HEART DISEASE

A MEDICAL DICTIONARY, BIBLIOGRAPHY,
AND ANNOTATED RESEARCH GUIDE TO
INTERNET REFERENCES

JAMES N. PARKER, M.D.
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FORWARD

In March 2001, the National Institutes of Health issued the following warning: "The number of Web sites offering health-related resources grows every day. Many sites provide valuable information, while others may have information that is unreliable or misleading."[1] Furthermore, because of the rapid increase in Internet-based information, many hours can be wasted searching, selecting, and printing. Since only the smallest fraction of information dealing with heart disease is indexed in search engines, such as **www.google.com** or others, a non-systematic approach to Internet research can be not only time consuming, but also incomplete. This book was created for medical professionals, students, and members of the general public who want to know as much as possible about heart disease, using the most advanced research tools available and spending the least amount of time doing so.

In addition to offering a structured and comprehensive bibliography, the pages that follow will tell you where and how to find reliable information covering virtually all topics related to heart disease, from the essentials to the most advanced areas of research. Public, academic, government, and peer-reviewed research studies are emphasized. Various abstracts are reproduced to give you some of the latest official information available to date on heart disease. Abundant guidance is given on how to obtain free-of-charge primary research results via the Internet. **While this book focuses on the field of medicine, when some sources provide access to non-medical information relating to heart disease, these are noted in the text.**

E-book and electronic versions of this book are fully interactive with each of the Internet sites mentioned (clicking on a hyperlink automatically opens your browser to the site indicated). If you are using the hard copy version of this book, you can access a cited Web site by typing the provided Web address directly into your Internet browser. You may find it useful to refer to synonyms or related terms when accessing these Internet databases. **NOTE:** At the time of publication, the Web addresses were functional. However, some links may fail due to URL address changes, which is a common occurrence on the Internet.

For readers unfamiliar with the Internet, detailed instructions are offered on how to access electronic resources. For readers unfamiliar with medical terminology, a comprehensive glossary is provided. For readers without access to Internet resources, a directory of medical libraries, that have or can locate references cited here, is given. We hope these resources will prove useful to the widest possible audience seeking information on heart disease.

The Editors

[1] From the NIH, National Cancer Institute (NCI): **http://www.cancer.gov/cancerinfo/ten-things-to-know**.

CHAPTER 1. STUDIES ON HEART DISEASE

Overview

In this chapter, we will show you how to locate peer-reviewed references and studies on heart disease.

The Combined Health Information Database

The Combined Health Information Database summarizes studies across numerous federal agencies. To limit your investigation to research studies and heart disease, you will need to use the advanced search options. First, go to **http://chid.nih.gov/index.html**. From there, select the "Detailed Search" option (or go directly to that page with the following hyperlink: **http://chid.nih.gov/detail/detail.html**). The trick in extracting studies is found in the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer, and the format option "Journal Article." At the top of the search form, select the number of records you would like to see (we recommend 100) and check the box to display "whole records." We recommend that you type "heart disease" (or synonyms) into the "For these words:" box. Consider using the option "anywhere in record" to make your search as broad as possible. If you want to limit the search to only a particular field, such as the title of the journal, then select this option in the "Search in these fields" drop box. The following is what you can expect from this type of search:

- **Kawasaki Syndrome and Acquired Heart Disease**

  Source: Journal of Musculoskeletal Medicine. 19(2): 65-68,74-75. February 2002.

  Summary: This journal article provides health professionals with information on the etiology, clinical presentation, diagnosis, and management of Kawasaki syndrome (KS). This multisystem inflammatory disorder of unknown cause most commonly affects young children. Although the cause of KS is unknown, an underlying genetic predisposition is suspected based on familial cases and twin studies. When KS is not recognized and managed early, children with the condition may develop coronary artery aneurysms or associated cardiac abnormalities. KS is the leading cause of acquired heart disease in children. All of the signs and symptoms of KS are common and nonspecific, which complicates the diagnosis. Although the diagnosis often involves uncertainty, the following clinical features help distinguish KS from illnesses that mimic

it: Children with KS are typically quite irritable and reluctant to be examined, conjunctival injection spares the zone immediately around the iris and is more prominent on the surface of the eyeball than on the mucosa of the eyelids, pharyngeal infection is typically nonfocal, lymphadenopathy is generally unilateral and confined to the anterior cervical region, the rash often has a perineal confluence and is very rarely vesicular, palmar and plantar erythema may come and go with the fever, and desquamation typically occurs 10 or more days after the fever begins. Although tests such as complete blood cell count and differential, erythrocyte sedimentation rate, microscopic urinalysis, and liver function are individually nonspecific, they can often be helpful in making the diagnosis. Aspirin has been the most widely used therapeutic agent, but intravenous immunoglobulin has been demonstrated to be more effective, especially for treating children with high or recurrent fever. 4 figures, 1 table, and 17 references. (AA-M).

- **Helicobacter Pylori Infection as a Risk Factor for Gastrointestinal Symptoms in Patients Using Aspirin to Prevent Ishaemic Heart Disease**

  Source: Alimentary Pharmacology and Therapeutics. 15(7): 1055-1059. July 2001.

  Contact: Available from Alimentary Pharmacology and Therapeutics. Blackwell Science Ltd., Osney Mead, Oxford OX2 OEL, UK. +44(0)1865 206206. Fax +44(0)1865 721205. E-mail: journals.cs@blacksci.co.uk. Website: www.blackwell-science.com.

  Summary: Aspirin use in the secondary prevention of ischemic heart disease may provoke gastrointestinal (GI) discomfort. This article reports on a study undertaken to register GI symptoms and complications in patients with cardiovascular disease using aspirin and to relate these symptoms to infection with H. pylori. Blood samples were obtained from 398 consecutive patients in the Coronary Care Unit at a hospital in the Netherlands; samples were analyzed for serum antibody levels to H. pylori infection. Questionnaires were sent 2 weeks after discharge to assess GI symptoms. Questionnaires were returned by 314 patients (79 percent). A total of 183 out of 314 patients (46 percent) reported GI symptoms. Of the 238 patients using 80 to 100 milligrams of aspirin daily, 145 (61 percent) recorded GI symptoms. Besides aspirin, the use of calcium antagonists was correlated with GI symptoms. Of the 128 patients using calcium antagonists, 84 (66 percent) reported GI symptoms. The prevalence of GI symptoms in H. pylori positive and negative patients using aspirin was 48 percent and 52 percent respectively. The authors conclude that 2 weeks after discharge, almost 50 percent of the patients with cardiovascular disease experienced GI symptoms, especially patients using aspirin or calcium antagonists. Patients seropositive for H. pylori and using aspirin or calcium antagonists did not have more GI discomfort compared to non infected patients. 2 tables. 16 references.

- **Lost In Darknes, Depression, Diabetes, And Heart Disease**

  Source: Diabetes Forecast. 56(5): 44-46. May 2003.

  Contact: Available from American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) 232-3472. Website: www.diabetes.org.

  Summary: This article helps readers with diabetes to understand the problem of depression and the risks that having a chronic disease may contribute to any likelihood of experiencing depression. The author notes that depression may occur as a reaction to illness and changing social circumstances, such as after the onset of type 1 diabetes or a divorce, but depression also seems to have a genetic basis in some patients. The author briefly reviews the biological basis of depression, stress hormones, diagnosing

depression, and treatment options. Although depression causes severe dysfunction, many people can be successfully treated by a combination of medication and by tested forms of behavioral therapy such as cognitive behavioral therapy, which is a form of coaching. Regular physical exercise has been shown to reduce depressive symptoms; exercise also improves blood glucose control and cardiovascular function.

- **NCEP-Defined Metabolic Syndrome, Diabetes, and Prevalence of Coronary Heart Disease Among NHANES III Participants Age 50 Years and Older**

  Source: Diabetes. 52(5): 1210-1214. May 2003.

  Contact: Available from American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) 232-3472. Website: www.diabetes.org.

  Summary: Although the individual components of the metabolic syndrome are clearly associated with increased risk for coronary heart disease (CHD), the authors of this study wanted to quantify the increased prevalence of CHD among people with metabolic syndrome. The authors used the Third National Health and Nutrition Examination Survey (NHANES III) to categorize adults over 50 years of age by presence of metabolic syndrome, with or without diabetes. Metabolic syndrome is very common, with approximately 44 percent of the United States population over 50 years of age meeting the criteria. In contrast, diabetes without metabolic syndrome is uncommon (13 percent of those with diabetes). Older Americans over 50 years of age without metabolic syndrome, regardless of diabetes status, had the lowest CHD prevalence. The prevalence of CHD markedly increased with the presence of metabolic syndrome. Among people with diabetes, the prevalence of metabolic syndrome was very high, and those with diabetes and metabolic syndrome had the highest prevalence of CHD. 2 figures. 4 tables. 31 references.

- **Heart Disease and Diabetes**

  Source: Clinical Diabetes. 21(1): 10. January 2003.

  Contact: Available from American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) 232-3472. Website: www.diabetes.org.

  Summary: This brief fact sheet reminds readers of the connection between heart disease and diabetes. The fact sheet notes that many conditions that increase one's changes of getting heart disease are more common in people with diabetes. These conditions include cholesterol problems, high blood pressure (hypertension), overweight, and blood clotting problems. Heart attacks, known in the medical community as myocardial infarctions, are one of the most common heart conditions. For most people, a heart condition leads to symptoms such as chest pain or pressure, jaw pain, arm pain, shortness of breath, sweating, and pounding heartbeat. However, many people with diabetes and heart disease do not notice any symptoms at all. This is called silent ischemia. Silent ischemia is very dangerous because it may prevent patients with heart problems from seeking medical care and getting early treatment. The fact sheet lists common risk factors for heart disease, and encourages readers with those risk factors to be tested.

- **Prospective Study of Obesity and Risk of Coronary Heart Disease Among Diabetic Women**

  Source: Diabetes Care. 25(7): 1142-1148. July 2002.

Contact: Available from American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) 232-3472. Website: www.diabetes.org.

Summary: This article reports on a study undertaken to examine the relationship of obesity, measured as BMI, and weight change to incidence of coronary heart disease (CHD) among women with diabetes. The authors followed 5,897 women with type 2 diabetes in the Nurses' Health Study for up to 20 years. Women were aged 40 to 74 years and had no history of cardiovascular disease or cancer at the beginning of the follow up period. During follow up, the authors document 418 incident cases of CHD (236 of nonfatal myocardial infarction and 182 of fatal CHD). After adjustment for age, smoking, and other coronary risk factors, current BMI (body mass index) was strongly associated with increased risk of CHD among women with diabetes. Increasing BMI values from age 18 years to 1976, before diagnosis of diabetes, were also positively associated with risk of CHD. Weight gain before the diagnosis of diabetes was related to increased risk of CHD. In contrast, weight change after diagnosis of diabetes was not associated with risk of CHD. The authors conclude that these findings provide strong evidence that obesity and weight gain before diagnosis of diabetes are associated with future risk of CHD among women with type 2 diabetes. 1 figure. 2 tables. 34 references.

- **Heart Disease: A Cardiologist's POV**

  Source: Diabetes Forecast. 54(9): 38-40. September 2001.

  Contact: Available from American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) 232-3472. Website: www.diabetes.org.

  Summary: This article, written by a cardiologist with a family history of diabetes, discusses the personal events that led to his interest in the cardiovascular effects of diabetes. The article begins with the author's recollections of his family's attitude about his grandfather's diabetes. This is followed by an account of his father's problems with high blood glucose and its effects on his heart. In addition, the author recounts his own experience with the effects of high blood glucose, its effects on his body, and his realization of the connection between diabetes and heart problems.

Federally Funded Research on Heart Disease

The U.S. Government supports a variety of research studies relating to heart disease. These studies are tracked by the Office of Extramural Research at the National Institutes of Health.[2] CRISP (Computerized Retrieval of Information on Scientific Projects) is a searchable database of federally funded biomedical research projects conducted at universities, hospitals, and other institutions.

Search the CRISP Web site at **http://crisp.cit.nih.gov/crisp/crisp_query.generate_screen**. You will have the option to perform targeted searches by various criteria, including geography, date, and topics related to heart disease.

For most of the studies, the agencies reporting into CRISP provide summaries or abstracts. As opposed to clinical trial research using patients, many federally funded studies use

[2] Healthcare projects are funded by the National Institutes of Health (NIH), Substance Abuse and Mental Health Services (SAMHSA), Health Resources and Services Administration (HRSA), Food and Drug Administration (FDA), Centers for Disease Control and Prevention (CDCP), Agency for Healthcare Research and Quality (AHRQ), and Office of Assistant Secretary of Health (OASH).

animals or simulated models to explore heart disease. The following is typical of the type of information found when searching the CRISP database for heart disease:

- **Project Title: 3D MYOCARDIAL STRAIN VECTORS IN ISCHEMIC HEART DISEASE**

  Principal Investigator & Institution: Cupps, Brian P. Surgery; Washington University Lindell and Skinker Blvd St. Louis, MO 63130

  Timing: Fiscal Year 2003; Project Start 01-AUG-2003; Project End 31-JUL-2007

  Summary: (provided by applicant): Directed intervention (coronary artery bypass surgery; intra-coronary stent placement) in the treatment of coronary artery disease is costly in patient morbidity, mortality and U.S. health care dollars. Methodologies that can improve the accuracy of application of these modalities deserve aggressive investigation. Magnetic Resonance Imaging (MRI) combined with radiofrequency tissue-tagging can non-invasively lay down transmural crosshatched grids in the myocardium that can be tracked throughout the cardiac cycle by MRI. Computer-based analysis techniques, developed in our laboratory, can track the grid intersections and generate regional point displacements and transmural 3D myocardial "strain" maps. Strain is a normalized description of direction and degree of myocardial point movement. From these 3D strain maps we can determine the direction of principal strain vectors at every point throughout the left ventricle. We hypothesize, based on our preliminary data, that the direction of these vectors is very sensitive to the regionally-varying influence of coronary artery disease on contractile function and can introduce an unprecedented level of objective quantification and accurate regional localization of ischemia. These strain vectors will be utilized in a familiar testing algorithm: 1) baseline strain vector determinations are made, and 2) repeat determinations are made after low-dose Dobutamine. Areas with abnormal resting vectors are examined at low dose Dobutamine to see if they "recruit," suggesting viability and potential for benefit from coronary intervention. The hypotheses do not test this well established algorithm, but rather the capability of these techniques to bring accurate regional localization and objective quantification to the output data from the algorithm, and thus improve accuracy of application of directed coronary intervention.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ACE INHIBITION IN SINGLE VENTRICLE/PULMON. HYPERTENSION**

  Principal Investigator & Institution: Gersony, Welton M. Pediatrics; Columbia University Health Sciences New York, NY 10032

  Timing: Fiscal Year 2001; Project Start 05-SEP-2001; Project End 31-AUG-2006

  Summary: (provided by applicant) The overall goal of this application is to examine treatment modalities which may improve the clinical care of two groups of patients with congenital heart disease: infants born with a single ventricle supplying blood flow to the lungs and body and children with pulmonary hypertension associated with congenital **heart disease.** The primary hypothesis in infants with single ventricle is that chronic angiotensin converting enzyme (ACE) inhibition favorably modifies the ventricular remodeling response to volume overload and improves ventricular function over the first year of life. Serial changes in ventricular geometry will be assessed using magnetic resonance imaging and compared with measurements of systolic and diastolic function, including the pressure/volume relation and the Tei index, and clinical outcome measures including post-operative course and changes in the Ross? heart failure

classification. The beneficial effect of ACE inhibition is expected to occur prior to and following volume unloading surgery with the bidirectional Glenn shunt or hemi-Fontan. The primary hypothesis of the study in congenital **heart disease** associated with pulmonary hypertension is that the effect of long-term treatment with an oral prostacyclin analogue or an oral endothelin receptor blocker has a salutory effect on exercise capacity, longevity, and quality of life. It will also be determined whether any of these patients carry a defect of the primary pulmonary hypertension-1 gene. Each of these studies could potentially lead to a significant improvement in prognosis: in the single ventricle group by preventing a long-term deterioration in ventricular function and in the pulmonary hypertension patients by improving quality of life and survival without transplantation.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: AGING, ESTROGEN, HSPS AND MYOCARDIAL ISCHEMIA**

  Principal Investigator & Institution: Knowlton, Anne A. Medicine; Baylor College of Medicine 1 Baylor Plaza Houston, TX 77030

  Timing: Fiscal Year 2001; Project Start 15-MAY-2001; Project End 30-JUN-2002

  Summary: (Adapted from the Investigator's abstract): The heat shock proteins (HSPs) are an important family of endogenous, protective proteins found in all tissues. In the heart, HSP72, the inducible HSP70, is increased by ischemia and is cardioprotective. Overexpression of other HSPs also protects against cardiac injury. Aging results in a decreased heat shock response in male rats; the effect on females in unknown. Women have less cardiovascular disease than men, but postmenopausal women have an increased incidence of **heart disease.** Although some of estrogen's protective effects can be explained by changes in lipoprotein profiles, other, unknown factors are involved. Studies in the Principal Investigator's laboratory have shown that estrogen increases levels of HSPs in male rat cardiac myocytes. We hypothesize that, as estrogen increases HSP expression and estrogen protects against CV disease, the decrease in estrogen with aging is associated with a decrease in HSP expression, and contributes to the increased susceptibility of the heart to injury. We propose to study the interaction of estrogen, HSPs and aging in three Specific Aims: 1 Determine the effect of aging versus hormones (17beta-estradiol and progesterone) on heat shock proteins in female rats. These experiments will test 2 hypotheses: 1. Estrogen affects HSP levels in the rat; and 2. Estrogen can increase HSP expression in aging. The expression of HSPs decreases in a number of settings with aging, and these experiments will address the importance of estrogen in this blunting of the heat shock (stress) response. 2. Determine the mechanism of selective induction of HSP72 by estrogen, and whether this is protective. These studies will investigate the mechanisms controlling HSP expression in the cardiac myocyte. E-Determine whether hormone replacement can increase HSP levels in vivo and protect against myocardial ischemia in aging and adult rats. This work will expand on the preliminary findings that estrogen increases HSP levels, and extend them to the in vivo setting of myocardial ischemia. These studies will address the important question of whether the heat shock response is blunted with aging or hormone changes in females, and provide new information about the effects of aging and hormone replacement on the response to ischemia. The long-term goal of this research initiative is to understand gender differences in the HSPs and changes in the HSPs with aging in the heart.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ALCOHOL INDUCED IMMUNOMODULATION--RETROVIRAL CARDIOPATH**

  Principal Investigator & Institution: Watson, Ronald R. Professor; None; University of Arizona P O Box 3308 Tucson, AZ 857223308

  Timing: Fiscal Year 2001; Project Start 03-JUL-1999; Project End 30-JUN-2004

  Summary: Cardiomyopathy and left ventricular dysfunction are prevalent in people with AIDS or chronic alcohol use. However almost nothing is known of the combined effects of retroviral infection plus alcohol on **heart disease.** Our murine retrovirus infection mimics much of the cytokine dysregulation found during HIV infection, prompting inflammatory damage for cardiac toxicity. We found that alcohol consumption exacerbated many immune, oxidative, and nutritional defects due to murine retrovirus infection. We found that alcohol + retrovirus exposure was particularly toxic, increasing Th2 and reducing Th1 cytokines, dramatically lowering cardiac vitamin E, increasing oxidation of cardiac lipids and synergistically promoting severe heart damage due to Coxsackie B3 infection. Our overall hypothesis is that the combination of ethanol + retroviral infection induces immune dysfunction and oxidation for increased cardiovascular disease. These effects should promote growth and pathogenesis of cardiotrophic pathogens. This hypothesis will be investigated using Coxsackie B infection of mice during retrovirus and/or ethanol exposure. Left ventricular function will be quantitated in vivo with interventricular catheter to define the ventricular dysfunction and characterize the effects of alcohol. We will determine the contribution of the inflammatory response, induced by alcohol and/or retroviral exposure, to myocardial ischemic events and infarctions. We will assert the immune mechanisms involving PMNs in amplifying ischemic injury during cocaine plus retroviral exposure. We will assess leukocyte adhesion and localization, platelet-leukocyte interactions, and blocking these cells' function with drugs to understand their role in ischemia and heart pump dysfunction induced by cocaine and/or retrovirus exposure. We will assert the cardiotoxic effects of alcohol exposure prior to as well as post retrovirus infection. Our model studies will increase understanding of etiology of alcohol + retrovirus-induced immune dysfunction in cardiac pathology. We will limit the deleterious cardiac effects of retroviral infection and alcohol exposure with our proven methods that prevented much of the cytokine dysregulation and oxidative damage to the heart: T-cell receptor Vbeta 8.1, multiple antioxidant, and vitamin E supplementation. We hypothesize that our treatments will restrict cytokine dysregulation and thus cardiotoxicity in retrovirally-infected, alcohol-exposed mice. Such studies could provide the basis for their application to reduce **heart disease** in alcohol-abusing, HIV-infected patients.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ANXIETY & VAGAL CONTROL OF THE HEART IN CORONARY DISEASE**

  Principal Investigator & Institution: Watkins, Lana L. Psychiatry; Duke University Durham, NC 27706

  Timing: Fiscal Year 2001; Project Start 01-JUN-1999; Project End 31-MAY-2003

  Summary: Coronary **heart disease** continues to be the leading cause of death in the United States, despite risk factor reduction and technological advances in treatment options. Prospective studies implicate chronic anxiety as an independent risk factor for fatal coronary **heart disease.** In particular, anxiety increases the risk of sudden cardiac death substantially. The primary objective of the proposed research is to examine the

role of reduced vagal control of heart rate in the increased risk of cardiac mortality associated with anxiety in a population with established coronary artery disease (CAD). A second objective is to determine whether the effects of anxiety are independent of the effects of depression. Nine hundred and fifty CAD patients will be recruited for this study from patients hospitalized for elective cardiac catheterization. Anxiety will be measured by the Hospital Anxiety Scale, the Spielberger Trait Anxiety Inventory, and the Crown-Crisp Phobic Anxiety Scale. Symptoms of depression will be measured by the Montgomery-Asberg Depression Rating Scale, the Hospital Depression Scale, and the Beck Depression Inventory. Vagal control of heart rate will be determined using power spectral analysis to measure two indices of vagal control: baroreceptor-mediated vagal reflex cardiac control, and respiratory sinus arrhythmia. Patients will be followed at 6 months, l year, 2 years, and 3 years postcatheterization, and cardiac mortality data will be obtained, including non-sudden and sudden cardiac death. The data generated by this study will be used to examine the involvement of impaired vagal cardiac control in the risk of fatal coronary **heart disease** and sudden cardiac death associated with anxiety. Specifically, the proposed study will examine: (1) the relationship between anxiety and cardiac mortality; (2) the relationship between anxiety and vagal control; (3) the role played by reduced vagal control in mediating anxiety-related risk; and (4) the relationship between depression, vagal control and cardiac risk. Findings of a relationship between anxiety, reduced vagal control and sudden cardiac death would suggest the potential importance of early intervention in cardiac patients with anxiety disorders and would underscore the benefit of aggressive monitoring of arrhythmias in this population, which may ultimately translate to reduced mortality rates.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: AZITHROMYCIN AND CORONARY EVENTS**

  Principal Investigator & Institution: Cohn, Peter; State University New York Stony Brook Stony Brook, NY 11794

  Timing: Fiscal Year 2001

  Summary: This study is to determine whether the antibotic, azithromycin, decreases the risk of having a heart attack or other complication related to coronary artery disease in people with **heart disease.** Azithromycin is an antibiotic that is used to treat infections with a bacteria called Chlamydia pneumoniae. Some studies suggest that Chlamydia pneumoniae infection may be a risk factor for heart attacks and other complications related to coronary artery disease. Chlamydia pneumoniae is spread from person-to-person by sneezing and coughing (unlike a related bacteria, Chlamydia trachomatis, it is not a sexually transmitted disease). Studies show that people who have been infected with Chlamydia pneumoniae in the past have a higher chance of having a heart attack. Other studies show that Chlamydia pnreumoniae is present in the plaque, or material blocking the heart vessel, in many people with **heart disease.**

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: BETA-RECEPTORS AND CARDIOVASCULAR PHARMACOGENOMICS**

  Principal Investigator & Institution: Johnson, Julie A. Professor of Pharmacy Practice & Medicin; Pharmacy Practice; University of Florida Gainesville, FL 32611

  Timing: Fiscal Year 2002; Project Start 01-JUL-2002; Project End 30-JUN-2007

  Summary: (provided by applicant): Pharmacogenetics/pharmacogenomics is a research field aimed at helping to describe the genetic contribution to variability in drug efficacy

and toxicity. The focus of the candidate?s research program is to determine the impact of beta-adrenergic receptor (betaAR) polymorphisms on drug response and cardiovascular disease. This broad research objective will be carried out through several studies. The first set of studies is focused in hypertension (HTN), which aim to test the hypotheses that the betaAR genes: a) are associated with HTN; b) are disease modifying in HTN, with a particular focus on the nocturnal blood pressure decline causing individuals to be either nocturnal "dippers" or "nondippers"; and c) are important determinants of the antihypertensive response to beta-blockers. The latter aim will be tested in a small population, with focus on the blood pressure, and also through a pharmacogenetic substudy of the INVEST trial, a 22,000 patient outcomes trial in patients with hypertension and documented ischemic **heart disease.** The second group of studies focus on beta-blocker pharmacogenetics in heart failure. Beta-blockers have been recently documented to prolong survival and slow disease progression in heart failure patients. These outcome benefits are thought to be associated with the "reverse remodeling effect" of the beta-blockers on the left ventricle (LV), which result in increases in ejection fraction, reductions in LV wall thickness and mass, and returning the ventricle to a more ellipitical shape. Despite these clear benefits, beta-blockers must be used cautiously during the titration phase to avoid worsening of heart failure, specifically, starting with very low doses, with close monitoring and cautious dose titration. These studies are focused on testing the hypotheses that certain polymorphisms of the beta1AR are associated with relatively poor initial tolerability of beta-blockers, and also with the most dramatic effects of "reverse remodeling" of the left ventricle. Additional patient-oriented research studies that will be conducted as part of this research career award include a study of dobutamine pharmacogenetics, and associations between the PAR genes and obesity or coronary microvascular dysfunction. The proposed studies are important because they will enhance our understanding of the genetic basis of various cardiovascular diseases, and will provide insight into genetic factors that influence response to drugs that act at the betaAR. The pharmacogenetic studies are significant in that they may provide information that will allow the drugs of interest to be targeted to the patients most likely to derive the greatest benefit from such therapy, thus leading to better individualization of therapy, and improved patient outcomes. These studies are also important as they provide excellent research training opportunities for clinicians. The proposed Research Career Award is important to the candidate?s career development in that it will provide sufficient protected time for the candidate to successfully complete the ongoing and planned studies described herein, will enhance the training of fellows in the emerging area of pharmacogenomics, and will allow for the continued growth of the candidate?s patient-oriented research program.

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- **Project Title: BONE MARROW ANGIOBLASTS FOR MYOCARDIAL VASCULOGENESIS**

  Principal Investigator & Institution: Itescu, Silviu; Assistant Professor; Surgery; Columbia University Health Sciences New York, NY 10032

  Timing: Fiscal Year 2002; Project Start 15-MAY-2002; Project End 30-APR-2007

  Summary: Congestive heart failure remains a major public health problem, and is frequently the result of left ventricular remodeling after myocardial infarction. We have recently shown that human adult bone marrow contains endothelial progenitors with phenotypic and functional characteristics of embryonic hemangioblasts, and that these can be used to induce vasculogenesis after experimental myocardial infarction. This results in decreased apoptosis of hypertrophied myocytes, long- term salvage and

survival of viable myocardium, reduction in collagen deposition, and sustained improvement in cardiac function. Thus, the use of cytokine-mobilized autologous human bone marrow-derived angioblasts offers the potential to significantly reduce morbidity and mortality associated with left ventricular remodeling. However, a number of studies have provided evidence that the ability of bone marrow-derived stem cells to respond to environmental demands such as injury, disease or other physiologic stimuli, may be compromised during aging. In the first part of this proposal we will investigate the relationship between increasing age or progression of ischemic **heart disease** and changes in the number, phenotypic characteristics, and in vitro and in vivo biologic properties of adult bone marrow-derived endothelial progenitors, or angioblasts. For these studies, 32 patients selected on the basis of incremental increase in age, and with a history of myocardial infarction (within previous 6-12 months with no evidence of heart failure, or greater than 5 years earlier and with heart failure), will be studied cross-sectionally and longitudinally. Phenotypic studies will be performed by flow cytometry of human bone marrow-derived cells obtained by leukapheresis after G-CSF mobilization. In vivo functional studies will involve intravenous injection of human angioblasts into athymic nude rats with experimental myocardial infarction. In the second part of this proposal we will seek to develop complementary strategies to augment the effects of angioblast- dependent vasculogenesis on cardiac function. We will first investigate whether angioblast migration to the infarct bed is regulated by chemokine-dependent interactions. We will then seek to increase angioblast trafficking to the heart and subsequent vasculogenesis by manipulating such interactions through use of monoclonal antibodies against chemokine receptors and induction of chemokine expression in the heart. Additional aims will be to evaluate whether the improvement in myocardial function obtained following angioblast-dependent vasculogenesis involves induction of myocyte proliferation/regeneration, and whether concomitant use of autologous mesenchymal stem cells or pharmacologic agents may provide synergistic, additive benefit. We believe that gaining an understanding of these issues will prove to be of critical importance in order to be able to rationally design and develop strategies for human clinical trials using autologous bone marrow-derived endothelial progenitors in the treatment of acute and chronic **heart disease.**

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- **Project Title: BRIDGING GENES AND HEART DISEASE IN DOWNS SYNDROME**

  Principal Investigator & Institution: Korenberg, Julie R. Professor; University of Texas Sw Med Ctr/Dallas Dallas, TX 753909105

  Timing: Fiscal Year 2001

  Summary: Down syndrome (DS) is a major cause of congenital **heart disease** (CHD), of which most is related to defective morphogenesis of the endocardial cushions (EC). Although the embryologic processes underlying cushion morphogenesis are elegantly described, the signalling pathways that lead from genes to defects are largely unknown. The ultimate goal of the research described in this proposal is to define the gen(s) responsible for DS-CHD, to define its role in cushion morphogenesis and to provide models in which to modify its expression. Previous work by this group has defined a DS-CHD region based on human and mouse models and has generated a sequence ready contig, with 300 kb of finished sequence and the remainder by 1/1999, and transcriptional map of 30 cDNAs in the 4 Mb candidate region on chromosome 21, as well as sequencing and characterization of a likely candidate., DS-CAM. Analysis of the human and mouse DS CAM clones reveal a highly conserved novel class of cell adhesion molecules (CAM) of the Ig superfamily with ten Ig2 and six fibronectin

domains and both extracellular and transmembrane forms. Expression in the endocardial cushions, neurons, neural crest and other sites of epithelial induction, promoter sites for known cardiogenic transcription factors (Mef2), combine with its map position with a small DS-CHD region, to make DS Cam a likely candidate. Not all mouse models of partial trisomy 16 including the DS-CHD region develop **heart disease.** To elucidate the role of DS-CAM in DS-CHD, the role of compartment specific expression (myocardium, endocardium, neural crest), mouse models of DS-CAM compartment specific over-expression (myocardium, endocardium, neural crest), mouse models of DS-CAM compartment specific over-expression and lack of expression will be generated as will models to address the possible contribution of the chromosome 21 gene for collagen VIa1/a2 located on MMU 10. We propose 4 aims; I and II narrow the region and examine human expression; III-V (in collaboration with the UCSD Mouse Core and Project 1) use mouse models to test the hypothesis that DS-CAM is the gene for DS-CHD, and VI defines the remainder of genes in the DS-CHD region. By defining the role of DS-CAM and its regulatory and interacting molecules, this work will provide the candidate causes for isolated AVSD, VSD, ASD and PS as well as other forms of monogenic CHD. Understanding DS CAM will provide insight into pathways on normal EC morphogenesis and their maldevelopment that cause the majority of deaths due to congenital anomalies.

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- **Project Title: CALCIUM REGULATION IN THE DIABETIC HEART**

  Principal Investigator & Institution: Davidoff, Amy J. Assistant Professor; Pharmacology; University of New England 605 Pool Rd Biddeford, ME 04005

  Timing: Fiscal Year 2001; Project Start 30-SEP-1997; Project End 31-AUG-2002

  Summary: Heart disease is the leading cause of death in diabetic patients, and considerable evidence is now available to support the existence of a specific diabetic cardiomyopathy that is independent of coronary artery disease and hypertension. Functional and biochemical data acquired from multicellular cardiac preparations of diabetic animals support the view that cellular mechanisms controlling cytosolic Ca2+ on a beat-to-beat basis are abnormal and contribute to impaired relaxation. The goal of this project is to characterize diabetes-induced changes in the expression and function of Ca2+ regulating proteins in isolated cardiac myocytes, and to determine the role of hyperglycemia in the pathogenesis and pathophysiology of diabetic cardiomyopathy. To test the hypothesis that abnormal Ca2+ handling occurs at the single cell level, biophysical assessment of excitation-contraction coupling will be carried out in ventricular myocytes isolated from diabetic rats. Voltage clamp techniques will be used to determine whole-cell Ca2+ and Na/Ca exchange currents as a measure of sarcolemmal L-type Ca2+ channel and Na/Ca exchanger function. Video edge-detection and Ca2+ fluorescence measurements (fura-2) will be used as additional means to evaluate Na/Ca exchange and to analyze SR Ca2+ ATPase and ryanodine receptor function. Abundance of mRNA for these Ca2+ regulating proteins will be determined using northern blot analysis and amount of protein will be assessed by western blot analyses. To test the hypothesis that hyperglycemia influences the expression and function of these Ca2+ regulating proteins, isolated ventricular myocytes from both diabetic and nondiabetic animals will be maintained in a "diabetic-like" medium (high glucose) in short-term primary culture. We will determine the specific role of hyperglycemia on expression, modification and function of calcium regulating proteins. Preliminary data show that within days, normal myocytes cultured in the "diabetic-like" medium exhibit prolonged relaxation, prolonged action potential durations, and slowed

cytosolic Ca2+ clearing, similar to that of diabetic myocytes. These experiments will provide important new insights into the pathogenesis and early events of diabetic cardiomyopathy. Taken together, these experiments will resolve the role of Ca2+ regulating proteins in diabetic cardiomyopathy by studying their function and expression at the single cell level. The adult myocyte culture system provides a well-defined method from elucidating fundamental mechanisms of high glucose that may contribute to the development of myocyte dysfunction in diabetes.

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- **Project Title: CANONICAL ANALYSIS OF #30 CARDIAC MOTION FROM TAGGED MRI**

  Principal Investigator & Institution: Metaxas, Dimitris N. Professor of Computer and Information Sc; Computer and Information Scis; University of Pennsylvania 3451 Walnut Street Philadelphia, PA 19104

  Timing: Fiscal Year 2001; Project Start 01-JUN-1998; Project End 31-MAY-2003

  Summary: (Adapted from the applicant's abstract): **Heart disease,** a major cause of morbidity and mortality in the Western World, generally leads to abnormalities of heart wall motion. However, there are still major difficulties in clinical assessment of heart wall disease due to the use of conventional imaging techniques (e.g., CT, conventional MRI), the lack of sufficient resolution in the extracted data, the absence of computational techniques for automatic extraction of the three-dimensional heart wall motion parameters in a way that is "useful" to physicians, and the absence of a database of normal patients against which normal and abnormal hearts can be compared. The aim of this proposal is to use the recently developed at the University of Pennsylvania, magnetic resonance imaging (MRI) technique based on magnetic tagging ("SPAMM") for modeling and clinically analyzing the cardiac motion. In particular this proposal aims to: 1) develop methods for ventricular analysis and modeling, 2) apply the methods to a combination of previously acquired normal subject data and some additional data in order to construct a novel canonical heart motion model, including dependence on age, gender, race and body size, 3) incorporate the resulting normal database representation in the analysis and modeling tools program to create for an individual subject a 3D visualization of their heart displaying those areas which move abnormally, and 4) use the tool developed in #3 and apply it to a limited series of patients as a preliminary assessment of the utility of these methods and as a guide on how to improve the analysis and display methods. The hypothesis is that due to this type of data (MRI-SPAMM) which shows a much greater degree of precision in 3D, we will be able to reliably distinguish in a quantitative way the above types of disease that are expected to affect cardiac function in characteristic ways. We will test this hypothesis by an already available small representative series of patients, each with a well-defined heart condition. The results of the analysis will be tested against all other available evaluations of the patients studied.

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- **Project Title: CARDIAC AMYLOIDOSIS IN AGING AFRICAN AMERICANS**

  Principal Investigator & Institution: Buxbaum, Joel N. Professor; Scripps Research Institute 10550 N Torrey Pines Rd San Diego, CA 920371000

  Timing: Fiscal Year 2001; Project Start 01-JUL-2001; Project End 30-JUN-2006

  Summary: (Verbatim from the application): If factors related to access to care are not considered, the increased mortality from cardiovascular disease in African-Americans

relative to other groups is due to the increased frequency of some diseases, a qualitatively different cardiac response to disorders affecting all ethnic groups and a relatively poor response to treatment of congestive heart failure. An undiagnosed, coexistent, relatively prevalent, treatment-resistant cardiomyopathy is a possible partial explanation. Late onset amyloidotic cardiomyopathy is fourfold more common in African-Americans than Caucasians. It causes congestive heart failure and arrhythmias, however these features are relatively non-specific and the clinical diagnosis is not always obvious. Digoxin and calcium channel blockers are toxic in patients with amyloid, thus, treatment of concomitant **heart disease** of other etiologies may be compromised; moreover misdiagnosis of amyloid **heart disease** may result in possibly harmful therapy. Unrecognized amyloidosis, in individuals over age 60, could contribute to some of the refractoriness seen in studies of congestive heart failure and to the higher cardiovascular morbidity and mortality in African-Americans. Our proposal examines a genetically determined form of late-onset amyloidosis due to a substitution of ILE for VAL at position 122 in the serum protein transthyretin (TTR). Approximately 4% of African-Americans are heterozygous for the allele that has an absolute risk for anatomic amyloid deposition after age 60 resulting in 154,000 African-Americans with some degree of cardiac amyloidosis. In a collaborative effort with two studies of cardiovascular risk in the community (ARIC and CFIS), with a combined African-American cohort of 5200, we will test the hypothesis that heterozygosity for the amyloidogenic allele is associated with clinical evidence of cardiac amyloidosis and a related increase in mortality. We will also assess the role of the allele in clinical **heart disease** by determining its prevalence in a cohort of African-American veterans, over 60, who are recognized as having **heart disease,** although their providers have not considered amyloidosis as a specific diagnosis. We will characterize the natural history of late onset cardiac amyloidosis in African-Americans, define its role in cardiovascular morbidity and mortality in this ethnic group and define guidelines for supportive treatment at present and specific therapy when available.

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- **Project Title: CARDIAC ESTROGEN RECEPTORS AND MI-- MOUSE MODELS**

  Principal Investigator & Institution: Karas, Richard H.; New England Medical Center Hospitals 750 Washington St Boston, MA 021111533

  Timing: Fiscal Year 2001

  Summary: Ischemic cardiovascular disease, specifically myocardial infarction (MI), is the leading cause of morbidity and mortality in Western society. There are clear gender-based differences in cardiac function and electrophysiology that influence the two major sequelae following MI: heart failure (NF), and susceptibility to arrhythmias. In women, MI is uncommon prior to menopause and postmenopausal estrogen replacement therapy decreases its incidence. These beneficial effects of estrogen have been attributed previously to indirect effects on classic risk factors. However, estrogen is now recognized to have direct effects on cardiovascular cells that are central to its beneficial effects on cardiovascular disease. Estrogen's effects are mediated by receptors that act as ligand-activated transcription factors. Two such receptors are currently known, ERALPHA, and the recently discovered ERbeta. We have developed and reported a series of novel murine models that provide unique tools for pursuing mechanistic questions related to the pathophysiology of cardiovascular diseases. These include a mouse model of MI and a mouse cardiac electrophysiology (EP) model. We now present preliminary data from murine models demonstrating: (a) gender- based differences in cardiac electrophysiology, susceptibility to ventricular arrhythmias, and post-MI cardiac

remodeling; (b) alteration in cardiac performance and electrophysiology in ERalpha (ERalphaKO) and ERbeta (ERbetaKO) knockout mice; (c) expression of both ERalpha and ERbeta and (d) ER-dependent effects on gene expression in cardiomyocytes. Taken together, these data identify the heart as a novel target organ for the direct effects of estrogen and form the basis for this Project's central hypothesis: Estrogen receptors regulate left ventricular remodeling and susceptibility to arrhythmias following myocardial infarction. We propose to test this hypothesis by pursuing two specific aims: Specific Aim 1: Investigation of the role of estrogen receptors an the effect of estrogen receptor modulators, including the SERM raloxifene, on left ventricular remodeling following myocardial infarction, using wild-type, ERalphaKO and ERbetaKO mice, and Specific Aim 2: Investigation of the role of estrogen receptors and the effect of estrogen receptor modulators on arrhythmias following myocardial infarction, using wild-type, ERalphaKO and ERbetaKO mice. These in vivo studies explore the molecular pathways that mediate gender-based and hormonal influences on cardiac remodeling and arrhythmias following MI. In addition, they provide a conceptual bridge from the genetic and clinical studies (Projects 1 and 2) to the other basic science projects (Projects 4 and 4) of this SCOR in ischemic **heart disease.**

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- **Project Title: CARDIAC FUNCTION AND METABOLISM IN HYPERTENSION**

  Principal Investigator & Institution: Davila-Roman, Victor G. Associate Professor of Medicine, Anesthe; Medicine; Washington University Lindell and Skinker Blvd St. Louis, MO 63130

  Timing: Fiscal Year 2002; Project Start 01-JUL-2002; Project End 30-JUN-2007

  Summary: This Midcareer Investigator Award in Patient-Oriented Research application focuses on training and mentoring of cardiovascular imaging techniques (i.e., echocardiography, cardiac MRI, and positron emission tomography) for the study of mechanisms of disease in patients with cardiovascular diseases. The goals are: 1) to continue the mentoring program of cardiology fellows established by the applicant, 2) to continue patient-oriented research in the applicant's laboratory, and 3) for the applicant to acquire additional training and skills in cardiac MRI. The applicant-PI has an established record of mentoring young investigators, and will use this grant as a mechanism to further mentoring of young investigators, and will use this grant as a mechanism to further the mentoring of young investigators in the use of non-invasive cardiovascular imaging techniques to answer clinically relevant questions through hypothesis-testing. Young trainees will be exposed to ongoing clinical investigations that use novel methods and techniques to explore mechanisms of human disease in a multi-disciplinary environment. The main themes of investigation of the applicant include elucidation of the relationship between myocardial blood flow, contractile function and metabolic function in various disease states such as hypertensive **heart disease** and heart failure. The applicant will continue to develop a clinical research program that builds upon recent developments in this area by our group and others. This multi- disciplinary approach to elucidate mechanisms of human disease in clinical medicine include the use of clinical and quantitative tools for hypothesis testing. Trainee support is obtained through several mechanisms: The Cardiovascular Division's NIH Training Grant, the NIH's Minority Investigator Research Supplements, the AHA, and private foundations (Fourjay, Dana, Doris Duke, and Robert Wood Johnson). Trainees will formulate hypothesis that require quantitative characterization of cardiovascular pathophysiology through the acquisition and processing of multi-imaging data, and test them through the use and application of biostatistical methods. The ultimate goal of the

PI is to expand the training program of cardi9logy fellows that want o become independent clinician-scientists. A funded research program that illustrates the research program for young investigators is described, to illustrate the applicant's involvement in mentoring in patient-oriented research.

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- **Project Title: CARDIAC MR OF SUBCLIN CVD: IMPACT OF AGE**

  Principal Investigator & Institution: Manning, Warren J. Assistant Professor; Beth Israel Deaconess Medical Center St 1005 Boston, MA 02215

  Timing: Fiscal Year 2001; Project Start 30-SEP-2001; Project End 31-AUG-2005

  Summary: (Verbatim from the Applicant's Abstract): Coronary **heart disease** and stroke are leading causes of mortality for men and women in the United States. Our current understanding of the pathogenesis of and the risk factors for cardiovascular disease (CVD) is derived largely from prospective studies of clinically overt disease. Unfortunately, clinical risk factors for CVD defined by these methods fail to predict a large proportion of CVD events, and some subjects at high clinical risk fail to develop overt disease. Subclinical disease precedes clinical events by years/decades but is difficult to quantify. For example, left ventricular hypertrophy (LVH) and aortic atherosclerosis are strong predictors of CVD events, but are difficult to accurately non-invasively quantify, especially among the elderly and overweight subjects (both growing populations in the U.S.). MRI perrnits accurate assessment of cardiac anatomy/function and subclinical aortic atherosclerosis. The underlying hypothesis of this proposal is that subclinical CVD is a precursor to overt CVD, and that MRI measures of subclinical aortic and cardiac anatomic disease are superior for the characterization of risk as compared with current measures of risk factors as well as more conventional imaging (e.g., carotid ultrasound, echo). Longitudinal/time-averaged indexes of all established risk factors for CVD have been collected in the Framingham Heart Study (FHS). These time-averaged indexes are stronger predictors of clinical CVD than single measures. In a Pilot study of 312 FHS Offspring subjects, MRI measures of LV mass were successfully acquired in a larger proportion of subjects than echo, and MR evidence of LVH and subclinical aortic disease correlated more strongly (than echo and carotid ultrasound measures) with these time-averaged indexes. Application of MRI methods in the FHS offers an opportunity to identify subclinical atherosclerosis and LVH in this well-characterized cohort and to relate these data with conventional imaging measures already acquired in this cohort. Importantly, the near-concurrent acquisition of brain MRI/neuropsychologic examination in the same FHS cohort offer the unique contemporaneous opportunity to examine subclinical cerebrovascular disease with MRI indexes of subclinical atherosclerosis. We propose to expand our Pilot study to perform heart and thoracic/abdominal aorta MRI studies in 2400 FHS participants to allow for identification of individual CVD risk factors for subclinical atherosclerosis. These population-based data will extend our knowledge of the distribution and severity of atherosclerosis in adult men and women and their relations to existing echo, carotid ultrasound and brain MRI measures. This study provides the rare opportunity to examine associations of quantitative MRI measures of aortic atherosclerosis and LVH with both cross-sectional and time-averaged measures of individual atherosclerotic risk factors (e.g., blood pressure, cigarette smoking, and cholesterol) and with novel inflammatory markers (e.g., C-reactive protein, MCP-1). Further, because the FHS consists of hundreds of sibships for which a DNA repository has been established, we propose to determine the heritability of MRI indexes of atherosclerosis and LVH, laying the groundwork for future genetic studies.

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- **Project Title: CARDIAC MYOCYTE APOPTOSIS--MECHANISM AND SIGNIFICANCE**

  Principal Investigator & Institution: Kitsis, Richard N. Professor; Medicine; Yeshiva University 500 W 185Th St New York, NY 10033

  Timing: Fiscal Year 2001; Project Start 01-AUG-1998; Project End 31-JUL-2003

  Summary: Numerous studies have demonstrated myocyte apoptosis during myocardial infarction, ischemia-reperfusion injury, and chronic heart failure. Despite these observations, the two most critical questions in the field remain unexplored: 1) What is the precise molecular mechanism of apoptosis in cardiac myocytes? 2) To what extent does myocyte apoptosis contribute to myocardial dysfunction in these disease states? The research program described herein addresses both of these interrelated questions. To facilitate a molecular genetic analysis, models of myocardial infarction and ischemia-reperfusion injury have been developed and characterized in the mouse. Using genetically altered mice, we have tested the necessity of proteins that mediate apoptosis in non-cardiac contexts for apoptosis during myocardial infarction. These studies have shown that one such protein, p53, while present in ischemic cardiac myocytes and sufficient to induce apoptosis in these cells, is not required for myocyte apoptosis. This result suggests that the apoptotic program in complex pathophysiologic states can be activated by multiple, redundant signaling pathways. In contrast, the caspases, a family of cysteine proteases, are components of the final common pathway for apoptosis in all metazoan cells from worm to mammal. Indeed we have shown that pharmacologic blockade of these enzymes markedly inhibits myocyte apoptosis during myocardial infarction in vivo. The potential significance of this result is two-fold: First, caspase inhibition may provide a direct means to determine the contribution of myocyte apoptosis to myocardial dysfunction. Second, caspase inhibition may provide a new therapeutic approach to ischemic **heart disease** and heart failure. We now propose to deepen our understanding of the mechanism and significance of cardiac myocyte apoptosis through the following specific aims: 1. To determine which caspases are expressed in adult cardiac myocytes and undergo proteolytic activation during myocardial infarction and ischemia-reperfusion injury. 2. To block myocyte apoptosis in these ischemic syndromes using caspase inhibition. Complementary pharmacologic (peptide pseudosubstrates) and transgenic (overexpression of a dominant caspase inhibitor) approaches will be employed. 3. To determine the contribution of apoptosis to changes in myocardial structure and function during and after infarction and ischemia-reperfusion injury. Using caspase inhibition, the contribution of myocyte apoptosis to infarct size, ventricular remodeling, and contractile dysfunction will be determined. These studies will increase our understanding of the mechanism of cardiac myocyte apoptosis and its role in the pathogenesis of ischemic **heart disease.**

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- **Project Title: CARDIOVASCULAR CELL AND GENE THERAPY CONFERENCE**

  Principal Investigator & Institution: Hajjar, Roger J. Assistant Professor of Medicine; Massachusetts General Hospital 55 Fruit St Boston, MA 02114

  Timing: Fiscal Year 2002; Project Start 01-JUN-2002; Project End 31-MAY-2005

  Summary: (provided by applicant): Cardiovascular disease is a major cause of morbidity and mortality in the United States. New treatments are being formulated based on a better understanding of the signaling pathways involved in the pathogenesis of

cardiovascular diseases. Furthermore cell replacement therapy has recently emerged as a novel way of correcting contractile and vascular deficiencies in cardiovascular diseases. The focus of this yearly symposium will be on the use of somatic gene transfer and cell therapy in cardiovascular diseases. Targeting genes to the heart through somatic gene transfer or transplanting stem cells have the potential to alter our approach to patients with cardiovascular diseases. Gene and cell therapy allow us to test hypotheses about mechanisms of disease, and, it is hoped, tailor therapy accordingly. This symposium will bring together scientists from industry, clinicians and basic scientists. It will be a multidisciplinary meeting that should bring together people who are beginning to have regular dialogues but whose traditions have been somewhat separate Through this combination of investigators with multidisciplinary backgrounds, diverse scientific perspectives will be brought into focus on gene and cell therapy. The conference will consist of cover nine separate sessions over two and a half days. The topics of the sessions are 1) Viral vectors, 2) Delivery approaches, 3) Lessons from development, 4) Cell therapy, 5) Targeting Ischemic **Heart Disease,** 6) Targeting hypertrophy and growth, 7) Targeting heart failure and arrhythmias, 8) Targeting vascular disease, and 9) NIH programs and regulatory issues.The conference will be organized on a yearly basis in April. All the logistics of the first conference along with speaker commitments have been completed and the assigned date of the first conference is April 8-20, 2002. The convergence of investigators from different fields which are typically separate will hopefully foster greater collaborative efforts in gene and cell therapy and provide better understanding and treatment modalities for cardiovascular diseases.

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- **Project Title: CARDIOVASCULAR DISEASE IN THE PIMA INDIANS**

  Principal Investigator & Institution: Howard, Barbara V. President; Medstar Research Institute Hyattsville, MD 20783

  Timing: Fiscal Year 2001; Project Start 30-SEP-1988; Project End 31-MAY-2005

  Summary: MedStar (formerly Medlantic) Research Institute proposes to continue its participation in the Strong Heart Study to manage the Arizona field center and the core laboratory. For the field center, this proposal describes methodology for 1) morbidity and mortality surveillance of the original Strong Heart Study cohort (1099 surviving out of 1500 original men and women ages 45-74 years in Phase I); 2) recruitment and examination of 30 families of at least 30 members, each 15 years and older; and 3) re-examination of the 900 family members from the Phase Ill pilot study. The Arizona field center comprises three American Indian communities: Gila River, Salt River, and Ak Chin. The Arizona center had a 71% recruitment rate in Phase I and 90%+ completion rates in Phases II and III. Morbidity and mortality surveillance obtained data on 99% of the participants. The core laboratory will provide accurate, reliable, stable, and comparable phenotypic measures of coronary **heart disease** risk factors in blood and urine samples. Measurements to be made for the family cohort include lipoprotein profile, glucose, HbA1c, insulin, LDL size, fibrinogen, PAI-1, apoE phenotype, apoB, apoA1, chemistry profile, and urinary albumin and creatinine. In addition, some exciting new markers of evolving importance in the etiology of atherosclerosis will be evaluated on stored baseline samples using a case-cohort design. sVCAM and endothelin-1 will be measured in approximately 400 definite cardiovascular disease cases and suitable controls. TSH also will be measured in these samples to allow evaluation of its role in cardiovascular disease in American Indians. The core laboratory will store blood, urine, and DNA in a safe and organized manner for effective inventory

so that the resources will be retrievable for other scientists and the American Indian communities. Laboratory performance during the previous exams has been excellent, with high completion rates and precision and accuracy exceeding those of most core laboratories.

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- **Project Title: CARDIOVASCULAR EFFECTS OF COCAINE**

  Principal Investigator & Institution: Knuepfer, Mark M. Professor; Pharmacological & Physiol Scis; St. Louis University St. Louis, MO 63110

  Timing: Fiscal Year 2001; Project Start 01-MAR-1989; Project End 28-FEB-2003

  Summary: Cocaine is still widely used despite increasing reports of apparent idiosyncratic myocardial toxicity. At present, we are unable to identify individuals at risk for cocaine-related cardiac disease. Individuals vary in their susceptibility to toxic responses yet no animal model for cocaine-induced cardiac disease had been described reflecting this variability. Using measurements of several cardiovascular variables (e.g. cardiac output, systemic vascular resistance, stroke volume, and heart rate), we have identified variability in response characteristics that is related to variability in cocaine-induced cardiomyopathies but not to pressor responses. In most of our studies, we have separated the population into two groups to facilitate our analysis using cardiac output (CO) responses. Cocaine administration elicits consistent decreases in CO in vascular responders (formerly named responders). Vascular responders have a greater incidence of ultrastructural myocardial abnormalities (eg., dilated sarcoplasmic reticulum, myofibrillar and mitochondrial abnormalities and focal myocytolysis) after repeated cocaine administration while these changes are less severe or absent in rats without a decrease in CO (mixed responders, formerly nonresponders). Vascular responders also have smaller increases in heart rate and greater increases in systemic vascular resistance (SVR). Several agents alter the CO and arterial pressure responses independently suggesting that different mechanisms are involved. In this application, we propose to focus on two aspects of our findings; the variability in cardiovascular and in cardiomyopathic responsiveness. First, we will determine the specific cause of the decrease in CO and enhanced increase in SVR by measuring specific parameters that could be responsible for the variability such as contractility, coronary and skeletal muscle vascular responsivity and sympathetic nerve activity. In addition, we will examine the relative contribution of parasympathetic and sympathetic tone before and after cocaine and the possible causes of differential cardiac sensitivity to adrenergic agents. These studies will define causes of the CO and SVR variability. Second, we will perform morphometry to characterize the ultrastructural alterations in the myocardium of cocaine-treated rats and compare these to catecholamine and CNS stimulation-induced cardiomyopathies. The causes of ultrastructural changes will be examined directly using selective antagonists and cardiac denervation. Our results will characterize the causes of differential sensitivity to cocaine-induced cardiovascular responses and myocardial disease and may provide specific treatments for patients sensitive to cocaine-induced cardiac disease. Furthermore, our studies offer a novel model by which individuals at greater risk for cocaine- or stress-related **heart disease** may be identified.

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- **Project Title: CARDIOVASCULAR HEALTH STUDY--ECHOCARDIOGRAPHY**

  Principal Investigator & Institution: Gottdiener, John S. Medicine; Georgetown University Washington, DC 20057

Timing: Fiscal Year 2001; Project Start 30-JUN-1993; Project End 31-MAY-2001

Summary: The Cardiovascular Health Study is a population-based, longitudinal study of risk factors for the development and progression of coronary **heart disease** and stroke in adults over the age of 65 years. Both risk factors established in middle-aged population and suspected risk factors are examined and include hypercholesterolemia, hypertension, glucose intolerance and diabetes, and cigarette smoking. Since atherosclerosis is prevalent in the elderly, the study focuses on factors thought to induce clinically overt disease. It does so in two ways: (1) It assesses the prediction of clinical disease from non-invasive measure of preclinical disease, such as carotid atherosclerosis, left ventricular impairment, and arrhythmias of episodes of myocardial ischemia. (2) Since cardiovascular events may occur in elderly people as a result of health or life circumstances which may have changed in the months preceding the event, the study contacts participants at frequent intervals to evaluate their status with respect to concurrent disease, social support networks, stressful life situations, diet, physical activity, and other risk factors. The study has three secondary objectives pertaining to the elderly populations: (1) to evaluate the factors associated with preclinical cardiovascular disease such as carotid atherosclerosis, left ventricular impairment and episodes of arrhythmia or myocardial ischemia; (2) to evaluate predictors of disability, institutionalization and mortality in participants who have coronary **heart disease** or stroke; and (3) to measure the utilization and impact of medical care services for coronary **heart disease** and stroke. Currently, risk associations are identified with clinical disease by the accumulation of events. Risk estimates are compared in subgroups of participants, such as women versus men, African-American versus Caucasian, those older versus younger than 75 years, or those with versus without prevalent clinical or subclinical disease. Risk estimates are compared in subtypes of disease, such as fatal versus non-fatal myocardial infarction, symptomatic versus silent myocardial ischemia, or fatal versus non-fatal stroke. Estimates are compared of longer-term (5-10 year) versus short-term (1-3 year) CVD risk. The study is also; determining whether presence or progression of subclinical disease (abnormalities detected non-invasively without signs or symptoms) are better predictors of clinical disease than traditional risk factors; identifying determinants of change in subclinical disease; identifying characteristics of subgroups at low risk for developing CVD (in whom preventive measures may be unnecessary). The contractor serves as the Echocardiography Reading Center. The duties of the Center include protocol development, instruction and supervision of the Field Centers in performance of echocardiography, measurement of parameters, and analysis and publication of data.

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- **Project Title: CARDIOVASOLOGY**

  Principal Investigator & Institution: Burnett, John C. Professor of Medicine & Physiology Direc; Mayo Clinic Rochester 200 1St St Sw Rochester, MN 55905

  Timing: Fiscal Year 2002; Project Start 01-JUL-1975; Project End 30-JUN-2006

  Summary: (Applicant?s Abstract) This renewal application represents a request for funding for years 26 to 30 of our training grant entitled Cardiovasology. This training program has been highly successful in preparing individuals for an independent research career in cardiovascular medicine and biology, and our current application is characterized by an increasing focus upon translational research that brings together an interdisciplinary team of established investigators. The proposed training program is focused upon the syndromes of heart failure and ischemic **heart disease.** These two interrelated themes were chosen based upon the current expertise of the faculty, the

central role played by these two disease entities in the US population and more specifically the patient population at the Mayo Clinic. An additional rationale for focusing upon heart failure and ischemic **heart disease** is that these are complex syndromes which involve multiple cell types, require multidisciplinary approaches and therefore provide a powerful mechanism to bring together the faculty and provide diverse research opportunities for trainees. The faculty consists of a collaborative and innovative team of basic scientists, physician-scientists, clinical investigators and population scientists with established track records in mentoring. Moreover, we have designed our competitive renewal to be in parallel with recommendations of the NHLBI "SPARKS" report and therefore our faculty incorporates genetics, cellular biology, research training at the tissue and whole animal level with human physiology and population sciences. It is the goal of our competitive renewal to train talented individuals in new biology and an emphasis upon innovative methodologies that will facilitate translation in the understanding of the pathophysiology of cardiovascular disease leading to novel diagnostics and therapeutics. With our current competitive renewal we propose a vibrant and highly talented multidisciplinary group of established investigators with documented mentoring skills and records. Current technologies represented in the training program faculty include molecular genetics, pharmacogenomics, functional genomics, epidemiology and outcomes research, gene therapy and transplantation biology which complement traditional departments of internal medicine, pediatrics, surgery, pharmacology, physiology, biochemistry and molecular biology. Thus, trainees are exposed to multiple technologies that are organized around the translational themes of heart failure and ischemic **heart disease.** Fundamental to this effort remains an unreserved commitment to diversity of trainees and faculty in addition to the commitment excellence. We believe that our competitive renewal lays down a strategy to continue this tradition of excellence in research training in cardiovascular biology and medicine.

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- **Project Title: CELLULAR RESPONSES TO STRESSORS OF CARDIOVASCULAR HEALTH**

  Principal Investigator & Institution: Flynn, Francis W. Professor; Psychology; University of Wyoming Box 3355, University Station Laramie, WY 82071

  Timing: Fiscal Year 2001; Project Start 15-SEP-2000; Project End 31-AUG-2005

  Summary: The scientific theme of this Center is the cellular responses to stressors that result in cardiovascular disease. Despite the enormous impact of heart diseases that kill more than half a million Americans each year, the systematic studies of the effects of various factors contributing to **heart disease** at the molecular level are lacking. This COBRE will pioneer the studies of global gene expression in progression of **heart disease** leading to myocardial infarction using DNA microarray technologies. The rat ischemia-reperfusion model system will be developed by the Center. Our principal objectives are to identify genes whose expression responds to hypoxia (ischemia) and reoxygenation (reperfusion) and to construct a gene expression database for this model. We will use our gene expression database to develop testable hypotheses within each area of interest to Center investigators. The areas of expertise of COBRE investigators are diverse and individual research projects will be carried out at various levels, from molecular through organismal. In addition to DNA microarray technologies, a number of common methodological approaches will be used, including confocal microscopy, protein overproduction, and protein structure-fiinction analysis. Defects in the hypoxia sensing proteins, accumulated DNA damage, and defective DNA repair pathways have

been linked to cardiac arrhythmias, ischemic neuronal damage, and hypertension. We propose to examine the mechanisms of hypoxia sensing and the structure of the PAS domain containing hypoxia sensors in prokaryotes and apply this knowledge to mammalian systems. The role of recombinational DNA repair pathways in the development of **heart disease** will be evaluated. Heat shock proteins, which appear to play an important role in protecting against cardiac damage caused by ischemia, also will be examined. The normal adaptive response of remaining myocardium surviving infarction is myocyte hypertrophy and reactive interstitial fibrosis. We propose to determine the nature and extent of hypertrophyinduced alterations in decorin-collagen interactions and collagen fibril architecture. Elevated salt intake is one factor contributing to development of hypertension and the progression of **heart disease.** We propose to identify the role of brain tachykinin neurotransmitters and in the control of salt intake and baroreflexes in normal and animal models of human essential hypertension. Collectively, the research proposals undertaken by this Center will elucidate cellular processes stimulated by stresses involved in the progression of cardiovascular disease.

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- **Project Title: CHOLESTEROL LOWERING BY NURSES TO CONTROL HEART DISEASE**

  Principal Investigator & Institution: Blumenthal, Roger S.; Johns Hopkins University 3400 N Charles St Baltimore, MD 21218

  Timing: Fiscal Year 2001

  Summary: The purpose of this study is to evaluate the effectiveness of a nurse-managed, case-management system of individualized lifestyle modification and pharmacologic intervention to manage lipid disorders in adults who have undergone coronary artery bypass graft surgery (CABG). Clinical trials have provided strong scientific evidence that lowering serum cholesterol will reduce morbidity and mortality from coronary **heart disease** (CHD) in patients with established CHD.1 Despite the clear benefits of cholesterol-lowering treatment, many patients with clinical evidence of atherosclerosis are not being treated effectively.2-4 Changes in the delivery of health care, including care for and after coronary events, mandate that we consider more effective and economical strategies for the management of lipid disorders in those with CHD so that the benefits of lipid lowering can be achieved outside of clinical drug trials. The primary aims of this study are to: 1. compare the effectiveness of a nurse-managed program for lipid modification with that of usual medical care in dyslipidemic adults who have undergone CABG in achieving the goal of a LDL-cholesterol 20% reduction in LDL-cholesterol; 2. determine the physiologic, behavioral, and demographic predictors of goal attainment; 3. determine the cost effectiveness of a nurse-managed lipid-lowering program; and 4. assess the impact of lipid-lowering therapy on patients quality of life. Our goal is to optimize lifestyle modification, thereby minimizing and improving the efficacy of pharmacologic intervention. We hypothesize that a significantly higher proportion of those who participate in the nurse-managed program for lipid modification will attain the goal for lipid lowering compared to those who receive usual care from their primary providers. Additionally, we propose that the intervention is a cost-effective model of delivering lipid-lowering therapy that lends itself to widespread application.

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- **Project Title: CIRCULATORY CONTROL DURING EXERCISE: EFFECT OF DISEASE**

  Principal Investigator & Institution: Smith, Scott A. Internal Medicine; University of Texas Sw Med Ctr/Dallas Dallas, TX 753909105

  Timing: Fiscal Year 2001; Project Start 01-MAR-2001

  Summary: (Applicant's abstract):Exercise capacity is reduced in patients with **heart disease** and hypertension limiting the therapeutic value of its prescription. The long-term goal of this proposal is to understand circulatory control during exercise mediated by intramuscular somatosensory input in chronic disease. The first objective is to develop a reproducible exercise model in which cardiovascular disease is readily induced. A decerebrate rat preparation will be used and the effect of mechanically and metabolically-sensitive hindlimb afferent activity on circulatory control will be investigated. The second objective is to characterize alterations in cardiovascular control during exercise after the development of heart failure. Myocardial infarction will be induced in rats via coronary artery ligation. Changes in heart rate, blood pressure, and renal sympathetic nerve activity will be measured in response to static muscle contraction and passive muscle stretch after decerebration. The third objective is to identify changes in circulatory regulation during exercise in hypertension using the methods presented above. A genetically engineered strain of spontaneously hypertensive rats will be employed and responses compared to a control group of normotensive rats. Results from these experiments will not only describe a novel animal preparation for the study of cardiovascular control by intramuscular neural mechanisms but will lead to an improved understanding of circulatory regulation during exercise after the manifestation of clinical pathologies.

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- **Project Title: CLINICAL TRIALS OF LOCAL PERCUTANEOUS DELIVERY OF BFGF IN CAD**

  Principal Investigator & Institution: Laham, Roger J. Associate Professor of Medicine; Harvard University (Medical School) Medical School Campus Boston, MA 02115

  Timing: Fiscal Year 2001; Project Start 01-APR-2001; Project End 02-APR-2001

  Summary: Ischemic coronary disease is the leading cause of morbidity and mortality in the Western world. Most available therapeutic approaches aim either at relieving symptoms by reducing myocardial oxygen demand, preventing further disease progression by modifying risk factors, restoring flow to a localized segment of the arterial tree (PTCA or CABG). Therapeutic angiogenesis may restore flow to the ischemic myocardium by creating new venues for blood flow. The purpose of the present investigation will be to examine the therapeutic potential of basic fibroblast growth factor (bFGF) in human ischemic **heart disease** using percutaneous intrapericardial delivery, define optimal outcome measures for clinical angiogenesis studies using a novel magnetic resonance imaging technique and Biosense electromechanical mapping, and explore novel growth factor deliver methods in animal models of myocardial ischemia, including intramyocardial delivery and gene therapy. We will conduct a clinical trial of therapeutic angiogenesis in patients with ischemic **heart disease** who are suboptimal candidates for standard revascularization strategies. This trial will examine the angiogenic efficacy of bFGF administered using a novel percutaneous subxyphoid intrapericardial delivery technique. We will investigate the effects of bFGF treatment on clinical parameters, left ventricular function, coronary angiography, and on the size and extent of myocardial ischemia using stress nuclear

perfusion scans. Biosense NOGA outcome variables in several ongoing clinical angiogenesis studies and laser myocardial revascularization studies comparing these two treatment strategies. In particular, we will validate two novel outcome measures: magnetic resonance imaging and Biosense NOGA mapping. Finally, we will develop novel delivery strategies in a porcine model of chronic myocardial ischemia and mouse matrigel and infarction models including intramyocardial delivery and gene therapy, and compare protein and gene therapy strategies for growth factor-induced angiogenesis. These novel delivery strategy, if successful, will be investigated clinically. These interrelated projects constitute a cohesive research program aimed at elucidating various aspects of therapeutic angiogenesis. Even though the problem, we wish to address, the techniques involved are necessarily broad, ranging from clinical trials, investigation of novel delivery strategies in animal models, and development of a standardized platforms for the conduction of future trials. This should lead to a novel approach to the treatment of ischemic **heart disease** and a better understanding of the mechanisms of growth- factor and laser induced "angiogenesis".

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- **Project Title: COMMUNITY SURVEILLANCE OF CARDIOVASCULAR DISEASE--RISK F**

  Principal Investigator & Institution: Arnett, Donna K. Associate Professor; Epidemiology; University of Minnesota Twin Cities 200 Oak Street Se Minneapolis, MN 554552070

  Timing: Fiscal Year 2001; Project Start 01-APR-1979; Project End 28-FEB-2004

  Summary: Data from the most recent survey (1995-97) of the Minnesota Heart Survey (MHS) indicate that previously favorable trends in cardiovascular risk factor levels are attenuating. While cigarette smoking and self-reported dietary fat intake continued to decline, mean body weight rose substantially and rapidly, physical activity decreased, and the previous decline in serum total cholesterol was no longer apparent. These trends may or may not continue in the future. As part of the Minnesota Heart Survey, we propose to conduct another population survey of 4,000 adults, ages 25-84 years in 2000-02, to detect current trends in cardiovascular disease risk factors, including serum lipids, blood pressure, cigarette smoking prevalence, dietary fat intake, obesity, diabetes, physical inactivity, fibrinogen, and serum vitamin E. The proposed survey will build upon four previous, independent cross-sectional surveys conducted in 1980-82, 1985-87, 1990-92, and 1995-97, which collectively examined 23,000 adults in the metropolitan Minneapolis-St. Paul (2.3 million residents in 1990). Using a sampling strategy identical to that of prior surveys, households will be randomly selected by a two-stage cluster design. A wide range of risk factors for cardiovascular disease will be measured using previously employed methods. Cohort and ecological analyses will link secular trends in risk factors to morbidity and mortality from coronary **heart disease,** congestive heart failure, and stroke within the same population. To estimate risk factor levels in children and adolescents, 1,000 youth ages 8-17, offspring of selected adults, will also be recruited and examined using youth-specific measurement instruments where appropriate (e.g., physical activity and smoking). New methodological studies are proposed to understand trends in reported dietary intake which are irreconcilable with trends in body weight and serum cholesterol. New information will be collected on measures of leisure time physical inactivity and diet validation. Serial ascertainment of risk factor levels in populations is crucial to understanding, predicting, and controlling population trends in cardiovascular disease. The Minnesota Heart Survey has been, and

continues to offer, a powerful resource to examine long-term trends in cardiovascular disease risk factors.

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- **Project Title: COMMUNITY SURVEILLANCE OF CHD AND SUDDEN DEATH (MHS)**

  Principal Investigator & Institution: Luepker, Russell V. Professor; Epidemiology; University of Minnesota Twin Cities 200 Oak Street Se Minneapolis, MN 554552070

  Timing: Fiscal Year 2001; Project Start 01-AUG-2000; Project End 30-JUN-2005

  Summary: The Minnesota Heart Survey (MHS) is among the few population- based longitudinal studies to monitor and explain trends in coronary **heart disease** (CHD) mortality and morbidity; the leading cause of death and disability in the United States. It encompasses a large and well defined community, the Minneapolis/St. Paul (Twin Cities) metropolitan area of Minnesota, comprising a population of 2.3 million (1990 census). For almost two decades, MHS has made contributions to: 1) understanding the components of the decline of coronary **heart disease** mortality including incidence rate, hospitalized attack rate, case fatality, and population levels of CHD risk factors; and 2) the methodology of disease surveillance in a time when classification and diagnostic technologies are constantly changing. In the last grant period, MHS morbidity and mortality surveillance has found: 1) a continued decline in age-adjusted CHD death rates through 1995 for both men and women; 2) continued improved survival of hospitalized AMI patients in the first half of the 1990's; 3) more modest declines of out-of-hospital sudden cardiac death during the 1990's resulting in an increasing preponderance of out-of-hospital compared to in-hospital CHD mortality; 4) declining rates of hospitalized AMI including incident and recurrent events for both men and women in the 1990's; 5) a modest change in event severity for AMI; 6) dramatic improvements in two year case fatality after validated hospitalized AMI; and 7) significant increases in the use of appropriate medications, diagnostic procedures and therapeutic procedures during AMI in the setting of dramatic declines in length of hospital stay. In this application, we propose to continue efficient MHS data collection in the following domains: 1) continue surveillance of coronary **heart disease** mortality through the year 2002; 2) monitoring trends in AMI occurrence and survival by surveillance of hospitalized AMI in the year 2000; 3) evaluation of the effect on AMI diagnosis of the widespread use of new, highly sensitive and specific biomarkers (troponins); and 4) evaluation of out-of- hospital sudden cardiac death (SCD) through an autopsy in a population sample of victims utilizing modern anatomical, histological, toxicologic and interview-based data to better characterize this fatal condition.

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- **Project Title: COMPREHENSIVE ASSESSMENT OF VALVULAR FUNCTION WITH MRI**

  Principal Investigator & Institution: Pauly, John M. Associate Professor; Electrical Engineering; Stanford University Stanford, CA 94305

  Timing: Fiscal Year 2003; Project Start 01-AUG-2003; Project End 31-JUL-2007

  Summary: (provided by applicant): The goal of this proposal is to develop and validate a comprehensive examination of valvular heart diseases. Valvular **heart disease** affects approximately 10% of the general population in the United States. Over the past 20 years, valvular diagnosis has undergone a revolution due to advances in cardiac ultrasound. However, ultrasound has inherent limitations with respect to tissue

characterization, spatial resolution, and the need for acoustic windows. Particularly difficult are the evaluation of valvular morphology, quantitation of valvular stenosis and identification and quantitation of valvular regurgitation. The examination of valvular **heart disease** includes the assessment of valvular morphology, cardiac output, intracardiac pressures, ventricular volume and volume regurgitations. Magnetic resonance imaging (MRI) is potentially the most appropriate technique for addressing all of these areas in a single examination. Current MR techniques for valvular imaging suffer from poor temporal and spatial resolutions, require prolonged acquisitions, and frequently require laborious post processing. As a result, there is a gap between what is scientifically feasible and what is currently applied clinically. Our goal in this proposal is to eliminate this gap between the potential of MRI and current clinical practice. Our group has pioneered many of the components that will be useful for the diagnosis of valvular **heart disease,** including real-time imaging, real-time color flow, and MR Doppler. In this proposal we will integrate and extend these components along with new developments to provide an integrated and comprehensive assessment of valvular function.

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- **Project Title: CONFERENCE ON MOLECULAR BIOLOGY OF THE HEART**

  Principal Investigator & Institution: Leinwand, Leslie A. Professor and Chair; Keystone Symposia Drawer 1630, 221 Summit Pl #272 Silverthorne, CO 80498

  Timing: Fiscal Year 2002; Project Start 01-JUN-2002; Project End 31-MAY-2003

  Summary: (provided by applicant): The focus of this symposium will be on the use of molecular and genetic tools to understand cardiovascular development, function and disease. It is a very exciting time in this field and we will take advantage of the veritable explosion in information that has resulted from the genome projects of multiple organisms. Gene discovery by the time of this meeting will undoubtedly be quite advanced and we should have information about the changes in gene expression that accompany cardiovascular disease in mice and men. Another exciting "hot" area of investigation is the potential for stem cells in the heart. We will devote a session to the promise and practice of cardiac stem cells. The use of model organisms has made it possible to develop paradigms for early heart formation and we will use examples of these studies to explore vertebrate and invertebrate heart development. Single gene disorders have contributed a great deal of information about congenital and adult cardiac disease ranging from valve and septal disorders to sudden death in young athletes. We will explore single gene disorders, but also approaches leading to the understanding of complex genetic disorders such as hypertension and predisposition for **heart disease.** Another emerging area of research is our ability to understand the molecular mechanisms leading to cardiac failure, the most prevalent disease and cause of death in North America. Sessions will be developed that are the essence of "bench-to-bedside" dealing with cardiac failure and how to develop treatment paradigms based on basic research findings. This symposium will bring together people from industry, clinicians and basic research in a way that serves the mission of the Keystone symposium. It will be a multidisciplinary meeting that should bring together people who are beginning to have regular dialogues but whose traditions have been somewhat separate.

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- **Project Title: CONGENITAL HEART DISEASE WITH LV NONCOMPACTION**

  Principal Investigator & Institution: Towbin, Jeffrey A. Professor; Baylor College of Medicine 1 Baylor Plaza Houston, TX 77030

  Timing: Fiscal Year 2001; Project Start 15-AUG-2001; Project End 31-JUL-2006

  Summary: Left ventricular non-compaction (LVNC), a form of myocardium disease which presents in infancy as heart failure, is characterized as a hypertrophic and dilated left ventricle with systolic dysfunction, deep endomyocardial recesses and trabeculations, and in some patients, congenital **heart disease** (CHD). When CHD occurs, it most commonly includes atrial septal defect (ASD), ventricular septal defect (VSD), right heart obstruction, or hypoplastic left heart syndrome. This disorder may be inherited as an autosomal dominant or X-LINKED trait. In the X- linked form, the gene G4.5 which encodes the tafazzin protein, has been found to be mutated in some patients but the mechanism of disease has not been discovered. No gene(s) has been identified for the autosomal dominant form. In the subproject, families with LVNC will be recruited and the genes for the disease will be pursued using a primary candidate gene approach utilizing our "final common pathway" hypothesis. In this hypothesis, we speculate that a central target protein is mutated directly or affected secondarily by an interacting cascade pathway, resulting in a specific phenotype. This hypothesis suggests that the final pathway or dilated cardiomyopathy is cytoskeletal/sarcolemmal abnormalities; hypertrophic cardiomyopathy is known to occur due to abnormalities of the sarcomere. Since these phenotypes are both involved in LVNC, genes encoding proteins involved in these pathways will be screened. In addition, this Program Project hypothesizes that transcription factors are disrupted in CHD, and therefore the interacting signaling cascade pathway(s) associated with the LVNC disease-causing gene will be identified. We have recently identified mutations in the alpha- dystrobrevin genes in patients with LVNC and this gene will be studied in a mutant mouse and the interacting proteins will be identified. The specific aims of this subproject include: (1) Identification and recruitment of families with LVNC; (2) Identification and characterization of genes responsible of genes responsible for LVNC; (3) Development and characterization of mouse models of LVNC, including alpha- dystrobrevin; and (4) Identification of protein-protein interactions and characterization of the mechanisms and pathways leading to associated CHD. Appropriate transcription factors and signaling pathways, particularly those interacting with the TGF-beta pathway studied in the other subprojects. Completion of this subproject will improve our understanding of the "final common pathways" involved in myocardial disease and CHD in children. In addition, successful completion of this subproject will clarify the role of signaling pathways in dilated and hypertrophic cardiomyopathy and lead to new paradigms in cardiac structure and function relationships.

