ensure hegemony of the party line. Ideology driven by affect rather than a monetary incentive determines a reviewer's comment of "that is ignorant" on the margin of my manuscript.

I will not beat a dead horse with a discussion and response to all of the inappropriate reviews my manuscripts have received. I will consider two of the more recent inappropriate reviews. One involves my correspondence with Griffith Edwards, dean of editors in the field of alcoholism studies. He has for many years been editor of the oldest journal in the English language dedicated to publishing in the field of addictions, *Addiction* (formerly the *British Journal of Addiction*). He retired as editor-in-chief on December 31, 2004, but continues his association with *Addiction* as a commission editor. A different kind of correspondence is with Mary Beth Kenkel, editor of an American Psychological Association publication, *Professional Psychology: Research and Practice*. She is not a specialist in alcoholism studies. Like editors of many general journals, she serves as an administrator, distributing manuscripts to associate editors knowledgable in the relevant specialty area who may distribute a manuscript to an expert on the editorial board, who in turn selects the manuscript reviewers.

My letters suggest a point that I have made before (Maltzman, 2000) and needs repeating: science and its institutions cannot regulate themselves. The Enron debacle is an example of the inability of big business to regulate itself. It is power and greed run rampant. Scientists are not free of the same motives. Coupled with the opportunity to profit financially, directly or indirectly, individuals lacking integrity and motivated by power and greed tend to rise to the top of the power echelon. When such people are united by a common ideology, a dogmatic fashion, they can do serious harm to science and the public at large. Freedom from such tyranny is only possible when there is dissent and a legitimate expression of criticism in professional journals. This is what keeps science on track in its search for the truth; this is what makes for a democratic society and a free and vigorous science. These are some of the ideas that Popper (1962) emphasized for half a century. Unfortunately, they may not be found in the specialty field of alcoholism studies within the CBT form of clinical psychology. Problems are most serious in the areas related to the application of alcoholism research and treatment. Areas in the public interest are directly affected by the lack of integrity of individual psychologists, administrators, investigators in professional and government institutions, and journal editors and reviewers.

A form of scientific misconduct, i.e. accepted coin of the realm, is evident in CBT quarters of alcoholism studies. We have seen this kind of corruption and misconduct exposed in big business, distinguished accounting firms, and the NIH. However, just as one cannot generalize from Arthur Andersen and Enron to all accounting firms and corporations that all of their executives are corrupt and guilty of misconduct, so too, one cannot generalize that all cognitive behavior therapists are guilty of scientific misconduct and lack integrity. Unfortunately, the guilty ones are highly visible in the field of alcohol studies, an area that has direct impact upon the well-being of the public. Misconduct in this field is especially abhorrent and contrary to the fundamental ethical codes of all science and its application: "Do no harm."

Manuscript rejections that will not be described in detail include my paper on the disease conception of alcoholism (Maltzman, 1994), finally published in a small circulation journal after it was rejected by Blane, an associate editor of the *Journal of Alcohol Studies* at the time.

The reviewers were all apparent revisionists. Each of their objections and criticisms was answered or shown to be inappropriate. Blane's response to my deconstruction of reviewers' criticisms was that he does not reconsider his judgments.

Andrasik, editor of *Behavior Therapy*, rejected my rebuttal of the reviewers' criticisms because the target of my manuscript's critical evaluation was Marlatt *et al*. (1993). Andrasik asserted that Marlatt *et al*.'s paper was a presidential address to the Association for the Advancement of Behavior Therapy, and that presidential addresses are not open to criticism. He did not inform me of this "rule" until after he submitted my paper to reviewers who had a variety of prevaricated criticisms, which he sent to me and which I disarmed. No matter, I criticized a presidential address, which is not acceptable.

The above brief examples may give the reader some idea of the problem of "who is watching over the watchdogs" of academic publishing, the journal editors. Letters to follow were selected from my collection of replies to nonscholarly rejections over the years because of the importance of the problems considered in the related manuscripts as well as the grounds and significance of their rejection.

Letters to Griffith Edwards

The first letter to follow was stimulated by two events. My book on alcoholism (Maltzman, 2000) was reviewed in only one professional journal, *Addiction*, edited by Griffith Edwards. The review was unusual, to say the least. It offered no description of the contents of the varied chapters in an almost 400-page book. Instead, Rehm (2000), the reviewer, characterized the book as a personal tirade and vendetta against the Sobells. I wrote a reply to the review providing a detailed response to Rehm's assertions. My reply was rejected. I asked for an explanation. Edwards offered none, other than his suggestion that I adopt an appropriate attitude toward life: "You win a few, you lose a few."

Not long afterwards, Edwards published an editorial asserting that professional journals must be more alert to authors' conflicts of interest. Manuscripts submitted for publication must recognize their sources of financial support, if any. Edwards' editorializing about the issue was

instigated by an incident in which two young investigators wrote a book on cigarette smoking analyzing all of the relevant research literature. They concluded that cigarette smoking is a serious health risk factor, but is not addictive and does not cause dependence because of its nicotine content; rather, cigarette smoking is addictive primarily because of its sensory stimulation of the throat. Frenk and Dar (2000), the authors of the book, failed to note that they had earlier received money from a law firm to review the cigarette smoking literature; apparently, the law firm also represented a cigarette company. As part of his editorial about the necessity of turning the camera on fraud in science, Edwards concluded that Frenk and Dar were unethical. They had a conflict of interest which was not divulged, a case of scientific misconduct.

My letter takes Edwards to task for selectively turning on the camera to catch fraud. He turns it on two young investigators for an arguably serious offense, not divulging a possible source of conflict of interest. I happen to know one of the coauthors who had been in my graduate school classes, Hannan Frenk. I also had Saul Shiffman, one of his critics, in my classes, as well as Jed Rose and Edward Levin who conducted the critical experiments described by Frenk and Dar (2000) showing that sensory stimulation of the throat produced by cigarette smoke is a critical variable in developing a dependence on cigarette smoking.

Edwards' editorial on turning on the camera to catch fraud and conflicts of interests, as well as letters in reply to his editorial, may be found in *Addiction* (2000, **97**, pp. 1–5). Edwards and his supporters ignore an important point: Frenk and Dar (2000) show that the studies purporting to demonstrate that nicotine is addictive are poorly designed experiments without adequate control groups; in contrast, evidence suggesting that sensory stimulation of the throat is a necessary condition for cigarette smoking dependence is based on well-designed experimental research (Levin *et al*., 1990; Rose & Hickman, 1987; Rose *et al*., 1993). Neither Edwards nor his supporters appear to have read Frenk and Dar's (2000) thoughtful book. I did. Frenk and Dar do not deny that cigarette smoking is a serious health risk; they deny that nicotine is the basis for dependence on cigarette smoking.

What follows are several of my preliminary letters followed by a long letter discussing the double standards employed by Edwards and the

arbitrary manner in which he decides to turn on the camera to catch instances of fraud. Edwards' insistence on footnotes to journal articles revealing the sources of funding and conflicts of interest seems to me to be a "paper tiger" in the fight against the lack of integrity in scientists. It reminds me of the situation when I joined the University of California, Los Angeles (UCLA) faculty in 1949. Because of the "red scare" (i.e. communism), we had to sign a loyalty oath pledging that we were not involved in political activities designed to overthrow the United States government. Does anyone now really think that if a potential faculty member were so inclined, they would refuse to sign the oath and therefore not be hired? How different is the requirement to provide a conflict of interest footnote? I wrote to Griffith Edwards, editor of *Addiction*, on July 31, 2001, as follows:

> The request to publish my reply to Rehm's review of my book is not a matter of egocentric philosophy. I ventured into alcohol studies more than 20 years ago, leaving my field of experimental psychology which had been my life-long interest, because I was concerned about the pursuit of truth in alcohology and the public good. It is time *Addiction* recognizes its responsibility towards these same goals.

I submitted a reply to Edwards' editorial, "No switching off the camera: how *Addiction* will respond to infringements of ethical publishing expectation", on January 2, 2002. It was quickly rejected. I wrote the following reply on February 28, 2002:

> Thank you for your letter of 28 January 2002. You stated that you gave thoughtful consideration to my letter which you rejected for publication in *Addiction* in response to your "switching off" editorial. It has been my experience, however, that respected science journals provide authors of rejected submissions the grounds for rejection. I would appreciate your adherence to this established principle by detailing for me the bases for your decision.

Edwards reply: it was rejected because there was nothing new in the letter. Of course there was nothing new in the letter — to him. He is

privy to my previous letters to him and to the free copy of my book the publisher sent to *Addiction* for review. The question is, is there anything new in my reply to his editorial for the general reader of *Addiction*? Of course there is. I now felt that there was no point in wasting my time replying to such an absurd explanation of the refusal to accept my critique of his editorial and the prior review of my book. Appendix 1 is the letter sent in reply to his "switching on the camera" editorial.

Discussion

How does one explain Edwards' editorial judgments? It is simple. He has a conflict of interest and little concern for the truth. As reported in Chapter 6, he is the senior coauthor of a skin-deep cognitivist textbook for helping professionals in the alcoholism treatment field. He writes approvingly of, and relies upon, the writings of Marlatt, Miller, and the Sobells. Does he have a footnote or foreword in the issues of *Addiction* he edits indicating that he has such a conflict of interest and is the senior coauthor of a textbook promoting a cognitivist interpretation and treatment for alcoholism? Of course not.

Letters to Kenkel

Appendix 2 is a cover letter and my response to the review of my manuscript, "Self-selection: harm reduction or induction", submitted to *Professional Psychology: Research and Practice*. The manuscript is now Chapter 5, with minor corrections and changes.

Discussion

Two editorial styles are apparent in *Addiction* and *Professional Psychology* (*PP*). Edwards, editor of *Addiction*, is authoritarian and rigid. Years ago, in dealing with my manuscript analyzing the Dickens Committee Report submitted to the *British Journal of Addiction*, he saw that home bases do not work in the case of investigations of fraud. However, this earlier experience had no effect upon his "camera" editorial, which emphasizes the use of home bases in investigating fraud (including conflicts of interest).

The irony is that involving the home base introduces a conflict of interest which he does not see.

Edwards appears to make editorial decisions without consulting associate editors. My manuscripts were not distributed to associate editors, who would have submitted them to reviewers providing Edwards with their evaluations. Whether this is his general practice, I do not know. I doubt that it is, but it certainly appears to be the case in relation to my "controversial" manuscripts. It is also a reasonable hypothesis that Rehm was selected to review my book (Maltzman, 2000) because his negative review was predictable. Any number of distinguished psychologists in the field of alcoholism treatment, therefore familiar with the issues and their history, could have been chosen to review my book. Instead, an epidemiologist, Rehm, with no track record in the alcoholism treatment area of specialty, was selected for the hatchet job.

Kenkel, editor of *PP*, acts as a high-level secretary distributing manuscripts to associate editors, who in turn submit the manuscript to reviewers. Kenkel echoes the opinions of the reviewers rather than making independent judgments. One obvious independent judgment is to reject *ad hominem* insulting, inappropriate, unscholarly comments such as "This is just ignorant." Kenkel insisted that my manuscript be greatly shortened. If I did this, it could be resubmitted for a new review. I refused because I saw little fat in it that could be eliminated without weakening the critique of the basic problem: harm reduction as practiced by Marlatt and others is a form of harm induction. The rejected manuscript is Chapter 5, except for minor grammatical changes as well as the deletion of a discussion of Sir Bradford Hill and the origin of RCTs, which was moved to Chapter 2.