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- **Project Title: CORE--GENE EXPRESSION**

  Principal Investigator & Institution: Oakey, Rebecca J.; Children's Hospital of Philadelphia 34Th St and Civic Ctr Blvd Philadelphia, PA 19104

  Timing: Fiscal Year 2001

  Summary: (Adapted from the Applicant's Abstract) The main reason for proposing to set up this core facility is that there are several investigators involved in the SCOR proposal whose projects require expression analysis of specific genes as part of understanding the development of the heart and pathogenesis of mis-expressed genes in **heart disease** (Projects 2, 3, 4 and 5). One of the crucial factors necessary for the

functional dissection of a gene is the analysis of its spatial and temporal expression pattern. Where and when a gene is expressed in a developing tissue is essential to understanding the molecular basis of heart development and vascular disease. Expression analyses are addressed definitively and easily by in situ hybridization of whole mouse embryos and sections of embryos using a gene probe of choice. By utilizing embryos from different developmental stages and a variety of embryo sections, both the temporal and spatial expression patterns of genes of interest can be determined. The developing mouse embryo provides an ideal model in which to study gene expression. Genes responsible for developmental defects in humans almost always have a closely related homologue in mouse (1). Moreover, due to extensive conservation of primary sequence between mouse and human genes, cross species hybridizations are frequently successful. Given the difficulties with obtaining samples of human embryos, the mouse provides an ideal alternative. In addition, location of the protein product of a gene facilitates analysis where antibodies are available. Accordingly, a core facility will be established that will bank whole mouse embryos and embryo sections from a range of developmental stages and perform in situ hybridization experiments and analyses using candidate gene probes and immunohistochemistry with specific antibodies. In situ hybridization of whole and sectioned embryos is complex and time consuming and requires a variety of skills in mouse husbandry, embryo staging, microscopy and molecular biology (2). This type of study is ideally suited to being performed by a core facility and as a service to a variety of investigators with different specific goals but a common interest in developmental and/or disease gene analysis of the heart. The main aim of this core will be to facilitate the identification of genes involved in the genetic basis of conotruncal malformations. This core will provide the technical expertise and facilities to perform spatial expression studies and to assist investigators of varying specialties with the tools to understand the biology of their heart specific genes. Projects 2, 3, 4 and 5 will be in a position to utilize this core for gene expression analyses. Expression studies require a wide variety of skills to set up. Moreover, this proposed core already has most of the necessary equipment and technical expertise in place to begin providing a service since The Children's Hospital of Philadelphia has already provided Dr. Oakey with seed money to begin this core.

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- **Project Title: CORONARY ARTERY VISUALIZATION SYSTEM**

  Principal Investigator & Institution: Kline-Schoder, Robert J.; Creare, Inc. Box 71, Etna Rd Hanover, NH 03755

  Timing: Fiscal Year 2001; Project Start 20-SEP-2001; Project End 30-APR-2002

  Summary: Heart disease is the cause of more deaths in America than any other disease. Coronary **heart disease** occurs when the walls of coronary arteries that carry blood to the heart are clogged with fatty deposits. A primary treatment for coronary **heart disease** is the coronary artery bypass graft (CABG). During a CABG, a detour is inserted around blocked portions of coronary arteries using segments of healthy vessels from other parts of the patient's body. For most patients, the internal condition of the affected coronary arteries is difficult to assess visually or by palpation. Further, for approximately 10% of patients, the affected arteries are obscured by fat or muscle and are difficult to locate. The aim of this project is to develop a system for use during surgery for visualizing coronary arteries, locating those that are obscured by fat or myocardial tissue, and assessing the quality of the resulting graft. The Creare system will allow cardiac surgeons to non-invasively view (before grafting) cross sections of the coronaries to assess their health and beneath the surface of obscuring layers to locate

buried vessels. The Creare system will provide cardiac surgeons with a new tool to optimize CABG procedures which will result in better, more consistent outcomes. PROPOSED COMMERCIAL APPLICATIONS: Creare will develop a new device for assessing and locating coronary arteries during bypass surgery. This device is intended to assist surgeons, will reduce the time for coronary artery bypass graft procedures, and result in better clinical outcomes. The device will be of interest to vendors of equipment for cardiac surgeons.

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- **Project Title: CORONARY DISEASE MORBIDITY AND MORTALITY IN A POPULATION**

  Principal Investigator & Institution: Roger, Veronique L.; Mayo Clinic Rochester 200 1St St Sw Rochester, MN 55905

  Timing: Fiscal Year 2001; Project Start 15-JAN-1998; Project End 14-JUL-2002

  Summary: (Adapted from Investigator's Abstract) Cardiovascular disease remains the leading cause of death in the U.S. Despite an encouraging decline in age-adjusted coronary **heart disease** (CHD) mortality, prevalent CHD continues to represent a major health burden, particularly in the elderly population. Most community surveillance programs, however, cannot fully characterize this problem because they are restricted to an upper age limit of 74 years and thus do not include the events occurring in an increasingly growing part of the population. Observational studies have questioned the existence of a change over time in the prevalence of anatomic coronary disease either at post-mortem examination or at coronary angiography. This remains to be further examined in a population-based setting. In addition, the natural history of myocardial infarction (MI) in the reperfusion area is unknown; in particular, there are no population-based data on the incidence of post-MI heart failure. The investigators propose to study the entire population of Olmsted County, Minnesota, including all age categories, to examine the secular trends in CHD mortality, MI incidence, and natural history, including MI severity, case fatality and post-MI morbidity. In addition, because of the uniquely high autopsy rate in Olmsted County, the time trends in the prevalence of CHD at post-mortem will be examined. The investigators point out that the Olmsted County population constitutes a valuable resource for ascertainment of MI incidence, morbidity and case fatality rate. In particular, since all inpatient and outpatient medical encounters are present in a single medical record unit, this study will have the capabilities of measuring the incidence of post-MI heart failure and its hypothesized change over time. The records of all Olmsted County residents with a hospital discharge diagnosis of MI between 1979 and 1999 will be reviewed, and standard epidemiologic MI validation criteria will be applied; post-MI outcome over time will be determined, including post-MI heart failure, angina, 30 day case fatality and long-term survival. In parallel with the analysis of time trends in CHD mortality, the autopsy reports will be reviewed to examine whether the prevalence of coronary disease at autopsy has changed over time. The investigators state that these studies will provide an assessment of the clinical and anatomical manifestations of CHD, including the outcome of acute MI, over a time period characterized by intensified primary prevention efforts and major changes in the treatment of acute CHD.

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- **Project Title: CORONARY HEART DISEASE INCIDENCE IN RELATION TO TOTAL HOMOCYSTEINE**

  Principal Investigator & Institution: Folsom, Aaron R.; Oregon Health & Science University Portland, OR 972393098

  Timing: Fiscal Year 2001

  Summary: We used a prospective case-cohort design to determine whether total homocysteine (tHcy)-related factors are associated with incidence of coronary **heart disease** (CHD) over an average of 3.3 years of follow-up in a biracial sample of middle-aged men and women. Age-, race-, and field center-adjusted CHD incidence was associated positively (P<0.05) with tHcy in women but not men, and CHD was associated negatively (P<0.05) with plasma folate (women only), plasma pyridoxal 5=-phosphate (both sexes), and vitamin supplementation (women only). However, after accounting for other risk factors, only plasma pyridoxal 5=-phosphate was associated with CHD incidence; the relative risk for the highest versus lowest quintile of pyridoxal 5=-phosphate was 0.28 (95% CI=0.1 to 0.7). There was no association of CHD with the C677 T mutation of the methylenetetrahydrofolate reductase gene or with three mutations of the cystathionine ?-synthase gene. Our prospective findings add uncertainty to conclusions derived mostly from cross-sectional studies that tHcy is a major, independent, causative risk factor for CHD. Our findings point more strongly to the possibility that vitamin B6 offers independent protection. Randomized trials, some of which are under way, are needed to better clarify the interrelationships of tHcy, B vitamins, and cardiovascular disease. FUNDING Collaboration with Dr. Aaron Folsom, University of Minnesota PUBLICATIONS Folsom AR, Nieto FJ, McGovern PG, McGovern PG, Tsai MY, Malinow MR, Eckfeldt JH, Hess DL, Davis CE. Prospective study of coronary **heart disease** incidence in relation to fasting total homocysteine, related genetic polymorphisms, and B vitamins The Atherosclerosis Risk in Communities (ARIC) Study. Circulation 98:204-210, 1998.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CORONARY HEART DISEASE: WOMEN'S VALUES, BELIEFS AND COGNITIVE PROCESSES**

  Principal Investigator & Institution: Arslanian-Engoren, Cynthia; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, MI 481091274

  Timing: Fiscal Year 2002; Project Start 30-SEP-2002; Project End 30-JUN-2007

  Summary: Coronary **heart disease,** which includes acute and old myocardial infarction, angina pectoris, and other acute, subacute, and chronic forms of ischemic **heart disease,** is the single largest killer of American women. In 1999, 262,391 United States women, of all ethnic and racial groups, lost their lives to coronary **heart disease.** However, Black women have the highest overall death rates from coronary **heart disease,** followed by White and Hispanic women. Despite these findings and the fact that women are more likely than men to die after a myocardial infarction, women are less likely than men to seek medical attention after the onset of initial symptoms. Explanations for these delays have been linked to low perceptions of susceptibility to **heart disease,** the lack of association of initial symptoms as significant indicators of an acute cardiac event, low socioeconomic status, and patient race. While not negating the importance of these variables, they do not speak to the values attitudes or beliefs that underlie women's decision to seek emergency care. Guided by The Health Belief Model, this triangulated, descriptive study will examine the values, beliefs, and cognitive process of a total of 78 women (26 Hispanic, and 26 white) relative to the manifestation and presentation of an

acute myocardial infarction, while the quantitative phase will focus on the symptoms women believe most likely indicate an acute myocardial infarction.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CVD RISK & HEALTH IN POSTMENOPAUSAL PHYTOESTROGEN USERS**

  Principal Investigator & Institution: Kritz-Silverstein, Donna C. Associate Adjunct Professor; Family and Preventive Medicine; University of California San Diego 9500 Gilman Dr, Dept. 0934 La Jolla, CA 92093

  Timing: Fiscal Year 2001; Project Start 01-APR-1997; Project End 09-MAR-2002

  Summary: (Adapted from Investigator's Abstract) In the United States, **heart disease** is the leading cause of death in postmenopausal women. Estrogen replacement therapy is beneficial for **heart disease** risk factors as well as for bone density. However, a large proportion of postmenopausal women are not compliant with therapeutic regimens. Phytoestrogens are naturally occurring compounds found in plants and soy products that have estrogenic effects, and may represent an alternative treatment for the prevention of **heart disease** and osteoporosis in postmenopausal women. However, few intervention trials have examined the extent to which it is possible to improve **heart disease** risk factors, bone density, and quality of life in postmenopausal women through use of a dietary supplement of Phytoestrogen. The proposed randomized, double-blind, placebo controlled study is designed to determine the acceptability and benefits of use of a dietary supplement of Phytoestrogen (genistein) versus placebo on **heart disease** risk factors, bone density and psychosocial outcomes in postmenopausal women aged 45-74. Approximately 300 women will be screened in order to enroll 200 (100 treatment, 100 placebo) who will each be followed for one year. Data will be collected at screening and baseline visits, 1 and 3-month follow-up telephone calls, and 6- and 12-month follow-up clinic visits. Measures of HDL, and other **heart disease** risk factors, hip and spine bone density, and depression, life satisfaction, and quality of well-being will be obtained. Cross-sectional and longitudinal comparisons of treatment and placebo groups will be performed before and after adjustment and stratification for potentially confounding covariates. It is expected that women treated with Phytoestrogen will have higher HDL and bone density, and more favorable psychosocial outcomes. It is also expected that women using Phytoestrogen will have more favorable total cholesterol, LDL, triglycerides, Lp(a), fibrinogen, blood pressure, fasting and postmenopausal challenge glucose and insulin, and fat distribution. Given that women can expect to live one-third of their lives after menopause, the investigators point out that it is important to know how Phytoestrogen may modify **heart disease** risk factors and bone density. They further state that by defining the influence Phytoestrogen use has, this study would contribute to understanding of how to prevent cardiovascular disease and osteoporosis in postmenopausal women and thereby improve their quality of life.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CX43 IN A GENETIC MODEL OF ALTERED MYOCARDIAL CONDUCTION**

  Principal Investigator & Institution: Saffitz, Jeffrey E. Professor of Pathology & Immunology; Pathology and Immunology; Washington University Lindell and Skinker Blvd St. Louis, MO 63130

  Timing: Fiscal Year 2001; Project Start 01-APR-1998; Project End 31-MAY-2005

Summary: The goal of the proposed research is to define the functional role of Cx43 in normal cardiac conduction and to delineate the role of altered coupling at gap junctions in the pathogenesis of conduction disturbances and arrhythmias. Proposed experiments will be performed using mice that are heterozygous for a null allele for the gene encoding the major cardiac gap junction protein, Cx43 (Cx43 plus/minus mice). These mice produce 50 percent of the wildtype level of Cx43 and have significant reduction in the number of gap junction interconnecting ventricular myocytes. The functional consequence of reduced Cx43 expression in adult mice is a 25-30 percent slowing of ventricular conduction velocity. Whereas the electrophysiological phenotype in Cx43 plus/minus mice is subtle under physiological conditions, a more dramatic phenotype can be elicited under pathophysiological condition. In response to acute regional ischemia, Cx43 plus/minus mice exhibit accelerated onset and increased incidence, frequency and duration of ventricular arrhythmias. The proposed research is focused on defining mechanisms by which reduced coupling promotes arrhythmias in accute and chronic ischemic **heart disease.** Studies in Specific Aim 1 will elucidate the mechanistic relationship between the rate and extent of electrical uncoupling at gap junctions and development of ventricular tachyarrhythmias induced by acute ischemia. Studies in Specific Aim 2 will define arrhythmia mechanisms in Cx43 plus/minus following acute coronary occlusion and delineate the roles of Cx43 and altered cell-to- cell coupling in electrical triggering events and sustained conduction abnormalities that underlie initiation and maintenance of ventricular arrhythmias in the setting of acute myocardial ischemia. In Specific Aim 3, the role of gap junction remodeling in the pathogenesis of arrhythmias in chronic ischemic **heart disease** will be elucidated by comparing arrhythmogenesis in Cx43 plus/minus and wildtype mice with healed myocardial infarcts. And in Specific Aim 4, molecular and structural determinants of conduction will be delineated using neonatal mouse ventricular myocytes grown in patterned arrays of defined structure and packing geometry, and analyzed by high resolution optical mapping. The results of the proposed research will define mechanisms by which reduced coupling promotes ventricular tachyarrhythmias in mouse models of acute and chronic ischemic **heart disease** in patients.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DEPRESSION IN ADAPTATION TO OPEN-HEART SURGERY**

  Principal Investigator & Institution: Goyal, Tanya M. Psychology; Rutgers the St Univ of Nj New Brunswick Asb Iii New Brunswick, NJ 08901

  Timing: Fiscal Year 2001; Project Start 01-JUN-2001

  Summary: Applicant?s The proposed study will examine the effects of depressive symptomatology on physical and psychological adaptation to open-heart surgery. There is growing evidence suggesting that depression may influence the development and progression of coronary **heart disease** as well as recovery following cardiac events. While several physiological and behavioral pathways have been proposed to explain these associations, potential psychological mechanisms have rarely been considered. In addition to evaluating effects of depression on adaptation to cardiac surgery, this study will examine the role of social cognitive variables as mediators of these effects. The specific aims of this study are to test the following hypotheses: (1) Lower levels of preoperative depression will predict shorter hospitalizations following surgery; (2) Lower levels of preoperative depression will predict less angina and better physical functioning six months after surgery; (3) Lower levels of preoperative depression will predict less anxiety six months after surgery; and (4) These effects will be partially mediated by outcome expectancies and efficacy expectancies regarding behavioral and

social activities associated with recovery. This project seeks to advance theoretical understanding of the relationship between psychosocial factors and physical illness. Additional long-term goals of this research include improving pre-surgical identification of cardiac patients at risk for poor outcomes, the development of psychosocial interventions for this population, and ultimately, the enhancement of surgical outcomes and long-term adaptation to chronic disease.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DETECTING CHANGES IN MYOCARDIAL PERFUSION AND FUNCTION**

  Principal Investigator & Institution: Faber, Tracy L. Associate Professor; Radiology; Emory University 1784 North Decatur Road Atlanta, GA 30322

  Timing: Fiscal Year 2002; Project Start 15-JUL-2002; Project End 30-JUN-2006

  Summary: Coronary **heart disease** caused 476,124 deaths in 1996 and continues to be the leading cause of death in America today. Over 12 million people alive today have a history of myocardial infarction, angina pectoris, or both. Every year in the US about 5 million perfusion studies are performed to evaluate extent and severity of CAD, thereby enabling clinical decisions regarding diagnosis, prognosis, and therapy for patients with **heart disease.** Of these, 1 million undergo angioplasty and about 500,000 have bypass surgery, and millions of others undergo drug therapy and or lifestyle changes to prevent progression of cardiac disease. It is widely recognized that computer quantification of myocardial perfusion images improves diagnostic accuracy and enhances confidence and reproducibility of interpretation. Theses quantitative approaches are well-established for assessing abnormalities in myocardial perfusion and function. However, they have not been developed or optimized for detecting changes in serial studies of the same patient such as is needed for assessing the effect of interventions, medical therapy, or disease progression. In this project, we will develop and validate computer-based methods to automatically quantify and visualize serial changes in myocardial perfusion and function from perfusion SPECT. The work can be separated into 4 projects: 1) To assess changes in myocardial perfusion, 2) to assess changes in myocardial function, 3) to design a virtual heart suitable for creating simulation data for optimizing and analyzing our algorithms, and 4) to validate the methods using both simulations and animal studies. Important subprojects include: a) development of 3-d and 4-d surface detection methods for defining LV endocardial and epicardial surfaces, b) development of algorithms for non-rigid alignment of static and/or dynamic serial SPECT images so that they may be more directly compared, c) development of motion analysis methods using similar non-linear alignment techniques to measure regional myocardial function and also to correct ungated SPECT scans for motion blur, and d) creation of new statistical approaches to determine significant changes in both global and regional perfusion and functional variables. The ultimate goal of this project is to create a clinically useful tool for detecting changes in serial SPECT studies. Most importantly, the tools will be extremely well characterized as to their sensitivity in detecting small changes as well as for overall accuracy.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DETECTION OF ATHEROSCLEROSIS IN SYSTEMIC LUPUS ERYTHEMATOSUS**

  Principal Investigator & Institution: Petri, Michelle; Johns Hopkins University 3400 N Charles St Baltimore, MD 21218

Timing: Fiscal Year 2001

Summary: Atherosclerosis (hardening of the arteries) is ten times more common in patients with SLE than in the general population. The purpose of this research study is to determine how frequently SLE patients have an abnormal heart scan, indicative of atherosclerosis. Research participants will undergo a nuclear medicine cardiac SPECT scan as an outpatient. The scan requires an injection of a small amount of radioactive tracer into an arm vein. The testing procedure requires a patient to be present for approximately 6 hours. If a patient weighs over 200 pounds, a second day of testing may be necessary. As part of the scan, patients exercise. Patients who have coronary **heart disease** (heart problems) might be at risk for angina (heart pain) or myocardial infarction (heart attack). No one with known **heart disease** will enter this study. The radiation exposure received from participating in this study is equivalent to an exposure of 3.4 rems to the whole body. Naturally occurring radiation (cosmic radiation, radon, etc.) produces whole body radiation exposures of about 0.3 rems per year. Occupationally exposed individuals are permitted to receive whole body exposures of 5 rems per year.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DEVELOPING CELLULAR CARDIOMYOPLASTY FOR INJURED HEART**

  Principal Investigator & Institution: Taylor, Doris A. Associate Professor; Medicine; Duke University Durham, NC 27706

  Timing: Fiscal Year 2001; Project Start 01-JUN-1997; Project End 31-MAY-2002

  Summary: Because the adult heart cannot regenerate or repair itself, the end result of ischemic **heart disease** (IHD) is often progression of acute myocardial infarction (AMI) to congestive heart failure (CHF) resulting in the death of over 41,000 persons annually. Currently, medical interventions to prevent the progression to heart failure following a severe AMI are limited. Treatment options for end-stage CHF are even more limited. A new therapeutic option in development, cellular cardiomyoplasty (CCM), or transplantation of autologous primary skeletal muscle cells into the myocardium, offers potential for augmenting cardiac function in myocardial disease. The overall aim of this project is to develop a basis for the use of cellular cardiomyoplasty in cardiac disease including AMI and CHF. The hypothesis is: in a rabbit model of myocardial infarction, decreased myocardial performance can be at least partly reversed by repopulating the damaged area with primary skeletal myoblasts to re- form a functional unit within an infarct region. The specific aims to test this hypothesis are to: 1) optimize marker gene expression in autologous primary skeletal muscle myoblasts to follow their fate in the heart; 2) compare the efficiency of tow methods of myoblast delivery to rabbit heart: localized delivery via direct injection or more dispersed delivery via infusion onto the coronary circulation; 3) use load-insensitive in dices of regional cardiac function to determine the temporal effects of directly injected myoblasts on regional contractile function and on ventricular morphology in control and infarcted hearts; 4) determine the temporal effects of myoblasts infused into the coronary circulation on global contractile function (by 2-d echocardiography) and ventricular morphology in control and infarcted hearts. Accomplishing these aims should allow an evaluation of the extent to which autologus skeletal myoblasts can survive implantation into the cardiac environment and contribute to cardiac function. Developing cellular cardiomyoplasty may contribute a promising therapeutic intervention for IHD or CHF both of which are major economic and management problems for all health care providers because of the substantial health care costs expended in the treatment of these severely debilitating

conditions and because of the limitations of definitive therapeutic interventions. R02MH49428 There is abundant evidence to suggest that neuropsychiatric disorders such as schizophrenia and autism are caused in many cases by genetic abnormalities that affect development and function of forebrain neural systems involved in cognition and emotion. The largest structures of the forebrain are the cerebral cortex and the striatum; both have been implicated as having a role in neuropsychiatric disorders. The goal of my research is to understand how genes regulate development of the striatum. To this end, my laboratory has identifies the D1x genes, which encoded a family of homeodomain transcription factors that are candidates for having a central role in striatal development. There are four known D1x genes that are expressed in the embryonic forebrain. The aims of the experiments proposed in this grant application are focused on: (1) elucidating the sequence of these genes and their encoded proteins; (2) characterizing the biochemical properties of the DLX proteins; (3) determining whether the DLX proteins are transcriptional regulators; (4) identifying proteins that interact with and modulate the function of the DLX proteins; (5) determining the intracellular location of the DLX proteins; (6) determining the temporal and spatial patterns expression of the D1x RNAs and proteins in the prenatal and postnatal forebrain; (7) begin to determine where the D1x genes are in the genetic hierarchy that regulates development of the forebrain using ectopic expression experiments.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DEVELOPMENTAL CORRELATES-- MEMBRANE/CONTRACTILE PROTEIN**

  Principal Investigator & Institution: Anderson, Page W. Professor; Pediatrics; Duke University Durham, NC 27706

  Timing: Fiscal Year 2001; Project Start 09-AUG-1991; Project End 30-APR-2005

  Summary: (Applicant's Description Verbatim): Heart failure is a major cause of premature death and disability in the United States. The basis of abnormal myofilament function in the failing heart is not known. Myofilament function is regulated by troponin, a complex made of three subunits (cTnI, cTnC, cTnT): cTnI inhibits actin-myosin interaction, the binding of Ca2+ to cTnC disinhibits this interaction, and cTnT binds the complex to tropomyosin and is essential for Ca2+ -dependent force development and myofibrillar ATPase activity. This proposal aims to determine the role of the cTnT isoforms in the regulation of myocardial function, and how they affect the failing heart. The function of the cTnT isoforms is not known. We have identified four TnT isoforms in the human heart, whose expression is regulated by development and affected by heart failure. In contrast, the same single isoform of cTnI and cTnC are expressed in the normal and failing adult human heart. We focus on the cTnT isoforms because cTnT isoform expression is correlated with the fall in myofibrillar ATPase activity in the failing human heart. We will test the following: Hypothesis 1(a) cTnT isoforms modulate the binding characteristics of cTnC to Ca2+ in troponin in vitro and the characteristics are further modulated by the incorporation of troponin into the thin filament. Hypothesis 1(b). In myocardium, the cTnT isoforms alter myofilament function by changing the myofilaments' sensitivity to Ca2+ and the sarcomere length-dependence of this sensitivity (and consequently, the Frank-Starling relation). Hypothesis 2. cTnT isoform expression in vivo alters ventricular function in vivo and myofilament function in vitro. Hypothesis 3. cTnT isoform expression in vivo preserves left ventricular function in **heart disease.** We will use reagents we have recently developed, including recombinant cTnT proteins and transgenic mice overexpressing cTnT isoforms. These will be used to examine the functional role of cTnT isoforms in

troponin, the thin filament, isolated myocytes, ventricular bundles and the in vivo heart and to test the effects of cTnI isoform expression of left ventricular structure and function in mouse models of **heart disease.** These models include constriction of the transverse aorta and over-expression of calsequestrin. Defining the role of cTnT isoforms in the normal and failing heart is expected to provide targets for potential new and effective pharmacological interventions in heart failure.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DIETARY ETILOGIES OF HEART DISEASE AND CANCER**

  Principal Investigator & Institution: Willett, Walter C. Professor and Chairman; Nutrition; Harvard University (Sch of Public Hlth) Public Health Campus Boston, MA 02460

  Timing: Fiscal Year 2001; Project Start 01-DEC-1985; Project End 30-NOV-2002

  Summary: (Adapted from Investigator's Abstract) The authors propose to continue the follow-up of 51,529 male health professionals, aged 40 to 75 years in 1986, to address a series of dietary hypotheses related to risk of coronary **heart disease,** stroke, and peripheral vascular disease. Diet has been assessed in 1986, 1990, and 1994 by a semiquantitative food frequency questionnaire developed and refined by the group over the last 15 years. Detailed studies in a subsample of participants demonstrate that this questionnaire performs well; the average correlation between the questionnaire and 14 days of diet recording for 16 nutrients of interest was 0.66. This study population has substantial variation in dietary intake, for example, fat intake varies from 25% to 42% of calories between extreme quintiles. Body fat distribution (assessed by waist and hip circumferences) has been measured twice (1987 and 1996) and weight is updated during every 2-year cycle. The first three follow-up cycles are complete; with a total questionnaire response of 94%. With the use of the National Death Index and cooperation of professional organizations, mortality follow-up is virtually complete. In the past five years, 24 papers (published or in press) were produced in the field of cardiovascular disease and related conditions, addressing such risk factors as obesity, smoking, and vitamin E, carotene, fish, fiber, and fat intake. The authors plan to continue following the cohort by questionnaires mailed at two-year intervals to update exposure information and ascertain nonfatal events. The proposed continuation is a highly cost-effective for studying diet and cardiovascular disease because funds for mailing and processing of the questionnaire and archiving of blood samples (n=18,000 collected) are provided from other sources (CA55075, DK45779, CA58684). Reported cases of nonfatal MI, stroke, peripheral arterial disease and cancer are documented with hospital records or pathology reports. Fatal events are identified by next-of-kin, postal service, or the National Death Index and confirmed by hospital records and other additional information. During the fourteen years of follow-up represented by this continuation the authors project a total of 2,068 cases of fatal or nonfatal myocardial infarction, 578 cases of stroke, and 745 cases of peripheral arterial occlusive disease. They will have substantial power to evaluate a series of continuing and new specific hypotheses to quantify dose-response relationships, and to assess the impact of change in diet and anthropometric variables using repeated measurements.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DIETARY ETIOLOGIES OF HEART DISEASE AND CANCER**

  Principal Investigator & Institution: Rimm, Eric B. Associate Professor; Nutrition; Harvard University (Sch of Public Hlth) Public Health Campus Boston, MA 02460

Timing: Fiscal Year 2003; Project Start 01-DEC-1985; Project End 31-MAY-2007

Summary: (provided by applicant): We propose to continue the biennial follow-up of cardiovascular disease among 51,529 male health professionals, age 40 to 75 years in 1986, to address a series of new dietary hypotheses related to risk of a coronary **heart disease** and stroke. We project over 4,000 incident MI, fatal CHD, and stroke cases through the end of the follow-up period. Nested within this cohort, over 18,000 participants provided blood samples in 1994 from which we propose to investigate several biological (plasma and genetic) determinants of disease. We will concentrate on several hypotheses related to nutritional and genetic determinants of cardiovascular disease (CVD). With this exceptional resource of repeated assessments of diet and lifestyle characteristics tied to potential genetic markers of disease, we will prospectively evaluate in relation to coronary **heart disease** 1) n-6 fatty acids across a wide range of n-3 fatty acid intake from fish and vegetable sources, 2) foods with a high glycemic load, specifically among men with a BMI > 25kg/m^2, 3) protein intake as a replacement for carbohydrate, and 4) putative functional variants and haplotypes in candidate genes thought to be insulin targets. Within this metabolic domain we seek to determine if lifestyle practices such as physical activity or a low glycemic load diet can modify underlying genetic risk. To investigate further the effect of diet on mediators of CHD we will investigate a) the interaction between n- 3 and n-6 fatty acids on inflammatory risk factors for CVD and b) glycemic load on adiponectin and the associated risk of this adipocyte-derived cytokine on risk of MI and fatal CHD. Also, we propose further to examine aims 1-3 with respect to stroke. Finally, within a small exploratory aim, we propose to document basic risk factors for congestive heart failure by utilizing the innovative methods we designed to study coronary **heart disease.** The ongoing Health Professionals Follow-up Study will provide follow-up of non-CVD endpoints (CA55075) in addition to information on important covariates for the proposed study. Overall, the large size of the prospective cohort, the high follow-up rate, the repeated assessment of dietary and lifestyle information, and the availability of archived bloods provide a unique cost-effective opportunity to test hypotheses related to CVD risk.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ECHOCARDIOGRAPHIC ASSESSMENT OF MYOCARDIAL PERFUSION**

  Principal Investigator & Institution: Kaul, Sanjiv; Professor of Cardiology; Internal Medicine; University of Virginia Charlottesville Box 400195 Charlottesville, VA 22904

  Timing: Fiscal Year 2001; Project Start 01-AUG-1992; Project End 31-MAY-2005

  Summary: (the applicant's description verbatim): Contrast-enhanced ultrasound has become a valuable experimental and clinical tool for assessing organ perfusion. It used gas-filled microbubbles as perfusion tracers, which scatter ultrasound during their transit through any vascular compartment. Because microbubbles have an intravascular rheology similar to that of red blood cells, contrast-enhanced ultrasound can be used to measure microbubble (or red blood cell) velocity in tissue. Additionally, because the microbubbles remain entirely within the intravascular space, contrast-enhanced ultrasound can also be used to measure the blood volume fraction (MBV). Over this previous grant cycle, we have used MCE to unravel many important aspects of MBV, which have major relevance in understanding the local control of myocardial blood flow (MBF) as well as the adaptive changes that occur consequent to coronary artery disease. In particular, we have gained insights into the role capillaries in regulating MBF. We have shown that capillaries are responsible for limiting the maximal hyperemic response seen in the normal coronary circulation and that capillary blood volume decreases distal

to a stenosis during hyperemia. We have also shown that perfusion defects on nuclear tracer imaging is related to reversible decreases in MBV distal to a stenosis rather than due to 'flow mismatch' as has heretofore been assumed. The aim of this proposal is to further define adaptive changes in MBV and capillary resistance that occur under physiological conditions and in atherosclerotic **heart disease.** The specific aims of this proposal are to assess: 1. How capillaries regulate MBF in the normal coronary circulation when autoregulation is exhausted. 2. What happens to capillaries distal to a stenosis during hyperemia. 3. Whether phasic changes in MBV can be used to detect the presence and magnitude of coronary stenosis at rest. 4. Whether changes in blood viscosity affect coronary blood flow reserve. Studies will be performed in open chest canine models using contrast-enhanced ultrasound and in the rat skeletal muscle using intravital micrscopy. The ultimate aim is the further understanding of the role of capillaries in the local regulation of MBF.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ECHOCARDIOGRAPHIC EVALUATION OF ENDOTHELIAL FUNCTION**

  Principal Investigator & Institution: Villanueva, Flordeliza S. Medicine; University of Pittsburgh at Pittsburgh 350 Thackeray Hall Pittsburgh, PA 15260

  Timing: Fiscal Year 2002; Project Start 01-AUG-1997; Project End 30-JUN-2006

  Summary: (provided by applicant): Coronary artery disease is the major cause of mortality in the United States. Although many patients come to medical attention because of hemodynamically significant coronary lesions, over one-half who die suddenly from coronary disease have had no pre-existing symptoms. It has been hypothesized that critical abnormalities in the coronary endothelial lining provide a milieu that ultimately promotes acute thrombotic occlusion in the absence of significant prior coronary stenoses. Moreover, in patients with angina due to severe stenoses, endothelial abnormalities likewise promote the development and progression of atherosclerotic plaques that culminate in acute ischemic syndromes. Aberrations in endothelial function in coronary disease involve the microcirculation and occur in other cardiovascular disease states such as heart transplant rejection and ischemia-reperfusion. Endothelial dysfunction is thus a critical mediator of the total burden of atherosclerotic **heart disease** and a major component of other cardiovascular pathologies, and the identification and treatment of this phenomenon would confer significant health benefits. Unfortunately, methods to identify patients with early endothelial dysfunction are limited. Studies have shown that endothelial overexpression of leukocyte adhesion molecules such as intercellular adhesion mnolecule-1 (ICAM1) promotes the development of the earliest lesions of atherosclerosis, suggesting that ICAM1 can serve as a marker of incipient endothelial disease. Using perfused cultured human coronary artery endothelial cells (ECs), we recently proved the principle that gas-filled microbubbles conjugated to a ligand that binds specifically to ICAM1 adhere to interleukin1a-activated ECs overexpressing ICAM1. Because these bubbles are acoustically active in the presence of ultrasound, we hypothesize that in vivo ultrasound imaging of bubbles targeted to specific cell surface markers of endothelial activation such as ICAM1 will permit non-invasive identification of pre-clinical endothelial dysfunction. To test this hypothesis, in vitro and in vivo models of endothelial activation will be used to address three Specific Aims with respect to bubbles engineered to bind to cell surface markers of endothelial disease: (1) Define shear conditions under which ICAM1-targeted bubbles attach to ECs and optimize bubble characteristics for binding; (2) Identify other EC surface proteins specific for endothelial dysfunction, including

selectins, vascular adhesion molecule-1, or vascular endothelial growth factor receptors, evaluate their suitability as imaging targets, and design microbubbles for these targets; and (3) Image and quantify targeted bubble adhesion in vivo under clinically relevant conditions. The ultimate goal of this proposal is to develop targeted ultrasound imaging in order to enhance the early diagnosis of ischemic **heart disease** or other cardiovascular disease states associated with endothelial dysfunction. Principles gleaned from these studies can be extended to function-specific ultrasound imaging of other targets such as angiogenic markers, and may also provide a basis for targeted therapeutic approaches using microbubble carriers.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: EFFECT OF HEART DISEASE EDUCATION ON OLDER WOMEN**

  Principal Investigator & Institution: Clark, Noreen M. Dean; Health Behavior and Hlth Educ; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, MI 481091274

  Timing: Fiscal Year 2001; Project Start 01-AUG-1997; Project End 31-JUL-2002

  Summary: This application is aimed at evaluating different health education approaches to improve the health-related quality of life and health care utilization of older women with **heart disease.** Approximately 1,100 women, who are 60 to 80 years old and have coronary **heart disease,** will be randomized to one of four treatment conditions: 1) usual care control, 2) group-based health education, 3) self-directed education via mail and phone contacts, or 4) a choice of either the self-directed or group program. The programs will be evaluated at 3, 12, and 18 months. The primary outcomes of interest are physical and psychological functioning, frequency and severity of symptoms, and health care utilization. It is hypothesized that both the self-directed and group health education formats will result in better long-term outcomes than usual care, and that the best outcomes will be observed when women are given a choice between either of the formats.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: EFFECTS OF MEDITATION ON MECHANISMS OF CHD**

  Principal Investigator & Institution: Bairey Merz, C. Noel. Associate Professor and Medical Director; Cedars-Sinai Medical Center Box 48750, 8700 Beverly Blvd Los Angeles, CA 90048

  Timing: Fiscal Year 2000; Project Start 30-SEP-1999; Project End 31-AUG-2004

  Summary: Coronary **heart disease** (CHD) is the leading cause of death, disability, and health care costs in the US. The majority of CHD patients continue to have acute cardiac events, many sudden and unexpected, despite identification and treatment of their disease and attention to the traditional risk factors. The pathophysiologic bases of these cardiac events are not fully established, but substantial evidence indicates that psychosocial stress and resulting sympathetic nervous system imbalance are major contributors. Evidence indicates that psychosocial stress and a hyperresponsive sympathetic nervous system have adverse effects on both vasomotor function and long-term autonomic balance. Recent advances in our understanding of the pathophysiology of acute cardiac events-specifically, identification of the roles that arterial vasomotor dysfunction and autonomic nervous system imbalances play in the interplay of psychosomatic stress and CHD. Preliminary evidence further suggests that Complementary and Alternative Medicine (CAM) practices, such as the Transcendental Meditation (TM) technique, can not only reduce stress but also reduce acute cardiac

events in patients with CHD. Based on these and related data, we propose a randomized, blinded, controlled study of the effects of one CAM practice, the TM technique, compared to a control group, on the primary outcomes of (1) arterial vasomotor dysfunction (brachial artery reactivity); (2) autonomic nervous system imbalances (heart rate variability); (3) transient ambulatory myocardial ischemia (ST segment depression); and (4) the secondary outcomes of psychological stress and quality of life (anger, hostility, anxiety, depression, perceived health, disease-specific symptoms, and life stress/social resources). We hypothesize that significance effects on these physiological and psychological mechanisms associated with practice of the TM program will elucidate the known effectiveness of certain CAM techniques as additive/alternative approaches to prevention of acute cardiac events in CHD patients. Results of this randomized controlled trial will: (a) yield new data regarding the reversal of pathophysiological mechanisms underlying CHD, (b) provide mechanism data to complement our ongoing NIH- sponsored trial of this CAM practices on CVD-related mortality, and (c) provide pilot data for an expanded study of the effect of the TM technique on the pathophysiological mechanisms underlying acute cardiac events.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ENDODONTIC INFLAMMATION AND CARDIOVASCULAR OUTCOMES**

  Principal Investigator & Institution: Caplan, Daniel J. Assistant Professor; Dental Ecology; University of North Carolina Chapel Hill Office of Sponsored Research Chapel Hill, NC 27599

  Timing: Fiscal Year 2001; Project Start 10-JUL-2001; Project End 30-JUN-2003

  Summary: Several large-scale epidemiologic investigations have uncovered relationships between chronic periodontal disease and adverse cardiovascular outcomes such as coronary **heart disease** and stroke. To date, endodontic inflammation has not received the same attention despite its being a commonly found sequel to bacterial infection of the Dental pulp space and its having several important characteristics in common with inflammation of periodontal origin. These similarities form the basis of the proposed epidemiologic study, which seeks to test the hypothesis that a greater history of endodontic inflammation is associated with 1) increased risk of coronary **heart disease** and stroke; and 2) increased carotid artery intimal-medial thickness and prevalence of coronary **heart disease.** These hypotheses will be addressed by linking data from three large, well-established, ongoing epidemiologic studies of aging populations with newly collected variables involving endodontic disease and treatment. Two of the three sub-studies (from populations of adult men in Boston and adult women in Sweden) will employ a review of existing intra- and extra-oral radiographs to assess variables related to apical periodontitis and frequency and quality of root canal therapy, and will relate these exposures to subsequent incidence of coronary **heart disease** and stroke. The third sub-study (from adult populations in four U.S. communities) will compare self-reports of endodontic treatment with prevalence of coronary **heart disease** and thickened carotid arterial walls in a cross-sectional fashion. Given the relatively high frequency of endodontic inflammation among adult populations, the proposed study describes a straightforward, fast, and inexpensive way to gain preliminary insight into the relationship between endodontic and cardiovascular disease. It also will serve as the epidemiologic foundation for future investigations into endodontic disease and other systemic outcomes.

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- **Project Title: ENDOTHELIAL CELL TARGETED GENE THERAPY STRATEGIES FOR CORONARY HEART DISEASE**

  Principal Investigator & Institution: Dzau, Victor J. Chairman; Brigham and Women's Hospital 75 Francis Street Boston, MA 02115

  Timing: Fiscal Year 2001

  Summary: The endothelium appears to play a central homeostatic role in maintaining normal vascular function and structure. Although the association between endothelial dysfunction and vascular disease is well established, its molecular basis and pathogenic significance remains poorly defined. Our central hypothesis poses that normal endothelial function is predicated upon a homeostatic balance between reactive nitrogen species such as nitric oxide (NO) and reactive oxygen species (ROS) such as superoxide anion. Furthermore, we postulate that the NO- ROS balance modulates several critical pathobiological processes involved in atherosclerotic coronary **heart disease** including : endothelial cell-leukocyte adhesion, vascular smooth muscle cell growth and migration, endothelial cell apoptosis, platelet aggregation, and decreased coronary blood flow. This project will test the postulate that the decline in NO bioactivity characteristic of endothelial dysfunction is a critical event in the pathogenesis of atherosclerotic coronary **heart disease** and that a gene therapy approach based upon restoring the normal NO-ROS homeostatic balance will be a novel and effective long- term treatment modality. We will focus on developing a strategy to "re- engineer" the endothelium to preserve the homeostatic NO-ROS balance by using three complementary approaches : 1) augment endothelial cell- nitric oxide synthase gene expression to override the increased catabolism of NO, 2) decrease oxidative stress-mediated catabolism of NO by augmenting the expression of superoxide dismutase (an endogenous anti-oxidant), and 3) prevent the emergence of the dysfunctional phenotype by inhibiting the cycle of oxidative stress-induced apoptosis and regeneration of dysfunctional cells by endothelial cell-targeted expression of anti-apoptotic genes. The success of this approach is predicated upon the development of stably integrating viral vectors for quiescent cells and vascular cell-specific targeting technologies under active investigation in this Gene Transfer Program. The initial phases of the project will utilize technologies of endothelial cell specification in genetically engineered mice to characterize the pathobiological significance of the NO-ROS balance in atherogenesis and cardiac ischemia-reperfusion injury. Based upon this characterization, we will utilize the novel viral vectors developed within the Program to further test this hypothesis in the context of animal models that simulate the treatment of human coronary artery disease.

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- **Project Title: ETIOLOGY AND DEVELOPMENT OF CONGENITAL HEART DISEASE**

  Principal Investigator & Institution: Patterson, Donald F. Professor; Clinical Studies; University of Pennsylvania 3451 Walnut Street Philadelphia, PA 19104

  Timing: Fiscal Year 2003; Project Start 01-MAY-1977; Project End 30-JUN-2007

  Summary: (provided by applicant): One to two percent of human infants are born with congenital **heart disease** (CHD). The common anatomic forms of CHD include conotruncal defects (CTD) and patent ductus arteriosus (PDA). Studies in human families have shown that CTD and PDA tend to occur in a familial pattern, each type being specific to a particular family. While it is clear that genetic factors are important in the cause of these and other forms of congenital **heart disease,** the patterns of inheritance in human families tend to be complex, indicating that the combined effects

of genes at more than one gene locus are involved. Despite the recent advances in mapping and. characterizing the human genome, progress in identifying the gene defects responsible for congenital heart diseases, including PDA and CTD, has been slow. This is because of the apparent complexity of inheritance, and the lack of large well-characterized families in which it can be assumed that all affected individuals have the same underlying genetic form of CHD or other study population with reduced genetic heterogeneity. Because the genetic instructions for development of the heart have been highly conserved during evolution, studies of naturally occurring congenital **heart disease** in other mammalian species can provide valuable clues to the genes underlying human CHD. Studies in dogs are of particular interest because dogs tend to have the same anatomic forms as in humans and with a similar frequency. In previous studies under this grant, the genetic transmission and embryologic defects in CTD and PDA were characterized, verifying that they are specific inherited traits. As in humans, the genetics of canine CTD and PDA initially appeared complex. However, subsequent studies provided evidence that the number of genes underlying each of these two forms of CHD is few. Using DNA collected from previous family studies of CTD and PDA and the developing canine genome map, we conducted a whole genome study of CTD. A region of linkage to CTD was found on each of 3 different canine chromosomes. The proposed further studies are aimed at identifying chromosome regions linked to PDA in separate families, and at exploring the gene content of the CTD- and PDA-linked regions to identify the defective genes involved. The findings are expected to aid in the discovery of the corresponding molecular genetic defects in humans, leading to advances in genetic counseling, prenatal diagnosis, and treatment of these common birth defects.

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- **Project Title: EVALUATING REASONS FOR VARIATIONS IN LDL LEVELS**

  Principal Investigator & Institution: Ho, P Michael. Medicine; University of Colorado Hlth Sciences Ctr P.O. Box 6508, Grants and Contracts Aurora, CO 800450508

  Timing: Fiscal Year 2002; Project Start 01-APR-2002

  Summary: (provided by applicant): Ischemic **heart disease** is a major health problem in the United States where secondary prevention therapies have been demonstrated to improve morbidity and mortality. Multiple guidelines have been published which recommend cholesterol lowering for patients with ischemic **heart disease.** Despite these recommendations, wide variations exist in the achievement of these target LDL levels. The purpose of the current project is to identify patient characteristics, processes of care and structures of care that are associated with these variations in compliance of target LDL levels. A cross-sectional study design will be used. Information will be gathered from secondary data sources, using the Veterans Health Administration databases and through primary data collection, by surveying care providers. Thirty clinics or facilities and approximately 10, 000 patients will be included in the sample. Once identified, these factors can serve as the focus for future quality improvement projects to increase the proportion of ischemic **heart disease** patients who achieve a target LDL-cholesterol level of less than or equal to 100 mg/dl as proposed in the Healthy People objectives.

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- **Project Title: EVALUATION OF CHD IN MEN AND WOMEN PRIOR TO FIRST AMI**

  Principal Investigator & Institution: Yawn, Barbara P. Director of Research; Olmsted Medical Group Box 4300, 210 9Th St Se Rochester, MN 55904

Timing: Fiscal Year 2001; Project Start 29-SEP-2000; Project End 31-AUG-2003

Summary: Coronary **heart disease** (CHD) is the leading cause of death in women in the United States. The recent proliferation of studies of woman and **heart disease** suggest that women may receive less aggressive treatment and thereby missing the important pre- and peri-CHD diagnostic phase. A recent vignette study confirmed a gender bis in proposed evaluation rather than actual practice information. No studies have addressed the possible the possible existence of gender differences in the critical pre- CHD diagnostic phase nor incorporated observed practice data across the total spectrum of primary to tertiary care. This study will focus on the important pre-diagnostic or differential diagnosis (hypothesis activation) phase. We will describe and compare the recorded CHD-related symptoms and their evaluation as well as timing of the CHD diagnosis in women and men during the ten years prior to their first acute myocardial infarction (AMI). In addition, we will describe and compare the evaluation and treatment of CHD risk factors in the ten years prior to the first AMI. These data will allow us to identify gender differences in timing of CHD diagnoses prior to AMI and differences in physician response to men and women's symptoms. Our extensive data collection will allow exploration of the association of other factors such as risk status, co-morbidity and age with gender differences in CHD-related testing or treatment. Control subjects (both men and women) who do not have known CHD will provide information on the role of prior probabilities on non-cardiac disease in gender differences in the evaluation of possible cardiac symptoms. The retrospective of look-back design starting at the time of the subject's first AMI, assures that subjects have CHD and will achieve greater efficiency in design than would be possible using a prospective cohort study. It also avoids the Hawthorne effect that might be observed in a clinical trial. Using a unique community population-based dataset maintained for > 70 years on all residents of Olmsted County avoids referral bias and will allow access to data from the entire spectrum of care, from primary ambulatory to tertiary hospital care. The information obtained will help fill important gaps in existing knowledge regarding gender differences in the recognition and evaluation of CHD. In addition, we will provide specific information on current gender-related practice patterns. This information can be used to inform future practice recommendations.

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- **Project Title: EXERCISE TRAINING & THE MYOCARDIUM--CELLULAR ADAPTATIONS**

Principal Investigator & Institution: Moore, Russell L. Professor; Integrated Physiology; University of Colorado at Boulder Boulder, CO 80309

Timing: Fiscal Year 2001; Project Start 01-JAN-1990; Project End 31-MAR-2005

Summary: (Applicant's abstract): It is well known that endurance exercise training elicits uniformly favorable adaptations in myocardial performance. Training is known to alter the configuration of the ventricular action potential (AP) and the inotropic responsiveness of heart to changes in extracellular (Ca2+) and (Na+). To date, however, the cellular basis for these adaptations has not been clearly identified. In the last funding period of this project, we have identified training adaptations in both the transient (Ito) and sustained (Is) components of the composite, outward repolarizing K+ current in rat left ventricular (LV) cardiocytes. These repolarizing K+ currents play a central role in defining AP configuration, and may well be the cellular basis for the effect of training on AP characteristics. Recently, composite Ito and Ito currents have been resolved into at least 4 other discrete outward currents. In addition, some of these discrete currents and the K+ channel proteins that are thought to be responsible for those currents are known

to be differentially distributed across specific regions of the LV. In this project, Specific Aim 1 is to determine the effect of training on the specific current components that contribute to the composite Ito and Isus in rat LV using whole cell, electrophysiological techniques. Specific Aim 2 is to determine whether or not the effects of training on the specific currents that contribute to the composite transient and sustained currents are regionally distributed in the heart. Finally, we recently acquired intriguing preliminary evidence for a subtle training-induced decrease in intracellular Ca2+ buffering capacity. This type of adaptation could provide a cellular explanation for the consensus observation that training alters the inotropic responsiveness of heart to changes in extracellular (Ca2+) and (Na+). Specific Aim 3 is to use electrophysiological techniques and more refined fluorescence microscopic techniques to confirm or refute this potentially important finding. Exercise training is known to be effective in the prevention and treatment of wide variety cardio pathologic conditions. Elucidation of the cellular changes that underlie these positive adaptations may be of particular importance in the design, development, and implementation of molecular and pharmacological **heart disease** treatment and prevention strategies. This is particularly significant in view of the fact that **heart disease** claims more North American lives than any other disease.

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- **Project Title: EXERCISE/TRAINING IN CHILDREN WITH HEART DISEASE**

  Principal Investigator & Institution: Chang, Ruey-Kang R. Medical Doctor; Harbor-Ucla Research & Educ Inst 1124 W Carson St Torrance, CA 90502

  Timing: Fiscal Year 2002; Project Start 15-MAY-2002; Project End 31-DEC-2002

  Summary: (provided by applicant): The principal investigator will develop expertise in exercise testing and training of children and obtain the experience and skill necessary to initiate independent research projects during the award period. The principal investigator's long-term career goal as an academic pediatric cardiologist is to become a clinical investigator with a research focus in exercise physiology and cardiac rehabilitation. Exercise intolerance is an under-recognized chronic morbidity in children with **heart disease.** Poor exercise capacity leads to low self-esteem and poor quality of life. Children with single ventricle after the Fontan operation and children after orthotopic heart transplantation (OHT) are among children with the lowest exercise capacity. Whether exercise intolerance in these children is due to cardiopulmonary dysfunction or deconditioning from decreased physical activity remains unclear. The role of skeletal muscle and humoral responses in determining exercise capacity and the effect of exercise training in children with **heart disease** are unknown. They propose a prospective, randomized and controlled study to compare physical activity level of healthy children and children with Fontan or OHT, to evaluate the skeletal muscle, catecholamine and cytokines in relation to exercise capacity, and to determine the effect of exercise training on cardiopulmonary function, skeletal muscle and quality of life. In order to conduct this study, prepubertal children six to 13 years of age, including 40 children after Fontan, 40 children after OHT, and 40 healthy children will be recruited. Subjects will be randomized to a six-week endurance training program or computer workshops (no training). The project will be conducted over five years and training sessions will be held in the summer. The following tests will be performed before and immediately after exercise training is completed: lean body mass by dual energy X-ray absorptiometry (DEXA) scan, thigh muscle volume by magnetic resonance imaging (MRI), echocardiogram, pulmonary function test, cardiopulmonary exercise test, catecholamine and cytokine tumor necrosis factor (TNF)-alpha, IL-1beta, and IL-6 levels

at rest and during exercise, physical activity level by activity monitor and quality of life measures by a questionnaire. They will compare these measurements between children with training and children without training and compare the effect of exercise training on children with Fontan, OHT and healthy children. The study is designed to define important mechanisms of exercise intolerance among children with severe forms of **heart disease.** The results of exercise training will provide evidence related to the physiologic and psychosocial benefits of increased physical activity and form the basis for future research on cardiopulmonary rehabilitation for children with **heart disease.** The principal investigator will take advanced courses to enhance knowledge and skills needed for patient-oriented research and work with the mentors to conduct the proposed project during the award period.

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- **Project Title: FETAL STRESSORS ALTER LONG-TERM MYOCYTE /CORONARY GROWTH**

  Principal Investigator & Institution: Thornburg, Kent L. Professor; Oregon Health & Science University Portland, OR 972393098

  Timing: Fiscal Year 2002; Project Start 01-JUN-2002; Project End 31-MAY-2007

  Summary: Babies born small at full term are 3-5 times more likely to contract ischemic **heart disease** than are heavy babies born to non-diabetic mothers. An index of fetal growth, like birth weight, is an independent risk factor more powerful than the well-known risk factors endorsed by the American Heart Association. However, the biological link between prenatal undergrowth and the propensity to contract coronary disease is elusive. This application is based on the hypothesis that intrauterine stressors such as hypoxia, hypertension and volume load will cause adaptive compensations of the gene expression pattern that will "program" the fetus for immediate survival adaptation but put the offspring at risk for adult-onset disease. During the past funding period, it was discovered that angiotensin II does not stimulate hypertrophy in vivo or in vitro, quite contrary to current dogma based on rat data. The proposal contains 3 aims designed to uncover adaptive mechanisms that are likely to bring about a life long propensity to cardiovascular disease. 1) Determine the physiological, histological, cellular and biochemical response to pressure loading and anemia in the fetus and determine the relationship between the myocyte and coronary growth. If the heart is born with too few cells, it may be failure-prone. 2) Determine the effects of fetal stressors on adult heart function and coronary conductance. If conductance is too high, the coronary endothelium may suffer from shear stress fatigue. 3) Determine the role of a specific member of the mitogen activated protein kinase (MAPK) signaling cascade (MAP kinase kinase called MEK) in regulating fetal cardiac hyperplasia versus hypertrophic growth. MEK will be blocked in both in vivo and in vitro experiments while the heart is being stimulated to grow. If MEK is blocked, pressure- induced cardiomyocyte proliferation may be prevented.

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- **Project Title: FUNCTIONAL MR IN ISCHEMIC CARDIOMYOPATHY**

  Principal Investigator & Institution: Grayburn, Paul A. Professor; Baylor Research Institute 3434 Live Oak St, Ste 125 Dallas, TX 75204

  Timing: Fiscal Year 2002; Project Start 30-SEP-2002; Project End 31-JUL-2006

  Summary: (provided by applicant): Functional mitral regurgitation (MR) is a common complication of ischemic **heart disease.** Two large clinical trials confirmed an adverse

effect of functional MR on survival after a heart attack. However, studies in heart failure are small and mainly limited to patients with nonischemic cardiomyopathy. Recent animal studies have challenged the traditional concept that functional MR is a consequence of mitral annular dilation, instead suggesting that functional MR is due to leaflet tethering by outward expansion of the left ventricular wall (i.e. LV remodeling). This has critical implications regarding the correct surgical approach to correcting functional MR. TO date, no large prospective studies have examined the mechanism(s) of functional MR in ischemic cardiomyopathy, nor has the interaction between mechanism and prognosis been explored. This is a crucial knowledge gap because 1) 70% of heart failure cases are caused by ischemic **heart disease,** and 2) functional MR occurs in around 60% of patients with ischemic cardiomyopathy. This proposal aims to fill these gaps by defining the mechanism(s) of functional MR by transesophageal echocardiography in a large clinical trial of patients with ischemic cardiomyopathy. The following specific aims will be addressed: Aim 1: To define the mechanism(s) of functional MR in ischemic cardiomyopathyAim 2: To define the effect of therapy on mechanism and severity of functional MRAim 3: To evaluate the effect of functional MR on prognosis is ischemic cardiomyopathyAim 4: To evaluate the effect of myocardial viability on functional MR and its response to treatment We propose to accomplish these aims as a ancillary study to the Surgical Treatment of Ischemic Heart Failure (STICH) Trial. The STICH Trial will compare surgical revascularization versus medical therapy for treatment of heart failure in 2800 patients with ischemic cardiomyopathy, and therefore affords a unique opportunity to investigate the mechanism(s) of functional MR. Despite its known clinical utility of assessing the mechanism and severity of MR, TEE is not currently included in STICH.

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- **Project Title: GENES DIFFERENTIALLY EXPRESSED DURING HEART FAILURE**

  Principal Investigator & Institution: Gwathmey, Judith K. Professor of Medicine and Physiology; Gwathmey, Inc. 763 E Concord Ave Cambridge, MA 02138

  Timing: Fiscal Year 2003; Project Start 01-APR-2001; Project End 31-AUG-2005

  Summary: (provided by applicant): The American Heart Association estimated the cost of cardiovascular disease in the United States in 2000 to be at $326.6 billion. This figure includes health expenditures and lost productivity resulting from morbidity and mortality. Heart failure is not a disease of the elderly or persons who live unhealthy lifestyles. The highest incidence occurs between 25-45 years of age. Although more patients are surviving their first myocardial infarction, they go on to develop progressive left ventricular dysfunction and end-stage heart failure. As a result, the incidence of congestive heart failure is increasing. Changes in gene expression profiles between normal tissue and diseased tissue can lead to identification of novel drug targets and to the development of drugs that will be able to interfere with disease development. Our hypothesis is that altered gene expression is the basis of the structural and functional changes that accompany the development of **heart disease** and that changes in gene expression profiles are important indicators of specific disease stages of heart failure. We predict that changes in the expression profile of a critical set of genes will be important indicators and diagnostic markers of **heart disease.** We have found that about 30 percent of the genes identified in our differential screening have no informative similarity to known genes in any of the public databases. These genes are excellent candidates as drug targets and/or as possible diagnostic markers. Our specific aims are 1) To fully characterize selected genes based on their differential expression in heart failure; 2) To validate the potential targets identified in Phase 1 and prioritize these

gene targets; 3) To determine if the levels of the corresponding gene products (proteins) have changed in a manner similar to the change in RNA levels; 4) To link information on the differences in gene expression and protein levels with consequences in cardiac myocytes and muscle strips by using adenoviral vectors to infect myocytes with transgenes that are over-expressed or knocked down (i.e., antisense); 5) To determine if changes in the gene expression pattern that we have identified in the turkey model are also present in the hearts of human patients with end-stage heart failure; 6) To produce the first "human heart failure chip"; 7) To patent novel therapeutic targets that have been identified and validated

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- **Project Title: GENETIC ANALYSIS OF INHERITED CONGENITAL HEART DISEASES**

  Principal Investigator & Institution: Seidman, Christine E.; Harvard University (Medical School) Medical School Campus Boston, MA 02115

  Timing: Fiscal Year 2001

  Summary: (Adapted from the Applicant's Abstract) Congenital heart defects are common human malformations that cause significant morbidity, mortality, in addition to substantial social and economic costs. Birth defect registries indicated congenital heart defects occur in approximately 1 percent of human lie births and 10 percent of stillbirths (1). Over the past 30 years major advances have occurred in the diagnosis and management of heart defects in infants and children. Far less is known about the normal molecular signals or pathways that direct human cardiac morphogenesis, nor how and why these processes sometimes fail. Application of human genetic analysis to the study of inherited congenital **heart disease** has enormous potential to provide novel insights into these complex human processes. The focus of this application is to define the molecular causes of inherited human congenital heart defects. The investigators have recently identified three loci that cause human cardiac malformations. (1) Holt-Oram syndrome which maps tot chromosome 12q2 is caused by mutations in human TBX5. This congenital malformation causes skeletal and ventricular septal defects and sinus or atrio- ventricular septal defects, and sinus or atrio-ventricular nodal abnormalities that arise independent of septation defects). (2) The investigators have recently mapped gene defects that cause non-syndromic secundum atrial septal defects with associated atrioventricular conduction delays to chromosome 5q and have demonstrated the causal gene at this locus to be Nkappax2.5 (also termed hCSx). (3) The investigators have defined a locus on chromosome 5p that causes non-syndromic secundum atrial septal defects without conduction defects. Individuals affected by mutations in the 5q and 5p loci may have normal cardiac structure, atrial septal aneurysm, bicuspid aortic valve, persistent left superior vena cava, or more complex structural defects (such as tetralogy of Fallot). This variable expressivity combined with reduced penetrance of these gene mutations have partially obscured the familial (and genetic) nature of these congenital heart defects. The investigators' data and studies by others clearly demonstrate genetic heterogeneity of human congenital **heart disease.** Further identification and characterization of mutations in known disease genes and those yet to be defined should therefore provide a better understanding of human cardiac morphogenesis and the molecular basis of cardiac malformations. Development and characterization of animal models with these mutations should help explain the variable expression of these mutations. Ultimately these studies may also improve the understanding of non-familial congenital **heart disease.** The investigators propose to address these issues through the following specific aims: (1) characterize further the clinical manifestations of human

TBX5 and Nkappax2.5 mutations to elucidate structure/function relationships; (2) identify other gene defects that cause heritable cardiac malformations using positional cloning and candidate gene analyses; (3) engineer and characterize mice with human TBX5 mutations in other cardiac malformation genes; and (5) test the hypothesis that TBX5 is subject to allelic exclusion in some cells.

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- **Project Title: GENETIC ARCHITECTURE OF HEART DISEASE IN RURAL BRAZIL**

  Principal Investigator & Institution: Williams-Blangero, Sarah A. Scientis and Chair; Southwest Foundation for Biomedical Res San Antonio, TX 782450549

  Timing: Fiscal Year 2001; Project Start 30-SEP-2001; Project End 31-JUL-2006

  Summary: (provided by applicant): This project has the ultimate goal of elucidating the genetic architecture of Chagas' disease in a population residing in rural Brazil. A leading cause of **heart disease** throughout Latin America, it affects between 16 and 18 million individuals. In Brazil alone, approximately 10 percent of the population is seropositive for T. cruzi, the parasitic cause of the disease, with sub-populations experiencing seropositivity rates as high as 65 percent. Given the large pool of primary hosts for this zoonotic disease, complete eradication of Chagas' disease through control of the arthropod vector is unlikely. Research with humans and animal models indicates that there is variation in susceptibility to infection, and disease outcome, and that this variation may be due to genetic factors. Thus, this form of **heart disease** represents a complex phenotype with potential genetic determinants to both susceptibility to infection and differential disease pathogenesis. The proposed project will test two general hypotheses: 1) that host genetic factors influence susceptibility to infection with Trypanosoma cruzi, cardiac consequences of T. cruzi infection, and immunological correlates of Chagas' disease, and 2) that individual loci have detectable effects on these Chagas' disease related traits. In the process of testing these hypotheses, we will 1) evaluate each individual's genotype for approximately 382 polymorphic short tandem repeats (STRs) spread throughout the genome in order to generate a population specific 10cM map; 2) quantify the effects of genetic and shared environmental factors on T. cruzi infection, ECG variables, and immunological correlates of Chagas' disease in the well-characterized population of Posse; and 3) utilize linkage analysis to localize the specific genes influencing susceptibility to T. cruzi infection, ECG variables, and immunological correlates by performing a genome wide scan for linkage using a multipoint variance component method that is appropriate for extended pedigrees. This study will be the first genome scan for susceptibility to Chagas' disease.

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- **Project Title: GENETIC BASIS OF CONGENITAL HEART DISEASE**

  Principal Investigator & Institution: Benson, D W. Professor; Washington University Lindell and Skinker Blvd St. Louis, MO 63130

  Timing: Fiscal Year 2001

  Summary: Secundum atrial septal defect (ASD) is a common congenital heart defect accounting for 10% of isolated congenital **heart disease.** Some individuals with ASD have a family history of this defect; some familial ASD kindreds also have other congenital heart defects and co-existing atrioventricular (AV) conduction abnormalities. The genetic basis for these observations remains unclear. Preliminary studies on seven kindreds in whom ASD was transmitted as an autosomal dominant trait confirmed these observations and also identified loci on chromosome 5p and 5q through genetic

linkage analysis. In a kindred mapping to the 59 locus, the clinical status of all family members can be accounted for by a model of incomplete penetrance and variable expressivity. In three kindreds mapping to the 5q locus, there is full disease penetrance, variable expressivity. In three kindreds mapping to the 5q locus, there is full disease penetrance, variable expressivity and affected individuals have associated AV conduction abnormalities. Thus, familial ASD is genetically heterogeneous; reduced disease penetrance and variable expressivity occur in some kindreds. A genome-wide search for a third locus is underway in one kindred, and three other kindreds are being clinically evaluated. The following studies are proposed: 1. To identify additional kindreds in whom ASD appears to be inherited as an autosomal dominant trait and map the kindreds to known loci on chromosome 5p and 5q. 2. To refine the genetic map of the chromosome 5p and 5q loci, construct a physical map, identify candidate genes and screen them for mutations. 3. To perform a genome-wide search to identify additional for familial ASD in kindreds that do not map to chromosome 5q or 5p. 4. To make a mouse model of familial ASD. These specific aims have been developed based upon the rationale and feasibility demonstrated in preliminary data. The availability of multiple kindreds at different stages of genetic evaluation is key to ensuring successful completion of the proposed studies. Based on preliminary studies, at least three genes can cause ASD. To date there have been no report of a gene whose mutation causes a simple, common heart defect such as ASD. Identification of such genes would provide an important perspective of both normal and abnormal cardiac development.

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- **Project Title: GENETIC REGULATION OF CORONARY HEART DISEASE SUSCEPTIBILITY**

  Principal Investigator & Institution: Breslow, Jan; Rockefeller University New York, NY 100216399

  Timing: Fiscal Year 2001

  Summary: Atherosclerotic coronary **heart disease** (CHD) is the number one public health problem in the United States. CHD has been increasing in prevalence and with the aging of the baby boomers and the increase in major cardiovascular risk factors in the population (smoking, obesity, and physical inactivity) over the last 10 years the problem will be even worse in the 21st century. This is not just a U.S. problem, but is occurring world wide. The WHO has recently predicted that by 2020 **heart disease** will replace infectious disease world wide as the number one cause of disability expressed as years of healthy life lost to death or disease. CHD is a complex genetic disease with many genes involved and important gene-environment interactions. In this proposal we will use the facilities of the Rockefeller Hospital GCRC to identify and study genes and gene-diet interactions that are important in determining CHD susceptibility.

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- **Project Title: GLOBAL ANALYSIS OF TECHNOLOGICAL CHANGE IN HEALTH CARE**

  Principal Investigator & Institution: Kessler, Daniel P. Medicine; Stanford University Stanford, CA 94305

  Timing: Fiscal Year 2001; Project Start 01-JUL-1999; Project End 30-JUN-2003

  Summary: Relatively little is known about differences across countries in changes in medical technology, and the potentially important dynamic implications of these differences for changes in medical expenditures and health outcomes. We will develop a

collaborative, global research network to analyze technological change at the "micro" level. We will begin with an analysis of changes in heart attach care, an important health problem for aging populations worldwide, and will gradually expand this focused analysis. Our study has three specific aims: 1. To develop a sustainable global network of collaborating health care researchers with expertise in clinical medicine, economics, epidemiology, and related fields, able to conduct detailed, valid studies with comparable microdata and standardized methods. 2. To conduct a detailed, quantitative analysis of technological change in the treatment of heart attacks in the participating countries, resulting in a series of publications that describe and quantify in detail how technological change for this disease has differed across countries, the economical and regulatory factors influencing these treatment differences, and their consequences for disease outcomes and resource use. 3. To generalize these results, by (a) extending our methods to study the more prevalent but less severe forms of ischemic **heart disease,** (b) relating our disease-specific, "micro" findings to existing evidence on aggregate "macro" differences in the levels and trends in medical expenditures and health outcomes across countries, and (c) conducting exploratory studies to extend our methods to the analysis of a more chronic illness. Our longitudinal approach and our initial emphasis on a cure, severe illness are likely to make it easier for us to isolate the effects of medical technology from many important but (relatively) stable cross- country differences that confound cross-sectional and "macro" studies. We have conducted extensive preliminary studies to explore the feasibility of this collaborative approach, and their encouraging results have led to this proposal to support the coordination of an international research network on technological change in health care.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: GLUCOSE METABOLISM IN HYPERTROPHIED HEART IN ISCHEMIA**

  Principal Investigator & Institution: Del Nido, Pedro J. Professor of Surgery and Chair; Children's Hospital (Boston) Boston, MA 021155737

  Timing: Fiscal Year 2001; Project Start 01-APR-2000; Project End 31-MAR-2004

  Summary: Myocardial hypertrophy remains an important risk factor for pediatric cardiac surgery of congenital and acquired **heart disease.** Clinical and experimental studies have shown that hypertrophied myocardium exhibits a worse recovery of contractile function post-ischemia. Glucose transport/utilization by myocytes is critical for normal function, and during ischemia and early reperfusion. Exogenous glucose for glycolysis enters the cell via a transporter protein (GLUT-1 and 4 in the heart), and at physiologic glucose concentrations, glucose entry into the cell is rate-limiting for its subsequent metabolism. Using a model of pressure overload hypertrophy (aortic banding at 10 days of age), we have shown that in hypertrophied hearts, glucose transport across the sarcolemma in response to insulin is impaired and this change is associated with worse recovery after ischemic injury. Inversely, improving glucose uptake significantly improves post-ischemic recovery in hypertrophied hearts. We therefore hypothesize that impaired glucose transport into myocytes is in large part responsible for the decreased tolerance of hypertrophied myocardium to ischemia. Insulin insensitivity, with resultant lack of activation of glucose transporters and downregulation of glucose transporter expression occurs in conjunction with the development of uncompensated hypertrophy. Proteins such as tumor necrosis factor which are elevated in congestive heart failure, can inhibit insulin response and cause downregulation of glucose transporters in myocytes. The overall goal of this project is to increase our understanding of the role and mechanism responsible for decreased

glucose uptake in hypertrophied myocardium and to develop novel therapies to improve tolerance to ischemia. The P.I. has brought together a multidisciplinary group of investigators with expertise in the various aspects of the project. We will pursue three specific aims to determine the mechanism responsible for insulin insensitivity in the hypertrophied heart (AIM I); test the efficacy of interventions aimed at bypassing the functional defect in insulin signaling (AIM II); and determine the effect and mechanism of action of tumor necrosis on glucose uptake and glucose transporter expression in cardiac myocytes in culture (AIM III). We will use a model of pressure overload hypertrophy generated by aortic banding of neonatal rabbits. Non-invasive assessment of LV muscle mass with trans-thoracic echocardiography will be used to monitor the development of hypertrophy and progression to heart failure. The hearts will be studied after the development of moderate and severe hypertrophy in an isolated blood perfused heart preparation. We will also perform studies using cardiomyocytes in culture to determine the mechanism responsible for glucose transporter downregulation.

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- **Project Title: HEART DISEASE AND THE EMERGENCE OF MODERN EPIDEMIOLOGY**

  Principal Investigator & Institution: Oppenheimer, Gerald M. Health & Nutritional Sciences; Brooklyn College 2900 Bedford Ave New York, NY 11210

  Timing: Fiscal Year 2003; Project Start 15-AUG-2003; Project End 14-AUG-2005

  Summary: (provided by applicant): Epidemiology is currently the science of public health mad file major intellectual system, along with economics, for studying, justifying and developing public health policy. Despite its deep influence on medicine, science, health policy and American culture over the past 40 years, there is presently no systematic history of epidemiology in the United States, particularly for the second half of the 20th century. The objective of this proposal is to write a social and intellectual history of the origins, development, and impact of one of the most important areas of epidemiology during the last century, that of coronary **heart disease** (CHD). The work will begin early in the 20th century, just before the "epidemiological transition," when mortality from **heart disease** and cancer surpassed deaths from communicable disorders. With this introductory period (1900-1945) as prologue, the book will then focus on the emergence, after World War II, of an epidemiology capable of defining, modeling, and quantifying chronic (heart) disease and of proposing clinical interventions. That history will continue into the 1960s, when epidemiology began to emerge from relative obscurity to become a recognized science and a party to health policy decision-making; then to the 1970s when epidemiology, with a body of knowledge, training programs, textbooks and journals, became a fully formed discipline at the heart of public health. It will end in the 1990s, when the successful paradigm developed in the decades after World War II--the multiple risk factor, individual-level analysis closely associated with coronary **heart disease** epidemiology--was severely questioned by experts both within and outside the field. Data for this project will come from archival sources, the medical and epidemiological literature, taped interviews, and publications in the history of medicine, public health, statistics, insurance and risk.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: HEART DISEASE IN RHEUMATOID ARTHRITIS**

  Principal Investigator & Institution: Gabriel, Sherine E. Professor; Mayo Clinic Rochester 200 1St St Sw Rochester, MN 55905

Timing: Fiscal Year 2001; Project Start 01-SEP-2000; Project End 31-AUG-2005

Summary: (Applicant's Abstract) The hypotheses to be tested in this proposal are built on findings from two intriguing, but rather disparate lines of investigation. The first is the recent data suggesting that the excess mortality experienced by people with rheumatoid arthritis (RA) may result from increased rates of coronary **heart disease** (CHD) among RA patients compared to the general population. The second is the rapidly growing body of evidence indicating that chronic systemic inflammation (such as that which occurs in RA) plays an important role of chronic inflammation in CHD. We propose 3 specific aims to investigate this subject: First, we will use a cohort study to test the hypothesis that the incidence of acute MI (the central manifestation of CHD) is higher in RA subjects compared to controls. Second, we will identify high-risk RA subgroups and, using a novel adaptation of the case-cohort design, investigate interactions between RA and the major CHD risk factors (e.g. smoking, hyperlipidemia, exogenous estrogens). Third, we will conduct studies on archived autopsy heart tissue to test the hypothesis that coronary atherosclerosis is more extensive in RA subjects compared to matched controls. A unique set of circumstances allows us to address each of these aims rigorously and efficiently. We will incorporate and extend our already assembled population-based RA incidence cohort and identify validated definite acute MI outcomes using the cardiovascular surveillance techniques developed through out NIH-funded companion study, "Coronary Disease Morbidity and Mortality in a Population" (HL59205). Our population-based data resources, with essentially complete enumeration of a geographically defined population, allowed us to design an analytic plan which nearly quadruples the statistical power of our risk factor analyses, compared with typical cohort analyses. Third, the availability of extensive autopsy material (the autopsy rate in this community is four-fold higher than the national rate and all autopsies have been performed at the same center since 1930) provides us with a unique opportunity to assess the pathologic characteristics of atherosclerosis among RA subjects compared to controls. When combined with our experienced multidisciplinary investigative team, these resources lend us a capability, not available elsewhere, to rigorously examine the risks and determinants of coronary **heart disease** in patients with RA. These results will lay the foundation for a program of research aimed at elucidating the mechanisms for CHD in RA patients and at improving our understanding of the role of inflammation in the pathogenesis of CHD in the general population.

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- **Project Title: HEPARIN INDUCED THROMBOCYTOPENIA IN CHILDREN WITH CPB**

  Principal Investigator & Institution: Mullen, Mary; Children's Hospital (Boston) Boston, MA 021155737

  Timing: Fiscal Year 2001

  Summary: This study examines the prevalence of anti-heparin/PF4 antibodies as well as heparin-induced thrombocytopenia in pediatric patients undergoing cardiopulmonary bypass surgery. Specific Aims: - To determine the prevalence of heparin-dependent platelet-reactive antibodies in a pediatric population undergoing cardiopulmonary bypass surgery for congenital **heart disease.** - To determine the incidence of heparin-induced thrombocytopenia in a pediatric population after exposure to heparin during cardiopulmonary bypass surgery - To examine the role of heparin-induced thrombocytopenia in clinical thrombotic or thromboembolic events occurring in pediatric patients after heparin exposure for cardiovascular procedures Heparin is used

frequently as an antithrombotic agent during cardiovascular procedures in patients with congenital **heart disease.** An increasingly recognized complication of heparin therapy is the development of heparin induced thrombocytopenia (HIT) and related thrombosis. Although the occurence of heparin associated thrombocytopenia has been noted anecdotally in the pediatric population, no study has examined the prevalence of heparin associated antibodies or the incidence of the syndrome in children. Generally thought to be an almost unavoidable consequence of prolonged hospitalization and central venous/arterial monitoring, some portion of postoperative thrombus formation in pediatric patients may represent the HIT syndrome. Heightened recognition of the disorder may be of major clinical significance to avoid complications in patients with congenital **heart disease** who face repeated exposures to intravenous and high dose heparin during catheterizations and surgical procedures. It will be of import to identify subpopulations at risk for HIT in order to offer alternative anticoagulation strategies. Additional insights into immune mechanisms and pathophysiology of associated vascular thrombosis may derive from investigation of this syndrome in patients of varying age groups, including those without prior heparin exposure and without the presence of superimposed atherosclerotic or other vascular diseases.