Conclusion

Trust, and the truth upon which it is based, is fundamental to the well-being of science as well as corporate America and the government. In the past several years, we have seen evidence of a lack of integrity in each of these areas. In psychology, leading cognitive behavior therapists in the field of alcohol studies have repeatedly dissembled. For example, Marlatt (1979) consistently argues that alcoholism is simply a bad habit,

obeying the same principles of learning as any other habit: "All drinking behavior, from social drinking to alcohol abuse, is assumed to be governed by similar principles of learning and reinforcement. As such it is assumed that there is no crucial difference that distinguishes the social drinker and the problem drinker, other than the amount of alcohol consumed" (1979, p. 324f). However, Hodgson *et al*. (1979) show quite the contrary. A dose of alcohol in the morning had a satiating effect on moderate drinkers in the afternoon, decreasing their alcohol consumption; and an appetizing effect on heavy drinkers, increasing their alcohol consumption later in the day. Their drinking behavior was not on a continuum.

Marlatt *et al*. (1993) cite Pendery *et al*. (1982) as conducting a follow-up of the Sobells' controlled drinking study participants 10 years after their treatment was completed, and state that Pendery *et al*. failed to report results for the control group and failed to compare the experimental and control groups. Both statements have been widely cited in textbooks and articles in professional journals. They are false, as described in detail in Maltzman (2000) and in Chapter 6.

Miller *et al*. (1995) state,

> The negative correlation between scientific evidence and application in standard practice remains striking, and could hardly be larger if one intentionally constructed treatment programs from those approaches with the *least* evidence of efficacy. Such a gap between science and practice will not be reduced without some disciplined and demanding changes. Clinicians, like scientists, must be willing to test their cherished assumptions against hard data and to relinquish views and practices that do not stand up to the test of evidence. [p. 33]

Miller's statement is contradicted by material presented in a book that he coedited (Laundergan, 1993). Miller cites the RAND Report as a fine multisite research study that was unreasonably attacked when it first appeared. He does not describe the major methodological flaws in the study and the unacceptably high levels of alcohol consumption used to define controlled drinking — levels that would produce brain damage and neuropsychological deficits in the majority of participants (Cala,

1987; Eckardt *et al*., 1998). He is a fabulist and cannot be trusted. As described at length in Maltzman (2000), Mark Sobell and Linda Sobell (1973a, 1973b) fabricated the results of their controlled drinking study and misrepresented their procedures. Investigations purportedly exonerating the Sobells of wrongdoing have been analyzed in detail and shown to be fundamentally flawed (Maltzman, 2000). Nevertheless, the Sobells' controlled drinking study is still widely cited approvingly. Miller, Wilbourne, & Hettema (2003) give it a high rating for its methodological strengths. Caddy, Addington, and Perkins' (1978) study procedures and results are also fabricated (Maltzman, 2000), but are also highly rated. I am sure Miller is aware of the evidence supporting my allegations of scientific misconduct in these studies (see Maltzman, 2000), but he prefers to wear conformist ideological blinders.

Marlatt, Miller, and the Sobells are violating the first principle of caregiving and research: "Do no harm." They, as well as the great majority of cognitive behavior therapists in the field of alcoholism research and practice, fail to consider the damage to brain structure and function produced by excessive alcohol consumption and the evidence that controlled drinking of as little as one drink/day will prevent the recovery of a neuropsychological deficit. After an extensive review of the world literature on the effects of moderate alcohol consumption on the central nervous system, Eckardt *et al*. (1998) concluded that more than approximately three or four drinks a day may produce measurable damage to the central nervous system and that more than three drinks/day is associated with measurable neuropsychological deficits. Nevertheless, a self-help book on moderation management for people who have a drinking problem but self-rate themselves as nonalcoholic recommends not more than four drinks/day as the goal of "responsible drinking". Neuropsychological assessments are never mentioned, much less demanded, of people with a drinking problem who will try to learn to drink "responsibly" (Rotgers *et al*., 2002). What about therapists engaging in responsible therapy? The laudatory foreword to the book by Rotgers *et al*. is written by Mark and Linda Sobell. Need anything more be said? Yes!! When will cognitive therapists initiate responsible therapy following ethical principles and punish those who do not?

What Is to be Done?

A complex multidetermined problem such as the one under consideration, i.e. deceit and a lack of integrity among professionals in the alcoholism studies field, has no simple solution. Those of us who recognize the problem must do our best to meet the challenge. Confront revisionists' fabulist publications with the facts. Write to psychology, psychiatry, and specialized alcoholism journals about the problems. Write to the authors of textbooks in abnormal psychology and in alcoholism and addictions that misrepresent the evidence concerning the etiology, characteristics, and treatments of alcoholism; write to their publishers, backing the criticisms with the facts. Check the course offerings on alcoholism and addictions in local colleges, and the textbooks used in these courses. Write articles and letters to alcoholism journals, calling attention to revisionist misrepresentations of the treatment literature. Give presentations and organize symposia for professional meetings on revisionist misrepresentations of the facts of alcoholism treatment as well as on the dangers of harm reduction, controlled drinking, and self-selection to brain structure and function. Fight back. The problem is too important, the public welfare is too much at risk, to stand by and do nothing.

If you teach, discuss the ethical principles of the Belmont Report with your students and colleagues. Consider the implications of the three basic ethical principles for research and practice. Evaluate research in terms of these principles. Do Project MATCH and other commonly cited research such as Miller's efficacy studies meet the standards of autonomy, beneficence, and justice? Discuss the views of Bronowski (1972, 1977) and Popper (1962), and their implications for alcoholism research and theory, with your students and colleagues. "Evil triumphs because good people stand by and do nothing."

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Appendix 1: Letter to Griffith Edwards

Sir:

Re: No switching off the camera: how *Addiction* will respond to infringements of ethical publishing expectation

Your editorial concerning no switching off the camera has a hollow ring to someone such as myself who has experienced your refusal to switch on the camera for more than 15 years. In response to my recent objection that Rehm's (2000) review of my book (Maltzman, 2000) avoids the facts involved in the ongoing Sobell affair, your camera once more was switched off. You offered me your egocentric philosophy of "winning a few and losing a few". This is your response when my reply to Rehm provided what I believe are repeated instances of the most serious case of fraud ever to occur in the field of alcohol studies?

My paper, "Criticisms of the Dickens Committee Enquiry into the Sobells' alleged fraud and Doob's effort at their defense", was submitted to the *British Journal of Addiction* in 1985. After extensive review by assessors, you informed me in 1987 that you wanted to publish my manuscript, but wished to first have the response of the Sobells, Dickens, Doob, and Trachtenberg. Following receipt of their replies you informed me that the Sobells, Dickens, and Doob threatened a law suit. You then rejected my paper as libelous under British law (Edwards, Sept. 27, 1988). What would you do today? Clearly your response would be the same, as

reflected by publication of the Rehm (2000) review in *Addiction* and your refusal to publish my reply. The camera is still switched off, selectively.

I demonstrated in my 1985 manuscript that the Dickens Committee investigation was fundamentally flawed (see Maltzman, 2000). The data they reviewed were provided by the Sobells and listed in their Report. This data base did not include the most vital data, the raw data, the information on the time-line follow-back interview forms the Sobells insist they used. The Dickens Committee never took the first essential step of an investigation into alleged fraud: Examine the raw data and determine whether they can reproduce the published results. In the Sobell's case they cannot. Neither can the data obtained by the investigators for the Alcohol, Drugs, and Mental Health Administration (ADAMHA) as described in their report (see Maltzman, 2000).

Years later, despite having known evidence indicating that they were guilty of scientific misconduct, you invited the Sobells (1995) to serve as editors of a section on controlled drinking. There never was an editorial note describing to the readers of *Addiction* the events that had taken place years earlier in my effort to expose the details of the alleged fraud and fundamentally flawed investigations by home bases. Readers of this once esteemed journal were kept in the dark. The camera remained switched off.

When Rehm (2000) wrote a review of my book, misrepresenting its contents and motivation, defaming me as well as Kluwer Academic Publishers and its editorial staff, Rehm's manuscript was not sent to me for comment prior to its publication. Rehm states that my book contains "seemingly endless personal tirades" and "personal allegations". I present in my book a photocopy of a letter written by former Vice President of the United States, Albert Gore, when he was head of the Congressional Committee responsible for investigating fraudulent use of government funds in science. His letter states categorically that there never was a Congressional investigation of the Sobells and none was planned, contradicting the Sobells' written assertions that they were exonerated by a congressional investigation.

In their book (Sobell & Sobell, 1978, p. 160) the Sobells present a vignette of a sober sociopath, JL, an alcoholic who they trained to control his drinking. But he remained a sociopath. He faked a marriage in order

to obtain wedding gifts. I present in my book a photocopy of JL's marriage license (Maltzman, 2000, p. 332). It shows that Mark Sobell was the minister officiating at the wedding and Linda Sobell was the witness. The marriage was legal. Mark Sobell was ordained a minister through a correspondence course conducted from Modesto, California. Is all this and more merely a personal tirade, or is Rehm defaming me?

The following comments concern specific inadequacies in the Editorial recommendations for dealing with unethical practices. These comments concern the recommendation that fraud should ultimately be dealt with by the home base of the investigator against whom allegations of fraud have been made. The case of the Sobells, however, provides an obvious contrary example. The recommendation also overlooks the question of what constitutes the home base of an investigator.

The Addiction Research Foundation (ARF) should not have been the agency investigating the Sobells. The investigation should have been conducted by the District Attorney for San Bernardino County, the County in which Patton State Hospital is located, where the initial alleged fraud occurred and where the Sobells were employed during the alleged fraud. Ideally, Patton State Hospital should have conducted the investigation. However, they, as is probably true of most state hospitals, had neither the resources nor personnel capable of conducting an investigation. If the District Attorney of San Bernardino or the District Attorney of California had conducted the investigation instead of the ARF, the investigation would have been conducted by a disinterested third party with judicial powers. Instead, the ARF, their home base some 10 years after the occurrence of the original alleged fraud, was asked by the Sobells to conduct an investigation. The ARF hired the Sobells because of their purported expertise in controlled drinking training. The Sobells had been hired by the ARF despite information the latter received questioning the Sobells' integrity. An inherent conflict of interest is present when the ARF, the "home base", conducted the investigation of allegations of fraud. They were asked to determine whether their employees were guilty of misconduct after they had hired them despite having been warned of such allegations. Finding the Sobells guilty of misconduct would result in a loss of credibility on the part of the ARF and a blow to the development of their program.

Broad and Wade (1982) describe a classic case of a home base failure to act forthrightly in the face of a serious matter of ethical misconduct, plagiarism. Morris Chafetz, a member of the Harvard University Department of Psychiatry, was found to have plagiarized extensive material for a book he had written. Jack Mendelson reported that the Executive Committee of their Psychiatry Department voted to do nothing in face of the threat of legal action by Chafetz's attorney. It was hoped that Chafetz would find employment elsewhere. In a few years he did. Chafetz left Harvard to become the head of the newly formed National Institute on Alcohol Abuse and Alcoholism (NIAAA).

Several years later when Pendery and I informed Chafetz of our allegation of fraud by the Sobells, is it any wonder that Chafetz did nothing? Years afterwards when Peter Nathan, Executive Editor, instructed Mendelson, editor of the *Journal of Studies on Alcohol*, to reject my paper (Maltzman, 1989) alleging fraud by the Sobells, is it surprising that Mendelson acquiesced?

Home bases are composed of people. They have their own motives, ambitions, and fears. They are not necessarily disinterested parties. On the contrary, they all too often have conflicting interests so they choose the safest alternative: Do not make waves. Do not alienate people of influence. Do not jeopardize your own future.

It is true that the University of California, San Diego investigated the Slutsky case forthrightly. This is the exception rather than the rule in my experience. The University of California system has been guilty of failure to act appropriately when confronted with fraud in more recent cases than the Slutsky incident. A striking example of a "home base" failure was widely publicized in the media some time after it occurred in the fertility clinic of the University of California, Irvine (UCI) Medical School Hospital. Women "treated" in the fertility clinic gave birth to children born of fertilized eggs that were not their own. Without the knowledge or consent of "donors", physicians removed eggs from women who were fertile and implanted them in women who were infertile. Neither party was aware of, nor provided consent to, the procedure. A staff member reported the switching to university authorities who did nothing,

and stonewalled, until the practice was reported to the newspapers. By the time the UCI investigated the matter, forced by the publicity, several of the physicians involved had fled the country. The University was sued by the families involved.

Another case of "home base" failure to act expeditiously is playing out at the present time. The University of California, Los Angeles (UCLA) Neuropsychiatric Insitute (NPI) is being sued by families of young adult schizophrenics who had participated in a drug treatment study conducted at the NPI. Families complain that they were never given proper informed consent indicating that neuroleptic drugs administered to the patients would be withdrawn for a time, producing a considerable risk of relapse. While off medication, the son of one family threatened his mother with bodily harm and another young man committed suicide by jumping off the roof of a campus building across the way from my office. University administrators failed to thoroughly investigate, and stonewalled. The families sued the University. Research is not the only kind of fraud home bases fail to investigate forthrightly and effectively. University of California medical schools are being sued by the Federal Government for 40 million (US) dollars as the result of a study by federal auditors. Medical services were written off as conducted by the chairmen of various departments in the medical schools, although the actual treatments were provided by subordinates. Whistleblowers called attention to the practice of defrauding Medicare, not to the oversight of the University, the home base.

Your recommendation concerning the role of the home base in ethical practices is unsatisfactory because of the inherent conflict of interest involved in home base investigations. If the institution finds the scientist or practitioner guilty of misconduct, the institution itself may become liable, and its administrators lose credibility. Poor judgment was shown in hiring people lacking integrity and then failing to properly supervise their research or practice. Alumni and other potential donors may hesitate to provide philanthropic support to an institution that has lost credibility and status in the eyes of the public.

Ideally, there must be an independent body conducting the investigation of alleged fraud, a body not subject to obvious monetary and

political influence, with the necessary resources for conducting a thorough investigation. Procedures for establishing effective oversight, prevention, and the investigation of fraud are described elsewhere (Maltzman, 2000).

Another obvious shortcoming in the suggestion that the home base conduct the investigation of alleged fraud is that not every investigator or practitioner need have a home base or one that is prepared to conduct investigations of scientific misconduct. Caddy and Perkins are examples. Caddy, Addington, and Perkins (1978) recruited students as research assistants and Perkins was a faculty member at Fullerton State College, California. They purportedly conducted an independent third-year follow-up study of the Sobells' patients. The overwhelming majority of patients that Pendery *et al*. (1982) followed up signed an affidavit affirming that they were never interviewed by people who tape-recorded an 80-minute interview as reported by Caddy *et al*.

Pendery *et al*. (1982, Table 3) show that retrospective reports of the patients differed fundamentally from the results reported by Caddy *et al*. (1978) purporting to support the Sobell's conclusions. I informed the Fullerton State College Chanceller of our findings and the likelihood of fraud. She informed me that Perkins' research was "none of her business". Although the State College had a standard procedure for investigating student fraud, plagiarism, and misconduct, there was no comparable procedure for investigating faculty misconduct.

Caddy was no longer employed at the University of California, Riverside by the time his alleged fraud was uncovered. He could not be investigated by a home base. After a stint at Nova University in Florida, Caddy established an independent consulting business, as do many psychologists and psychiatrists in the USA. There is no "home base" to investigate individuals of this kind or others who work in small colleges, hospitals, or clinics, conduct research, and who may be guilty of alleged misconduct.

Professional societies such as the American Psychological Association, American Psychiatric Association, and American Medical Association are also inadequate home bases. They have no judicial powers. They cannot subpoena records and provide immunity so that witnesses under oath are protected by law from libel suits. Professional societies are essentially guilds designed to promote the business of their

membership and the reputation of their profession. They are not free of conflict of interest and personal pressures, and have neither the expertise nor the will to conduct a thorough investigation. At least, that has been my experience with the American Psychological Association. Perhaps conditions have changed in the past 20 years. I doubt it.

In addition to your journal, I have had different manuscripts rejected by editors of *Behavior Therapy*, *Behaviour Research and Therapy*, *Psychology of Addictive Behaviors*, and the *Journal of Studies on Alcohol*. All have used the excuse of possible libel action by the Sobells and others if my article in question were to be published. It is significant that the Sobells never threatened to sue me for libel.

My experience with the *Journal of Studies on Alcohol* took a different turn. After my manuscript was approved by assessors and I signed my copyright waiver, the manuscript was listed in a *Journal* issue among its articles forthcoming. Marlatt and the Sobells upon reading the announcement telephoned Peter Nathan, at the time Executive Editor of the *Journal* by virtue of his position as Director of the Rutgers Institute of Alcoholism, owner of the *Journal*, complaining about the possible publication of my manuscript (see Maltzman, 2000). Nathan instructed Mendelson, editor of the *Journal*, to reject my manuscript which he did. This was a breach of our contract. Fortunately, I was able to obtain the services of the oldest and most prestigious law firm in Los Angeles to represent me *pro bono*, in the public interest. We sued the *Journal of Studies on Alcohol*, Rutgers University, and the State of New Jersey for breach of contract and torts. After more than three years of haggling over the word "fraud" in my manuscript, and legal maneuverings by a large and prestigious Los Angeles law firm Rutgers was forced to hire to represent them, the judge set a date for the trial to begin. The attorneys for the *Journal* capitulated and I published my unaltered manuscript (Maltzman, 1989). Replies by the Sobells, Timothy Baker, and Cook were included in a special section of the *Journal*. The lead article was an editorial commentary by Peter Nathan. He likened me to the Ayatollah Khomeini ruthlessly pursuing Salman Rushdie, i.e., the Sobells. My reply to the commentaries was rejected by Mendelson without review. A revealing interview with the Sobells was published not long afterwards in *The Chronicle of Higher Education* (McDonald, 1989). The Sobells

acknowledged that they threatened to sue the *Journal of Studies on Alcohol* and the *British Journal of Addiction* if they published my manuscript. But they expressed surprise that any one would think that they sought to intimidate the journals. These are the kind of people you expect to apologize if their wrongdoings are exposed?

The above incidents and more are discussed in detail in my book (Maltzman, 2000) described by Rehm in *Addictions* (Rehm, 2000) as nothing more than "personal tirades". In contrast to your concern about fairness as a matter of policy expressed in your Editorial, your response to my reply to Rehm, which you refused to publish, was that I accept your philosophy of "you win a few and lose a few". Why did you not write that to the Sobells in 1987 or respond to me in 2001 as you did to the Sobells in 1987? No doubt you will turn the switch on young scholars without influential connections who are guilty of minor ethical infractions. But will you ever switch on the camera when it really matters?

All of the above raises a serious question ignored by your Editorial: Who is going to watch over the watchdogs, the journals? Your editorial is out of touch with the reality of misconduct in science and particularly the ongoing misconduct by major figures in the field of alcohol studies.

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Appendix 2: Letter to Mary Beth Kenkel

7/01/01

Dr. Mary Beth Kenkel
Editor
California School of Professional Psychology
5130 E. Clinton Way
Fresno, CA 93727

Dear Dr. Kenkel:

I know that it may appear ridiculous to provide a reply to editorial reviews that is almost as long as the original manuscript, which was uniformly judged to be overly long. Unfortunately, the reply is so lengthy because the reviews in large part are ridiculous. Please take the time to read my reply, because I think some very serious issues are involved.

I have been an associate, member, and fellow of the American Psychological Assoiation (APA) for more than half a century. I started publishing in APA journals in 1950, in the *Journal of Experimental Psychology* and *Psychological Review* initially, and over the years in the *Psychological Bulletin, Journal of Abnormal Psychology,* and *Psychological Monographs,* the as well as a variety of non-APA journals. I was on the editorial board of the *Journal of Experimental Psychology* from the mid-1960s to the mid-1970s, as well as a number of non-APA journals, including associate

editor of one. I have seen journal editing from the inside as well as the outside. I would say that the set of reviews of my manuscript are the most uninformed and nonprofessional reviews I have ever seen in relation to an APA journal in my more than 50 years' experience.

My replies are in order of the length and detail of the three original reviews, concluding with my response to your general criticisms and a suggestion that I hope you will consider.

Sincerely yours,

Irving Maltzman
Professor Emeritus

enc
cc Bennett

Reviewer #4

Other than comments on usage and grammar, the principal criticism by Reviewer #4 is on p. 26 of the manuscript: "This is just ignorant." This is simply inappropriate. If Reviewer #4 disagrees with the application of a statement by Alasdair MacIntyre, one of the leading moral philosophers of our time, he/she ought to point out why that is the case. The "review" is insulting in form as well as content. This is not the sort of review that should be acceptable to a scholarly professional journal.

Reviewer #1

> Some issues seemed confused: for example, in several places the authors seemed to assume that cognitive behavioral approaches to alcoholism treatment uniformly involved treatment goals of controlled drinking. I have marked other examples on the manuscript. In many places the logic of the argument the authors were making was difficult to follow.

In no place or form does the manuscript assume that cognitive behavior therapists uniformly adopt controlled drinking as a treatment

goal. It happens that the leading figures in the controlled drinking treatment movement are all cognitive behavior therapists. It is also the case that a study of undergraduate textbooks of abnormal psychology written by cognitive behavior therapists all present controlled drinking as a treatment goal for alcoholics in a positive light. This treatment is not true of other clinical psychologist authors of abnormal psychology texts (see Maltzman, 2000, Chapter 9). Since no one that I know of has polled all cognitive behavior therapists treating alcohol-dependent people, there is no way to know what they all think the treatment goal for alcoholics ought to be. That the manuscript assumes that all cognitive behavior therapists favor controlled drinking as a treatment goal for alcoholics is an erroneous impression of Reviewer #1. It would take only a sentence or two to clarify explicitly the position of the manuscript on this point.

> Because issues were confusing and were presented with only superficial coverage (a great deal of the literature bearing on the main issues raised is simply not presented in the manuscript), I recommend the manuscript not be published.

A proper review would indicate where there was a failure to cover the literature and the importance of the omissions. Due to the length of the manuscript, the number of references and descriptions of the literature were limited to save space. This general kind of statement without specifics is an inappropriate criticism because of its vagueness. What issues were confusing? How were they confusing? What was superficial? Notations on the manuscript are often illegible and hardly critical and certainly not informative.

> [I]t is not clear what relevance many portions of the manuscript have for the practicing psychologist. For example, a vague suggestion that psychologists screen alcoholic clients with a neuropsychological battery (pp. 34, 40) is not helpful to most practitioners.

This is hardly a major criticism. It would be a simple matter to provide specific information concerning the kind of tests to be used, their

purpose, and where they may be obtained. Duffy (1995) provides a table listing "bedside" tests for assessing brain damage that may produce a form of denial. They were not described in the manuscript, but it would be a simple matter to describe them and other assessment tools including computerized tests that score individual test results and aggregate them.

> I don't believe most readers of *PP* [*Professional Psychology*] care to read another article advancing the notion of alcoholism as a disease. Most practitioners are less interested in theoretical models of alcoholism and more interested in the practical realities of treating the alcoholic patient. The current manuscript contributes little new in this latter regard.

First, the reviewer did not poll the readership concerning their interest in reading another article about the disease concept. Second, when was there an article on the disease concept published in *PP*? If there had been, it would most probably be a demonized construction, not a current conceptualization such as may be found in Maltzman (2000) or as one briefly stated in the manuscript. A quick review of every issue of *PP* for the past 10 years was undertaken; there has not been an article on the disease concept of alcoholism in *PP* during that period. Such sophistry on the part of Reviewer #1 is inappropriate. Furthermore, at least some articles in a professional journal should stimulate thinking and curiosity. Its readers are not merely a group of technicians. They were trained as scientist practitioners. In that regard, a great deal is contributed by the present manuscript.