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- **Project Title: HIGHLY AUTOMATED ANALYSIS OF 4-D CARDIOVASCULAR MR DATA**

  Principal Investigator & Institution: Sonka, Milan; Electrical and Computer Engineering; University of Iowa Iowa City, IA 52242

  Timing: Fiscal Year 2003; Project Start 01-AUG-2003; Project End 31-JUL-2007

  Summary: (provided by applicant): Magnetic resonance (MR) imaging plays an increasingly important role in the diagnosis and management of congenital **heart disease.** Often, cardiovascular MR data are analyzed qualitatively. Enhanced computing power and quantitative image analysis should provide rapid, comprehensive and reproducible assessment of 4-dimensional MR data sets. Starting with development of a general-purpose cardiac image segmentation method, this proposal focuses on two groups of subjects - postoperative tetralogy of Fallot patients and patients with connective tissue disorders. These patients require accurate, serial assessment of right ventricular function and aortic dimensions, respectively. In this proposal, an image analysis methodology based on Active Appearance Models (AAM) will be applied to both tasks. During training, the AAM is built automatically from manually analyzed image examples. In the analysis stage, the AAM allows fully automated segmentation of image data using its learned knowledge of allowed shapes and appearances of objects of interest - the ventricles and the thoracic aorta. Hypotheses driving this proposal are that a) active appearance model-based segmentation can provide automated, reproducible assessment of cardiovascular MR images and increase the information content of these studies by analyzing data in four dimensions (3-D + time), eliminating operator variability and labor-intensive border tracing, and that b) complete 4-D data sets of ventricular and aortic surface morphology and motion will provide novel quantitative indices of disease status. We propose to: I) Develop and validate an active appearance model (AAM) based method for 3-D and 4-D (3-D + time) segmentation of the left and right ventricles and the thoracic aorta from volumetric MR images. 2) Use the 4-D AAM segmentation approach to develop and validate a patient-specific method for highly automated and reproducible serial analysis of the right and left ventricles and the thoracic aorta. 3) Develop a set of novel quantitative indices of ventricular and aortic morphology and function and validate the reproducibility of these measurements in

postoperative tetralogy of Fallot patients and connective tissue disorder pa tients. The relationship between disease status, standard measures of ventricular function and aortic size, and novel quantitative indices will be assessed.

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- **Project Title: HOMEBOX GENES IN CARDIAC DEVELOPMENT AND FUNCTION**

  Principal Investigator & Institution: Kern, Michael J. Cell Biology and Anatomy; Medical University of South Carolina 171 Ashley Ave Charleston, SC 29425

  Timing: Fiscal Year 2001; Project Start 10-APR-1998; Project End 31-MAR-2002

  Summary: Heart disease is a major cause of morbidity and mortality; care for pediatric and adult heart patients represents a significant portion of the health care dollars spent in this country. Understanding the genetic processes of heart development and function will enhance future treatments for cardiac pathologies. Our work focuses on the Prx2 homeobox gene, a regulatory gene which encodes a DNA binding transcription factor. Prx2 is highly expressed in the embryonic heart, but decreases markedly at birth. The developing limbs and craniofacial mesenchyme also express Prx2. Gene targeting experiments have demonstrated that Prx2 is critical for correct limb and craniofacial morphogenesis as well as normal adult cardiac function. We have recently shown that the Prx2 mutant heart has defined alterations in cardiac contractility and action potential at the organ and cellular level. Thus the Prx2 gene targeted mouse is an excellent model to examine developmental gene regulation that is critical for normal cardiac function. The cardiac deficiencies in the Prx2 mutant may result from altered gene expression established during cardiac development and/or altered expression in the adult heart. Based on our data we hypothesize that Prx2 regulation of collagen expression in the primary mechanism whereby Prx2 is critical for normal cardiac development and function. Specific Aim Number 1 Test the hypothesis that mutations in Prx2 are implicated in human cardiovascular disease. Clone and chromosomally localize the human Prx2 gene to determine if it maps to any regions implicated in cardiovascular disease. Interbreed the Prx2 mutant allele onto different genetic strains of mice. Specific Aim Number 2 Test the hypothesis that collagen expression is regulated by Prx2 in the heart. Determine the pattern and level of collagen expression in the Prx2 mutant heart. Define when in development the Prx2 regulation of collagen begins. Establish and utilize two cell culture systems to examine the Prx2 regulation of collagen in vitro: cardiac fibroblastic cells from the mutant and wild type heart as well as NIH3T3 cells that are stably transfected with sense or antisense constructs of Prx2. Specific Aim Number 3 Evaluate the physiological parameters of altered cardiac function in the Prx2 mutant. Exercise and perform aortic banding of the Prx2 mice to assess cardiac function under stress. Evaluate the beta-adrenergic signalling pathway in the Prx2 mutant heart.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: HOMOCYSTEINE DIET STUDY: EFFECTS OF DIFFERENT FOLATE SOURCES**

  Principal Investigator & Institution: Farquhar, John W.; Stanford University Stanford, CA 94305

  Timing: Fiscal Year 2001

  Summary: Homocysteine is a factor found in blood that has been found to be elevated among adults with **heart disease,** similar to elevated blood cholesterol levels. This relatively risk factor for **heart disease** has been found to respond to the dietary vitamin known as either folate or folic acid. There are three different sources of folate in the diet.

One is from typical multivitamins, another is from breakfast cereals and others that have been with folate, and a third is from beans and leafy greens, which have naturally high folate levels. The purpose of this study was to determine which of those three sources of folate would be most effective in lowering homocysteine levels in adults with moderately elevated homocysteine. Sixty participants were enrolled and randomly assigned to be in one of four groups: Multivitamin, fortified foods, beans and greens, or a placebo group. After six weeks it was found that blood levels of homocysteine dropped the most for the group taking the multivitamin. The group eating the fortified foods did almost as well as the multivitamin group. The group eating beans and greens did no better than the placebo group, both showed negligible change. Knowledge of the form of folate found in each of these sources might explain the observed differences. In the multivitamins and fortified foods, the form of folate used is the smallest, best absorbed form of folate, whereas in the beans and greens the folate is found in a larger, more complex form, and is not as readily absorbed. In conclusion, two safe and readily available approaches for increasing folate in the diet were effective at lowering elevated homocysteine levels, but the approach of increasing daily consumption of beans and greens was not effective for improving this one particular **heart disease** risk factor.

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- **Project Title: HOSTILITY, MARITAL INTERACTION AND HEALTH IN AGING**

  Principal Investigator & Institution: Smith, Timothy W. Department Chair; Psychology; University of Utah 200 S University St Salt Lake City, UT 84112

  Timing: Fiscal Year 2001; Project Start 01-JUN-2001; Project End 31-MAY-2005

  Summary: Hostility confers increased risk of coronary **heart disease,** presumably through the mechanism of cardiovascular reactivity to interpersonal stressors. Marriage is an important context for this mechanism. However, an adult developmental perspective suggests that marital conflict may be a more central issue for trait hostility among middle- aged spouses, whereas the stress of collaboration may be important for hostility in older couples. Guided by a model of individual, spouse, and couple effects of hostility, the proposed study examines the effects of hostility on immediate behavioral and psychophysiological responses to marital conflict and collaboration, and on health outcomes of ambulatory blood pressure, coronary artery disease, marital adjustment, and cognitive functioning. The major aims are to examine (a) how the effect of hostility on behavioral and cardiovascular responses may differ for middle-aged and older adults, during conflict and collaborative problem solving, (b) the effect of hostility on ambulatory blood pressure and coronary artery disease, and (c) the role of hostility in the frequency and quality of collaborative problem solving. One-hundred and fifty middle-aged (40-50 years) and 150 older married couples (60-70 years) will be involved in a 4-day study. Hostility will be measured in a multi-method approach with interview, self-report, and spouse report measures. Marital interaction will be examined as couples discuss a source of marital conflict and solve a planning task. Interaction will be coded for components typical of interactions of hostile persons and also detrimental to collaborative cognition. Psychophysiological reactivity will be examined via blood pressure, heart rate, and impedance cardiography during the two tasks. The effects of hostility will be examined on health outcomes such as coronary artery disease (assessed via computed tomography) ambulatory blood pressure, marital adjustment, and general cognitive function (e.g., fluid and crystallized intelligence). The long-term goal of the research is to identify potentially modifiable determinants of cardiovascular risk, marital adjustment, and cognitive aging in adulthood.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: IDENTIFYING A GENE FOR CANINE CARDIOMYOPATHY**

  Principal Investigator & Institution: Jakobs, Petra M. Medicine; Oregon Health & Science University Portland, OR 972393098

  Timing: Fiscal Year 2002; Project Start 10-JUL-2002; Project End 30-JUN-2006

  Summary: (provided by applicant): Atrial fibrillation (AF) causes significant morbidity, disability, and mortality related to **heart disease** and stroke in the human population. Dilated cardiomyopathy (DCM) is characterized by ventricular dilatation and systolic contractile dysfunction and is an important cause of heart failure. In some canine breeds, DCM is a relatively common, lethal disease. A recent study of 500 Irish Wolfhounds (IW) found that 24% had DCM; 88% of these affected dogs also had AF. The DCM/AF phenotype appears to be inherited as an autosomal dominant trait. We have collected DNA and clinical data from a large family of IW in which AF and progressive DCM is segregating. Our hypothesis is that a mutation in a single gene causes DCM/AF in IW, and we propose to map this gene by linkage analysis and to use the tools of positional cloning and candidate gene analysis to identify the gene. One of the most common genetic causes of human DCM is mutation in the lamin A/C gene. The phenotype caused by lamin A/C mutations in such patients also includes conduction system disease and therefore resembles the DCM/AF phenotype in IW. In preliminary studies, we have excluded lamin A/C as the locus of the defect in IW. Hence our proposed work presents an ideal opportunity to identify a novel DCM/AF disease gene. If successful, this may lead to improved knowledge of the mechanisms of DCM and AF in humans.

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- **Project Title: IMMUNOLOGIC BASIS OF CORONARY DISEASE IN WOMEN**

  Principal Investigator & Institution: Reis, Steven E. Medicine; University of Pittsburgh at Pittsburgh 350 Thackeray Hall Pittsburgh, PA 15260

  Timing: Fiscal Year 2001; Project Start 01-MAY-2001; Project End 30-APR-2003

  Summary: (Abstract from University of Pittsburgh application) This reapplication proposes to extend the follow-up of the Women's Ischemia Syndrome Evaluation (WISE) patients for a minimum of 5 years and is being submitted jointly with the applications, "Altered Renin Angiotensin System as a Mechanism for Coronary Microvascular Dysfunction in Women" (C. Pepine PI) and "Immunologic Basis For Coronary Disease in Women" (S.Reis OI). The WISE contract began in September 1996 as a 4-center study to 1) optimize symptom evaluation and diagnostic testing for ischemic **heart disease** in women: 2) explore mechanisms for symptoms and myocardial ischemia in the absence of epicardial coronary artery stenoses: and 3) evaluate the influence of reproductive hormones on symptoms and diagnostic test response. An extensive contemporary database has been assembled on 936 women referred for coronary angiography because of suspected ischemia. Data include demographic, clinical, symptomatic, functional, and psychosocial variables. Coronary angiography and ventriculography data, brachial artery reactivity testing, ECG monitoring, and blood determinations are all assessed by core laboratories, Site-specific-innovative technologies have been used to develop potential markers of myocardial ischemia. We seek to 1) Determine the incremental long- term prognostic value of novel testing developed in WISE: 2) Develop sex- specific incremental outcome models to evaluate the prognostic value of female reproductive variables: 3) Assess the incremental cost effectiveness and resource of the WISE innovative testing techniques as compared with traditional tests: 4) Continue ongoing analyses and ancillary projects, collaborate with other WISE investigators' R01s submitted in this cluster and maintain a WISE database

and infrastructure to facilitate further investigations into the mechanisms underlying ischemia syndromes in women. To address these aims, a longer follow-up is necessary. Follow-up will consist of annual telephone contacts by experienced site coordinators. WISE will continue to use the well-established methods to implement study coordination, data management, quality control, statistical analyses, and manuscript preparation. Cox regression models will be used to analysis events with demographics risk factors, diagnostic testing and reproductive factors considered as explanatory variables. A hybrid decision model will be used that compares resource use patterns and sums cost estates. The results of these studies will enhance our understanding of both the significance and pathophysiology of ischemic **heart disease** in women and serve as a foundation for diagnostic and therapeutic clinical trials aimed at reducing disease-related morbidity and mortality.

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- **Project Title: IMPACT OF CHD RISK PERCEPTION ON HEALTH BEHAVIORS AND O***

  Principal Investigator & Institution: Barnhart, Janice M. Epidemiology & Social Medicine; Yeshiva University 500 W 185Th St New York, NY 10033

  Timing: Fiscal Year 2002; Project Start 15-MAY-2002; Project End 30-APR-2006

  Summary: (Applicant's abstract) The overall goal of the proposed study is determine if perceived risk of **heart disease** among postmenopausal women enrolled in the Observational Study (OS) of the Women's Health Initiative (WHI) in the New York Clinical Center, differs by race, and if risk perception is related to health behaviors. The applicant's long-term career goals are to become an independent investigator with special scientific interest in determining the key psychosocial contributors to racial and gender disparity in **heart disease** morbidity and mortality. In the proposed study, the first phase will be to develop and pilot test a methodologically sound instrument that measures perceived risk of **heart disease.** The second phase will be to administer this instrument to 300 White, African-American and Hispanic women at higher risk of **heart disease** because of smoking status, hypertension, diabetes or high cholesterol (requiring pills) and to determine the relationship of risk perception to risk reduction and other specific health behaviors. There will be 2 annual follow-ups to determine if changes in risk perception or health behavior occur. Data collected will be used as the basis of an independent grant application to expand this study to additional WHI centers and to provide information for future cardiac interventions using culturally- sensitive risk communication strategies to lessen the disease burden in African Americans and other population at high-risk for **heart disease.** The applicant's immediate career goals are to devote no less than 75 percent of her time to a highly structured and intensely focused research experience, over the next 4 years, to nurture development of her theoretical and practical skills in research design, implementation and analysis and in survey instrument development and test construction. This research and career development plan includes: 1) formal graduate course work in biostatistics, test construction, and decision analysis; 2) mentoring from the WHI Principal Investigator (PI) at the study site and investigators who have done pioneering work in the emerging field of risk perception in diabetes; 3) becoming thoroughly versed in the WHI operations; 4) having a formal evaluation by a Trainee Advisory Committee (TAC). The applicant will work closely under the guidance of the primary mentor (WHI PI). She will learn the rigorous methods employed in the conduct of a major, multi-center study, the WHI, will learn about the rigorous process of identifying and adjudicating coronary outcomes, and about procedures to assure research integrity.

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- **Project Title: INFLAMMATORY RESPONSE TO FETAL CARDIAC SURGERY**

  Principal Investigator & Institution: Malhotra, Sunil P. Surgery; University of California San Francisco 500 Parnassus Ave San Francisco, CA 94122

  Timing: Fiscal Year 2001; Project Start 01-APR-2001

  Summary: This research endeavor is aimed at understanding the effects of cardiac bypass on the fetus, with the hope that this knowledge will contribute to the development of safe corrective surgery for congenital **heart disease** in the human fetus. The advantages of early surgical intervention are obvious in defects that would require a much more challenging repair after birth. Prenatal repair would prevent the subsequent development of complex lesions during the fetal period. However, a detailed understanding of the effect of cardiac surgery and extracorporeal circulation on the fetus is required. The focus of this project is to identity the inflammatory mediators responsible for the fetal and placental pathophysiologic response to cardiac bypass using a fetal primate model. The role of cytokines, eicosanoids, and autocoids as candidate mediators of fetal and placental dysfunction will be examined. The effectiveness of attenuating this response with pharmacological antagonists of these putative mediators will also be explored.

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- **Project Title: INTEGRATED MECHANISM OF ISCHEMIC MITRAL REGURGITATION**

  Principal Investigator & Institution: Levine, Robert A. Professor; Massachusetts General Hospital 55 Fruit St Boston, MA 02114

  Timing: Fiscal Year 2001; Project Start 01-JUL-1987; Project End 30-JUN-2003

  Summary: (Adapted from Applicant's Abstract): An integrated mechanism that explains mitral valve function based on the three-dimensional geometry of the leaflet attachments has been useful in understanding systolic anterior motion of the mitral valve. A conceptually related problem is the mechanism of ischemic mitral regurgitation (MR), a common complication of coronary artery disease that conveys adverse prognosis. Its mechanism and therapy remain controversial. This proposal aims to address the hypothesis that the mechanism of ischemic mitral regurgitation can be understood in terms of an altered force balance on the mitral leaflets in systole: a combination of increased tethering forces, restraining the leaflets from closing, and resulting from an altered three-dimensional geometry of leaflet attachments, and decreased ventricular forces acting to close the mitral leaflets. This mechanism will be studied using two recently developed noninvasive physiologic tools in experimental and clinical settings: three-dimensional echocardiographic reconstruction, to provide repeated, detailed and complete assessment of the geometric changes capable of altering the force balance on the leaflets; and the Doppler flow convergence technique, which can describe variations in the MR orifice area within the cardiac cycle to provide mechanistic insight. Elucidating this mechanism can suggest practical clinical approaches for reducing mitral regurgitation at the time of surgical coronary revascularization. Preliminary studies have successfully applied these research techniques in acute and chronic animal models of global and segmental ischemia as well as in patients; they have also suggested that 3D echo findings can guide the development of interventions that restore a more favorable leaflet tethering configuration to reduce or eliminate MR. Specific and measurable factors that will be

tested in subhypotheses include the effect on MR of altering the three-dimensional geometry of mitral leaflet attachments, decreasing left ventricular force, increasing mitral leaflet and chordal length, reducing mitral annular size by ring implantation (which may not improve or may even exacerbate the abnormal tethering geometry), and reducing ventricular size through left ventricular remodeling surgery or angiotensin converting enzyme inhibition. These factors will be addressed in a series of in vivo models of ischemic MR that reflect the heterogeneity of wall motion abnormalities in patients with ischemic **heart disease,** and dissect out the rule of the different factors in a controlled manner to study the detailed force balance on the mitral leaflets.

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- **Project Title: INTERMITTENT HYPOXIA: CV IMPACT AND BIOLOGIC MARKERS**

  Principal Investigator & Institution: Rodway, George W. Acute/Tertiary Care; University of Pittsburgh at Pittsburgh 350 Thackeray Hall Pittsburgh, PA 15260

  Timing: Fiscal Year 2003; Project Start 01-MAY-2003; Project End 30-APR-2005

  Summary: (provided by applicant): It is estimated that 12-20 million people in the United States have obstructive sleep apnea (OSA), a disorder typified by nightly intermittent hypoxia (IH) exposure due to upper airway obstruction during sleep. Epidemiologic, retrospective, and cross-sectional studies have identified associations between OSA and an increased risk for cardiovascular disease, e.g., stroke, ischemic **heart disease,** and systemic hypertension. There is emerging evidence that IH may be a more potent sympathetic nervous system stimulus than continuous hypoxia (CH), and that the pattern of hypoxia (repetitive or continuous) is a critical factor in determining physiologic response. Prior studies indicate that the episodic reoxygenation that occurs during IH represents an oxidative stress (OS) that causes cellular generation of reactive oxygen species. Additional studies suggest that OS is likely to be influential in the transcriptional activation of specific genes through which hypoxia influences adaptive (or maladaptive) responses. There are major gaps in knowledge regarding the linkage of genomic responses to IH with physiological adaptation. The proposed study represents an initial step in increasing understanding of the physiological importance of episodic periods of IH and CH on selected cardiovascular variables (blood pressure [BP], heart rate [HR]) and the potential modulator roles of OS and Hypoxia-inducible factor-1 alpha (HIF-1alpha) transcription activation. The study will use a within-subjects repeated measures design. Normal subjects will be enrolled to avoid potential confounders due to the underlying disease process. Each subject will participate in three blocks (control, IH, CH). During the IH and CH blocks, BP, HR, exhaled biomarkers (nitric oxide [NO], carbon monoxide [CO]), and circulating biomarkers of HIF-1alpha and heme-oxygenase-1 (target gene controlling cellular generation of CO) will be measured. Statistical analysis will include repeated measures analysis of variance (ANOVA), correlations, and linear regression.

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- **Project Title: INTERMOUNTAIN PEDIATRIC HEART DISEASE RESEARCH CENTER**

  Principal Investigator & Institution: Minich, L L. Pediatrics; University of Utah 200 S University St Salt Lake City, UT 84112

  Timing: Fiscal Year 2001; Project Start 01-SEP-2001; Project End 31-AUG-2006

Summary: (provided by applicant) **Heart disease** is a leading cause of morbidity and mortality in children. A network of research centers with both a sufficient clinical population and team of researchers trained to conduct multi-center trials could provide standardized, blinded, quality research that has the statistical power to change the way children with **heart disease** are managed. Primary Children?s Medical Center, located in Salt Lake City, Utah, is uniquely qualified to participate as a research center within such a network. It serves as the leading referral hospital for children with **heart disease** in the Intermountain West. The overall goal of the proposed Intermountain Pediatric **Heart Disease** Research Center is to collaborate in prospective, randomized, multi-institutional trials that provide robust data leading to the advancement of knowledge regarding pediatric **heart disease** and evidence-based pediatric medicine. The specific aims of the Intermountain Pediatric **Heart Disease** Research Center are: to propose scientifically sound, statistically robust research protocols that can be performed within the Pediatric **Heart Disease** Clinical Research Network; to enroll eligible children at Primary Children?s Medical Center; and to collect complete data and readily communicate results within the Pediatric **Heart Disease** Clinical Research Network so as to facilitate the rapid dissemination of research findings. We propose two clinical studies for the Pediatric **Heart Disease** Clinical Research Network. The overall goal of protocol 1, "Efficacy of Abciximab in Treating Children with Large Coronary Aneurysms," is to compare the effectiveness of abciximab added to conventional therapy versus conventional treatment alone for dissolving or preventing thrombi and for facilitating remodeling in large coronary artery aneurysms attributed to Kawasaki disease, The overall goal of protocol 2, "Decellularized Human Allograft Valve Preparation (Cryovalve SGR) versus Standard Cryopreserved Valved Allografts for Vascular Reconstruction in Children," is to determine the function and long-term durability of decellularized human allograft valve preparation versus the current state-of-the- art cryopreserved valved allograft. The investigators agree to enthusiastically participate in and diligently adhere to any protocol selected by the Pediatric **Heart Disease** Clinical Research Network. The applicant organization, Primary Children?s Medical Center, agrees to the per patient capitation of operational costs for the study protocols selected by the Pediatric **Heart Disease** Clinical Research Network.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ISCHEMIA/REPERFUSION INJURY IN DIABETIC HEART**

  Principal Investigator & Institution: Mcdonagh, Paul F. Professor; Surgery; University of Arizona P O Box 3308 Tucson, AZ 857223308

  Timing: Fiscal Year 2001; Project Start 01-AUG-1997; Project End 31-JUL-2003

  Summary: Diabetes is now considered a prime risk actor for cardiovascular disease, particularly ischemic **heart disease.** The risks for myocardial infarction, reinfarction, ischemic heart failure, stroke and the associated mortalities are all significantly increased in diabetes. The pathobiology underlying the excessive and severe ischemic **heart disease** in diabetes is unclear. Myocardial ischemia-reperfusion (I'R)injury involves both early and late phases. In the early phase, the initial deposition of leukocytes amplifies cardiac injury via an acute inflammatory response. The initial step in acute inflammation is leukocyte, particularly PMN, deposition in the coronary microcirculation. It is not clear how PMNs initially accumulate in the microcirculation, but recent studies suggest that this step is amplified in the diabetic heart following ischemia. Once accumulated, the PMNs activate and produce oxygen free radicals, causing further damage to the vasculature and myocytes. Following ischemia, if PMN deposition is excessive in the diabetic coronary microcirculation an(LIor if diabetic

PMNs are hyper-responsive to cytokines released from ischemic tissue, then the severity of leukocytemediated reperfusion injury may be excessive as well. In this project, we will test the hypothesis that diabetes causes alterations in both the blood and the coronary blood vessels. These alterations set the stage for an excessive leukocyte-mediated reperfusion injury in the diabetic heart. If so, then pharmacologically blocking early PMN-mediated inflammation will reduce reperfusion injury and improve the recovery of myocardial contractile function. We will first investigate specific mechanisms, suspected to cause the excessive blood-coronary microvessel interactions observed in diabetes. We will then compare leukocyte adhesion protein characteristics and the "reactivity" of PMNs to stimulation in Type I and Type II diabetic animals and in patients with Type II diabetes. We will determine if platelets and plasma complement, modulate leukocyte function and leukocyte reactivity in diabetes. The therapeutic potential of limiting leukocyte-mediated inflammation in diabetes will be evaluated. Those pharmacologic agents and antibodies that prove to attenuate the early PMN-mediated response will be tested for efficacy to improve the recovery of myocardial contractile function in the diabetic. The lessons learned from these studies will aid in developing improved therapies to reduce the excessive ischemic **heart disease** observed in diabetes.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ISCHEMIC HEART DISEASE IN WOMEN--CLINICAL CENTER**

  Principal Investigator & Institution: Pepine, Carl J. Professor and Chief; Medicine; University of Florida Gainesville, FL 32611

  Timing: Fiscal Year 2001; Project Start 03-SEP-1996; Project End 30-JUN-2002

  Summary: The primary objective of the Women's Ischemic Syndrome Evaluation Study (WISE)- previously referred to as Evaluation of Ischemic **Heart Disease** in Women (EIHDW) under RFP NHLBI-HC-94-13, is to perform clinical studies to improve the diagnostic reliability of cardiovascular testing in evaluation of ischemic **heart disease** in women. Innovative approaches proposed in evaluation of ischemia will include physiologic or functional measurements such as impaired metabolism, perfusion, or endothelial function as well as assessment of epicardial coronary arteries by angiography. Objectives of this study are to develop safe, accurate, and cost effective diagnostic approaches for evaluating women with suspected ischemic **heart disease,** and to determine the frequency of myocardial ischemia in the absence of significant epicardial coronary stenosis, as well as the frequency of non-ischemic or non-cardiac chest pain. Key to these goals is the improved understanding of chest pain in women. Whether there are angina equivalents that are more accurate clinical descriptors of myocardial ischemia in women will be investigated. WISE will use new or innovative techniques such as Doppler flow, contrast perfusion or indirect measures of the microcirculation to assess myocardial function, perfusion, coronary tone and anatomy, or endothelial function, and to correlate these results with coronary arteriography. The study consists of four clinical centers that will evaluate innovative diagnostic methods designed to improve the diagnostic reliability of cardiovascular testing in evaluation of ischemic **heart disease** in women. The clinical centers will evaluate diagnostic methods and perform uniform study protocols, including angiography, on 234 participants over three years. The centers will perform analysis of the data in the fourth year. One clinical center will provide central collection, management, and analysis of uniform data and research evaluation data obtained by all of the WISE Clinical Centers. The period of performance is from September 3, 1996 through August 31, 2000.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: JACKSON HEART STUDY--COORDINATING CENTER**

  Principal Investigator & Institution: Garrison, Robert J. None; Jackson State University 1400 John R. Lynch St Jackson, MS 39217

  Timing: Fiscal Year 2001; Project Start 01-JUN-1999; Project End 31-MAY-2005

  Summary: Despite encouraging declines over the past three decades, cardiovascular disease (CVD) remains the number one cause of death in the U.S. A number of risk factors for coronary **heart disease** (CHD) and stroke have been identified; however, relatively few population-based studies have examined CVD in a large group of African-Americans. Existing evidence indicated that death rates in Mississippi are the highest in the nation and particularly high among African-Americans. Between 1980 and 1995, the decline in CVD death rates has been the slowest among African-American men and women in Mississippi relative to other groups in the state and nation. The Jackson Heart Study (JHS) is a single-site prospective epidemiologic investigation of CVD among approximately 6,500 African-Americans ages 35 to 84, from Jackson, Mississippi metropolitan area. The primary objective of the JHS is to investigate the causes of CVD in African- Americans to learn how to best prevent this group of diseases in the future. More specific objectives include: (1) Identifying factors which influence the development and worsening of CVD in African-Americans, with an emphasis on Manifestations related to high blood pressure (such as enlargement of the left ventricle of the heart, coronary artery disease, heart failure, stroke and disorders affecting the blood vessels of the kidney). (2) Building research capabilities in minority institutions at the undergraduate and graduate level by developing partnerships between minority and majority institutions and enhancing participation of minority investigators in large-scale epidemiologic studies. (3) Attaching minority students to and preparing them for careers in public health and epidemiology. This project serves as the Coordinating Center where all data collected during the study will be managed, analysis of the data will be performed, and community involvement will be coordinated.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: JACKSON HEART STUDY--EXAM CENTER**

  Principal Investigator & Institution: Jones, Daniel W. Professor; Medicine; University of Mississippi Medical Center 2500 N State St Jackson, MS 39216

  Timing: Fiscal Year 2001; Project Start 01-JUN-1999; Project End 31-MAY-2005

  Summary: Despite encouraging declines over the past three decades, cardiovascular disease (CVD) remains the number one cause of death in the U.S. A number of risk factors for coronary **heart disease** (CHD) and stroke have been identified; however, relatively few population-based studies have examined CVD in a large group of African-Americans. Existing evidence indicated that death rates in Mississippi are the highest in the nation and particularly high among African-Americans. Between 1980 and 1995, the decline in CVD death rates has been the slowest among African-American men and women in Mississippi relative to other groups in the state and nation. The Jackson Heart Study (JHS) is a single-site prospective epidemiologic investigation of CVD among approximately 6,500 African-Americans ages 35 to 84, from Jackson, Mississippi metropolitan area. The primary objective of the JHS is to investigate the causes of CVD in African- Americans to learn how to best prevent this group of diseases in the future. More specific objectives include: (1) Identifying factors which influence the development and worsening of CVD in African-Americans, with an emphasis on Manifestations related to high blood pressure (such as enlargement of the left ventricle of the heart, coronary artery disease, heart failure, stroke and disorders affecting the

blood vessels of the kidney). (2) Building research capabilities in minority institutions at the undergraduate and graduate level by developing partnerships between minority and majority institutions and enhancing participation of minority investigators in large-scale epidemiologic studies. (3) Attaching minority students to and preparing them for careers in public health and epidemiology. This project serves as the Coordinating Center where all data collected during the study will be managed, analysis of the data will be performed, and community involvement will be coordinated.

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- **Project Title: JACKSON HEART STUDY--TRAINING CENTER**

  Principal Investigator & Institution: Srinivasan, Asoka; None; Tougaloo College Tougaloo, MS 39174

  Timing: Fiscal Year 2001; Project Start 01-JUN-1999; Project End 31-MAY-2005

  Summary: Despite encouraging declines over the past three decades, cardiovascular disease (CVD) remains the number one cause of death in the U.S. A number of risk factors for coronary **heart disease** (CHD) and stroke have been identified; however, relatively few population-based studies have examined CVD in a large group of African-Americans. Existing evidence indicated that death rates in Mississippi are the highest in the nation and particularly high among African-Americans. Between 1980 and 1995, the decline in CVD death rates has been the slowest among African-American men and women in Mississippi relative to other groups in the state and nation. The Jackson Heart Study (JHS) is a single-site prospective epidemiologic investigation of CVD among approximately 6,500 African-Americans ages 35 to 84, from Jackson, Mississippi metropolitan area. The primary objective of the JHS is to investigate the causes of CVD in African- Americans to learn how to best prevent this group of diseases in the future. More specific objectives include: (1) Identifying factors which influence the development and worsening of CVD in African-Americans, with an emphasis on Manifestations related to high blood pressure (such as enlargement of the left ventricle of the heart, coronary artery disease, heart failure, stroke and disorders affecting the blood vessels of the kidney). (2) Building research capabilities in minority institutions at the undergraduate and graduate level by developing partnerships between minority and majority institutions and enhancing participation of minority investigators in large-scale epidemiologic studies. (3) Attaching minority students to and preparing them for careers in public health and epidemiology. This project serves as the Coordinating Center where all data collected during the study will be managed, analysis of the data will be performed, and community involvement will be coordinated.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MCP-1 INDUCED GENE EXPRESSION IN CARDIOVASCULAR DISEASE**

  Principal Investigator & Institution: Kolattukudy, Pappachan E. Professor and Directpr; Neurobiotechnology Center; Ohio State University 1800 Cannon Dr, Rm 1210 Columbus, OH 43210

  Timing: Fiscal Year 2002; Project Start 20-JUL-2002; Project End 31-DEC-2002

  Summary: (provided by applicant): Many recent reports provide strong evidence that MCP- 1 plays a major role in the development of ischemic **heart disease** (IHD) that is responsible for the majority of the 5 million human heart failure cases in the US, but the underlying mechanism is not known. We postulate that MCP-1-induced gene expression changes in the monocytes and the consequent production of cytokines and

other biologically active molecules, as well as possible direct effects of MCP- 1 on the major cell types in the myocardium can lead to the development of IHD. IHD probably develops as a net consequence of the interplay of these events which involve many cell types and many biologically active molecules. Therefore in vitro studies on isolated cells cannot accurately reflect the interactions involved in IND. On the other hand a genomic approach can discover the genes whose altered expression in the heart leads to the development of IHD. Such an approach, although not possible on humans, can be done on a suitable animal model. We have developed a transgenic murine model that faithfully recapitulates most features of human IRD. Use of this model has a high likelihood of discovering novel genes as illustrated by our discovery of a previously unknown MCP-1-induced protein (MCPIP). With this overall objective we propose to pursue the following specific aims: 1.) Identify gene expression changes that occur in the ventricle during IHD development induced by targeted expression of MCP 1 in the CMC of transgenic mice using gene chip technology. 2.) Determine the function of the novel MCPIP: a) Determine whether a) MCPIP expression causes cell death, b) this death is enhanced by MCP-l and its receptor CCR2, c) whether the cell death shares features characteristic of apoptosis, d) MCPIP has a transcription factor-like activity and whether structural features required for this activity are also required for the cell death-inducing activity. 3.) Assess the role of apoptosis in the development of MCP 1-induced cardiovascular disease by testing whether a) prevention of monocyte apoptosis by targeted expression of Bc12 or b) prevention of apoptosis by absence of Fas or expression of sFas in the myocytes would delay or rescue the development of the disease. The approach proposed here will identify MCP-l induced alterations in the expression of known genes not previously implicated in IHD and novel genes of unknown function that are involved in IHD. These genes are likely to reveal novel targets for intervention in IND and genes with prognostic value.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MECHANISMS UNDERLYING PSYCHOSOCIAL ASSOCIATIONS WITH IHD**

  Principal Investigator & Institution: Kaplan, George A. Professor and Chair; Epidemiology; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, MI 481091274

  Timing: Fiscal Year 2001; Project Start 01-JUL-1990; Project End 31-AUG-2002

  Summary: (Adapted from Investigator's Abstract) Considerable epidemiologic evidence suggests that a variety of behavioral, psychosocial, and socioeconomic factors are associated with ischemic **heart disease.** However, little is available concerning the pathophysiologic pathways that link these factors with clinically important events, on the impact of changes in these factors on morbidity or mortality, or subclinical manifestations of atherosclerotic disease. Little is also known about the contributions which these factors make to explaining cardiovascular disease in women or sex differences in CVD in later life. The Kuopio Ischemic **Heart Disease** Risk Factor Study (KIHD) is the most comprehensive, population-based study to prospectively assess the association between behavioral, psychosocial, and socioeconomic factors and the extent and progression of atherosclerosis, and the incidence of ischemic **heart disease.** Data from a baseline examination, a 4-year re-examination, and a proposed 1-year re-examination of ultrasonographically assessed carotid and femoral atherosclerosis, and surveillance for myocardial infarctions and other outcomes, will be used to examine the progression or incidence of these outcomes in relation to changes in behavioral, psychosocial and socioeconomic factors. It will also be possible to see to what extent the

input of these behavioral, psychosocial and socioeconomic factors on CVD in men and women is mediated by lipid peroxidation, hemostatic factors, and cardiovascular reactivity. Finally, the investigators state that this will be the first population-based epidemiologic study to examine the association between a carefully developed set of measures of cardiovascular reactivity to stress and the progression of carotid atherosclerosis, risk of myocardial infarction and death, and development of hypertension.

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- **Project Title: MENTORED PATIENT ORIENTED RESEARCH CAREER DEVELOPMENT AW**

  Principal Investigator & Institution: Garlow, Stepheng J. Psychiatry and Behavioral Scis; Emory University 1784 North Decatur Road Atlanta, GA 30322

  Timing: Fiscal Year 2001; Project Start 01-MAY-2000; Project End 30-APR-2005

  Summary: PROPOSAL (Adapted from the applicant's abstract): Major depression is a significant independent risk factor for the development of ischemic **heart disease** and is a potentially lethal comorbid condition in post-myocardial infarction patients. The pathophysiology that links major depression to the occurrence of **heart disease** is not known. Preliminary observations indicate that platelet reactivity is increased in depression, which implies that depressed patients may be prone to thrombus formation, hence at increased risk for catastrophic cardiac events. There are considerable data indicating that the serotonergic system is altered in depression, both in the central nervous system (CNS) and in the platelets. In the periphery, the most notable and consistently replicated observation of serotonergic alteration is an increased B max for the serotonin-2A (5-HT2A) receptor on the platelets of depressed individuals. The platelet 5-HT2A receptor plays a central role in platelet reactivity and thrombus formation, and may be involved in regulating the expression of platelet specific genes in megakaryocytes, the cells from which platelets are derived. While not tested directly, the overarching hypothesis for this proposal is that major depression adversely increases platelet reactivity, which leads to increased risk of developing **heart disease.** The principal hypotheses that will be tested are that: 1) Platelets from depressed patients are produced in a "upregulated" state, with increased amounts of a number of transcripts that encode platelet specific genes, the result being the platelets are more reactive and prone to thrombus formation. 2) The platelet serotonergic system, in particular the 5-HT2A receptor, is altered in depression which in turn contributes to the increased platelet reactivity observed in depression, and the relevant alteration may occur in the megakaryocytes. 3) Alterations in the concentration of one or more humoral factors (interleukins, cytokines, stress hormones) orchestrate the alterations in platelet reactivity and serotonergic functioning observed in depression. Patients suffering from major depression will be recruited and their condition treated. Their platelets will be sampled before and after treatment and the amounts of transcripts that encode platelet reactivity molecules will be measured. The circulating concentration of three hormones, cortisol, interleukin-1 and interleukin-6 will be measured before and after treatment, and correlated to platelet reactivity.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MOLECULAR DETERMINANTS OF PEDIATRIC HEART DISEASE**

  Principal Investigator & Institution: Kelly, Daniel P. Pediatrics; Washington University Lindell and Skinker Blvd St. Louis, MO 63130

Timing: Fiscal Year 2001; Project Start 01-JAN-1999; Project End 31-DEC-2003

Summary: The long term objective of this Pediatric SCOR is to determine the molecular bases of defective human cardiac morphogenesis and myocardial function which results in congenital **heart disease** (CHD) and pediatric cardiomyopathy (CM). The underlying hypothesis is that single gene defects at multiple loci in the human genome cause most pediatric cardiac structural and myopathic diseases. Two corollaries will also be explored, that (i) genetic abnormalities at the same locus have variable expressivity and can result in different phenotypes and (ii) genotype- phenotype correlations exist. A multi-disciplinary approach encompassing 14 investigators, 6 clinical and laboratory projects, and 4 core units at three locations is proposed. Molecular genetic studies of a St. Louis family with dominantly-inherited dilated CM; CM or sudden death secondary to mutations in mitochondrial fatty acid oxidation enzymes; patients with CHD associated with at the human at the human 8p23 locus encompassing the GATA-4 gene, a critical transcription factor in heart; elastin in heart; elastin mutations in supravalvar aortic stenosis; and dominantly-inherited atrial septal defects (ASD) mapped to 5p and 5q are the clinical focus. Mouse gene ablation and transgenic models to delineate (i) the pathogenesis of CM and sudden death in fatty acid oxidation defects, (ii) the essential role and downstream targets of GATA-4 expressed in mouse embryonic endoderm for mesoderm-mediated and downstream morphogenesis, (iii) the mechanisms by which elastin mutations disrupt elastic fiber assembly and vasculogenesis, and (iv) the mechanisms by which mutant mouse homologs of genes defective in human familial ASD result in CHD will be created. The molecular role of syndecans in determination of left-right asymmetry will be studied in the uniquely-manipulable Xenopus embryo system as a model for mechanisms underlying human heterotaxy syndromes and CHD. Characterization of defective human genes causing pediatric **heart disease** and the mechanisms through which mutations alter cardiac development and myocardial function is necessary before manipulations to prevent CHD and inherited CM are feasible.

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- **Project Title: MOLECULAR GENETICS OF CARDIOPROTECTION**

  Principal Investigator & Institution: Baker, John E. Professor; Medical College of Wisconsin Po Box26509 Milwaukee, WI 532264801

  Timing: Fiscal Year 2001

  Summary: Ischemic **heart disease** is the leading cause of death in African- Americans. Two of the risk factors that contribute to ischemic **heart disease,** hypertension and insulin resistance are a substantial medical problem in the African-American population. Our goal is to use an animal model of insulin resistance and hypertension (Dahl S rat) to map the genes responsible for increased sensitivity of the heart to ischemic injury. Our preliminary studies suggest that susceptibility to global ischemia in the isolated heart from Dahl S (SS/Mcw) rats is two-fold greater than in hearts from Brown Norway (BN/SsN/Mcw) rats using multiple independent measures of tissue injury. We hypothesize that genetic factors are responsible for increased susceptibility to myocardial ischemia in the Dahl S (SS/Mcw) compared with the Brown Norway (BN/SsN/Mcw) rat. This hypothesis will be tested in the progeny of a cross between the BN/SsN/Mc2 (a normotensive, non-insulin resistant rat, resistant to myocardial ischemia) and SS/Mc2 rat (a hypertensive, insulin resistant rat, used to facilitate discrete, well-controlled investigations of resistance to ischemia in the inbred rat strains. Congenic rats will be developed to further reduce the complexity to a "single gene trait". Specifically we shall: (1) Determine the phenotypic differences of isolated hearts and

coronary vessels from the parental strains in greater detain by studying the responses to: ischemia alone, adaptation to hypoxia and preconditioning prior to ischemia. (2) Map the gene(s) responsible for susceptibility to myocardial ischemia. The phenotyping protocol developed in Specific Aim 1 will be used to phenotype 300 animals in a cross between BN/SsN/Mcw and SS/Mcw. A total genome scan will be used to map the quantitative trait loci (QTLs) responsible for susceptibility to ischemia in this cross. Regions with QTLs identified in the total genome scan will be converted to the human homologous region by comparative mapping, using developing informatics tools and radiation hybrid mapping. (3) Determine the phenotype of isolated hearts and blood vessels from the congenic strains in response to: ischemia alone, adaptation to hypoxia, and preconditioning prior to ischemia. This will facilitate our understanding and help identify the causal gene(s). This information will provide candidate genome regions for our clinical projects, and will contribute to our understanding of the genetic basis underlying susceptibility to myocardial ischemia, and will provide information expected to facilitate the development of strategies to manage ischemic **heart disease** in African-Americans.

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- **Project Title: MOLECULAR MECHANISMS OF CONGENITAL HEART DISEASE**

  Principal Investigator & Institution: Izumo, Seigo; Director of Cardiovascular Research; Medicine; Harvard University (Medical School) Medical School Campus Boston, MA 02115

  Timing: Fiscal Year 2001; Project Start 01-JAN-1999; Project End 31-DEC-2003

  Summary: The goal of this SCOR project is to further the understanding of the fundamental mechanism of congenital **heart disease** with the use of the state-of-the-art technology of molecular biology, developmental biology, human genetics, small animal cardiac physiology, and clinical pediatric cardiology. The Harvard SCOR in Pediatric Cardiology would bring together some of the finest basic science research faculty of the Harvard Medical School and its affiliated teaching hospitals. Each participant of this SCOR has a strong track record of making original and fundamental contributions to cardiovascular sciences as it relates to the aim of this RFA. The projects described in this SCOR application are designed to take the maximum advantage of studying the closely related scientific theme using the different model organisms and system to dissect the role of transcription factors and secreted factors in the cardiac morphogenesis and their mutations in human and in experimental animals, as well as their linkage into the population of pediatric patients with congenital **heart disease.** Accordingly, this Harvard SCOR application is entitled "Molecular Mechanisms of Congenital **Heart Disease,**" and consists of five independent but highly interactive projects and three cores that provide highly specialized technological support as well as administrative support to facilitate the progress of each project. Specific projects include: Project I: Functional Analysis of the Cardiac Specific Homeobox Gene Csx/Nkx (P.I.-Seigo, Izumo). Project II: Genetic Analytic of Inherited Congenital Heart Diseases (P.I.-Christine E. Seidman; Co-P.I.-Jon Seidman). Project III. Identification of Signaling Molecules that Induce Heart Formation (P.I.-Andrew B. Lasser). Project IV: T-Box Gene in Congenital Heart and Limb Deformities (P.I.-Clifford J. Tabin). Project V: Genetic Determinants of Arrythmia Development Associated with Congenital Heart Malformation (P.I.-Charles I. Berul; Co-Investigators Mark E. Josephson and Edward Walsh). Small Animal Physiology Core (Director- James P. Morgan) will provide the state-of-the-art physiological analysis of the murine cardiovascular system using a multi-disciplinary approach. The Embryo Physiology and Imaging Laboratory will be a

unique resource to the Harvard community. Morphology Core will be developed to facilitate morphological studies by each Project. The program is designed to have clear focus on the investigations of the molecular basis for cardiac development and their genetic alterations with particular emphasis on the studies of cardiac transcription factors and secreted factors that play critical roles in early cardiogenesis. All projects are extensively interconnected both in scientific theme and the technological aspects while taking advantage of both experimental and clinical methodologies. The program unites both basic science departments and clinical cardiology programs of Harvard Medical Campus. The SCOR Director has extensive experiences in organizing interactive multi-disciplinary programs that encompasses both clinical and academic disciplines. The SCOR has full institutional support to make the program successful. We believe that this SCOR will generate many new findings fundamental to our understanding of congenital **heart disease** and at the same time is designed to provide rich training opportunities for the next generation of cardiovascular scientists and clinicians.

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- **Project Title: MONITORING COMMUNITY TRENDS IN HEART FAILURE**

  Principal Investigator & Institution: Goldberg, Robert J. Professor; Medicine; Univ of Massachusetts Med Sch Worcester Office of Research Funding Worcester, MA 01655

  Timing: Fiscal Year 2002; Project Start 01-APR-2002; Project End 31-MAR-2007

  Summary: (provided by applicant): Heart failure is a syndrome of profound clinical and public health importance. Heart failure (HF) is estimated to contribute to nearly 1 million hospitalizations and approximately 250,000 deaths annually in the U.S. The number of new cases of HF in the U.S. is estimated to exceed 400,000 annually. Though reliable estimates of the magnitude, incidence, and mortality of HF remain sorely lacking, HF is associated with a grim prognosis. However, little recent data exist, particularly from a community-wide perspective, to determine whether the incidence or survival associated with HF, and the management of this clinical syndrome, has changed over time. This study proposes to examine temporal trends (1995 and 2000) in the incidence rates of HF, its therapeutic management, and changes over time in the hospital and long-term survival of patients with HF from a multi-hospital, population-based perspective. The study will take place in residents of the Worcester (MA) metropolitan area (1990 census 437,000) and will examine changes over time in these and additional outcomes for patients with validated HF during 1995 and 2000. Complimenting the hospital surveillance of HF, newly diagnosed cases of HF occurring in members of the largest HMO in Central Massachusetts during 1995 and 2000 will be identified and monitored over time. The proposed project will build on the investigators' clinical and epidemiological experience and on data collection efforts and methodologies used in the ongoing community-wide study of coronary **heart disease** in greater Worcester residents. To accomplish the study objectives, the medical records of residents of the Worcester metropolitan area hospitalized with a discharge diagnosis of HF and related diagnostic rubrics will be individually reviewed and validated according to pre-established diagnostic criteria. The use of traditional criteria for HF as well as development of new criteria for the epidemiological study of HF will be an important focus of this observational study. Records for additional hospitalizations and death certificates will be reviewed to examine trends in long-term survival of discharged hospital patients through the year 2005. The results of this study will provide much needed information about the epidemiology of HF from a more generalizable population-based perspective. Information would be provided about the clinical and descriptive epidemiology of this prevalent and disabling condition in men and women

and individuals of different age groups. The proposed surveillance system will provide insights and guidance to public health and clinical efforts of HF and in monitoring trends in this newly emerging chronic disease.

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- **Project Title: MRI OF COLLATERAL DEVELOPMENT IN THE HEART**

  Principal Investigator & Institution: Jerosch-Herold, Michael; Radiology; University of Minnesota Twin Cities 200 Oak Street Se Minneapolis, MN 554552070

  Timing: Fiscal Year 2001; Project Start 20-SEP-2000; Project End 31-JUL-2003

  Summary: (Adapted from Applicants Abstract): In ischemic **heart disease** the presence and extent of collateral circulation can prevent myocardial infarction, or at least limit the damage and reduce the size of infarcts. The objective of this proposal is to characterize the development of coronary collaterals in the heart with methods based on magnetic resonance imaging (MRI). MRI-based measures of collateral growth can define the functional capacity of collaterals to preserve myocardial blood flow and contractile function in regions at risk of infarction. This would be a significant advantage over current angiographic methods used to define the extent of collateral circulation in the heart. The proposed studies will also determine to what degree collateral development in heart translates into an increased capacity of the heart to prevent stress-induced ischemia. The studies will be carried out in an experimental animal model of collateral development with slow progressive occlusion of the left circumflex coronary artery ('ameroid constrictor' model). This animal model of collateral development approximates the progression of coronary artery disease as it occurs in humans. The proposed diagnostic MRI techniques for assessment of collateral blood flow in the heart may in the future allow improved prediction of outcome. Understanding how collateral growth can be followed non-invasively also means that one can assess more effectively new therapies that boost collateral development in the heart and which could markedly change the natural history of coronary artery disease.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MRI OF VASCULAR FUNCTION IN THE HYPERTENSIVE HEART**

  Principal Investigator & Institution: Beache, Garth M. Radiology; University of Maryland Balt Prof School Baltimore, MD 21201

  Timing: Fiscal Year 2001; Project Start 01-JUL-1998; Project End 30-JUN-2003

  Summary: (Adapted from applicant's abstract) This proposal will provide the candidate with in-depth training in the concepts and methods relevant to the noninvasive imaging of vascular function in **heart disease** using magnetic resonance imaging (MRI). The proposed research will build on the candidate's previous focus on the use of MRI to characterize mechanical and perfusion abnormalities in **heart disease.** The long-term goal is to develop broad-based skills that are essential for elucidating the multifactorial pathophysiology underlying many heart conditions. The candidate will be guided by a team of basic scientists and clinicians who are currently conducting related cardiac research. Through closely working with this interdisciplinary team of senior investigators, the candidate is expected to develop a model for his own career. Specifically, the research aims to refine and implement a noninvasive MRI method, based on the sensitivity of MRI to blood oxygenation changes that accompany changes in hemodynamics and oxygen metabolism, to detect and qualify microvascular dysfunction in the heart. The method will be applied to the study of hypertension since it is a disease with major public health impact, affecting 10-20% of US adults, in which

microvascular dysfunction is believed to contribute to symptoms and cardiac performance, and for which adequate noninvasive vascular function methods do not exist. The applicants will then relate a new MR measure of vascular dysfunction to establish indicators of cardiac disability. They have obtained preliminary data confirming the ability to detect these effects in rats and in human subjects. This research has the potential to characterize the functional manifestations of hypertension and has the potential to contribute to a more comprehensive explanation of symptoms, which will aid in tailoring therapeutic regimen related to the vascular aspects of this disease. (End of Abstract)

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- **Project Title: MUTATIONAL ANALYSIS OF TGFBETA FUNCTION**

  Principal Investigator & Institution: Doetschman, Thomas C. Professor; Molecular Genetics, Biochemistry & Microbiology; University of Cincinnati 2624 Clifton Ave Cincinnati, OH 45221

  Timing: Fiscal Year 2001; Project Start 01-MAY-1990; Project End 30-JUN-2006

  Summary: Mutational Analysis of TGF beta Function. Congenital **heart disease** occurs in nearly 1 percent of all live births in the United States. Over 1/3 of these involve ventral and atrial septal defects and outflow tract defects. About a quarter of all heart defects result in critical disease, and even with modern medical practices about 1/3 of these still do not support life past one year. Even though congenital heart defects have both environmental as well as genetic components, genetic models of these abnormalities will be useful for identifying the molecular pathways through which both environmental and genetic factors can interfere. Therefore, identification of these pathways will likely lead to improved diagnostic, intervention and treatment strategies that will be applicable to both the environmental and genetic components of congenital **heart disease.** Transforming Growth Factor beta 2 (TGFbeta2) knockout mice have congenital heart abnormalities that are commonly found in humans: double outlet right ventricle, double inlet left ventricle, ventral and atrial septal defects, and valve defects. For the 4-chambered heart to form with proper alignment of the great vessels with their respective ventricles, considerable morphogenesis and remodeling must occur. Defects in either cushion formation or the subsequent myocardialization of those cushions could lead to such congenital abnormalities. We propose an in depth analysis of 1) the process of cushion formation, in which an epithelial- mesenchymal transformation of the endocardial cells into cushion mesenchyme occurs, and 2) the process of myocardialization, in which inner curvature myocardial cells, with the help of neural crest cells, invade and muscularize the cushion mesenchyme. We will apply immunohistochemical, in situ hybridization and electron mircroscopy techniques to normal and knockout embryonic hearts to analyze alterations in expression of the cell adhesion, migration, extracellular matrix and differentiation molecules that may be involved in these processes. We will also determine which cells are responsible for producing the TGFbeta2 needed for these processes to occur normally by transgenic rescue and conditional knockout, both in myocardial and neural crest cells. Finally, we will use functional genomics to discover new candidate genes involved in these essential processes of heart development. With this study we will obtain a much better understanding of the molecular mechanisms underlying the most common congenital heart abnormalities in man.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MYOSIN STRUCTURE /FUNCTION RELATIONSHIPS IN HEART FAILURE**

  Principal Investigator & Institution: Robbins, Jeffrey; Professor & Director; University of Cincinnati 2624 Clifton Ave Cincinnati, OH 45221

  Timing: Fiscal Year 2001; Project Start 01-FEB-2001; Project End 31-JAN-2002

  Summary: Project 5's long term goal is to investigate both the basic biology underlying normal cardiac development and function and the physiological consequences of the myosin heavy chain (MyHC) isoform shifts that occur during the development of heart failure. Many of the signaling pathways activated during compensated hypertrophy and decompensated failure result in fetal gene program activation. Consistent with these data, it has recently been shown that substantial amounts of the alpha-myosin heavy chain gene transcript are present in the normal human heart, and that these levels are dramatically down regulated during heart failure. What is the functional significance of the different cardiac proteins that constitute the main component of the pump? Can modulation of the myosin heavy chain isoform content impact significantly upon the heart's ability to maintain normal cardiac output under normal or pathologic conditions? Project 5 addresses these questions by modulating the myosin isoform content of the mouse heart. SPECIFIC AIM 1 will, using cardiac-specific transgenic expression in the mouse, replace the normal alpha-MyHC isoform with the beta1 beta-MyHC protein. Analyses at the molecular, cellular, whole organ and whole animal levels over the lifetime of the transgenic cohorts will provide a comprehensive picture of the consequences of varying myosin isoform content. SPECIFIC AIM 2 will challenge these mice, using both a surgically-induced pressure overload model, and breeding them into a number of genetically defined, cardiac-compromised transgenic lines, in order to define how altered motor content impacts on the progression and severity of cardiac disease. SPECIFIC AIM 3 will test the relevance of the mouse models to the human condition by creating the exact isoform shift that occurs in man in a transgenic rabbit heart. The alpha-MyHC will be linked to the beta-MyHC promoter in order to effect a partial or complete replacement of the normal beta-MyHC in the rabbit heart. The effects at the molecular and cellular levels, as well as hypertrophy and failure. Finally, SPECIFIC AIM 4 will measure both the RNA and protein levels for the alpha- and beta- MyHC's in populations with defined **heart disease.** These data will establish the normal and abnormal levels of these molecules during various, well-defined stages of human cardiac disease. The levels of the V1 and V3 will be correlated with the mechanical properties of the fibers. Together, these experiments will, for the first time, unambiguously establish the structure-function relationships of the cardiac myosin isoforms in the intact animal and will help establish the consequences of altered isoform content on compensated hypertrophy and its progression to heart failure.

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- **Project Title: MYOSIN--A LINK BETWEEN STREPTOCOCCI AND HEART**

  Principal Investigator & Institution: Cunningham, Madeleine W. Professor; Microbiology and Immunology; University of Oklahoma Hlth Sciences Ctr Health Sciences Center Oklahoma City, OK 73126

  Timing: Fiscal Year 2001; Project Start 01-AUG-1989; Project End 29-FEB-2004

  Summary: Rheumatic fever is a sequela of group A streptococcal infection primarily in children. Manifestations of the disease include carditis, arthritis and chorea. Our hypothesis is that autoimmune mechanisms due to molecular mimicry between the group A streptococcus and human tissues are responsible for the disease. Our data

support this hypothesis. We have identified host and streptococcal antigens which react with anti-strep/heart antibodies and T cells, and we have identified streptococcal and human cardiac myosin epitopes which produce carditis and valvulitis in animal models of disease. Despite our progress, we do not know how these crossreactive autoantibodies function in the pathogenesis of acute rheumatic fever(ARF) or the exact nature and antigenic specificities of the T cells in rheumatic carditis. Therefore, the goal and objectives propose to answer questions about the potential role of antibody in disease and to investigate the nature of the T cells which are crossreactive and appear to be responsible for valvulitis. The objectives are 1) to produce a panel of cytotoxic/crossreactive monoclonal antibodies (mAbs) from humans and transgenic mice and passively transfer IgM and IgG mAbs to test for tissue deposition in vivo; 2) to determine the nucleotide sequences of crossreactive antibody V, D, and J region genes; 3) to produce transgenic mice containing the VDJ genes(H and L) of human and mouse crossreactive and/or cytotoxic mAbs; 4) to investigate the Lewis rat model of valvulitis by producing and characterizing T cell clones from rats immunized with rM6 protein and cardiac myosin and in passive transfer experiments determine if these T cells produce disease; 5) to compare valves immunohistochemically from rheumatic carditis and Lewis rat valvulitis to identify similarities. These studies will attempt to define the steps in the pathogenesis of rheumatic carditis and will continue to support the growing body of evidence that infectious agents play a role in the development of autoimmunity in man.

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- **Project Title: NOTCH SIGNALING PATHWAY LIGANDS IN CARDIOVASCULAR DISEASE**

  Principal Investigator & Institution: Spinner, Nancy B.; Children's Hospital of Philadelphia 34Th St and Civic Ctr Blvd Philadelphia, PA 19104

  Timing: Fiscal Year 2001

  Summary: (Adapted from the Applicant's Abstract) Identification of genes causing human cardiac disease provides insight into the molecular pathways involved in heart development. The investigators have recently identified Jagged1 (JAG1) as the disease gene causing Alagille syndrome, a genetic disorder associated with heart, liver, and several other anomalies. JAG1 is a ligand in the Notch signaling pathway, shown in multiple organisms to be involved in cell fate determination. Alagille syndrome is a dominant disorder, with extreme variability in the expression of phenotypic features. Some individuals with JAG1 mutations have only a single clinical feature, rather than the multi- system involvement characteristic of Alagille syndrome, which led the investigators to hypothesize that JAG1 mutations would be identified in patients with isolated **heart disease.** The preliminary data support this hypothesis, as the investigators have identified three patients with cardiac disease and JAG1 mutations who do not manifest the liver abnormalities associated with Alagille syndrome. The investigators will extend this work to study a cohort of patients to determine the frequency of JAG1 mutations associated with cardiac defects. In order to understand how JAG1 is involved in cardiovascular development, the investigators will analyze location and timing of JAG1 expression in the developing mouse embryo. The investigators will also address the mechanism by which mutations in JAG1 cause **heart disease.** In Drosophila, mutations in Notch ligands which cause truncated proteins similar to those predicted in Alagille syndrome patients, act in a dominant negative fashion. However, there is compelling data from human studies that the mechanism for the effect of JAG1 is haploinsufficiency. The investigators propose to overexpress the

mutations seen in Alagille syndrome patients in the mouse embryo and determine their effect on early vascular development. The investigators further propose that other members of the Notch signaling pathway may be associated with cardiac abnormalities. The Notch ligand Delta has recently been mapped to 6q27, a region of the genome associated with cardiac disease in patients deleted for this region. The investigators will determine if Delta is the gene responsible for **heart disease** in these patients. If they are unable to show a role for Delta, they will use a positional cloning approach to identify other gene(s) associated with cardiac disease from 6q27. In summary, this work will provide an increased understanding of the role of JAG1 in normal and abnormal development of the heart, and lay the foundation for identifying additional genes contributing to cardiac disease.

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- **Project Title: OSTEOBLASTOGENESIS IN AORTIC VALVE CALCIFICATION**

  Principal Investigator & Institution: Rajamannan, Nalini M. Feinberg Cardiovascular Inst; Northwestern University Office of Sponsored Programs Chicago, IL 60611

  Timing: Fiscal Year 2003; Project Start 01-SEP-2003; Project End 31-AUG-2007

  Summary: (provided by applicant): This proposal is for a new K08 application which proposes a detailed plan for the candidate's research and a 4-year goal driven plan for further career development. The candidate's career goal is to become a successful independent scientific investigator. A career development plan is described that delineates the specific goals over the award period and the mechanisms that will be used to achieve them. Through didactic training and ongoing supervision through mentors and advisory committee, the candidate expects to 1) further develop as a basic science investigator, 2) conduct the proposed study with the highest degree of quality 3) present and publish studies in valvular **heart disease** at National conferences and leading journals 5) compete for advanced sources of funding and 6) develop insights in to the pathogenesis of valvular **heart disease.** The underlying mechanism for the pathogenesis of calcific aortic stenosis is unknown. This trend is becoming more pronounced with the aging population. Clinical studies indicate parallel risk factors for vascular atherosclerosis and aortic valve disease, the principle causative factor is elevated cholesterol levels. Due to these clinical observations, the PI has developed a translational approach to studying aortic valve disease. It is our hypothesis that calcification of the aortic valve is a direct result of valvular cellular transformation to an osteoblast phenotype that is initiated by hypercholesterolemia. The PI plans to test the hypothesis by the following specific Aims: Aim 1: Using the in vivo rabbit model, analyze the effects of hypercholesterotemia with and without statins in the atherosclerotic and proliferative aortic valve. Aim 2: Using the in vivo rabbit model, analyze the effects of hypercholesterolemia with and without statins in the calcification and mineralization of aortic valve. Aim 3: Using the in vivo rabbit model, determine the specific signaling pathways in the aortic valve which mediate the development of aortic valve atherosclerosis and osteoblastogenesis. Aim 4: Imaging the in vivo rabbit model of aortic valve disease. These studies provide the first in vivo model of aortic valve calcification which correlate the epidemiologic studies of atherosclerotic risk factors to the development of calcific aortic valve disease. If hypercholesterolemia stimulates the development of calcification in the aortic valve then the use of medical therapy such as HMG CoA reductase medications may have a role in the medical therapy of calcific aortic stenosis.

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- **Project Title: OXIDATIVE MODIFICATIONS OF PROTEINS AND FIBRINOGEN IN ATHEROGENESIS**

  Principal Investigator & Institution: Ischiropoulos, Harry; Associate Professor; University of Pennsylvania 3451 Walnut Street Philadelphia, PA 19104

  Timing: Fiscal Year 2002; Project Start 01-MAY-2002; Project End 31-MAR-2007

  Summary: (provided by the applicant): A potential major cause of vascular injury leading to the development of atherosclerosis is oxidative stress. Oxidative stress is the result of overproduction of reactive species that overwhelms the cellular antioxidant capacity leading to inactivation of key cellular functions and ultimately to cell death. Proteins are major targets of reactive species, and nitration of tyrosine residues is a selective protein modification induced by reactive nitrogen species in human disorders as well as animal and cellular models of disease. We have discovered that circulating fibrinogen is nitrated in patients with acute respiratory distress syndrome (ARDS). Nitration of fibrinogen significantly altered the function of fibrinogen by accelerating the rate of fibrin clot formation producing of fibrin clot with abnormal structure upon electron microscopic examination. Fibrin deposits are abundant in the lungs of patients with ARDS, a common complication of hemorrhagic injury and sepsis, and likely the result of abnormal clotting rather than failure in the fibrinolytic pathways, which are functioning normally in ARDS patients. A number of epidemiological studies have indicated that high levels of circulating fibrinogen is an independent predictor of coronary **heart disease** and in some cases of premature death from cardiovascular and **heart disease** although a causative correlation between high levels of fibrinogen and cardiovascular disease has not been established. Based on these data we developed the hypothesis that plasma protein nitration is a marker of oxidative stress and independent predictor of coronary **heart disease** and that nitration of fibrinogen is a critical post-translational modification responsible for abnormal functioning of the hemostatic system in atherosclerosis. To test the critical aspects of this hypothesis we propose to: 1) Quantify by the use of LC-MS the levels of 3-nitrotyrosine, dityrosine, the oxidation product of tyrosine and 3- chlorotyrosine, a marker of inflammation, in plasma proteins of smokers and nicotine users (Project 1), subjects in the prospective study of progression in coronary plaque burden (Project 5), the Apobec-l/LDLR double knock out mouse (Project 1) and in the plasma of mice with altered ApoA-I levels (Project 5). Evaluate the degree of nitration of specific proteins, fibrinogen, LDL/ApoB-100, and determine the site(s) of tyrosine nitration by the use of the Proteomics Core. 2) Evaluate the effects of nitration and/or oxidation on the biochemical, biophysical and viscoelastic properties of fibrinogen and fibrin clots by the use of scanning and transmission electron microscopy, and a Plazek torsion pendulum. 3) Determine the effect of nitration on critical functional aspects of fibrinogen and fibrin clot, ADP-induced platelet aggregation, endothelial cell gene expression (Genomics Core), endothelial-inflammatory cell interaction (in collaboration with Project 3), and kinetics of fibrinolysis. Overall this project is focused on investigating the possible biochemical and biophysical mechanisms that may underline abnormalities in the hemostatic factors that regulate critical pro- and anti-thrombotic functions in human cardiovascular disorders.

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- **Project Title: PEDIATRIC HEART DISEASE CLINICAL RESEARCH NETWORK - DCC**

  Principal Investigator & Institution: Sleeper, Lynn A. Director, Center for Statistical Analysi; New England Research Institutes, Inc. 9 Galen St Watertown, MA 02472

  Timing: Fiscal Year 2001; Project Start 06-SEP-2001; Project End 31-AUG-2006

Summary: (provided by applicant) The Pediatric **Heart Disease** Clinical Research Network will be a cooperative network of up to six Clinical Centers, a Data Coordinating Center (DCC), and the Division of Heart and Vascular Diseases of the NHLBI. The specific aim of this network is to efficiently conduct multi-center clinical studies (including but not limited to randomized clinical trials), and to evaluate new and existing treatments and management approaches for children with structural congenital **heart disease,** inflammatory **heart disease,** heart muscle disease, and arrhythmias. The role of the DCC is to support protocol development for the clinical studies, to provide statistical design expertise, to identify, train, and maintain central laboratories for echocardiography and other cardiac specialties; to collect and monitor the data and provide quality assurance in the form of personnel training and site visits; to provide overall study coordination including plans for patient recruitment and retention; and to analyze the data collected and promote rapid dissemination of study findings. New England Research Institutes (NERI) proposes that in the first year of Network funding, that patient registries of the cardiac conditions most likely to be the focus of Network studies be established in order to facilitate patient recruitment at the time of study approval and a system of modular data collection forms be developed that can be selectively utilized by Network studies in later years to ensure efficient start-up. Data collection and data management for all studies will be conducted using NERI?s proprietary web-based system ADEPT (Advanced Data Entry and Protocol Tracking System). Sites will have immediate access to site-specific enrollment reports, patient follow-up reminders and data summaries on each study participant, NERI proposes a stepwise approach with objective criteria for the selection of core laboratories and offers key personnel with medical training for implementation of quality assurance procedures. Experienced research staff from NERI will coordinate both shipments and electronic submissions of laboratory specimens and diagnostic cardiac data for the core laboratories. The NERI statistical team has extensive experience in the field of cardiology will provide expert guidance in the design of both observational studies and randomized trials, including design of early stopping rules, This application also includes a proposal for collection of cost-effectiveness data to be analyzed by NERI?s health economist. NERI has extensive experience in all areas of multi-center study coordination and, more specifically, currently coordinates one of the only such studies in the field of pediatric cardiology, the Pediatric Cardiomyopathy Registry.

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- **Project Title: PEDIATRIC HEART DISEASE NETWORK: CHOP MEMBERSHIP**

  Principal Investigator & Institution: Vetter, Victoria L.; Children's Hospital of Philadelphia 34Th St and Civic Ctr Blvd Philadelphia, PA 19104

  Timing: Fiscal Year 2001; Project Start 01-SEP-2001; Project End 31-AUG-2006

  Summary: (provided by applicant) As a Clinical Center in the new Pediatric **Heart Disease** Clinical Research Network (PHDCRN), the Children?s Hospital of Philadelphia (CHOP) will participate collaboratively with the Network to improve outcomes for children with **heart disease,** provide an evidential base for therapies currently used or considered, develop new therapies, and disseminate that information to the medical community. To achieve this goal, we will address the following aims: Aim 1: Develop an infrastructure and investigational team for to assure full participation in PHDCRN; Aim 2: Develop procedures and policies to assure proper implementation of approved PHDCRN protocols; Aim 3: Fully participate in the development new protocols and dissemination of information to the scientific community. Two protocols are proposed for the PHDCRN?s scientific agenda that emphasize the research strengths of the

Cardiology Division, the Cardiac Center and CHOP. The short-term study investigates the use of biventricular pacing or cardiac resynchronization in children with severe congestive heart failure and dilated cardiomyopathy. The primary aim of the study is to evaluate the acute effects of biventricular pacing on cardiac function as measured by oxygen consumption during exercise in a 6-week randomized controlled clinical trial. After the 6-week randomized comparison, all patients are paced and longer-term effects on cardiac function, functional capacity and quality of life will be evaluated out to 12 months. The long-term project investigates whether enalapril (ACE inhibition) can reduce the time-related decline in exercise performance experienced by patients with single ventricle who have undergone the Fontan procedure. The primary aim will be to evaluate the effects of ACE inhibition over a 4-year period on maximal oxygen consumption measured during exercise testing in a double blinded randomized clinical trial design. The Children?s Hospital of Philadelphia Cardiology Division and Cardiac Center has a distinguished history of clinical innovation and excellence in cardiac care, a high volume program with expertise in all areas of pediatric cardiology including cardiac arrhythmias, echocardiography, exercise physiology catheterization including interventions, intensive care, cardiac magnetic resonance imaging, transplantation, and surgery for **heart disease.** Patient care is integrated through a multidisciplinary Cardiac Center. The faculty of the Cardiac Center are experienced investigators and maintain a highly productive program of multicenter and institutional clinical trials and studies. CHOP has made major investments in the clinical research infrastructure that support essential functions in clinical investigations. These programmatic and institutional strengths support the aims and enhance the likelihood that the long-term goals of the PHDCRN will be achieved.

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- **Project Title: PERIODONTAL DISEASE AND RECURRENT CHD EVENTS**

  Principal Investigator & Institution: Trevisan, Maurizio M. Professor and Chair and Acting Dean; Social and Preventive Medicine; State University of New York at Buffalo 402 Crofts Hall Buffalo, NY 14260

  Timing: Fiscal Year 2001; Project Start 01-SEP-2000; Project End 30-JUN-2005

  Summary: (Adapted from the Investigator's Abstract) This study will analyze the association between periodontal disease and recurrent coronary **heart disease** (CHD) in individuals who have survived a myocardial infarction (MI). The overall goal is to test the hypothesis that periodontal disease is a significant and independent risk factor for recurrent CHD events. In addition potential mechanisms linking periodontal disease to CHD events will be investigated. At baseline 1,200 participants will undergo a detailed interview and health examination that will include information on lifestyle, sociodemographic, and anthropometric variables and CVD risk factors, and a complete oral health examination including measurement of clinical attachment levels and radiographic assessment of interproximal alveolar crestal height. The two aims are to examine the association between recurrent **heart disease** and periodontal disease as measured by gingival attachment level and radiographic assessment, and to examine the association between recurrent **heart disease** and levels of bacterial infection. Participants will be followed prospectively annually for an average of 3 years. The investigators state that the proposed study should provide important new information about the temporal association between periodontal disease and CHD.

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- **Project Title: PERIODONTAL INTERVENTION FOR CARDIAC EVENTS: PILOT TRIAL**

  Principal Investigator & Institution: Genco, Robert J. Distinguished Professor and Chair; Oral Biology; State University of New York at Buffalo 402 Crofts Hall Buffalo, NY 14260

  Timing: Fiscal Year 2001; Project Start 15-JUL-2001; Project End 30-JUN-2004

  Summary: (abstract verbatim) This proposal is to develop the clinical trial organization for studies of periodontal treatment in the secondary prevention of cardiovascular events and to develop the institutional teams to accomplish the research. With the appropriate infrastructure and procedures developed, a future definitive randomized clinical trial of the relationship between periodontal infection and cardiovascular disease can be successfully carried out. There is growing evidence of a strong relationship between infection and atherosclerosis as well as a specific link between periodontal infection and **heart disease.** Previous studies have established possible effective treatments of periodontal disease. These treatments may lead to a diminution of subsequent occurrence of myocardial events in persons at high risk for cardiovascular disease. A clinical trial process to demonstrate the feasibility of a periodontal intervention trial in heart patients requires the establishment of a new team of cardiologists, periodontists, epidemiologists, infectious disease specialists, biostatisticians, research nurses in periodontics and cardiology, and data managers. In addition, such a study requires a large number of institutional investigative centers. In contrast to previous **heart disease** studies, the primary treatment in the new trial process would be directed toward infectious agents with specific attention to periodontal sites. We propose a multi-center Planning and Pilot Study involving 5 Investigational Centers at the University of Buffalo, University of North Carolina, Boston University, Kaiser Permanente/Oregon Health Science University, University of Maryland, and a Coordinating Center at the University of North Carolina. This study will be supported by Central Microbiology and Cytokine Labs, and a Biologic Specimen Bank. The establishment of the investigative machinery to determine the relationships and treatments linking infection, periodontal disease and **heart disease** requires a careful, cost-effective approach. This planning proposal outlines a process designed to develop and demonstrate the procedures for such studies, to conduct a pilot randomized clinical trial to test the feasibility of these procedures, and to select and refine a periodontal intervention. We propose that this planning and pilot study will ultimately lead to a definitive randomized clinical trial of the relationship between periodontal infection and cardiovascular disease, and may be a vanguard study for such a trial.

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- **Project Title: PH III: AZIMILIDE CONTROLLED TRIAL**

  Principal Investigator & Institution: Bahnson, Tristram D.; Duke University Durham, NC 27706

  Timing: Fiscal Year 2001

  Summary: Purpose: The purpose of this study is to assess the safety of long-term use of azimilide in patients with atrial fibrillation/flutter and/or paroxysmal sypraventricular tachycardia. Atrial fibrillation is the most common cause of clinically significant sustained tachycardia and occurs in up to 12% of individuals over age 75. Incidence increases with each decade of life. Furthermore, atrial fibrillation is responsible for up to 1/3 of thromboembolic strokes. Anticoagulation, pharmacologic control of the ventricular response, and antiarrhythmic pharmacotherapy have been the main stays of

treatment for atrial fibrillation. However, the selection of available antiarrhythmic drugs, and their efficacy, are limited. Specifically, of available antiarrhythmic agents, only sotalol and amiodarone are considered safe in patients with underlying **heart disease.** Atrial fibrillation is most often associated with valvular, ischemic, or hypertrophic **heart disease,** limiting the use of available type I antiarrhythmic agents. Further, the class III agents sotalol and amiodarone are potent negative chronotropic agents which limits their usefulness in patients with atrial fibrillation in association with sinus node disease (brady-tachy syndrome), also a frequent clinical occurence. Accordingly, there is an important need for the development of new antiarrhythmic drugs with the following characteristics: 1) safety in patients with structural **heart disease.** 2) absence of significant negative chronotropic effects. One such drug is Azimilide, a class III antiarrhythmic agent with very weak bradycardic effects at test dosing levels, and which appears to be safe when used in individuals with structural **heart disease.** Methods: This is a multi-center double-blind, placebo-controlled, parallel design clinical trial studying a daily oral dose of 125 mg of azimilide dihydrochloride. Results: Results of the double blind trial of azimilide has been reported in preliminary form (n=89) with demonstration of a significant decreases in mean time to AF recurrence in azimilide treated patients (130 days; 100mg/d and 125mg/d) comparted with control (17 days, p (log rank) of 0.002). Significance: The significance of this clinical research program is to test the safety and efficacy of this promising new antiarrhythmic drug in the treatment of atrial fibrillation. Atrial fibrillation is the most common cause of clinically significant sustained tachycardia and occurs in up to 12% of individuals over age 75. Incidence increases with each decade of life. Furthermore, atrial fibrillation is responsible for up to 1/3 of thromboembolic strokes. Future Plans: Phase III trials of Azimilide are ongoing, and it is anticipated that currently enrolled patients will be followed prospectively for a total of 24 months. After completion of the phase III trials of Azimilide for treatment of atrial fibrillation, further studies of the safety and efficacy of this new antiarrhythmic agent to control paroxysmal atrial fibrillation in specific clinical circumstances are envisioned. These studies will be conducted under separate protocol.

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- **Project Title: PREMENOPAUSAL RISK FACTORS FOR CORONARY HEART DISEASE IN BLACK & WHITE WOMEN**

  Principal Investigator & Institution: Gerhard, Glenn; Oregon Health & Science University Portland, OR 972393098

  Timing: Fiscal Year 2001

  Summary: Premenopausal black women have a 2- to 3-fold greater rate of coronary **heart disease** (CHD) than premenopausal white women. The purpose of this study was to provide greater insight into the reasons for this difference, which are currently unclear. We compared CHD risk factors in 99 black and 100 white, healthy, premenopausal women, aged 18 to 45 years, and of relatively advantaged socioeconomic status. Compared with white women, black women had a higher body mass index (32.0 _ 9.2 vs 29.0 _ 9.4 kg/m2, p=0.021), and higher systolic (124 _17 vs 115 _ 14 mm Hg, p<0.0001) and diastolic (79 _ 14 vs 75 _ 11 mm Hg, p=0.048) blood pressures. The mean plasma lipoprotein(a) concentration was markedly higher in the black women (40.2 _ 31.3 mg/dl) than in the white women (19.2 _ 23.7 mg/dl, p<0.0001). The plasma total homocysteine level was also higher in the black women (8.80 _ 3.38 vs 7.81 _ 2.58 umol/L, p=0.013). The black women, however, had lower plasma triglyceride levels (0.91 _ 0.46 vs 1.22 _ 0.60 mmol/L, p<0.0001), and a trend toward higher high-density lipoprotein (HDL) cholesterol levels (1.37 _ 0.34 vs 1.29 _ 0.31 mmol/L, p=0.064) than

the white women. Plasma total and low-density lipoprotein (LDL) cholesterol levels were similar, despite a greater consumption of saturated fat and cholesterol by the black women. Rates of cigarette smoking and alcohol intake were low and similar between the races. Premenopausal black women had a higher mean body mass index, blood pressure, lipoprotein(a), and plasma total homocysteine level, and a greater consumption of saturated fat and cholesterol than white women. These differences in coronary risk factors may place the black women in our study at increased risk for CHD compared with the white women. FUNDING Collaboration with Dr. Gerhard at OHSU PUBLICATIONS Gerhard GT, Sexton G, Malinow MR, Wander RC, Connor SL, Pappu AS, Connor WE. Premenopausal black women have more risk factors for coronary **heart disease** than white women. Am J Cardiol 82:1040-1045, 1998.