The manuscript forces the practitioner to think about the ethical and legal problems involved in treating autonomous people as well as using them in experiments. It raises important issues about informed consent and the necessity of determining whether or not individuals treated for alcohol dependence are truly informed: (a) do they comprehend the information, and (b) are they provided with material information? In both cases, validated assessments that may be employed are described. Information about these assessments and their application can be expanded in a revision. The discussion of the problem of denial

produced by brain damage needs to be expanded. It is reasonable to assume that this aspect of denial raised in the manuscript would be new to the overwhelming majority of the readership of *PP*. Again, specific assessments are described. Their application to treatment could be developed further, and I would do that in a revision.

Response to Reviewer #1 comments written on the manuscript:

p. 11.	Last paragraph, states that wording is very awkward and logic is very difficult to follow. This can easily be improved.
p. 17.	Questions whether "falsified" is the "wrong word". It is not.
p. 18.	Asks, "What do you mean here?" I distinguish between the legal terms of incompetence and mental capacity in the material that followed the example.
p. 22.	Asks why figures for Gual *et al*. do not add up, and whether I mean medical or psychological when I describe "42% of the patients suffered some clinical pathology. . . ." The latter reference is to medical pathology. Figures do not add up to 100% because 7.6% of the participants had died and 3.9% were missing.
pp. 24, 37, 38.	Deletes "infrahuman". . . leaves "animal". This correction is inappropriate. "Nonhuman" is used in the title of an article by Higley *et al*. in the *Proceedings of the National Academy of Science* included among the manuscript References. It is common usage in biobehavioral studies. That is a good enough standard for me. What are the grounds for the Reviewer's deletion?
p. 33.	Comment "suggests it is conscious" is an inappropriate inference. The meaning of denial as anosognosia is expanded in the manuscript following the comment in the margin. This material needs to be expanded further because it is apparent that the Reviewers have no understanding of the nature of the problem.
p. 34.	Minor grammatical, terminological preferences.

p. 35. Bottom of page states "wording" is awkward. I don't think it is, but I will ask my wife who is a retired English teacher.

Specific comments written on the manuscript's margins do not indicate that a particular subject lacks citations or is superficial in its treatment. For the most part, the comments are on grammar, awkward wording, etc.; nothing on substance. Reviewer #1 does not support his/her broad and ambiguous generalization that coverage is superficial by marking specific passages or pages that can be characterized as superficial, much less indicating how they could be improved. This is not a scholarly peer review of a scholarly paper.

Reviewer #2

This is the most substantial of the three reviews. However, it contains numerous errors of fact, interpretation, and reasoning. Each of the Reviewer's criticisms is examined in turn. A series of incorrect interpretations are found in the very first paragraph and reappear several times during the course of the review. They will be treated in detail as they reappear. The fact that Reviewer #2 fails to grasp the essence of the paper and uses disparaging terms such as "polemical" and "rambling" sets the tone for the remainder of the review.

> This manuscript is an extraordinarily long (53 pages) and rambling polemical argument, the essence of which is that because alcohol impairs cognitive functioning, heavy drinkers and people with alcohol use disorders must not be permitted to make a choice between moderate drinking and abstinence. The authors argue for psychologists to exercise 'paternalistic' protection of drinkers under the Belmont Report guidelines, as people with diminished capacity to make autonomous choices. They conclude that it is unethical to permit consideration of any goal other than abstinence unless the psychologist has performed comprehensive neuropsychological testing to ensure the absence of cognitive impairment that might impede comprehension of informed consent.

Unfortunately, the Reviewer from the outset conflates the ethical issues and their implications. As a consequence, in part, he/she repeatedly misinterprets and misrepresents the contents of the manuscript. A neuropsychological assessment should be available for all people with alcohol use disorders because they are at risk for brain damage, which may be reflected in impaired cognitive functioning. Paternalism is required if a person with alcohol use disorders shows evidence of brain dysfunction, even though they comprehend the informed consent, because of the basic ethical principle of nonmaleficence: "Do no harm." "Moderate" drinking, which the Reviewer does not define, may exacerbate and prevent reversal of brain dysfunction and neuropsychological deficits responsible for impaired "cognitive functioning". This would be true even if the person in question does not have diminished capacity, can fully comprehend the information in an informed consent. Neuropsychological assessment does not reveal whether the person fails to comprehend the informed consent or not. A structured interview is needed for such an assessment, as discussed in the manuscript and below.

Treatment guidelines are not based on the Belmont Report, but as repeatedly stated in the manuscript, they are based on case law, legal decisions in malpractice suits brought in clinical medicine. Contrary to the Reviewer's interpretation that paternalism is needed because of diminished capacity, the final sentence to the Abstract states: " . . . [U]ntil brain research and neuropsychology can establish markers distinguishing between who can and who cannot drink without exacerbating brain structure and function, practitioners ought to continue to adopt beneficence, paternalism, and provide abstinence oriented treatment." Diminished capacity is another reason brain structure and function and neuropsychological deficits would be found in a majority, as indicated in empirical research (e.g., Cala, 1987; Glass, Chan, & Rentz, 2000).

> The article's rhetoric conceptualizes psychologists as divided into two opposing camps: those favoring the authors' disease conception of alcoholism, and others termed 'revisionists' or 'autonomists' who favor patient choice of goals.

This assertion is false. The manuscript does not make such a division. On the contrary, it states there is hegemony of the cognitive behavior therapists who are the major proponents of "autonomy". This judgment is based on reading the published professional literature. Publications in the professional psychology literature are dominated by the revisionist view and are anti-disease in concept — as they understand "disease". It actually is a gross distortion of the classic disease theory of Jellinek. Cognitive behavior therapists do not provide an accurate description of Jellinek's work or of modern interpretations of disease and alcoholism as a disease as represented by the work of Maltzman (1991, 1994, 1998, 2000) and numerous philosophers of medicine, philosophers of science, and medical practitioners cited by Maltzman, some of whom are cited in the manuscript. The disease concept is overwhelmingly accepted by MDs and alcoholism counselors. Alcoholism as it appears to be treated in professional psychology journals and books is not divided. It is dominated by one position, the one represented by Marlatt, William Miller, the Sobells, and their numerous students, colleagues, and others. This does not mean that all cognitive behavior therapists reject a disease conception of alcoholism. It means that those psychologists in the alcoholism treatment field who publish and are recognized leaders are primarily cognitive behavior therapists who reject their own distorted construction of a disease conception of alcoholism.

If the above analysis is in error, Reviewer #2 should name psychologists who, like the above, have been president of the Association for Advancement of Behavior Therapy (AABT), APA Divisions 12 and 50, and who write about alcoholism using a disease framework. There are none. Professional psychology as represented in the professional literature and the power structure of the APA and AABT are not made up of two equally divided camps. They are dominated by a group of active cognitive behavior therapists. It is my impression that psychologists working in treatment facilities have a different view of matters. But they are not for the most part the ones who write articles in professional journals, conduct treatment evaluation, research, write handbooks of treatment evaluation, and hold office in the professional associations.

> The manuscript begins with eight pages of direct quotations, to characterize the revisionist position and educate psychologists about the fundamental principles underlying the Belmont Report. These principles, designed to protect human research participants, are then extended to cover patients entering treatment.

This statement is incorrect and misleading. As noted in the manuscript, the Belmont Report — designed to protect experimental subjects — was based in part on case law developed over the years from malpractice lawsuits in clinical medicine. The Canterbury vs. Spence case is explicitly cited several times in the manuscript to emphasize that ethical treatment of patients based on the common law preceded the writing of the Belmont Report. In particular, rules of informed consent protecting patients in treatment were in place prior to the writing of the Belmont Report. This priority is described at length by Faden and Beauchamp (1986), cited several times in the manuscript.

> The authors correctly point out that informed consent to treatment (of any kind) is rarely preceded by neuropsychological assessment to determine that the patient is free from diminished cognitive capacity to consent. Such neurocognitive testing is argued to be essential before patients are permitted to make a choice between abstinence and moderation (although not for any other of the treatment choices mentioned, such as surgery or admission for inpatient alcoholism treatment).

Two different ethical requirements are conflated by Reviewer #2 in this implied criticism, as they are in the opening paragraph of the review. One is to perform an assessment prior to treatment in order to ensure that the person is not suffering from diminished capacity, the capacity to understand and use the information provided. A second ethical requirement is to perform a neuropsychological assessment for brain damage, which may be further exacerbated by alcohol consumption.

The MacArthur Competence Assessment Tool for Treatment (MacCAT-T) (Grisso & Appelbaum, 1998), for example, is a structured interview designed to determine the mental capacity of persons to

provide a truly informed consent. It assesses the patient's capacity to comprehend, understand the information provided, integrate the diverse information, and apply it. The MacCAT-T should be used in any case where there is a question concerning an individual's capacity to understand the consent form. It is not a neuropsychological test such as the Wisconson Card Sorting Test (WSCT) that has been validated as a measure of a particular kind of brain dysfunction, damage to the executive functions of the prefrontal cortex. It is used, among others, for assessing brain damage which would be exacerbated by alcohol consumption. A person may have a high % perseverative error score on the WSCT indicating prefrontal cortical damage, but thoroughly understand the informed consent. Neuropsychological tests are not ethically required prior to surgery or admission to inpatient alcoholism treatment unless the treatment goal is controlled drinking. I believe a neuropsychological assessment should be given prior to the start of any treatment, including abstinence. Information indicating the presence of brain dysfunction that would be exacerbated by further drinking would probably provide an additional incentive to maintain abstinence. The WSCT is not given prior to surgery because it does not assess the capacity to understand informed consent. The MacCAT-T would be used prior to surgery or inpatient treatment if there is any question concerning understanding the informed consent, if there is any question concerning mental capacity. Presence of brain damage is material information which must be provided in an ethical informed consent prior to controlled drinking treatment. Furthermore, an assessment of denial by a validated assessment tool may be needed. A validated assessment of denial is needed to distinguish between rationalization, making of excuses, from anosognosia, unawareness of symptoms. Such an assessment is needed because a person with the latter form of denial may select controlled drinking because they believe they do not have the symptoms of an alcoholic when in fact they display such symptoms as well as brain damage as reflected by neuropsychological testing or brain imaging.

A developmentally disabled adult may not be able to live independently, pay rent, buy groceries, etc., and therefore have a guardian appointed to care for them. However, such a developmentally disabled

adult may, as determined by a MacCAT-T, comprehend the information concerning the costs and benefits of having surgery on a malignancy. To repeat, Reviewer #2 fails to distinguish between a neuropsychological test designed to obtain material information for a proper informed consent, e.g., is brain damage present, and a structured interview that assesses whether the person can comprehend the information contained in an informed consent.

Failure to understand this distinction on the part of Reviewers #1 and #2 does not reflect positively on the thoughtfulness of their reviews. Of course, a lack of clarity in the manuscript could have contributed to Reviewers' conflation of issues. A revised manuscript would make a greater attempt to clarify the distinctions among the different kinds of assessments and describe these assessments more explicitly. However, if the Reviewers had been familiar with the pertinent literature, as they should be, such confusion should not occur.

The Reviewer's bias and lack of critical expertise is evident in the following criticism:

> The authors' highly selective use and characterization of research is evident throughout the manuscript. In reviewing research on 'treatment self-selection as efficacious,' for example, the authors dismiss one of the clearer RCTs [randomized controlled trials] on the subject (Sanchez-Craig & Lei, 1986) because its sample was judged to be too 'biased' by exclusion criteria. A study by Booth *et al*., is dismissed because the authors perceive the groups to have been non-equivalent at baseline. The Walsh *et al.* (1991) study, in contrast, is cited in defense of the authors' position, without pointing out that randomization was violated by allowing EAPs [employee assistance programs] to reassign patients from the AA or choice groups into inpatient treatment, thereby clouding any between-group comparisons.