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- **Project Title: PROSPECTIVE STUDY OF HEALTH IN RUNNERS AND WALKERS**

  Principal Investigator & Institution: Williams, Paul T. Nuclear Science Division; University of Calif-Lawrenc Berkeley Lab Lawrence Berkeley National Laboratory Berkeley, CA 94720

  Timing: Fiscal Year 2001; Project Start 01-JUN-1998; Project End 31-MAY-2003

  Summary: (Adapted from Investigator's Abstract) Current government physical fitness guidelines state that: 1) the majority of the health benefits from physical activity can be obtained by walking 2 miles briskly on most days of the week; and 2) the health benefits of physical activity depend principally on the total amount of activity rather than the intensity of the activity. Nevertheless, there are currently no prospective epidemiological studies extant, designed specifically to directly contrast the health benefits and costs of moderate exercise (e.g., walking) versus vigorous exercise (e.g., running). The proposed study plans to compare rates of coronary **heart disease** (CHD), cancer, total mortality and exercise injuries in 68,000 runners and 68,000 walkers during four years of surveillance. Questionnaires concerning running and other physical activities in 56,000 runners have already been obtained, and additional questionnaires from 13,000 runners are expected before March 1997. The runners will be resurveyed in 1997 along with 68,000 walkers. The walkers will also be solicited through the publication of the questionnaire in Walking magazine followed by a direct mailing of the questionnaire to 425,000 subscribers. Total and cause-specific mortality will be determined from the National Death Index; fatal and nonfatal cancers will be identified from the SEER and 46 state registries; nonfatal coronary **heart disease** and injuries will be determined from questionnaires. Survival analyses will be used to test whether runners have greater reduction in **heart disease,** total mortality, and cancer per unit of exercise. Exercise-related injuries from walking and running will also be examined. Power calculations suggest that detection of differences between runners and walkers, as small as 11% for total mortality, 16% for CHD, 12% for total cancers, and 36% for breast cancer, will be possible. The differences will be adjusted for weekly kilocalories expended by walking and running, for walking and running distance, and for time spent on each activity to test whether these variables account for differences in disease rates between walkers and runners. Before the start of the study, 233,000 person-years of follow-up in 56,000 runners (between 1991 and 1997) will have been accumulated. By the end of the study, 517,000 person years in 68,000 runners (between 1991 and 2001) will be available for analysis. Survival analysis will be used to test for a dose-response relationship between running mileage and CHD and cancer risk, and whether this relationship is affected by running intensity, running frequency, running history, gender, adiposity, age or medication use. Using conservative rates (25% below

published values), statistical power calculations suggest that detectable reduction in coronary **heart disease** risk as small as 0.71% per mile will be possible, which is far below the estimated reduction from other published studies (2.1%). Additionally, a detectable reduction in breast cancer risk as small as 1.5% per mile run in women is calculated, which is below the 1.7% reduction in risk estimated from other published data.

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- **Project Title: QUANTIFICATION OF HEART BETA ADRENERGIC RECEPTORS**

  Principal Investigator & Institution: Muzic, Raymond F. Radiology; Case Western Reserve University 10900 Euclid Ave Cleveland, OH 44106

  Timing: Fiscal Year 2001; Project Start 01-APR-1999; Project End 31-MAR-2003

  Summary: (Adapted from applicant's abstract): Health Relevance: Beta-adrenergic receptors (beta-ARs) play a fundamental role in the regulation of heart function. Changes in the amount and binding properties of beta-ARs are implicated in coronary **heart disease,** congestive and ischemic heart failure, cardiomyopathy, sudden death, arrhythmia, and mitral valve disease. Drugs that interact with the beta-ARs, beta-blockers, are widely prescribed to treat **heart disease.** Since the in vitro behavior of receptors often differs from their in vivo behavior, a method to assess beta-ARs in vivo is essential for improving our understanding and treatment of heart diseases. Moreover, a relatively noninvasive test could be used to assess patients individually. Proposed Work: a significant component of the tissue uptake of (S)-[18F]fluorocarazolol as measured by positron emission tomography (PET) reflects specific binding to beta-ARs. However, it also reflects nonspecific uptake, radioactive metabolites in the myocardium, and possibly uptake related to the norepinephrine transporter. Therefore, quantitative assessment of beta-AR specific binding and of beta-AR concentration requires a mathematical model of fluorocarazolol pharmacokinetics. To formulate this model, details of fluorocarazolol pharmacokinetics will be clarified via in vitro and in vivo experiments (Aims 1 to 2) and via computer simulation to compare compartmental and distributed pharmacokinetic models (Aim 3). A mathematical model of fluorocarazolol pharmacokinetics will be formulated in accordance with the results of Aims 1 to 3. This model will then be used to analyze PET data collected from pigs with normal and elevated concentrations of beta-AR. The validity of the model and its utility to assess beta-AR concentration and binding properties in vivo will be evaluated based on comparison to results obtained via in vitro assay of myocardial samples (Aim 4). Significance: Although [11C]CGP 12177 has been used to estimate myocardial beta-AR concentration in vivo, there are numerous advantages for using [18F]fluorocarazolol. Perhaps the most significant is that fluorocarazolol reaches internalized receptors whereas CGP 12177 does not. Completion of the proposed work could lead to a method for estimating the fraction of receptors that are internalized. It would entail two PET experiments using [18F]fluorocarazolol; one at baseline and one following administration of unlabeled 9commercially available) CGP 12177 to block surface receptors.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: RANDOMIZED CLINICAL TRIALS FOR PEDIATRIC HEART DISEASE**

  Principal Investigator & Institution: Saul, J P. Pediatrics; Medical University of South Carolina 171 Ashley Ave Charleston, SC 29425

Timing: Fiscal Year 2001; Project Start 01-SEP-2001; Project End 31-AUG-2006

Summary: (provided by the applicant) The Children?s Heart Program of South Carolina is a statewide consortium of pediatric cardiologists, who care for 90% of the 3.7 million residents in the state. This consortium has all the critical elements for a center in the proposed research network: adequate patient volume, clinical research infrastructure, a track record of subject enrollment, and a demonstrated dedication to hypothesis driven clinical research. The applicant center, MUSC, is the tertiary referral center for the Children?s Heart Program. Current MUSC faculty have participated as PI?s in a total of 20 multicenter clinical trials or registries (10 open, 2 under IRB review). The PI of this application has been the lead investigator nationally in 4 of the 20. These protocols range from industry sponsored drug or device trials, to an NIH sponsored drug trial for fetal heart block, to an NIH prospective registry. The faculty also currently runs 11 local clinical research protocols. Participation in all of these protocols is supported by a dedicated pediatric cardiac research support group with 2 full time RN coordinators and an additional RN FTE. The combined resources of high volume and strong research infrastructure have enabled the PI?s at MUSC to be one of the top 2 subject recruiters in 6 of the 18 completed or active multicenter studies. As requested, the proposal contains a short-term and a long-term protocol. Short-Term. Randomized Trial of Aortopulmonary Collateral Coil Occlusion Prior to Fontan. Multiple factors influence morbidity and mortality for single ventricle patients undergoing Fontan operation. One considered recently is the presence of APC?s. However, multiple retrospective studies have failed to clearly delineate the role of APC?s or their optimum management. This protocol will prospectively evaluate the role of APC?s in postoperative Fontan hemodynamics and morbidity, and determine the importance of preoperative coil embolization in their management. Long-Term. Randomized Trial of Amiodarone vs Cooled- Tip Catheter Ablation for Treatment of Recurrent Intra-atrial Reentry Tachycardia (IART) in Patients with Congenital **Heart Disease.** IART, the single largest cause of morbidity late after repair of congenital **heart disease,** is often life-threatening, frustrating to treat and has no clearly superior therapy. This protocol will prospectively compare the most successful medical and catheter therapies for IART. The primary endpoint during a minimum of 2 years follow-up will be IART recurrence after successful ablation, or after drug loading and cardioversion.

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- **Project Title: RANDOMIZED THERAPEUTIC TRIALS IN PEDIATRIC HEART DISEASE**

  Principal Investigator & Institution: Newburger, Jane W. Associate Cardiologist-In-Chief; Children's Hospital (Boston) Boston, MA 021155737

  Timing: Fiscal Year 2001; Project Start 01-SEP-2001; Project End 31-AUG-2006

  Summary: (provided by applicant) This grant application proposes two randomized, double-blind, placebo-controlled trials to be performed in the Pediatric **Heart Disease** Clinical Research Network. Our shorter-term trial will study the efficacy and safety of pulse steroid therapy, when added to conventional therapy with intravenous gamma globulin (IVIG) plus aspirin, in treatment of acute Kawasaki disease. Patients will be randomly assigned to receive either methylprednisolone (IVMP), 30 mg/kg, plus conventional therapy (i.e., "IVMP plus IVIG") versus placebo plus conventional therapy ("IVIG alone"). Our first aim is to test the hypothesis that treatment of acute Kawasaki disease with IVMP plus IVIG is more effective than treatment with IVIG alone. Our primary efficacy outcome variables will be BSA-adjusted coronary artery dimensions (z-scores) for the proximal right, left main, and proximal left anterior descending coronary

arteries; number of days of fever after completion of initial IVIG infusion; and C-reactive protein at 2 weeks after illness onset. Our second aim is to test the hypothesis that children treated with IMP plus IVIG will have fewer adverse effects than those treated with IVIG alone. Our primary safety outcome will be the prevalence of all adverse side effects. The structure of the study will allow us to explore and identify factors other than the two treatment strategies (e.g., immune gene polymorphisms) that relate to the occurrence of-coronary artery abnormalities. The KD trial will span less than two years from onset of enrollment to preliminary data. Our longer-term trial evaluates the efficacy of beta-blocker therapy in retarding progressive aortic root dilation and valvular aortic regurgitation in patients after the arterial switch operation (ASO). Patients will be randomly assigned to receive either propranolol (2-4 mg/kg/day) or placebo. Our first aim is to assess the effect of propranolol therapy on the rate of aortic root dilation after the ASO. The primary outcome variable is the change in aortic root size during two years of treatment, assessed as the aortic root diameter adjusted for body surface area (BSA). A second specific aim is to assess the incidence and magnitude of adverse effects of propranolol therapy. The primary end-point will be the change in the Physical Health Summary and Psychosocial Health Summary scores of the CHQ-50 at one and two years of therapy compared to pre-therapy. A third specific aim is to evaluate the role of collagen and fibrillin in the pathogenesis of aortic root dilation after the ASO by analyzing single nucleotide polymorphisms (SNPs) in candidate genes: fibrillin and Collagen types 3al, 5al, and 5a2 genes. The primary endpoint is the identification of SNPs that are significantly associated with severity of aortic root dilation. The ASO trial will involve an enrollment period of two years and a follow-up period of 2 years. Both trials are expected to yield information important to clinical practice.

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- **Project Title: REDUCING HEART DISEASE RISK WITH 'GET-THE-FAT-OUT'**

  Principal Investigator & Institution: Southard, Barbara H.; Health Management Consultants of Va of Virginia, Inc. Blacksburg, VA 24060

  Timing: Fiscal Year 2001; Project Start 01-AUG-2001; Project End 31-JAN-2002

  Summary: (provided by investigator): Despite enormous public health efforts, **heart disease** remains the number one killer, partly due to the prevalence of high dietary fat intake and sedentary lifestyles. At the same time and for many of the same reasons, obesity is becoming increasingly prevalent, bringing with it increased risk of **heart disease,** hypercholesterolemia, diabetes, hypertension, and some forms of cancer. In an effort to address the dietary behaviors that promote both **heart disease** and obesity, Health Management Consultants of Virginia proposes to develop Get-the-Fat-Out, an Internet data based behavior modification program targeted for at-risk adults at varying stages of readiness for changing dietary fat intake and physical activity level. The ultimate goal of Get-the-Fat-Out is to facilitate life-long habits of maintaining a healthy weight, an active lifestyle, and a low-fat diet. Specific aims for Phase I are: 1) Develop initial and on going "intervention-integrated" readiness for change assessments; 2) Develop stage-specific contents and strategies; 3) Develop a reward system; 4) Program 1, 2 and 3 for delivery on-line via PC and/or PDA; 5) Formatively evaluate the program. Get-the-Fat-Out could be offered to adults as part of a cardiac rehabilitation or other disease management or wellness program, or as an independent intervention via on-line subscription. PROPOSED COMMERCIAL APPLICATION: NOT AVAILABLE

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: REDUCING PREHOSPITAL DELAY ACUTE MYOCARDIAL INFARCTION**

  Principal Investigator & Institution: Dracup, Kathleen A. Dean and Professor; Dean's Office; University of California San Francisco 500 Parnassus Ave San Francisco, CA 94122

  Timing: Fiscal Year 2001; Project Start 01-JUN-2001; Project End 28-FEB-2006

  Summary: Background - Individuals who experience the symptoms of acute myocardial infarction (AMI) often delay hours or even days before seeking medical treatment. Median prehospital delay times (i.e., between the onset of cardiac symptoms and arrival at the hospital) have been documented to be 2.0 to 6.0 hours in numerous studies conducted over the past three decades. Such delays result in significant morbidity and mortality. Purpose - We propose a randomized experimental trial to determine whether a one-to-one education and counseling intervention delivered specifically to patients with documented ischemic **heart disease** will 1) reduce prehospital delay, 2) increase 911 use, and 3) increase aspirin use in those patients who experience AMI symptoms. Other aims to be tested relate to hypothesized changes in resource utilization; knowledge, attitudes, and beliefs about **heart disease** and AMI symptoms; and perceived control and anxiety. The effect of various sociodemographic characteristics and means of health care payment on prehospital delay will also be tested. Methods - 5,400 patients with documented ischemic **heart disease** will be randomized to the experimental intervention or to a care-as-usual control group at six participating sites. The intervention group will receive face-to-face counseling about the symptoms of AMI and the importance of seeking treatment early, as well as 911 and aspirin use. They will also receive telephonic reinforcement of the intervention at one-month follow-up. Data will be collected at baseline, immediately following the intervention, 3, 12, 24 and 36 months. Instruments to be used include the Response Questionnaire (knowledge, attitudes and beliefs), Cardiac Control Index (perceived control), Brief Symptom Inventory Anxiety Subscale, and the Resource Utilization Interview. Prehospital delay, 911 use, aspirin use and resource utilization will be evaluated by means of medical record review and patient interview. It is anticipated that approximately 3% of patients will experience AMI symptoms annually. Analyses. Standard multivariate and repeated measures analysis of variance techniques will be used to test study hypotheses. Significance - All previous interventions have focused on educating e public using mass media, with disappointing results. This study will be the first to test the effectiveness of a one-to-one intervention to reduce prehospital delay. The proposed intervention, if effective, could result in significant improvement in morbidity and mortality of patients with **heart disease.**

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- **Project Title: REMATCH TRIAL**

  Principal Investigator & Institution: Rose, Eric A. Professor of Surgery; Surgery; Columbia University Health Sciences New York, NY 10032

  Timing: Fiscal Year 2001; Project Start 11-APR-1997; Project End 31-AUG-2004

  Summary: 600,000 Americans die each year as a result of **heart disease,** despite improvements in the diagnosis and treatment of cardiac problems. Recent dramatic success with heart transplantation has shown that replacement of the human heart can be achieved with remarkable prolongation of high-quality of life. Un fortunately, the limited availability of donor organs severely restricts the use of cardiac transplantation to a small minority of patients with end-stage heart failure. Recent developments in

pump design and materials, however, suggest that the previously elusive goal of successful cardiac replacement with man-made pumps may soon be achieved. Experience with advanced-design wearable left ventricular assist devices used as "bridges" to transplantation has been recently extended to periods of 100 to more than 500 days. Patients can enjoy a high quality of life outside of hospital, with low fail-safe mechanisms in device design allowing expeditious and effective treatment in rare instances of mechanical device failure. In light of the ongoing high incidence and poor prognosis of end-stage **heart disease** and encouraging process in mechanical circulatory assistance, we propose a three-center randomized clinical trial of the wearable Thermo cardiosystems left ventricular assist device versus medical therapy of heart failure in patients who are not candidates for cardiac transplantation. Our primary hypothesis is that such devices can reduce two-year mortality by 33% (from 75% to 50%). Randomization of total of 130 patients allows an 80% power to demonstrate the anticipated survival benefit. Rigorous assessment of quality of life and the cost-effectiveness of medical versus device therapy will also be conducted.

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- **Project Title: RETINAL ARTERIOLAR ABNORMALITIES AND CV MORTALITY**

  Principal Investigator & Institution: Klein, Ronald; Professor; Ophthalmology and Visual Sci; University of Wisconsin Madison 750 University Ave Madison, WI 53706

  Timing: Fiscal Year 2001; Project Start 01-MAR-2001; Project End 28-FEB-2003

  Summary: The application describes a population-based case-cohort study to determine whether retinal arteriolar changes (generalized narrowing, focal narrowing and arterio-venous nicking) and retinopathy are associated with 10-year stroke-and ischemic heart disease-related mortality. The study population will be selected from participants of the Beaver Dam Eye Study, a well-characterized population of predominantly white persons aged 43-86 years at the baseline examination in 1988-90. Cases are defined as participants who have died from either stroke or ischemic **heart disease** since the baseline examination. Three participants per case will be selected from the cohort at baseline as controls, matched on gender and 5-year age intervals to cases. Focal arteriolar narrowing, arteriovenous nicking and retinopathy have been graded to baseline using a standardized photographic grading protocol. To evaluate generalized arteriolar narrowing, a method modified from the Atherosclerosis Risk in Communities Study will be used. Retinal photographs will be digitized and processed using a high-resolution scanner. Retinal arteriole and venule widths will then be measured with the help of a computer program based on pixel density contrast between the vessel and the background retina. Finally, the average width of the arterioles will be summarized as a retinal arteriole to venule ratio (AVR). Standard case-control methodology will be applied to calculate the relative odds of association between generalized retinal arteriolar narrowing (using different categories of AVR) and other changes with stroke-and ischemic heart disease-related mortality. Logistic regression models controlling for potential confounders (e.g., blood pressure, serum lipid levels) will be employed to evaluate the independent association between retinal arteriolar characteristics and stroke- and ischemic **heart disease** mortality. Data from this study will improve the understanding of the association between microvascular characteristics and cardiovascular mortality.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: RISK FACTORS FOR CVD IN WOMEN**

  Principal Investigator & Institution: Manson, Joann E. Associate Professor; Brigham and Women's Hospital 75 Francis Street Boston, MA 02115

  Timing: Fiscal Year 2001; Project Start 01-DEC-1984; Project End 30-AUG-2002

  Summary: The plan is to continue investigation of determinants of cardiovascular disease (CVD) in a large, prospective cohort study of long duration, comprised of 121,700 US female registered nurses who have been followed since 1976 and are currently age 51-76 years. Continued follow-up will permit evaluation of a number of new and timely hypotheses regarding dietary and hormonal risk factors for coronary **heart disease** (CHD) as well as ischemic and hemorrhagic stroke. In addition, follow-up of two new and important endpoints, sudden cardiac death (SCD) and congestive heart failure (CHF), will be established, allowing them to examine the effect of atherogenic risk factors, physical activity and weight change on risk of SCD, as well as implement a methodology for confirming CHF. The Nurses' Health Study was begun in 1976 to study risk factors for breast cancer. In 1979, funding for risk factors of CVD was secured. Biennial mailed questionnaires gather detailed, updated information about a large number of exposures, including diet, smoking physical activity, postmenopausal hormone use, and psychosocial and behavioral factors. Incident reports of myocardial infarction and stroke are confirmed an classified by review of medical records, and similar procedures will be extended to confirm CHF and SCD. Fatal CVD events are documented by death certificates and confirmed by review of hospital medical records, autopsy reports and interviews with next of kin. Mortality follow-up exceeds 98 percent. The large size, prospective design, high follow-up rates, detailed and reliable long-term exposure and outcome information, and the availability of blood specimens on a large subgroup, combined with the relatively low cost, make this cohort a valuable and unique resource to study nutritional, hormonal and behavioral determinants of CVD in women. Continued funding of this grant will provide important new information about risk factors for CHD, stroke, SCD and CHF in this population of women who are now entering the age where CVD becomes the leading killer.

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- **Project Title: RNA ISOLATION FROM HEART SAMPLES FOR EXPRESSION ANALYSIS**

  Principal Investigator & Institution: Perreault-Micale, Cynthia L.; Gwathmey, Inc. 763 E Concord Ave Cambridge, MA 02138

  Timing: Fiscal Year 2003; Project Start 15-AUG-2003; Project End 31-JAN-2004

  Summary: (provided by applicant): The overall objective of this study is to determine the changes in gene expression responsible for the development of endstage heart failure. The first step will be to construct two interactive databases 1) for human heart, vascular and skeletal muscle samples and 2) for similar samples from rodent models of heart failure. Gwathmey, Inc has collected hearts and blood vessels, and all the corresponding medical records from human samples that are stored as paper copies only. The investigators propose to develop a database to aid in the management of the tremendous amount of medical information that is available with each human heart. The proposed approach will make it possible for us to access all patients' records according to diagnosis, age, gender, existing medical conditions, medications, race, serology, and other specific clinical tests. Also the investigators will create a database of commonly used rat models of heart failure for comparison. Similarly they will be able to recall heart samples from animal models based on age, gender, and severity of heart

failure as well as cause of the **heart disease.** The human database will allow for continued expansion with an on-line update upon receiving new heart tissue that occurs on a weekly basis. The second goal of this study is to extract total and polyA RNA from all of the normal and diseased heart samples. Initially, the investigators intend to extract RNA from about 400 left ventricular control and myopathic human and rodent hearts that will represent the most common disease states (dilated, ischemic and hypertensive myopathies), as well as appropriate controls. The investigators will examine the quality, purity and quantity of RNA obtained and enter it into both databases so that they can keep a running log of the results. The human and rodent heart databases, in combination with the extensive collection of high quality RNA, will be an extremely valuable research tool and product available to the investigators and clients for innovative studies of differential gene expression during heart failure.

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- **Project Title: ROLE OF BOP IN CARDIAC DEVELOPMENT AND FUNCTION**

  Principal Investigator & Institution: Gottlieb, Paul D. Molecular Genetics & Microbiol0gy; University of Texas Austin 101 E. 27Th/Po Box 7726 Austin, TX 78712

  Timing: Fiscal Year 2003; Project Start 15-MAY-2003; Project End 30-APR-2007

  Summary: (provided by applicant): Congenital **heart disease** and acquired **heart disease** are the leading non-infectious causes of death in children and adults, respectively. The long term objectives of this project are to understand the role of m-Bop proteins in cardiac myocyte differentiation and cardiac development. Bop encodes m-Bop proteins specifically expressed in the heart field and myotome of the mouse and chick early in development as well as in fetal and adult mouse myocardium and skeletal muscle. m-Bop proteins contain both a MYND domain, shown in other proteins to recruit histone deacetylases (HDACs), and a S-ET domain, shown elsewhere to affect chromatin structure, sometimes through intrinsic histone methyltransferase (HMT) activity. Both activities can repress gene expression through epigenetic effects involving chromatin modifications. Targeted inactivation of Bop in mice leads to death at embryonic day 10 (El0 0), and hearts of Bop-null fetuses lack a right ventricle and have abnormal cardiomyocyte differentiation. Absence of the transcription factor, Hand2, from the heart primordia of E7 75 Bop-null embryos suggests a role for m-Bop in an early gene expression cascade that leads to right ventricular development. m-Bop has repressive activity in vitro due to recruitment of HDACs, and it physically interacts directly or indirectly with HDACs. m-Bop interacts with skNAC, a heart- and skeletal muscle-specific transcription factor, and co-localizes with skNAC during myogenesis in vitro and during cardiogenesis in vivo. m-Bop also interacts with MITR, a co-repressor of myogenesis, and HRT2, a heart ventricle-specific transcription factor. We plan to test the hypotheses that 1) m-Bop is a cardiac-specific regulator of chromatin modifications early in cardiomyocyte development and functions through direct and indirect interactions with DNA-binding proteins, and 2) that m-Bop and skNAC interact in a physiologically meaningful manner during myogenesis in vitro and cardiac development in vivo. Specific Aims are 1) To define the mechanisms by which the MYND, SET and other domains of m-Bop promote alterations in chromatin structure and regulate transcription, and 2) To determine the biological significance of m-Bop/skNAC interaction and the role of skNAC during cardiogenesis. These studies are relevant to mechanisms that may underlie ventricular hypoplasia in congenital **heart disease.** That m-Bop appears to specifically affect heart development by promoting histone modifications and hence chromatin reorganization in a lineage-specific fashion places it

among a very few proteins known to operate in this way. This gives a broader relevance to the mechanisms to be investigated in the proposed studies.

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- **Project Title: ROLE OF CALCINEURIN IN CARDIAC HYPERTROPHY**

  Principal Investigator & Institution: Bueno, Orlando F.; Children's Hospital Med Ctr (Cincinnati) 3333 Burnet Ave Cincinnati, OH 45229

  Timing: Fiscal Year 2001; Project Start 01-MAY-2001

  Summary: The theme of this application is to characterize reactive intracellular signaling pathways that promote cardiac hypertrophy. In response to various disease states the myocardium undergoes hypertrophic growth as a means of compensation. This compensation eventually leads to greater pathology and progressive deterioration of function. An understanding of the intracellular signaling pathways that cause or regulate hypertrophic growth of the heart will permit development of new therapies for certain forms of **heart disease.** The laboratory has recently identified a novel calcium responsive intracellular signaling pathway that likely plays an important role in regulating cardiac hypertrophy. It was shown that the calcium regulated phosphatase calcineurin (PP2B) and the downstream transcription factor NFAT3 are key components of cardiomyocyte cellular hypertrophy (Molkentin et al., 1998; Sussman et al., 1998). However, the physiologic relevance of this novel signaling pathway is presently uncertain and the subject of debate. Accordingly, the goals of the proposed study are: 1) To utilize transgenic mice expressing a peptide inhibitor of calcineurin in the heart to evaluate the role of calcineurin in mediating intrinsic forms of **heart disease.** 2) To utilize dominant negative calcineurin transgenic mice to determine the role of calcineurin signaling in pathophysiologic (extrinsic) forms of cardiac hypertrophy. Transgenic and pathophysiologic animal models are proposed to test the hypothesis that calcineurin/NF-AT act as a parallel regulatory pathway for cardiac reactive responses in vivo.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ROLE OF CYP2E1 AND ADH IN HUMAN TRI TERATOGENICITY**

  Principal Investigator & Institution: Mccarver, D Gail. Assoc. Prof. Pediatrics/Pharmacol. Pediatrics; Medical College of Wisconsin Po Box26509 Milwaukee, WI 532264801

  Timing: Fiscal Year 2001; Project Start 30-SEP-2001; Project End 29-SEP-2006

  Summary: Congenital **heart disease** is a major cause of mortality and morbidity. Exposure to halogenated hydrocarbons, specifically trichloroethylene (TRI), during pregnancy has been associated with congenital **heart disease** in animal models and retrospective epidemiology studies. Multiple studies support trichloracetic acid (TCA), a TRI metabolite, as the metabolite, as the more proximate teratogen. Overall hypothesis is that commonly observed levels of trichlorinated hydrocarbon exposure during human pregnancy are associated with an increase for offspring congenital **heart disease** and that genetic and phenotypic differences in the key enzymes responsible for the formation of TCA are associated with differences in offspring susceptibility. The specific aims are to 1) prospectively determine the risk of congenital **heart disease** from commonly observed levels of maternal exposure to trichlorinated hydrocarbons during pregnancy and 2) determine the differences in the offspring risk for congenital **heart disease** from maternal intersubject variation in the enzymes catalyzing the disposition of TRI among mother-infant pairs with documented trichlorinated hydrocarbon

exposure during pregnancy. To complete these aims, we will determine the presence of congenital **heart disease** by performing echocardiography on infants selected using a stratified recruitment strategy based on 1) maternal urinary trihalogenated hydrocarbon concentrations measured during pregnancy using a sensitive GCMS assay 2) maternal CYP2E1 genotype for a polymorphism associated with increased CYP2E1 activity in the presence of inducers and 3) maternal ADH2 genotype, a polymorphism which impacts the metabolism of other small molecular weight hydrocarbons. In addition, because multiple environmental factors alter CYP2E1 metabolic ability, some by post-transcriptional mechanisms, we will evaluate maternal white blood cell microsomal immunoreactive CYP2E1 during pregnancy as an important biomaker of increased offspring risk. Because the genotypes of interest are more common in the African-American population and because TRI emissions are documented in the Milwaukee area, an urban Milwaukee, African-American population will be studied. Maternal exposure to volatile organic solvents and ethanol will be measured and induced as potential confounders. Ultimately, the data generated in this proposal, will leaf to a better understanding of the genetic and environmental mechanisms determining susceptibility for congenital **heart disease** and provide the knowledge necessary for future public health prevention/intervention strategies.

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- **Project Title: ROLE OF DYSTROGLYCAN IN HEART DEVELOPMENT**

  Principal Investigator & Institution: Campbell, Kevin P.; University of Iowa Iowa City, IA 52242

  Timing: Fiscal Year 2001

  Summary: (Adapted from the Applicant's Abstract) The ultimate aim of this research is to determine the potential role of dystroglycan dysfunction in congenital **heart disease.** The overall objective of this project is to investigate the function of dystroglycan in cardiac embryogenesis. Extracellular matrix components and receptors are critical for the morphogenesis of the heart and are excellent candidates for involvement in congenital **heart disease** including atrioventricular canal defects and perimembranous interventricular septal defects. Dystroglycan is a broadly expressed cell surface extracellular matrix receptor that is linked to the cytoskeleton. Recent studies have indicated that dystroglycan plays a critical role in myogenesis and organogenesis. To investigate the role of dystroglycan in developmental processes, the investigators disrupted the dystroglycan gene in the mouse. The null mutation results in early embryonic lethality, prior to the onset of gastrulation. This phenotype stems from the failed development of Reichert's membrane, an extraembryonic basement membrane structure in which dystroglycan is also expressed. From these studies, dystroglycan seems to be required for either the anchorage of cells to the extracellular matrix or the assembly of networks of extracellular matrix proteins. Dystroglycan's involvement with extracellular matrix and its expression in various cell types suggest that it could be involved in many aspects of heart morphogenesis. To begin to address the role of dystroglycan in cardiac embryogenesis, the investigators will first define the developmental expression of dystroglycan in the heart (Specific Aim 1). To directly examine dystroglycan's function in heart embryogenesis, the investigators have proposed experiments to circumvent the early lethality of the dystroglycan null mutation so that the investigators may analyze dystroglycan's role later during heart development (Specific Aims 2 to 4). The first specific aim will utilize normal mouse embryos to establish a map of the spatial and temporal pattern of expression of dystroglycan during heart development. The second aim will analyze the cellular role of

dystroglycan in the development of cardiomyocytes in embryoid bodies. The third aim will use tetraploid complementation in order to rescue the Reichert's membrane defect and thus allow them to examine dystroglycan-null embryos that develop to later stages after the onset of heart development. The fourth aim will be the myocardium-specific disruption of the dystroglycan gene. The goal of the last two specific aims is to examine heart development in embryos that lack dystroglycan in all cells (Specific Aim 3) or just cardiomyocytes (Specific Aim 4). The complementary approaches outlined in these specific aims will yield a new understanding of the role of dystroglycan in heart development and will constitute a foundation for future investigations directed toward the identification of congenital **heart disease** patients with abnormalities in dystroglycan function.

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- **Project Title: ROLE OF NKX 2.5 IN THE CARDIAC CONDUCTION SYSTEM**

  Principal Investigator & Institution: Pashmforoush, Mohammad; University of California San Diego 9500 Gilman Dr, Dept. 0934 La Jolla, CA 92093

  Timing: Fiscal Year 2003; Project Start 01-JUL-2003; Project End 30-JUN-2008

  Summary: (provided by applicant): Dr. Pashmforoush is a highly trained clinician and scientist with training in molecular biology and clinical cardiology. The purpose of this grant proposal is to obtain support to enable Dr. Pashmforoush to become an independent investigator by the end of the proposed funding period. The proposed research will be performed in the laboratory of Dr. Kenneth Chien in the Institute of Molecular Medicine at the University of California, San Diego. During this time, Dr. Pashmforoush will receive the necessary training for electrophysiologic methodology to apply to murine models of human conduction system disease. UCSD and specifically, Dr. Chien's laboratory in the IMM will provide an ideal environment that has been extraordinarily successful for promoting and developing similar research interests. The long-term goals of this project are to study the mechanisms of human conduction system disease by using animal models. In this regard, cardiac arrhythmias remain important causes of cardiovascular morbidity mortality worldwide. Mutations in gene encoding the homeobox transcription factor NKX 2.5 are found to cause a non-syndromic human congenital **heart disease** with progressive conduction system disease leading to complete heart block. Following these observations, our hypothesis is that NKX 2.5 plays a critical role in the developmental maturation and maintenance of the cardiac conduction system and in particular the AV node. In the current proposal we will utilize a mouse model system to gain insight into the mechanistic pathways that link defects in the pattern of expression of NKX 2.5 with the associated cardiac arrhythmia. Similar to observations made in families with heart block, mice deficient for NKX 2.5 develop progressive AV nodal degeneration leading to complete heart block. Accordingly, our Specific Aims in this proposal are: To determine the role of NKX 2.5 in the development, maturation and maintenance of AV node and the distal conduction system. To define the role of NKX 2.5 in the development and maintenance of the atrial conduction system including the SA node, and to assess the postnatal requirement for the NKX 2.5 in the maintenance of the cardiac conduction system including the SA node, AV node, and the HIS-Purkinje system. We now have all the necessary tools to successfully accomplish the specific aims of this project.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ROLE OF TITIN IN HEART FUNCTION AND DISEASE**

  Principal Investigator & Institution: Granzier, Henk L. Professor; Vet & Comp Anat/Pharm/Physiol; Washington State University 423 Neill Hall Pullman, WA 99164

  Timing: Fiscal Year 2003; Project Start 30-SEP-1998; Project End 31-AUG-2008

  Summary: (provided by applicant): The long-term goals of this work are to understand the roles of titin in both diastolic and systolic dysfunction, and to provide important new insights applicable to human cardiac function and disease. Titin is the third myofilament of the sarcomere, with critical roles in myofibrillar assembly and generation of passive and restoring forces. In normal hearts, titin-based forces are the main determinants of passive myocardial stiffness at physiological sarcomere lengths. Our work has shown that mammals may tune passive myocardial stiffness by co-expressing varying mixes of stiff and compliant titin isoforms, obtained via post-transcriptional switching of splice patterns. To understand the role of titin in diastolic dysfunction, we propose to study various **heart disease** models and test the hypothesis that adjusting the titin isoform expression ratio is a widely used mechanism for passive stiffness modulation in **heart disease.** In addition, titin's elastic properties may be adjusted via post-translational processes (e.g., PKA-based phosphorylation), mechanisms that are faster than those that require isoform switching. Hence, we will also study how post-translational modification affects stiffness of the different cardiac titin isoforms. Our hypothesis is that as the expression of stiff titins increases, passive stiffness becomes more sensitive to post-translational regulation. Because titin may play a role in the Frank-Starling (FS) mechanism of the heart, by sensing sarcomere stretch, changes in passive tension due to post-transcriptional and post-translational processes may not only modulate passive stiffness, but also the FS mechanism. Thus, titin may also impact systolic function in heart failure and disease. Therefore, we will study the length dependence of calcium sensitivity in myocardium with different isoform expression ratios, to test the hypothesis that changes in titin isoform expression in disease may affect contractile performance. Finally, in addition to acquired alterations in titin expression in **heart disease,** genetic defects of the titin filament, including a recently reported frame shift mutation that eliminates titin's M-line region with its kinase domain, cause familial dilated cardiomyopathy (DCM). To understand the role of mutant titins in this type of DCM, mouse models will be studied in which gene targeting is used to remove segments of titin's M-line region. We will test the hypothesis that titin's M-line region is critically important for the structural integrity of contracting sarcomere.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SAFETY AND EFFICACY OF SERTRALINE FOR DEPRESSION CHF**

  Principal Investigator & Institution: Krishnan, Ranga R. Chairman and Professor; Psychiatry; Duke University Durham, NC 27706

  Timing: Fiscal Year 2003; Project Start 20-FEB-2003; Project End 31-JAN-2008

  Summary: (provided by applicant): The significance of co-morbid depression upon the medically ill has recently been recognized in the medical literature. Higher prevalence rates of mood disorders above that of the normal population has been found in patients who suffer from chronic medical illnesses, including vascular disease (cerebrovascular and coronary artery disease). Additional work has shown increased in-patient hospitalizations, cost of care, morbidity and mortality in these patients. More than 2 million United States citizens suffer from congestive heart failure (CHF), accounting for the highest category for hospitalization in the Medicare population, with annual

expenses exceeding $10 billion. One leading source of heart failure is ischemic **heart disease.** Despite knowledge that depressive disorders lead to increased morbidity, mortality and poorer outcomes in ischemic **heart disease,** little is currently known about the association of CHF and depression. There is evidence that the rate of depression may be high in the CHF population, but no studies have addressed the impact on morbidity and mortality in CHF patients when depression is adequately treated. Funding is requested for a two site, prospective placebo treatment of patients with congestive heart failure and clinically diagnosed major depression. Patients will be enrolled in this study with clinically diagnosed heart failure of NYHA functional > II. Patients will be interviewed and evaluated for major depression by use of the protocol developed by the NIMH-supported Duke Center for the Study of Depression in the Elderly. This includes sections that assess depressive symptoms, psychiatric co-morbidity, cognitive status, functional status and disability, daily and chronic stress, and social support, the longitudinal component of this study will include collecting data on all enrolled subjects. Information collected in these follow-up contacts will include deaths, re-hospitalizations, cardiac events, functional status/quality of life measures, and level of depressive symptoms.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SCOR - ISCHEMIC HEART DISEASE IN BLACKS**

  Principal Investigator & Institution: Chilian, William M. Professor; Physiology; Medical College of Wisconsin Po Box26509 Milwaukee, WI 532264801

  Timing: Fiscal Year 2001; Project Start 30-SEP-2000; Project End 31-JUL-2005

  Summary: The SCOR in Ischemic **Heart Disease** in Blacks at the Medical College of Wisconsin represents a multi-disciplinary proposal that focuses on solving basic (physiological, molecular, and genetic) and clinical (physiological and genetic) problems underlying this pathology. We have focused our efforts on diabetic **heart disease,** because this disease is of near epidemic proportions in the Black population, especially those with proven coronary artery disease, but they may also abrogate collateral growth in patients and augment the deleterious consequences of myocardial ischemia. Our program is composed of 4 Projects and 2 Core Facilities. Our strategy is to use experimental approaches that deviate from traditional methods to investigate the problems and to utilize corroborative experimental tools to test hypotheses at one level (documentation of physiological or pathophysiological responses) and then elucidate fundamental mechanisms at cellular, ion challenge molecular, and/or genetic levels. Projects 1 and 2 study mechanisms of coronary collateralization. Project 1, "Integrative Analysis of Coronary Adaptations to Ischemia" will delineate the temporal sequence of expression of growth factors, and their respective receptors from the initiation to the final stages of collateralization in dogs. This project also elucidate the mechanisms by which diabetes compromises coronary collateral growth. Project 2 (Genetic Basis of Coronary Artery Disease and Coronary Collateralization) will define the genetic basis of coronary artery disease and coronary collateralization in Black and White patients. This project will also examine the familial transmission of alleles involved in coronary artery disease. In Project 3, "Oxidant Stress and Human Coronary Microvascular Function," the effects of diabetes on vascular function, vessels on vascular cells obtained from human hearts of Black and White patients. We will also utilize molecular strains of rats: Brown Norway, which are resistant to ischemia and Dah, which are sensitive to ischemia and Dahl, which are sensitive to ischemia (Project 4: "Molecular Genetics of Cardioprotection"). The Dahl strain is also insulin resistant and demonstrates salt sensitive hypertension. The two cores will be involved as repositories for data and

administration, and data analysis. The Administration Core will contain a centralized file server for all results and will be accessible to all investigators. The Biostatistics Core will analyze all experimental results, and perform the association analyses of phenotype with genotype. We believe the science generated by this program will provide the template for rational pharmacological therapies to treat ischemic **heart disease** and will elucidate mechanisms contributing to ischemic **heart disease** in Blacks.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SCOR IN ISCHEMIC HEART DISEASE IN BLACKS**

  Principal Investigator & Institution: Loscalzo, Joseph; Wade Professor and Chairman; Medicine; Boston University Medical Campus 715 Albany St, 560 Boston, MA 02118

  Timing: Fiscal Year 2001; Project Start 30-SEP-1995; Project End 31-AUG-2005

  Summary: The Ischemic **Heart Disease** in Blacks Specialized Center of Research at Boston University School of Medicine will provide a multi-disciplinary approach to the study of vascular function and dysfunction relevant to the pathogenesis of cardiovascular disease in blacks. We postulate that the principal cardiovascular disorders common among blacks-hypertension, left ventricular hypertrophy, and myocardial ischemia without epicardial coronary disease-result from a specific vascular diathesis that is a consequence of increased oxidant stress. This unifying theme for our proposal represents a direct outgrowth of the first five years of SCOR support during which we demonstrated that one component of the vascular diathesis of blacks is nitric oxide insufficiency, which we find to be a consequence of its increased oxidative inactivation. The fundamental mechanism(s) underlying this increased oxidant stress is a major focus of this program, and we proposed to address the issue using a combination of approaches, including molecular, cellular, and genetic studies; animal experiments; and human studies. The projects presented in this program will focus on critical cellular mechanisms essential for protection form oxidant stress, and, in particular, will focus on one whose deficiency is common among African Americans, glucose-6-phosphate dehydrogenase (G6PD). Project 1 will test the hypothesis that a deficiency of G6PD, as the most important cellular source of reduced nicotinamide adenine dinucleotide phosphate (NADH) and, indirectly, glutathione, will lead to vascular dysfunction by decreased antioxidant protection and enhanced oxidative inactivation of nitric oxide. Project 2 poses the central hypotheses that G6PD is a critical determinant of superoxide production by NAD(P)H oxidases in the atherosclerotic milieu. In Project 3, the role of G6PD deficiency in myocardial ischemia-reperfusion injury will be assessed and the potential benefits of enhancing antioxidant capacity in G6PD-deficient states evaluated. Project 4, a new project for the program, will focus on the hypothesis that depletion of G6PD in cardiac myocytes will lead to increased reactive oxygen species generation resulting in hypertrophy, fetal gene expression, and apoptosis, serving as one cellular mechanism for adverse post-infarction remodeling. Human subject studies will be performed in Project 5 to test the hypothesis that G6PD deficiency is associated with an increase in oxidant stress in the vasculature and in accompanying loss of arterial endothelial-derived nitric oxide bioactivity; importantly, G6PD deficiency will e defined by specific activity measurements and gene sequencing for each individual studied. Thus, with this broad spectrum of approaches centered around a well- developed and well-integrated cardiovascular theme, this SCOR in Ischemic **Heart Disease** in Blacks should continue to provide the opportunity to identify important new mechanisms on the causes and consequences of vascular disease in blacks, and should lead to new approaches for its prevention and treatment.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SITOSTANOL & N-3 FATTY ACIDS ON STEROLS, FATTY ACIDS, LIPOPROTEINS, CHOLESTEROL**

  Principal Investigator & Institution: Connor, William E. Professor of Medicine; Oregon Health & Science University Portland, OR 972393098

  Timing: Fiscal Year 2001

  Summary: Participants in this research study will consist of 2 groups. One group will have a moderately high blood cholesterol level. The purpose of this study is to provide a nutritional, non-drug therapy to lower blood cholesterol levels and reduce the risk for **heart disease.** A substance in plants called sitostanol has been shown to block the absorption of dietary cholesterol that leads to a lower blood cholesterol level. Several companies have developed sitostanol-containing products. They have put sitostanol into margarine for use in this study. Another food that has been shown to decrease the risk of **heart disease** is fish oil. Fish oil contains omega-3 fatty acids. These fatty acids lower the blood triglyceride level, decrease the formation of blood clots that can block an artery and cause a heart attack and keep the heart beating in a normal rhythm. This study will show if sitostanol and fish oil are a good combination therapy to decrease the risk of **heart disease.** The second group of participants in this research study include those with an inherited disorder, sitosterolemia. The purpose of this study is to provide a nutritional, non-drug therapy to lower blood cholesterol and sitosterol levels and reduce the risk for **heart disease.** A substance in plants called sitostanol has been shown to block the absorption of dietary cholesterol that leads to a lower blood cholesterol level. This study will determine if sitostanol will also lower the blood sitosterol level. Several companies have developed sitostanol-containing products. They have put sitostanol into margarine for use in this study.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SLEEP HEART HEALTH STUDY**

  Principal Investigator & Institution: O'connor, George T. Associate Professor; Medicine; Boston University Medical Campus 715 Albany St, 560 Boston, MA 02118

  Timing: Fiscal Year 2001; Project Start 30-SEP-1994; Project End 31-AUG-2004

  Summary: The Sleep Heart Health Study (SHHS) was started in 1994 as a multicenter cohort study of the cardiovascular consequences of sleep-disordered breathing (SDB). The study's principal aims are to assess SDB as a risk factor for adverse cardiovascular outcomes, including incident coronary **heart disease** events, stroke, and hypertension, and accelerated increase in blood pressure with age. The SHHS protocol added an assessment of SDB to ongoing cohort studies of cardiovascular and other diseases, including the Framingham Offspring and Omni cohorts, the Hagerstown and Minneapolis/St. Paul sites of the Atherosclerosis Risk in Communities (ARIC) Study, the Hagerstown, Sacramento, and Pittsburgh sites of the Cardiovascular Health Study (CHS), the Strong Heart Study (SHS) sites South Dakota, Oklahoma, and Arizona, and cohort studies of respiratory disease in Tucson and of hypertension in New York. During its first four years (1994-1998), the SHHS was successfully started with full and high quality polysomnography (PSG) data obtained in the home from 6,440 participants, exceeding the recruitment target. The SHHS cohort, includes 3,039 men and 3,401 women 40 years of age or more, of whom 8.2 percent are African American, 9.6 percent are Native American, 1.3 percent are Asian, and 4.2 percent are Hispanic. In addition to PSG, data collection covered snoring and sleepiness and quality of life (QOL). Outcome assessment protocols are in place for all cohorts and the second SHHS examination is now in progress. Initial cross-sectional findings show that SDB is common and

associated with hypertension and self-reported cardiovascular disease (CVD). This application requests five years additional support to continue the SHHS. Further follow-up is needed to have sufficient power to test the primary SHHS hypotheses. Additionally in Years 7-9, PSG will be repeated to further characterize SDB in the participants and to describe the natural history of SDB. During the first five years, the SHHS has shown that large-scale research on sleep, SDB, and disease risk can be conducted in the community. Follow-up of the SHHS cohort will provide the data needed to characterize the cardiovascular consequences of SDB, along with its natural history.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SOCIAL AND OCCUPATIONAL INFLUENCES ON HEALTH AND ILLNESS**

  Principal Investigator & Institution: Marmot, Michael G.; U of L University College London University College London London,

  Timing: Fiscal Year 2001; Project Start 30-SEP-1993; Project End 31-AUG-2002

  Summary: The inverse gradient in Coronary **Heart Disease** (CHD) by socioeconomic status is one of the major unsolved puzzles in epidemiology. Whitehall II is a longitudinal cohort study of 10,308 female and male British Civil Servants set up in 1985 to test the hypothesis that psychosocial factors and differences in nutrition might explain this inverse gradient. NHLBI has been instrumental in supporting the measurement of psychosocial characteristics, in particular the psychosocial work environment and social support. There have now been four phases of data collection in the study, of which two have involved clinical screening with measurement of blood pressure, ECG and a blood sample. Five year incidence data, based on self-report coronary endpoints suggest that standard coronary risk factors explain no more than a third of the gradient, but factors in the psychosocial environment and height appear to explain the major part of the social gradient in CHD. There are also clear social gradients in plasma fibrinogen and in markers of the metabolic syndrome suggestive of insulin resistance. It is not yet clear whether these biochemical factors may mediate the link between psychosocial factors and CHD. In the next phase of data collection (1997), mostly funded by core British support, the investigators plan a further clinical examination combined with assessment of neurohumoral (hypothalamic-pituitary-adrenal axis and adrenal medullary) functioning as markers of physiological pathways linking the psychosocial environment and CHD. In the next three years the investigators seek funding from NHLBI to analyze the association between biochemical factors and validated coronary endpoints, to examine whether biomedical factors mediate the relationship between the psychosocial environment and coronary **heart disease.** To do this they need to continue to collect information on sickness absence from the Civil Service, mortality from National Health Service Central Records, records of illness from family physicians and hospitals. They also request funding to validate our self-report measures of work characteristics and to measure 24 hour urinary catecholamines as a component of assessment of adrenal medullarv function to support measures of heart rate variability.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SOCIAL CHANGE, HEART DISEASE, AND DEPRESSION**

  Principal Investigator & Institution: Fechner, Mary J. Anthropology; University of Oregon Eugene, OR 97403

Timing: Fiscal Year 2001; Project Start 01-SEP-2001

Summary: INVESTIGATOR'S The research is part of a larger goal to understand the role of culture on the experience of **heart disease,** depression, and their co-morbidity. The research begins with a cultural investigation of post-socialism in eastern Germany, but will culminate in an understanding of illness as a meaningful experience in a context of rapid social change. Research suggests that **heart disease** (and **heart disease** co-morbid with depression) is sensitive to biologic and psychosocial variables, but few studies have investigated the role of culture in **heart disease,** or its co-morbid state. This work builds on research into culture and hypertension, and culture and depression, to fill a gap in our understanding of the cultural processes at work in the expression of **heart disease,** but adds a new dimension by exploring co-morbidity. The research will take place in eastern Germany, where individuals are at increased risk to both conditions since 1989. Research aims include: 1) a community-based cultural study of the political-economic transition; 2) a community-based cultural study of cardiac and mental health; and 3) a clinic- or hospital based appraisal of cultural factors among patients expressing: a) **heart disease;** or, b) co-morbid **heart disease** and depression. Data sources will include: ethnographic interviews of post-socialism; idioms of distress, explanatory models, illness narratives, and semantic illness networks for **heart disease** and depression; a cultural consonance measure; measures for depression; demographics; and, local health statistics.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SODIUM IMAGING IN ISCHEMIC HEART DISEASE**

  Principal Investigator & Institution: Bottomley, Paul A. Russell H. Morgan Professor; Radiology; Johns Hopkins University 3400 N Charles St Baltimore, MD 21218

  Timing: Fiscal Year 2001; Project Start 01-JUL-1999; Project End 30-JUN-2003

  Summary: The exchange of intra- and extra-cellular sodium and potassium ions is essential to cell function and integrity. In the heart, evidence from animal and human radionuclide imaging studies of potassium and potassium analogs, and from biochemical and magnetic resonance spectroscopy (MRS) studies of animal models, indicates that sodium- potassium pump function is compromised during periods of myocardial ischemia and that it is lost in non-viable, infarcted tissue as intra- and extra-cellular pools equilibrate. Sodium (23Na) magnetic resonance imaging (MRI) is uniquely able to image and measure noninvasively naturally abundant, endogenous sodium in the body. 23Na MRI at magnetic fields of > 2.7 Tesla (T) in animal models demonstrate a 2-fold increase in 23Na signal levels in nonviable, histologically-confirmed, acute reperfused myocardial infarction (MI). Owing to its higher tissue concentration and sensitivity and its short relaxation time, 23Na MRI has an enormous sensitivity advantage compared, for example, with the detection of high-energy phosphate metabolites by phosphorus (31P) MRI (approximately 80- fold). Thus 23Na MRI is a potentially unique and important tool for assessing cellular metabolic and ionic function through altered sodium levels in patients with ischemic **heart disease** and/or MI. Yet 23Na is not now routinely possible on clinical 1.5T MRI scanners. Moreover, human 23Na MRI has never benefitted from new MRI hardware and software technology. In preliminary studies we implemented 23Na MRI on a clinical MRI scanner, and demonstrate altered 23Na MRI levels in MI. We show preliminary stress-23Na MRI data from patients with stress-induced ischemia detected metabolically by 31P MRS. Here we propose to develop and optimize human cardiac 23Na MRI on a clinical 1.5 T MRI/MRS system, by implementing high-speed MRI, 23Na phased-array detection, resolution-enhancement using a priori anatomic information, and methods of suppressing 23Na

signals from ventricular blood. We will use optimized 23Na MRI to characterize normal and infarcted human myocardium, and to test the hypotheses that 23Na MRI can differentiate normal from non-viable reperfused MI in patients as detected by radionuclide imaging, and compared with contrast- enhanced MRI. Further, the hypothesis that optimized 23Na MRI can detect stress-induced changes in sodium in energetically- compromised myocardium will be tested in combined stress-23Na/3 1P metabolic studies. The availability of thousands of clinical MRI scanners offers a great opportunity for advancing 23Na MRI as a tool for assessing sodium pump function in human **heart disease.**

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SODM FOR MANAGEMENT OF ISCHEMIC HEART DISEASE**

  Principal Investigator & Institution: Salvemini, Daniela; Metaphore Pharmaceuticals, Inc. 1910 Innerbelt Bus Center Dr St. Louis, MO 63114

  Timing: Fiscal Year 2001; Project Start 30-SEP-2001; Project End 29-MAR-2003

  Summary: (provided by applicant): Recent studies in models of cardiac ischemia/reperfusion injury have shown that a significant reduction in tissue damage from infarction can be achieved using MetaPhor's proprietary superoxide dismutase mimetics (SODm). In preliminary studies we found that M40403 a selective and potent SODm preserved cardiac function following ischemia/reperfusion injury. Based on the data that free radical generation, especially superoxide, may play a critical role in mediating tissue damage and death in heart following an ischemic event, such as a heart attack, we propose to develop a SODm mimetic of the M40403 class as a novel parenteral agent for administration after cardiac infarction or ischemia. M40403 will not be developed for this indication for reasons that are discussed in the proposal. Instead M40401, an SODm with higher catalytic activity, similar stability and improved toxicity to M40403 will be pursued. Given the enormous need for improving the outcome of cardiac ischemia and infarction, the novel findings of these SODm in protecting tissues from damage following ischemia/reperfusion injury, and the potential impact of this class of drugs on current management of **heart disease** in this country, we propose to move the program aggressively forward to clinical application and thus, are submitting this proposal for consideration. The Phase I application is focused on developing our mechanistic understanding of the role of superoxide in cardiac ischemia/reperfusion injury and to evaluate the effects of M40401 in protection against cardiac ischemia and to explore the potential use of this drug in post-ischemia management of **heart disease.** PROPOSED COMMERCIAL APPLICATION: NOT AVAILABLE

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SPECIALIZED CENTER OF RESEARCH IN ISCHEMIC HEART DISEASE**

  Principal Investigator & Institution: Mendelsohn, Michael E. Professor; New England Medical Center Hospitals 750 Washington St Boston, MA 021111533

  Timing: Fiscal Year 2001; Project Start 01-FEB-2000; Project End 31-JAN-2005

  Summary: This proposal describes a Specialized Center for Research (SCOR) in Ischemic **Heart Disease** that tests the hypothesis that the genetics, expression and function of cardiovascular estrogen receptors (ER) and estrogen-regulated target genes mediate protection against ischemic diseases and their sequelae, including vascular dysfunction, post- myocardial infarction remodeling, and arrhythmias. The program is based on widely noted gender differences in ischemic disease, beneficial effects of estrogen on

cardiovascular diseases in women, and recent data demonstrating: 1) the presence of functional ER in cardiovascular diseases in women and men; 2) important ER target genes in cardiovascular tissues; and 3) an important role for ER and the genes they regulate in vascular and myocardial physiology. The hypothesis is investigated through genetic, molecular, cellular, animal and human studies in five highly integrated and cooperative projects that expand on existing strengths at New England Medical Center and Tufts University, as well as MIT, Boston University, and the Framingham, Heart Study. The five projects Proposed include: Project 1: "Genetics of Estrogen and Cardiovascular Responses", a genetic analysis of cardiovascular phenotypes and ER/ER-related genes from subjects in the Framingham Offspring study; Project 2: "Selective ER Modulation: Effects in Post Menopausal Women Following Myocardial Infarction", a secondary prevention trial at New England Medical Center with the selective ER modulator raloxifene; Project 3: "Cardiac ER and MI: Mouse Models", studies of ventricular remodeling, arrhythmogenic changes, and myocardial gene expression in wild-type, ERalphaKO and ERbetaKO ventricular remodeling, arrhythmogenic changes, and myocardial gene expression in wild-type, ERalphaKO and ERbetaKO mice following MI; Project 4: "ER Regulation of NO Synthases," studies of the rapid activation of endothelial eNOS by ERalpha, and the longer-term induction of vascular smooth muscle iNOS gene expression by ERbeta; and Project 5: "ER and Smooth Muscle BKCa Channels", studies of ER regulation of ion channel function ins ingle vascular smooth muscle cells. These Projects are served by three Cores: Administrative, Mouse (with Transgenic, Physiology, Cell Culture and Histology components), and Statistical, all build on existing Cores at the Molecular Cardiology Research Institute and Tufts-NEMC. Together, these five Projects define a broad-based program to explore new mechanisms of coronary myocardial regulation by ER with direct consequences for the diagnosis and management of ischemic cardiovascular diseases, and the potential to evolve a new class of therapies for these disorders.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: STRESS REDUCTION AND ATHEROSCLEROTIC CVD IN BLACKS**

  Principal Investigator & Institution: Schneider, Robert H. Dean; None; Maharishi University of Management 1000 N 4Th St, Ste 2 Fairfield, IA 52557

  Timing: Fiscal Year 2001; Project Start 01-MAR-1994; Project End 31-MAY-2004

  Summary: The President's Racial and Ethnic Health Disparities Initiative of 1998 emphasizes that African Americans suffer from disproportionately high rates of cardiovascular disease (CVD) morbidity and mortality compared to white Americans. Numerous controlled studies suggest that this disparity is associated with chronic psychosocial and environmental stress. For these reasons, the NHLBI Working Group on Research in Coronary **Heart Disease** in Blacks has mandated behavior and prevention in this minority population as a national research priority. Our research team has previously demonstrated in NHLBI-supported randomized controlled trials that hypertension can be effectively treated in high risk African Americans with stress reduction using the Transcendental Meditation (TM) program compared to control procedures. These and other studies have also reported clinically significant improvements in other CVD risk factors, psychosocial stress, myocardial ischemia, left ventricular mass and mortality rates from CVD and all-causes in high risk subjects randomized to active stress reduction. Preliminary findings from our recently completed, NHLBI-supported clinical trial of stress reduction in the prevention of hypertensive **heart disease** in inner city African Americans has indicated that atherosclerotic CVD, assessed by carotid artery intima-media thickness (IMT),

significantly regresses after eight months of active stress reduction intervention compared to control education. These results were comparable to effects of lipid lowering therapies or extensive lifestyle modification. This proposed continuation project will extend these findings by investigating the effectiveness of active stress reduction in the secondary prevention of atherosclerotic CVD in African Americans. This five year study will be a randomized, single-blind, controlled, community-based trial involving 184 African American subjects with known coronary **heart disease.** Male and female subjects will be enrolled at our on-going field site, Martin Luther King-Drew Medical Center in inner city Los Angeles. After baseline testing, subjects will be randomized to either active stress reduction with TM or health education control-both in addition to usual medical care-and posttested after 12 months. The primary outcome will be carotid artery atherosclerosis (IMT) measured non-invasively by quantitative B-mode ultrasonography. Secondary measures will include traditional CVD risk factors (blood pressure, lipids, smoking, exercise), psychosocial stress, quality of life and cost effectiveness. The results of this clinical trial will yield valuable new knowledge for the prevention of CVD through behavioral means in high risk African Americans.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SUBCLINICAL HEART DISEASE IN INSULIN-DEPENDENT DIABETES**

  Principal Investigator & Institution: Rewers, Marian J. Professor of Pediatrics; Preventive Med and Biometrics; University of Colorado Hlth Sciences Ctr P.O. Box 6508, Grants and Contracts Aurora, CO 800450508

  Timing: Fiscal Year 2001; Project Start 01-SEP-1999; Project End 31-AUG-2004

  Summary: Approximately 10 percent of premature coronary artery disease (CAD) morbidity and mortality in the general population is due to insulin dependent diabetes mellitus (IDDM). By age 55, 35 percent of IDDM patents die of CAD, in contrast to only 8 percent of nondiabetic men and 4 percent of women. In the U.S., IDDM affects at least 750,000 persons and this number is growing rapidly as the effect of increasing incidence and improved survival. Tight blood glucose control ran slow the development of microvascular complications but a protective effect on **heart disease** has not been convincingly demonstrated. This observational population-based study will evaluate cross- sectionally a population-based group of 800 IDDM patients aged 20-49 years and 600 of their non-diabetic spouse/partner controls using the electron-beam computed tomography (EBCT). We will compare the patients and controls in terms of the amount and anatomical distribution of coronary artery calcium (CAC), a marker of atherosclerosis, and the left ventricular (LV) area, a marker of LV hypertrophy and diabetic cardiomyopathy. We will define the demographic, metabolic, and behavioral factors associated with increased CAC and LV area. Using standard epidemiological methods, we will determine the prevalence of clinical CAD, defined by previous MI, revascularization, or angina in the study population. In 100 asymptomatic high-risk IDDM patients (CAC greater than or equal to 20 or LV area greater than 60 cm2), in 50 low-risk patients (CAC and LV area below these cut-offs), and in 20 nondiabetic controls age-sex matched to the high-risk patients, we will perform ECG-gated rest-stress technetium-99m sestamibi single-photon emission computed tomographic imaging (MIBI SPECT). This will help us to determine the presence of myocardial perfusion defects and to quantify myocardial perfusion reserve as well as to relate these findings anatomically to the distribution of CAC by EBCT. In addition, we will determine the LV volumes, ejection fraction, wall motion and thickening, and relate these findings to LV area by EBCT. Finally, the study cohort of 800 IDDM patients and 600 non diabetic

spouses/partners will be followed up for a period of 3 years to measure the change in CAC and LV area using a repeat EBCT and to identify the metabolic and behavioral risk factors for progression in these indices. We will also monitor cause-specific mortality and ascertain all fatal and non-fatal cardiac events. In the subgroup of 100 high-risk IDDM patients studied with the MIBI SPECT at the baseline and in all low-risk patients whose CAC increased by more than 50 during the follow-up, we will evaluate using MIBI SPECT the change in myocardial perfusion, LV volumes, ejection fraction, wall motion and thickening, as well as to relate these findings to the change in CAC and LV area by EBCT. This proposed study will better define the causes of increased **heart disease** risk in IDDM patients, develop appropriate screening methods, and set the stage for effective primary prevention.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SURGICAL TREATMENT OF CARDIAC ARRHYTHMIAS**

  Principal Investigator & Institution: Boineau, John P. Medical Science Service; Surgery; Washington University Lindell and Skinker Blvd St. Louis, MO 63130

  Timing: Fiscal Year 2001; Project Start 08-AUG-1983; Project End 31-JUL-2003

  Summary: This renewal application requests five years' support for work now in continuous progress for over 15 years at Washington University. Dr. John Boineau, the new PI, has replaced Dr. James Cox, the former P1, who transferred to Georgetown University Hospital. Dr. Cox remains as a special consultant. The broad aims continue to be the direct or surgical ablation of cardiac arrhythmias. The emphasis of the current renewal is focused upon the development of a new procedure, the radial incisions approach (RIA), to eradicate atrial fibrillation (AF) and restore atrial transport function and is directed primarily toward patients undergoing surgery for valvular or ischemic **heart disease.** Conventional valve or CABG surgery does not eliminate and may not prevent AF in these patients. The availability of an effective means of eradicating this arrhythmia in these patients at the time of surgery would permit control of rate and rhythm, limit embolic stroke, and improve cardiac performance, outcome, and the quality of life. Whereas the Maze and RIA assume randomly distributed and changing reentry which are eliminated without prior activation mapping, new data indicate that some forms of AF result from (spatially) stable reentry which can be identified by new mapping methods and focally ablated. Thus, a second project is directed toward map guided, focal cryoablation of AF. This could be performed off bypass as a more limited and rapid alternative to the more extensive and (bypass) time consuming RIA procedure. A third project is targeted at prevention and correction of postoperative atrial flutter (AFL) after the Fontan operation in congenital heart patients or after lung transplant surgery. Studies will be performed in both realistic animal models with atrial enlargement and patients with AF and AFL and will center about the use of new automated, 3-D mapping techniques and rapid numerical analysis of potentials recorded simultaneously from to 512 electrodes during the arrhythmias. Preliminary observations indicate that the proposed studies are feasible, will provide new information regarding the different mechanisms of AF and AFL that are related to atrial enlargement and/or atrial surgery, and this data will be used to develop the new surgical ablation techniques to control or prevent these arrhythmias.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SURVEILLANCE AND ANALYSIS OF THE UNC ALUMNI HEART STUDY**

  Principal Investigator & Institution: Siegler, Ilene C. Professor of Medical Psychology; Psychiatry; Duke University Durham, NC 27706

  Timing: Fiscal Year 2001; Project Start 01-MAY-1996; Project End 31-JUL-2005

  Summary: (investigator's abstract): The UNC Alumni Heart Study continues to examine the impact of hostility on health behaviors and psychological status at midlife to test the prospective associations of hostility with coronary **heart disease** (CHD) events and other health outcomes. The Specific Aims of the proposed research are: [1] To better understand the dynamic interrelationships of psychosocial and behavioral risk factors of the adult life span, we will map the trajectories of hostility, depression, smoking, body mass, exercise patterns, and alcohol consumption using multiple assessments from age 19 to age 60. It is predicted that a significant proportion of the change in risk behavior will be due to trajectories of hostility and depression, operating singly and in combination over time. [2] To test the prospective associations of hostility, depression, and other psychosocial variables (e.g., social support and job strain) with coronary events and mortality observed while the cohort is middle-aged. [3] To broaden the scope of the psychosocial variables to examine individual differences in personality over the life course and dietary practices at midlife in addition to the indicators noted above, and [4] To better understand the effect of gender on the natural history of coronary disease and coronary risk profiles in women, we will monitor changes in menopausal status, and patterns of hormone replacement therapy use among women during midlife and study the associations of these factors with the other risk indicators. In sum, although the literature suggesting that psychosocial factors play a significant role in the etiology of CHD in older samples is convincing, major gaps remain with respect to understanding the associations between psychosocial factors and premature coronary **heart disease** and mortality during the middle years. Adding additional measures to the present rich data base, places the UNC Alumni Heart Study in an excellent position to help fill these gaps in the next 5 years, as these members of the early Baby Boom Cohort approach age 60.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SYSTEMIC EFFECTS OF PERIODONTITIS**

  Principal Investigator & Institution: Beck, James D. Professor and Chairman; Dental Ecology; University of North Carolina Chapel Hill Office of Sponsored Research Chapel Hill, NC 27599

  Timing: Fiscal Year 2001; Project Start 01-MAY-1996; Project End 30-APR-2003

  Summary: It is our central hypothesis that periodontal diseases, which are chronic gram-negative infections represent a previously unrecognized risk factor for atherosclerosis and thromboembolic events Previous studies have demonstrated an association between periodontal disease severity and risk of coronary artery disease and stroke. We hypothesize that this association may be due to an intrinsic underlying inflammatory response trait that places an individual at high risk for developing both periodontal disease and atherosclerosis. We further suggest that periodontal disease, once established, provides a biological burden of endotoxin (LPS, lipopolysaccharide) and inflammatory cytokines (especially TxA2, IL- 1B, PGE2 and TNFa) which serve to initiate and exacerbate atherogenesis and thromboembolic events. We propose to test these hypotheses by performing cross-sectional study on 14,000 participants in a longitudinal study of Atherosclerosis Risk Communities (ARIC) to determine the

contribution of periodontal infection variables to existing multivariate models of atherosclerosis. Using a cross-sectional design we will measure periodontal disease variables including periodontal probing depths and clinical attachment levels. Plaque samples will be collected for storage and later quantitation of Porphyromonas gingivalis, as S. Sanguis. Gingival crevicular fluid will be collected for the quantitation of Prostaglandin E2 (PGE2) thromboxane B2 (TxB2), Interleukin-1B (IL-1B) and tumors necrosis factor a (TNFa). Serum sample will be analyzed for whole-cell and LPS-specific antibody titers against selected periodontal pathogens. These measures will be sued to test associations with clinical measures of **heart disease,** heart attack, death from **heart disease,** and direct ultrasound measures of carotid and popliteal intima-media thickening and lesions as well as atherogenic risk factors that continue to be gathered in the ARIC Study. More specifically, we will determine whether the local gingival crevicular fluid levels of TxB2, IL-1B, TNFa and PGE2 are elevated in cases of severe atherosclerotic stenosis and whether elevated levels of these mediators are associated with other atherosclerosis risk factor including elevated serum lipid variables, serum TxB2 and fibrinogen.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: TEACHING NUTRITION TO PREVENT CARDIOVASCULAR DISEASES**

  Principal Investigator & Institution: Walker, W Allan. Director; Pediatrics; Harvard University (Medical School) Medical School Campus Boston, MA 02115

  Timing: Fiscal Year 2001; Project Start 01-SEP-2000; Project End 31-AUG-2005

  Summary: As Director of the Division of Nutrition at Harvard Medical School (HMS), Dr. Allan Walker will direct the development and implementation of a curriculum to teach nutrition principles and clinical practice skills for the prevention of cardiovascular diseases, hypertension, diabetes and obesity to medical students, house staff and practicing physicians. The nutrition theme will enrich and extend the current curriculum and core clinical clerkships and create a new elective clinical rotation. The specific aims are to: 1) develop a four-year, cardiovascular disease specific nutrition curriculum for HMS students by a) developing didactic lectures; b) developing clinical cases based on patients in Dr. Welty's secondary prevention program, Heart and Sole, and women's and lipid clinics for case-based teaching; c) developing a teaching module on women, diet and **heart disease** in collaboration with the NIH funded Harvard Center of Excellence in Women's Health. All lectures, cases and minicourses will be Web-based for HMS students, other medical schools and as CME credit for practicing physicians; 2) add content to the clinical years by a) providing a Nutrition and **Heart Disease** Prevention Inpatient Teaching Service which will teach students to apply nutrition skills and knowledge on Medicine and Ambulatory Clerkships; b) adding a new outpatient Preventive Cardiology Clinical Rotation for medical students, residents and fellows; c) developing the Heart and Sole program on a Native-American reservation to provide an elective on nutrition and **heart disease** for medical students from many schools; and d) increasing research opportunities in nutrition and prevention of cardiovascular diseases; 3) designing and implementing faculty development and training materials; 4) evaluating the impact of the nutrition curriculum, a portion of which involves adding nutrition stations to the objective structured clinical exam (OSCE) already in place at HMS; the OSCE could be disseminated to other medical schools; and 5) broaden exposure to the heart- disease nutrition connection among health care professionals by providing a CME course and web site. This career award will establish Dr. Walker as a leader in medical education in nutrition, his long-term career goal.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: THE CLINICAL UTILITY OF ENDOTHELIAL FUNCTION IN PAD**

  Principal Investigator & Institution: Vita, Joseph A. Associate Professor; Boston Medical Center Gambro Bldg, 2Nd Fl, 660 Harrison Ave, Ste a Boston, MA 02118

  Timing: Fiscal Year 2003; Project Start 22-SEP-2003; Project End 31-AUG-2008

  Summary: (provided by applicant): Peripheral arterial disease (PAD) produces considerable morbidity and mortality, and the precise factors that determine disease progression and the responses to therapy remain largely unknown, In addition to their risk for critical limb ischemia or graft failure, PAD patients also have markedly increased risk for coronary **heart disease,** particularly during the stress of vascular surgery, It is clear that new approaches are needed for optimal risk assessment and therapy. Targeting endothelial function represents a major new departure from traditional methods for assessing cardiovascular disease risk. The central hypothesis of this proposal is that endothelial dysfunction is a critical mediator of both PAD and coronary **heart disease** events and measuring endothelial function will enhance both the risk assessment and therapy in PAD patients. Recent studies by the applicants strongly support this contention and establish the prognostic value of endothelial dysfunction in PAD patients undergoing vascular surgery. A key unresolved question is whether reversing endothelial dysfunction will directly reduce risk. This finding would more firmly establish endothelial dysfunction as a mediator of both PAD and coronary **heart disease** risk and further validate its clinical utility. We propose the following specific aims: 1. To determine whether reversing endothelial dysfunction ameliorates perioperative risk in PAD patients. Patients referred for elective vascular surgery will be treated with high dose atorvastatin (80 mg/day), ascorbic acid (500 mg/day), or placebo in a randomized, double blind, fashion beginning a month prior to surgery and continuing for a month after surgery. Non-invasive assessment of vascular function will be performed at baseline and immediately prior to surgery. Patients will be monitored for cardiovascular events (cardiac death, myocardial infarction, unstable angina, and stroke) in the 30-day postoperative period. The goal is to determine whether improvement in vascular function independently predicts outcome (irrespective of which treatment produces the improvement). 2. To determine whether endothelial dysfunction predicts long-term (2-year) PAD and coronary **heart disease** risk in PAD patients. 3. To determine whether systemic markers of oxidative stress and inflammation relate to endothelial dysfunction and long-term PAD and coronary **heart disease** risk. This work will provide novel information about the pathogenesis and management of PAD.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: THE GENETIC ETIOLOGY OF LEFT-SIDED CARDIAC DEFECTS**

  Principal Investigator & Institution: Goldmuntz, Elizabeth; Children's Hospital of Philadelphia 34Th St and Civic Ctr Blvd Philadelphia, PA 19104

  Timing: Fiscal Year 2003; Project Start 01-SEP-2003; Project End 30-JUN-2008

  Summary: (provided by applicant): Congenital **heart disease** (CHD) is the most common major birth defect affecting 4-8 per 1000 livebirths. Left-sided cardiac defects (LSCD) include valvar aortic stenosis, coarctation of the aorta and hypoplastic left heart syndrome, and account for at least 10% of CHD. Although LSCD are associated with significant morbidity and mortality, their etiology is largely unknown. Very few, if any, environmental causes have been identified and LSCD are associated with relatively few

consistent chromosomal alterations or genetic syndromes. Nonetheless, studies point to a genetic contribution to these conditions, given an increased risk of recurrence and small, multiplex families. LSCD appear to share a common genetic etiology as different defects can occur in different members of a single family. Large multiplex families or affected sibling pairs amenable to genome wide linkage analyses are very rare, consistent in part with a complex mode of inheritence. The goal of this program is to identify genetic factors that contribute to the etiology of LSCD using techniques which dissect complex disorders. This investigation proposes that there are susceptibility loci and novel mutations that contribute to the etiology of LSCD. To investigate this hypothesis, this program will: (I) identify and characterize common susceptibility loci for LSCD using family-based association studies, and (II) identify and characterize rare or novel mutations in candidate genes in subjects with LSCD. A large cohort of subjects has been recruited. Family based association analyses and mutation analyses have already identified potential disease-related alleles and mutations respectively. To accomplish our goals, we will continue to recruit subjects, genotype them for a chosen set of common variants, and test them for association to LSCD. The role of maternal genetic effects for a subset of the putative susceptibility loci will also be evaluated. To complement the association studies, we will screen subjects for mutations of candidate genes by Conformation Sensitive Gel Electrophoresis and direct sequencing. If a sequence variant is identified, control populations will be tested for the same variant to determine disease relevance. The functional significance of mutations will also be assessed. This investigation will begin to dissect the genetic factors that contribute to the development of LSCD. The findings will allow for improved understanding of the disease-mechanism, more precise family counseling, and may identify preventive measures. In addition, these investigations will lead to future studies that assess the relationship of genotype to clinical outcome, and allow us to improve upon our clinical management accordingly.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: THE ROLE OF IGLONS IN CARDIAC DEVELOPMENT AND DISEASE**

  Principal Investigator & Institution: Grossfeld, Paul D. Pediatrics; University of California San Diego 9500 Gilman Dr, Dept. 0934 La Jolla, CA 92093

  Timing: Fiscal Year 2003; Project Start 01-APR-2003; Project End 31-MAR-2008

  Summary: (provided by applicant): Dr. Grossfeld is a highly trained, board-certified pediatric cardiologist who is devoted to studying the genetic basis of congenital **heart disease.** The purpose of this grant proposal is to obtain support to enable Dr. Grossfeld to become an independent investigator by the end of the proposed funding period. The proposed research will be performed in the laboratory of Dr. Anthony Wynshaw-Boris in the Department of Pediatrics at the University of California, San Diego. During this time, Dr. Grossfeld will receive the necessary training for incorporating human and animal genetic model system approaches for studying genes causing hypoplastic left heart syndrome (HLHS). UCSD and specifically, Dr. Wynshaw-Boris, provide an ideal environment that has been extraordinarily successful for promoting and developing similar research interests. The long-term goal of this project is to study the role of OBCAM and Neurotrimin, two members of the IgLON subfamily of genes, in cardiac development and in the generation of congenital heart defects, including HLHS. HLHS is one of the most severe congenital heart defects, accounting for 20% of all deaths in infants with congenital **heart disease.** To date, no causative gene for HLHS has been reported. The specific aims of this project are: Specific Aim 1): Characterization of the

expression of OBCAM and Neurotrimin in fetal and adult heart; Specific Aim 2): Alteration in the expression of OBCAM and Neurotrimin utilizing mouse and chicken genetic model systems, and Specific Aim 3): Mutation analysis for point mutations and microdeletions in OBCAM and Neurotrimin in patients with isolated heart defects that occur in Jacobsen syndrome, and for single nucleotide polymorphisms in OBCAM and Neurotrimin in patients with Jacobsen syndrome.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: THE SLEEP HEART HEALTH STUDY**

  Principal Investigator & Institution: Quan, Stuart F. Professor of Medicine; Medicine; University of Arizona P O Box 3308 Tucson, AZ 857223308

  Timing: Fiscal Year 2001; Project Start 30-SEP-1994; Project End 31-AUG-2004

  Summary: The Sleep Heart Health Study (SHHS) was started in 1994 as a multicenter cohort study of the cardiovascular consequences of sleep-disordered breathing (SDB). The study's principal aims are to assess SDB as a risk factor for adverse cardiovascular outcomes, including incident coronary **heart disease** events, stroke, and hypertension, and accelerated increase in blood pressure with age. The SHHS protocol added an assessment of SDB to ongoing cohort studies of cardiovascular and other diseases, including the Framingham Offspring and Omni cohorts, the Hagerstown and Minneapolis/St. Paul sites of the Atherosclerosis Risk in Communities (AMC) Study, the Hagerstown, Sacramento, and Pittsburgh sites of the Cardiovascular Health Study (CHS), the Strong Heart Study (SHS) sites in South Dakota, Oklahoma, and Arizona, and cohort studies of respiratory disease in Tucson and of hypertension in New York. During its first four years (1994-1998), the SHHS was successfully started with full and high quality polysomnography (PSG) data obtained in the home from 6,440 participants, exceeding the recruitment target. The SHHS cohort, includes 3,039 men and 3,401 women 40 years of age or more, of whom 8.2 percent are African American, 9.6 percent are Native American, 1.3 percent are Asian, and 4.2 percent are Hispanic. In addition to PSG, data collection covered snoring and sleepiness and quality of life (QOL). Outcome assessment protocols are in place for all cohorts and the second SHHS examination is now in progress. Initial cross-sectional findings show that SDB is common and associated with hypertension and self-reported cardiovascular disease (CVD). This application requests five years additional support to continue the SHHS. Further follow-up is needed to have sufficient power to test the primary SHHS hypotheses. Additionally in Years 8-9, PSG will be repeated to further characterize SDB in the participants and to describe the natural history of SDB. During the first five years, the SHHS has shown that large-scale research on sleep, SDB, and disease risk can be conducted in the community. Follow-up of the SHHS cohort will provide the data needed to characterize the cardiovascular consequences of SDB, along with its natural history. This proposal is a request to fund the University of Arizona Field Center of the SHHS to fulfill its role performing repeat PSG and cardiovascular follow-up of 909 participants in this multicenter study.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: THE SLEEP HEART HEALTH STUDY: READING CENTER APPLICATION**

  Principal Investigator & Institution: Redline, Susan S. Professor of Pediatrics, Medicine & Epid; Medicine; Case Western Reserve University 10900 Euclid Ave Cleveland, OH 44106

Timing: Fiscal Year 2001; Project Start 30-SEP-1999; Project End 31-AUG-2004

Summary: The Sleep Heart Health Study (SHHS) was started in 1994 as a multicenter cohort study of the cardiovascular consequences of sleep-disordered breathing (SDB). The study's principal aims are to assess SDB as a risk factor for adverse cardiovascular outcomes, including incident coronary **heart disease** events, stroke, and hypertension, and accelerated increase in blood pressure with age. The SHHS protocol added an assessment of SDB to ongoing cohort studies of cardiovascular and other diseases. During its first four years (1994-1998), the SHHS was successfully started with full and high quality polysomnography (PSG) data obtained in the home from 6,440 participants. Initial cross-sectional findings show that SDB is common and associated with hypertension and self-reported cardiovascular disease (CVD). The clinical centers are now requesting another five years of support to collect additional endpoints needed for testing primary SHHS hypotheses. Additionally in Years 7-9, PSG will be repeated to further characterize SDB in the participants and to describe the natural history of SDB. This application requests support that will allow Case Western Reserve University to continue to serve as the Polysmongraphy Reading Center for the SHHS. In this capacity, we will: a. Provide centralized training for aspects of SHHS related to the performance and interpretation of sleep studies. b. Provide ongoing technical support to the clinical sites for the performance of sleep studies; c. Provide timely review (for quality and medical alerts) and scoring of all records, generating reports needed for participant feedback and data analysis; d. Participate in on-going quality assurance efforts to maintain high levels of scoring accuracy and reliability; e. Develop, implement and monitor the technical performance of PSGs at clinical the field sites; f. Provide reports on study quality to the Steering Committee and QA Committee; g. Collaborate with other SHHS investigators to assist in protocol development, data analysis, data interpretation, and manuscript preparation. The SHHS has shown that large-scale research on sleep, SDB, and disease risk can be conducted in the community and high quality PSG data can be collected from multiple sites by using centralized and intensive methods for training filed technicians and polysomnologists, and for monitoring data quality. Follow-up of the SHHS cohort will provide reliable and accurate data needed to characterize the cardiovascular consequences of SDB, along with its natural history.

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- **Project Title: TISSUE ENGINEERING OF A CARDIAC PATCH**

  Principal Investigator & Institution: Terracio, Louis; Associate Dean for Resarch; Basic Science and Craniofacial Biology; New York University 15 Washington Place New York, NY 10003

  Timing: Fiscal Year 2002; Project Start 30-SEP-2002; Project End 31-JUL-2005

  Summary: (provided by applicant): Despite a dramatic decline over the past 15 years, cardiovascular disease remains the leading cause of death and disability in the Western world. The adult onset disease processes that compromise cardiac function include, infarction, ischemic **heart disease,** myocarditis, and a variety of idiopathic cardiomyopathies. The focal loss of muscular tissue, as a result of a congenital defect or a disease process, alters the unique architectural arrangement of the heart and impairs its function. An engineered segment of artificial myocardium (cardiac patch) potentially offers a nearly unlimited source of material for reconstructive surgery. In preliminary experiments from our lab we have successfully constructed small, multilayered cultures of fetal and neonatal cardiac myocytes that exhibit a tissue-like pattern of organization. These multilayered cultures are composed of myocytes that have an elongated, rod-like cell shape, but require further development before they would be suitable for

transplantation. This proposal takes advantage of the Pl's experience in cardiac development, in vitro cultivation of cardiac myocytes on various ECM components, in vitro mechanical stimulation of cells in culture, morphological characterization of tissues, proof of concept studies, and established collaborations with bioengineering colleagues. The Specific Aims of this proposal are: 1) To use specially fabricated collagen substrates and a series of bioreactors to produce histotypic cultures of cardiac cells suitable for transplantation. 2) Characterize and compare the artificial myocardium to the intact heart by morphometric, biochemical and molecular techniques. 3) Transplant the artificial myocardium to in vivo locations that will allow vascularization of the tissue. The transplanted cultures will be compared by morphometric, biochemical, and molecular techniques to the structure and function of the intact heart. Data from these studies will identify the mechanical and chemical parameters necessary to produce the three-dimensional organization of the heart patches. These cultures will provide a potential source of biological material for repairing focal damage to the myocardium.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ULTRAFINE PARTICULATE MATTER & CARDIORESPIRATORY HEALTH**

  Principal Investigator & Institution: Delfino, Ralph J. Associate Professor; Medicine; University of California Irvine Campus Dr Irvine, CA 92697

  Timing: Fiscal Year 2003; Project Start 30-SEP-2003; Project End 31-JUL-2008

  Summary: (provided by applicant) **Heart disease** is the leading cause of death and hospitalization among the elderly population, which makes the identification of preventable causes for **heart disease** morbidity and mortality a major goal of epidemiologic research. Numerous studies have shown associations of outdoor particulate matter (PM) air pollution with cardiovascular hospital admissions and mortality. The causal pollutant components and physiologic mechanisms for these associations are not fully understood. There is evidence that airway inflammation resulting from airway deposition of ultrafine particles (< 0.1 fm in diameter) could lead to an increase in thrombogenic and inflammatory activity in the blood, and to a disturbance in cardiovascular function, resulting from oxidant stress responses at extra-pulmonary sites, including the vascular endothelium of the heart. This is expected to increase the risk of adverse cardiovascular outcomes, particularly in people with underlying coronary **heart disease** (CHD). We propose to conduct a panel study with repeated measurements to evaluate acute cardiovascular and respiratory health effects of ultrafine PM personal, indoor and outdoor exposures. Over seven month periods, we will follow 72 nonsmoking elderly individuals with CHD living in areas with high air pollution levels in the Los Angeles Air Basin of California. The design will maximize the utility of intensive exposure assessments by measuring multiple interrelated clinical, physiological and biochemical outcomes. The specific aims will address the following hypotheses: 1) Exposure to ultrafine particles will be associated with increased circulating biomarkers of inflammation and thrombosis, increased blood pressure, adverse cardiac clinical outcomes, and increases in a biomarker of airway inflammation, exhaled nitric oxide; and 2) These associations will be stronger for measurements of particle components and certain ambient sources thought to influence inflammatory processes through oxidant damage. We will also evaluate relationships of outcomes with accumulation mode PM (0.18-2.5 fm) and coarse mode PM (2.5-10 fm). We will assess whether estimates of association for predicted (adjusted) personal or indoor exposure to ultrafine or accumulation mode PM of outdoor origin are stronger than estimates of association for unadjusted (raw) personal or indoor exposures. Results of

this study will advance knowledge on the cardiovascular and respiratory effects of ultrafine particles. Our results are expected to clarify findings in the literature of associations between ambient particulate air pollution (PM10 and PM2.5) and severe cardiovascular outcomes, including mortality and hospital admissions.

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- **Project Title: VEGF TRANSFER TO PROMOTE ANGIOGENESIS IN ADVANCED CHF**

  Principal Investigator & Institution: Mccarthy, Patrick M.; St. Elizabeth's Medical Center of Boston 736 Cambridge St Boston, MA 02135

  Timing: Fiscal Year 2001; Project Start 30-SEP-2001; Project End 31-AUG-2006

  Summary: The clinical investigations outlined in this Project are designed to test the hypothesis that direct intramyocardial injections of naked DNA encoding for vascular endothelial growth factor (phVEGF165) in patients with advanced heart failure is safely tolerated and may in some patients lead to improvement in their clinical status. The clinical trials that we have proposed incorporate a strategy that is designed to address patients in whom all medial measures to treat advanced congestive heart failure (CHF) have failed, leaving these patients in need of cardiac transplantation. Owing to the mismatch that currently exists between the number of patients in need of cardiac transplantation and the number of available donors, implantation of a left ventricular assist device (LVAD) Is often required for patients as a so- called "bridge" to transplantation. It is this population of patients-those undergoing LVAD implantation for advanced heart failure-that re intended to be addressed in the current Proposal. For the purpose of our clinical studies, these patients have been divided into two large subgroups, based on associated evidence on extramural coronary artery disease (CAD). Accordingly, the specific aims of this Proposal are as follows: 1. Specific Aim #1: To evaluate the safety and impact of phVEGF/165 gene transfer on LV function in patients with CHF due to coronary artery disease. 2. Specific Aim #2: To evaluate the safety and impact of phVEGF165 gene transfer on LV function in patients with CHF due to idiopathic dilated cardiomyopathy, excluding patients with significant narrowing of the extramural coronary arteries of primary valvular **heart disease.** 3. Sp4ecific Aim #3: To evaluate the efficacy of phVEGF gene transfer to allow for LVAD bridge-to-recovery (BTR) as an alternative to transplantation.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: WOMEN'S HEART PROJECT**

  Principal Investigator & Institution: Lipner, Robyn; Jacobs Institute of Women's Health 409 12Th St, Sw Washington, DC 20024

  Timing: Fiscal Year 2002; Project Start 30-SEP-2002; Project End 29-SEP-2003

  Summary: The overall goal of the "Women and Heart Disease-Putting Prevention into Primary Care" conference is to increase awareness of coronary **heart disease** (CHD) in women and to improve the health services that women receive for prevention of **heart disease** (CHD) in women and to improve the health services that women receive for prevention of **heart disease.** Given the high number of women at risk, the lack of awareness among both women and their providers, and the existence of a number of proven strategies to reduce the risks of developing CHD, the specific focus of the conference will be on primary and secondary prevention in the primary care setting. The conference will take place on Wednesday, December 4, 2002, from 8:00 am to 4:15 PM and Thursday, December 5th, 2002 from 8:30 am to 3:30 pm. The first day of the

conference is intended to follow a format of defining the problem of women and **heart disease** and updating knowledge of current prevention practices and barriers to implementation for improving primary and secondary prevention of **heart disease** in women in women in the primary care setting. A multi-disciplinary work group meeting on day two of the conference will synthesis the information presented at the conference and make recommendations for improving health services for women for the primary and secondary prevention of **heart disease.** Recommendations would address four main areas: clinical practice, research, payers, and advocacy/policy. The recommendations complied by the working group will be summarized into a final report. The report will include recommendations for improving clinical practice and will outline agendas for research and policy to increase the number of women who receive effective and appropriate preventing services (i.e. counseling and screening for known risk factors) and to improve the quality of the services women receive for the prevention of **heart disease.** The final report will be distributed to a wide audiences including conference participants, health services researchers, health care professionals, women, policy makers, and large insurers.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: WOMENS POOLING PROJECT**

  Principal Investigator & Institution: Mosca, Lori J. Associate Professor of Medicine; Medicine; Columbia University Health Sciences New York, NY 10032

  Timing: Fiscal Year 2001; Project Start 01-SEP-1997; Project End 31-AUG-2002

  Summary: (Adapted from applicant's abstract) Dr. Lori Mosca is a Junior faculty member in the Division of Cardiology at the University of Michigan who recently completed an NRSA training program in Preventive Cardiology and received her Ph.D. in Epidemiology from Columbia University in May, 1996. Over the past two years she has been actively involved in the initiation of The Women's Pooling Project, which was established to address the critical need for information about cardiovascular risk factors and disease in women. The goal of the WPP is to combine data collected over the past 40 years from long term, community-based studies in the United States, for the purpose of examining health issues in women that cannot be adequately addressed in a single study. The WPP is a historical prospective study which has well characterized cohorts with nearly 100% follow-up, large numbers and ethnic diversity. Cardiovascular risk in women will be examined by pooling data on over 12,000 women aged 30 - 79 years from the Charleston Heart Study, the Evan's County Study, the Framingham Heart Study, the Rancho Bernardo Study, the San Antonio Heart Study, and the Tecumseh Community Health Study. The WPP includes risk factors such as age, blood pressure, cholesterol, diabetes, body mass index, smoking pattern, educational level, and electrographic findings. An NHLBI sponsored pilot study was conducted that demonstrated pooling data from the above cohorts was feasible. Preliminary results suggest there are striking differences in the burden of cardiovascular risk factors among ethnic subgroups in the WPP. The prevalence of traditional cardiovascular disease (CVD) risk factors and their relation to mortality due to all causes, CVD and coronary **heart disease** (CHD) will be determined overall and by ethnic group. The proposed study will also evaluate the corresponding male data from each cohort and will examine gender differences in the burden of cardiovascular risk factors on mortality due to all causes, CVD and CHD. This study will generate timely data regarding cardiovascular risk in women and how it differs from men that may be useful for developing appropriate preventive strategies for diverse populations. Dr. Mosca will conduct the proposed research under the guidance of Dr. Millicent Higgins, an internationally recognized Cardiovascular Epidemiologist.

The goal of this award will be to develop Dr. Mosca into an independent researcher so that she will be able to make contributions to the field of Cardiovascular Epidemiology, specifically in the area of Women's Health. (End of Abstract)

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: WOMEN'S PRODROMAL AND ACUTE SYMPTOMS OF MYOCARDIAL INFAR**

  Principal Investigator & Institution: Mcsweeney, Jean C. None; University of Arkansas Med Scis Ltl Rock 4301 W Markham St Little Rock, AR 72205

  Timing: Fiscal Year 2001; Project Start 15-SEP-1999; Project End 31-AUG-2003

  Summary: Despite decades of research, cardiovascular disease kills more than half a million women each year, a greater number than the subsequent 16 causes of death combined. After an acute myocardial infarction (MI), women have a 16% mortality rate compared with 11% for men. Therefore, further investigation of acute MI in women is essential. Research investigating delay in seeking treatment associated with MI consistently reports women delay seeking treatment because they do not recognize symptoms of MI since their symptoms differ from expectations. Since delay in seeking treatment is associated with less effective and fewer treatment options, longer hospitalizations, and higher mortality and morbidity rates, women's symptomatology must be delineated. There is a growing consensus among clinicians and researchers that women with MI experience different symptoms than men do. However, few studies have specifically focused on women's symptoms of MI and **heart disease** or addressed prodromal symptoms, those symptoms that come and go prior to and change after the MI. To fill this void in research on women with MI and **heart disease,** the objective of this proposal is to delineate prodromal and acute symptoms of MI. White, black, and Hispanic women will be targeted since they delay seeking treatment longer than other groups, are more likely to have cardiac risk factors, and have higher mortality rates after MI. The researchers will conduct a telephone survey of 1540 women from 8 medical centers. To ensure representation of ethnic minorities, quotas will be set at a minimum of 186 black and 171 Hispanic women. The women will be queried about their prodromal and acute symptoms of MI and cardiovascular risk status four to six months after their MI. This allows them time to identify prodromal symptoms of their MI. The data will be analyzed to compare prodromal and acute symptoms of white, black, and Hispanic women when contributing for cardiovascular risk status. Results of this study will help us to more fully describe prodromal and acute symptomatology in women with **heart disease** and MI.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: YOUTH TAKE HEART (PHASE I & II)**

  Principal Investigator & Institution: Ratner, Buddy D. Distinguished Profess of Bioengineering; Bioengineering; University of Washington Seattle, WA 98195

  Timing: Fiscal Year 2003; Project Start 30-SEP-2003; Project End 29-SEP-2008

  Summary: (provided by applicant): As atherosclerosis, obesity, and sedentary habits are known to be formed in childhood and adolescence, cardiovascular education among our youth today could have a major impact on reducing the incidence and prevalence of **heart disease** in the future. The Youth Take Heart (YTH) program is a collaborative project between the University of Washington Engineered Biomaterials Engineering Research Center (UWEB ERC), The Hope Heart Institute (HHI), and the Washington Mathematics, Engineering, Science Achievement (MESA) program. Phase I will focus on

four main components: 1. Development/testing of an interactive Internet and CD-ROM program titled "Guy Simplant: The Heart" (the character "Guy Simplant" was created by UWEB to introduce biomaterials to middle school students) to teach about the anatomy and physiology of the heart, **heart disease** prevention, and newly emerging bioengineering solutions for repairing or replacing the heart; 2. Development of a corresponding cardiovascular health laboratory kit and curriculum module by leading health/science teachers and scientists; 3. Development of a YTH public lecture and parent training series and; 4. Creation of a YTH newsletter and brochures. Phase II will focus on the dissemination of the Guy Simplant game, curriculum, laboratory kits, newsletters, brochures, and implementation of the lecture series using a collaborative model of marketing and dissemination. We will primarily target approximately 5,000 MESA students (80% of whom are under-represented minorities and women). Our ultimate goal is to reach all youth in WA and the nation specifically targeting under-served populations who traditionally lack information on careers in math, engineering, medicine and science, and who are also at higher risk for cardiovascular disease. The project will fulfill WA State's Essential Academic Learning Requirements in Science and Health 1-3 and provide tools for understanding scientific concepts and principles. The project uses the federal government's Healthy People 2010 initiative guidelines, which challenge individuals and communities to take steps to ensure good health and a long life are enjoyed by all. Youth Take Heart will help enhance the cardiovascular health and quality of life for numerous students through the teaching of medical management, prevention, and control of risk factors for **heart disease.**

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

E-Journals: PubMed Central[3]

PubMed Central (PMC) is a digital archive of life sciences journal literature developed and managed by the National Center for Biotechnology Information (NCBI) at the U.S. National Library of Medicine (NLM).[4] Access to this growing archive of e-journals is free and unrestricted.[5] To search, go to **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Pmc**, and type "heart disease" (or synonyms) into the search box. This search gives you access to full-text articles. The following is a sample of items found for heart disease in the PubMed Central database:

- **"Unwarranted survivals" and "anomalous deaths" from coronary heart disease: prospective survey of general population.** by McConnachie A, Hunt K, Emslie C, Hart C, Watt G. 2001 Dec 22;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=61054
- **Analysis of predicted coronary heart disease risk in England based on Framingham study risk appraisal models published in 1991 and 2000.** by Nanchahal K, Duncan JR, Durrington PN, Jackson RT. 2002 Jul 27;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=117447

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[3] Adapted from the National Library of Medicine: **http://www.pubmedcentral.nih.gov/about/intro.html**.

[4] With PubMed Central, NCBI is taking the lead in preservation and maintenance of open access to electronic literature, just as NLM has done for decades with printed biomedical literature. PubMed Central aims to become a world-class library of the digital age.

[5] The value of PubMed Central, in addition to its role as an archive, lies in the availability of data from diverse sources stored in a common format in a single repository. Many journals already have online publishing operations, and there is a growing tendency to publish material online only, to the exclusion of print.

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The National Library of Medicine: PubMed

One of the quickest and most comprehensive ways to find academic studies in both English and other languages is to use PubMed, maintained by the National Library of Medicine.[6] The advantage of PubMed over previously mentioned sources is that it covers a greater number of domestic and foreign references. It is also free to use. If the publisher has a Web site that offers full text of its journals, PubMed will provide links to that site, as well as to sites offering other related data. User registration, a subscription fee, or some other type of fee may be required to access the full text of articles in some journals.

To generate your own bibliography of studies dealing with heart disease, simply go to the PubMed Web site at **http://www.ncbi.nlm.nih.gov/pubmed**. Type "heart disease" (or synonyms) into the search box, and click "Go." The following is the type of output you can expect from PubMed for heart disease (hyperlinks lead to article summaries):

- **A 21-year-old woman with complex congenital heart disease and cardiac arrest.**
  Author(s): Listernick R.
  Source: Pediatric Annals. 2002 October; 31(10): 615-9.
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- **A case of congenital midline cervical cleft with congenital heart disease.**
  Author(s): Hirokawa S, Uotani H, Okami H, Tsukada K, Futatani T, Hashimoto I.
  Source: Journal of Pediatric Surgery. 2003 July; 38(7): 1099-101.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12861550&dopt=Abstract

[6] PubMed was developed by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine (NLM) at the National Institutes of Health (NIH). The PubMed database was developed in conjunction with publishers of biomedical literature as a search tool for accessing literature citations and linking to full-text journal articles at Web sites of participating publishers. Publishers that participate in PubMed supply NLM with their citations electronically prior to or at the time of publication.

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- **Ask the doctor. I'm a 51-year-old man with no risk factors for heart disease other than being male. My blood pressure and cholesterol levels are fine. I exercise, and my diet is pretty good. I've never smoked. Yet I recently underwent a triple bypass for sudden chest pain. What could have caused this blockage and can I prevent future problems?**
  Author(s): Lee TH.
  Source: Harvard Heart Letter : from Harvard Medical School. 2002 December; 13(4): 8.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12499159&dopt=Abstract

- **Ask the doctor. In early July, the New York Times Magazine published an article that had good things to say about the Atkins high-protein diet. It also claimed that low-fat diets might be harmful. This shocked many of us who have been following a low-fat diet to reduce our risk from heart disease. Have we been barking up the wrong tree all these years?**
  Author(s): Lee TH.
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  Author(s): Wannamethee SG, Shaper AG.
  Source: Heart (British Cardiac Society). 2002 January; 87(1): 32-6.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11751661&dopt=Abstract

- **Tea consumption and the prevalence of coronary heart disease in Saudi adults: results from a Saudi national study.**
  Author(s): Hakim IA, Alsaif MA, Alduwaihy M, Al-Rubeaan K, Al-Nuaim AR, Al-Attas OS.
  Source: Preventive Medicine. 2003 January; 36(1): 64-70.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12473426&dopt=Abstract

- **Temporary endoepicardial atrioventricular sequential pacing for complete heart block following complex surgery for congenital heart disease.**
  Author(s): Pandurangi UM, Ruth PJ, Toal SC, Kulkarni S, Murthy KS, Cherian KM.
  Source: Indian Heart J. 2003 January-February; 55(1): 68-70.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12760592&dopt=Abstract

- **Test characteristics of a level I or II fetal ultrasound in detecting structural heart disease.**
  Author(s): Skeels M, Taylor D, Park J, Parrish M, Choy M.
  Source: Pediatric Cardiology. 2002 November-December; 23(6): 594-7.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12530490&dopt=Abstract

- **Testosterone for secondary prevention in men with ischaemic heart disease?**
  Author(s): Malkin CJ, Pugh PJ, Jones TH, Channer KS.
  Source: Qjm : Monthly Journal of the Association of Physicians. 2003 July; 96(7): 521-9. Review.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12881595&dopt=Abstract

- **The applicability of the Framingham coronary heart disease prediction function to black and minority ethnic groups in the UK.**
  Author(s): Quirke TP, Gill PS, Mant JW, Allan TF.
  Source: Heart (British Cardiac Society). 2003 July; 89(7): 785-6.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12807859&dopt=Abstract

- **The association between body height and coronary heart disease among Finnish twins and singletons.**
  Author(s): Silventoinen K, Kaprio J, Koskenvuo M, Lahelma E.
  Source: International Journal of Epidemiology. 2003 February; 32(1): 78-82.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12690014&dopt=Abstract

- **The big mush: psychometric measures are confounded and non-independent in their association with age at initial diagnosis of Ischaemic Coronary Heart Disease.**
  Author(s): Ketterer MW, Denollet J, Goldberg AD, McCullough PA, John S, Farha AJ, Clark V, Keteyian S, Chapp J, Thayer B, Deveshwar S.
  Source: Journal of Cardiovascular Risk. 2002 February; 9(1): 41-8.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11984216&dopt=Abstract

CHAPTER 2. NUTRITION AND HEART DISEASE

Overview

In this chapter, we will show you how to find studies dedicated specifically to nutrition and heart disease.

Finding Nutrition Studies on Heart Disease

The National Institutes of Health's Office of Dietary Supplements (ODS) offers a searchable bibliographic database called the IBIDS (International Bibliographic Information on Dietary Supplements; National Institutes of Health, Building 31, Room 1B29, 31 Center Drive, MSC 2086, Bethesda, Maryland 20892-2086, Tel: 301-435-2920, Fax: 301-480-1845, E-mail: ods@nih.gov). The IBIDS contains over 460,000 scientific citations and summaries about dietary supplements and nutrition as well as references to published international, scientific literature on dietary supplements such as vitamins, minerals, and botanicals.[7] The IBIDS includes references and citations to both human and animal research studies.

As a service of the ODS, access to the IBIDS database is available free of charge at the following Web address: **http://ods.od.nih.gov/databases/ibids.html**. After entering the search area, you have three choices: (1) IBIDS Consumer Database, (2) Full IBIDS Database, or (3) Peer Reviewed Citations Only.

Now that you have selected a database, click on the "Advanced" tab. An advanced search allows you to retrieve up to 100 fully explained references in a comprehensive format. Type "heart disease" (or synonyms) into the search box, and click "Go." To narrow the search, you can also select the "Title" field.

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[7] Adapted from **http://ods.od.nih.gov**. IBIDS is produced by the Office of Dietary Supplements (ODS) at the National Institutes of Health to assist the public, healthcare providers, educators, and researchers in locating credible, scientific information on dietary supplements. IBIDS was developed and will be maintained through an interagency partnership with the Food and Nutrition Information Center of the National Agricultural Library, U.S. Department of Agriculture.

The following is a typical result when searching for recently indexed consumer information on heart disease:

- **Ironing out heart disease: deplete or not deplete?**
  Author(s): Purdue University.
  Source: Proulx, W.R. Weaver, C.M. Nutrition-today (USA). (February 1995). volume 30(1) page 16-23. cardiovascular diseases aetiology iron nutrition physiology nutritional requirements nutrient excesses nutrient deficiencies nutrients blood composition contraceptives biological rhythms aging risk 0029-666X
- **Pungent, powerful garlic may help fight infection, heart disease.**
  Source: Golub, C. Environmental-nutrition (USA). (December 1995). volume 18(12) page 1, 6. garlic heart diseases pathogenesis disease control medicinal properties neoplasms food intake 0893-4452
- **Therapeutic nutrition: an alternative approach to coronary heart disease management.**
  Author(s): North Colorado Medical Center, Greeley, CO.
  Source: Nelson, K. Nutrition-today (USA). (June 1995). volume 30(3) page 114-122. fats usa diet cardiovascular diseases aetiology disease control therapeutic diets health nutrition physiology human nutrition research medical sciences cultural development sociology 0029-666X

Additional consumer oriented references include:

- **3. Milk and heart disease.**
  Source: Ashwell, M. B-N-F-Nutr-Bull-Br-Nutr-Found. London : The Foundation. January 1991. volume 16 page 6-7. 0141-9684
- **A fish oil story: omega-3's return to fight heart disease, cancer.**
  Source: Hrabak, D. Environmental-nutrition (USA). (July 1994). volume 17(7) page 1, 4. fish oils fatty acids circulatory disorders neoplasms disease control nutrients 0893-4452
- **Alcohol and coronary heart disease.**
  Source: Bingham, S. B-N-F-Nutr-Bull-Br-Nutr-Found. London : The Foundation. January 1992. volume 17 page 10-13. 0141-9684
- **Ask the doctor. I'm a 51-year-old man with no risk factors for heart disease other than being male. My blood pressure and cholesterol levels are fine. I exercise, and my diet is pretty good. I've never smoked. Yet I recently underwent a triple bypass for sudden chest pain. What could have caused this blockage and can I prevent future problems?**
  Source: Lee, T H Harv-Heart-Lett. 2002 December; 13(4): 8 1051-5313
- **Ask the doctor. In early July, the New York Times Magazine published an article that had good things to say about the Atkins high-protein diet. It also claimed that low-fat diets might be harmful. This shocked many of us who have been following a low-fat diet to reduce our risk from heart disease. Have we been barking up the wrong tree all these years?**
  Source: Lee, T H Harv-Heart-Lett. 2002 November; 13(3): 8 1051-5313
- **B vitamins and heart disease.**
  Source: Anonymous Harv-Health-Lett. 1998 October; 23(12): 8 1052-1577
- **B vitamins may cut heart disease risk.**
  Source: Anonymous Harv-Health-Lett. 1998 April; 23(6): 8 1052-1577
- **By the way, doctor. I recently read that eating cereal will reduce my risk of heart disease. What is it in the cereal that produces this beneficial effect?**
  Source: Nicholson, C R Harv-Womens-Health-Watch. 1999 September; 7(1): 8 1070-910X

- **By the way, doctor.My father died of a heart attack at age 72, but he smoked and ate poorly. Now that I'm in my 50s, I'm starting to wonder if I fall into a high-risk category for heart disease.**
  Source: Lee, T H Harv-Health-Lett. 2001 November; 27(1): 8 1052-1577
- **Coping with cholesterol. The waxy buildup in our blood vessels is a leading cause of fatal heart disease. Do dietary supplements add anything to the arsenal against it?**
  Source: Kalb, C Newsweek. 2000 June 19; 135(25): 73, 75 0028-9604
- **Coronary heart disease -- "Doing the wrong things.".**
  Source: Nutr-Today. Baltimore, Md. : Williams & Wilkins. July/August 1985. volume 20 (4) page 12-14. 0029-666X
- **Diet, serum lipids and coronary heart disease in women.**
  Author(s): University of Texas, Southwestern Medical Center, Dallas, TX
  Source: Denke, M.A. Food-and-nutrition-news (USA). (Nov-December 1992). volume 64(5) page 31-33. diet circulatory disorders lipaemia women 0015-6310
- **Estrogen for heart disease: risk or benefit?**
  Source: Anonymous Harv-Womens-Health-Watch. 2000 June; 7(10): 1-2 1070-910X
- **Food, heart disease and stroke.**
  Source: Marlett, J.A. Cheung, T.F. BNF-nutr-bull. London : The Foundation,. January 1995. volume 20 (73) page 34-41. 0141-9684
- **Halt heart disease or beat breast cancer? A woman's quandary.**
  Source: Golub, C. Environmental-nutrition (USA). (May 1998). volume 21(5) page 1, 6. diet fats alcoholic beverages disease control risk 0893-4452
- **Health claim: for foods that could lower heart disease risk.**
  Source: House, S. FDA-consum. Rockville, Md. : Food and Drug Administration, Department of Health & Human Services. Nov/December 2000. volume 34 (6) page 11. 0362-1332
- **Heart disease handbook. 1. Rating your risks and taking action.**
  Source: Hudnall, M. Environmental-nutrition (USA). (February 1997). volume 20(2) page 1, 4. heart diseases risk diet disease control health services health 0893-4452
- **Heart disease handbook. 2. Deciphering blood cholesterol.**
  Source: Forman, A. Environmental-nutrition (USA). (March 1997). volume 20(3) page 1, 4. heart diseases cholesterol lipoproteins diet drug therapy 0893-4452
- **Heart Disease Handbook. 3. Triglycerides turn troublesome.**
  Source: Hudnall, M. Environmental-nutrition (USA). (April 1997). volume 20(4) page 1, 4. heart diseases lipoproteins triglycerides diet lipid metabolism 0893-4452
- **Heart Disease Handbook. 4. Eating to your heart's content.**
  Source: Ternus, M. Environmental-nutrition (USA). (May 1997). volume 20(5) page 1, 4. heart diseases dietary fibres diet fats fatty acids vitamin e 0893-4452
- **Heart disease risk and prevention: not just cholesterol anymore.**
  Source: Klausner, A. Environ-nutr. New York : Environmental Nutrition, Inc.,. August 1999. volume 22 (8) page 1, 4. 0893-4452
- **Heart disease: a cardiologist's POV.**
  Source: Gottlieb, S.H. Diabetes-forecast. Alexandria, Va. : American Diabetes Association Inc. Sept 2001. volume 54 (9) page 38-40. 0095-8301

- **Helping in preventing heart disease.**
  Source: Kurtzweil, P. FDA-consumer (USA). (December 1994). volume 28(10) page 19-24. nutrition labelling human nutrition heart diseases disease control 0362-1332
- **Herbs and spices may be barrier against cancer, heart disease.**
  Source: Berkowitz, K.F. Environmental-nutrition (USA). (June 1993). volume 16(6) page 1, 4. culinary herbs spices disease control 0893-4452
- **Homocysteine & heart disease.**
  Source: Anonymous Health-News. 2000 October; 6(10): 4 1081-5880
- **How soy fares in fight against cancer, heart disease, osteoporosis.**
  Source: Walsh, J. Environ-nutr. New York : Environmental Nutrition, Inc.,. May 2002. volume 25 (5) page 1, 6. 0893-4452
- **HRT vs. heart disease: down but not out.**
  Source: Anonymous Harv-Womens-Health-Watch. 2001 September; 9(1): 1-2 1070-910X
- **I'm a 44-year-old former smoker. I have just been diagnosed with colitis, and I've heard that smoking can help my condition. There is no cancer or heart disease in my family. Should I start smoking again?**
  Source: Simon, H B Harv-Mens-Health-Watch. 1998 September; 3(2): 8 1089-1102
- **In one HealthNews article, you wrote that vitamin C can lower your blood pressure, and in another you said that it increases the intima-media thickness (IMT), thereby increasing the risk of heart disease. Should I or shouldn't I take extra vitamin C?**
  Author(s): Massachusetts Veterans Epidemiology Research and Information Center, USA.
  Source: Gaziano, J M Health-News. 2001 March; 7(3): 10 1081-5880
- **Is total serum cholesterol level a reliable indicator of latent risk of coronary heart disease?**
  Source: Nutrition-today (USA). (Jan-February 1985). volume 20(1) page 11-12. heart diseases cholesterol blood composition risk 0029-666X
- **Low-fat diets, triglycerides and coronary heart disease risk.**
  Source: Nestle, M. BNF-nutr-bull. London : The British Nutrition Foundation. March 2000. volume 25 (1) page 49-53. 0141-9684
- **Magnesium and coronary heart disease.**
  Source: Cwiertka, K. BNF-nutr-bull. London : The British Nutrition Foundation. Sept 1996. volume 21 page 190-195. 0141-9684
- **Mercury and your heart. Does mercury in fish increase your risk of heart disease?**
  Source: Ornato, J P Health-News. 2003 January; 9(1): 3 1081-5880
- **NIH panel embraces changes in diet to lower heart disease risks.**
  Source: Nutr-Action-Health-Lett. Washington, D.C. : Center for Science in the Public Interest. Jan/February 1985. volume 12 (1) page 3. ill. 0199-5510
- **Therapeutic nutrition--an alternative approach to coronary heart disease management.**
  Source: Blair, S.N. Nutr-today. Baltimore, Md. : Williams & Wilkins. June 1995. volume 30 (3) page 114-122. 0029-666X
- **Trio of vitamins are recruited in the fight against heart disease.**
  Source: Berkowitz, K.F. Environmental-nutrition (USA). (September 1992). volume 15(9) page 1, 4. vitamins circulatory disorders 0893-4452

- **Vitamin C-rich foods help fend off heart disease, cancer--even scurvy.**
  Source: Walsh, J. Environ-nutr. New York : Environmental Nutrition, Inc.,. October 2000. volume 23 (10) page 1, 4. 0893-4452
- **Vitamin E and coronary heart disease.**
  Source: Arens, U. BNF-nutr-bull. London : The Foundation,. Sept 1993. volume 18 (69) page 157-159. 0141-9684
- **Vitamin E seen lowering heart disease risk.**
  Source: Wash-Post. [Washington, D.C.] : The Washington Post Co. May 20, 1993. page A3. 0190-8286
- **Vitamin E shows no benefit in heart disease.**
  Source: Anonymous Health-News. 2000 February; 6(2): 6 1081-5880

The following information is typical of that found when using the "Full IBIDS Database" to search for "heart disease" (or a synonym):

- **A study of hormone replacement therapy in postmenopausal women with ischaemic heart disease: the Papworth HRT atherosclerosis study.**
  Author(s): Papworth Hospital, Cambridge, UK.
  Source: Clarke, S C Kelleher, J Lloyd Jones, H Slack, M Schofiel, P M BJOG. 2002 September; 109(9): 1056-62 1470-0328
- **Alcohol, heart disease, and mortality: a review.**
  Author(s): Division of Cardiology, University of Maryland School of Medicine, Baltimore, MD, USA.
  Source: Vogel, R A Rev-Cardiovasc-Med. 2002 Winter; 3(1): 7-13 1530-6550
- **An update: vitamin E supplementation and heart disease.**
  Author(s): Jean Mayer USDA Human Nutrition Research Center on Aging, Tufts University Boston, Massachusetts, USA. blumberg@hnrc.tufts.edu
  Source: Blumberg, J B Nutr-Clin-Care. 2002 Mar-April; 5(2): 50-5 1096-6781
- **Ask the doctors. I was always told that low-fat diets are most appropriate for patients with heart disease. Now I'm hearing that high-fat diets are the way to go. What should I believe?**
  Source: Anonymous Heart-Advis. 2003 January; 6(1): 8 1523-9004
- **Atrial fibrillation threshold predicted long-term efficacy of pharmacological treatment of patients without structural heart disease.**
  Author(s): Kyoto Prefectural University of Medicine, Second Department of Medicine, Kawaramachi Hirokoji, Japan. sirayama@koto.kpu-m.ac.jp
  Source: Shirayama, T Shiraishi, H Yoshida, S Matoba, Y Imai, H Nakagawa, M Europace. 2002 October; 4(4): 383-9 1099-5129
- **Beraprost sodium for pulmonary hypertension with congenital heart disease.**
  Author(s): Department of Pediatrics, Yamagata University School of Medicine, Yamagata, Japan. hrsuzuki@med.id.yamagata-u.ac.jp
  Source: Suzuki, H Sato, S Tanabe, S Hayasaka, K Pediatr-Int. 2002 October; 44(5): 528-9 1328-8067
- **Diet medications and valvular heart disease: the current evidence.**
  Author(s): University of Medicine and Dentistry of New Jersey-New Jersey Medical School, Newark, USA. barasaff@cs.com
  Source: Barasch, A Safford, M M Spec-Care-Dentist. 2002 May-June; 22(3): 108-14 0275-1879

- **Dietary fibre and coronary heart disease.**
  Author(s): Department of Biochemistry, Mymensingh Medical College.
  Source: Mia, M A Siddiqui, M N Haque, M S Islam, M N Rukunzzaman, M Deb, K Mymensingh-Med-J. 2002 July; 11(2): 133-5 1022-4742
- **Dioxin exposure is an environmental risk factor for ischemic heart disease.**
  Author(s): Center for Environmental Genetics and Department of Environmental Health, University of Cincinnati Medical Center, Cincinnati, OH 45267-0056, USA.
  Source: Dalton, T P Kerzee, J K Wang, B Miller, M Dieter, M Z Lorenz, J N Shertzer, H G Nerbert, D W Puga, A Cardiovasc-Toxicol. 2001; 1(4): 285-98 1530-7905
- **Dose-dependent effects of folic acid on plasma homocysteine in a randomized trial conducted among 723 individuals with coronary heart disease.**
  Source: Neal, B MacMahon, S Ohkubo, T Tonkin, A Wilcken, D Eur-Heart-J. 2002 October; 23(19): 1509-15 0195-668X
- **Fibrotic valvular heart disease subsequent to bromocriptine treatment.**
  Author(s): Service de Medecine Interne, the Service de Cardiologie B, CHU Timone, Marseille Cedex 5, France.
  Source: Serratrice, J Disdier, P Habib, G Viallet, F Weiller, P J Cardiol-Revolume 2002 Nov-December; 10(6): 334-6 1061-5377
- **Frequent nut intake and risk of death from coronary heart disease and all causes in postmenopausal women: the Iowa Women's Health Study.**
  Author(s): Division of Epidemiology, School of Public Health, University of Minnesota, 1300 South Second Street, Suite 300, Minneapolis, MN 55454-1015, USA.
  Source: Ellsworth, J L Kushi, L H Folsom, A R Nutr-Metab-Cardiovasc-Dis. 2001 December; 11(6): 372-7 0939-4753
- **Growth hormone therapy in Noonan's syndrome: non-cardiomyopathic congenital heart disease does not adversely affect growth improvement.**
  Author(s): Child Life & Health, University of Edinburgh, London, UK. donald.brown@luht.scot.nhs.uk
  Source: Brown, D C Macfarlane, C E McKenna, W J Patton, M A Dunger, D B Savage, M O Kelnar, C J J-Pediatr-Endocrinol-Metab. 2002 June; 15(6): 851-2
- **Heart disease and its related risk factors in Asian Indians.**
  Author(s): Department of Internal Medicine, St. Mary's Health Center, St. Louis, Missouri 63117, USA. cuppaluri@aol.com
  Source: Uppaluri, Chitra R Ethn-Dis. 2002 Winter; 12(1): 45-53 1049-510X
- **Histamine-dependent changes in free radical processes during coronary heart disease.**
  Author(s): Department of Internal Diseases No 2, Therapeutic Faculty, I. M. Sechenov Moscow Medical Academy.
  Source: Stremoukhov, A A Bull-Exp-Biol-Med. 2001 December; 132(6): 1157-9 0007-4888
- **Hostility, the metabolic syndrome, and incident coronary heart disease.**
  Author(s): Department of Psychiatry and Human Behavior, Brown Medical School, Centers for Behavioral and Preventive Medicine, The Miriam Hospital, Providence, Rhode Island 02903, USA. raymond_niaura@brown.edu
  Source: Niaura, R Todaro, J F Stroud, L Spiro, A 3rd Ward, K D Weiss, S Health-Psychol. 2002 November; 21(6): 588-93 0278-6133
- **How soy fares in fight against cancer, heart disease, osteoporosis.**
  Source: Walsh, J. Environ-nutr. New York : Environmental Nutrition, Inc.,. May 2002. volume 25 (5) page 1, 6. 0893-4452

- **I read recently that patients with heart disease are more likely to develop dementia. How can I prevent dementia as I age?**
  Author(s): Coronary Intensive Care Unit, Cleveland Clinic, USA.
  Source: Francis, G S Heart-Advis. 2002 September; 5(9): 8 1523-9004
- **Ischaemic heart disease, Type 1 diabetes, and cow milk A1 beta-casein.**
  Author(s): Health New Zealand, Auckland, New Zealand. laugesen@healthnz.co.nz
  Source: Laugesen, M Elliott, R N-Z-Med-J. 2003 January 24; 116(1168): U295 1175-8716
- **Lycopene, atherosclerosis, and coronary heart disease.**
  Author(s): Research Institute of Public Health, University of Kuopio, Kuopio, Finland 70211. Tiina.rissanen@uku.fi
  Source: Rissanen, T Voutilainen, S Nyyssonen, K Salonen, J T Exp-Biol-Med-(Maywood). 2002 November; 227(10): 900-7 1535-3702
- **Lycopene, tomatoes, and the prevention of coronary heart disease.**
  Author(s): Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada M5S 3E2. v.rao@utoronto.ca
  Source: Rao, A V Exp-Biol-Med-(Maywood). 2002 November; 227(10): 908-13 1535-3702
- **Meaning of low-density lipoprotein-apheresis for hypercholesterolemic patients at high risk for recurrence of coronary heart disease.**
  Author(s): Shin-Koga Hospital, Kurume, Japan. akikokhp@kureme.ktarn.or.jp
  Source: Koga, N Ther-Apher. 2002 October; 6(5): 372-80 1091-6660
- **Mediterranean diet and prevention of coronary heart disease.**
  Author(s): Department of Geriatrics and Metabolic Diseases, Cardiovascular Research Center, Second University of Naples, Italy. katherine.esposito@unina2.it
  Source: Esposito, K Giugliano, D J-Endocrinol-Invest. 2002 March; 25(3): 296-9 0391-4097
- **Mercury and the risk of coronary heart disease in men.**
  Author(s): Department of Nutrition, Harvard School of Public Health, Boston, USA.
  Source: Yoshizawa, K Rimm, E B Morris, J S Spate, V L Hsieh, C C Spiegelman, D Stampfer, M J Willett, W C N-Engl-J-Med. 2002 November 28; 347(22): 1755-60 1533-4406
- **Mercury and your heart. Does mercury in fish increase your risk of heart disease?**
  Source: Ornato, J P Health-News. 2003 January; 9(1): 3 1081-5880
- **Migraine and coronary heart disease in women and men.**
  Author(s): Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02215, USA.
  Source: Cook, N R Bensenor, I M Lotufo, P A Lee, I M Skerrett, P J Chown, M J Ajani, U A Manson, J E Buring, J E Headache. 2002 September; 42(8): 715-27 0017-8748
- **Myths and science of dietary fat and coronary heart disease.**
  Source: Higgs, J Nurs-Times. 2002 August 20; 98(34): 49-52 0954-7762
- **Oxidized lipids as mediators of coronary heart disease.**
  Author(s): Department of Medicine, Cardiology, University of California Los Angeles, 90095-1679, USA. mnavab@mednet.ucla.edu
  Source: Navab, M Hama, S Y Ready, S T Ng, C J Van Lenten, B J Laks, H Fogelman, A M Curr-Opin-Lipidol. 2002 August; 13(4): 363-72 0957-9672
- **Recognizing heart disease. Helping women seek treatment faster.**
  Author(s): University of Michigan School of Nursing, USA.
  Source: Arslanian Engoren, Cynthia AWHONN-Lifelines. 2002 Apr-May; 6(2): 114-9, 122 1091-5923

- **Roles of drinking pattern and type of alcohol consumed in coronary heart disease in men.**
  Author(s): Division of General Medicine and Primary Care, Beth Israel Deaconess Medical Center, Boston, MA 02215, USA. kmukamal@caregroup.harvard.edu
  Source: Mukamal, K J Conigrave, K M Mittleman, M A Camargo, C A Jr Stampfer, M J Willett, W C Rimm, E B N-Engl-J-Med. 2003 January 9; 348(2): 109-18 1533-4406
- **Selenium supplementation decreases coxsackievirus heart disease during murine AIDS.**
  Author(s): Microbiology and Immunology, College of Medicine, University of Arizona, Tucson, AZ, 85724, USA.
  Source: Sepulveda, R T Zhang, J Watson, R R Cardiovasc-Toxicol. 2002; 2(1): 53-61 1530-7905
- **Socio-economic position and coronary heart disease risk factors in children and young people. Evidence from UK epidemiological studies.**
  Author(s): Epidemiology Unit, Department of Epidemiology and Population Health, London School of Hygiene & Tropical Medicine, Keppel Street, London WC1E 7HT, UK.
  Source: Batty, G D Leon, D A Eur-J-Public-Health. 2002 December; 12(4): 263-72 1101-1262
- **The paraoxonase gene family and coronary heart disease.**
  Author(s): University Department of Medicine, Manchester Royal Infirmary, UK. bmack@central.cmht.nwest.nhs.uk
  Source: Mackness, B Durrington, P N Mackness, M I Curr-Opin-Lipidol. 2002 August; 13(4): 357-62 0957-9672
- **Understanding the Mediterranean diet. Could this be the new "gold standard" for heart disease prevention?**
  Author(s): Corvallis Clinic, Corvallis, Oregon, USA.
  Source: Curtis, B M O'Keefe, J H Jr Postgrad-Med. 2002 August; 112(2): 35-8, 41-5 0032-5481
- **Vitamin C-rich foods help fend off heart disease, cancer--even scurvy.**
  Source: Walsh, J. Environ-nutr. New York : Environmental Nutrition, Inc.,. October 2000. volume 23 (10) page 1, 4. 0893-4452
- **Wine defences against bacteria, heart disease, and cancer.**
  Source: Paul, H W Clio-Med. 2001; 64: 269-304 0045-7183

Federal Resources on Nutrition

In addition to the IBIDS, the United States Department of Health and Human Services (HHS) and the United States Department of Agriculture (USDA) provide many sources of information on general nutrition and health. Recommended resources include:

- healthfinder®, HHS's gateway to health information, including diet and nutrition: **http://www.healthfinder.gov/scripts/SearchContext.asp?topic=238&page=0**
- The United States Department of Agriculture's Web site dedicated to nutrition information: **www.nutrition.gov**
- The Food and Drug Administration's Web site for federal food safety information: **www.foodsafety.gov**

- The National Action Plan on Overweight and Obesity sponsored by the United States Surgeon General: **http://www.surgeongeneral.gov/topics/obesity/**
- The Center for Food Safety and Applied Nutrition has an Internet site sponsored by the Food and Drug Administration and the Department of Health and Human Services: **http://vm.cfsan.fda.gov/**
- Center for Nutrition Policy and Promotion sponsored by the United States Department of Agriculture: **http://www.usda.gov/cnpp/**
- Food and Nutrition Information Center, National Agricultural Library sponsored by the United States Department of Agriculture: **http://www.nal.usda.gov/fnic/**
- Food and Nutrition Service sponsored by the United States Department of Agriculture: **http://www.fns.usda.gov/fns/**

Additional Web Resources

A number of additional Web sites offer encyclopedic information covering food and nutrition. The following is a representative sample:

- AOL: **http://search.aol.com/cat.adp?id=174&layer=&from=subcats**
- Family Village: **http://www.familyvillage.wisc.edu/med_nutrition.html**
- Google: **http://directory.google.com/Top/Health/Nutrition/**
- Healthnotes: **http://www.healthnotes.com/**
- Open Directory Project: **http://dmoz.org/Health/Nutrition/**
- Yahoo.com: **http://dir.yahoo.com/Health/Nutrition/**
- WebMD®Health: **http://my.webmd.com/nutrition**
- WholeHealthMD.com: **http://www.wholehealthmd.com/reflib/0,1529,00.html**

The following is a specific Web list relating to heart disease; please note that any particular subject below may indicate either a therapeutic use, or a contraindication (potential danger), and does not reflect an official recommendation:

- **Vitamins**

  Ascorbic Acid
  Source: Integrative Medicine Communications; www.drkoop.com

  Ascorbic Acid
  Alternative names: Vitamin C (Ascorbic Acid)
  Source: Integrative Medicine Communications; www.drkoop.com

  Folic Acid
  Source: Healthnotes, Inc. www.healthnotes.com

  Folic Acid
  Source: Integrative Medicine Communications; www.drkoop.com

Folic acid
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,887,00.html

Multiple Vitamin-Mineral Supplements
Source: Healthnotes, Inc. www.healthnotes.com

Provitamin A
Source: Integrative Medicine Communications; www.drkoop.com

Pyridoxine
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin B
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10067,00.html

Vitamin B complex
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,962,00.html

Vitamin B1
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin B12
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin B12
Source: Prima Communications, Inc.www.personalhealthzone.com

Vitamin B12 (Cobalamin)
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin B3
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin B6
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin B6
Source: Prima Communications, Inc.www.personalhealthzone.com

Vitamin B6 (Pyridoxine)
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin B9 (Folic Acid)
Alternative names: Folate, Folic Acid
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin C
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin C
Source: Prima Communications, Inc.www.personalhealthzone.com

Vitamin C
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,904,00.html

Vitamin C (Ascorbic Acid)
Alternative names: Ascorbic Acid
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin D
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin D
Alternative names: Calciferol, Calcitrol, Cholecalciferol, Erocalciferol
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin E
Source: Healthnotes, Inc. www.healthnotes.com

Vitamin E
Alternative names: Alpha-Tocopherol, Beta-Tocopherol, D-Alpha-Tocopherol, Delta-Tocopherol, Gamma-Tocopherol
Source: Integrative Medicine Communications; www.drkoop.com

Vitamin E
Source: Prima Communications, Inc.www.personalhealthzone.com

Vitamin E
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,906,00.html

- **Minerals**

Alpha-Tocopherol
Source: Integrative Medicine Communications; www.drkoop.com

Betaine Hydrochloride
Source: Healthnotes, Inc. www.healthnotes.com

Beta-Tocopherol
Source: Integrative Medicine Communications; www.drkoop.com

Calcium
Source: Healthnotes, Inc. www.healthnotes.com

Calcium/magnesium
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,937,00.html

Carnitine
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10012,00.html

Carnitine (L-Carnitine)
Source: Integrative Medicine Communications; www.drkoop.com

Chondroitin
Alternative names: chondroitin sulfate, sodium chondroitin sulfate
Source: Integrative Medicine Communications; www.drkoop.com

Chromium
Source: Integrative Medicine Communications; www.drkoop.com

Chromium
Source: Prima Communications, Inc.www.personalhealthzone.com

Chromium
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10018,00.html

Copper
Source: Healthnotes, Inc. www.healthnotes.com

Copper
Source: Prima Communications, Inc.www.personalhealthzone.com

Copper
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,886,00.html

Creatine
Source: Prima Communications, Inc.www.personalhealthzone.com

D-Alpha-Tocopherol
Source: Integrative Medicine Communications; www.drkoop.com

Delta-Tocopherol
Source: Integrative Medicine Communications; www.drkoop.com

Folate
Source: Integrative Medicine Communications; www.drkoop.com

Folate
Source: Prima Communications, Inc.www.personalhealthzone.com

Gamma-Tocopherol
Source: Integrative Medicine Communications; www.drkoop.com

HMG-CoA Reductase Inhibitors (Statins)
Source: Integrative Medicine Communications; www.drkoop.com

Iron
Source: Healthnotes, Inc. www.healthnotes.com

Iron
Alternative names: Ferrous Sulfate
Source: Integrative Medicine Communications; www.drkoop.com

Iron
Source: Prima Communications, Inc.www.personalhealthzone.com

L-Carnitine
Source: Integrative Medicine Communications; www.drkoop.com

Lovastatin
Source: Healthnotes, Inc. www.healthnotes.com

Magnesium
Source: Integrative Medicine Communications; www.drkoop.com

Magnesium
Source: Prima Communications, Inc.www.personalhealthzone.com

Magnesium
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,890,00.html

Potassium
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10086,00.html

Potassium-Sparing Diuretics
Source: Integrative Medicine Communications; www.drkoop.com

Pravastatin
Source: Healthnotes, Inc. www.healthnotes.com

Quercetin
Source: Healthnotes, Inc. www.healthnotes.com

Quercetin
Source: Integrative Medicine Communications; www.drkoop.com

Quercetin
Source: Prima Communications, Inc.www.personalhealthzone.com

Selenium
Source: Healthnotes, Inc. www.healthnotes.com

Selenium
Source: Integrative Medicine Communications; www.drkoop.com

Selenium
Source: Prima Communications, Inc.www.personalhealthzone.com

Selenium
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10055,00.html

Simvastatin
Source: Healthnotes, Inc. www.healthnotes.com

Vanadium
Alternative names: Vanadate, Vanadyl
Source: Integrative Medicine Communications; www.drkoop.com

- **Food and Diet**

Apples
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,44,00.html

Asparagus
Source: Healthnotes, Inc. www.healthnotes.com

Atkins Diet
Source: Healthnotes, Inc. www.healthnotes.com

Beef
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,85,00.html

Beets
Source: Healthnotes, Inc. www.healthnotes.com

Blood Type Diet
Source: Healthnotes, Inc. www.healthnotes.com

Brazil nuts
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com

Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,115,00.html

Brussels Sprouts
Source: Healthnotes, Inc. www.healthnotes.com

Cancer Prevention and Diet
Source: Healthnotes, Inc. www.healthnotes.com

Carbo-Loading Diet
Source: Healthnotes, Inc. www.healthnotes.com

Carrots
Source: Healthnotes, Inc. www.healthnotes.com

Celery
Source: Healthnotes, Inc. www.healthnotes.com

Chocolate
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,179,00.html

Chondroitin Sulfate
Source: Healthnotes, Inc. www.healthnotes.com

Coffee
Source: Healthnotes, Inc. www.healthnotes.com

Complex carbohydrates
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,993,00.html

Fat Alternatives and Fat Replacers
Source: Healthnotes, Inc. www.healthnotes.com

Ferrous Sulfate
Source: Integrative Medicine Communications; www.drkoop.com

Fish, lean
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,93,00.html

Flaxseeds
Source: Healthnotes, Inc. www.healthnotes.com

Garlic
Source: Prima Communications, Inc.www.personalhealthzone.com

Garlic
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,786,00.html

Hazelnuts
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,307,00.html

High Cholesterol
Source: Healthnotes, Inc. www.healthnotes.com

High-Fiber Diet
Source: Healthnotes, Inc. www.healthnotes.com

Hypertension
Source: Healthnotes, Inc. www.healthnotes.com

Kale
Source: Healthnotes, Inc. www.healthnotes.com

Lentils
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,99,00.html

Low-Fat Diet
Source: Healthnotes, Inc. www.healthnotes.com

Mackerel
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,310,00.html

Macrobiotic Diet
Source: Healthnotes, Inc. www.healthnotes.com

Monounsaturated Fats
Source: Healthnotes, Inc. www.healthnotes.com

Mushrooms
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10046,00.html

Okra
Source: Healthnotes, Inc. www.healthnotes.com

Olives
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com

Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,318,00.html

Omega-3 Fatty Acids
Source: Integrative Medicine Communications; www.drkoop.com

Omega-3 fatty acids
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,992,00.html

Omega-6 Fatty Acids
Source: Integrative Medicine Communications; www.drkoop.com

Omega-6 fatty acids
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,1037,00.html

Peanuts
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,110,00.html

Polyunsaturated Fats
Source: Healthnotes, Inc. www.healthnotes.com

Salmon
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,102,00.html

Saturated Fats
Source: Healthnotes, Inc. www.healthnotes.com

Shiitake Mushrooms
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,308,00.html

Soy
Source: Prima Communications, Inc.www.personalhealthzone.com

Soy products
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,135,00.html

Soybeans
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,105,00.html

Spinach
Source: Healthnotes, Inc. www.healthnotes.com

Sweet Potatoes
Source: Healthnotes, Inc. www.healthnotes.com

Tea
Source: Healthnotes, Inc. www.healthnotes.com

The Dean Ornish Diet
Source: Healthnotes, Inc. www.healthnotes.com

The Pritikin Diet Program
Source: Healthnotes, Inc. www.healthnotes.com

The Zone Diet
Source: Healthnotes, Inc. www.healthnotes.com

Tomatoes
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/foods_view/0,1523,41,00.html

Trans-Fats
Source: Healthnotes, Inc. www.healthnotes.com

Turnips
Source: Healthnotes, Inc. www.healthnotes.com

Vegetarian Diet
Source: Healthnotes, Inc. www.healthnotes.com

Wound Healing
Source: Healthnotes, Inc. www.healthnotes.com

CHAPTER 3. ALTERNATIVE MEDICINE AND HEART DISEASE

Overview

In this chapter, we will begin by introducing you to official information sources on complementary and alternative medicine (CAM) relating to heart disease. At the conclusion of this chapter, we will provide additional sources.

The Combined Health Information Database

The Combined Health Information Database (CHID) is a bibliographic database produced by health-related agencies of the U.S. federal government (mostly from the National Institutes of Health) that can offer concise information for a targeted search. The CHID database is updated four times a year at the end of January, April, July, and October. Check the titles, summaries, and availability of CAM-related information by using the "Simple Search" option at the following Web site: **http://chid.nih.gov/simple/simple.html**. In the drop box at the top, select "Complementary and Alternative Medicine." Then type "heart disease" (or synonyms) in the second search box. We recommend that you select 100 "documents per page" and to check the "whole records" options. The following was extracted using this technique:

- **Soybeans: Good for Your Heart. Patient Education**

  Source: Advance for Nurse Practitioners. 10(5): 85. May 2002.

  Summary: This article provides information on the health benefits of soybeans and foods made from soybeans. The possible benefits that soy proteins may have on certain diseases, such as coronary **heart disease,** menopause, osteoporosis, cancer, allergies, diabetes, and kidney disease, are briefly noted. The article also describes the soy products currently available, including edamame, miso, tofu, soy nuts, tempeh, soy milk, soy sauce, and natto. It lists two Web sites for additional information on soybeans.

- **Soy: Health Claims for Soy Protein, Questions About Other Components**

  Source: FDA Consumer. 34(3): 13-15, 18-20. May-June 2000.

Contact: Available from Food and Drug Administration. 5600 Fishers Lane, Rockville, MD 20857. (888) 463-6332. PRICE: Free.

Summary: This Food and Drug Administration (FDA) Consumer magazine article discusses FDA permission to food manufacturers to put labels on products high in soy protein indicating that these foods may help lower **heart disease** risk. It includes information on concerns about certain components in soy products, particularly isoflavones, which have engulfed the health claim regulation in controversy. The article features quotes from Elizabeth A. Yetley, Ph.D., lead scientist for nutrition at FDA's Center for Food Safety and Applied Nutrition; Christine Lewis, acting Director of the Center for Food Safety and Applied Nutrition's Office of Nutritional Products, Labeling, and Dietary Supplements; Margo Woods, D.Sc., associate professor of medicine at Tufts University; and Daniel Sheehan, Ph.D., director of the Estrogen Knowledge Base Program at FDA's National Center for Toxicological Research. The article also includes sections on soy benefits, the different types of soy products, trends in soy consumption, adding soy to the diet, and soy health claims. Organizations where consumers can obtain additional information on soy are listed at the end of the article.

- **Using the Body To Heal the Body: Exercise and Disease Intervention**

  Source: Alternative and Complementary Therapies. 4(3): 169-172. June 1998.

  Summary: This journal article discusses the multiple health benefits of exercise, highlighting the work in this area by Dr. L. Goldberg and D. L. Elliot at the Human Performance Laboratory, Division of Health Promotion and Sports Medicine, Oregon Health Sciences University in Portland. Drs. Goldberg and Elliot have focused their work on the effects of exercise in hypertension, neuromuscular diseases, certain metabolic conditions, and obesity. Clinicians at the Human Performance Laboratory offer patients a choice of testing options, and develop individualized exercise plans specifying exercise mode, intensity, duration, frequency, and progression. They also help patients develop strategies to overcome potential barriers to plan compliance. Exercise, along with diet, stress reduction, and social support, also is an integral component of the Opening Your Heart Program developed by Dr. D. Ornish at the Preventive Medicine Research Center in Sausalito, California. Dr. Ornish emphasizes that exercise does not have to be vigorous to be beneficial, noting that consistency is more important than intensity. In addition to its benefits in **heart disease,** recent research suggests a role for exercise in reducing cancer risk and improving function in older age. The article includes a list of recommended readings and 14 references.

National Center for Complementary and Alternative Medicine

The National Center for Complementary and Alternative Medicine (NCCAM) of the National Institutes of Health (**http://nccam.nih.gov/**) has created a link to the National Library of Medicine's databases to facilitate research for articles that specifically relate to heart disease and complementary medicine. To search the database, go to the following Web site: **http://www.nlm.nih.gov/nccam/camonpubmed.html**. Select "CAM on PubMed." Enter "heart disease" (or synonyms) into the search box. Click "Go." The following references provide information on particular aspects of complementary and alternative medicine that are related to heart disease:

- **A prospective study of calcium intake from diet and supplements and risk of ischemic heart disease among men.**
  Author(s): Al-Delaimy WK, Rimm E, Willett WC, Stampfer MJ, Hu FB.

Source: The American Journal of Clinical Nutrition. 2003 April; 77(4): 814-8.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12663277&dopt=Abstract

- **A survey of attitudes and experiences of women with heart disease.**
Author(s): Marcuccio E, Loving N, Bennett SK, Hayes SN.
Source: Women's Health Issues : Official Publication of the Jacobs Institute of Women's Health. 2003 January-February; 13(1): 23-31.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12598056&dopt=Abstract

- **Adult bone marrow stem cells regenerate myocardium in ischemic heart disease.**
Author(s): Orlic D.
Source: Annals of the New York Academy of Sciences. 2003 May; 996: 152-7.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12799293&dopt=Abstract

- **Cardiology Grand Rounds from the University of North Carolina at Chapel Hill. The antioxidant vitamins and coronary heart disease: Part 1. Basic science background and clinical observational studies.**
Author(s): Riley SJ, Stouffer GA.
Source: The American Journal of the Medical Sciences. 2002 December; 324(6): 314-20. Review.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12495298&dopt=Abstract

- **Cardiology Grand Rounds from the University of North Carolina at Chapel Hill. The antioxidant vitamins and coronary heart disease: Part II. Randomized clinical trials.**
Author(s): Riley SJ, Stouffer GA.
Source: The American Journal of the Medical Sciences. 2003 January; 325(1): 15-9. Review.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12544080&dopt=Abstract

- **Dietary monounsaturated versus polyunsaturated fatty acids: which is really better for protection from coronary heart disease?**
Author(s): Lada AT, Rudel LL.
Source: Current Opinion in Lipidology. 2003 February; 14(1): 41-6. Review.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12544660&dopt=Abstract

- **Does chelation therapy work for ischemic heart disease?**
Author(s): Anand A, Evans MF.
Source: Can Fam Physician. 2003 March; 49: 307-9. No Abstract Available.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12675543&dopt=Abstract

- **Dynamics of Interrelationships between the Content of Lipoprotein Particles, Fibrinogen, and Leukocyte Count in the Plasma from Patients with Coronary Heart**

Disease Treated with Kwai.
Author(s): Chernyad'eva IF, Shil'nikova SV, Rogoza AN, Kukharchuk VV.
Source: Bulletin of Experimental Biology and Medicine. 2003 May; 135(5): 436-9.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12910280&dopt=Abstract

- **Effect of dietary intervention and lipid-lowering treatment on brachial vasoreactivity in patients with ischemic heart disease and hypercholesterolemia.**
Author(s): Sondergaard E, Moller JE, Egstrup K.
Source: American Heart Journal. 2003 May; 145(5): E19.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12766751&dopt=Abstract

- **Epidemiologic studies on dietary fats and coronary heart disease.**
Author(s): Ascherio A.
Source: The American Journal of Medicine. 2002 December 30; 113 Suppl 9B: 9S-12S.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12566133&dopt=Abstract

- **Fish and long-chain omega-3 fatty acid intake and risk of coronary heart disease and total mortality in diabetic women.**
Author(s): Hu FB, Cho E, Rexrode KM, Albert CM, Manson JE.
Source: Circulation. 2003 April 15; 107(14): 1852-7. Epub 2003 March 31.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12668520&dopt=Abstract

- **Genetics and heart disease.**
Author(s): Chamsi Pasha H.
Source: Saudi Med J. 2003 January; 24(1): 11-8.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12590266&dopt=Abstract

- **Management of grown up congenital heart disease.**
Author(s): Deanfield J, Thaulow E, Warnes C, Webb G, Kolbel F, Hoffman A, Sorenson K, Kaemmer H, Thilen U, Bink-Boelkens M, Iserin L, Daliento L, Silove E, Redington A, Vouhe P, Priori S, Alonso MA, Blanc JJ, Budaj A, Cowie M, Deckers J, Fernandez Burgos E, Lekakis J, Lindahl B, Mazzotta G, Morais J, Oto A, Smiseth O, Trappe HJ, Klein W, Blomstrom-Lundqvist C, de Backer G, Hradec J, Mazzotta G, Parkhomenko A, Presbitero P, Torbicki A; Task Force on the Management of Grown Up Congenital Heart Disease, European Society of Cardiology; ESC Committee for Practice Guidelines.
Source: European Heart Journal. 2003 June; 24(11): 1035-84.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12868424&dopt=Abstract

- **Mercury and your heart. Does mercury in fish increase your risk of heart disease?**
Author(s): Ornato JP.
Source: Health News. 2003 January; 9(1): 3. No Abstract Available.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12545947&dopt=Abstract

- **Mindfulness meditation, anxiety reduction, and heart disease: a pilot study.**
  Author(s): Tacon AM, McComb J, Caldera Y, Randolph P.
  Source: Family & Community Health. 2003 January-March; 26(1): 25-33.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12802125&dopt=Abstract

- **n-3 Long-chain polyunsaturated fatty acids reduce risk of coronary heart disease death: extending the evidence to the elderly.**
  Author(s): Harris WS.
  Source: The American Journal of Clinical Nutrition. 2003 February; 77(2): 279-80.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12540382&dopt=Abstract

- **n-3 Polyunsaturated fatty acids, fatal ischemic heart disease, and nonfatal myocardial infarction in older adults: the Cardiovascular Health Study.**
  Author(s): Lemaitre RN, King IB, Mozaffarian D, Kuller LH, Tracy RP, Siscovick DS.
  Source: The American Journal of Clinical Nutrition. 2003 February; 77(2): 319-25.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12540389&dopt=Abstract

- **New directions in strategies using cell therapy for heart disease.**
  Author(s): Itescu S, Schuster MD, Kocher AA.
  Source: Journal of Molecular Medicine (Berlin, Germany). 2003 May; 81(5): 288-96. Epub 2003 April 16.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12698252&dopt=Abstract

- **Selectin-P and interleukin-8 plasma levels in coronary heart disease patients.**
  Author(s): Romuk E, Skrzep-Poloczek B, Wojciechowska C, Tomasik A, Birkner E, Wodniecki J, Gabrylewicz B, Ochala A, Tendera M.
  Source: European Journal of Clinical Investigation. 2002 September; 32(9): 657-61.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12486864&dopt=Abstract

- **Serum plant sterols as a potential risk factor for coronary heart disease.**
  Author(s): Sudhop T, Gottwald BM, von Bergmann K.
  Source: Metabolism: Clinical and Experimental. 2002 December; 51(12): 1519-21.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12489060&dopt=Abstract

- **Steeped in health. Ordinary tea may help fight cancer, heart disease and, now, infection.**
  Author(s): Gorman C.
  Source: Time. 2003 May 5; 161(18): 86.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12747035&dopt=Abstract

- **The dietary prevention of ischaemic heart disease.**
  Author(s): Saracci R.