The above comments concerning each of the studies in question will be reviewed in turn. In each case, it will be shown that the Reviewer's judgment is incorrect and omits critical details of the studies. All of the errors of omission and commission are in one direction, in support of the Reviewer's bias.

Reviewer #2 complains that the Sanchez-Craig & Lei (1986) study was "judged to be too 'biased' by exclusion criteria." The term "judged" as used suggests that the author of the manuscript makes a subjective and possibly biased assertion. Reviewer #2 fails to consider the following important details: subjects were volunteers solicited for the study, and volunteers who believed in the disease concept or had attended AA meetings were excluded. Of course this results in a bias. A sample selected in this fashion does not represent the people in the community who receive treatment for alcoholism. Unfortunately, Sanchez-Craig does not indicate how many volunteers were so rejected on these grounds. She states in a later paper (Sanchez-Craig *et al*., 1984, p. 399) that she did not present the disease concept in sufficient detail, and admits that asking the patients to keep a diary of their drinking suggests that drinking is permissible despite the fact that their treatment goal is abstinence. Furthermore, the small number of participants in the study means that it lacks power. Its results are not interpretable for all these reasons. The fact that participants were eventually randomly assigned does not make it a methodologically sound experiment with an unbiased sample.

There are many factors determining the quality of an experiment in addition to the fact that subjects are randomly assigned to treatments. The original RCT — the model which is the gold standard when properly done — does not have such shortcomings. It did not employ volunteers, but people who entered the hospital for treatment of tuberculosis (TB). The subjects in the experiment represented those in the community who would seek treatment. Studies using volunteers obtained through advertisements and often paid for their services do not represent the community of people entering treatment, especially when they are excluded for a belief related to a particular treatment. The original RCT had external validity, which Sanchez-Craig's study does not, in addition to other shortcomings. Furthermore, as is true of the other studies favored by Reviewer #2, the people conducting the treatment evaluation are also the people or their student assistants conducting the treatment. Investigators are not blind to the treatment received by participants. Such studies are open to experimenter effects.

Again, this shortcoming was not present in the original RCT — or in any methodologically sound RCT.

Reviewer #2 states that Booth *et al*. is dismissed because the manuscript authors perceive the groups to have been nonequivalent at baseline. This, again, is a misleading prejudicial assertion. The term "perceived" suggests that the author of the manuscript is making a subjective evaluation. In the Prescribed Abstinence (PA) group, 10/10 participants had liver disease at baseline; in the Chose Abstinence (CA) group, 7/15 had liver disease; and in the Chose Controlled Drinking (CD) group, 4/12 had liver disease. There is a statistically significant difference between these groups in the frequency of baseline liver disease as indicated by a chi-square test I conducted. This is not a subjective perception. The groups were not randomly assigned. They differed significantly in the percentage of individuals suffering from liver disease. Differences in treatment outcome among the groups are unreliable, PA = 20% functioning well at follow-up as compared to 27% and 42% for CA and CD, respectively. A shift in two people would eliminate outcome differences among the groups. Reviewer #2 also fails to note the criterion of successful controlled drinking, the criterion which makes CD superior to the other two groups: 8 drinks/day for men and 6 drinks/day for women. The highly regarded RCTs on naltrexone (O'Malley *et al*., 1992; Volpicelli *et al*., 1992) used 5 drinks/day as their measure of relapse. Gual *et al*. reports that 5 drinks/day was the average consumed by his alcohol-dependent men. It is apparent that Booth *et al.*'s criteria for successful controlled drinking is unacceptably high. Nevertheless, this methodologically unsound paper is defended by Reviewer #2.

Reviewer #2 omits, again, important information in his/her criticism of the Walsh *et al*. study. Independent clinical and research staffs were employed, in contrast to the Sanchez-Craig and Booth *et al*. studies. For ethical reasons, the emergency practice associate (EPA) staff — the clinical staff — had the authority to provide supplementary hospital treatment to individuals judged to be in need of hospitalization because of the seriousness of their alcoholism, they were suicidal, etc. These patients were retained in their original groups, and received their designated

treatment *in addition* to inpatient hospital treatment. Their results were analyzed as part of their original group. This is in contrast to Sanchez-Craig, who did not admit people who believed in the disease concept into any treatment condition, a violation of the ethical principle of justice as well as poor research methodology producing the loss of external validity. Reviewer #2 misrepresents the nature of the Walsh *et al*. experiment. The patients were not "reassigned into inpatient treatment". They received inpatient treatment in addition to their assigned treatments AA or Choice, and were analyzed as part of that group to which they were originally assigned. Despite the "clouding" of differences between the groups as a result of adding hospital treatment to the other treatments for ethical medical reasons, hospital treatment was significantly better than the other two groups on many measures. If the data of the people receiving hospital inpatient treatment as well as outpatient treatment were discarded, the superiority of hospital inpatient treatment over the other two in all likelihood would have been greater than what was reported. The criticism that the manuscript selectively uses and characterizes research is without merit.

The most inappropriate criticism of the manuscript of all is the following:

> The authors then maintain (erroneously) that there is only one study . . . that approximates the classic RCT in experimental medicine and cite Rychtarik *et al.* (1987). Sanchez Craig's study clearly qualifies as an RCT on any standard, and no mention at all is made of the classic RCT by Sobell and Sobell. The RCT of treatment goal selection by Graber [*sic*] is cited elsewhere in the article, but apparently also does not qualify as evidence in the authors' view.

There is much to criticize in the above. First, Reviewer #2 fails to recognize the importance of external validity. As stated correctly in the manuscript, only Rychtarik *et al*.'s study corresponds to the classic RCT. In their study, patients already in a hospital for treatment were randomly assigned to treatment conditions. The studies by William Miller and student colleagues (including Graber and Miller), Sanchez-Craig, and the Sobells solicited volunteers and then randomized them. As previously

mentioned, an important characteristic of the RCT in experimental medicine, including the first classic study, is that different staff conduct the clinical and research aspects of the experiment. This is true of the Walsh *et al*. study and the Foy *et al*. study. It is not true of the Sanchez-Craig study. It is not true of the Sobell and Sobell study, which was not mentioned in the manuscript because the Sobell and Sobell study (1972, 1973, 1976, 1987) does not belong in the science canon. Nothing so much identifies Reviewer #2 as a biased and uninformed reviewer as his/her comments concerning the Sobell and Sobell study. The following are some of the major methodological flaws in the Sobell study ignored by Reviewer #2. We shall turn to the verisimilitude of the Sobells' study later. A more detailed discussion and evidence may be found in Maltzman (2000).

Sobell and Sobell did not randomly assign a population sample of patients to experimental and control groups. They asked a hospital population for volunteers to participate in a controlled drinking treatment study. Patients volunteered thinking they would be taught how to control their drinking. Instead, half of the participants in the critical arm were placed in an abstinence group and half in a controlled drinking group. Subjects purportedly were randomly assigned. However, Pendery, Maltzman, and West (1982) show that there was a statistically significant order effect in their participation in the experiment. The majority of patients in the controlled drinking condition were treated and followed up before the abstinence group. During the time some of the abstinence treatment patients were still in treatment and almost all were still to be followed up, reports appeared in the media lauding the success of the new treatment of controlled drinking. Patients in the two groups were able to communicate with each other, and their identities and treatment assignments were known to Linda Sobell, who was responsible for most of the follow-up interviews in the first year and all of the interviews in the second year follow-up. All of the above information and more is available in the professional literature (Maltzman, 1989, 2000; Pendery *et al*., 1982). The Sobells' criterion of controlled drinking was not more than 6 drinks/day. Their criterion for controlled, moderate drinking is now considered evidence of relapse. As noted earlier, Volpicelli *et al*. (1992) and O'Malley *et al*. (1992) in their RCT studies of naltrexone

used 5 drinks/day as a criterion of relapse. Turning to the question of verisimilitude, evidence is presented by Maltzman (2000) which he believes beyond reasonable doubt demonstrates that the Sobells are guilty of scientific misconduct. They intentionally misrepresented their research procedures, fabricated results, and misrepresented results and events surrounding the study.

For example, a photocopy of a medical record (Maltzman, 2000, p. 156) shows that a patient represented in the results of the abstinence group actually received controlled drinking treatment. It is also shown that the Sobells necessarily had to know, by virtue of the time-line follow-back method they insist they used, that they did not interview the patients every 3–4 weeks for two full years (Maltzman, 2000, p. 148). The Sobells (1984) insist that they have been exonerated by a congressional investigation. Maltzman (2000, p. 173) shows a photocopy of a letter written by the former Vice President of the United States when he was head of the Congressional Committee responsible for investigating fraud in science stating unequivocally that a congressional investigation of the Sobells' research never occurred and none was planned.

A photocopy is presented of the marriage license of a patient (Maltzman, 2000, p. 332) the Sobells (1978, p. 160) say became a sober sociopath following treatment who then faked a marriage to obtain gifts. The marriage license shows that Mark Sobell served as the minister performing the marriage and Linda Sobell was the witness. The preceding is only some of the evidence of the alleged misconduct. Analysis showing the fundamental flaws in the investigations by the Addiction Research Foundation and ADAMHA are also detailed in Maltzman (2000). The facts mentioned above are good reason not to consider the Sobells' study a model RCT. This manuscript should not be victimized by a Reviewer's lack of scholarship.

Shortcomings in the Graber and Miller study may be found detailed in Maltzman (2000) and need not be repeated here. A lack of critical scholarship on the part of Reviewer #2 is further displayed by his/her additional comments on treatment outcome research.

> Decrying that 'Few public or private alcoholism treatment facilities are large enough to offer alternative treatments provided by experienced

> counselors and a large enough patient population to meet the requirements of a comparative treatment outcome study with sufficient statistical power to obtain reliable results,' the authors then dismiss the largest and most powerful RCT in the field — Project MATCH [Matching Alcoholism Treatments to Client Heterogeneity] — because it failed to include a 28-day inpatient comparison group, but laud the uncontrolled VA [Department of Veterans Affairs] study by Moos *et al*. When evidence in support of a Minnesota Model is sought, the authors cite the uncontrolled survey research of Hoffman *et al*. [*sic*] without concern for sample bias or the lack of any control or comparison group.

There are several good reasons why Project MATCH is not a powerful RCT in addition to its failure to use a standard treatment employed in the community as a comparison, a practice used for good reason in RCTs in experimental medicine. Its purpose is to maintain external validity, generalizability to the community. MATCH used volunteers who were randomly assigned to treatment, not patients entering treatment who were randomly assigned. There were exclusionary criteria which markedly reduces its external validity. Perhaps one of the most serious violations of representativeness was the exclusion of individuals dependent on other drugs. Another serious shortcoming overlooked by Reviewer #2 as well as other commentators on MATCH is its gross overgeneralization due to a poor experimental design. MATCH employed only a single low intensity of treatment — 12 meetings for twelve-step facilitation (TSF) and CBT, and 4 for motivational enhancement therapy (ME). This is not the level of treatment employed in the community. It is a level at the borderline of minimal effectiveness, as shown by Monahan and Finney (1996). If the RCT model as employed in experimental medicine had been employed, treatment would have been approximately 28 days or varied to suit the needs of the patient as determined by an experienced counselor, not one who must follow a manual. Instead, the designers of MATCH in my judgment replaced good science, a parametric study of treatment intensity, with what would appeal to managed care: the briefer the treatment, the better.