Source: Iarc Sci Publ. 2002; 156: 531-3. No Abstract Available.
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12484252&dopt=Abstract

- **The Garden of Eden-plant based diets, the genetic drive to conserve cholesterol and its implications for heart disease in the 21st century.**
  Author(s): Jenkins DJ, Kendall CW, Marchie A, Jenkins AL, Connelly PW, Jones PJ, Vuksan V.
  Source: Comparative Biochemistry and Physiology. Part A, Molecular & Integrative Physiology. 2003 September; 136(1): 141-51.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=14527636&dopt=Abstract

- **The natural cure of coronary heart disease.**
  Author(s): Withnell A.
  Source: Nutr Health. 2003; 17(1): 55-60. Review.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12803281&dopt=Abstract

- **Understanding the transition from alternative medicine to mainstream science: the homocysteine theory of heart disease and the crucial role of effective mentoring.**
  Author(s): Podell RN.
  Source: Medical Hypotheses. 2003 September; 61(3): 340-5.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12944102&dopt=Abstract

- **Vitamin C and risk of coronary heart disease in women.**
  Author(s): Osganian SK, Stampfer MJ, Rimm E, Spiegelman D, Hu FB, Manson JE, Willett WC.
  Source: Journal of the American College of Cardiology. 2003 July 16; 42(2): 246-52.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12875759&dopt=Abstract

- **Xuezhikang decreases serum lipoprotein(a) and C-reactive protein concentrations in patients with coronary heart disease.**
  Author(s): Liu L, Zhao SP, Cheng YC, Li YL.
  Source: Clinical Chemistry. 2003 August; 49(8): 1347-52.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12881451&dopt=Abstract

Additional Web Resources

A number of additional Web sites offer encyclopedic information covering CAM and related topics. The following is a representative sample:

- Alternative Medicine Foundation, Inc.: **http://www.herbmed.org/**
- AOL: **http://search.aol.com/cat.adp?id=169&layer=&from=subcats**
- Chinese Medicine: **http://www.newcenturynutrition.com/**

- drkoop.com®: **http://www.drkoop.com/InteractiveMedicine/IndexC.html**
- Family Village: **http://www.familyvillage.wisc.edu/med_altn.htm**
- Google: **http://directory.google.com/Top/Health/Alternative/**
- Healthnotes: **http://www.healthnotes.com/**
- MedWebPlus: **http://medwebplus.com/subject/Alternative_and_Complementary_Medicine**
- Open Directory Project: **http://dmoz.org/Health/Alternative/**
- HealthGate: **http://www.tnp.com/**
- WebMD®Health: **http://my.webmd.com/drugs_and_herbs**
- WholeHealthMD.com: **http://www.wholehealthmd.com/reflib/0,1529,00.html**
- Yahoo.com: **http://dir.yahoo.com/Health/Alternative_Medicine/**

The following is a specific Web list relating to heart disease; please note that any particular subject below may indicate either a therapeutic use, or a contraindication (potential danger), and does not reflect an official recommendation:

- **General Overview**

  Alzheimer's Disease
  Source: Integrative Medicine Communications; www.drkoop.com

  Amyloidosis
  Source: Integrative Medicine Communications; www.drkoop.com

  Anaphylaxis
  Source: Integrative Medicine Communications; www.drkoop.com

  Angina
  Source: Healthnotes, Inc. www.healthnotes.com

  Angina
  Source: Integrative Medicine Communications; www.drkoop.com

  Atherosclerosis
  Source: Healthnotes, Inc. www.healthnotes.com

  Atherosclerosis and Heart Disease Prevention
  Source: Prima Communications, Inc.www.personalhealthzone.com

  Bone Marrow Disorders
  Source: Integrative Medicine Communications; www.drkoop.com

  Cancer Prevention (Reducing the Risk)
  Source: Prima Communications, Inc.www.personalhealthzone.com

Cardiomyopathy
Source: Healthnotes, Inc. www.healthnotes.com

Cardiovascular Disease Overview
Source: Healthnotes, Inc. www.healthnotes.com

Cataracts (Prevention)
Source: Prima Communications, Inc.www.personalhealthzone.com

Chronic Myelogenous Leukemia
Source: Integrative Medicine Communications; www.drkoop.com

Congestive Heart Failure
Source: Healthnotes, Inc. www.healthnotes.com

Congestive Heart Failure
Source: Integrative Medicine Communications; www.drkoop.com

Cough
Source: Integrative Medicine Communications; www.drkoop.com

Depression
Source: Integrative Medicine Communications; www.drkoop.com

Depression (Mild to Moderate)
Source: Prima Communications, Inc.www.personalhealthzone.com

Diabetes
Source: Healthnotes, Inc. www.healthnotes.com

Diabetes Mellitus
Source: Integrative Medicine Communications; www.drkoop.com

Endocarditis
Source: Integrative Medicine Communications; www.drkoop.com

Fainting
Source: Integrative Medicine Communications; www.drkoop.com

Heart Attack
Source: Healthnotes, Inc. www.healthnotes.com

Heart Attack
Source: Integrative Medicine Communications; www.drkoop.com

High Blood Pressure
Source: Integrative Medicine Communications; www.drkoop.com

High Cholesterol
Source: Integrative Medicine Communications; www.drkoop.com

High Cholesterol
Source: Prima Communications, Inc.www.personalhealthzone.com

High Homocysteine
Source: Healthnotes, Inc. www.healthnotes.com

High Triglycerides
Source: Healthnotes, Inc. www.healthnotes.com

Hypercholesterolemia
Source: Integrative Medicine Communications; www.drkoop.com

Hypertension
Source: Integrative Medicine Communications; www.drkoop.com

Hypertension
Alternative names: High Blood Pressure
Source: Prima Communications, Inc.www.personalhealthzone.com

Insomnia
Source: Integrative Medicine Communications; www.drkoop.com

Insulin Resistance Syndrome
Source: Healthnotes, Inc. www.healthnotes.com

Intermittent Claudication
Source: Healthnotes, Inc. www.healthnotes.com

Iron-Deficiency Anemia
Source: Healthnotes, Inc. www.healthnotes.com

Lung Cancer
Source: Healthnotes, Inc. www.healthnotes.com

Macular Degeneration
Source: Integrative Medicine Communications; www.drkoop.com

Menopausal Symptoms (Other Than Osteoporosis)
Source: Prima Communications, Inc.www.personalhealthzone.com

Menopause
Source: Healthnotes, Inc. www.healthnotes.com

Menopause
Source: Integrative Medicine Communications; www.drkoop.com

Myelofibrosis
Source: Integrative Medicine Communications; www.drkoop.com

Myeloproliferative Disorders
Source: Integrative Medicine Communications; www.drkoop.com

Myocardial Infarction
Source: Integrative Medicine Communications; www.drkoop.com

Obesity
Source: Integrative Medicine Communications; www.drkoop.com

Parkinson's Disease
Source: Integrative Medicine Communications; www.drkoop.com

Polycythemia Vera
Source: Integrative Medicine Communications; www.drkoop.com

Pulmonary Edema
Source: Integrative Medicine Communications; www.drkoop.com

Pulmonary Hypertension
Source: Integrative Medicine Communications; www.drkoop.com

Raynaud's Disease
Source: Healthnotes, Inc. www.healthnotes.com

Shock
Source: Integrative Medicine Communications; www.drkoop.com

Sleeplessness
Source: Integrative Medicine Communications; www.drkoop.com

Sprains and Strains
Source: Healthnotes, Inc. www.healthnotes.com

Stress
Source: Integrative Medicine Communications; www.drkoop.com

Stroke
Source: Healthnotes, Inc. www.healthnotes.com

Stroke
Source: Integrative Medicine Communications; www.drkoop.com

Syncope
Source: Integrative Medicine Communications; www.drkoop.com

Systemic Lupus Erythematosus
Source: Healthnotes, Inc. www.healthnotes.com

Thrombocytosis
Source: Integrative Medicine Communications; www.drkoop.com

TIAs
Source: Integrative Medicine Communications; www.drkoop.com

Transient Ischemic Attacks
Source: Integrative Medicine Communications; www.drkoop.com

Weight Loss and Obesity
Source: Healthnotes, Inc. www.healthnotes.com

- **Alternative Therapy**

Apitherapy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,669,00.html

Ayurveda
Source: Integrative Medicine Communications; www.drkoop.com

Ayurveda
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,672,00.html

Biofeedback
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,675,00.html

Chelation therapy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,679,00.html

Chirognomy
Alternative names: cheirognomy chirognosy chiromancy chirosophy
Source: The Canoe version of A Dictionary of Alternative-Medicine Methods, by Priorities for Health editor Jack Raso, M.S., R.D.
Hyperlink: http://www.canoe.ca/AltmedDictionary/c.html

Colon therapy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,682,00.html

Craniosacral therapy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,685,00.html

Fasting
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,694,00.html

Homeopathy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,703,00.html

Macrobiotics
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,714,00.html

Massage therapy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,716,00.html

Meditation
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,717,00.html

Native American medicine
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,721,00.html

Naturopathy
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,722,00.html

Nutrition
Source: Integrative Medicine Communications; www.drkoop.com

Prayer
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,728,00.html

Qigong
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,729,00.html

Reflexology
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,730,00.html

Relaxation Techniques
Source: Integrative Medicine Communications; www.drkoop.com

Spirituality
Source: Integrative Medicine Communications; www.drkoop.com

Tai Chi
Source: Integrative Medicine Communications; www.drkoop.com

Tai chi
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,737,00.html

Yoga
Source: Integrative Medicine Communications; www.drkoop.com

Yoga
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,746,00.html

- **Chinese Medicine**

Bawei Chenxiang San
Alternative names: Bawei Chenxiang Powder
Source: Pharmacopoeia Commission of the Ministry of Health, People's Republic of China
Hyperlink: http://www.newcenturynutrition.com/cgi-local/patent_herbs_db/db.cgi?db=default&Chinese=Bawei%20Chenxiang%20San&mh=10&sb=---&view_records=View+Records

Guanxin Danshen Pian
Alternative names: Guanxin Danshen Tablets
Source: Pharmacopoeia Commission of the Ministry of Health, People's Republic of China
Hyperlink: http://www.newcenturynutrition.com/cgi-local/patent_herbs_db/db.cgi?db=default&Chinese=Guanxin%20Danshen%20Pian&mh=10&sb=---&view_records=View+Records

Jingzhi Guanxin Pian
Alternative names: Jingzhi Guanxin Tablets
Source: Pharmacopoeia Commission of the Ministry of Health, People's Republic of China
Hyperlink: http://www.newcenturynutrition.com/cgi-local/patent_herbs_db/db.cgi?db=default&Chinese=Jingzhi%20Guanxin%20Pian&mh=10&sb=---&view_records=View+Records

Shuxin Koufuye
Alternative names: Shuxin Oral Liquid; Shuxin Koufuye
(Shu Xin Kou Fu Ye)
Source: Pharmacopoeia Commission of the Ministry of Health, People's Republic of China
Hyperlink: http://www.newcenturynutrition.com/cgi-local/patent_herbs_db/db.cgi?db=default&Chinese=Shuxin%20Koufuye&mh=10&sb=---&view_records=View+Records

Shuxiong Pian
Alternative names: Shuxiong Tablets; Shuxiong Pian
(Shu Xiong Pi An)
Source: Pharmacopoeia Commission of the Ministry of Health, People's Republic of China
Hyperlink: http://www.newcenturynutrition.com/cgi-local/patent_herbs_db/db.cgi?db=default&Chinese=Shuxiong%20Pian&mh=10&sb=---&view_records=View+Records

Tanxiang
Alternative names: Sandalwood; Lignum Santaii Albi
Source: Chinese Materia Medica

Tinglizi
Alternative names: Pepperweed Seed; Semen Lepidii
Source: Chinese Materia Medica

- **Herbs and Supplements**

5-Aminosalicylic Acid Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Activated charcoal
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,832,00.html

ALA
Source: Integrative Medicine Communications; www.drkoop.com

Alpha2-Adrenergic Agonists
Source: Integrative Medicine Communications; www.drkoop.com

Alpha-Linolenic Acid (ALA)
Source: Integrative Medicine Communications; www.drkoop.com

Alpha-Lipoic Acid
Source: Integrative Medicine Communications; www.drkoop.com

Alpha-lipoic acid
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10002,00.html

American Ginseng
Alternative names: Panax quinquefolium
Source: Integrative Medicine Communications; www.drkoop.com

Amino acids
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10003,00.html

Aminoglycosides
Source: Integrative Medicine Communications; www.drkoop.com

Ananas comosus
Source: Integrative Medicine Communications; www.drkoop.com

Androstenedione
Source: Healthnotes, Inc. www.healthnotes.com

Androstenedione
Source: Prima Communications, Inc.www.personalhealthzone.com

Angelica sinensis
Source: Integrative Medicine Communications; www.drkoop.com

Angkak
Source: Integrative Medicine Communications; www.drkoop.com

Antibiotic Combination: Sulfa Drugs
Source: Integrative Medicine Communications; www.drkoop.com

Antioxidants
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10004,00.html

Antituberculosis Agents
Source: Integrative Medicine Communications; www.drkoop.com

Aortic Glycosaminoglycans
Source: Prima Communications, Inc.www.personalhealthzone.com

Arnica
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,753,00.html

Asian Ginseng
Alternative names: Panax ginseng
Source: Integrative Medicine Communications; www.drkoop.com

Astragalus
Alternative names: Astragalus membranaceus
Source: Healthnotes, Inc. www.healthnotes.com

Astragalus
Alternative names: Astragalus membranaceus, Astragalus membranaceus var. mongholicus, Huang-qi, Milk-Vetch Root
Source: Integrative Medicine Communications; www.drkoop.com

Astragalus mem
Alternative names: Huang-Qi; Astragalus membranaceus
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org
Astragalus membranaceus
Source: Integrative Medicine Communications; www.drkoop.com

Astragalus mongholicus
Alternative names: Astragalus membranaceus, Astragalus membranaceus var. mongholicus, Huang-qi, Milk-Vetch Root
Source: Integrative Medicine Communications; www.drkoop.com

Barbiturates
Source: Integrative Medicine Communications; www.drkoop.com

B-carotene
Source: Integrative Medicine Communications; www.drkoop.com

Beni-koji
Source: Integrative Medicine Communications; www.drkoop.com

Beta-Blockers
Source: Integrative Medicine Communications; www.drkoop.com

Beta-Carotene
Alternative names: b-carotene, Trans-beta Carotene; Provitamin A, Betacarotenum
Source: Integrative Medicine Communications; www.drkoop.com

Beta-Carotene
Source: Prima Communications, Inc.www.personalhealthzone.com

Beta-carotene
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10103,00.html

Betacarotenum
Source: Integrative Medicine Communications; www.drkoop.com

Betaine
Alternative names: Trimethylglycine
Source: Integrative Medicine Communications; www.drkoop.com

Biguanides
Source: Integrative Medicine Communications; www.drkoop.com

Bile Acid Sequestrants
Source: Integrative Medicine Communications; www.drkoop.com

Black Cohosh
Source: Prima Communications, Inc.www.personalhealthzone.com

Black cohosh
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10009,00.html
Blackberry
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,837,00.html

Bloodroot
Alternative names: Sanguinaria canadensis
Source: Healthnotes, Inc. www.healthnotes.com

Borago
Alternative names: Borage; Borago officinalis
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org

Brahmi
Alternative names: Centella asiatica , Centella, March Pennywort, Indian Pennywort, Hydrocotyle, Brahmi (Sanskrit), Luei Gong Gen (Chinese)(Note: Gotu kola should not be confused with kola nut.)
Source: Integrative Medicine Communications; www.drkoop.com

Bromelain
Source: Healthnotes, Inc. www.healthnotes.com

Bromelain
Alternative names: Ananas comosus, Bromelainum
Source: Integrative Medicine Communications; www.drkoop.com

Bromelainum
Source: Integrative Medicine Communications; www.drkoop.com

CACTUS GRANDIFLORUS
Source: The Canadian Internet Directory for Holistic Help, WellNet, Health and Wellness Network; www.wellnet.ca

Calciferol
Source: Integrative Medicine Communications; www.drkoop.com

Calcitrol
Source: Integrative Medicine Communications; www.drkoop.com

Capsaicin
Source: Integrative Medicine Communications; www.drkoop.com

Capsicum frutescens
Source: Integrative Medicine Communications; www.drkoop.com

Carotenoids
Source: Healthnotes, Inc. www.healthnotes.com

Carotenoids
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,763,00.html

Catechins
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,1023,00.html

Cayenne
Alternative names: Capsicum frutescens, Capsicum spp., Capsaicin, Chili Pepper, Red Pepper
Source: Integrative Medicine Communications; www.drkoop.com

Cayenne
Source: Prima Communications, Inc.www.personalhealthzone.com

Cayenne
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,765,00.html

Centella
Source: Integrative Medicine Communications; www.drkoop.com

Centella asiatica
Alternative names: Centella asiatica , Centella, March Pennywort, Indian Pennywort, Hydrocotyle, Brahmi (Sanskrit), Luei Gong Gen (Chinese)(Note: Gotu kola should not be confused with kola nut.)
Source: Integrative Medicine Communications; www.drkoop.com

Cephalosporins
Source: Integrative Medicine Communications; www.drkoop.com

Cherry fruit extract
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10015,00.html

Chili Pepper
Source: Integrative Medicine Communications; www.drkoop.com

Chinese Angelica
Source: Integrative Medicine Communications; www.drkoop.com

Cholecalciferol
Source: Integrative Medicine Communications; www.drkoop.com

Cobalamin
Source: Integrative Medicine Communications; www.drkoop.com

Coenzyme Q
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,768,00.html

Coenzyme Q10
Source: Integrative Medicine Communications; www.drkoop.com
Coenzyme Q10 (CoQ10)
Source: Prima Communications, Inc.www.personalhealthzone.com

CoQ10
Source: Integrative Medicine Communications; www.drkoop.com

CORN SILK
Source: The Canadian Internet Directory for Holistic Help, WellNet, Health and Wellness Network; www.wellnet.ca

Crataegus
Alternative names: Hawthorn; Crataegus oxyacantha L.
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org

Danggui
Alternative names: Angelica sinensis, Chinese Angelica, Dang Gui, Danngui, Dong Qua, Tang Kuei, Tan Kue Bai zhi(Note: Dong quai should not be confused with Angelica root or Angelica seed.)
Source: Integrative Medicine Communications; www.drkoop.com

Dehydroepiandrosterone (DHEA)
Source: Healthnotes, Inc. www.healthnotes.com

Dehydroepiandrosterone (DHEA)
Source: Integrative Medicine Communications; www.drkoop.com

DHA
Source: Integrative Medicine Communications; www.drkoop.com

DHEA
Source: Integrative Medicine Communications; www.drkoop.com

DHEA
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10022,00.html

Dipyridamole
Source: Healthnotes, Inc. www.healthnotes.com

Docosahexaenoic Acid
Source: Healthnotes, Inc. www.healthnotes.com

Docosahexaenoic Acid (DHA)
Source: Integrative Medicine Communications; www.drkoop.com

Dong Quai
Alternative names: Angelica sinensis, Chinese Angelica, Dang Gui, Danngui, Dong Qua, Tang Kuei, Tan Kue Bai zhi(Note: Dong quai should not be confused with Angelica root or Angelica seed.)
Source: Integrative Medicine Communications; www.drkoop.com

EDTA
Source: Integrative Medicine Communications; www.drkoop.com

Eicosapentaenoic Acid (EPA)
Source: Integrative Medicine Communications; www.drkoop.com

Electrolytes
Source: Integrative Medicine Communications; www.drkoop.com

Eleuthero
Alternative names: Siberian Ginseng, Eleuthero; Acanthopanax/Eleutherococcus senticosus Rupr. & Maxim.
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org

EPA
Source: Integrative Medicine Communications; www.drkoop.com

Ephedra
Source: Prima Communications, Inc.www.personalhealthzone.com

Ephedra (Ma huang)
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,777,00.html

Erocalciferol
Source: Integrative Medicine Communications; www.drkoop.com

Estrogens (Combined)
Source: Healthnotes, Inc. www.healthnotes.com

Ethylenediaminetetraacetic Acid (EDTA)
Source: Integrative Medicine Communications; www.drkoop.com

Fenofibrate
Source: Healthnotes, Inc. www.healthnotes.com

Fiber
Source: Healthnotes, Inc. www.healthnotes.com

Fiber
Source: Integrative Medicine Communications; www.drkoop.com

Fibric Acid Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Flavonoids
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,782,00.html

Forskolin
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10025,00.html

Gamma-Linolenic Acid (GLA)
Source: Integrative Medicine Communications; www.drkoop.com

Ginkgo biloba
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,788,00.html

Ginseng (Panax)
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10029,00.html

GLA
Source: Integrative Medicine Communications; www.drkoop.com

GLA (Gamma-Linolenic Acid)
Source: Prima Communications, Inc.www.personalhealthzone.com

Glucomannan
Source: Healthnotes, Inc. www.healthnotes.com

Glutamic Acid
Source: Healthnotes, Inc. www.healthnotes.com

Glutathione
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,854,00.html

Goldenseal
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com

Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,791,00.html

Gotu Kola
Alternative names: Centella asiatica
Source: Healthnotes, Inc. www.healthnotes.com

Gotu Kola
Alternative names: Centella asiatica , Centella, March Pennywort, Indian Pennywort, Hydrocotyle, Brahmi (Sanskrit), Luei Gong Gen (Chinese)(Note: Gotu kola should not be confused with kola nut.)
Source: Integrative Medicine Communications; www.drkoop.com

Grape Seed
Alternative names: Vitis vinifera
Source: Integrative Medicine Communications; www.drkoop.com

Grape seed extract
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,793,00.html

Green Tea
Source: Prima Communications, Inc.www.personalhealthzone.com

Green tea
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10032,00.html

Gugulipid
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10033,00.html

Hawthorn
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10035,00.html

Histamine H2 Antagonists
Source: Integrative Medicine Communications; www.drkoop.com

Hong Qu
Source: Integrative Medicine Communications; www.drkoop.com

Huang-qi
Source: Integrative Medicine Communications; www.drkoop.com

Hung-chu
Source: Integrative Medicine Communications; www.drkoop.com

Hydantoin Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Hydrocotyle
Source: Integrative Medicine Communications; www.drkoop.com

Indian Pennywort
Source: Integrative Medicine Communications; www.drkoop.com

Inhalant, Systemic, and Topical Corticosteroids
Source: Integrative Medicine Communications; www.drkoop.com

Inosine
Source: Prima Communications, Inc.www.personalhealthzone.com

Ipriflavone
Source: Prima Communications, Inc.www.personalhealthzone.com

Isoflavones
Source: Prima Communications, Inc.www.personalhealthzone.com

Ispaghula
Source: Integrative Medicine Communications; www.drkoop.com

Kava
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,798,00.html

Licorice
Source: Prima Communications, Inc.www.personalhealthzone.com

Licorice
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,801,00.html

Lipoic Acid
Source: Prima Communications, Inc.www.personalhealthzone.com

Loop Diuretics
Source: Healthnotes, Inc. www.healthnotes.com

Loop Diuretics
Source: Integrative Medicine Communications; www.drkoop.com

Lubricant Laxatives
Source: Integrative Medicine Communications; www.drkoop.com

Lycopene
Source: Healthnotes, Inc. www.healthnotes.com

Macrolides
Source: Integrative Medicine Communications; www.drkoop.com

Maitake
Source: Prima Communications, Inc.www.personalhealthzone.com

Marsh Pennywort
Alternative names: Centella asiatica , Centella, March Pennywort, Indian Pennywort, Hydrocotyle, Brahmi (Sanskrit), Luei Gong Gen (Chinese)(Note: Gotu kola should not be confused with kola nut.)
Source: Integrative Medicine Communications; www.drkoop.com

Melatonin
Source: Healthnotes, Inc. www.healthnotes.com

Melatonin
Source: Integrative Medicine Communications; www.drkoop.com

Melatonin
Source: Prima Communications, Inc.www.personalhealthzone.com

Melatonin
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,804,00.html

Metformin
Source: Healthnotes, Inc. www.healthnotes.com

Methionine
Source: Healthnotes, Inc. www.healthnotes.com

Milk-Vetch Root
Source: Integrative Medicine Communications; www.drkoop.com

Monascus
Source: Integrative Medicine Communications; www.drkoop.com

Monophasic, Biphasic, and Triphasic Preparations
Source: Integrative Medicine Communications; www.drkoop.com

NAC (N-acetylcysteine)
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,809,00.html

N-Acetyl Cysteine (NAC)
Source: Prima Communications, Inc.www.personalhealthzone.com

Natural progesterone cream
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com

Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10099,00.html

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)
Source: Integrative Medicine Communications; www.drkoop.com

OPCs (Oligomeric Proanthocyanidins)
Source: Prima Communications, Inc.www.personalhealthzone.com

Oral Contraceptives
Source: Prima Communications, Inc.www.personalhealthzone.com

Panax
Alternative names: Ginseng; Panax ginseng
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org

Panax ginseng
Source: Integrative Medicine Communications; www.drkoop.com

Panax quinquefolium
Source: Integrative Medicine Communications; www.drkoop.com

Penicillin Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Phenothiazine Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Plantago isphagula
Source: Integrative Medicine Communications; www.drkoop.com

Progesterone
Source: Healthnotes, Inc. www.healthnotes.com

Proton Pump Inhibitors (Gastric Acid Secretion Inhibitors)
Source: Integrative Medicine Communications; www.drkoop.com

Psyllium
Alternative names: Ispaghula,Plantago isphagula
Source: Integrative Medicine Communications; www.drkoop.com

Quinolones
Source: Integrative Medicine Communications; www.drkoop.com

Red Clover
Alternative names: Trifolium pratense , beebread, cow clover, cow grass, meadow clover, purple clover
Source: Integrative Medicine Communications; www.drkoop.com

Red Koji
Source: Integrative Medicine Communications; www.drkoop.com

Red Leaven
Source: Integrative Medicine Communications; www.drkoop.com

Red Pepper
Source: Integrative Medicine Communications; www.drkoop.com

Red Rice
Source: Integrative Medicine Communications; www.drkoop.com

Red Yeast Rice
Alternative names: Angkak, Beni-koju, Hong Qu, Hung-chu, Monascus, Red Leaven, Red Rice, Red Koji, Zhitai, Xue Zhi Kang
Source: Integrative Medicine Communications; www.drkoop.com

Red yeast rice
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10054,00.html

Resveratrol
Source: Healthnotes, Inc. www.healthnotes.com

Resveratrol
Source: Prima Communications, Inc.www.personalhealthzone.com

Resveratrol
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,1040,00.html

Salicylates
Source: Integrative Medicine Communications; www.drkoop.com

Sambucus
Alternative names: Black Elderberry; Sambucus nigra L.
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org

SAMe
Source: Healthnotes, Inc. www.healthnotes.com

Soy isoflavones
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10057,00.html

Spirulina and kelp
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10058,00.html

St. John's Wort
Source: Prima Communications, Inc.www.personalhealthzone.com

Sulfonylureas
Source: Integrative Medicine Communications; www.drkoop.com

Tang Kuei
Source: Integrative Medicine Communications; www.drkoop.com

Taurine
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10059,00.html

Tetracycline Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Theophylline Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Thiazide Diuretics
Source: Healthnotes, Inc. www.healthnotes.com

Thiazide Diuretics
Source: Integrative Medicine Communications; www.drkoop.com

Thioxanthene Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

TMG (Trimethylglycine)
Source: Prima Communications, Inc.www.personalhealthzone.com

Trace minerals
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,10061,00.html

Trans-Beta-Carotene
Source: Integrative Medicine Communications; www.drkoop.com

Tribulus Puncture
Alternative names: Puncture Vine, Goathead; Tribulus terrestris L.
Source: Alternative Medicine Foundation, Inc. www.amfoundation.org

Tricyclic Antidepressants (TCAs)
Source: Integrative Medicine Communications; www.drkoop.com

Trimethylglycine
Source: Integrative Medicine Communications; www.drkoop.com

Turmeric
Source: Prima Communications, Inc.www.personalhealthzone.com

Uricosuric Agents
Source: Integrative Medicine Communications; www.drkoop.com

Valproic Acid Derivatives
Source: Integrative Medicine Communications; www.drkoop.com

Vanadate
Source: Integrative Medicine Communications; www.drkoop.com

Vanadyl
Source: Integrative Medicine Communications; www.drkoop.com

Vasodilators
Source: Integrative Medicine Communications; www.drkoop.com

Vitis vinifera
Source: Integrative Medicine Communications; www.drkoop.com

Yohimbe
Alternative names: Pausinystalia yohimbe
Source: Healthnotes, Inc. www.healthnotes.com

Yohimbe
Source: WholeHealthMD.com, LLC. www.wholehealthmd.com
Hyperlink:
http://www.wholehealthmd.com/refshelf/substances_view/0,1525,830,00.html

Zhitai
Source: Integrative Medicine Communications; www.drkoop.com

Zue Zhi Kang
Source: Integrative Medicine Communications; www.drkoop.com

General References

A good place to find general background information on CAM is the National Library of Medicine. It has prepared within the MEDLINEplus system an information topic page dedicated to complementary and alternative medicine. To access this page, go to the MEDLINEplus site at **http://www.nlm.nih.gov/medlineplus/alternativemedicine.html**. This Web site provides a general overview of various topics and can lead to a number of general sources.

CHAPTER 4. DISSERTATIONS ON HEART DISEASE

Overview

In this chapter, we will give you a bibliography on recent dissertations relating to heart disease. We will also provide you with information on how to use the Internet to stay current on dissertations. **IMPORTANT NOTE:** When following the search strategy described below, you may discover non-medical dissertations that use the generic term "heart disease" (or a synonym) in their titles. To accurately reflect the results that you might find while conducting research on heart disease, we have not necessarily excluded non-medical dissertations in this bibliography.

Dissertations on Heart Disease

ProQuest Digital Dissertations, the largest archive of academic dissertations available, is located at the following Web address: **http://wwwlib.umi.com/dissertations**. From this archive, we have compiled the following list covering dissertations devoted to heart disease. You will see that the information provided includes the dissertation's title, its author, and the institution with which the author is associated. The following covers recent dissertations found when using this search procedure:

- **A Comparison of Alienation and Information about Heart Disease between Normal and Cardiac Patients** by Morrison, Mary Jane, Phd from The University of Connecticut, 1971, 67 pages
  http://wwwlib.umi.com/dissertations/fullcit/7214246
- **A Demographic Analysis of Heterogeneous Coronary Heart Disease Mortality Rates over Space and Time** by El-bolkiny, Mohamed Tawfik, Phd from The University of Connecticut, 1986, 183 pages
  http://wwwlib.umi.com/dissertations/fullcit/8709026
- **A Mathematical Model of Aging Processes, with Reference to Heart Disease** by Brown, Kenneth S; Phd from University of Waterloo (canada), 1974
  http://wwwlib.umi.com/dissertations/fullcit/NK21589
- **A Model of Hostility and Coronary Heart Disease Based on Orientation to Self and Others** by Altum, Sharyl Ann; Phd from University of Cincinnati, 2002, 149 pages
  http://wwwlib.umi.com/dissertations/fullcit/3041135

- **A Nested Case-control Study of the Association between Chlamydia Pneumoniae and Cytomegalovirus and Coronary Heart Disease** by Arcari, Christine Marie; Phd from The Johns Hopkins University, 2003, 185 pages
  http://wwwlib.umi.com/dissertations/fullcit/3068112
- **A Qualitative Assessment of Susceptibility to Coronary Heart Disease As Perceived by Selected Middle-aged Women** by Boudreau, Margaret A., Phd from Southern Illinois University at Carbondale, 1995, 133 pages
  http://wwwlib.umi.com/dissertations/fullcit/9536518
- **A Study of Adults with Congenital Heart Disease** by Rosenberg, Deborah Miriam, Phd from University of South Florida, 1999, 200 pages
  http://wwwlib.umi.com/dissertations/fullcit/9922451
- **A Study of Some of the Psychological Reactions to Heart Disease and Their Implications for Pastoral Care** by Hester, Larry Ronald, Ddiv from Vanderbilt University Divinity School, 1971, 120 pages
  http://wwwlib.umi.com/dissertations/fullcit/7126145
- **Adjustment of Children to Congenital Heart Disease: the Significance of the Mother-child Dyad** by Bertram, Susan, Edd from Harvard University, 1988, 174 pages
  http://wwwlib.umi.com/dissertations/fullcit/8907627
- **Administrators, Stress, and Coronary Heart Disease.** by Dale, Rosemary Louise, Edd from Ball State University, 1976, 139 pages
  http://wwwlib.umi.com/dissertations/fullcit/7708654
- **Air Pollution and Daily Hospital Admissions for Ischemic Heart Disease in the South Coast Air Basin of California** by Mann, Jennifer Kate; Phd from University of California, Berkeley, 2002, 129 pages
  http://wwwlib.umi.com/dissertations/fullcit/3082309
- **An Application of the Health Belief Model to a Preventive Health Behavior among High-risk Heart Disease Screening Program Participants.** by Schelzel, George Walter, Phd from The Pennsylvania State University, 1977, 163 pages
  http://wwwlib.umi.com/dissertations/fullcit/7803366
- **An Identification and Description of Community Health Education Activities in Jackson County for the Risk Factors Associated with Heart Disease, Cancer, and Stroke (illinois)** by Soha, Carol Jean, Phd from Southern Illinois University at Carbondale, 1983, 141 pages
  http://wwwlib.umi.com/dissertations/fullcit/8326567
- **An Instrument to Measure the Tendency towards Heart Disease Risk in an Adolescent Population: the Heart Health Profile.** by Golaszewski, Thomas Jude, Edd from State University of New York at Buffalo, 1978, 160 pages
  http://wwwlib.umi.com/dissertations/fullcit/7817037
- **Bodies at Risk: Heart Disease in Everyday Life** by Wheatley, Elizabeth Ellen, Phd from University of California, Santa Cruz, 1998, 453 pages
  http://wwwlib.umi.com/dissertations/fullcit/9907395
- **Case Study of a Health Promotion Coalition (heart Disease, Coalitions)** by Rollins, Lisa Kay, Phd from University of Virginia, 1992, 184 pages
  http://wwwlib.umi.com/dissertations/fullcit/9316932

- **Comparative Analysis of Rehabilitation Through Exercise and Traditional Treatment on Coronary Heart Disease.** by Karatun, Ozdemir, Phd from University of Kansas, 1975, 384 pages
  http://wwwlib.umi.com/dissertations/fullcit/7530050
- **Comparison of Risk Factors for Coronary Heart Disease in Sedentary and Physically Active College Students** by Jensen, Marian, Edd from Brigham Young University, 1992, 112 pages
  http://wwwlib.umi.com/dissertations/fullcit/9220176
- **Coronary Artery Bypass Surgery: Education of the Family (adult Heart Disease, Support, Prevention, Patient)** by Kroon, Janet Ellen Rohr, Edd from University of South Dakota, 1985, 67 pages
  http://wwwlib.umi.com/dissertations/fullcit/8609385
- **Coronary Heart Disease Risk Factor Variables in Black and White Females Aged 18-24 Years (discriminant, Regression, Race)** by Myers, Barbee Claudette, Phd from The University of Tennessee, 1984, 154 pages
  http://wwwlib.umi.com/dissertations/fullcit/8506909
- **Coronary Heart Disease Risk Factors and Health-related Fitness of Adolescents in Niteroi, Rio De Janeiro, Brazil** by Rosendo Da Silva, Rosane Carla, Phd from Michigan State University, 1998, 323 pages
  http://wwwlib.umi.com/dissertations/fullcit/9922369
- **Coronary Heart Disease Risk Factors in Newfoundland Children** by Balram, Bodhnarine Christofer Maraj; Phd from Memorial University of Newfoundland (canada), 1982
  http://wwwlib.umi.com/dissertations/fullcit/NK57250
- **Coronary Heart Disease, Jungian Psychological Traits and a College Based Primary Care Paradigm** by Roberts, Ernst Edward, Ii, Edd from Texas Tech University, 1987, 253 pages
  http://wwwlib.umi.com/dissertations/fullcit/8717338
- **Correlates and Risk Factors Associated with Coronary Heart Disease and the Likelihood of Engaging in Selected Nutrition Health-promoting Behaviors among Women** by Covington, Carolyn Frances; Phd from Howard University, 2002, 241 pages
  http://wwwlib.umi.com/dissertations/fullcit/3066487
- **Depression As a Predictor of Ischemic Heart Disease** by Rowan, Paul John; Phd from The University of Alabama, 2002, 173 pages
  http://wwwlib.umi.com/dissertations/fullcit/3067307
- **Development and Evaluation of a School Curriculum on Nutrition and Heart Disease for Young Adolescents** by Bottom, Julia Stewart, Phd from Colorado State University, 1987, 178 pages
  http://wwwlib.umi.com/dissertations/fullcit/8725618
- **Development and Significance of Cardiac Myosin Autoimmunity Induced by Trypanosoma Cruzi Infection in Experimental Acute Chagas Heart Disease** by Leon, Juan Shedan; Phd from Northwestern University, 2003, 231 pages
  http://wwwlib.umi.com/dissertations/fullcit/3087937
- **Diet and Heart Disease: a Political Case Study** by Mills, Michael P., Phd from University of Essex (united Kingdom), 1989, 348 pages
  http://wwwlib.umi.com/dissertations/fullcit/D-92382

- **Diet, Tobacco, Alcohol and Stress As Causes of Coronary Heart Disease: a Longitudinal Causal Analysis (polyunsaturated Fats, Risk Factors, Cholesterol)** by Lynch, Wendy Diane, Phd from University of Colorado at Boulder, 1986, 173 pages
http://wwwlib.umi.com/dissertations/fullcit/8700370
- **Differences in Knowledge of Coronary Heart Disease Risks and Modifiable Risk Factor Behaviors by Gender, Age, and Ethnicity** by Reitz, Sharon Margaret, Phd from University of Maryland College Park, 1997, 333 pages
http://wwwlib.umi.com/dissertations/fullcit/9736622
- **Do Women with Heart Disease Experience More Distress Than Men?** by Woodend, Anne Kirsten; Phd from University of Toronto (canada), 2002, 288 pages
http://wwwlib.umi.com/dissertations/fullcit/NQ69063
- **Effects of Coronary Heart Disease Risk Factor Reduction on Exercise Performance in Men at High Risk of Future Coronary Heart Disease** by Ringhofer, Kevin Richard, Phd from University of Minnesota, 1991, 142 pages
http://wwwlib.umi.com/dissertations/fullcit/9122205
- **Family Based Investigation of the Genetic Basis of Ischaemic Heart Disease** by Spence, Mark S. Md from Queen's University of Belfast (northern Ireland), 2002, 257 pages
http://wwwlib.umi.com/dissertations/fullcit/f661921
- **Family Response to the Crisis of Coronary Heart Disease** by Dhooper, Surjit Singh Bachint Singh, Phd from Case Western Reserve University, 1982, 232 pages
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- **Health Behavior Change in Women with Coronary Heart Disease: a Phenomenological Study** by Katz, Laurie Baskin; Phd from Alliant International University, Fresno, 2002, 283 pages
http://wwwlib.umi.com/dissertations/fullcit/3043006
- **Health-related Beliefs and Behaviors of Adolescents with and without Congenital Heart Disease** by Burgess, Erika Swift; Phd from Case Western Reserve University, 2002, 116 pages
http://wwwlib.umi.com/dissertations/fullcit/3058329
- **Heart Disease among Middle-aged and Elderly Persons in the United States: Trends and a Multistate Model** by Ueda, Hiroshi; Phd from University of Southern California, 2000, 219 pages
http://wwwlib.umi.com/dissertations/fullcit/3018138
- **Heart Disease Beliefs of Urban Elderly African-american Women** by Ward, Velma Lewis, Phd from Wayne State University, 1996, 255 pages
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- **Hemodynamic Determinants of Exercise Capacity in Heart Disease: a Path-analytic Model** by Myers, Jonathan, Phd from University of Southern California, 1991
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- **Non-high-density Lipoprotein Cholesterol and Coronary Heart Disease** by Liu, Jian; , Phd from State University of New York at Buffalo, 2002, 252 pages
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- **Opposing Cardioprotective Effects of Delta and Epsilonpkc: Therapeutic Peptides for the Treatment of Ischemic Heart Disease** by Chen, Leon Ernest; Phd from Stanford University, 2002, 133 pages
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- **Parents' Reaction to the Diagnosis of Congenital Heart Disease in Infants** by Cohn, Joan Kirschenbaum, Dsw from Adelphi University, School of Social Work, 1986, 123 pages
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- **Patient Socioeconomic Status and Treatment of Heart Disease in the Emergency Room** by Nordquist, Gigi; Phd from Case Western Reserve University, 2002, 128 pages
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- **Proinsulin and Insulin Sensitivity As Predictors for Type 2 Diabetes Mellitus and Coronary Heart Disease: Clinical Epidemiological Studies with Up to 27 Years of Follow-up** by Zethelius, Bjorn; Md from Uppsala Universitet (sweden), 2003, 67 pages
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- **Psychosocial Risk Factors of Heart Disease in Older Hispanic Women** by Martinez, Sylvia L. Phd from Alliant International University, Los Angeles, 2002, 174 pages
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- **Psychosocial Variables, Type a Behavior Pattern, and Coronary Heart Disease in Women** by Low, Kathryn Graff, Phd from Stanford University, 1991, 202 pages
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CHAPTER 5. CLINICAL TRIALS AND HEART DISEASE

Overview

In this chapter, we will show you how to keep informed of the latest clinical trials concerning heart disease.

Recent Trials on Heart Disease

The following is a list of recent trials dedicated to heart disease.[8] Further information on a trial is available at the Web site indicated.

- **Body Water Content in Cyanotic Congenital Heart Disease**

  Condition(s): Heart Defects, Congenital

  Study Status: This study is currently recruiting patients.

  Sponsor(s): National Center for Research Resources (NCRR)

  Purpose - Excerpt: Adults with cyanotic congenital heart disease have elevated levels of plasma proatrial natruretic peptide (proANP) which most likely results in chronic dehydration, leading to reduced oxygen transport to tissues and shortness of breath with activity. The purpose of this study is to characterize adults with cyanotic congenital heart defects with respect to their body composition (water and fat-free mass) and resting metabolic rates. The study consists of several measures of how much body water, fat and lean tissue a subject has, and measures the number of calories the subject's body uses at rest. Adult subjects with cyanotic congenital heart disease will be recruited along with healthy noncyanotic control subjects matched for age, gender, and body weight.

  Phase(s): Phase I

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00000107

[8] These are listed at **www.ClinicalTrials.gov**.

- **Cardio Vascu-Grow for treatment of coronary heart disease**

  Condition(s): Coronary Disease; Coronary Heart Disease; Myocardial Ischemia; Coronary Arteriosclerosis

  Study Status: This study is currently recruiting patients.

  Sponsor(s): Cardio Vascular Genetic Engineering; Cardiovascular Clinical Research

  Purpose - Excerpt: Treatment for no-options heart patients with coronary artery disease. Procedure involves the injection into the heart of a protein growth factor that stimulates the growth of new blood vessels around blocked coronary arteries.

  Phase(s): Phase I; Phase II

  Study Type: Interventional

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00032318

- **Effects of Meditation on Mechanism of Coronary Heart Disease**

  Condition(s): Coronary Heart Disease

  Study Status: This study is currently recruiting patients.

  Sponsor(s): National Center for Complementary and Alternative Medicine (NCCAM)

  Purpose - Excerpt: To study the effects of Transcendental Meditation on Coronary Heart Disease

  Phase(s): Phase II

  Study Type: Interventional

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00010738

- **Evaluation of Patients with Heart Disease Not Eligible for Research Protocols**

  Condition(s): Heart Disease

  Study Status: This study is currently recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: An important mission of the National Institutes of Health (NIH) is to develop and carryout research studies designed to improve understanding of disease processes and treatments. Clinical research continues to become more focused on specific diseases and the signs and symptoms of diseases in specific groups of people and patients. This study is designed to permit inpatient evaluation and care of patients with heart disease who do not qualify to participate in research studies being conducted by the Cardiology Branch of the National Heart, Lung, and Blood Institute (NHLBI). These patients are valuable to the Cardiology Branch of the NHLBI because they help to improve training and experience of its researchers.

  Study Type: Interventional

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00001458

- **Evaluation of Patients with Known or Suspected Heart Disease**

  Condition(s): Chest Pain; Coronary Disease; Heart Disease; Hypertrophic Cardiomyopathy; Syndrome X

  Study Status: This study is currently recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: In this study researchers will admit and evaluate patients with known or suspected heart disease referred to the Cardiology Branch of the National Heart, Lung, and Blood Institute (NHLBI). Patients participating in this study will undergo a general medical evaluation, including blood tests, urine, examination, chest x-ray and electrocardiogram (EKG). In addition, patients may be asked to have an echocardiogram (ultrasound scan of the heart) and to perform an exercise stress test. These tests are designed to assess the types and causes of patient's heart diseases and to determine if they can participate in other, specific research studies.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00001313

- **Pediatric Heart Disease Clinical Research Network**

  Condition(s): Arrhythmia; Defect, Congenital Heart; Pediatric Heart Disease; Kawasaki Disease

  Study Status: This study is currently recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To evaluate novel treatment methods and management strategies to benefit children with structural congenital heart disease, inflammatory heart disease, heart muscle diseases, and arrhythmia.

  Study Type: Interventional

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00023517

- **Behavioral Factors in Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To elucidate the role of biobehavioral factors in the etiology, pathogenesis and course of coronary heart disease (CHD) and to use this knowledge to devise more effective prevention, treatment and rehabilitation approaches.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005314

- **Enhancing Recovery in Coronary Heart Disease (ENRICHD) Patients**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Depression; Heart Diseases; Myocardial Infarction; Myocardial Ischemia

Study Status: This study is no longer recruiting patients.

Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

Purpose - Excerpt: To evaluate the effect of psychosocial intervention on mortality and reinfarction in coronary heart disease patients at high psychosocial risk.

Phase(s): Phase III

Study Type: Interventional

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00000557

- **Genetic Architecture of Heart Disease in Rural Brazil**

  Condition(s): Chagas Disease; Heart Diseases; Arrhythmia; Heart Failure, Congestive

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine the genetics of human susceptibility to Chagas' disease, a leading cause of heart disease throughout Latin America.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00023556

- **Genetic Epidemiology of Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To relate observations at the DNA level to the distribution of coronary heart disease in the population at large.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005209

- **Mental Stress, Autonomic Function, and Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine the vagal, vascular sympathetic, and mechno-structural components of baroreflex regulation in coronary artery disease (CAD) patients and healthy age-matched controls during rest and acute laboratory stress.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005524

- **Risk of Coronary Heart Disease in Women with Polycystic Ovary Syndrome**

  Condition(s): Atherosclerosis; Cardiovascular Diseases; Heart Diseases; Carotid Artery Diseases; Coronary Disease; Polycystic Ovary Syndrome

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To investigate whether women with Polycystic Ovary syndrome (PCOS) have evidence of an increased prevalence rate of subclinical atherosclerosis as measured by the presence of plaque, increased intima-medial carotid artery wall thickness and lower brachial artery flow mediated vasodilation.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005459

- **Subclinical Heart Disease in Insulin-Dependent Diabetes**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease; Diabetes Mellitus; Diabetes Mellitus, Insulin-Dependent

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To investigate the occurrence and associated risk factors for subclinical heart disease in persons with insulin-dependent diabetes mellitus (IDDM).

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005754

- **VLDL and LDL Particle Types as Coronary Heart Disease Risk Factors**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease; Myocardial Infarction; Diabetes Mellitus; Diabetes Mellitus, non-insulin dependent

  Study Status: This study is no longer recruiting patients.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To evaluate very low density lipoprotein (VLDL) and low density lipoprotein (LDL) particle types as predictors of initial coronary events.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00065793

- **AIDS-Associated Heart Disease -- Incidence and Etiology**

  Condition(s): Heart Diseases; Acquired Immunodeficiency Syndrome; Myocardial Diseases; HIV Infections

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To detect by Doppler echocardiography the incidence of cardiac abnormalities in HIV-positive patients in a prospective, longitudinal study.

Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00005228

- **Anger Expression, Self-focus and Coronary Heart Disease Risk Factors**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To identify behavioral factors underlying the development of cardiovascular risk in young adults.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005244

- **Apolipoprotein Polymorphisms and Risk of Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Arteriosclerosis; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine the relative risk in a defined population of angiographically demonstrated coronary artery disease due to genetic polymorphisms at the four apolipoprotein genomic regions.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005225

- **Change in Coronary Heart Disease Risk Factors in Young Adults**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To prospectively examine changes in lipids, lipoproteins, and blood pressure among 295 young adults who had been followed for sixteen years in the Beaver County Lipid Study. The Beaver County Lipid Study tracked the cholesterol values of 295 children, ages 11 to 14 at baseline.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005406

- **Chronic Stress as a Risk Factor in the Etiology of Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases

  Study Status: This study is completed.

Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

Purpose - Excerpt: To conduct a prospective, longitudinal, analysis of the psychophysiological effects of chronic exposure to environmental stress. The study took advantage of a unique, naturally occurring experiment caused by the relocation of a major international airport.

Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00005352

- **Coronary Heart Disease Incidence, Mortality, and Risk Factor Relationships**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases; Myocardial Infarction; Angina Pectoris

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine the incidence, secular trends, and outcomes of coronary heart disease in the population of Rochester, Minnesota.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005148

- **Coronary Heart Disease Risk Factors in Blacks Vs Whites**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To use data from the Epidemiologic Follow-up Study of the National Health and Nutrition Examination Survey (NHANES) to develop models to predict morbidity and mortality as a function of risk factors for coronary artery disease. Bivariate dependent survival distributions were examined.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005431

- **Coronary Heart Disease Risk Factors in Upwardly Mobile Blacks and Whites**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Hypertension; Heart Diseases; Obesity

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To evaluate the relation between blood pressure and socioeconomic status, electrolyte intake, obesity and psychosocial factors in Black and white students. Also, to compare blood pressure, cardiovascular risk factors, sodium and potassium excretion in United States Blacks with West African Blacks.

  Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00005175

- **Endogenous Estrogen and Coronary Heart Disease in Women**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases; Myocardial Infarction; Postmenopause

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To investigate the relation between endogenous levels of estrogen in postmenopausal women and the subsequent development of coronary heart disease.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005446

- **Epidemiology of Coronary Heart Disease in Blacks**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease; Myocardial Infarction

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To conduct an analysis of the epidemiology of coronary heart disease (CHD) in Blacks using data collected from the 'Survival and Ventricular Enlargement (SAVE) Following Myocardial Infarction' study.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005410

- **Epidemiology of Coronary Heart Disease in Men Aged 40 and Over**

  Condition(s): Atherosclerosis; Coronary Arteriosclerosis; Coronary Disease; Heart Diseases; Cardiovascular Diseases

  Study Status: This study is not yet open for patient recruitment.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine whether the prevalence of subclinical coronary and aortic atherosclerotic disease is different among Japanese in Japan, Japanese in Hawaii, and black and white Americans.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00069797

- **Epidemiology of Decline in Heart Disease Mortality**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

Purpose - Excerpt: To continue a study of premature coronary heart disease mortality among men and women aged 35-54 in Allegheny County, Pennsylvania.

Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00005162

- **Epidemiology of Insulin and Dehydroepiandrosterone Sulfate and Coronary Heart Disease Mortality**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine whether serum insulin is a risk factor for coronary heart disease morbidity and mortality and whether dehydroepiandrosterone sulfate (DHEAS) is a risk factor for coronary heart disease mortality. Also, to ascertain the determinants of serum insulin levels among middle-aged men.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005246

- **Evaluation and Treatment of Heart Disease in Patients not Participating in Research**

  Condition(s): Arrhythmia; Congenital Heart Defect; Heart Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: There is an important need to maintain clinical skills, provide quality training and expertise, and provide an environment that stimulates ideas for clinical research. This study permits inpatient evaluation and management of patients with heart disease who do not qualify to participate in studies currently being conducted by the Cardiology Branch of the National Heart, Lung, and Blood Institute.

  Study Type: Interventional

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00001460

- **Genetic Epidemiology of Coronary Heart Disease Risk in Women Twins**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine genetic and environmental influences on several recently identified coronary heart disease risk factors in identical and fraternal adult women twins. The risk factors included low density lipoprotein (LDL) subclass patterns, plasma apolipoprotein levels, body fat distribution, and serum insulin levels.

  Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00005239

- **Green Tea Consumption and Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine the prospective association of green tea consumption to the incidence and mortality of coronary heart disease (CHD) using multivariate analysis while controlling for the potential confounding effects of cholesterol, triglycerides, glucose and dietary nutrients.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005548

- **Heart Disease and the Black Health Disadvantage**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To utilize national population data sets prepared by the National Center for Health Statistics, to (1) examine the current Black disadvantage in cardiovascular (CV) health, (2) explore potential clinical and epidemiologic causes, (3) incorporate emerging knowledge of new risk factors and (4) compare trends in medical treatment and risk factors for the four sex-race groups.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005403

- **HIV-Associated Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Myocardial Diseases; Acquired Immunodeficiency Syndrome; HIV Infections

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To develop natural history data regarding the incidence, clinical course, prognosis, and effects of treatment with anti-viral and immunosuppressive agents on HIV-associated heart disease. A second part of the study evaluated a number of possible mechanisms underlying the development of HIV heart disease.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005229

- **Hostility and Pathogenic Mechanisms of Coronary Heart Disease in Women**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine the combined effects of hostility, harassment, lipids, and oral contraceptive (0C) use on physiological responses in young and middle-aged premenopausal women.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005435

- **Hyperapo B and Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases; Diabetes Mellitus; Obesity; Hypercholesterolemia, Familial

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine the role of apolipoprotein B and apolipoprotein A1 in the etiology of coronary artery disease.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005168

- **Immunogenetic Factors of Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases; Hypertension; Obesity; Diabetes Mellitus; Hypothyroidism

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To assess the association of immunogenetic factors with onset of coronary heart disease and the interrelationship of these factors with standard coronary heart disease risk factors.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005184

- **Implementation of Ischemic Heart Disease Clinical Practice Guidelines**

  Condition(s): Myocardial Ischemia; Heart Disease; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): Department of Veterans Affairs; Department of Veterans Affairs Health Services Research and Development Service; Department of Veterans Affairs - Veterans Integrated Services Network

  Purpose - Excerpt: The purpose of this study is to design and evaluate targeted implementation strategies to fully integrate the VHA clinical practice guidelines for

ischemic heart disease into VHA clinical practice. Effectively implementing the guideline will enhance the quality, appropriateness, timeliness, and cost effectiveness of care delivered to veterans with ischemic heart disease. The long-term objective of this study is to identify the best strategies for implementing the IHD guidelines to improve guideline adherence and provider acceptance. The specific objectives of the study are to: (1) describe the temporal aspects of guideline acceptance and adherence, over three periods of time: pre-implementation, post implementation of general strategies, and post- implementation of targeted strategies; (2) assess the relative effectiveness of targeted intervention strategies on guideline adherence; (3) identify the relationships among provider beliefs, attitudes, and their intentions to use guidelines; (4) identify the costs associated with implementation of general and targeted implementation strategies; and (5) describe provider satisfaction with targeted implementation strategies. Qualitative (focus groups, interviews with key informants) and quantitative methods (surveys, chart reviews) are used to address study objectives. A survey will be sent to providers to measure beliefs and attitudes that predict provider acceptance to clinical practice guidelines. Patient level data will be collected from reviews of charts from each facility. Based on provider focus groups we will design targeted strategies to overcome system barriers and address needs identified by providers to support clinical practice guideline implementation. A randomized trial will be conducted to assess the relative effectiveness of the targeted intervention strategies.

Study Type: Interventional

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00013104

- **Ischemic Heart Disease Incidence and Indices of Body-fat Distribution**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases; Myocardial Ischemia; Obesity

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine the association between ischemic heart disease incidence and anthropometric indices of body-fat distribution.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005255

- **Lipoprotein Subfractions and Coronary Heart Disease During 25 Year Follow-up**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine the relationship of fatal and nonfatal coronary heart disease to lipoprotein fractions and other risk factors in a prospective epidemiologic study conducted between 1954 and 1957.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005215

- **Mechanisms Underlying Psychosocial Associations with Ischemic Heart Disease (Kuopio)**

  Condition(s): Cardiovascular Diseases; Carotid Artery Diseases; Arrhythmia; Myocardial Ischemia; Thrombosis; Heart Diseases; Atherosclerosis

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine the relationships among psychosocial factors and carotid atherosclerosis, myocardial ischemia, arrhythmias, and thrombosis.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005260

- **Multiple Risk Factor Intervention Trial for the Prevention of Coronary Heart Disease (MRFIT)**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Heart Diseases; Myocardial Ischemia

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To determine for a group of men at high risk of death from coronary heart disease whether a special intervention program to lower serum cholesterol, reduce blood pressure, and eliminate cigarette smoking would result in a significant reduction in mortality from coronary heart disease.

  Phase(s): Phase III

  Study Type: Interventional

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00000487

- **Natural History of Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Coronary Disease; Myocardial Infarction; Heart Diseases; Death, Sudden, Cardiac; Heart Failure, Congestive; Heart Failure

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To examine the natural history of mortality due to coronary heart disease in post-myocardial infarction patients from the Beta-Blocker Heart Attack Trial (BHAT) and the Aspirin Myocardial Infarction Study (AMIS).

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005265

- **Outpatient Evaluation of Patients with Known or Suspected Heart Disease**

  Condition(s): Heart Disease

  Study Status: This study is completed.

Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

Purpose - Excerpt: This study is designed to screen patients with heart diseases who may qualify to participate in other research studies being conducted by the Cardiology Branch of the National Heart, Lung, and Blood Institute (NHLBI). Patients participating in this research study will be seen on an outpatient basis and undergo a general medical evaluation, including blood tests, urine, examination, chest x-ray and electrocardiogram (EKG). In addition, patients may be asked to have an echocardiogram (ultrasound scan of the heart) and to perform an exercise stress test. Patients participating in this study will not receive any investigational treatments.

Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00001400

- **Physical Activity, Hypertension, Diabetes, and Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Diabetes Mellitus; Coronary Disease; Hypertension; Diabetes Mellitus, non-insulin dependent

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To study the influences of physical activity on the incidence of hypertension, non-insulin-dependent diabetes (NIDDM), and coronary heart disease (CHD), taking into account the influences of other life-style elements such as body size, cigarette habit, alcohol consumption habits, and parental history of disease on these same chronic diseases.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005419

- **Psychosocial Risk Factors for Coronary Heart Disease in Swedish Women**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To study psychosocial risk factors for coronary heart disease in Swedish women.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005691

- **Quality of Life in Patients with Chronic Ischemic Heart Disease**

  Condition(s): Ischemic Heart Disease; Myocardial Ischemia

  Study Status: This study is completed.

  Sponsor(s): Warren G Magnuson Clinical Center (CC)

  Purpose - Excerpt: This study will examine the health-related quality of life in patients being treated for chronic heart disease. Patients 18 years or older with chronic ischemic

heart disease and left ventricular dysfunction enrolled in protocols in the National Heart Lung and Blood Institute's Cardiology Branch may participate in this study. Participants will complete five questionnaires at 3 separate times during the study-once during hospitalization at the NIH Clinical Center and again at home 6 months and 1 year later. The questionnaires, described below, require a total of about 30 minutes to complete. 1. Demographic Information Sheet -General information such as age, marital status, employment, education, and history of cardiac medical procedures. 2. General Health Survey -Patient's self-assessment, on a rating scale, of physical and emotional well being. Questions are related to the ability to perform work and daily living activities, mood and state of mind, limitations on social activities, energy level, pain level, general quality of life, etc. 3. Heart Disease Survey - Patient's self-assessment, on a rating scale, of the level of physical, social, emotional and functional well being related to his or her heart condition. Questions concern fatigue level, emotional outlook, social well being, etc. 4. Angina Survey - Information on the frequency of chest pain, chest tightness, or angina. 5. Symptom Distress Survey - Patient's ranking of the degree of symptom distress from chest discomfort, difficulty breathing, heart rate irregularities, wheezing and coughing. All information provided in the questionnaires will be kept confidential. Upon request, patients will be sent a summary of the study results when the study is completed.

Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00005671

- **Re-evaluating Triglycerides in Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease; Hypertriglyceridemia

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To conduct a comprehensive epidemiologic investigation into the relationship between serum triglyceride (TG) levels and coronary heart disease (CHD).

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005442

- **Screening for Inherited Heart Disease**

  Condition(s): Heart Disease; Hypertrophic Cardiomyopathy

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: Genetically inherited heart diseases like hypertrophic cardiomyopathy (HCM) are conditions affecting the heart passed on to family members by abnormalities in genetic information. These conditions are responsible for many heart related deaths and illnesses. Presently, there are several research studies being conducted in order to improve the understanding of disease processes and symptoms associated with genetically inherited heart diseases. This study is designed to determine the eligibility of patients diagnosed with or suspected to have inherited heart disease to participate in these research studies.

Study Type: Observational

Contact(s): see Web site below

Web Site: http://clinicaltrials.gov/ct/show/NCT00001746

- **Signs and Symptoms Associated with Molecular Defects in Genetically Inherited Heart Disease**

  Condition(s): Congenital Heart Defect

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: Genetically inherited heart diseases (familial cardiopathies) are conditions affecting the heart passed on to family members by abnormalities in genetic information. These conditions are responsible for many heart related deaths and illnesses. Researchers are interested in learning more about the specific genetic abnormalities causing heart diseases. In addition, they would like to find out how these abnormal genes can contribute to the development of other medical problems. In order to do this, researchers plan to study patients and family members of patients diagnosed with genetically inherited heart disease. Those people participating in the study will undergo a variety of tests including blood tests, echocardiograms, and magnetic resonance imaging studies (MRI). These tests will be used to help researchers find the genetic problem causing the familial cardiopathy. Researchers hope that the information gathered from this study can be used to develop better medical care through early diagnosis, management, and treatment plans.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00027196

- **Speech Characteristics in Coronary Heart Disease**

  Condition(s): Cardiovascular Diseases; Heart Diseases; Coronary Disease

  Study Status: This study is completed.

  Sponsor(s): National Heart, Lung, and Blood Institute (NHLBI)

  Purpose - Excerpt: To improve the predictive validity of Structured Interview assessments of Type A behavior by comparing interviewer techniques in the Multiple Risk Factor Intervention Trial (MRFIT) and the Western Collaborative Group Study (WCGS). To assess whether there were interviewer differences in the predictiveness of Type A behavior and its components for coronary heart disease incidence in MRFIT and WCGS and if so, to assess whether the interviewer differences in disease predictiveness were related to interviewer stylistics.

  Study Type: Observational

  Contact(s): see Web site below

  Web Site: http://clinicaltrials.gov/ct/show/NCT00005157

Keeping Current on Clinical Trials

The U.S. National Institutes of Health, through the National Library of Medicine, has developed ClinicalTrials.gov to provide current information about clinical research across the broadest number of diseases and conditions.

The site was launched in February 2000 and currently contains approximately 5,700 clinical studies in over 59,000 locations worldwide, with most studies being conducted in the United States. ClinicalTrials.gov receives about 2 million hits per month and hosts approximately 5,400 visitors daily. To access this database, simply go to the Web site at **http://www.clinicaltrials.gov/** and search by "heart disease" (or synonyms).

While ClinicalTrials.gov is the most comprehensive listing of NIH-supported clinical trials available, not all trials are in the database. The database is updated regularly, so clinical trials are continually being added. The following is a list of specialty databases affiliated with the National Institutes of Health that offer additional information on trials:

- For clinical studies at the Warren Grant Magnuson Clinical Center located in Bethesda, Maryland, visit their Web site: **http://clinicalstudies.info.nih.gov/**
- For clinical studies conducted at the Bayview Campus in Baltimore, Maryland, visit their Web site: **http://www.jhbmc.jhu.edu/studies/index.html**
- For cancer trials, visit the National Cancer Institute: **http://cancertrials.nci.nih.gov/**
- For eye-related trials, visit and search the Web page of the National Eye Institute: **http://www.nei.nih.gov/neitrials/index.htm**
- For heart, lung and blood trials, visit the Web page of the National Heart, Lung and Blood Institute: **http://www.nhlbi.nih.gov/studies/index.htm**
- For trials on aging, visit and search the Web site of the National Institute on Aging: **http://www.grc.nia.nih.gov/studies/index.htm**
- For rare diseases, visit and search the Web site sponsored by the Office of Rare Diseases: **http://ord.aspensys.com/asp/resources/rsch_trials.asp**
- For alcoholism, visit the National Institute on Alcohol Abuse and Alcoholism: **http://www.niaaa.nih.gov/intramural/Web_dicbr_hp/particip.htm**
- For trials on infectious, immune, and allergic diseases, visit the site of the National Institute of Allergy and Infectious Diseases: **http://www.niaid.nih.gov/clintrials/**
- For trials on arthritis, musculoskeletal and skin diseases, visit newly revised site of the National Institute of Arthritis and Musculoskeletal and Skin Diseases of the National Institutes of Health: **http://www.niams.nih.gov/hi/studies/index.htm**
- For hearing-related trials, visit the National Institute on Deafness and Other Communication Disorders: **http://www.nidcd.nih.gov/health/clinical/index.htm**
- For trials on diseases of the digestive system and kidneys, and diabetes, visit the National Institute of Diabetes and Digestive and Kidney Diseases: **http://www.niddk.nih.gov/patient/patient.htm**
- For drug abuse trials, visit and search the Web site sponsored by the National Institute on Drug Abuse: **http://www.nida.nih.gov/CTN/Index.htm**

- For trials on mental disorders, visit and search the Web site of the National Institute of Mental Health: **http://www.nimh.nih.gov/studies/index.cfm**
- For trials on neurological disorders and stroke, visit and search the Web site sponsored by the National Institute of Neurological Disorders and Stroke of the NIH: **http://www.ninds.nih.gov/funding/funding_opportunities.htm#Clinical_Trials**

CHAPTER 6. PATENTS ON HEART DISEASE

Overview

Patents can be physical innovations (e.g. chemicals, pharmaceuticals, medical equipment) or processes (e.g. treatments or diagnostic procedures). The United States Patent and Trademark Office defines a patent as a grant of a property right to the inventor, issued by the Patent and Trademark Office.[9] Patents, therefore, are intellectual property. For the United States, the term of a new patent is 20 years from the date when the patent application was filed. If the inventor wishes to receive economic benefits, it is likely that the invention will become commercially available within 20 years of the initial filing. It is important to understand, therefore, that an inventor's patent does not indicate that a product or service is or will be commercially available. The patent implies only that the inventor has "the right to exclude others from making, using, offering for sale, or selling" the invention in the United States. While this relates to U.S. patents, similar rules govern foreign patents.

In this chapter, we show you how to locate information on patents and their inventors. If you find a patent that is particularly interesting to you, contact the inventor or the assignee for further information. **IMPORTANT NOTE:** When following the search strategy described below, you may discover non-medical patents that use the generic term "heart disease" (or a synonym) in their titles. To accurately reflect the results that you might find while conducting research on heart disease, we have not necessarily excluded non-medical patents in this bibliography.

Patents on Heart Disease

By performing a patent search focusing on heart disease, you can obtain information such as the title of the invention, the names of the inventor(s), the assignee(s) or the company that owns or controls the patent, a short abstract that summarizes the patent, and a few excerpts from the description of the patent. The abstract of a patent tends to be more technical in nature, while the description is often written for the public. Full patent descriptions contain much more information than is presented here (e.g. claims, references, figures, diagrams, etc.). We will tell you how to obtain this information later in the chapter. The following is an

[9] Adapted from the United States Patent and Trademark Office: **http://www.uspto.gov/web/offices/pac/doc/general/whatis.htm**.

example of the type of information that you can expect to obtain from a patent search on heart disease:

- **Acetylsalicylic acid and micronutrient supplementation for nutritional losses and coronary heart disease**

  Inventor(s): Christakis; George (Sunrise, FL), Riley; Patricia A. (Sunrise, FL)

  Assignee(s): Medical Doctor's Research Institute, Inc. (Sunrise, FL)

  Patent Number: 5,948,443

  Date filed: February 21, 1997

  Abstract: The present invention pertains generally to the field of Public Health, including the prevention and treatment of coronary heart disease which is currently the first cause of death in the American population. More specifically, the present invention concerns a total modular system of multivitamin and mineral supplementation composed of 7 distinct modules for improving public health by insuring adequate intake of micronutrients needed for disease prevention and protection against nutritional losses and deficiencies due to, for example, lifestyle factors and common inadequate dietary patterns. A module, as used herein throughout, is defined as a separate and distinct combination of vitamin-mineral and other health promoting compounds which are directed to specific target populations. The formulations of the present invention which, when combined in one capsule or tablet or as separate modules, exert a joint and enhancing effect on the major pathogenetic factors involved in the atherosclerotic process. Moreover, certain modular formulations of the present invention incorporate both antioxidants and acetylsalicylic acid (aspirin) as a single preventive modality. Such a combination of antioxidants and aspirin is believed to act to prevent oxidation of low density lipoproteins within coronary arterial walls and to cause platelet deagluttination thereby inhibiting thrombus formation. The benefit of preventing these two major processes is believed to reduce the risk of coronary heart disease.

  Excerpt(s): The present invention concerns a total modular system of multivitamin and mineral supplementation composed of 7 distinct modules for improving public health by insuring adequate intake of micronutrients needed for disease prevention and protection against nutritional losses and deficiencies due to, for example, lifestyle factors and common inadequate dietary patterns. A module, as used herein throughout, is a separate and distinct combination of vitamin-mineral and other health promoting compounds which are directed to specific target populations. Micronutrients are elements or compounds which are present in foods in small or trace amounts and includes vitamins, minerals or other elements; and compounds found in foods for which a Recommended Dietary Allowance (RDA) has not yet been determined (pantothenic acid, biotin, choline, etc.). The macronutrients consist of carbohydrates, fats and proteins which supply nutrients and calories. Some elements such as calcium, sodium, potassium, chloride and phosphorus are elements consumed in relatively large amounts, while many such as iron, iodine, and zinc are consumed in small amounts (milligrams). Vitamins such as vitamin B12, and folic acid and the minerals copper, selenium and chromium are consumed in very small, or trace amounts (micrograms). Inasmuch as the human body, does not synthesize many "essential compounds", these specific vitamins and minerals can be obtained from only two sources: foods and supplements. The primary source of all nutrients is food. Over the past four decades, ample evidence documents that major portions of various subgroups of Americans stratified by age, gender, socioeconomic status and other variables, cannot meet the "Recommended Dietary Allowances" of the foods containing these essential compounds

and elements. Thus vitamin and mineral supplementation has become a recognized method of meeting accepted medical and public health nutrition standards. The enrichment of bread with iron and B vitamins in 1940 is considered a major factor in assuring favorable nutritional status for the general population of that time, and has been retained as a major advance for public health. In 1993, the Interagency Board of Nutrition Monitoring and Related Research reported that women did not meet the RDA's of 6 out of 15 micronutrients: B6, E, calcium, iron, magnesium and zinc. Men also failed to met the RDA's for 4 of 15 micronutrients: B6, E, magnesium and zinc. Their findings reveal significant prevalence and incidence of various population subgroups deficient in specific vitamins and minerals. The importance of these findings relate to the prevention of micronutrient deficiency diseases such as scurvy (vitamin C deficiency), pellagra (niacin deficiency), beri-beri (vitamin B1 deficiency), iron deficiency anemia and other vitamin and mineral deficiency states. The effect of marginal deficiency states is only now being considered as a cause of suboptimal health status. Moreover, research conducted and published in the past three decades indicates that antioxidant micronutrients are involved in preventing molecular biological processes affecting health and disease at the subcellular and submolecular level. It is current thought that free-radical effects on cells and tissues can be modified by antioxidant micronutrients reducing cellular damage. Specific micronutrients maintain immune system integrity, moderate the aging process, and play a role in the prevention of atherogenesis and cancer. Furthermore, substantial segments of the population do not manifest desirable eating patterns, that is, an adequate intake in both the quantity and variety of food to fulfill the Recommended Dietary Allowances, as indicated by recent government survey. Only 22% of the subjects of a National Cancer Institute Study, consumed the recommended number of dietary servings of fruits and vegetables.

Web site: http://www.delphion.com/details?pn=US05948443__

- **Antianginal film and method of treating ischemic heart disease**

  Inventor(s): Davydov; Anatoly B. (Moscow, SU), Piotrovsky; Vladimir K. (Moscow, SU), Metelitsa; Vladimir I. (Moscow, SU), Khromov; Gennady L. (Moscow, SU), Utyamyshev; Rustam I. (Moscow, SU), Novikova; Elizaveta B. (Moscow, SU), Savvateev; Konstantin L. (Moscow, SU), Vikhert; Anatoly M. (Moscow, SU), Gerasimova; Galina A. (Moscow, SU), Babaian; Eduard A. (Moscow, SU)

  Assignee(s): Vsesojuny Kardiologichesky Nauchny Tsentr Adkaemii Meditsinski Nauk SSSR (Moscow, SU), Vsesojuzny Nauchno-Issledovatelsky I Ispytateiny Institut Meditsinskoi (Moscow, SU)

  Patent Number: 4,713,239

  Date filed: November 19, 1985

  Abstract: The antianginal film is a plate, 0.1 to 1.5 thick, consisting of a biologically soluble and resolvable carrier, which is a hompolymer of acrylamide or vinylpyrrolidone, or a copolymer thereof with acrylate, containing from 99 to 70 percent by weight of acrylamide with vinylpyrrolidone and from 1 to 30 percent by weight of acrylate having a molecular mass from 50,000 to 1,000,000, and the active principle having antianginal action, the components being taken in the following proportions, in percent by weight:active substance having antianginal action 3.0-30.0biologically soluble and resolvable carrier 70.0-97.0The method of treating ischemic heart disease with the proposed antianginal film by individual doses, consists in that a biologically soluble and resolvable film without any active substance is first applied to a selected site of the

mouth mucosa and the time during which this film is fully resolved is determined, this time being characteristic of the period during which the active substance is delivered into the patient's body. Next a medicinal film containing the active substance in the quantity which should be delivered into the patient's body during the time of the film resolution is selected. Finally the film containing the required quantity of the antianginal preparation is applied once or several times, to the selected site of the mouth mucosa to ensure continuous and optimal therapeutic effect during the entire period of the full resolution of the proposed film.

Excerpt(s): This invention relates to pharmacy, and more particularly it relates to a new medicinal form, a medicinal film having antianginal action, and the method of treating ischemic heart disease. Said antianginal films are used in cardiology to treat ischemic heart disease. Known in the prior art are antianginal preparations used to arrest attacks of stenocardia containing active substances such as glyceryl trinitrate, isosorbide dinitrate, pentaerythritol tetranitrate, and others. In addition to said active substances these medicinal preparations contain an inert carrier which is resolved in liquid media of the body. For example, used in the prior art are granules of glyceryl trinitrate containing sugar, starch, and other substances as filling materials (Soviet State Pharmacopoeia, 10th edition).

Web site: http://www.delphion.com/details?pn=US04713239__

- **Antianginal plate for treating ischemic heart disease**

Inventor(s): Savvateev; Konstantin L. (ulitsa Chusovskaya 15, kv. 75, Moscow, SU), Metelitsa; Vladimir I. (Khimki, Jubileiny prospekt, 35, kv. 186, Moscow, SU), Utyamyshev; Rustam I. (Prospekt Mira 118, kv. 222, Moscow, SU), Babaian; Eduard A. (ulitsa Novatorov 36, Korpus 9, kv. 12, Moscow, SU), Piotrovsky; Vladimir K. (ulitsa Akademika Skryabina, 3, korpus 1, kv. 59, Moscow, SU), Novikova; Elizaveta B. (Petrovsko-Razumovskaya alleya 18, kv. 25, Moscow, SU), Vikhert; Anatoly M. (ulitsa Burdenko 11, kv. 8-a, Moscow, SU), Khromov; Gennady L. (2 Frunzenskaya ulitsa 10, kv. 100, Moscow, SU), Davydov; Anatoly B. (ulitsa Krasny Kazanets 19, Korpus 1, kv. 283, Moscow, SU), Gerasimova; Galina A. (ulitsa Shepilovskaya 36, Korpus 2, kv. 131, Moscow, SU)

Assignee(s): none reported

Patent Number: 4,921,695

Date filed: March 9, 1989

Abstract: An antianginal composition in the form of a plate is provided for treating ischemic heart disease and is comprised of a biologically soluble and resolvable film and an antianginal coronary vasodilating nitrate. This film is placed buccally on the gum or mouth mucosa for rapid arresting or prevention of angina pectoris attacks.The antianginal film is in the form of a plate, 0.1 to 1.5 mm thick, consisting of a biologically soluble and resolvable carrier. Preferably, the film is comprised of a homoploymer of acrylamide or vinylpyrrolidone, or a copolymer thereof with acrylate, containing from 99 to 70 percent by weight of acrylamide with vinylpyrrolidone and from 1 to 30 percent by weight of acrylate having a molecular mass from 50,000 to 1,000,000, and the active principle having antianginal action, the components being taken in the following proportions, in percent by weight:

Excerpt(s): This invention relates to pharmacy, and more particularly it relates to a new medicinal form, a medicinal plate having antianginal action, and the method of treating

ischemic heart disease. Such antianginal plates are used in cardiology to treat ischemic heart disease. Known in the prior art are antianginal preparations used to arrest attacks of angina pectoris containing active substances such as glycerol trinitrate, isosorbide dinitrate, pentaerythritol tetranitrate, and others. In addition to said active substances, these medicinal preparations contain an inert carrier which is resolved in liquid media of the body. For example, used in the prior art are granules of glycerol trinitrate containing sugar, starch, and other substances as filling materials (Soviet State Pharmacopoeia, 10th edition).

Web site: http://www.delphion.com/details?pn=US04921695__

- **Chromosome 11-linked coronary heart disease susceptibility gene CHD1**

  Inventor(s): Wagner; Susanne (Murray, UT), Ding; Wei (Salt Lake City, UT), Hess; Mark A. (Salt Lake City, UT), Ballinger; Dennis G. (Menlo Park, CA)

  Assignee(s): Myriad Genetics, Inc. (Salk Lake City, UT)

  Patent Number: 6,225,451

  Date filed: March 4, 1999

  Abstract: Human coronary heart disease susceptibility gene (CHD1), some alleles of which are related to susceptibility to coronary heart disease. Germline mutations in the CHD1 gene and their use in the diagnosis of predisposition to coronary heart disease and to metabolic disorders, including hypoalphalipoproteinemia, familial combined hyperlipidemia, insulin resistant syndrome X or multiple metabolic disorder, obesity, diabetes and dyslipidemic hypertension. Presymptomatic therapy of individuals who carry deleterious alleles of the CHD1 gene (including gene therapy, protein replacement therapy, and administration of protein mimetics and inhibitors). The screening of drugs for dyslipidemic therapy.

  Excerpt(s): The present invention relates generally to the field of human genetics. The present invention specifically relates to a human coronary heart disease susceptibility gene (CHD1), some alleles of which are related to susceptibility to coronary heart disease. More specifically, the present invention relates to germline mutations in the CHD1 gene and their use in the diagnosis of predisposition to coronary heart disease and to metabolic disorders, including hypoalphalipoproteinemia, familial combined hyperlipidemia, insulin resistant syndrome X or multiple metabolic disorder, obesity, diabetes and dyslipidemic hypertension. The invention also relates to presymptomatic therapy of individuals who carry deleterious alleles of the CHD1 gene (including gene therapy, protein replacement therapy, and administration of protein mimetics and inhibitors). Also within the scope of this invention is the screening of drugs for coronary heart disease or metabolic disorder therapy.

  Web site: http://www.delphion.com/details?pn=US06225451__

- **Congenital heart disease proteins and products related thereto**

  Inventor(s): Korenberg; Julie R. (Los Angeles, CA)

  Assignee(s): Cedars-Sinai Medical Center (Los Angeles, CA)

  Patent Number: 6,040,429

  Date filed: August 30, 1999

Abstract: In accordance with the present invention, there are provided novel Congenital Heart Disease (CHD) proteins. Nucleic acid sequences encoding such proteins and assays employing same are also disclosed. The invention CHD proteins can be employed in a variety of ways, for example, for the production of anti-CHD antibodies thereto, in therapeutic compositions and methods employing such proteins and/or antibodies.

Excerpt(s): The present invention relates to nucleic acids and receptor proteins encoded thereby. Invention nucleic acids encode novel congenital heart disease related proteins. The invention also relates to methods for making and using such nucleic acids and proteins. Down syndrome (DS) usually caused by chromosome 21 trisomy and is a major cause of mental retardation and congenital heart (CHD) and gut disease affecting over 200,000 persons in the United States alone. The characteristic heart abnormalities in DS-CHD include atrioventricular canal and ventricular septal defects. Other features of DS include a set of characteristic facies, thymic abnormalities, increased risk of leukemia and early onset of Alzheimer-like dementia. Molecular analysis of rare patients with DS and partial chromosome 21 duplications and monosomies has led to the association of certain chromosomal regions with specific DS phenotypes (Korenberg et al., Proc. Natl. Acad. Sci. USA M91:4997-5001 (1994); Delebar et al., Eur. J. Human. Genet. 1:114-124 (1993); Antonarakis et al., Progress in Clinical and Biological Research 311:29-43 (1989)). Chromosome 21 is therefore a model for the study of human chromosomal aneuploidy and the construction of its physical map is of special interest. Human chromosome 21 has a nearly complete physical map with a well characterized contiguous set of overlapping YACs spanning most of its length (Chumakov et al., Nature 359:380-387 (1992); Shimizu et al., Cytogenet. Cell Genet. 70:147-182 (1995); Korenberg et al., Genome Research 5:427-443 (1995)). The demand for sequence ready contigs and clones for gene isolation efforts has prompted the construction of numerous higher resolution contigs in cosmids (Patil et al., Hum. Molec. Genet. 3:1811-1817 (1995)) and more recently in PACs (Osoegawa et al., Genomics 32:375-387 (1996)). Considerable mapping efforts exist in the region from CBR to D21S55 due to the duplication of this region in partially trisomic individuals with several phenotypic features of DS including mental retardation. However, the distal and adjacent, 4-5 Mb D21S55 to MX1 region is also of interest due to its association with Down syndrome congenital heart disease (DS-CHD) as well as other characteristic features of DS (Korenberg et al., Am. J. Hum. Genet. 50:294-302 (1992), Korenberg et al. (1994)). Although non-chimeric YACs span this interval, three are not as yet any higher resolution physical maps are available for the entire D21S55 to MX1 region. Thus, higher resolution physical maps for the entire D21S55 to MX1 region are desired. In addition, there is a need in the art for an isolated nucleic acid, and isolated protein encoded thereby, associated with congenital heart disease (CHD).

Web site: http://www.delphion.com/details?pn=US06040429__

- **Coronary heart disease treated with 17.beta.oestradiol**

  Inventor(s): Collins; Peter (Richmond, GB2)

  Assignee(s): National Heart and Lung Institute (London, GB2)

  Patent Number: 5,512,557

  Date filed: October 7, 1993

  Abstract: Use of 17.beta.-oestradiol in the treatment of coronary heart disease.

Excerpt(s): The present invention relates to the treatment of coronary heart disease and provides a pharmaceutical composition for such treatment, as well as a method for the treatment of coronary heart disease. Coronary heart disease arises from damage to the cardiac muscle, the myocardium, caused by insufficient flow of blood in the coronary arteries. The reduced flow of blood is termed myocardial ischemia, and the resulting heart damage is reflected in severe attacks of pain known as angina pectoris. The attacks of pain may be relieved or prevented using drugs, for example by sublingual administration of nitroglycerin or by the oral use of.beta.-blocking agents and calcium antagonists. Apart from relieving or preventing the angina, coronary heart disease may be treated by a combination of further methods, including the use of other drugs and the use of surgery. Coronary heart disease is particularly prevalent in women who have past the time of menopause. Furthermore, an increase in the incidence of heart conditions, including coronary artery disease, angina pectoris and vasomotor disturbance, is associated with the menopause.

Web site: http://www.delphion.com/details?pn=US05512557__

- **Coronary heart disease treated with oestradiol**

  Inventor(s): Collins; Peter (Richmond, GB)

  Assignee(s): Sterix Limited (Oxford, GB)

  Patent Number: RE36,701

  Date filed: April 29, 1998

  Abstract: Use of 17.beta.-oestradiol in the treatment of coronary heart disease.

  Excerpt(s): The present invention relates to the treatment of coronary heart disease and provides a pharmaceutical composition for such treatment, as well as a method for the treatment of coronary heart disease. Coronary heart disease arises from damage to the cardiac muscle, the myocardium, caused by insufficient flow of blood in the coronary arteries. The reduced flow of blood is termed myocardial ischemia, and the resulting heart damage is reflected in severe attacks of pain known as angina pectoris. The attacks of pain may be relieved or prevented using drugs, for example by sublingual administration of nitroglycerin or by the oral use of.beta.-blocking agents and calcium antagonists. Apart from relieving or preventing the angina, coronary heart disease may be treated by a combination of further methods, including the use of other drugs and the use of surgery. Coronary heart disease is particularly prevalent in women who have past the time of menopause. Furthermore, an increase in the incidence of heart conditions, including coronary artery disease, angina pectoris and vasomotor disturbance, is associated with the menopause.

  Web site: http://www.delphion.com/details?pn=US0RE36701__

- **Diagnostic agent for heart disease and use thereof**

  Inventor(s): Yazaki; Yoshio (Tokyo, JP), Sugi; Masahito (Choshi, JP)

  Assignee(s): Yamasa Shoyu Kabushiki Kaisha (Chiba, JP)

  Patent Number: 4,943,427

  Date filed: March 25, 1985

Abstract: A diagnostic agent for heart disease comprising a radiolabeled monoclonal antibody having specificity to cardiac myosin heavy chain or its active fragment is disclosed. The diagnostic agent is useful for topographic diagnosis of heart disease such as myocardial infarction and myocardial disease by imaging.

Excerpt(s): The present invention relates to a diagnostic agent useful for diagnosis of heart disease such as myocardial infarction and myocardial disease by imaging, and a method for the diagnosis of heart disease with use of the diagnostic agent. In recent years, in the diagnosis of heart disease such as myocardial infarction, diagnosis by imaging which involves administering a radiolabeled tracers into a body, detecting.gamma.-rays emitted by the radioisotope to convert the same into an image, processing the image with a computer to obtain a two- or three-dimensional image, and diagnosing the site or size of the myocardial infarction on the basis of the image thus obtained has made rapid progress. However, tracers used heretofore in the diagnosis by imaging in cardiac nuclear medicine have not always been able to depict specifically the site of myocardial infarction. For example, T1 scintigraphy of myocardium using thallium-201 (.sup.201 T1) applies the mechanism wherein the T1 behaves in vivo similarly as potassium ion and is taken into cells of the heart liver, kidneys, endocrine organs, tumors and the like where turnover rate is relatively fast, whereby normal cardiac muscle is depicted while the T1 is not ingested into necrotized or ischemic cardiac muscle at an infarction site, which site is depicted as a defect. Accordingly, the T1 does not always depict cardiac muscle specifically, and it has also been difficult to determine by this method whether the infarction occurred recently or in the past.

Web site: http://www.delphion.com/details?pn=US04943427__

- **Dietary supplement and method for lowering risk of heart disease**

Inventor(s): Bell; Stacey J. (Belmont, MA), Bistrian; Bruce R. (Ipswich, MA), Forse; R. Armour (Brookline, MA)

Assignee(s): Beth Israel Deaconess Medical Center (Boston, MA)

Patent Number: 6,210,686

Date filed: December 18, 1998

Abstract: Yeast-derived fiber has been demonstrated, as described herein, to effectively improve the serum lipid profile in humans, when provided as a dietary supplement, without some of the disadvantages known to accompany dietary supplementation with oat fiber or psyllium fiber. Described herein are dietary supplements comprising yeast fiber, e.g.,.beta.-glucan or glucomannan, and further comprising folic acid or a salt thereof, vitamin B.sub.6, vitamin B.sub.12, and vitamin E. The dietary supplements of the invention can further comprise fats, carbohydrates and proteins, for example, and other ingredients added to formulate a food product. Such food products can be in the form, for example, of solid or semi-solid foods, such as food bars, pudding, or spreads. By including folate and vitamin B.sub.6, the dietary supplement provides a second benefit of suppressing the level of homocysteine in the blood. A third benefit is provided by the anti-oxidant properties of vitamin E, particularly the effect of preserving low density lipoproteins from oxidation. Further embodiments of the invention are methods for improving the serum lipid profile in a human, methods for lowering risk of heart disease, and methods for improving cardiovascular health in a human, comprising administering to the human a dietary supplement of the invention.

Excerpt(s): Ischemic heart disease is a major health problem in the United States. One and half million new cases of heart disease are diagnosed annually; 700,000 of these are diagnosed at the occurrence of an acute myocardial infarction. Nine hundred thousand people die annually of heart disease (http://www.amhrt.org). The relative risk of mortality from heart disease increases four-fold as the serum cholesterol concentration of the individual goes from 4.32 mmol/L (167 mg/dL) to.gtoreq.6.83 mmol/L (264 mg/dL). (Schaefer, E. J., et al., "Individual Variability in Lipoprotein Cholesterol Response to the National Cholesterol Education Program Step 2 Diet," Am J. Clin. Nutr. 65:823-830 (1997)). At least 25% of the U.S. population has serum cholesterol levels outside the desirable range. (Sempos, C., et al., "The prevalence of High Blood Cholesterol Levels Among Adults in the United States," JAAM, 262:45-52 (1988)). However, a 1% reduction in serum cholesterol concentrations could reduce heart disease by 2%. (Lipid Research Clinics Programs. The Lipid Research Clinics Coronary Primary Prevention Trial Results I: Reduction in Incidence of Coronary Heart Disease, JAMA, 251:351-364 (1984); Lipid Research Clinics Programs. The Lipid Research Clinics Coronary Primary Prevention Trial Results II: The Relationship of Reduction in Incidence of Coronary Heart Disease to Cholesterol Lowering, JAAM, 251:365-374 (1984). It is known that changing the fat intake in the diet can significantly alter levels of cholesterol, LDLs and HDLs. The National Cholesterol Education Program (NCEP) limits intakes of total fat (<30% of total energy), saturated fat (<10% of total energy), and cholesterol (<300 mg) (Schaefer, E. J., et al., "Individual Variability in Lipoprotein Cholesterol Response to the National Cholesterol Education Program Step 2 Diet," Am J. Clin. Nutr. 65:823-830 (1997)). Patients can expect to experience a reduction in LDL-C (cholesterol in low density lipoproteins, or so-called "bad cholesterol") of 16% to 19%; HDL-C (cholesterol in high density lipoproteins, or so-called "good cholesterol") also declined, 11% for women and 17% for men.

Web site: http://www.delphion.com/details?pn=US06210686__

- **Implantable cardiac stimulation device for and method of monitoring progression or regression of heart disease by monitoring evoked response features**

  Inventor(s): Bradley; Kerry (Glendale, CA)

  Assignee(s): Pacesetter, Inc. (Sylmar, CA)

  Patent Number: 6,473,647

  Date filed: October 18, 2000

  Abstract: An implantable cardiac stimulation device has a system and method that monitors progression or regression in a patient's heart disease. A pulse generator delivers pacing pulses to the heart to cause evoked responses of the heart. A sensing circuit senses the evoked responses of the heart and generates evoked response signals. A processor is programmed to analyze the evoked response signals, to isolate a given characteristic of the evoked response signals and to quantify the isolated characteristics to provide corresponding quantized values. Relative changes in the quantized values over time are indicative of the progression or regression in the patient's heart disease. A memory stores the quantized values and a telemetry circuit transmits the stored quantized values to an external receiver for analysis.

  Excerpt(s): The present invention is generally directed to an implantable device for monitoring the progression or regression of heart disease. The present invention is more particularly directed to a system and method for use in an implantable cardiac stimulation device, which quantifies and stores evoked response features. Relative

changes in the quantified evoked response features, over time, are indicative of the progression or regression of the heart disease. More people die of heart disease than any other single cause. One common form of heart disease is congestive heart failure. Congestive heart failure (CHF) is a debilitating, end-stage disease in which abnormal function of the heart leads to inadequate bloodflow to fulfill the needs of the body's tissues. Typically, the heart loses propulsive power because the cardiac muscle loses capacity to stretch and contract. Often, the ventricles do not appropriately fill with blood between heartbeats and the valves regulating blood flow may become leaky, allowing regurgitation or back-flow of blood. The impairment of arterial circulation deprives vital organs of oxygen and nutrients. Fatigue, weakness, and inability to carry out daily tasks may result.

Web site: http://www.delphion.com/details?pn=US06473647__

- **Ischemic heart disease detection**

  Inventor(s): Ferek-Petric; Bozidar (Zagreb, HR)

  Assignee(s): Medtronic, Inc. (Minneapolis, MN)

  Patent Number: 6,514,195

  Date filed: April 28, 2000

  Abstract: An implantable medical device system for detecting cardiac conditions such the long-term ischemic heart disease, an occlusion of a coronary artery by a thrombus or an impending as a myocardial infarction. The implantable medical device (IMD) system includes a sensor that outputs a blood flow rate signal representing a rate of blood flow through a coronary sinus of a patient's heart. An implantable medical device (IMD) includes a microcomputer circuit configured to analyze the blood flow rate signal and detect a cardiac condition as a function of the blood flow rate signal. The system can also includes an implantable lead that senses electrical activity from the patient's heart. The microcomputer circuit monitors an ST segment of the electrical activity signal and detects a cardiac condition as a function of blood flow rate signal in conjunction with the electrical activity signal.

  Excerpt(s): This invention relates generally to the field of implantable medical devices, and more particularly to implantable heart monitors and therapy delivery devices. A wide variety of implantable heart monitors and therapy delivery devices have been developed including pacemakers, cardioverter/defibrillators, heart pumps, cardiomyostimulators, ischemia treatment devices, and drug delivery devices. Most of these cardiac systems include electrodes for sensing and sense amplifiers for recording and/or deriving sense event signals. All patents listed in Table 1 above are hereby incorporated by reference herein in their respective entireties. As those of ordinary skill in the art will appreciate readily upon reading the Summary of the Invention, Detailed Description and Claims set forth below, many of the devices and methods disclosed in the patents of Table 1 may be modified advantageously by using the teachings of the present invention.

  Web site: http://www.delphion.com/details?pn=US06514195__

- **Lysine containing peptides for treatment of heart disease**

  Inventor(s): Deghenghi; Romano (Chesaux-Dessus, St. Cergue, CH)

  Assignee(s): none reported

  Patent Number: 5,932,548

  Date filed: June 3, 1998

  Abstract: The present invention relates a number of different lysine containing peptides which can be administered to a mammal to normalize cardiac pressure for treatment of heart disease conditions such as myocardial ischemia. These peptides include certain known peptides, some of which are capable of liberating growth hormone to various degrees when administered to a mammal. Other peptides useful in the invention are novel peptide sequences which include a spirolactam, bicyclic or tricyclic peptidomimetic unit. The peptides disclosed herein exhibit binding to cardiac tissue and normalize cardiac pressure after administration, thus imparting cardiac protecting activity by a mechanism which at the present is unknown. One common feature of the peptides of this invention is that at least one lysine unit is present.

  Excerpt(s): The present invention relates to the administration of certain lysine containing peptides to a mammal for normalizing cardiac pressure and treating heart disease. Under the general term heart disease, a variety of cardiac ailments, including myocardial ischemia, heart failure and related vascular dysfunction, are treated with drugs such as organic nitrates, calcium channel blockers,.beta.-adrenergic receptor antagonists, antiplatelet and antithrombotic agents, cardiac glycosides, angiotensin converting enzyme inhibitors and angiotensin receptor antagonists. A general review of the field is found, for example, in Goodman & Gilman's "The Pharmacologic Basis of Therapeutics", IX edition, McGraw-Hill, New York, (1996), chapters 32 and 34. Recently, the protective effect of a peptide known as Hexarelin (also called examorelin) having the structure His-D-2-methyl-Trp-Ala-Trp-D-Phe-Lys-NH.sub.2 was described in an article by V. De Gennaro Colonna et al., European J. Pharmacology, 334, (1997), 201-207. Hexarelin was found to reverse the worsening of cardiac dysfunction in growth hormone deficient rats. At least part of its beneficial effect on myocardial ischemia was attributed to the growth hormone liberating properties of the peptide.

  Web site: http://www.delphion.com/details?pn=US05932548__

- **Method for detecting a risk of cancer and coronary heart disease and kit therefor**

  Inventor(s): Salonen; Jukka (Jannevirta, FI)

  Assignee(s): Oy Jurilab Ltd. (Jannevirta, FI)

  Patent Number: 6,242,186

  Date filed: June 1, 1999

  Abstract: The present invention is directed to a method and a kit for detecting a risk of cancer and coronary heart disease in a subject, comprising isolating genomic DNA from said subject, determining the allelic pattern for the codon 54 of the paraoxonase encoding PON1 gene in the genomic DNA, identification of M54L mutation indicating said risk being reduced.

  Excerpt(s): The present invention relates to a method for detecting or predicting the risk of, or predisposition to, cancer and coronary heart disease in a subject. The present invention also relates to a kit for carrying out the said method. The present invention

was based on the hypothesis that homozygosity of the L54 allele in the PON1 gene might protect against certain diseases associated with oxidative stress, in particular cancer and coronary heart disease. For this purpose, the said hypothesis was tested in a prospective population-based cohort study. The results of this study show that our hypothesis is true and that there is a clear association between homozygosity of the L54 allele, and a reduced risk for cancer and coronary heart disease. The present invention is thus directed to a method for detecting a risk of cancer and coronary heart disease, in a subject, comprising isolating genomic DNA from said subject, determining the allelic pattern for the codon 54 of the paraoxonase encoding PON1 gene in the genomic DNA, and identification of M54L mutation indicating said risk being reduced.

Web site: http://www.delphion.com/details?pn=US06242186__

- **Method for predicting coronary heart disease**

Inventor(s): Cuypers; Dirk (Bogaarden, BE)

Assignee(s): Bristol-Myers Squibb Company (Princeton, NJ)

Patent Number: 5,396,886

Date filed: July 1, 1993

Abstract: A device or system is provided for calculating and visualising the risk for developing coronary heart disease which is in the form of a cardiovascular risk diagram which includes a series of cardiovascular risk scales projected on cardiovascular risk areas (for example, delineated by different colours) each scale representing a different cardiovascular risk factor. Data for each cardiovascular risk is recorded on its appropriate risk scale, and data points-on adjacent risk scales are connected to form enclosed area superimposed over the risk areas. The resulting enclosed area is visually or otherwise compared to the total area of all cardiovascular risk areas, to determine a score of cardiovascular risk.

Excerpt(s): The present invention relates to a system and method for the visualisation of different cardiovascular risk factors on a scale, projected in different risk areas, to give a cardiovascular risk diagram which can be used to predict coronary heart disease. The major risk factors for coronary artery diseases are widely recognised. Age and gender have a powerful effect, but are immutable. Hypertension, cigarette smoking, total cholesterol and low density lipoprotein cholesterol double or more the risk. Other important recognised risk factors are overweight, left ventricular hypertrophy, glucose intolerance, hyperinsulinaemia and physical inactivity (Brach, Cholesterol and coronary heart disease prevention. A transatlantic consensus. European Heart Journal, 1989, 10:702-711; Grundy, Cholesterol and coronary heart disease. Jama, November 1986, Vol. 256:2849-2858; The prospective cardiovascular Munster study: Prevalence and prognostic significance of hyperlipidemia in men with systemic hypertension. Am. J. Card., 1987, 59:9G-17G; Egan et al., Comparative effect of overweight on cardiovascular risk in younger versus older men. Am. J. Card., February 1991, Vol. 67:248-252).

Web site: http://www.delphion.com/details?pn=US05396886__

- **Method for reducing infarct size in subjects afflicted with ischemic heart disease**

  Inventor(s): Kitakaze; Masafumi (Osaka, JP)

  Assignee(s): Otsuka Pharmaceutical Co., Ltd. (Tokyo, JP)

  Patent Number: 5,700,803

  Date filed: December 12, 1995

  Abstract: A method for reducing infarct size in a subject afflicted with ischemic heart disease, is disclosed, wherein said method uses, as the active agent, a carbostyril derivative.

  Excerpt(s): The present invention relates to a method for reducing infarct size in a subject afflicted with ischemic heart disease, wherein said method uses, as the active agent, a carbostyril derivative. are well-known in the art (U.S. Pat. No. 4,415,572, which is incorporated by reference herein in its entirety). These carbostyrils have been found to be an oral inotropic agent that augments myocardial contractility in model systems, with little effect on the heart rate or myocardial oxygen consumption (Feldman et al, N. Engl. J. Med., 329:149-155 (1993)), and are useful for treatment of patients with congestive heart failure (U.S. Pat. No. 4,415,572; and Hori et al, Jpn. Circ. J., 50:659-666 (1986)). Several studies have demonstrated that the above carbostyrils improve hemodynamic indexes, and exercise capacity in congestive heart failure patients (Inoue et al, Heart Vessels, 2:166-171 (1986); Sasayama et al, Heart Vessels, 2:23-28 (1986); and Feldman et al, Am. Heart J., 116:771-777 (1988)). In addition, multi-center randomized placebo-controlled trials both in Japan and in the United States have demonstrated that these carbostyrils improved quality of life and reduced the risk of death in patients with congestive heart failure (OPC-8212 Multicenter Research Group, Cardiovasc. Drugs Ther., 4:419-425 (1990); Feldman et al, Am. J. Cardiol., 68:1203-1210 (1991); and Feldman et al, N. Engl. J. Med., 329:149-155 (1993)).

  Web site: http://www.delphion.com/details?pn=US05700803__

- **Method for treating heart disease and cardiovascular disease in diabetic and non-diabetic patients**

  Inventor(s): Aoki; Thomas T. (1021 El Sur Way, Sacramento, CA 95825)

  Assignee(s): none reported

  Patent Number: 6,579,531

  Date filed: June 15, 2001

  Abstract: The present invention is a system and method capable of improving the dietary fuel capabilities and diabetics impaired patients and correct an overutilization of free fatty acids associated with heart disease in diabetic and non-diabetic patients. The current invention is the treating of heart disease and cardiovasular disease using insulin pulses to a patient utilizing Chronic Intermittent Intravenous Insulin Therapy to achieve an increase dietary fuel capabilities and correct overutilization of free fatty acids associated with heart disease in both diabetic and non-diabetic patients.

  Excerpt(s): This invention relates to the treatment of heart disease and cardiovascular disease in diabetic and non-diabetic patients. More specifically, the invention relates to a system and method for treating heart and cardiovascular diseases in diabetic and non-diabetic patients with Chronic Intermittent Intravenous Insulin Therapy. The main cause of death for patients with diabetes mellitus is cardiovascular disease in its various

forms. Existing evidence indicates that diabetic patients are particularly susceptible to heart failure, primarily in association with atherosclerosis of the coronary arteries and autonomic neuropathy. Furthermore, recent data also supports the existence of a disease entity called "diabetic cardiomyopathy" which occurs in the absence of angiographic signs of coronary artery disease. There is little doubt that a metabolic component is present in various forms of cardiovascular disease in diabetic patients. Altered lipid metabolism (excessive lipolysis, increased free fatty acids (FFA) levels and enhanced FFA oxidation in the myocardium) and altered carbohydrate metabolism (impaired glucose oxidation in the myocardium through reduced rate of glucose utilization and depressed pyruvate dehydrogenase complex activity) lead to depressed myosin ATPase activity, decreased ability of the sarcoplasmic reticulum to take up calcium, and depression of other membrane enzymes such as Na.sup.+ /K.sup.+ -ATPase and Ca.sup.2+ -ATPase (Rodrigues et al. J Mol Cell Cardiol, 1995, 27:169-79). The cardiac dysfunction (lower stroke volume, cardiac index and ejection fraction and a higher left ventricular end diastolic pressure) frequently manifested by patients with type 1 diabetes, could be explained at least partially by the metabolic abnormalities outlined above, and is likely secondary to insulin deficiency since appropriate insulin administration can restore normal patterns of cardiac metabolism (Avogaro et al, Am J Physiol 1990,258:E606-18). There is little dispute that an attempt should be made to lower elevated plasma triglyceride and FFA levels, thus decreasing the heart's reliance on FFA and, hence, overcoming the FFA inhibition of myocardial glucose utilization. The abnormalities in left ventricular systolic function may be partially reversible with improvement of metabolic control of diabetes. Recently, the DIGAMI (Diabetes mellitus, Insulin Glucose infusion in Acute Myocardial Infarction) study indicated that diabetic patients with acute myocardial infarction had a 28% reduced mortality at 1 year when treated with an insulin-glucose infusion followed by multidose insulin, compared to conventional therapy (controls) (DIGAMI, Malmberg K. Br Med J, 1997,314:1512-15). What is needed is a system and method that increases stroke volume, that improves cardiac index, that increases ejection fraction, and that lowers ventricular end diastolic pressure, thus improving cardiac function, as well as improving the quality of life of the patient. It is a further objective of this treatment to significantly reverse the cardiac dysfunction common to diabetic patients with heart disease, as well as heart disease in those who are not diabetic.

Web site: http://www.delphion.com/details?pn=US06579531__

- **Method of and arrangement for diagnosing heart disease**

  Inventor(s): Feng; Genquan (P.O. Box 1796, New York, NY 10185-0016)

  Assignee(s): none reported

  Patent Number: 5,649,544

  Date filed: January 17, 1992

  Abstract: Heart disease is non-invasively, accurately diagnosed at an early stage. A plurality of functions descriptive of the patient are mathematically determined. A set of indices for each function is established in advance. Each index has two states indicative of the patient's condition. An integrated pattern of the states of the indices from a plurality of the functions is generated and matched against a stored collection of index patterns whose diagnosis is known.

  Excerpt(s): This invention generally relates to a method of, and an arrangement for, determining a condition of a sample to be analyzed and, more particularly, to the early

and accurate diagnosis of heart and/or brain disease in human patients. Heart and brain disease are still the leading causes of death around the world. Conventional detection of such disease relies on electrocardiograph (EKG) and electroencephalograph (EEG) devices for measuring heart and brain wave activity by sensing electrical signals at various sites on the human body, and by recording these signals as waveforms. A cardiologist or a neurologist evaluates the EKG/EEG waveforms to determine abnormalities therein. Such evaluation requires considerable training and skill. Even despite a high degree of training and skill, an EKG/EEG waveform can still be interpreted as indicating normal heart/brain activity even in the presence of advanced coronary artery disease and brain epilepsy. Experience has shown that conventional EKG/EEG devices, although useful, are not sufficiently reliable to diagnose heart/brain disease, either due to insufficient sensitivity or specificity, and certainly not at an early stage of heart/brain disease. It has been estimated that over 50% of people with occlusive coronary artery disease or brain epilepsy have been reported to have normal EKG/EEG waveforms. The prior art has proposed several approaches to extract more information from the EKG/EEG signals. U.S. Pat. No. 4,924,875 teaches the extraction of information regarding ischemia, propensity to ventricular tachycardia and other disorders in the heart which affect cardiac electrical activity. U.S. Pat. No. 4,579,125 teaches the determination of the frequency content of EEG signals from the brain. U.S. Pat. No. 4,421,122 teaches the topographic mapping of a person's brain.

Web site: http://www.delphion.com/details?pn=US05649544__

- **Method of treating the syndrome of coronary heart disease risk factors in humans**

  Inventor(s): Clemens; Anton H. (Madison, WI)

  Assignee(s): CPD, LLC (Madison, WI)

  Patent Number: 6,262,062

  Date filed: August 15, 2000

  Abstract: A method of treating a human suffering from one or more conditions included within the Coronary Heart Disease Risk Factor (CHDRF) syndrome. The method includes administering, by a pharmaceutically effective mode, a drug composition having an active ingredient which is selected from opiate antagonists, opiates having.mu. agonist activity and combinations thereof.

  Excerpt(s): Coronary Heart Disease Risk Factors (CHDRFs) are major causes of death in the industrialized world. CHD risk factors include Type 2 Diabetes (and its precursor, Impaired Glucose Tolerance (IGT)), hyperlipidemia or dislipidemia, overweight, obesity and essential hypertension, i.e., a form of hypertension that occurs without a discoverable organic cause. The CHDRF syndrome may, therefore, be defined as a group of interrelated disorders: Type 2 Diabetes, IGT, Dislipidemia, Overweight, Obesity and essential hypertension. It has also become apparent that Type 2 Diabetes, by itself, represents a syndrome of various, in part sequential, disease states which interact with other components of the CHDRF syndrome. However, the exact interrelationships between the disease states that make up these syndromes is not fully understood. A wide variety of chemical and physical abnormalities associated with these syndromes exist. They include elevations in fasting blood glucose and gluconeogenesis in spite of significant increases in fasting insulin and C-peptide concentrations and increases in lipogenesis. Typically associated with lipogenesis are increases in levels of fasting Free Fatty Acid (FFA), fasting triglycerides (TG) and total cholesterol concentrations, increases in levels of fasting Low Density Lipoprotein (LDL)-

cholesterol, decreases in levels of fasting High Density Lipoprotein (HDL)-cholesterol, an increased LDL/HDL ratio, increases in body weight and increases in systolic and diastolic blood pressure. Although these syndromes are interrelated and typically result from derangements in nutrient metabolism, all the associated symptoms may not be present in individual patients. Accordingly, in some patients lipid metabolism problems may predominate, while in others, carbohydrate metabolism problems may be predominant. While these factors, which lend one aspect of the syndrome to dominate over another, are not well understood, it is clear that each portion of the syndrome, or combinations of portions of the syndrome, represents risk factors in coronary heart disease. A common denominator in the etiology of the syndromes of Type 2 Diabetes and the CHDRFs appears to be Insulin Resistance (IR). IR is characterized as a state in which a normal amount of insulin produces a subnormal biological response in carbohydrate metabolism. This may be the case for subjects afflicted with the non-insulin-dependent diabetes form of Type 2 Diabetes and in pre-diabetic subjects affected by Impaired Fasting Glucose (IFG) or Impaired Glucose Tolerance (IGT). These subjects require (and endogenously produce) higher than normal levels of insulin to compensate for their insulin resistance and to normalize their blood glucose levels. Traditionally, IR has been expressed as the insulin/glucose ratio (I/G). More recently, several more complex models have been proposed to define Insulin Resistance or the Insulin Sensitivity Index. Only recently have other biological functions of insulin become the focus of more intense scientific interest, e.g. the role of insulin in endogenous lipogenesis. Although an interaction between insulin resistance and the CHDRF components has been established, the cause and effect relationship between insulin resistance, obesity, dislipidemia and IGT/Type 2 Diabetes is still subject to debate. IR increases FFA levels, which further contributes to IR, thereby creating a vicious circle. Therapeutic modalities for lowering any one of the lipid fractions in dislipidemia have not proven capable of correcting the entire hyperlipidemic complex with a single therapeutic agent.

Web site: http://www.delphion.com/details?pn=US06262062__

- **Method of treating the syndrome of coronary heart disease risk factors in humans**

  Inventor(s): Clemens; Anton H. (5854 Schumann Dr., Madison, WI 53711)

  Assignee(s): none reported

  Patent Number: 6,026,817

  Date filed: April 11, 1998

  Abstract: A method of treatment of humans suffering from the Coronary Heart Disease Risk Factor (CHDRF) Syndrome which comprises the steps of 1) administering, by a pharmaceutically effective mode, a priming dose of drug composition selected from the group consisting of opiate antagonists, and drugs which substantially equally reduce the amounts of catecholamines bound to catecholamine binding sites for a period of about one to four weeks, and 2) administering a maintenance dose of said selected drug, is disclosed.

  Excerpt(s): Coronary heart disease is one of the major causes of death in the industrialized world. The major coronary heart disease (CHD) risk factors are hyperlipidemia, type 2 diabetes / impaired glucose tolerance (IGT), obesity and essential hypertension, that is, a form of hypertension occurring without discoverable organic cause. The coronary heart disease risk factor syndrome (CHDRF syndrome) may be defined as a group of diseases, that is, hyperlidemia, Type 2 diabetes / impaired

glucose tolerance (IGT), obesity and essential hypertension which individually, and together are among the major risk factors in coronary heart disease. The disease states which make up the syndrome of the coronary heart disease risk factors (CHDRF syndrome) are all interrelated. However, the exact interrelationship between the disease states making up the syndrome is not frilly understood. There are a wide variety of chemical and physical abnormalities associated with the CHDRF syndrome. These abnormalities include elevation in fasting blood glucose, elevation of HgbAlc, elevation of C-peptide, elevation of fasting total cholesterol, elevation of fasting LDL-cholesterol, decrease in fasting HDL-cholesterol, a high LDL/HDL ratio, elevation of fasting triglycerides, elevation of fasting free fatty acid concentration, elevation in body weight, elevation of systolic blood pressure, and elevation of diastolic blood pressure. Although the entire CHDRFS is interrelated, individual patients may not present all the symptoms associated with the syndrome. Accordingly, in some patients the lipid metabolism problems may predominate, while in others, the glucose metabolism problems play a more major role. The factors, which lead one aspect of the syndrome to predominate over another, are not well understood. However, it is clear that each portion of the syndrome, or combination of portions of the syndrome, represents a risk factor in coronary heart disease. Hyperlipidemia, also called dislipidemia, is characterized by elevated levels of total and low density lipoprotein cholesterol (LDL cholesterol), elevated levels of triglycerides and low levels of high density lipoprotein cholesterol (HDL cholesterol), as well as elevated levels of free fatty acids. State-of-the-art therapeutic regimens have failed to treat and correct the entire complex of hyperlipidemia with a single pharmaceutical agent. Drugs such as clofibrate/gemfibrozil lower triglycerides, by some unknown mechanism, but have no effect of the free fatty acid level, and no effect upon the total cholesterol level. However, the drugs may shift the proportion of cholesterol found in the form of low and high density lipoprotein cholesterol. In patients suffering from an elevated level of low density lipoprotein cholesterol, the drugs may actually further increase the level of low density lipoprotein cholesterol. Drugs like lovastatin, on the other hand, lower the level of both total and low density lipoprotein cholesterol, while slightly increasing the level of high density lipoprotein cholesterol. However, these drugs have no effect on free fatty acids and little or no effect on triglyceride levels.

Web site: http://www.delphion.com/details?pn=US06026817__

- **Method of treating the syndrome of coronary heart disease risk factors in humans**

  Inventor(s): Clemens; Anton H. (Madison, WI)

  Assignee(s): CPD, LLC (Madison, WI)

  Patent Number: 6,528,520

  Date filed: June 11, 2001

  Abstract: The invention provides an improved method of treating a human suffering from one or more conditions included within the Coronary Heart Disease Risk Factor (CHDRF) syndrome. The method includes administering, by a pharmaceutically effective mode, a drug composition having an opioidergic agent including an opiate antagonist, opiate having.mu.-agonist activity or combination thereof, and an insulin secretagogue.

  Excerpt(s): Coronary Heart Disease Risk Factors (CHDRFs) are major causes of death in the industrialized world. CHD risk factors include Type 2 Diabetes (and its precursor, Impaired Glucose Tolerance (IGT)), hyperlipidemia dyslipidemia, overweight, obesity

and essential hypertension, i.e., a form of hypertension that occurs without a discoverable organic cause. The CHDRF syndrome may, therefore, be defined as a group of interrelated disorders: Type 2 Diabetes, IGT, Dyslipidemia, Overweight, Obesity and essential hypertension. It has also become apparent that Type 2 Diabetes, by itself, represents a syndrome of various, in part sequential, disease states which interact with other components of the CHDRF syndrome. However, the exact interrelationships between the disease states that make up these syndromes is not fully understood. A wide variety of chemical and physical abnormalities associated with these syndromes exist. They include elevations in fasting blood glucose and gluconeogenesis in spite of significant increases in fasting insulin and C-peptide concentrations and increases in lipogenesis. Typically associated with lipogenesis are increases in levels of fasting Free Fatty Acid (FFA), fasting triglycerides (TG) and total cholesterol concentrations, increases in levels of fasting Low Density Lipoprotein (LDL)-cholesterol, decreases in levels of fasting High Density Lipoprotein (HDL)-cholesterol, an increased LDL/HDL ratio, increases in body weight and increases in systolic and diastolic blood pressure. Although these syndromes are interrelated and typically result from derangements in nutrient metabolism, all the associated symptoms may not be present in individual patients. Accordingly, in some patients lipid metabolism problems may predominate, while in others, carbohydrate metabolism problems may be predominant. While these factors, which lend one aspect of the syndrome to dominate over another, are not well understood, it is clear that each portion of the syndrome, or combinations of portions of the syndrome, represents risk factors in coronary heart disease. Common denominators in the etiology of the syndromes of Type 2 Diabetes and the CHDRFs appear to be Insulin Resistance (IR) and Beta-Cell Dysfunction. IR is characterized as a state in which a normal amount of insulin produces a subnormal biological response in carbohydrate metabolism. This may be the case for subjects afflicted with the non-insulin-dependent diabetes form of Type 2 Diabetes, in pre-diabetic subjects affected by Impaired Fasting Glucose (IFG) or Impaired Glucose Tolerance (IGT), and in overweight and obese subjects. These subjects require (and endogenously produce) higher than normal levels of insulin to compensate for their insulin resistance and Beta-Cell Dysfunction to normalize their blood glucose levels. Traditionally, IR has been expressed as the insulin/glucose ratio (I/G). More recently, several more complex models have been proposed to define Insulin Resistance or the Insulin Sensitivity Index. Only recently have other biological functions of insulin become the focus of more intense scientific interest, e.g. the role of insulin in endogenous lipogenesis. Although an interaction between insulin resistance and the CHDRF components has been established, the cause and effect relationship between insulin resistance, obesity, dyslipidemia and IGT/Type 2 Diabetes is still subject to debate. IR increases FFA and Triglyceride (TG) levels, which further contribute to IR, thereby creating a vicious circle. Therapeutic modalities for lowering any one of the lipid fractions in dyslipidemia have not proven capable of correcting the entire hyperlipidemic complex with a single therapeutic agent.

Web site: http://www.delphion.com/details?pn=US06528520__

- **Mitochondrial DNA damage as a predictor of coronary atherosclerotic heart disease**

  Inventor(s): Runge; Marschall S. (Galveston, TX), Ballinger; Scott W. (Santa Fe, TX), VanHouten; Bennett (Galveston, TX)

  Assignee(s): Research Development Foundation (Carson City, NV)

  Patent Number: 6,322,974

  Date filed: January 14, 1999

  Abstract: The present invention demonstrates that mitochondrial DNA damage occurs prior to, or simultaneous with, atherosclerotic lesion development, that aortic mitochondrial DNA damage increases with age, and that genotype and diet both influence the level of mitochondrial DNA damage. Hence, the present invention demonstrates that mitochondrial DNA damage occurs early in atherosclerosis, and may be an initiating event in atherogenesis, and provides methods to predict coronary atherosclerotic heart disease based upon the amount of mitochondrial DNA damage.

  Excerpt(s): The present invention relates generally to the field of physiology and molecular biology. More specifically, the present invention relates to DNA damage and the effects of DNA damage on atherosclerosis. Reactive oxygen species (reactive oxygen species) have been suggested to play a critical role in the pathogenesis of atherosclerotic lesions (1-6), but the underlying mechanisms have not yet been elucidated. For example, reactive oxygen species-mediated mechanisms are likely to be a significant factor in the oxidation of LDL (ox-LDL), a key event in atherogenesis (3,7,8). Studies have shown that both superoxide (O.sub.2.sup.-) and peroxynitrite (peroxynitrite; formed from O.sub.2.sup.- +nitric oxide) are capable of oxidizing LDL(9-11). Hence, reactions involving nitric oxide and/or O.sub.2.sup.- are believed to play a critical role in the pathogenesis of atherosclerotic lesions and impaired vascular function (i.e. endothelial cell dysfunction), with the actions of their oxidizing products (H.sub.2 O.sub.2, peroxynitrite) not yet well defined. The mitochondrion is a major source of cellular reactive oxygen species (O.sub.2.sup.-), which are formed during electron transport (12-16). These reactive oxygen species are capable of preferentially damaging the mitochondrial membranes and proteins (17-19), affecting key cell functions, including mitochondrial respiration, which, if altered, leads to increased reactive oxygen species production (20-22), mediating lipid peroxidation (23, 24) and DNA damage (25, 26). Because mitochondrial oxidative phosphorylation (OXPHOS) capacities decline as mitochondrial DNA (mtDNA) damage and mutations accumulate with age (6, 27-29), mitochondrial damage and reactive oxygen species generation may act as catalysts for age-related degenerative disease, such as coronary artery disease (CAD). It was hypothesized that free radicals generated within the endothelial and smooth muscle cell environment mediate mitochondrial damage within these cells, establishing a vicious cycle of further reactive oxygen species generation and mitochondrial damage leading to vascular cell dysfunction.

  Web site: http://www.delphion.com/details?pn=US06322974__

- **Nucleic acid encoding congenital heart disease protein and products related thereto**

  Inventor(s): Korenberg; Julie R. (Los Angeles, CA)

  Assignee(s): Cedars-Sinai Medical Center (Los Angeles, CA)

  Patent Number: 5,945,305

  Date filed: September 11, 1997

Abstract: In accordance with the present invention, there are provided novel Congenital Heart Disease (CHD) proteins. Nucleic acid sequences encoding such proteins and assays employing same are also disclosed. The invention CHD proteins can be employed in a variety of ways, for example, for the production of anti-CHD antibodies thereto, in therapeutic compositions and methods employing such proteins and/or antibodies.

Excerpt(s): The present invention relates to nucleic acids and receptor proteins encoded thereby. Invention nucleic acids encode novel congenital heart disease related proteins. The invention also relates to methods for making and using such nucleic acids and proteins. Down syndrome (DS) usually caused by chromosome 21 trisomy and is a major cause of mental retardation and congenital heart (CHD) and gut disease affecting over 200,000 persons in the United States alone. The characteristic heart abnormalities in DS-CHD include atrioventricular canal and ventricular septal defects. Other features of DS include a set of characteristic facies, thymic abnormalities, increased risk of leukemia and early onset of Alzheimer-like dementia. Molecular analysis of rare patients with DS and partial chromosome 21 duplications and monosomies has led to the association of certain chromosomal regions with specific DS phenotypes (Korenberg et al., Proc. Natl. Acad. Sci. USA M91:4997-5001 (1994); Delebar et al., Eur. J. Human. Genet. 1:114-124 (1993); Antonarakis et al., Progress in Clinical and Biological Research 311:29-43 (1989)). Chromosome 21 is therefore a model for the study of human chromosomal aneuploidy and the construction of its physical map is of special interest. Human chromosome 21 has a nearly complete physical map with a well characterized contiguous set of overlapping YACs spanning most of its length (Chumakov et al., Nature 359:380-387 (1992); Shimizu et al., Cytogenet. Cell Genet. 70:147-182 (1995); Korenberg et al., Genome Research 5:427-443 (1995)). The demand for sequence ready contigs and clones for gene isolation efforts has prompted the construction of numerous higher resolution contigs in cosmids (Patil et al., Hum. Molec. Genet. 3:1811-1817 (1995)) and more recently in PACs (Osoegawa et al., Genomics 32:375-387 (1996)). Considerable mapping efforts exist in the region from CBR to D21S55 due to the duplication of this region in partially trisomic individuals with several phenotypic features of DS including mental retardation. However, the distal and adjacent, 4-5 Mb D21S55 to MX1 region is also of interest due to its association with Down syndrome congenital heart disease (DS-CHD) as well as other characteristic features of DS (Korenberg et al., Am. J. Hum. Genet. 50:294-302 (1992), Korenberg et al. (1994)). Although non-chimeric YACs span this interval, three are not as yet any higher resolution physical maps are available for the entire D21S55 to MX1 region. Thus, higher resolution physical maps for the entire D21S55 to MX1 region are desired. In addition, there is a need in the art for an isolated nucleic acid, and isolated protein encoded thereby, associated with congenital heart disease (CHD).

Web site: http://www.delphion.com/details?pn=US05945305__

- **Peptides capable of modulating inflammatory heart disease**

  Inventor(s): Penninger; Josef Martin (Toronto, CA), Neu; Nickolaus (Innsbruck, AT), Hessel; Andrew John (Toronto, CA), Bachmaier; Kurt (Toronto, CA)

  Assignee(s): Amgen Canada Inc. (Mississauga, CA)

  Patent Number: 5,962,636

  Date filed: August 12, 1998

Abstract: Disclosed are novel peptides that modulate inflammatory heart disease. Also disclosed are DNA molecules encoding the peptides, and methods of making the peptides.

Excerpt(s): This invention relates to novel peptides, and to genes encoding the peptides, which are capable of inducing inflammatory cardiomyopathy in vivo. Cardiovascular diseases are a major cause of death in Western societies. Various risk factors have been associated with the pathogenesis of cardiovascular disease, including such factors as high cholesterol levels, smoking, stress, high blood pressure, obesity, and hyperglycemia. Recent evidence suggests that certain bacterial infections may be a causative event in the development of certain heart diseases (Danesh et al., Lancet, 350: 430-436 ›1997!). In particular, Chlamydia infections have recently been shown to be linked, both epidemiologcally and experimentally, to heart disease (Danesh et al., supra; Ossewaarde et al., Epidemiol. and Infect., 120: 93-99 ›1998!). Inflammatory heart disease and dilated cardiomyopathy similar to that which occurs in humans can be induced in mice by immunization of the mice with myosin protein obtained from heart muscle (Neu et al., J. Immunol., 139: 3630-3636 ›1987!). Immunization of Balb/c mice with a peptide consisting of amino acids 614-643 of cardiac alpha-myosin can induce inflammatory heart disease in the mice (Pummerer et al., J. Clin Invest., 97: 2057-2062 ›1996!).

Web site: http://www.delphion.com/details?pn=US05962636__

- **Pharmaceutical compositions effective against coronary heart disease and hypertension**

  Inventor(s): Berthold; Richard (Bottmingen, CH)

  Assignee(s): Sandoz Ltd. (Basel, CH)

  Patent Number: 4,510,150

  Date filed: August 1, 1983

  Abstract: Pharmaceutical composition containing as active agents (A) a calcium antagonist of a 4-(2,1,3-benz-oxa- or thiadiazoly)-1,4-dihydro pyridine type and (B) a.beta.-blocker and/or a vasodilating nitrate, effective against coronary heart disease and hypertension.

  Excerpt(s): The components (A), (B) and (C) of the composition are generally known. Particularly the compounds of formula I are known from e.g. European Patent Publication No. EP 150 (application No. 78100165.6), U.K. Patent Publication No. 20 41 358 (application No. 7943112) and U.K. Patent Publication No. 20 37 766 (application No. 7943113). In any of the above radicals for active component (A) of formula I, alkyl of 1 to 6 carbon atoms is preferably of 1 to 4 carbon atoms, especially of 1 to 2 carbon atoms. Any alkyl, alkoxy, alkylthio or alkylsulfonyl radical of 1 to 4 carbon atoms is preferably of 1 to 2 carbon atoms. The hydroxy, alkoxy, hydroxyalkoxy, amino or alkylamino group of the hydroxyalkyl, alkoxyalkyl, hydroxyalkoxyalkyl, aminoalkyl or alkylaminoalkyl moiety in COOR.sub.7 is preferably not attached to the.alpha.-carbon atom and is preferably attached to the distant terminal carbon atom. Any hydroxyalkyl, alkoxyalkyl, hydroxyalkoxyalkyl, aminoalkyl or alkylaminoalkyl radical preferably has an ethylene or propylene moiety substituted by hydroxy, alkoxy, hydroxyalkoxy, amino or alkylamino respectively. The alkyl moiety of cycloalkylalkyl is conveniently methyl. Halogen means fluorine, chlorine or bromine and is especially chlorine. Cycloalkyl or the cycloalkyl moiety of cycloalkylalkyl is conveniently cyclopropyl or cyclopentyl or

cyclohexyl. The multiple bond of alkenyl, alkinyl or phenylalkenyl in R.sub.1 or COOR.sub.7 is preferably not in the.alpha.,.beta. position. Alkenyl or alkinyl preferably has 3 to 5 carbon atoms. Alkenyl is conveniently allyl or 2-methylallyl. Alkinyl is conveniently propinyl. Phenylalkenyl preferably has the trans-configuration and is for example cinnamyl. When R.sub.1 is optionally substituted phenylalkyl, the phenyl group is preferably unsubstituted. When the phenyl group is di- or tri-substituted, preferably the substituents are the same. When R.sub.7, R.sub.8 or R.sub.9 contain a heterocyclic ring this may be for example furyl, thienyl, pyrrolyl, thiazolyl, isothiazolyl, thiadiazolyl, oxazolyl, isoxazolyl, oxadiazolyl, imidazolyl, pyrazolyl, triazolyl, tetrazolyl, pyridyl, pyrimidinyl, pyrazinyl, pyridazinyl, piperidinyl, morpholinyl or triazinyl. When R.sub.10 and R.sub.11 together with the nitrogen atom to which they are bound, form a heterocyclic ring, this is preferably saturated and may be for example pyrrolidine, piperidine, piperazine, N-alkylpiperazine, morpholine, azepane, diazepane or N-alkyl-diazepane.

Web site: http://www.delphion.com/details?pn=US04510150__

- **System and method for detecting and locating heart disease**

  Inventor(s): Fang; Dan Oun (P.O. Box 6787, Rosemead, CA 91770), Liu; Hai Xiang (P.O. Box 6787, Rosemead, CA 91770)

  Assignee(s): none reported

  Patent Number: 6,148,228

  Date filed: March 5, 1998

  Abstract: A method is delineated for detecting and locating coronary artery and heart disease comprising the steps of obtaining electrocardiograph (EKG) signals from a patient, modifying the EKG signals, and establishing a base value for use in evaluating modified EKG signals. The step of modifying includes the steps of amplifying the EKG signals, digitizing amplified EKG signals, mathematically modifying the amplified and digitized EKG signals to obtain 12 lead signals in the time domain, and converting the 12 lead signals into power spectrum signals in the frequency domain. The base value is obtained by taking a patient's resting heart rate in beats per minute, converting it to beats per second, and multiplying by a scaling quantity between approximately 3 and 7, inclusively. Then, a first area is calculated by integrating a selected one of the power spectrum signals from zero Hertz to the base value. Similarly, a second area results from integrating the selected power spectrum signal from the base value to infinity. Then, one takes the ratio of the first to second areas to obtain an evaluation standard indicative of the patient's coronary health. Peak analysis of the power spectrum signals is also available, and a scheme for locating detected heart disease is also provided. Lastly, a system corresponding to the aforementioned methodology is shown.

  Excerpt(s): This invention is in the field of cardiology and methods therefor and, more particularly, is a system and method for detecting and locating heart disease, and especially coronary artery disease including myocardial ischaemia and infarction; however, the act of locating is with reference to myocardial ischemia and infarction. Coronary artery disease is the leading cause of death in the United States, yet the disease remains "silent" or dormant in the majority of patients until the fourth or fifth decade of life. At that point, coronary artery disease typically moves from the "silent" phase to a symptomatic phase, at which time the patient may experience as the first symptoms, angina pectoris, myocardial infarction, and/or sudden death. The prevalence of coronary artery disease in the United States has been estimated as affecting over 4

million persons. Over 1 million are expected to suffer myocardial infarctions or sudden death before attaining the age of 60. Furthermore, once coronary artery disease is symptomatic--regardless of whether the symptoms comprise angina or myocardial infarction--the mortality rate is increased to 4% per year overall and 8% per year in those patients with an abnormal electrocardiogram or hypertension. This increased mortality rate is largely due to increases in the occurrence of sudden death, or the complications of repeated myocardial infarction.

Web site: http://www.delphion.com/details?pn=US06148228__

- **Therapeutics and diagnostics for congenital heart disease based on a novel human transcription factor**

  Inventor(s): Sheffield; Val C. (Coralville, IA), Nishimura; Darryl (Coralville, IA), Stone; Edwin M. (Iowa City, IA), Patil; Shiva (Iowa City, IA), Alward; Wallace L. M. (Iowa City, IA)

  Assignee(s): The University of Iowa Research Foundation (Iowa City, IA)

  Patent Number: 6,087,107

  Date filed: May 22, 1998

  Abstract: Methods and compositions for treating a congenital heart disease and methods and compositions for prognosing or diagnosing a congenital heart disease in a subject are disclosed.

  Excerpt(s): Many congenital heart diseases have a genetic basis. However, surgery offers the only therapeutic option for many of these disorders. In addition, current identification and diagnosis of congenital heart disease depends on the recognition of affected cardiac function, such as heart murmurs representing turbulent flow, altered systemic and pulmonary blood flow, shunting in either direction, and evidences of altered work load of the cardiac chambers. Routine history, physical examination, ECG, and chest x-ray are usually performed for specific anatomic diagnosis, with supportive and confirmatory data from echocardiography, cardiac catheterization, angiocardiography and other laboratory data. Improved therapies and diagnostics for genetically based congenital heart diseases are needed. The present invention is based, at least in part, on the discovery of a novel human gene, which encodes a novel human protein. These newly identified genes and proteins are referred to herein as "FKHL7". FKHL7 is a monomeric DNA binding protein that shares a core binding site (RTAAAYA; SEQ ID NO:22) with four other FKHL7-like proteins. In addition, the forkhead domain of this protein shows strong homology to the human gene, FKHL14 and the mouse genes, Fkh1 and Fkh14, by BLASTN analysis.

  Web site: http://www.delphion.com/details?pn=US06087107__

- **Treatment of heart disease with cox-2 inhibitors**

  Inventor(s): Delgado, III; Reynolds M. (Houston, TX), Willerson; James T. (Houston, TX)

  Assignee(s): Board of Regents, The University of Texas System (Austin, TX), Texas Heart Institute (Houston, TX)

  Patent Number: 6,323,226

  Date filed: October 16, 2000

Abstract: COX-2 selective inhibitors are disclosed as useful in treating or preventing heart disease, and in particular, congestive heart failure.

Excerpt(s): The present invention relates to the treatment of heart disease using cyclooxygenase-2 (COX-2) inhibiting drugs. Heart disease can be congenital or caused by an initial precipitating insult. Examples of initial insults include a lack of adequate blood flow, infection, toxins and autoimmune type reactions. Typically cardiac function continues to deteriorate post-initial insult, and heart muscle (myocyte) performance declines. The term "heart disease" is used in the general sense and includes conditions ranging, for example, from those in which positive inotropic medications are useful to those in which coronary vessel occlusion is predominant, to arrhythmias or cardiotoxicity, such as that which may be observed as a side effect of cardiotoxic drugs, e.g., doxorubicin. In these conditions, it is evident that COX-2 expression and the inflammation that is attendant therewith contribute to the overall disease state. Congestive heart failure (CHF, cardiac failure) is a form of heart disease in which weakened heart function exists with concommitant edema. Congestive heart failure has many different causes, including narrowing of the arteries supplying blood to the heart muscle (coronary heart disease); prior heart attack (myocardial infarction) resulting in scar tissue large enough or located so to interfere with normal electrocardiac function; high blood pressure; heart valve disease, such as due to past rheumatic fever or congenital valve abnormality; primary disease of the heart muscle itself (cardiomyopathy); other defects in the heart present at birth (congenital heart disease); and infection of the heart valves and/or heart muscle itself (endocarditis and/or myocarditis). Each of these disease processes can lead to congestive heart failure.

Web site: http://www.delphion.com/details?pn=US06323226__

- **Use of phenylglyoxylic acids and derivatives thereof in the treatment of ischemic heart disease**

Inventor(s): Page; Michael G. (Waterford, CT), Morville; Malcolm (Margate, GB2), Danilewicz; John C. (Ash, Nr. Canterbury, GB2), Cross; Peter E. (Canterbury, GB2), Barnish; Ian T. (Ramsgate, GB2)

Assignee(s): Pfizer Inc. (New York, NY)

Patent Number: 4,239,779

Date filed: September 14, 1979

Abstract: The use of phenylglyoxylic acids and derivatives thereof in the treatment of ischemic heart disease and the hyperglycemia of diabetes.

Excerpt(s): The invention relates to the novel use of certain phenylglyoxylic acids and derivatives thereof in the treatment of ischemic heart disease and the hyperglycemia of diabetes. The invention also relates to a pharmaceutical composition of the useful compounds in a unit dosage form. Ischemic heart disease is characterized by the obstruction of the major blood vessels which service the heart, resulting in decreased oxygen supply to myocardial tissue. During physical stress the reduction in the myocardial oxygen supply results in extreme cardiac pain known as angina pectoris. Although nitrates, such as nitroglycerin, are the drugs of choice as vasodilators in the treatment of ischemic heart disease, they suffer from a short duration of action.

Web site: http://www.delphion.com/details?pn=US04239779__

Patent Applications on Heart Disease

As of December 2000, U.S. patent applications are open to public viewing.[10] Applications are patent requests which have yet to be granted. (The process to achieve a patent can take several years.) The following patent applications have been filed since December 2000 relating to heart disease:

- **Ischemic heart disease detection**

  Inventor(s): Ferek-Petric, Bozidar; (Zagreb, HR)

  Correspondence: Thomas F. Woods; Medtronic, Inc. MS LC 340; 710 Medtronic Parkway; Minneapolis; MN; 55432-5604; US

  Patent Application Number: 20020120205

  Date filed: March 1, 2002

  Abstract: An implantable medical device system for detecting cardiac conditions such the long-term ischemic heart disease, an occlusion of a coronary artery by a thrombus or an impending as a myocardial infarction. The implantable medical device (IMD) system includes a sensor that outputs a blood flow rate signal representing a rate of blood flow through a coronary sinus of a patient's heart. An implantable medical device (IMD) includes a microcomputer circuit configured to analyze the blood flow rate signal and detect a cardiac condition as a function of the blood flow rate signal. The system can also includes an implantable lead that senses electrical activity from the patient's heart. The microcomputer circuit monitors an ST segment of the electrical activity signal and detects a cardiac condition as a function of blood flow rate signal in conjunction with the electrical activity signal.

  Excerpt(s): This invention relates generally to the field of implantable medical devices, and more particularly to implantable heart monitors and therapy delivery devices. A wide variety of implantable heart monitors and therapy delivery devices have been developed including pacemakers, cardioverter/defibrilla- tors, heart pumps, cardiomyostimulators, ischemia treatment devices, and drug delivery devices. Most of these cardiac systems include electrodes for sensing and sense amplifiers for recording and/or deriving sense event signals. All patents listed in Table 1 above are hereby incorporated by reference herein in their respective entireties. As those of ordinary skill in the art will appreciate readily upon reading the Summary of the Invention, Detailed Description and Claims set forth below, many of the devices and methods disclosed in the patents of Table 1 may be modified advantageously by using the teachings of the present invention.

  Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

[10] This has been a common practice outside the United States prior to December 2000.

- **Method for diagnosing heart disease, predicting sudden death, and analyzing treatment response using multifractial analysis**

  Inventor(s): Flick, James T. (Little Rock, AR), Joseph, Jacob; (Little Rock, AR)

  Correspondence: WRIGHT LINDSEY & JENNINGS, LLP; 200 WEST CAPITOL AVENUE; SUITE 2200; LITTLE ROCK; AR; 722013699

  Patent Application Number: 20030163057

  Date filed: February 22, 2002

  Abstract: A method of analyzing electrocardiogram (EKG) data for use in the diagnosis of heart disease, prognosis of cardiac conditions, and the monitoring of heart disease therapies is disclosed. The method utilizes a wavelet-based multifractal analysis with one or more of (1) a discrete wavelet smoothing step to remove the effects of abnormal beats; (2) "Levy flight" analysis to detect the frequency of abnormal beats known to adversely affect the multifractal (MF) analysis; and (3) MF alpha analysis, a multifractal extension of monofractal short term (ST) alpha analysis. The invention further comprises an EKG test battery comprising Levy flight anomalous beat/beat cluster screening, followed by (ST) MF alpha analysis and MF Holder analysis (when validated by the Levy flight analysis). The wavelet smoothing step can also be used to classify human EKGs by observing the effect of sequential smoothing on the MF Holder coefficient. Alternative choices to the wavelet smoothing approach to removal of abnormal beat effects include probability distribution function analysis to determine the MF Holder coefficient directly, abnormal beat ridge skeleton removal to remove the offending beats based on a direct multifractal spectrum calculation, and the calculation of various types of entropy coefficients for the EKG time series.

  Excerpt(s): The present invention is directed to a battery of multifractal-based tests developed for the analysis of electrocardiogram (EKG) data. The invention is preferably for clinical use in a novel integrated approach to diagnosis of heart disease, prognosis of cardiac conditions, and monitoring of heart disease therapies. This multifractal approach is not available with current, clinically available cardiographic methods. Cardiovascular disease currently affects approximately 20 million Americans. Roughly 12 million Americans are affected by coronary artery disease (CAD), and 5 million suffer from congestive heart failure (CHF). More importantly, millions suffer from undiagnosed heart disease; the prevalence of undiagnosed CHF is estimated to be approximately 20 million in the U.S. In addition, 400,000 Americans with CAD or CHF die from sudden cardiac death each year. Although there are a number of conventional nonfractal tests for the diagnosis of heart disease (several of them highly invasive), none of these tests can effectively predict which patients in this group of 17 million (CAD+CHF) are at risk of sudden death. Since it is impractical and undesirable to treat every person with heart disease (CAD or CHF) as if such a person were at risk for sudden death, a reliable predictive test (or test battery) to determine which patients are at high risk would be of great value. In addition, new drugs and other treatments continue to be developed to treat heart disease. There are, however, no currently available analytic test regimens to rapidly evaluate how well the patient may respond to treatment and whether the risk of sudden death has been decreased by anti-arrhythmic therapy. As a result, the efficacy of these treatments must be determined empirically with slow, multi-year, prospective-controlled studies, which are very difficult to extrapolate to each individual patient. Therefore, an analytic method that would monitor a patient for signs of potential improvement on therapy would also be of great value. The diagnosis of CAD and CHF would also be greatly improved by a very sensitive and specific but noninvasive test to replace or supplement expensive and

invasive modalities like heart catheterization and allow screening of patients with asymptomatic heart disease.

Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **Method of treating the syndrome of coronary heart disease risk factors in humans**

  Inventor(s): Clemens, Anton H. (Madison, WI)

  Correspondence: MICHAEL BEST & FRIEDRICH, LLP; ONE SOUTH PINCKNEY STREET; P O BOX 1806; MADISON; WI; 53701

  Patent Application Number: 20030149076

  Date filed: March 3, 2003

  Abstract: The invention provides an improved method of treating a human suffering from one or more conditions included within the Coronary Heart Disease Risk Factor (CHDRF) syndrome. The method includes administering, by a pharmaceutically effective mode, a drug composition having an opioidergic agent including an opiate antagonist, opiate having.mu.-agonist activity or combination thereof, and an insulin secretagogue.

  Excerpt(s): This application is a continuation-in-part of and claims priority to U.S. application Ser. No. 09/639,061 filed on Aug. 15, 2000. Coronary Heart Disease Risk Factors (CHDRFs) are major causes of death in the industrialized world. CHD risk factors include Type 2 Diabetes (and its precursor, Impaired Glucose Tolerance (IGT)), hyperlipidemia or dyslipidemia, overweight, obesity and essential hypertension, i.e., a form of hypertension that occurs without a discoverable organic cause. The CHDRF syndrome may, therefore, be defined as a group of interrelated disorders: Type 2 Diabetes, IGT, Dyslipidemia, Overweight, Obesity and essential hypertension. It has also become apparent that Type 2 Diabetes, by itself, represents a syndrome of various, in part sequential, disease states which interact with other components of the CHDRF syndrome. However, the exact interrelationships between the disease states that make up these syndromes is not fully understood. A wide variety of chemical and physical abnormalities associated with these syndromes exist. They include elevations in fasting blood glucose and gluconeogenesis in spite of significant increases in fasting insulin and C-peptide concentrations and increases in lipogenesis. Typically associated with lipogenesis are increases in levels of fasting Free Fatty Acid (FFA), fasting triglycerides (TG) and total cholesterol concentrations, increases in levels of fasting Low Density Lipoprotein (LDL)-cholesterol, decreases in levels of fasting High Density Lipoprotein (HDL)-cholesterol, an increased LDL/HDL ratio, increases in body weight and increases in systolic and diastolic blood pressure. Although these syndromes are interrelated and typically result from derangements in nutrient metabolism, all the associated symptoms may not be present in individual patients. Accordingly, in some patients lipid metabolism problems may predominate, while in others, carbohydrate metabolism problems may be predominant. While these factors, which lend one aspect of the syndrome to dominate over another, are not well understood, it is clear that each portion of the syndrome, or combinations of portions of the syndrome, represents risk factors in coronary heart disease.

  Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **Methods for diagnosing and treating heart disease**

  Inventor(s): Fishman, Mark C. (Newton Center, MA)

  Correspondence: Karen L. Elbing, Ph.D. Clark & Elbing LLP; 176 Federal Street; Boston; MA; 02110; US

  Patent Application Number: 20020182599

  Date filed: January 12, 2001

  Abstract: The invention provides methods of diagnosing heart disease, such as heart failure, screening methods for identifying compounds that can be used to treat or to prevent heart disease, and methods of using these compounds to treat or to prevent heart disease. The invention also provides animal model systems for carrying out the screening methods.

  Excerpt(s): This application claims priority from U.S. provisional patent application 60/175,786, filed Jan. 12, 2000. This invention relates to methods for diagnosing and treating heart disease. Heart disease is a general term used to describe many different heart conditions. For example, coronary artery disease, which is the most common heart disease, is characterized by constriction or narrowing of the arteries supplying the heart with oxygen-rich blood, and can lead to myocardial infarction, which is the death of a portion of the heart muscle. Heart failure is a condition resulting from the inability of the heart to pump an adequate amount of blood through the body. Heart failure is not a sudden, abrupt stop of heart activity, but, rather, typically develops slowly over many years, as the heart gradually loses its ability to pump blood efficiently. Risk factors for heart failure include coronary artery disease, hypertension, valvular heart disease, cardiomyopathy, disease of the heart muscle, obesity, diabetes, and a family history of heart failure.

  Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **Methods of use of fibroblast growth factor, vascular endothelial growth factor and related proteins in the treatment of acute and chronic heart disease**

  Inventor(s): Franco, Wayne P. (Rocky Hill, CT)

  Correspondence: Cummings & Lockwood; 700 State Street; P.O. Box 1960; New Haven; CT; 06509-1960; US

  Patent Application Number: 20020058612

  Date filed: April 6, 2001

  Abstract: Disclosed herein is a rational, multi-tier approach to the administration of growth factor proteins in the treatment of heart disease. Also disclosed is a method to evaluate the effectiveness of the administration of growth factor proteins comprising the clinical assay of CPK-MB levels in a patient undergoing treatment with growth factor proteins. In addition, there is disclosed a method for treatment of heart disease comprising administration of a therapeutically effective amount of a growth factor protein by oral inhalation therapy.

  Excerpt(s): This application claims priority under Title 35, U.S.C.sctn. 119(e) of United States Application No. 60/195,624, Filed Apr. 6, 2000. The present invention relates generally to strategies and methods for the treatment of chronic and acute heart disease through the delivery of one or more related protein growth factors such as fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF). Chronic myocardial

ischemia is the leading cardiac illness affecting the general population in the Western world. Since the occurrence of angina symptoms or objective physiological manifestations of myocardial ischemia signifies a mismatch between myocardial oxygen demand and the available coronary blood flow, the goal of therapy is to restore this balance. This can be achieved either by attempting to prevent further disease progression through modification of risk factors, or by more aggressive modes of therapy such as reducing the myocardial oxygen demand (i.e. reducing the heart rate, myocardial contractility or blood pressure) by using anti-anginal medications, or by restoring the blood supply by means of mechanical interventions such as percutaneous transluminal angioplasty or its variants, or coronary artery bypass surgery, coronary angioplasty (PTCA) or bypass surgery (CABG). When CABG is selected as the treatment option, its success may be limited by the inability to provide complete revascularization in those patients in whom the artery that supplies a viable but underperfused myocardial territory is not graftable because of diffuse-disease, calcifications, or small size. Complete revascularization cannot be achieved in up to 37% of patients undergoing CABG. This number is probably much lower today. However, patients who undergo complete revascularization have improved 5-year survival and angina-free survival compared with patients who have incomplete revascularization. Therefore, an adjunctive treatment strategy is warranted in patients undergoing CABG if complete revascularization is not possible. Percutaneous catheter-based revascularization is often precluded secondary to the same attributes that made the myocardial territory ungraftable: diffuse disease and small or calcified vessels.

Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **Milk and milk products for preventing or treating heart disease**

  Inventor(s): McLachlan, Corran Norman Stuart; (North Shore, NL)

  Correspondence: YOUNG & THOMPSON; 745 SOUTH 23RD STREET 2ND FLOOR; ARLINGTON; VA; 22202

  Patent Application Number: 20020166129

  Date filed: July 18, 2001

  Abstract: A milk which is free of.beta.-casein A.sup.1 protein in the prevention or treatment of coronary heart disease is disclosed. In addition, a process for the testing of DNA from cells obtained from lactating bovines for the presence of DNA encoding certain.beta.-casein proteins, selecting the bovines on the basis of the testing, and then milking those bovines to produce milk free of.beta.-casein A.sup.1 for use in the prevention or treatment of coronary heart disease is disclosed.

  Excerpt(s): This invention relates to the use of milk which is free of the.beta.-casein A.sup.1 protein in the prevention or treatment of coronary heart disease. The invention also relates to the testing of DNA from cells obtained from lactating bovines for the presence of DNA encoding certain.beta.-casein proteins, selecting the bovines on the basis of the testing, and then milking those bovines to produce milk free of.beta.-casein A.sup.1 for use in the prevention or treatment of coronary heart disease. Coronary heart disease is a major cause of death, particularly in countries where the populations are well-nourished, such as in the western world. Many factors are implicated as risk factors for this disease including obesity, smoking, genetic predisposition, diet, hypertension, and cholesterol. Dairy products, especially milk, are a major contributor to the dietary intake of humans, again particularly in western world populations. Milk contains numerous components of nutritional and health benefit. Calcium is one example.

However, milk is also a significant source of dietary fat. It is widely accepted that saturated fats found in milk are a risk factor for coronary heart disease. However, the inventor has discovered an additional risk factor present in some bovine milk unrelated to the fat content. What is entirely surprising is the source of the risk. The source is not dependent on the fat content of milk. Instead, it is a milk protein,.beta.-casein, which is linked to coronary heart disease.

Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **Mitochondrial DNA damage as a predictor of coronary atherosclerotic heart disease**

  Inventor(s): Ballinger, Scott W. (Santa Fe, TX), VanHouten, Bennett; (Durham, NC), Runge, Marschall S. (Chapel Hill, NC)

  Correspondence: Benjamin Aaron Adler; ADLER & ASSOCIATES; 8011 Candle Lane; Houston; TX; 77071; US

  Patent Application Number: 20030040495

  Date filed: April 10, 2001

  Abstract: The present invention demonstrates that mitochondrial DNA damage occurs prior to, or simultaneous with, atherosclerotic lesion development, that aortic mitochondrial DNA damage increases with age, and that genotype and diet both influence the level of mitochondrial DNA damage. Hence, the present invention demonstrates that mitochondrial DNA damage occurs early in atherosclerosis, and may be an initiating event in atherogenesis, and provides methods to predict coronary atherosclerotic heart disease based upon the amount of mitochondrial DNA damage.

  Excerpt(s): This non-provisional patent application is a continuation in part of U.S. Ser. No. 09/231,093, filed Jan. 14, 1999. The present invention relates generally to the field of physiology and molecular biology. More specifically, the present invention relates to DNA damage and the effects of DNA damage on atherosclerosis. Reactive oxygen species (reactive oxygen species) have been suggested to play a critical role in the pathogenesis of atherosclerotic lesions (1-6), but the underlying mechanisms have not yet been elucidated. For example, reactive oxygen species-mediated mechanisms are likely to be a significant factor in the oxidation of LDL (ox-LDL), a key event in atherogenesis (3,7,8). Studies have shown that both superoxide (O.sub.2.sup.31) and peroxynitrite (peroxynitrite; formed from O.sub.2.sup.-+nitric oxide) are capable of oxidizing LDL (9-11). Hence, reactions involving nitric oxide and/or O.sub.2.sup.- are believed to play a critical role in the pathogenesis of atherosclerotic lesions and impaired vascular function (i.e. endothelial cell dysfunction), with the actions of their oxidizing products (H.sub.2O.sub.2, peroxynitrite) not yet well defined.

  Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **System and method for treating heart disease and cardiovascular disease in diabetic and non-diabetic patients**

  Inventor(s): Aoki, Thomas T. (Sacramento, CA)

  Correspondence: Law Office of Eric G. Masamori; 6520 Ridgewood Drive; Castro Valley; CA; 94552; US

  Patent Application Number: 20010053380

  Date filed: June 15, 2001

  Abstract: The present invention is a system and method capable of improving the dietary fuel capabilities and metabolically impaired patients and correct an overutilization of free fatty acids associated with heart disease in diabetic and non-diabetic patients. The current invention is the treating of heart disease and cardiovasular disease using insulin pulses to a patient utilizing Chronic Intermittent Intravenous Insulin Therapy to achieve an increase dietary fuel capabilities and correct overutilization of free fatty acids associated with heart disease in both diabetic and non-diabetic patients.

  Excerpt(s): This application claims the benefit of U.S. Provisional Patent Application Number 60/212,135 filed Jun. 16, 2000. This invention relates to the treatment of heart disease and cardiovascular disease in diabetic and non-diabetic patients. More specifically, the invention relates to a system and method for treating heart and cardiovascular diseases in diabetic and non-diabetic patients with Chronic Intermittent Intravenous Insulin Therapy. The main cause of death for patients with diabetes mellitus is cardiovascular disease in its various forms. Existing evidence indicates that diabetic patients are particularly susceptible to heart failure, primarily in association with atherosclerosis of the coronary arteries and autonomic neuropathy. Furthermore, recent data also supports the existence of a disease entity called "diabetic cardiomyopathy" which occurs in the absence of angiographic signs of coronary artery disease. There is little doubt that a metabolic component is present in various forms of cardiovascular disease in diabetic patients. Altered lipid metabolism (excessive lipolysis, increased free fatty acids (FFA) levels and enhanced FFA oxidation in the myocardium) and altered carbohydrate metabolism (impaired glucose oxidation in the myocardium through reduced rate of glucose utilization and depressed pyruvate dehydrogenase complex activity) lead to depressed myosin ATPase activity, decreased ability of the sarcoplasmic reticulum to take up calcium, and depression of other membrane enzymes such as Na.sup.+/K.sup.+-ATPase and Ca.sup.2+-ATPase (Rodrigues et al. J Mol Cell Cardiol, 1995, 27:169-79). The cardiac dysfunction (lower stroke volume, cardiac index and ejection fraction and a higher left ventricular end diastolic pressure) frequently manifested by patients with type 1 diabetes, could be explained at least partially by the metabolic abnormalities outlined above, and is likely secondary to insulin deficiency since appropriate insulin administration can restore normal patterns of cardiac metabolism (Avogaro et al, Am J Physiol 1990,258:E606-18). There is little dispute that an attempt should be made to lower elevated plasma triglyceride and FFA levels, thus decreasing the heart's reliance on FFA and, hence, overcoming the FFA inhibition of myocardial glucose utilization. The abnormalities in left ventricular systolic function may be partially reversible with improvement of metabolic control of diabetes. Recently, the DIGAMI (Diabetes mellitus, Insulin Glucose infusion in Acute Myocardial Infarction) study indicated that diabetic patients with acute myocardial infarction had a 28% reduced mortality at 1 year when treated with an insulin-glucose infusion followed by multidose insulin, compared to conventional therapy (controls) (DIGAMI, Malmberg K. Br Med J, 1997,314:1512-15).

Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **System for treating heart disease and cardiovascular disease in diabetic and non-diabetic patients**

  Inventor(s): Aoki, Thomas T. (Sacramento, CA)

  Correspondence: Law Office of Eric G. Masamori; 6520 Ridgewood Drive; Castro Valley; CA; 94552; US

  Patent Application Number: 20030176322

  Date filed: March 19, 2003

  Abstract: The present invention is a system capable of improving the dietary fuel capabilities of diabetics and metabolically impaired patients and correct an overutilization of free fatty acids associated with heart disease in diabetic and non-diabetic patients. The current invention is the treating of heart disease and cardiovascular disease using insulin pulses to a patient utilizing Chronic Intermittent Intravenous Insulin Therapy to achieve an increase dietary fuel capabilities and correct overutilization of free fatty acids associated with heart disease in both diabetic and non-diabetic patients.

  Excerpt(s): This application claims the benefit of U.S. Non-Provisional Patent Application No. 09/881,824, filed on Jun. 15, 2001 which further claims the benefit of U.S. Provisional Patent Application No. 60/212,135 filed Jun. 16, 2000. This invention relates to the treatment of heart disease and cardiovascular disease in diabetic and non-diabetic patients. More specifically, the invention relates to a system for treating heart and cardiovascular diseases in diabetic and non-diabetic patients with Chronic Intermittent Intravenous Insulin Therapy. The main cause of death for patients with diabetes mellitus is cardiovascular disease in its various forms. Existing evidence indicates that diabetic patients are particularly susceptible to heart failure, primarily in association with atherosclerosis of the coronary arteries and autonomic neuropathy. Furthermore, recent data also supports the existence of a disease entity called "diabetic cardiomyopathy" which occurs in the absence of angiographic signs of coronary artery disease. There is little doubt that a metabolic component is present in various forms of cardiovascular disease in diabetic patients. Altered lipid metabolism (excessive lipolysis, increased free fatty acids (FFA) levels and enhanced FFA oxidation in the myocardium) and altered carbohydrate metabolism (impaired glucose oxidation in the myocardium through reduced rate of glucose utilization and depressed pyruvate dehydrogenase complex activity) lead to depressed myosin ATPase activity, decreased ability of the sarcoplasmic reticulum to take up calcium, and depression of other membrane enzymes such as Na.sup.+/K.sup.+-ATPase and Ca.sup.+-ATPase (Rodrigues et al. J Mol Cell Cardiol, 1995, 27:169-79). The cardiac dysfunction (lower stroke volume, cardiac index and ejection fraction and a higher left ventricular end diastolic pressure) frequently manifested by patients with type 1 diabetes, could be explained at least partially by the metabolic abnormalities outlined above, and is likely secondary to insulin deficiency since appropriate insulin administration can restore normal patterns of cardiac metabolism (Avogaro et al, Am J Physiol 1990,258:E606-18). There is little dispute that an attempt should be made to lower elevated plasma triglyceride and FFA levels, thus decreasing the heart's reliance on FFA and, hence, overcoming the FFA inhibition of myocardial glucose utilization. The abnormalities in left ventricular systolic function may be partially reversible with improvement of metabolic control of diabetes. Recently, the DIGAMI (Diabetes mellitus, Insulin Glucose infusion in Acute Myocardial

Infarction) study indicated that diabetic patients with acute myocardial infarction had a 28% reduced mortality at 1 year when treated with an insulin-glucose infusion followed by multidose insulin, compared to conventional therapy (controls) (DIGAMI, Malmberg K. Br Med J, 1997,314:1512-15).

Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

- **Treatment or prophylaxis of ischemic heart disease**

  Inventor(s): Kitakaze, Masafumi; (Osaka, JP)

  Correspondence: HUNTON AND WILLIAMS; 1900 K STREET N W; WASHINGTON; DC; 20006; US

  Patent Application Number: 20010027181

  Date filed: January 3, 2001

  Abstract: The present invention provides a pharmaceutical composition and a method for reducing an infarct region resulting from the ischemic necrosis of cells, especially, a pharmaceutical composition and a method for suppressing ischemia-reperfusion injury in the treatment of ischemic heart disease. The pharmaceutical composition and the method utilize a substance, as an active ingredient, which can increase intracellular cGMP production by acting on a natriuretic peptide receptor, and which has the effect of reducing an infarct region. The substance is preferably a natriuretic peptide. The present invention is particularly useful for the treatment or prophylaxis of ischemic disease.

  Excerpt(s): This invention relates to a pharmaceutical composition for reducing an infarct region resulting from the ischemic necrosis of cells, the pharmaceutical composition containing a substance, as an active ingredient, which can increase intracellular cGMP production by acting on a natriuretic peptide receptor. This invention also relates to a method for reducing an infarct region resulting from the ischemic necrosis of cells, comprising administering said substance or pharmaceutical composition to a patient with ischemic disease. In recent years, ischemic heart disease has posed a major problem in an aging population. Of cardiac diseases which are diseases of circulatory organs, myocardial infarction ascribed to cardiovascular disorder, in particular, is a serious, potentially fatal disease which either obstructs the coronary artery or substantially decreases the blood flow resulting in ischemic necrosis of myocytes and deteriorating cardiac function. The direct cause of myocardial infarction is a decrease or interruption of the blood flow to the myocardium due to coronary arteriosclerosis or thrombus formation in the coronary artery. The disease can result in either acute or chronic cardiac failure. Methods adopted for treatment of ischemic heart disease include the dilatation of the obstructed coronary artery by use of an intravascularly inserted balloon, maintenance of blood flow by intravascular insertion of a stent, and dissolution and removal of a thrombus formed in the blood vessel with the use of a thrombolytic agent. With any of such treatments, it is known that as blood flow is restored in the coronary artery, Ca overload or free radicals occur, increasing the region of cellular necrosis. Prevention of the occurrence of such ischemia-reperfusion injury is difficult, and no effective method of treatment has been established.

  Web site: http://appft1.uspto.gov/netahtml/PTO/search-bool.html

Keeping Current

In order to stay informed about patents and patent applications dealing with heart disease, you can access the U.S. Patent Office archive via the Internet at the following Web address: **http://www.uspto.gov/main/patents.htm**. Under "Services," click on "Search Patents." You will see two broad options: (1) Patent Grants, and (2) Patent Applications. To see a list of granted patents, perform the following steps: Under "Patent Grants," click "Quick Search." Then, type "heart disease" (or synonyms) into the "Term 1" box. After clicking on the search button, scroll down to see the various patents which have been granted to date on heart disease. You can also use this procedure to view pending patent applications concerning heart disease. Simply go back to the following Web address: **http://www.uspto.gov/main/patents.htm**. Under "Services," click on "Search Patents." Select "Quick Search" under "Patent Applications." Then proceed with the steps listed above.

CHAPTER 7. BOOKS ON HEART DISEASE

Overview

This chapter provides bibliographic book references relating to heart disease. In addition to online booksellers such as **www.amazon.com** and **www.bn.com**, excellent sources for book titles on heart disease include the Combined Health Information Database and the National Library of Medicine. Your local medical library also may have these titles available for loan.

Book Summaries: Federal Agencies

The Combined Health Information Database collects various book abstracts from a variety of healthcare institutions and federal agencies. To access these summaries, go directly to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. You will need to use the "Detailed Search" option. To find book summaries, use the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer. For the format option, select "Monograph/Book." Now type "heart disease" (or synonyms) into the "For these words:" box. You should check back periodically with this database which is updated every three months. The following is a typical result when searching for books on heart disease:

- **Living a Healthy Life With Chronic Conditions: Self-Management of Heart Disease, Arthritis, Stroke, Diabetes, Asthma, Bronchitis, Emphysema and Others**

  Source: Palo Alto, CA: Bull Publishing Company. 1994. 296 p.

  Contact: Available from Bull Publishing Company. P.O. Box 208, Palo Alto, CA 94302-0208. (800) 676-2855 or (415) 322-2855. Fax (415) 327-3300. E-mail: BullPublishing@msn.com. PRICE: $14.95. ISBN: 0923521283.

  Summary: This book is a complete self-management guide for people with chronic diseases. The authors focus on day-to-day living skills, in the context of the specific chronic diseases, including heart disease, arthritis, stroke, diabetes, asthma, bronchitis, and emphysema. General topics include the psychological aspects to self-management; finding resources; smoking and quitting; understanding common symptoms; using one's mind to manage symptoms; exercising for fun and fitness; exercising for flexibility and strength; exercising for endurance; exercising tips for people with specific chronic

diseases; the importance of communication; durable powers of attorney for health care; eating well; and managing medications. The chapter on diabetes covers diabetes and its causes; maintaining an appropriate blood glucose level; symptoms of hyperglycemia and hypoglycemia; dietary management; exercise; insulin injections; oral medications; emotions; self-monitoring of blood glucose and urine; the complications of diabetes; and diabetes resources. Each chapter includes limited references and a subject index concludes the volume.

- **What women need to know: From headaches to heart disease and everything in between**

  Source: Chicago, IL: Olmstead Press. 2000. 239 pp.

  Contact: Available from LPC Group, Olmstead Press, 1436 West Randolph Street, Chicago, IL 60607. Telephone: (800) 243-0138 / fax: (800) 334- 3892, (312) 432-7603 (local). $15.95.

  Summary: This book presents answers from an internist specializing in women's health to a variety of questions women have about their health, including everyday health questions, symptoms, prevention, and treatments. The information is presented as a means to close the communication gap between women and their physicians. The information is organized alphabetically by subject. A list of resources for further information is included.

- **Heart health for black women: A natural approach to healing and preventing heart disease**

  Source: New York, NY: Marlowe. 2000. 244 pp.

  Contact: Available from Marlowe and Company, 841 Broadway, 4th Floor, New York, NY 10003. $15.95.

  Summary: This book for health and community service professionals and the general public, focuses on heart disease risks common among black women. Topics include high blood pressure, obesity, diabetes, nutrition, cholesterol, estrogen replacement therapy, and smoking. Alternative treatments such as herbs, vitamins, stress reduction techniques, and visualization techniques are discussed. Appendices include questions for healthcare providers, resources, a stay-on-track checklist, an exercise checklist, and a glossary. An index is provided.

- **Women and heart disease: An atlas of racial and ethnic disparities in mortality. (2nd ed.)**

  Source: Morgantown, WV: Office for Social Environment and Health Research, West Virginia University. 2000. 239 pp.

  Contact: Available from National Center for Chronic Disease Prevention and Health Promotion, 4770 Buford Highway, N.E., Mailstop K-30, Atlanta, GA 30341-3724. Telephone: (404) 488-5080 / fax: (404) 488-5969 / e-mail: ccdinfo@cdc.gov / Web site: http://www.cdc.gov/nccdphp/nccdhome.htm. Available at no charge; also available from the Web site at no charge.