Moos *et al*. studied patients in the VA hospital system who self-selected one of three different treatments provided by experts in their

field. Traditional treatment had significantly superior outcomes than CBT and a mixture of CBT and traditional treatment. Detailed statistical analyses showed that most of the variance in treatment outcome was accounted for by attendance in AA meetings during aftercare. In what sense was the study uncontrolled, other than the fact that subjects were not randomly assigned volunteers? Of course it is a biased sample; it includes only male veterans. But they are a substantial population in our society. The study has external validity because the three groups, although not randomly assigned to treatments, self-selected standard treatments offered in the VA community. If Reviewer #2 knows of a serious lack of control, why does he/she fail to indicate what it is? Failing to do so is sophistry not worthy of a review for a scholarly journal.

Reviewer #2 further states, "When evidence in support of a Minnesota Model is sought, the authors cite the uncontrolled survey research of Hoffmann *et al*. [*sic*] without concern for sample bias or the lack of any control or comparison group." Reviewer #2 misrepresents Harrison, Hoffmann, & Streed (1991).

Harrison *et al*. (1991) did not conduct uncontrolled survey research. They report results from a sample of patients from the CATOR (Comprehensive Assessment and Treatment Outcome Research) registry of patients in treatment programs that pay to have an independent organization — CATOR — provide assessments of their patients entering treatment and to conduct independent follow-up evaluations. The Registry contains data from some 50 000 patients. Harrison *et al*. report results from a sample of 5000 inpatients and 1000 outpatients.

What are the many reasons for the 'inflated success' rates reported by Harrison *et al.*? A scholarly review would state explicitly why the rates are purportedly inflated, not require the reader to trust the Reviewer's judgment. From everything discussed so far, there is no good reason to trust the scholarly judgment of Reviewer #2. Hester and Miller (1995) certainly give no reason for their ignoring Harrison *et al.*, as they do other studies, including studies with comparison groups whose results fail to suit their bias (see Maltzman, 2000). Reviewer #2 as well as Hester and Miller (1995) fail to consider the aversion conditioning results from the Schick Shadel hospitals that were compared with the outcomes from the Minnesota Model programs in the CATOR

registry (Smith & Frawley, 1993). The Schick Shadel outcomes, as assessed by independent CATOR staff, were significantly better than those of the Minnesota Model–matched patients from the CATOR registry. Both kinds of treatments provide results greatly superior to those reported by Project MATCH or any of the small RCTs conducted by Miller and his colleagues who use volunteers answering advertisements. None of this is mentioned by Reviewer #2. It must also be noted that patients in the CATOR Registry are not excluded because they are dependent on drugs other than alcohol, in contrast to Project MATCH and other small trials conducted by cognitive behavior therapists. Instead, Harrison *et al*. present informative outcome results for subgroups, those dependent only on alcohol, alcohol and cocaine, etc., and overall results. Reasons why success rates of the largely Minnesota Model treatment programs in CATOR are superior, I suspect, are because they provide more intensive treatment, better treatment, and more aftercare than the low-intensity RCTs conducted by cognitive behavior therapists. Monahan and Finney (1996) demonstrate, contrary to Miller, that intensity of treatment is a significant factor affecting treatment outcome.

> The aggregate outcomes reported by Moos *et al*. from the VA study are, in fact, highly similar to those resulting from other large clinical trials of the past 30 years, including Project MATCH.

This latter statement is one more assertion demonstrating a lack of critical scholarship. Why does Reviewer #2 not cite these other "clinical trials"? I suspect because there is only one other study, the RAND report, and it was not an RCT. Patients were not randomly assigned to treatment facilities or treatments. Its profound defects are detailed at length elsewhere (Maltzman, 2000; Wallace, 1989) and need not be repeated here. I doubt very much that he has in mind the Küfner and Feuerlein (1989) large multisite study conducted in Germany that obtained far superior results than those reported by MATCH and Moos *et al*., and is never mentioned by Hester and Miller [see Maltzman, 2000 for an extended discussion of the results of Feuerlein and Küfner (1989) and Küfner and Feuerlein (1989)]. Treatments used

in Germany are traditional clinical treatments with an abstinence goal. A scholarly review does not make assertions unsupported by evidence such as citations to the relevant literature so that the truth status of the assertion can be assessed.

The similarity between Moos *et al*. and Project MATCH results does not prove the effectiveness of MATCH. It only proves the uncritical nature of the Reviewer's judgment. There are probably important differences in the populations studied by Moos' VA comparative project and Project MATCH. Patients in VA hospitals generally lack stable social support and employment; otherwise, they would not be in a VA hospital. They are not excluded if they are dependent on other drugs. That these people have the same outcomes as Project MATCH, which had middle-class participants who fit rigid exclusionary criteria, reveals the inferiority of the brief treatments provided by MATCH.

> The authors oddly reference Hester & Miller's . . . as the citation for their statement that 'Reading the descriptions of cognitive behavior therapy procedures designed to produce harm reduction/controlled drinking. . . .' Most of the content of their volume has nothing to do with harm reduction. . . .

There is nothing odd about the reference. Harm reduction in the present context is an euphemism for controlled drinking. Hester and Miller attempt to show the superiority of cognitive behavior therapy methods — which are primarily directed towards controlled drinking — as vastly superior to out-of-date traditional treatments, although they do state that cognitive behavior therapy may also be used with abstinence as a goal. Hester and Miller's bias is evident in their failure to cite studies in Australia, Germany, and the United States, showing superior results than any CB therapy with abstinence or controlled drinking as treatment goals (see Maltzman, 2000, Chapter 7).

Reviewer #2 comments on the manuscript's criticism of Miller, Hester, and colleagues for failing to provide neuropsychological assessments and assessments of mental capacity. Reviewer #2 again proceeds to a conflation of issues, reflecting his/her failure to understand the

distinction between mental capacity to provide an informed consent and information essential to an ethical informed consent.

> From here the argument proceeds that even moderate drinking yields cognitive impairment serious enough to impair informed consent, selectively citing only one study [Cala] (from an enormous literature) which 'found. . . . [F]ollows from this, the authors reason, that moderation can never be ethically provided as a choice, presumably for any drinker.' On this ground they critique Marlatt's RCTs demonstrating effective secondary prevention strategies to reduce heavy drinking among college students, claiming that in fact this constitutes "harm induction." They propose, without supporting evidence, that 'the most effective approach to reducing the problem of heavy drinking on college campuses is primary prevention' which, it would follow, should promote total abstinence, and argue that such strategies 'must be employed in the college community.' This argument is further supported by the authors' quotation of Wagenaar's statement of the prevention paradox that 'the majority of alcohol-related death and disability is attributable to moderate drinkers.'

Errors in fact and interpretation in the above statement will be addressed in turn.

1. The manuscript does not state that "even moderate drinking yields cognitive impairment serious enough to impair informed consent, selectively citing only one study [Cala] (from an enormous literature)". This statement is a misrepresentation and reflects the repeated conflation of issues on the part of the Reviewer. The manuscript states that even "moderate" drinking over the long term can produce brain damage (Parsons, 1998). This implies that individuals entering treatment for alcohol problems must be assessed for brain damage, especially if they are to be offered the choice of controlled drinking vs. abstinence. The evidence overwhelmingly indicates that continued drinking will exacerbate brain damage or prevent its reversibility. The manuscript does not suggest that moderate drinking causes brain damage, which will prevent informed consent. Reviewer #2

continues to confuse the assessment of mental capacity for informed consent, the capacity to understand information provided, and neuropsychological assessment for brain damage. The latter is material information that must be part of an informed consent involving controlled drinking. The manuscript does state that alcoholics, people with alcohol dependence, may suffer more severe brain damage, sufficiently so that when sober as well as under the influence, they may have diminished capacity and therefore may not be able to give a proper informed consent. A MacCAT-T would be needed to assess the capacity to give a proper informed consent. Whether the capacity to give informed consent is normal or not, if there is any brain damage, as indicated by a neuropsychological assessment or more direct neuroimaging assessments of brain structure and function, this information is material to a proper informed consent and must be provided.

2. There is not an "enormous" literature on the brain damage effects of moderate drinking. Reviewer #2 is probably referring, erroneously, to all studies of brain damage produced by alcohol. By far the largest number of such studies refer to people with evidence of severe alcohol dependence. The manuscript explicitly indicates that it is referring to people consuming alcohol who do not even approximate the Korsakoff syndrome. Furthermore, a number of such studies, in fact probably the majority, are cited in the manuscript. These include Parsons (1998) who concludes, after an intensive study and reanalysis of his own long-term research and the research of other investigators on the negative consequences of "moderate drinking" by social drinkers, that there is a continuum of damage from the Korsakoff syndrome to moderate drinking. The approximate cut-off is 21 drinks/week. This is discussed in the manuscript as well as reference to a Swedish population study, and others. Reviewer #2 should have offered citations to support his/her position. On the basis of the available information, a reasonable conclusion is that he/she is confused about the issues and the criticisms are without foundation.
3. Given this basic confusion on the part of Reviewer #2, he/she reaches the *reductio ad absurdum*, "[it] follows from this, the authors reason, that moderation can never be ethically provided as a choice,

presumably for any drinker." There are marked individual differences in vulnerability to brain damage and to its reversibility (Cala, 1987). There is no evidence that 1 drink/day for females and 2/day for males causes brain damage in social drinkers. However, as the manuscript discusses, Sanchez-Craig (Wilkinson & Sanchez-Craig, 1981) found that 1 drink/day for recovering alcoholics who have shown neuropsychological deficits prevents reversal of those deficits. Moderate drinking — as defined by the World Health Organization (WHO) above — can be undertaken for someone without a history of alcohol problems. For some one recovering from alcoholism, even 1/day is a risk factor. To determine the extent of the risk, a neuropsychological — and ideally — neuroimaging assessment would be needed. It is unclear what Reviewer #2 means by "drinker". If he/she means someone who has had problems with drinking, then the answer is yes, it cannot be an ethically provided choice in the absence of an assessment showing that the individual is free of brain damage. Is that unreasonable given the research literature which shows that even 1 drink/day may prevent reversal of a neuropsychological deficit (Wilkinson & Sanchez-Craig, 1981)?

4. Reviewer #2 fails to grasp that binge drinkers, college students who drink 5 or more drinks on at least 1 occasion in the previous month, are at risk for brain damage. He/she confuses the issue by talking about heavy drinkers, undefined. Marlatt's secondary prevention was applied to binge drinkers, as defined above.

> On this ground they critique Marlatt's RCTs demonstrating effective secondary prevention strategies to reduce heavy drinking among college students, claiming that in fact this constitutes "harm induction." They propose, without supporting evidence, that 'the most effective approach to reducing the problem of heavy drinking on college campuses is primary prevention' which, it would follow, should promote total abstinence.

The manuscript argues that a reasonable implication from the evidence presented in the manuscript is that Marlatt's harm reduction is harm induction. The evidence is that Sher *et al*., cited in the

manuscript (p. 25), demonstrated that college binge drinkers show a significant correlation between their negative consequences, such as missing classes, etc., and neuropsychological deficits. Since binge drinkers have a high incidence of neuropsychological deficits, as indicated by Sher *et al*., it places them at risk for exacerbation of those deficits if they continue to drink. The manuscript also cites experiments which show that a blood alcohol level (BAL) below 0.08, a BAL lower than what would usually be produced by binge drinking, results in greater deleterious effects on memory in men in their early 20s than those in their late 20s. All of the participants in Marlatt's study were 19 years or younger. Additional research is cited in the manuscript showing that there is a final growth spurt in brain development, particularly in the frontal lobes, from approximately years 18–24, which makes the brain particularly vulnerable to damage during that period.

Since submitting this manuscript to *PP*, an article has been published by Marlatt and his students (Blume, Marlatt, & Schmalion, 2000) in which they report neuropsychological deficits in executive functions in college binge drinkers. They do not discuss the possible connection between neuropsychological test results and brain dysfunction or the consequences of continued binge drinking on neuropsychological deficits. They are apparently oblivious to the fact, as is apparently true of the Reviewer, that neuropsychological tests have been validated as indicators of brain damage.

There is a reasonable basis for the manuscript's conclusion that Marlatt's attempt to train binge drinking college students, all of whom are minors particularly vulnerable to brain damage, to drink moderately without assessing their state of brain function or informing them of the risk of exacerbation of existing brain damage is harm inducing. Failing to obtain neuropsychological assessments in Marlatt's study of harm reduction as secondary prevention and to provide material information for a proper informed consent is engaging in unethical research as well as harm induction. Nothing Reviewer #2 has stated undermines the conclusion reached in the manuscript.

A significant problem Reviewer #2 does not address, but is noted in passing by Reviewer #3 in an inappropriate notation on a margin of the manuscript, is that the participants in Marlatt's study are minors. It is illegal for them to drink. Nevertheless, Marlatt and the University of Washington paid binge-drinking minor students to participate in this illegal practice. Furthermore, there is no indication that Marlatt *et al*. required binge-drinking minor women who were given controlled drinking training to provide evidence that they were not pregnant and that they were informed of the risks of fetal alcohol effects even with moderate alcohol consumption.

5. They propose, without supporting evidence, that 'the most effective approach to reducing the problem of heavy drinking on college campuses is primary prevention' which, it would follow, should promote total abstinence, and argue that such strategies 'must be employed in the college community.' This argument is further supported by the authors' quotation of Wagenaar's statement of the prevention paradox that 'the majority of alcohol-related death and disability is attributable to moderate drinkers.'

   Again, Reviewer #2 misrepresents the manuscript. It is apparent that primary prevention would have greater impact on drinking problems on college campuses because this is true in the community at large. The manuscript provides an analysis of the impact Marlatt's secondary prevention program would have on binge drinkers versus all other drinkers on campus (p. 26f). A far greater number of problems are produced by the nonbinge drinkers on campus than the binge drinkers because there are far more of the former than the latter. The analysis provided in the manuscript supports the need for primary prevention. This material is ignored by Reviewer #2. Wagenaar's statement supports in general terms what was demonstrated in the manuscript. Contrary to the assertion by Reviewer #2 that no evidence is provided in support of primary prevention, results of a study by Weitzman and Kawachi (2000) are cited in the manuscript (p. 27), showing that schools promoting student participation in voluntary associations have a 26% lower rate of binge drinking than schools that do not promote

such activities. This is a far greater impact than the one produced by Marlatt's secondary prevention. Results are based on a national sample of 17 000 students. Mills, Neal, and Peed-Neal (1983) provide an extensive discussion of primary and secondary prevention efforts on the college campus. They also offer a detailed description of the application of primary prevention techniques for the college campus that they believe are superior to secondary prevention efforts.

The Reviewer's assertion that the manuscript's emphasis on primary prevention promotes total abstinence is false. No such assertion or implication is present. Abstinence is only argued as necessary for students under the age of 21 because that is the law. Abstinence is particularly important for minors suffering neuropsychological deficits at risk for further brain damage as it is essential for students of all ages suffering from brain damage.

> Selective standards of evidence persist throughout the article. The authors maintain ignoring scientific evidence to the contrary, that 'denial' is characteristic of alcoholism, citing as evidence Marty Mann's 1958 *New Primer on Alcoholism*. The problem of denial is incorrectly claimed to be 'largely ignored by cognitive behavior therapists,' ignoring twenty years of psychological research to understand and address motivational processes underlying change in addictive behaviors.

The above passage contains a series of misrepresentations and shows a lack of understanding of the problem of 'denial'. The manuscript states (pp. 33–34) that a reasonable hypothesis is that some of the variance in denial is due to brain dysfunction. The distinctions should have been made more explicit in the manuscript. A validated denial scale (Ward & Rothaus, 1991) derived two factors, denial and rationalization. My hypothesis is that unawareness of symptoms characterize the denial factor and excuses for drinking characterize the rationalization scale factor. There is no evidence that the motivational problems that Miller and Rolnick are studying and that others have studied over the years involve the kind of denial for which standardized assessments

have been developed, the unawareness of illness which I hypothesize to be a form of anosognosia. Three studies of standardized scales for assessment of denial are cited in the manuscript. They are apparently ignored, and obviously have not been read by the Reviewer. That Miller and Rolnick and others are addressing the problem the manuscript is calling attention to is not apparent. They and other cognitive behavior therapists do not cite the use of validated assessment tools in their discussion of "motivational" problems. Mann (1958) was not cited as experimental evidence, but to note that from the outset, people knowledgable about the behavior of alcoholics consider denial — or rationalization — an important problem. It is probably the case that Miller and Rolnick conflate denial and rationalization, calling both denial.

Reviewer #2 seems to be questioning the evidence that 'denial' is a characteristic of alcoholism. If it is not a characteristic, how is it possible that validated scales of denial of alcoholism have been developed (Newsome & Ditzler, 1993; Ward & Rothaus, 1991)? It is one of the symptoms Jellinek (1952) describes in the progression of symptoms found in alcoholics based upon his analysis of data from 2000 respondents. Validated denial scales have been used to predict treatment outcomes with considerable success, and serve as guidelines for counselors to adapt treatments to the needs of patients during the course of treatment (Ward & Rothaus, 1991). These studies of denial were not discussed in detail in the manuscript, but were cited so that the interested reader could discern the importance of using validated assessments of denial and their usefulness in treatment. Obviously, Reviewer #2 did not make the effort to read the citations. Evidence for the kind of denial that can be described as a form of anosognosia is contained in the papers cited in the manuscript and ignored by Reviewer #2. Discussions of interpretations of denial in terms of ego mechanisms and avoidance have been repeatedly presented and may be found in Duffy (1995), as well the presentation of a case report of the kind of denial described as a form of anasognosia in the manuscript.

"Twenty years of psychological research to understand and address motivational processes underlying change in addictive behaviors" are ignored in the manuscript because they are not relevant to the question of whether some individuals with denial are suffering from a brain

dysfunction as demonstrated in cases of denial with schizophrenics (Flashman, McAllister, Andreasen, & Saykin, 2000; Mohamed *et al*., 1999). The manuscript urges that similar research be conducted on denial expressed by alcoholics. The rationale is evident. Damasio and his colleagues have shown in exquisite neurological and behavioral detail that a form of denial is anosognosia (Damasio,1994; Bechara, Tranel, Damasio, & Damasio, 1996). Duffy (1995) has also discussed the neurological basis for a form of denial and described some of its implications for assessment and treatment.

Reviewer #2 states, "as evidence for the veracity of a 1930s disease model, the authors cite a polemical book by Maltzman which did not benefit from scientific peer review." It is not apparent how Reviewer #2 would know that Maltzman's book did not benefit from "scientific peer review", since only Maltzman and his editor would be privy to that information — and possible reviewers. Reviewer #2's comment is inappropriate sophistry. Maltzman does not support a 1930s disease model (whatever that means). Once more, Reviewer #2 fails to offer any citations to such a disease model. Jellinek's disease "model" was published in 1960. Maltzman (2000, pp. 13–15) cites 12 studies demonstrating a symptom progression supporting Jellinek's formulation. The manuscript does not cite or discuss Jellinek's progression and the research on the problem by other investigators because the citation to Maltzman (2000) contains an extended discussion of the problem of symptom progression. The importance of a lawful symptom progression is that it demonstrates that alcohol dependence, alcoholism, is a syndrome. It shows a lawful pattern of observable, objective, recurring signs and symptoms. A syndrome is one of the two essential features of a disease. The second essential characteristic of a disease is that it is judged to be a significant deviation from accepted standards of health, a feature generally agreed upon for alcoholism. The manuscript briefly makes this statement. Maltzman's book devotes more than a chapter to the question of alcoholism as a disease. Maltzman's book is not a demonstration of the veracity of a "1930s disease model", but builds on Jellinek's formulation and suggests that the disease conception of alcoholism is not merely an abstraction, a hypothetical construct, but a disease entity where the symptoms are a consequence of a continuous interaction between the brain and social

environment. Changes in brain function as a consequence of such interaction gives rise to the observable signs and symptoms. Maltzman's book also suggests how brain chemistry may be normalized as a result of interactions with the social environment.

Reviewer #2 states, "in fact those studies [of symptom progression] show that the further a sample departs from white American males, the less resemblance there is to Jellinek's tentatively hypothesized progression." Once again, there are no citations to support this false and misleading assertion. It can only be surmised that the Reviewer is referring to the out-of-date and irrelevant studies by Cahalan and colleagues (e.g., Cahalan, Cisin, & Crossley, 1969). Cahalan *et al*. report results of drinking surveys in terms of the % individuals in one of several categories: abstainers, light drinkers, moderate drinkers, and heavy drinkers. Percentages in these different categories differ between black women and white women and between women and men, as well as between different religious groups, ethnic groups, etc. These data do not address the issue of Jellinek's progression of symptoms for people who are alcohol-dependent. Cahalan's results cannot address this problem because the dependent variables, measures of drinking employed by Cahalan and colleagues, do not differentiate among people classifiable as alcohol-dependent, alcohol abusers, or preclinical heavy social drinkers. The measures of heavy drinking versus moderate drinking do not permit such differentiations as defined by DSM-III-R, DSM-IV, or ICD-10. Individuals classified as alcohol-dependent, e.g. by DSM or the criteria of gamma alcoholism used in early studies, confirm Jellinek and contradict Reviewer #2. For example, data on symptom progression were obtained from the US National Comorbidity Survey, a nationwide survey of US households based on a stratified, multistage probability sample of a noninstitutionalized civilian population with a supplemental sample of students living in campus group housing. Face-to-face household interviews were conducted with 8000 respondents (Nelson *et al*., 1996). Diagnostic assessments were based on definitions and criteria of alcohol-dependence for DSM-III-R and ICSD-10. In contrast to Cahalan, the symptom progression was determined for each person classified as alcohol-dependent according to diagnostic criteria. Results reported by Nelson *et al*. confirm the conception of a lawful progression among three different symptom clusters for people who are

alcohol-dependent. The rank order of appearance of symptoms in men and women correlated 0.97, a result flatly contradicting the Reviewer's assertion. The point is that whether the progression from social drinking to dependent drinking is faster in women than in men, or varies in speed of progression for different ethnic groups, cultures, etc., within each group core symptoms appear in essentially the same order. Whether the progression is fast or slow may be a function of a variety of different risk factors: age at which drinking begins, family history, ethnicity, culture, etc. Once started, however, there is a lawful pattern to the sequence of symptoms.

It is true that Nelson *et al*. did not analyze ethnic and religious differences, but it is reasonable to assume that a stratified probability sample of the magnitude conducted included various ethnic, socioeconomic, and religious groups. If these groups differed in the order of appearance of symptoms, then it is unlikely that an overall lawful progression would have been obtained. It must also be noted that these results are for a nonclinical sample, not all members of AA, further demonstrating the generality of Jellinek's formulation of a progression of symptoms for people classified as alcohol-dependent. Park and Whitehead (1973) report a corresponding progression of alcohol symptoms in Finnish and in American male alcoholics despite their cultural differences. Maltzman (2000) lists some dozen research studies supporting Jellinek's generalization. There is no point in listing them here. They are available to any critic or reviewer who is curious.

The Reviewer's final paragraph concludes with another series of false and misleading assertions: "The manuscript became painful to read at the 'demonization' section. Complex cognitive behavioral models are reduced to a single simplistic straw man: 'Alcoholism for them is nothing but a bad habit.'" "Complex cognitive behavioral models" are not reduced to a single simplistic straw man. That alcoholism is nothing more than learning is the core notion explicitly stated by Marlatt, the Sobells, and others. For example, according to Marlatt (1985):

> [I]n recent years, a third approach has emerged as an alternative to the moral and disease modes [*sic*] of addiction. Derived from the principles of social-learning theory, cognitive psychology, and experimental

> social psychology, the addictive behavior model makes a number of assumptions that differ markedly from the disease and moral models. From a social-learning perspective addictive behaviors represent a category of 'bad habits' including such behaviors as problem drinking, smoking, substance abuse. . . . In terms of frequency of occurrence, addictive behaviors are presumed to lie along a continuum of use rather than being defined in terms of discrete or fixed categories such as excessive use (loss of control) or total abstinence. In contrast, *all* points along this continuum of frequency of occurrence, from very infrequent to 'normal' to excessive use, are assumed to be governed by similar processes of learning. [p. 9]

Thombs (1994) states:

> The principal aims of 'behaviorism' are to elucidate the conditions of human learning and to develop a technology for behavior change. Behaviorists believe that most or all human behavior is learned; this includes not only adaptive but also maladaptive behavior (e.g., addiction). One of the major premises, then, is that certain fundamental laws (known and unknown) govern the initiation, maintenance, and cessation of human behavior. Alcohol or drug use is considered a behavior that is subject to the same principles of learning as driving a car, typing a letter, or building a house. [p. 74]

"[T]he prevailing behavioral view is that the behavior of most alcoholics is learned" (Nathan & Lipscomb, 1979, p. 306).

> I want to talk today about the latest in behavioral approaches to alcoholism. Technically, this approach is now known as cognitive social learning theory and therapy. There are a variety of reasons for the name change. For several unfortunate reasons, behavior therapy has acquired an undeserved bad name, especially in the whole controversy over controlled drinking. We are now calling it cognitive social learning theory, and it is different in more than in name. . . . What are the distinguishing characteristics of the cognitive social learning strategy as it applies to the treatment of alcoholism? . . . [T]he approach

> focuses on the observable characteristics of behavior, rather than, for example, on theory confirmation or the search for historical antecedents or unconscious determinants of behavior. . . . Such variables are the amount, frequency and duration of drinking; the problems associated with excessive use; and situational and environmental factors in abusive drinking. . . . Especially important are an individual's processes associated with drinking: expectations about the effect of drinking; expectations of its effects on behavior (it makes me more powerful, it makes me sexy, it makes me better). In a real sense, expectations exert their effect regardless of what alcohol actually does. [Nathan, 1985, p. 169]

Nathan continues, "[T]he approach focuses on the observable characteristics of behavior . . ." in relation to the claim that: "Especially important are an individual's processes associated with drinking: expectations about the effect of drinking; expectations of its effects on behavior. . . . In a real sense, expectations exert their effect regardless of what alcohol actually does."

According to Maltzman (2000),

> On the one hand these statements describe behaviorism as focused on observable characteristics of behavior and on the other Nathan affirms expectations as determiners of behavior. Undefined, unobservable expectancies replace undefined, unobservable unconscious psychodynamic processes. When it first evolved behavior therapy explicitly revolted against the latter kind of theorizing. How can an unobservable reified mental process given a label "expectancy" and nothing more, cause behavior? Expectancies in the hands of behavior therapists such as Nathan are a return to Cartesian ghosts. It is word magic. [p. 35f]

Complex multiple regression models can be formulated showing that "expectancy" statistically predicts outcomes, accounts for some of the variance in dependent variables, is affected by "prompts", etc. "Expectancy" in these models is a label attached to a series of questions in a questionnaire. As Wittgenstein noted years ago, there is no necessity

that reality corresponds to a word. Because the term "expectancy" exists does not mean that a thing or process of expectancy exists. Reification of the term, "expectancy", no matter how complicated the model in which it is buried, does not change the basic approach: alcoholism is nothing but a bad habit.

It might be further noted that the Reviewer's comment concerning how "painful" it was reading the 'demonization' section of the manuscript are his private feelings and should be kept just that.

> The imagined coven of 'revisionists,' accused of demonizing the authors' position, is itself demonized as unethical, scientifically uncritical, 'lacking verisimilitude,' mired in 'myth,' motivated by 'monetary gain,' and seeking to silence criticism. Citing as examples two articles with unclear connection to the point, the authors allege that: 'A goal of the leading cognitive behavior therapists in the alcoholism field is to enlarge market share vs. traditional Minnesota Model treatment.' At this point, the article enters a realm that is neither professional nor psychology.

The inability of Reviewer #2 to view the field dispassionately is evident in the above passage. Use of the inflammatory term 'coven' is an expression of the very attitude that he/she denies exists. Reviewer #2 reflects an interesting example of one kind of denial: avoidance of unpleasant, painful, information. Grounds supporting each of the negative evaluations described in the above quotation of Reviewer #2 is provided in Maltzman (2000). Briefly, as already indicated, Maltzman (2000) provides evidence demonstrating that the Sobells are guilty of alleged scientific misconduct, that so-called exonerations never occurred or are based on fundamentally flawed investigations; the flaws are delineated in Maltzman (2000). Some of that evidence is presented in Maltzman (1989).

The story of the latter article, among others (Maltzman, 2000), describes an organized attempt by several influential cognitive behavior therapists to suppress criticism. It has been described briefly elsewhere as well (Maltzman, 1992). A telephone call by Marlatt and the Sobells to Peter Nathan, the executive editor of a leading

journal in the field of alcoholism studies, the *Journal of Studies on Alcohol*, was sufficient to censor publication of a manuscript (Maltzman, 1989) already accepted for publication following blind peer review. A common ideology was all that was needed to produce censorship and the investment of vast amounts of public funds in a failed court battle to suppress Maltzman's paper. He was able, by luck, to obtain the services of the oldest and most prestigious law firm in Los Angeles, the law firm of former secretary of State Warren Christopher, to represent him *pro bono*, in the public interest. They sued the *Journal of Studies on Alcohol*, its publisher, Rutgers University, and the State of New Jersey for breach of contract. Nathan and the *Journal* then hired an equally large Los Angeles law firm to defend their action. After more than three years and an enormous expenditure of public funds, the *Journal* capitulated. It published Maltzman's article (1989), accompanied by an editorial and a series of papers criticizing Maltzman's paper. It refused to accept a reply by Maltzman. Further details of this effort at censorship by a group of cognitive behavior therapists, as well as other such incidents, are described at length by Maltzman (2000).

By tossing Maltzman's (2000) book into the dustbin of history as 'not receiving scientific peer review', Reviewer #2 is able to deny a history of suppression and censorship practiced by cognitive behavior therapy journals and handbooks. Reviewer #2 is also able by these same means — avoidance of the evidence — to avoid reading an entire chapter devoted to an examination of the uncritical acceptance of poorly designed RCTs by cognitive behavior therapists and the demonization of their straw man construction of a "disease model". It was the Sobells (e.g., 1995) who made this "controversy" into a clash between two cultures — science (them) versus the Luddites (whoever disagrees with them).

Once more, Reviewer #2 engages in sophistry by asserting that two citations in the manuscript are beside the point but does not indicate which two citations are in question and why he/she thinks they are irrelevant. That, of course, requires that the references have been read.

Strategies of some cognitive behavior therapists to enlarge market share and gain hegemony over the alcoholism treatment field are described

in papers presented at the 1986 meeting of the AABT (Miller, 1987; Sobell, 1987). There is no point in reiterating that information in an overly long manuscript — or in this overly long reply to reviewers. The information is available to the curious, either in the words of the authors above or a critic (Maltzman, 2000).

Reply to Editor Kenkel's comments:

1. *The manuscript reads like an attack on harm reduction approaches specifically*. It is an attack on harm reduction in the form of controlled drinking as a treatment for alcoholics or people with brain damage. Harm reduction takes many different forms depending upon the drug involved, problems, and circumstances. Needle exchange programs for heroin addicts that would reduce the spread of AIDS is a very different form of harm reduction than the provision of controlled drinking to people in treatment for alcohol dependence or abuse who may have brain damage and do not receive a neuropsychological assessment. Each problem with a harm reduction approach has to be evaluated in its own right. Since many people with alcohol use disorders have brain damage, it is essential that if controlled drinking, now called harm reduction, is considered, an assessment of brain function must be made.
2. *I appear to attack cognitive behavior therapy per se*. That is not my intent. See my discussion of Baxter and Schwartz on p. 37. I can readily make this more explicit. It so happens that the most prominent exponents of harm reduction in the alcoholism treatment field are Marlatt, William Miller, and the Sobells, all cognitive behavior therapists.
3. *There are inaccuracies in reporting of results and in the interpretations of results*. I believe I have shown each of these criticisms is without merit. Inaccuracies in every case are those of the reviewer, not the manuscript.
4. *The manuscript does not fit the PP format; it does not have direct practice implications*. On the contrary, the manuscript has specific implications of enormous import for the practitioner who deals with individuals suffering from alcohol use disorders. These involve the nature of a proper informed consent which must include all material

information; assessments for mental capacity to provide a fully informed consent, assessments to evaluate possible brain damage as a result of heavy alcohol use and therefore at heightened risk for exacerbation of the damage or prevention of its reversal with harm reduction; and assessments for denial in the sense of anosognosia, unawareness of illness as a result of brain damage which affects informed consent as well as treatment progress. There are specific validated assessments for each of these problems. Their applications were not described in detail in the manuscript, but this would be a simple matter to remedy in a revision. The important general point is that the manuscript increases practitioners' awareness of problems and approaches that have important implications for their practice and the well-being of their patients. Practitioners are not exposed to this information in the usual articles in APA journals. This is evident in that even purported experts in the field such as the reviewers of this manuscript show that they are poorly informed concerning alcoholism treatment research and related problems.

Lack of critical information is also apparent in an article appearing in a recent *PP* (Read *et al*., 2001). It purports to present empirically supported alcoholism treatments. Instead, it uncritically parrots Hester and Miller (1995), presenting the methodologically weak RCTs of cognitive behavior therapists as the empirically supported base for treatment of alcohol use disorders. I examine some of these in the present manuscript. They are examined in detail in Maltzman (2000) and found seriously flawed, whereas the best studies of treatment effectiveness are ignored by Hester and Miller (1995) and by Read *et al*. The latter review ignores the article by Seligman and Levant (1998) pointing out the differences in the external validity of efficacy and effectiveness research. Read *et al*. rightly note the serious negative consequences of excessive alcohol use such as violence and morbidity and mortality, but never mention brain damage and the ethical problems involved in informed consent when controlled drinking is offered as a treatment choice in the absence of a neuropsychological assessment, in the absence of material information, essential information for a truly informed consent. The article by

Read *et al*. cites none of the outstanding studies of the effectiveness of traditional clinical approaches to alcoholism treatment, such as the CATOR studies (Harrison *et al*., 1991) and the elegant multisite study conducted in Germany by Feuerlein and Küfner (1989) and Küfner and Feuerlein (1989) and others. Obviously the Read *et al*. paper was given a positive peer review, exposing the *PP* readership to misinformation and the lack of important information with immediate application. Rejection of my manuscript contributes to the continued failure to properly inform the readership of *PP* concerning treatments for alcohol use disorders. The public in need of proper care is the ultimate victim.

5. *Manuscript is too long*. True, following the suggestions for more emphasis on specific applications, it would be even longer. I agree the paper is unusually long. I respectfully request a reconsideration of your decision to close the file on this manuscript. Give me the opportunity to improve the paper. Its clarity and greater emphasis on specific applications can be improved. It has the potential for helping to fill an obvious gap in the knowledge base of professional psychologists. They are being poorly informed about alcohol use problems. The most important contribution to application made by this manuscript is one which needs to be heard. It is the first ethical principle of caregiving: "Do no harm."

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