  Summary: This publication provides local, state, and national statistical information essential to identify populations of women at greatest risk of heart disease and in greatest need of prevention efforts. Contained in five sections are topics on racial and ethnic disparities in heart disease among women; a reader's guide to understanding and

interpreting the statistical maps; local social environment and women's risk for heart disease mortality; national maps of heart disease mortality among women; and state maps of heart disease mortality among women. The appendices include: state rankings of heart disease mortality among women; methodological and technical notes; and resources for federal and state agencies, health organizations, and patient information. The atlas concludes with an index.

Book Summaries: Online Booksellers

Commercial Internet-based booksellers, such as Amazon.com and Barnes&Noble.com, offer summaries which have been supplied by each title's publisher. Some summaries also include customer reviews. Your local bookseller may have access to in-house and commercial databases that index all published books (e.g. Books in Print®). **IMPORTANT NOTE:** Online booksellers typically produce search results for medical and non-medical books. When searching for "heart disease" at online booksellers' Web sites, you may discover non-medical books that use the generic term "heart disease" (or a synonym) in their titles. The following is indicative of the results you might find when searching for "heart disease" (sorted alphabetically by title; follow the hyperlink to view more details at Amazon.com):

- **21st Century Complete Medical Guide to Heart Disease, Heart Attack, Cholesterol, Coronary Artery Disease, Bypass Surgery, Angioplasty ¿ Authoritative Federal Government Documents and Clinical References for Patients and Physicians with Practical Information on Diagnosis and Treatment Options** by PM Medical Health News; ISBN: 1931828342; http://www.amazon.com/exec/obidos/ASIN/1931828342/icongroupinterna
- **8 Steps to a Healthy Heart: The Complete Guide to Heart Disease Prevention and Recovery from Heart Attack and Bypass Surgery** by Robert E. Kowalski; ISBN: 0446516643; http://www.amazon.com/exec/obidos/ASIN/0446516643/icongroupinterna
- **Acute Coronary Syndromes: A Companion to Braunwald's Heart Disease** by Pierre Theroux (Editor); ISBN: 0721696139; http://www.amazon.com/exec/obidos/ASIN/0721696139/icongroupinterna
- **Acute Myocardial Infarction and Other Acute Ischemic Syndromes (Atlas of Heart Diseases, Vol 8)** by Robert M., MD Califf (Editor), et al; ISBN: 1878132326; http://www.amazon.com/exec/obidos/ASIN/1878132326/icongroupinterna
- **Alpha Lipoic Acid Breakthrough: The Superb Antioxidant That May Slow Aging, Repair Liver Damage, and Reduce the Risk of Cancer, Heart Disease, and Diabetes** by Burt Berkson, et al (1998); ISBN: 0761514570; http://www.amazon.com/exec/obidos/ASIN/0761514570/icongroupinterna
- **Alternative Medicine Guide to Heart Disease** by Burton Goldberg, et al (1997); ISBN: 1887299106; http://www.amazon.com/exec/obidos/ASIN/1887299106/icongroupinterna
- **Atlas of Heart Diseases** by Eugene Braunwald (Editor); ISBN: 1878132210; http://www.amazon.com/exec/obidos/ASIN/1878132210/icongroupinterna
- **Atlas of Heart Diseases: Vascular Disease (Atlas of Heart Diseases, Vol 7)** by Eugene Braunwald (Editor), et al; ISBN: 1878132261; http://www.amazon.com/exec/obidos/ASIN/1878132261/icongroupinterna

- **Atlas of Valvular Heart Disease: Clinical and Pathologic Aspects** by James T. Willerson (Editor), et al (1998); ISBN: 0443079536; http://www.amazon.com/exec/obidos/ASIN/0443079536/icongroupinterna
- **BEAT HEART DISEASE] - PHG**; ISBN: 0906348056; http://www.amazon.com/exec/obidos/ASIN/0906348056/icongroupinterna
- **Beta-Carotene and Other Carotenoids: The Antioxidant Family That Protects Against Cancer and Heart Disease and Strengthens the Immune System (Keats Good Health Guides)** by Richard A. Passwater, Susan Davis (Editor) (1996); ISBN: 0879837055; http://www.amazon.com/exec/obidos/ASIN/0879837055/icongroupinterna
- **Beyond Cholesterol: The Johns Hopkins Complete Guide for Avoiding Heart Disease** by Peter O., Jr. Kwiterovich (1989); ISBN: 0801838282; http://www.amazon.com/exec/obidos/ASIN/0801838282/icongroupinterna
- **Braunwald's Heart Disease: Review and Assessment to Accompany Braunwald's Heart Disease 6th Edition** by Leonard S. Lilly; ISBN: 0721694462; http://www.amazon.com/exec/obidos/ASIN/0721694462/icongroupinterna
- **Cardiac Problems in Pregnancy: Diagnosis and Management of Maternal and Fetal Heart Disease, 3rd Edition** by Uri Elkayam (Editor), Norbert Gleicher (Editor); ISBN: 0471163589; http://www.amazon.com/exec/obidos/ASIN/0471163589/icongroupinterna
- **Cholesterol and Coronary Heart Disease: The Great Debate** by Phil, M.D. Gold, et al; ISBN: 1850704147; http://www.amazon.com/exec/obidos/ASIN/1850704147/icongroupinterna
- **Chronic Ischemic Heart Disease: Chronic Ischemic Heart Disease (Atlas of Heart Diseases, Vol 5)** by George A. Beller (Editor), et al; ISBN: 1878132296; http://www.amazon.com/exec/obidos/ASIN/1878132296/icongroupinterna
- **Color Atlas of Cardiac Surgery: Acquired Heart Disease** by James L. Monro; ISBN: 0838511643; http://www.amazon.com/exec/obidos/ASIN/0838511643/icongroupinterna
- **Color Atlas of Congenital Heart Disease** by Siew Yeu Ho, et al (1995); ISBN: 0723417121; http://www.amazon.com/exec/obidos/ASIN/0723417121/icongroupinterna
- **Congenital Heart Disease Adult** by Welton M., Md Gersony, et al; ISBN: 0070329095; http://www.amazon.com/exec/obidos/ASIN/0070329095/icongroupinterna
- **Contemporary Diagnosis and Management of Valvular Heart Disease** by Jeffrey S. Borer; ISBN: 1884065589; http://www.amazon.com/exec/obidos/ASIN/1884065589/icongroupinterna
- **Coronary Heart Disease & Risk Factor Management: A Nursing Perspective** by Nalini Jairath, et al; ISBN: 0721668550; http://www.amazon.com/exec/obidos/ASIN/0721668550/icongroupinterna
- **Coronary heart disease and the mucopolysaccharides (glycosaminoglycans)** by Lester M. Morrison; ISBN: 0398029032; http://www.amazon.com/exec/obidos/ASIN/0398029032/icongroupinterna
- **Coronary Heart Disease Epidemiology: From Aetiology to Public Health (Oxford Medical Publications)** by Michael Marmot (Editor), et al (1997); ISBN: 0192625462; http://www.amazon.com/exec/obidos/ASIN/0192625462/icongroupinterna

- **Coronary Heart Disease in the Elderly** by Nanette K. Wenger (Editor), et al; ISBN: 0444011285;
  http://www.amazon.com/exec/obidos/ASIN/0444011285/icongroupinterna
- **Coronary Heart Disease Prevention** by Frank G. Yanowitz (Editor); ISBN: 0824787137;
  http://www.amazon.com/exec/obidos/ASIN/0824787137/icongroupinterna
- **Coronary Heart Disease: A Guide to Diagnosis and Treatment** by Barry M., Md. Cohen, et al; ISBN: 1886039585;
  http://www.amazon.com/exec/obidos/ASIN/1886039585/icongroupinterna
- **Coronary Heart Disease: The Facts (The Facts Series)** by Desmond Gareth Julian, Claire Marley (Contributor); ISBN: 0192619349;
  http://www.amazon.com/exec/obidos/ASIN/0192619349/icongroupinterna
- **Diagnosis and Management of Adult Congenital Heart Disease** by Michael A., Md Gatzoulis, et al (2003); ISBN: 0443071039;
  http://www.amazon.com/exec/obidos/ASIN/0443071039/icongroupinterna
- **Diagnosis: Heart Disease: Answers to Your Questions about Recovery and Lasting Health** by John W., Md Farquhar, et al; ISBN: 0393050122;
  http://www.amazon.com/exec/obidos/ASIN/0393050122/icongroupinterna
- **Diagnostic and Interventional Catheterization in Congenital Heart Disease** by James E. Lock (Editor), et al; ISBN: 0792385977;
  http://www.amazon.com/exec/obidos/ASIN/0792385977/icongroupinterna
- **Dr. Dean Ornish's Program for Reversing Heart Disease: The Only System Scientifically Proven to Reverse Heart Disease Without Drugs or Surgery** by Dean Ornish; ISBN: 0804110387;
  http://www.amazon.com/exec/obidos/ASIN/0804110387/icongroupinterna
- **Earl Mindell's Food As Medicine: What You Can Eat to Help Prevent Everything from Colds to Heart Disease to Cancer** by Earl Mindell (2002); ISBN: 0743226623;
  http://www.amazon.com/exec/obidos/ASIN/0743226623/icongroupinterna
- **Echocardiographic Diagnosis of Congenital Heart Disease: An Embryologic and Anatomic Approach** by Lilliam M. Valdes-Cruz (Editor), Raul O. Cayre (Editor); ISBN: 0781714338;
  http://www.amazon.com/exec/obidos/ASIN/0781714338/icongroupinterna
- **Echocardiography in Congenital Heart Disease Made Simple (Cardiopulmonary Medicine Series)** by S. Y. Ho, et al (2004); ISBN: 1860941249;
  http://www.amazon.com/exec/obidos/ASIN/1860941249/icongroupinterna
- **Echocardiography in Pediatric Heart Disease** by A. Rebecca Snider, et al (1997); ISBN: 0815178514;
  http://www.amazon.com/exec/obidos/ASIN/0815178514/icongroupinterna
- **Eisenhower's Heart Attack: How Ike Beat Heart Disease and Held on to the Presidency** by Clarence G. Lasby (1997); ISBN: 0700608222;
  http://www.amazon.com/exec/obidos/ASIN/0700608222/icongroupinterna
- **Essential Atlas of Heart Diseases** by Eugene Braunwald (Editor), et al; ISBN: 0071376453;
  http://www.amazon.com/exec/obidos/ASIN/0071376453/icongroupinterna
- **Evaluation of the Patient with Heart Disease: Integrating the Physical Exam and Echocardiography** by Jonathan, Md. Abrams (Editor), Carlos A.,MD Roldan (Editor);

ISBN: 0781724791;
http://www.amazon.com/exec/obidos/ASIN/0781724791/icongroupinterna

- **Greens Are Good for You!: How Green Power Protects You Against Heart Disease, Cancer, Diabetes, Macular Degeneration, Poor Night Vision, Senile Dementia, Liver Disease, fatigue** by Tony O'Donnell; ISBN: 1591200369;
http://www.amazon.com/exec/obidos/ASIN/1591200369/icongroupinterna
- **Has Heart Disease Been Cured** by Douglas Mulhall, Katja Hansen (2003); ISBN: 1932133739;
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- **Heal Your Heart: How You Can Prevent or Reverse Heart Disease** by K. Lance, Md. Gould (2000); ISBN: 0813528968;
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- **Health Journeys for People With High Blood Pressure or Heart Disease** by Belleruth Naparstek (Reader); ISBN: 1570420149;
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- **Healthy Heart Cookbook: Easy Delicious Recipes for Preventing and Managing Heart Disease** by Health Magazine (Editor) (2003); ISBN: 0848727665;
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- **Heart & Soul: A Psychological and Spiritual Guide to Preventing and Healing Heart Disease** by Bruno Cortis (1997); ISBN: 067155140X;
http://www.amazon.com/exec/obidos/ASIN/067155140X/icongroupinterna
- **Heart Disease and Erectile Dysfunction (Contemporary Cardiology)** by Robert A. Kloner (Editor) (2004); ISBN: 1588292169;
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- **Heart Disease and High Blood Pressure: How You Can Benefit from Diet, Vitamins, Minerals, Herbs, Exercise, and Other Natural Methods (Getting Well Naturally)** by Michael T. Murray (1997); ISBN: 0761506586;
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- **Heart Disease and Rehabilitation** by Michael L. Pollock (Editor), Donald H. Schmidt (Editor) (1995); ISBN: 0873225880;
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- **Heart Disease Diagnosis and Therapy: A Practical Approach** by M. Gabriel Khan, et al; ISBN: 0683046144;
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- **Heart Disease For Dummies®** by James M. Rippe (Author) (2004); ISBN: 0764541552;
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- **Heart Disease in Women** by Susan Wilansky (Editor), et al; ISBN: 0443079005;
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- **Heart Disease: A Textbook of Cardiovascular Medicine (2-Volume Set)** by Eugene Braunwald (Editor), et al; ISBN: 0721685617;
http://www.amazon.com/exec/obidos/ASIN/0721685617/icongroupinterna

- **Heart Disease: What You Should Know** by Douglas L. Wetherill, et al; ISBN: 0632045299; http://www.amazon.com/exec/obidos/ASIN/0632045299/icongroupinterna
- **Heart Failure: A Companion to Braunwald's Heart Disease** by Douglas L. Mann (Editor); ISBN: 0721694454; http://www.amazon.com/exec/obidos/ASIN/0721694454/icongroupinterna
- **Heart Fitness for Life: The Essential Guide for Preventing and Reversing Heart Disease** by Mary P. McGowan, et al (1999); ISBN: 0195129091; http://www.amazon.com/exec/obidos/ASIN/0195129091/icongroupinterna
- **Heart Fitness for Life: The Essential Guide to Preventing and Reversing Heart Disease** by Mary P., Md. McGowan, et al; ISBN: 0195116240; http://www.amazon.com/exec/obidos/ASIN/0195116240/icongroupinterna
- **Heart Healthy for Life: The Ultimate Guide to Preventing and Reversing Heart Disease** by Peter Jaret, Readers Digest (2003); ISBN: 076210452X; http://www.amazon.com/exec/obidos/ASIN/076210452X/icongroupinterna
- **Heart to Heart: A Cleveland Clinic Guide to Understanding Heart Disease and Open Heart Surgery** by Norman Richard, et al; ISBN: 0689118546; http://www.amazon.com/exec/obidos/ASIN/0689118546/icongroupinterna
- **Heart-Aches: Heart Disease and the Psychology of the Broken Heart** by Rudiger Dahlke, Rudiger Daklke (1996); ISBN: 1885394144; http://www.amazon.com/exec/obidos/ASIN/1885394144/icongroupinterna
- **Heartbreak and Heart Disease: A Mind/Body Prescription for Healing the Heart** by Stephen T., MD Sinatra; ISBN: 0879837233; http://www.amazon.com/exec/obidos/ASIN/0879837233/icongroupinterna
- **Her Healthy Heart: A Woman's Guide to Preventing and Reversing Heart Disease Naturally** by Linda Ojeda (1998); ISBN: 0897932250; http://www.amazon.com/exec/obidos/ASIN/0897932250/icongroupinterna
- **How to Fight Heart Disease & Win** by William L. Fischer; ISBN: 189143411X; http://www.amazon.com/exec/obidos/ASIN/189143411X/icongroupinterna
- **Internal Cleansing : Rid Your Body of Toxins to Naturally and Effectively Fight Heart Disease, Chronic Pain, Fatigue, PMS and Menopause Symptoms, and More (Revised 2nd Edition)** by Linda Berry, Jan Fawcett; ISBN: 0761529322; http://www.amazon.com/exec/obidos/ASIN/0761529322/icongroupinterna
- **Ischemic Heart Disease: A Rational Basis for Clinical Practise and Clinical Research** by Attilio Maseri; ISBN: 0443079102; http://www.amazon.com/exec/obidos/ASIN/0443079102/icongroupinterna
- **Ischemic Heart Disease: Clinical and Pathophysiological Aspects** by James T. Willerson; ISBN: 0890045631; http://www.amazon.com/exec/obidos/ASIN/0890045631/icongroupinterna
- **Ischemic Heart Disease: Surgical Management** by Brian Buxton (Editor), et al; ISBN: 0723429111; http://www.amazon.com/exec/obidos/ASIN/0723429111/icongroupinterna
- **Keep Your Heart Pumping: A Practical Guide to Heart Disease and Stroke Prevention** by Dr Lola Greene, Kate Kate Ewing (2003); ISBN: 0972941150; http://www.amazon.com/exec/obidos/ASIN/0972941150/icongroupinterna

- **Living a Healthy Life with Chronic Conditions: Self-Management of Heart Disease, Arthritis, Diabetes, Asthma, Bronchitis, Emphysema & Others** by Kate Lorig (Editor), et al; ISBN: 0923521534; http://www.amazon.com/exec/obidos/ASIN/0923521534/icongroupinterna
- **Living With Heart Disease** by Marie R. Squillace, et al; ISBN: 0737300825; http://www.amazon.com/exec/obidos/ASIN/0737300825/icongroupinterna
- **Manual of Lipid Disorders: Reducing the Risk for Coronary Heart Disease** by Antonio M., Jr., Md. Gotto, et al; ISBN: 078173584X; http://www.amazon.com/exec/obidos/ASIN/078173584X/icongroupinterna
- **Manual of Neonatal and Paediatric Heart Disease** by Fiona S., BscRgn Rscn Horrox, Guy Heaton (Illustrator); ISBN: 1861562446; http://www.amazon.com/exec/obidos/ASIN/1861562446/icongroupinterna
- **Miracle Heart: Preventing and Curing Heart Disease With Diet and Supplements** by Jean Carper (2003); ISBN: 0743403878; http://www.amazon.com/exec/obidos/ASIN/0743403878/icongroupinterna
- **Moss and Adams' Heart Disease in Infants, Children, and Adolescents : Including the Fetus and Young Adult (2 Volume Set)** by Hugh D., MD Allen (Editor), et al; ISBN: 0683307428; http://www.amazon.com/exec/obidos/ASIN/0683307428/icongroupinterna
- **Natural Alternatives to Hrt: Overcome Osteoporosis, Heart Disease, and Other Menopausal Conditions Without Risky Synthetic Hormone Replacement** by Rita Elkins (2003); ISBN: 1580543693; http://www.amazon.com/exec/obidos/ASIN/1580543693/icongroupinterna
- **Natural Medicine for Heart Disease: The Best Alternative Methods for Prevention and Treatment: High Cholesterol, High Blood Pressure, Stroke, Chest Pain, Other Circulatory Problems** by Glenn S., Md Rothfeld, et al; ISBN: 0875962890; http://www.amazon.com/exec/obidos/ASIN/0875962890/icongroupinterna
- **NovaCon - Coronary Heart Disease (CD-ROM)** by Frank H. Netter; ISBN: 0914168479; http://www.amazon.com/exec/obidos/ASIN/0914168479/icongroupinterna
- **Nutrition and Biotechnology in Heart Disease and Cancer (Advances in Experimental Medicine and Biology, 369)** by John B. Longenecker, et al; ISBN: 0306449943; http://www.amazon.com/exec/obidos/ASIN/0306449943/icongroupinterna
- **Nutrition and Heart Disease: Causation and Prevention** by Ronald R. Watson (Editor), Victor R. Preedy (Editor) (2003); ISBN: 084931674X; http://www.amazon.com/exec/obidos/ASIN/084931674X/icongroupinterna
- **Passing on Bypass Using External CounterPulsation : An FDA Cleared Alternative to Treat Heart Disease Without Surgery, Drugs or Angioplasty. SECOND EDITION** by George J. Juetersonke; ISBN: 096781281X; http://www.amazon.com/exec/obidos/ASIN/096781281X/icongroupinterna
- **Passing on Bypass Using External Counterpulsation : an FDA Cleared Alternative, to Treat Heart Disease Without Surgery, Drugs or Angioplasty!** by George J. Juetersonke, George J. Juetersonke; ISBN: 0967812801; http://www.amazon.com/exec/obidos/ASIN/0967812801/icongroupinterna
- **Pathophysiology of Heart Disease: A Collaborative Project of Medical Students and Faculty** by Leonard S. Lilly (Editor), Harvard Medical School (2002); ISBN: 0781740274; http://www.amazon.com/exec/obidos/ASIN/0781740274/icongroupinterna

- **Pathophysiology, Evaluation and Management of Valvular Heart Diseases: Developed from Valves in the Heart of the Big Apple Evaluation and Management of Valvular Heart Diseases, May 10-12, 2001, New York, N.Y. (Advances in Cardiology, 39)** by Jeffrey S. Borer (Editor), O. Wayne Isom (Editor) (2002); ISBN: 3805574029; http://www.amazon.com/exec/obidos/ASIN/3805574029/icongroupinterna
- **Permanent Remissions : Life-Extending Diet Stategies That Can Help Prevent and Reverse Cancer, Heart Disease, Diabets, and Osteoporosis** by Robert Haas, Kristin Massey (1997); ISBN: 0671007769; http://www.amazon.com/exec/obidos/ASIN/0671007769/icongroupinterna
- **Permanent Remissions: Life-Extending Diet Strategies That Can Help Prevent and Reverse Cancer, Heart Disease, Diabetes, and Osteoporosis** by Robert Haas; ISBN: 0671007777; http://www.amazon.com/exec/obidos/ASIN/0671007777/icongroupinterna
- **Plague Time: How Stealth Infections Cause Cancer, Heart Disease, and Other Deadly Ailments [BARGAIN PRICE]** by Paul Ewald (2000); ISBN: B0000C37E8; http://www.amazon.com/exec/obidos/ASIN/B0000C37E8/icongroupinterna
- **Practical Echocardiography of Congenital Heart Disease: From Fetus to Adult** by D. T., MD Linker (2000); ISBN: 0443076405; http://www.amazon.com/exec/obidos/ASIN/0443076405/icongroupinterna
- **Preventing Silent Heart Disease: Detecting and Preventing America's Number 1 Killer** by Harold L. Karpman, H. J. C. Swan; ISBN: 0517573008; http://www.amazon.com/exec/obidos/ASIN/0517573008/icongroupinterna
- **Prevention and rehabilitation in ischemic heart disease**; ISBN: 0683051504; http://www.amazon.com/exec/obidos/ASIN/0683051504/icongroupinterna
- **Primary Care Management of Heart Disease** by George Jesse Taylor (Editor), Sc Charleston; ISBN: 0323002560; http://www.amazon.com/exec/obidos/ASIN/0323002560/icongroupinterna
- **Recovering From Heart Disease in Body & Mind: Medical and Psychological Strategies for Living with Coronary Artery Disease** by Brian Harvey Baker, et al; ISBN: 0737303603; http://www.amazon.com/exec/obidos/ASIN/0737303603/icongroupinterna
- **Reversing Heart Disease: A Vital New Program to Help Prevent, Treat, and Eliminate Cardiac Problems Without Surgery** by Julian, MD Whitaker (2002); ISBN: 0446676578; http://www.amazon.com/exec/obidos/ASIN/0446676578/icongroupinterna
- **Soy Smart Health: Discover the "Super Food" That Fights Breast Cancer, Heart Disease, Osteoporosis, Menopausal Discomforts, and Estrogen Dominance** by Rita Elkins, et al; ISBN: 1580540449; http://www.amazon.com/exec/obidos/ASIN/1580540449/icongroupinterna
- **Stress and the Heart: Psychosocial Pathways to Coronary Heart Disease** by Bmj Books, et al; ISBN: 0727912771; http://www.amazon.com/exec/obidos/ASIN/0727912771/icongroupinterna
- **Syndrome X, the Silent Killer: The New Heart Disease Risk** by Gerald M. Reaven, et al (2001); ISBN: 0684868636; http://www.amazon.com/exec/obidos/ASIN/0684868636/icongroupinterna

- **Take a Load Off Your Heart: 109 Things You Can Do to Prevent or Reverse Heart Disease** by Joseph C. Piscatella, Barry A., Ph.D. Franklin (2003); ISBN: 0761126767; http://www.amazon.com/exec/obidos/ASIN/0761126767/icongroupinterna
- **The 10% Solution for a Healthy Life: How to Reduce Fat in Your Diet and Eliminate Virtually All Risk of Heart Disease and Cancer** by Raymond Kurzweil, et al (1995); ISBN: 0517883015; http://www.amazon.com/exec/obidos/ASIN/0517883015/icongroupinterna
- **The ABC's of Coronary Heart Disease** by James J. Maciejko (2001); ISBN: 1886947996; http://www.amazon.com/exec/obidos/ASIN/1886947996/icongroupinterna
- **The Bible Cure for Heart Disease (Health and Fitness)** by Don Colbert (1999); ISBN: 088419647X; http://www.amazon.com/exec/obidos/ASIN/088419647X/icongroupinterna
- **The Blood Thinner Cure : A Revolutionary Seven-Step Lifestyle Plan for Stopping Heart Disease and Stroke** by Kenneth R. Kensey M.D., Carol A. Turkington; ISBN: 0809298414; http://www.amazon.com/exec/obidos/ASIN/0809298414/icongroupinterna
- **The Carbohydrate Addict's Healthy Heart Program: Break Your Carbo-Insulin Connection to Heart Disease** by Richard F., Dr. Heller, et al; ISBN: 0345426118; http://www.amazon.com/exec/obidos/ASIN/0345426118/icongroupinterna
- **The Cardiac Recovery Handbook: The Complete Guide to Heart Disease and Recovery for Patients and Their Families** by Paul, Md. Kligfield, Paul Kligfield; ISBN: 1578261422; http://www.amazon.com/exec/obidos/ASIN/1578261422/icongroupinterna
- **The Carnitine Defense: A Nutraceutical Formula to Prevent and Treat Heart Disease, the Nation's #1 Killer** by Stephen L. DeFelice, et al; ISBN: 157954133X; http://www.amazon.com/exec/obidos/ASIN/157954133X/icongroupinterna
- **The Cholesterol Myths : Exposing the Fallacy that Saturated Fat and Cholesterol Cause Heart Disease** by Uffe Ravnskov; ISBN: 0967089700; http://www.amazon.com/exec/obidos/ASIN/0967089700/icongroupinterna
- **The Clinical Recognition of Congenital Heart Disease** by Joseph K. Perloff (2003); ISBN: 0721697305; http://www.amazon.com/exec/obidos/ASIN/0721697305/icongroupinterna
- **The Female Heart : The Truth About Women and Heart Disease** by Marianne J. Legato (Author), Carol Colman (Author) (2000); ISBN: 0688180655; http://www.amazon.com/exec/obidos/ASIN/0688180655/icongroupinterna
- **The Healing Power of Exercise : Your Guide to Preventing and Treating Diabetes, Depression, Heart Disease, High Blood Pressure, Arthritis, and More** by Linn Goldberg (Author), Diane L. Elliot (Author); ISBN: 0471348007; http://www.amazon.com/exec/obidos/ASIN/0471348007/icongroupinterna
- **The Heart Disease Breakthrough : What Even Your Doctor Doesn't Know about Preventing a Heart Attack** by Thomas Yannios M.D. (Author) (1999); ISBN: 0471255335; http://www.amazon.com/exec/obidos/ASIN/0471255335/icongroupinterna
- **The Heart Disease Sourcebook** by Roger, MD Cicala, Judith E. Brown (1998); ISBN: 0737300205; http://www.amazon.com/exec/obidos/ASIN/0737300205/icongroupinterna

- **The Heart of the Matter : A Revolutionary Program for Preventing Coronary Heart Disease** by Peter Salgo (Author), Joe Layden (Author) (2004); ISBN: 0060544287; http://www.amazon.com/exec/obidos/ASIN/0060544287/icongroupinterna
- **The High Blood Pressure Solution: A Scientifically Proven Program for Preventing Strokes and Heart Disease** by Richard D. Moore M.D. Ph.D., Ph.D., Richard, D. Moore M.D. ISBN: 0892819758; http://www.amazon.com/exec/obidos/ASIN/0892819758/icongroupinterna
- **The Human Heart: A Basic Guide to Heart Disease** by Brendan, Md. Phibbs, Christopher Wikoff (Illustrator) (1997); ISBN: 0316705136; http://www.amazon.com/exec/obidos/ASIN/0316705136/icongroupinterna
- **The Immortality Enzyme: Aging, Cancer & Heart Disease** by Phillip Minton; ISBN: 1884367054; http://www.amazon.com/exec/obidos/ASIN/1884367054/icongroupinterna
- **The Inflammation Cure : How to Combat the Hidden Factor Behind Heart Disease, Arthritis, Asthma, Diabetes, & Other Diseases** by William Joel Meggs, et al; ISBN: 0071413200; http://www.amazon.com/exec/obidos/ASIN/0071413200/icongroupinterna
- **The Inflammation Syndrome: The Complete Nutritional Program to Prevent and Reverse Heart Disease, Arthritis, Diabetes, Allergies, and Asthma** by Jack Challem (Author); ISBN: 0471202711; http://www.amazon.com/exec/obidos/ASIN/0471202711/icongroupinterna
- **The Johns Hopkins White Papers 2002, Volume 1: Arthritis, Coronary Heart Disease, Depression and Anxiety, Diabetes, Digestive Disorders** by Simeon Margolis (2002); ISBN: 0929661710; http://www.amazon.com/exec/obidos/ASIN/0929661710/icongroupinterna
- **The Johns Hopkins White Papers: Coronary Heart Disease** by Gary Gerstenblith, Simeon Margolis (2002); ISBN: 0929661125; http://www.amazon.com/exec/obidos/ASIN/0929661125/icongroupinterna
- **The McDougall Program for a Healthy Heart: A Life-Saving Approach to Preventing and Treating Heart Disease** by Mary McDougall, John A., M.D. McDougall (1998); ISBN: 0452272661; http://www.amazon.com/exec/obidos/ASIN/0452272661/icongroupinterna
- **The Miracle Heart : The Ultimate Guide to Preventing and Curing Heart Disease With Diet and Supplements** by Jean Carper (Author); ISBN: 0061013838; http://www.amazon.com/exec/obidos/ASIN/0061013838/icongroupinterna
- **The Modern Nutritional Diseases: And How to Prevent Them: Heart Disease, Stroke, Type-2 Diabetes, Obesity, Cancer** by Fred Ottoboni, et al (2002); ISBN: 091524103X; http://www.amazon.com/exec/obidos/ASIN/091524103X/icongroupinterna
- **The Multiple Sclerosis Diet Book: A Low-Fat Diet for the Treatment of M.S., Heart Disease, and Stroke** by Roy Laver, Swank, Mary-Helen Pullen; ISBN: 0385120923; http://www.amazon.com/exec/obidos/ASIN/0385120923/icongroupinterna
- **The New 8-Week Cholesterol Cure : The Ultimate Program for Preventing Heart Disease** by Robert E. Kowalski (Author) (2002); ISBN: 0061031763; http://www.amazon.com/exec/obidos/ASIN/0061031763/icongroupinterna

- **The New American Heart Association Cookbook: Fighting Heart Disease and Stroke** by American Heart Association (Editor) (2001); ISBN: 0609808907; http://www.amazon.com/exec/obidos/ASIN/0609808907/icongroupinterna
- **The Right Heart in Congenital Heart Disease** by Robert H. Anderson (Editor), et al; ISBN: 1900151847; http://www.amazon.com/exec/obidos/ASIN/1900151847/icongroupinterna
- **The Taste for Living WORLD Cookbook: More of Mike Milken's Favorite Recipes for Fighting Cancer and Heart Disease** by Beth Ginsberg, et al; ISBN: 0967365503; http://www.amazon.com/exec/obidos/ASIN/0967365503/icongroupinterna
- **The Total Guide to a Healthy Heart: Integrative Strategies for Preventing and Reversing Heart Disease** by Seth J. Baum (2000); ISBN: 157566562X; http://www.amazon.com/exec/obidos/ASIN/157566562X/icongroupinterna
- **The Vitamin E Factor : The Miraculous Antioxidant for the Prevention and Treatment of Heart Disease, Cancer, and Aging** by Andreas Papas (1999); ISBN: 0060984430; http://www.amazon.com/exec/obidos/ASIN/0060984430/icongroupinterna
- **The Wellness Lowfat Cookbook: Hundreds of Delicious Recipes & A Revolutionary New Eating Plan That Can Help Prevent Heart Disease** by Editors of the University of Ca. Et Al., et al; ISBN: 0929661117; http://www.amazon.com/exec/obidos/ASIN/0929661117/icongroupinterna
- **Thriving With Heart Disease : A Unique Program for You and Your Family / Live Happier, Healthier, Longer** by Wayne Sotile (Author), Robin Cantor-Cooke (Contributor); ISBN: 0743243641; http://www.amazon.com/exec/obidos/ASIN/0743243641/icongroupinterna
- **Thriving With Heart Disease : The Leading Authority on the Emotional Effects of Heart Disease Tells You and Your Family How to Heal and Reclaim Your Lives** by Wayne Sotile (Author) (2004); ISBN: 074324365X; http://www.amazon.com/exec/obidos/ASIN/074324365X/icongroupinterna
- **Tomato Power: Lycopene: The Miracle Nutrient That Can Prevent Aging, Heart Disease and Cancer** by James F. Scheer, et al (1999); ISBN: 1889462047; http://www.amazon.com/exec/obidos/ASIN/1889462047/icongroupinterna
- **Valvular Heart Disease** by Catherine M. Otto (2004); ISBN: 0721697879; http://www.amazon.com/exec/obidos/ASIN/0721697879/icongroupinterna
- **Valvular Heart Disease** by Joseph S. Alpert (Editor), et al; ISBN: 0781723108; http://www.amazon.com/exec/obidos/ASIN/0781723108/icongroupinterna
- **Valvular Heart Disease** by Muayed Al Zaibag, et al; ISBN: 0824788613; http://www.amazon.com/exec/obidos/ASIN/0824788613/icongroupinterna
- **Valvular Heart Disease and Endocarditis (Atlas of Heart Diseases, V. 11)** by Shahbudin H. Rahimtoola (Editor), et al; ISBN: 187813230X; http://www.amazon.com/exec/obidos/ASIN/187813230X/icongroupinterna
- **What Every Woman Must Know About Heart Disease: A No-Nonsense Approach to Diagnosing, Treating, and Preventing the #1 Killer of Women** by Siegfried J. Kra, Kra M D Siegfried J (1997); ISBN: 0446395323; http://www.amazon.com/exec/obidos/ASIN/0446395323/icongroupinterna
- **What to Eat if You Have Heart Disease : Nutritional Therapy for the Prevention and Treatment of Cardiovascular Disease** by Maureen Keane, Daniella Chace; ISBN:

0809229676;
http://www.amazon.com/exec/obidos/ASIN/0809229676/icongroupinterna

- **What Women Need to Know: From Headaches to Heart Disease and Everything in Between** by Carol Colman, Marianne Legato; ISBN: 1587541009;
http://www.amazon.com/exec/obidos/ASIN/1587541009/icongroupinterna
- **Women & Heart Disease** by Desmond G. Julian, Nanette Kass Wenger; ISBN: 1853172871;
http://www.amazon.com/exec/obidos/ASIN/1853172871/icongroupinterna
- **Women Are Not Small Men: Life-Saving Strategies for Preventing and Healing Heart Disease in Women** by Nieca Goldberg (2002); ISBN: 0345440986;
http://www.amazon.com/exec/obidos/ASIN/0345440986/icongroupinterna
- **Women, Stress, and Heart Disease** by Kristina Orth-Gomer (Editor), et al (1998); ISBN: 0805821244;
http://www.amazon.com/exec/obidos/ASIN/0805821244/icongroupinterna
- **You Can Beat Heart Disease: How to Defeat America's #1 Killer** by Lester R. Sauvage, Jerry Goldstone (2002); ISBN: 0966378857;
http://www.amazon.com/exec/obidos/ASIN/0966378857/icongroupinterna

The National Library of Medicine Book Index

The National Library of Medicine at the National Institutes of Health has a massive database of books published on healthcare and biomedicine. Go to the following Internet site, **http://locatorplus.gov/**, and then select "Search LOCATORplus." Once you are in the search area, simply type "heart disease" (or synonyms) into the search box, and select "books only." From there, results can be sorted by publication date, author, or relevance. The following was recently catalogued by the National Library of Medicine:[11]

- **Carcinoid heart disease: a clinical, pathologic, and therapeutic update** Author: Strickman, Neil E.; Year: 1959; Chicago: Year Book Medical Publishers, c1982; ISBN: 0815199155
http://www.amazon.com/exec/obidos/ASIN/0815199155/icongroupinterna
- **Cor pulmonale; pulmonary heart disease.** Author: Bhargava, R. K.; Year: 1962; [Mount Kisco, N. Y.] Futura Pub. Co., 1973; ISBN: 087993007
- **Nature of rheumatic heart disease; with special reference to myocardial disease and heart failure.** Author: Murphy, George Edward,; Year: 1966; Baltimore, Williams; Wilkins [c1960]
- **Rheumatic and coronary heart disease; a medical-surgical symposium, sponsored by St. Barnabas Hospital, New York City.** Author: Bailey, Charles Philamore,; Year: 1965; Philadelphia, Lippincott [c1967]

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[11] In addition to LOCATORPlus, in collaboration with authors and publishers, the National Center for Biotechnology Information (NCBI) is currently adapting biomedical books for the Web. The books may be accessed in two ways: (1) by searching directly using any search term or phrase (in the same way as the bibliographic database PubMed), or (2) by following the links to PubMed abstracts. Each PubMed abstract has a "Books" button that displays a facsimile of the abstract in which some phrases are hypertext links. These phrases are also found in the books available at NCBI. Click on hyperlinked results in the list of books in which the phrase is found. Currently, the majority of the links are between the books and PubMed. In the future, more links will be created between the books and other types of information, such as gene and protein sequences and macromolecular structures. See **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Books**.

- **Rheumatic fever and rheumatic heart disease; a review of research grants supported by the National Heart Institute 1949 to 1966, by May Sherman. Subject index by Ellen M. Barnes. Bibliography by Judith L. Ellin.** Author: National Heart Institute (U.S.); Year: 1961; Bethesda, Md., National
- **Rheumatic heart disease and mitral valve disease** Author: Fitzmaurice, Joan B.; Year: 1966; New York: Appleton-Century-Crofts, c1980; ISBN: 083858439X http://www.amazon.com/exec/obidos/ASIN/083858439X/icongroupinterna
- **Rheumatic heart disease; pathology and clinical implications, a summary of five hundred and nine autopsied cases, by Jacques B. Wallach and Edgar F. Borgatta, in collaboration with Alfred A. Angrist.** Author: Wallach, Jacques B. (Jacques Burton),; Year: 1967; Springfield, Ill., Thomas [c1962]
- **What you can do for rheumatic fever & rheumatic heart disease control in Michigan; a summary of the Michigan Rheumatic Fever study.** Author: Michigan Rheumatic Fever Study for the Prevention and Control of Rheumatic Fever and Rheumatic Heart Disease in Michigan.; Year: 1967; Southfield, Michigan

Chapters on Heart Disease

In order to find chapters that specifically relate to heart disease, an excellent source of abstracts is the Combined Health Information Database. You will need to limit your search to book chapters and heart disease using the "Detailed Search" option. Go to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. To find book chapters, use the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer, and the format option "Book Chapter." Type "heart disease" (or synonyms) into the "For these words:" box. The following is a typical result when searching for book chapters on heart disease:

- **Reduction in Risk of Coronary Heart Disease and Diabetes**

  Source: in Devlin, J.T. and Schneider, S.H., eds. Handbook of Exercise in Diabetes. Alexandria, VA: American Diabetes Association. 2002. p. 155-181.

  Contact: Available from American Diabetes Association (ADA). Order Fulfillment Department, P.O. Box 930850, Atlanta, GA 31193-0850. (800) 232-6733. Fax (770) 442-9742. Website: www.diabetes.org. PRICE: $69.95 plus shipping and handling. ISBN: 1580400191.

  Summary: This chapter is from a book that provides a practical, comprehensive guide to diabetes and exercise for health care professionals involved in patient care. In this chapter, the authors consider strategies that may result in a reduction in risk of coronary heart disease (CHD) and type 2 diabetes. Increased physical activity improves the cardiovascular risk factor profile; its effects include reducing adiposity, blood pressure, dyslipidemia, and platelet adhesives, as well as enhancing fibrinolysis. Increased physical activity may also reduce CHD risk independently of favorably alterations in traditional coronary risk factors. The estimated reduction in the risk of CHD with the maintenance of an active, compared with a sedentary, lifestyle is estimated to be 35 to 55 percent. Physical activity improves insulin sensitivity and glycemic control among nondiabetic individuals, as well as among those with impaired glucose tolerance (IGT) or overt type 2 diabetes. The addition of exercise to caloric restriction facilitates loss of adipose tissue, assists in maintenance of reduced body weight, and may independently improve insulin sensitivity. The potential reduction in the risk of type 2 diabetes

associated with an active, compared with a sedentary, lifestyle is 30 to 50 percent. 2 tables. 101 references.

- **Regional Body Fat Distribution, the Insulin Resistance-Dyslipidemic Syndrome, and the Risk of Type 2 Diabetes and Coronary Heart Disease**

  Source: in Devlin, J.T. and Schneider, S.H., eds. Handbook of Exercise in Diabetes. Alexandria, VA: American Diabetes Association. 2002. p. 197-234.

  Contact: Available from American Diabetes Association (ADA). Order Fulfillment Department, P.O. Box 930850, Atlanta, GA 31193-0850. (800) 232-6733. Fax (770) 442-9742. Website: www.diabetes.org. PRICE: $69.95 plus shipping and handling. ISBN: 1580400191.

  Summary: Visceral adipose tissue accumulation is an important factor to consider in the evaluation of health risks associated with obesity. This chapter is from a book that provides a practical, comprehensive guide to diabetes and exercise for health care professionals involved in patient care. In this chapter, the authors consider regional body fat distribution, the insulin resistance-dyslipidemic syndrome, and the risk of type 2 diabetes and coronary heart disease. The simultaneous presence of hyperinsulinemia, hyperapolipoprotein B, and small, dense LDL particles is associated with a 20 fold increase in the risk of ischemic heart disease. A simple and inexpensive screening test to identify a high risk form of abdominal obesity is to determine if the patient has the following: a waist circumference greater than 90 centimeters and triglyceride levels greater than 2.0 mmol per liter. Improvements in the metabolic risk profile resulting from endurance exercise training are more related to the volume of exercise than to its intensity. The authors conclude that from a public health standpoint, the greatest benefit would be to transform the largely sedentary population into moderately active individuals. Physicians and health professionals should keep in mind that the changes in lifestyle associated with the best compliance are those that are likely to have the greatest long term impact on cardiovascular health. 8 figures. 3 tables. 148 references.

- **Diabetic Neuropathy and Coronary Heart Disease**

  Source: in Reece, E.A. and Coustan, D.R., eds. Diabetes Mellitus in Pregnancy. 2nd ed. New York, NY: Churchill Livingstone. 1995. p. 345-351.

  Contact: Available from Churchill Livingstone. 300 Lighting Way, Secaucus, NJ 07094. (800) 553-5426. PRICE: $92.00. ISBN: 0443089795.

  Summary: This chapter, from a text on diabetes mellitus in pregnancy, focuses on diabetic neuropathy and coronary heart disease. The authors discuss the potential serious impact of autonomic neuropathy on the pregnancy complicated by diabetes, and reviews other forms of neuropathy so that the reader will be able to recognize them, should they become manifested during pregnancy. Topics include autonomic neuropathy, peripheral neuropathy, cranial neuropathy, neuropathic fractures, and coronary artery disease. The authors conclude that the presence of either coronary artery disease or gastroparesis may lead to increased morbidity and mortality risks for pregnant women with IDDM. 2 figures. 1 table. 26 references. (AA-M).

- **Rheumatic Fever, Rheumatic Heart Disease, and Murmurs**

  Source: in Little, J.W., et al. Dental Management of the Medically Compromised Patient. 5th ed. St. Louis, MO: Mosby, Inc. 1997. p. 131-143.

Contact: Available from Harcourt Health Sciences. 11830 Westline Industrial Drive, St. Louis, MO 63146. (800) 325-4177. Fax (800) 874-6418. Website: www.harcourthealth.com. PRICE: $48.00 plus shipping and handling. ISBN: 0815156340.

Summary: A working knowledge of the multitude of compromised health states is essential for dental professionals, as the majority of medically compromised patients need or want oral health care. This chapter on rheumatic fever, rheumatic heart disease, and murmurs is from a text that provides the dental practitioner with an up to date reference work describing the dental management of patients with selected medical problems. Patients with a history of rheumatic fever may have residual cardiac damage and rheumatic heart disease. These patients need to be given prophylactic antibiotic therapy during dental treatment to prevent infective endocarditis. The authors discuss incidence and prevalence of these conditions, pathophysiology and complication, signs and symptoms, the medical management of patients with a history of rheumatic fever, and considerations for the dental management of this population. A final section discusses patients with heart murmurs: innocent, or functional murmurs are sounds caused by turbulence in the absence of any cardiac abnormality; they do not require antibiotic prophylaxis. Organic murmurs are sounds caused by a pathologic abnormality in the heart; they do require antibiotic prophylaxis. The authors stress that dentists, for the most part, are not trained to detect or evaluate heart murmurs and should thus rely on a physician colleague to perform these tasks. 6 figures. 3 tables. 25 references.

- **Congenital Heart Disease**

  Source: in Little, J.W., et al. Dental Management of the Medically Compromised Patient. 5th ed. St. Louis, MO: Mosby, Inc. 1997. p. 144-155.

  Contact: Available from Harcourt Health Sciences. 11830 Westline Industrial Drive, St. Louis, MO 63146. (800) 325-4177. Fax (800) 874-6418. Website: www.harcourthealth.com. PRICE: $48.00 plus shipping and handling. ISBN: 0815156340.

  Summary: A working knowledge of the multitude of compromised health states is essential for dental professionals, as the majority of medically compromised patients need or want oral health care. This chapter on congenital heart disease is from a text that provides the dental practitioner with an up to date reference work describing the dental management of patients with selected medical problems. The prime concern of the dentist in dealing with a patient who has congenital heart disease is the prevention of bacterial endocarditis (BE) and bacterial endarteritis (BEA). The authors discuss incidence and prevalence of the condition, its etiology (including genetic causes), pathophysiology and complication, specific congenital heart defects, signs and symptoms (clinical presentation and laboratory findings), the medical management of patients with congenital heart disease, and the dental management of this population. 8 figures. 9 tables. 16 references.

- **Ischemic Heart Disease**

  Source: in Little, J.W., et al. Dental Management of the Medically Compromised Patient. 5th ed. St. Louis, MO: Mosby, Inc. 1997. p. 192-205.

  Contact: Available from Harcourt Health Sciences. 11830 Westline Industrial Drive, St. Louis, MO 63146. (800) 325-4177. Fax (800) 874-6418. Website: www.harcourthealth.com. PRICE: $48.00 plus shipping and handling. ISBN: 0815156340.

Summary: A working knowledge of the multitude of compromised health states is essential for dental professionals, as the majority of medically compromised patients need or want oral health care. This chapter on ischemic heart disease is from a text that provides the dental practitioner with an up to date reference work describing the dental management of patients with selected medical problems. Ischemic heart disease is coronary atherosclerosis that is symptomatic; the symptoms are the result of oxygen deprivation consequent to reduced vascular flow to the heart. Other conditions such as embolism, coronary ostial stenosis, coronary artery spasm, and congenital abnormalities also may cause ischemic heart disease. The authors discuss incidence and prevalence of the condition, its etiology (including lifestyle factors), pathophysiology and complications, specific congenital heart defects, signs and symptoms (clinical presentation and laboratory findings), the medical management of patients with angina pectoris or myocardial infarction (heart attack), and the dental management of this population. 4 figures. 5 tables. 37 references.

Directories

In addition to the references and resources discussed earlier in this chapter, a number of directories relating to heart disease have been published that consolidate information across various sources. The Combined Health Information Database lists the following, which you may wish to consult in your local medical library:[12]

- **Who? What? Where?: Resources for women's health and aging**

  Source: Bethesda, MD: National Institute on Aging, U.S. Department of Health and Human Services. 1992. 82 pp.

  Contact: Available from National Institute on Aging Information Center, P.O. Box 8057, Gaithersburg, MD 20898-8057. Telephone: (301) 587-2528 or (800) 222-2225 or (800) 222-4225 TDD / fax: (301) 589-3014 / e-mail: NIAINFO@access.digex.net / Web site: http://www.nih.gov/nia. Available at no charge.

  Summary: This directory lists resources available to women which can help them cope with the processes of aging. The first section provides resources for health promotion and prevention. Topics included here are skin care, nutrition and exercise, and menopause. The second section concentrates on common disorders associated with age such as osteoporosis, urinary incontinence, cancer, and **This director.** The third section focuses on other aspects of women's lives such as widowhood, finances, and caregiving. The final sections provided information on women's health research and other organizations and readings.

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[12] You will need to limit your search to “Directory” and “heart disease” using the "Detailed Search" option. Go directly to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. To find directories, use the drop boxes at the bottom of the search page where “You may refine your search by.” For publication date, select “All Years.” Select your preferred language and the format option “Directory.” Type “heart disease” (or synonyms) into the “For these words:” box. You should check back periodically with this database as it is updated every three months.

CHAPTER 8. MULTIMEDIA ON HEART DISEASE

Overview

In this chapter, we show you how to keep current on multimedia sources of information on heart disease. We start with sources that have been summarized by federal agencies, and then show you how to find bibliographic information catalogued by the National Library of Medicine.

Video Recordings

An excellent source of multimedia information on heart disease is the Combined Health Information Database. You will need to limit your search to "Videorecording" and "heart disease" using the "Detailed Search" option. Go directly to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. To find video productions, use the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer, and the format option "Videorecording (videotape, videocassette, etc.)." Type "heart disease" (or synonyms) into the "For these words:" box. The following is a typical result when searching for video recordings on heart disease:

- **Diabetes and Heart Disease**

  Source: Timonium, MD: Milner-Fenwick. 2000. (videocassette).

  Contact: Available from Milner-Fenwick, Inc. 2125 Greenspring Drive, Timonium, MD 21093-3100. (800) 432-8433. Fax (410) 252-6316. PRICE: $125.00; bulk orders available; plus shipping and handling.

  Summary: The goal of this video program is to help patients with diabetes understand and minimize their risk of heart disease. The program helps viewers assess their individual risks and demonstrates that it is never too late to make positive lifestyle changes to reduce those risks that can be controlled. The program covers working with the health care team, health food choices, exercise, smoking cessation, stress management, and the importance of regular check ups. The program also explains the signs and symptoms of a heart attack and what to do if they occur. The videotape was produced in cooperation with the American Association of Diabetes Educators (AADE), which defined the content of the video, selected the program consultants, and approved

production at each stage of development. The program is closed-captioned and available in English or Spanish.

Bibliography: Multimedia on Heart Disease

The National Library of Medicine is a rich source of information on healthcare-related multimedia productions including slides, computer software, and databases. To access the multimedia database, go to the following Web site: **http://locatorplus.gov/**. Select "Search LOCATORplus." Once in the search area, simply type in heart disease (or synonyms). Then, in the option box provided below the search box, select "Audiovisuals and Computer Files." From there, you can choose to sort results by publication date, author, or relevance. The following multimedia has been indexed on heart disease:

- **A Roentgenologic approach to heart disease [slide]** Source: Larry P. Elliott; Year: 1974; Format: Slide; New York: Medcom, c1974
- **Acquired heart disease: radiologic aspects [slide]** Source: Robert N. Cooley; Year: 1976; Format: Slide; New York: Medcom, 1976
- **Coronary atherosclerotic heart disease [videorecording]** Source: [South Carolina Health Communications Network]; Year: 1971; Format: Videorecording; [Charleston, S. C.]: The Network, 1971
- **Cyanotic congenital heart disease [motion picture]** Source: University of Washington, School of Medicine; produced in the Department of Medical Illustration; Year: 1953; Format: Motion picture; Seattle: The School; [for loan by the Health Sciences Center for Educational Resources, 1953]
- **Diagnosis and management of congenital heart disease [videorecording]** Source: Children's National Medical Center; Year: 2003; Format: Videorecording; Cherry Hill, NJ: CMEinfo.com., 2003
- **Diagnosis of atherosclerotic heart disease [videorecording]** Source: Emory University School of Medicine; Year: 1973; Format: Videorecording; Atlanta: Georgia Regional Medical Television Network: [for loan or sale by A. W. Calhoun Medical Library, 1973]
- **Diagnosis of congenital heart disease [motion picture]** Source: Univ. Centre of Cardiology, Geneva. [et al.]; produced by Condor Films; Year: 1964; Format: Motion picture; Ardsley, N. Y.: Geigy Pharmaceuticals, [1964]
- **Introduction to congenital heart disease [slide]** Source: National Medical Audiovisual Center in cooperation with the Medical College of Georgia; Year: 1973; Format: Slide; [Atlanta]: The Center; [Washington: for sale by National Audiovisual Center], 1973
- **Oxygen therapy in heart disease [motion picture]** Source: presented by the Linde Air Products Company, Unit of Union Carbide and Carbon Corporation; produced in collaboration with the American Heart Association, Inc. produced for the Linde Air Products Compan; Year: 1946; Format: Motion picture; [United States]: The Company, c1946
- **Recognition and management of valvular heart disease [videorecording]** Source: produced for Central Washington Project by the Information and Education Resource Support Unit; Format: Videorecording; United States: [s.n., 196-?]
- **Risk factors: the odds for heart disease [slide]** Source: William Hazzard, John Bolles; Year: 1974; Format: Slide; Seattle, Wash.: Univ. of Washington; [for loan and sale by its Health Sciences Center for Educational Resources, 1974]

- **Surgical management of coronary atherosclerotic heart disease [videorecording]** Source: Emory University School of Medicine; Year: 1977; Format: Videorecording; Atlanta: Georgia Regional Medical Television Network: [for loan or sale by A. W. Calhoun Medical Library, 1977]
- **The electrocardiogram in arteriosclerotic heart disease [motion picture]** Source: [production company unknown; C.J. Lundy]; Year: 1933; Format: Motion picture; [S.l.: s.n., 1933]
- **The electrocardiogram in rheumatic heart disease [motion picture]** Source: [production company unknown; C.J. Lundy]; Year: 1933; Format: Motion picture; [S.l.: s.n., 1933]
- The normal heart sounds [motion picture]: recorded simultaneously with the electrocardiogram; abnormal heart sounds in rheumatic heart disease; the systolic murmur Source: [production company unknown; C.J. Lundy]; Year: 1933; Format: Motion picture; [S.l.: s.n., 1933]
- **The surgical management of valvular heart disease [videorecording]** Source: produced for Central Washington Project by the Information and Education Resource Unit; Format: Videorecording; United States: [s.n., 196-?]
- **Tricuspid and pulmonic valve replacement for carcinoid heart disease [videorecording]** Source: Mount Sinai School of Medicine, Division of Cardiothoracic Surgery; authors, Mohey K. Saleh, Arisan Ergin, Randall B. Griepp; Year: 1987; Format: Videorecording; [New York, N.Y.]: The School, c1987

CHAPTER 9. PERIODICALS AND NEWS ON HEART DISEASE

Overview

In this chapter, we suggest a number of news sources and present various periodicals that cover heart disease.

News Services and Press Releases

One of the simplest ways of tracking press releases on heart disease is to search the news wires. In the following sample of sources, we will briefly describe how to access each service. These services only post recent news intended for public viewing.

PR Newswire

To access the PR Newswire archive, simply go to **http://www.prnewswire.com/**. Select your country. Type "heart disease" (or synonyms) into the search box. You will automatically receive information on relevant news releases posted within the last 30 days. The search results are shown by order of relevance.

Reuters Health

The Reuters' Medical News and Health eLine databases can be very useful in exploring news archives relating to heart disease. While some of the listed articles are free to view, others are available for purchase for a nominal fee. To access this archive, go to **http://www.reutershealth.com/en/index.html** and search by "heart disease" (or synonyms). The following was recently listed in this archive for heart disease:

- **Heart disease worse killer than cancer, women told**
  Source: Reuters Health eLine
  Date: September 26, 2003
  http://www.reutershealth.com/archive/2003/09/26/eline/links/20030926elin001.html

- **Family history of heart disease tied to birth weight**
  Source: Reuters Health eLine
  Date: September 19, 2003

- **Family history of heart disease tied to higher risk of birth of low birth weight infant**
  Source: Reuters Medical News
  Date: September 19, 2003

- **Revascularization ups survival in kidney disease patients with heart disease**
  Source: Reuters Medical News
  Date: September 18, 2003

- **High dietary fiber intake can reduce coronary heart disease risk**
  Source: Reuters Medical News
  Date: September 09, 2003

- **Silent heart disease should be considered in stroke patients**
  Source: Reuters Medical News
  Date: September 08, 2003

- **Silent heart disease often seen in stroke patients**
  Source: Reuters Health eLine
  Date: September 08, 2003

- **Most heart disease attributable to common risk factors**
  Source: Reuters Medical News
  Date: August 19, 2003

- **Subtle signs point to early diabetic heart disease**
  Source: Reuters Health eLine
  Date: August 12, 2003

- **Subtle LV dysfunction seen with early diabetic heart disease**
  Source: Reuters Medical News
  Date: August 12, 2003

- **HRT does not slow progression of heart disease in postmenopausal women**
  Source: Reuters Industry Breifing
  Date: August 06, 2003

- **Myocardial perfusion measured by MRI useful in detecting heart disease**
  Source: Reuters Medical News
  Date: August 01, 2003

- **MRI may detect heart disease**
  Source: Reuters Health eLine
  Date: August 01, 2003

- **Hemophilia carriers protected from heart disease**
  Source: Reuters Health eLine
  Date: August 01, 2003

- **Hemophilia carriers are protected against fatal ischemic heart disease**
  Source: Reuters Medical News
  Date: July 31, 2003

- **High blood pressure in teens predicts heart disease**
  Source: Reuters Health eLine
  Date: July 29, 2003

- **HIV patients have more heart disease risk factors**
  Source: Reuters Health eLine
  Date: July 23, 2003

- **Coronary heart disease risk factors more common in HIV-infected patients**
  Source: Reuters Medical News
  Date: July 23, 2003

- **Anti-diabetes drug may prevent heart disease**
  Source: Reuters Health eLine
  Date: July 23, 2003

- **Acarbose may cut heart disease risk in patients with impaired glucose tolerance**
  Source: Reuters Industry Breifing
  Date: July 23, 2003

- **Depression tied to myocardial ischemia in patients with ischemic heart disease**
  Source: Reuters Medical News
  Date: July 22, 2003

- **U.S. approves test to help predict heart disease**
  Source: Reuters Health eLine
  Date: July 21, 2003

- **Subclinical atherosclerosis associated with parental coronary heart disease**
  Source: Reuters Medical News
  Date: July 21, 2003

- **ICAM-1 levels may explain heart disease risk in women with prior preeclampsia**
  Source: Reuters Medical News
  Date: July 17, 2003

- **Antibodies to oral pathogens associated with heart disease**
  Source: Reuters Medical News
  Date: July 17, 2003

- **Vitamin C use may lower heart disease risk**
  Source: Reuters Medical News
  Date: July 15, 2003

The NIH

Within MEDLINEplus, the NIH has made an agreement with the New York Times Syndicate, the AP News Service, and Reuters to deliver news that can be browsed by the public. Search news releases at **http://www.nlm.nih.gov/medlineplus/alphanews_a.html**. MEDLINEplus allows you to browse across an alphabetical index. Or you can search by date at the following Web page: **http://www.nlm.nih.gov/medlineplus/newsbydate.html**. Often, news items are indexed by MEDLINEplus within its search engine.

Business Wire

Business Wire is similar to PR Newswire. To access this archive, simply go to **http://www.businesswire.com/**. You can scan the news by industry category or company name.

Market Wire

Market Wire is more focused on technology than the other wires. To browse the latest press releases by topic, such as alternative medicine, biotechnology, fitness, healthcare, legal, nutrition, and pharmaceuticals, access Market Wire's Medical/Health channel at **http://www.marketwire.com/mw/release_index?channel=MedicalHealth**. Or simply go to Market Wire's home page at **http://www.marketwire.com/mw/home**, type "heart disease" (or synonyms) into the search box, and click on "Search News." As this service is technology oriented, you may wish to use it when searching for press releases covering diagnostic procedures or tests.

Search Engines

Medical news is also available in the news sections of commercial Internet search engines. See the health news page at Yahoo (**http://dir.yahoo.com/Health/News_and_Media/**), or you can use this Web site's general news search page at **http://news.yahoo.com/**. Type in "heart disease" (or synonyms). If you know the name of a company that is relevant to heart disease, you can go to any stock trading Web site (such as **http://www.etrade.com/**) and search for the company name there. News items across various news sources are reported on indicated hyperlinks. Google offers a similar service at **http://news.google.com/**.

BBC

Covering news from a more European perspective, the British Broadcasting Corporation (BBC) allows the public free access to their news archive located at **http://www.bbc.co.uk/**. Search by "heart disease" (or synonyms).

Newsletters on Heart Disease

Find newsletters on heart disease using the Combined Health Information Database (CHID). You will need to use the "Detailed Search" option. To access CHID, go to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. Limit your search to "Newsletter" and "heart disease." Go to the bottom of the search page where "You may refine your search by." Select the dates and language that you prefer. For the format option, select "Newsletter." Type "heart disease" (or synonyms) into the "For these words:" box. The following list was generated using the options described above:

- **Health Span. [Newsletter]**

  Source: Novato, CA: Buck Center for Research in Aging. 1990 -. [8 p. average].

  Contact: Available from Buck Center for Research in Aging. 505A San Marin Drive, Suite 300, Novato, CA 94945. (415) 899-1800. PRICE: Free.

  Summary: This newsletter, published by the Buck Center for Research in Aging in Marin County, California, includes articles on all aspects of health concerns affecting the aged. The issue examined (Fall 1991) contains articles about Alzheimer's disease research, **This newslett,** skin cancers, and osteoporosis. The articles range from short reports of recent research activities, to health hints, to reports on national needs in the field of aging. The newsletter also deals specifically with the needs of the aging in Marin County and with the goals of Buck Center For Research in Aging, which is seeking county permission to build a scientific research facility.

Newsletter Articles

Use the Combined Health Information Database, and limit your search criteria to "newsletter articles." Again, you will need to use the "Detailed Search" option. Go directly to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. Go to the bottom of the search page where "You may refine your search by." Select the dates and language that you prefer. For the format option, select "Newsletter Article." Type "heart disease" (or synonyms) into the "For these words:" box. You should check back periodically with this database as it is updated every three months. The following is a typical result when searching for newsletter articles on heart disease:

- **Ask the Doctor: What Is the Connection Between Lupus and Heart Disease?**

  Source: Lupus Foundation Network. 4; Summer 1996.

  Contact: Lupus Network Inc. 230 Ranch Drive, Bridgeport, CT 06606. (203) 372-5795.

  Summary: This newsletter article for individuals with systemic lupus erythematosus (SLE) discusses the connection between SLE and heart disease. The most common cardiac manifestation of SLE is pericarditis, which is an inflammation of the lining

around the heart. Myocarditis, an inflammation of the heart muscle itself, occurs only in a small number of patients. Recent data have shown an increased rate of heart attacks in young women with lupus. Although the specific cause of this increased rate of heart attacks is unknown, it is probably related to long-term use of steroids, inflammation of blood vessels, and the presence of lupus antibody. The symptoms, diagnosis, and treatment of some of these cardiac manifestations are presented.

- **Diabetes and Heart Disease: New Strategies Emerge**

  Source: Harvard Heart Letter. 10(11): 1-4. July 2000.

  Contact: Available from Harvard Medical School Health Publications Group. Harvard Heart Letter, P.O. Box 420300, Palm Coast, FL 32142-0300. (800) 829-9045. E-mail: harvardmed@palmcoastd.com. Website: www.health.harvard.edu.

  Summary: This article explores the relationship between diabetes and cardiovascular disease. Diabetes is a risk factor for atherosclerosis in the blood vessels of the heart and throughout the body. In addition, other risk factors for heart disease are closely associated with diabetes, including obesity, hypertension, and lipid abnormalities. Although the death rates due to coronary heart disease have been steadily declining over the last few decades, this has not been the case for people who have diabetes. Middle aged women with diabetes have the same increased risk for heart disease as do men. In addition, people who have diabetes and have had heart attacks have a less favorable prognosis than heart attack victims without diabetes. Therefore, most experts recommend that physicians regard all people who have diabetes as heart disease patients, even if they show no signs of cardiovascular problems. Studies have shown that beta blockers such as atenolol, metoprolol, nadolol, and propranolol are among the best drugs to treat coronary artery disease. Doctors traditionally have avoided prescribing beta blockers for people who have diabetes because they can mask the warning signs of low blood glucose and can worsen some problems common in people who have diabetes such as impotence and fatigue. However, research suggests that people who have diabetes may derive even greater benefits from beta blockers when compared with people who do not have diabetes. In addition, research suggests that tight diabetes control can reduce the risk of other diabetes complications. Other studies have investigated the outcomes between people with and without diabetes following balloon angioplasty. Results suggest that angioplasty in people who have diabetes leaves more heart muscle in danger than does bypass surgery. Thus, most physicians create treatment plans under the assumption that bypass surgery is the best form of treatment for people who have diabetes and severe symptoms of coronary disease that has not responded to drug treatment.

- **Heart Disease Handbook, Part 3: Triglycerides Turn Troublesome**

  Source: Environmental Nutrition. 20(4): 1, 6. April 1997.

  Contact: Available from Environmental Nutrition. P.O. Box 420451, Palm Coast, FL 32142-0451. (800) 829-5384.

  Summary: This newsletter article describes triglycerides, or fat (lipids). Triglycerides from food travel through the bloodstream by way of chylomicrons, the largest of the lipid carriers (needed because fat and blood do not mix). Other triglycerides come from the liver, which manufactures them from excess carbohydrates and alcohol as a way to store energy. These triglycerides travel through blood via carriers called very low-density lipoproteins (VLDLs). The author discusses the problems occurring with high triglyceride levels, notably risk for heart disease. A recent study of 201 men and women

found that high triglycerides increase heart disease risk independently. Treatment should include ways to control levels of triglycerides. Diet is the first line of treatment; drugs are used only when patients already have heart disease or when triglyceride levels exceed 500. For people who are overweight, simply losing weight can lower triglycerides. Other dietary suggestions include getting or staying physically active, cutting back on saturated fats, limiting carbohydrates to 55 percent of calories or less, avoiding alcohol, and discussing fish oil supplements with a physician. (AA-M).

- **Oral Health and Heart Disease**

  Source: Harvard Health Letter. 11(7): 1-3. March 2001.

  Contact: Available from Harvard Health Publications. P.O. Box 420300, Palm Coast, FL 32142-0300. (800) 829-9045. Website: www.health.harvard.edu.

  Summary: This article explores the recent evidence that by averting gum disease, patients might actually be reducing their chances for developing heart disease. The author notes that, at the very least, it seems clear that people with worse dental health have a higher risk of heart attack. Recent findings link periodontal disease to cardiovascular disease. Periodontal disease is any disease, including gingivitis or periodontitis, that affects the gums and associated membranes. However, not all of these studies adequately controlled for other risk factors (for example, socioeconomic status, age, or unhealthy behaviors). Poor dental health may consequently have been an indication of poor personal hygiene or suboptimal health habits. The author cautions that the observed increase in heart disease risk among those with poor dental health may have reflected a general lack of health care, rather than a lack of dental care in particular. The article also reports on present Harvard studies that are evaluating the role of inflammation and diet as potential mediators. For example, periodontal disease and resulting tooth loss may lead to poor dietary habits that, in turn, might increase heart disease risk. The article concludes by hypothesizing the role of inflammation (the body's response to infection or injury) in heart disease. 1 figure.

- **Bad Teeth and Gums a Risk Factor for Heart Disease?**

  Source: Harvard Heart Letter. 9(3): 6-7. November 1998.

  Contact: Available from Harvard Heart Letter. P.O. Box 420379, Palm Coast, FL 32142-0379. (800) 829-9171. E-mail: harvardhhl@palmcoastd.com. Website: www.countway.harvard.edu/publications/Health_Publications.

  Summary: This newsletter article informs readers about recent studies that are investigating the role of bad teeth and gums as risk factors for heart disease. Several studies found that patients with heart disease had more tooth decay and higher rates of gum disease. A link between oral health and heart disease was also found in epidemiological studies that examined people's teeth and then followed them for several years to see if those with poor dental health were more likely to get heart disease. For example, people with inflammation of the gums (periodontitis) had a 25 percent increased risk of heart disease over the follow up period, compared to those with minimal periodontitis. The author notes that one reason why many scientists have been skeptical about poor dental health as a cause of heart disease is that there was no obvious way in which the two conditions could be linked. Some experts now speculate that the bacteria that accumulate in the gums and teeth of people with poor dental hygiene cause inflammation that leads to an increase in blood clotting factors. If so, these clotting factors might increase the chances that a blood clot could form in the heart. Another theory considers the role of white blood cells that fight infection in the

gums and elsewhere. The author concludes that these theories sound promising, but at present remain unproven.

- **For Women Only: 14 Questions That Could Reveal Hidden Heart Disease**

  Source: Tufts University Health and Nutrition Letter. 17(12):4-5. February 2000.

  Contact: Tufts University Health and Nutrition Letter, 500 Broadway, 15th Floor, New York, NY 10004.

  Summary: According to Laura Kimble, associate professor of nursing at Emory University, a woman's ability to do housework can be an indication of how healthy her heart is. Because the state of their house is very important to most women, they will clean house even when they do not feel well. Therefore, if they begin to let household chores go, then they must be feeling very ill. Further, most physicians ask about a patient's ability to do physical activity, but most women do not consider housework to be physical activity. However, for many women, housework is the only physical activity they do on a regular basis. Kimble therefore recommends that physicians ask women, especially older women, how well they are able to do their household work as a measure of their heart health. Kimble has created a Household Activities Scale to help women measure their ability to perform physical activities.

- **Phytochemical Watch: Resveratrol Helps Prevent Cancer, Heart Disease**

  Source: American Institute for Cancer Research Newsletter. Issue 69, p.8. Fall 2000.

  Contact: American Institute for Cancer Research. 1759 R St. NW, Washington, DC 20009. (202)328-7744.

  Summary: Scientists have identified the phytochemical resveratrol as a promising cancer preventative. Resveratrol seems to fight cancer in three ways: blocking the action of cancer-causing agents, inhibiting the development and growth of tumors, and causing precancerous cells to revert to normal. Resveratrol also shows significant promise in controlling heart disease by working as an antioxidant to help prevent the oxidation and build-up of fatty plaques in blood vessels, and as an anticoagulant, similar to aspirin. Resveratrol is found in at least 72 different plants, including mulberries and peanuts, but grapes and grape products are the richest sources. Research thus far has been limited to cell cultures and animal studies. It is as yet unknown if results will be similar in humans and what amount of resveratrol is needed to produce beneficial effects.

Academic Periodicals covering Heart Disease

Numerous periodicals are currently indexed within the National Library of Medicine's PubMed database that are known to publish articles relating to heart disease. In addition to these sources, you can search for articles covering heart disease that have been published by any of the periodicals listed in previous chapters. To find the latest studies published, go to **http://www.ncbi.nlm.nih.gov/pubmed**, type the name of the periodical into the search box, and click "Go."

If you want complete details about the historical contents of a journal, you can also visit the following Web site: **http://www.ncbi.nlm.nih.gov/entrez/jrbrowser.cgi**. Here, type in the name of the journal or its abbreviation, and you will receive an index of published articles. At **http://locatorplus.gov/**, you can retrieve more indexing information on medical

periodicals (e.g. the name of the publisher). Select the button "Search LOCATORplus." Then type in the name of the journal and select the advanced search option "Journal Title Search."

APPENDICES

APPENDIX A. PHYSICIAN RESOURCES

Overview

In this chapter, we focus on databases and Internet-based guidelines and information resources created or written for a professional audience.

NIH Guidelines

Commonly referred to as "clinical" or "professional" guidelines, the National Institutes of Health publish physician guidelines for the most common diseases. Publications are available at the following by relevant Institute[13]:

- Office of the Director (OD); guidelines consolidated across agencies available at **http://www.nih.gov/health/consumer/conkey.htm**
- National Institute of General Medical Sciences (NIGMS); fact sheets available at **http://www.nigms.nih.gov/news/facts/**
- National Library of Medicine (NLM); extensive encyclopedia (A.D.A.M., Inc.) with guidelines: **http://www.nlm.nih.gov/medlineplus/healthtopics.html**
- National Cancer Institute (NCI); guidelines available at **http://www.cancer.gov/cancerinfo/list.aspx?viewid=5f35036e-5497-4d86-8c2c-714a9f7c8d25**
- National Eye Institute (NEI); guidelines available at **http://www.nei.nih.gov/order/index.htm**
- National Heart, Lung, and Blood Institute (NHLBI); guidelines available at **http://www.nhlbi.nih.gov/guidelines/index.htm**
- National Human Genome Research Institute (NHGRI); research available at **http://www.genome.gov/page.cfm?pageID=10000375**
- National Institute on Aging (NIA); guidelines available at **http://www.nia.nih.gov/health/**

[13] These publications are typically written by one or more of the various NIH Institutes.

- National Institute on Alcohol Abuse and Alcoholism (NIAAA); guidelines available at **http://www.niaaa.nih.gov/publications/publications.htm**
- National Institute of Allergy and Infectious Diseases (NIAID); guidelines available at **http://www.niaid.nih.gov/publications/**
- National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS); fact sheets and guidelines available at **http://www.niams.nih.gov/hi/index.htm**
- National Institute of Child Health and Human Development (NICHD); guidelines available at **http://www.nichd.nih.gov/publications/pubskey.cfm**
- National Institute on Deafness and Other Communication Disorders (NIDCD); fact sheets and guidelines at **http://www.nidcd.nih.gov/health/**
- National Institute of Dental and Craniofacial Research (NIDCR); guidelines available at **http://www.nidr.nih.gov/health/**
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK); guidelines available at **http://www.niddk.nih.gov/health/health.htm**
- National Institute on Drug Abuse (NIDA); guidelines available at **http://www.nida.nih.gov/DrugAbuse.html**
- National Institute of Environmental Health Sciences (NIEHS); environmental health information available at **http://www.niehs.nih.gov/external/facts.htm**
- National Institute of Mental Health (NIMH); guidelines available at **http://www.nimh.nih.gov/practitioners/index.cfm**
- National Institute of Neurological Disorders and Stroke (NINDS); neurological disorder information pages available at **http://www.ninds.nih.gov/health_and_medical/disorder_index.htm**
- National Institute of Nursing Research (NINR); publications on selected illnesses at **http://www.nih.gov/ninr/news-info/publications.html**
- National Institute of Biomedical Imaging and Bioengineering; general information at **http://grants.nih.gov/grants/becon/becon_info.htm**
- Center for Information Technology (CIT); referrals to other agencies based on keyword searches available at **http://kb.nih.gov/www_query_main.asp**
- National Center for Complementary and Alternative Medicine (NCCAM); health information available at **http://nccam.nih.gov/health/**
- National Center for Research Resources (NCRR); various information directories available at **http://www.ncrr.nih.gov/publications.asp**
- Office of Rare Diseases; various fact sheets available at **http://rarediseases.info.nih.gov/html/resources/rep_pubs.html**
- Centers for Disease Control and Prevention; various fact sheets on infectious diseases available at **http://www.cdc.gov/publications.htm**

NIH Databases

In addition to the various Institutes of Health that publish professional guidelines, the NIH has designed a number of databases for professionals.[14] Physician-oriented resources provide a wide variety of information related to the biomedical and health sciences, both past and present. The format of these resources varies. Searchable databases, bibliographic citations, full-text articles (when available), archival collections, and images are all available. The following are referenced by the National Library of Medicine:[15]

- **Bioethics:** Access to published literature on the ethical, legal, and public policy issues surrounding healthcare and biomedical research. This information is provided in conjunction with the Kennedy Institute of Ethics located at Georgetown University, Washington, D.C.: **http://www.nlm.nih.gov/databases/databases_bioethics.html**
- **HIV/AIDS Resources:** Describes various links and databases dedicated to HIV/AIDS research: **http://www.nlm.nih.gov/pubs/factsheets/aidsinfs.html**
- **NLM Online Exhibitions:** Describes "Exhibitions in the History of Medicine": **http://www.nlm.nih.gov/exhibition/exhibition.html**. Additional resources for historical scholarship in medicine: **http://www.nlm.nih.gov/hmd/hmd.html**
- **Biotechnology Information:** Access to public databases. The National Center for Biotechnology Information conducts research in computational biology, develops software tools for analyzing genome data, and disseminates biomedical information for the better understanding of molecular processes affecting human health and disease: **http://www.ncbi.nlm.nih.gov/**
- **Population Information:** The National Library of Medicine provides access to worldwide coverage of population, family planning, and related health issues, including family planning technology and programs, fertility, and population law and policy: **http://www.nlm.nih.gov/databases/databases_population.html**
- **Cancer Information:** Access to cancer-oriented databases: **http://www.nlm.nih.gov/databases/databases_cancer.html**
- **Profiles in Science:** Offering the archival collections of prominent twentieth-century biomedical scientists to the public through modern digital technology: **http://www.profiles.nlm.nih.gov/**
- **Chemical Information:** Provides links to various chemical databases and references: **http://sis.nlm.nih.gov/Chem/ChemMain.html**
- **Clinical Alerts:** Reports the release of findings from the NIH-funded clinical trials where such release could significantly affect morbidity and mortality: **http://www.nlm.nih.gov/databases/alerts/clinical_alerts.html**
- **Space Life Sciences:** Provides links and information to space-based research (including NASA): **http://www.nlm.nih.gov/databases/databases_space.html**
- **MEDLINE:** Bibliographic database covering the fields of medicine, nursing, dentistry, veterinary medicine, the healthcare system, and the pre-clinical sciences: **http://www.nlm.nih.gov/databases/databases_medline.html**

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[14] Remember, for the general public, the National Library of Medicine recommends the databases referenced in MEDLINE*plus* (**http://medlineplus.gov/** or **http://www.nlm.nih.gov/medlineplus/databases.html**).

[15] See **http://www.nlm.nih.gov/databases/databases.html**.

- **Toxicology and Environmental Health Information (TOXNET):** Databases covering toxicology and environmental health: **http://sis.nlm.nih.gov/Tox/ToxMain.html**
- **Visible Human Interface:** Anatomically detailed, three-dimensional representations of normal male and female human bodies: **http://www.nlm.nih.gov/research/visible/visible_human.html**

The Combined Health Information Database

A comprehensive source of information on clinical guidelines written for professionals is the Combined Health Information Database. You will need to limit your search to one of the following: Brochure/Pamphlet, Fact Sheet, or Information Package, and "heart disease" using the "Detailed Search" option. Go directly to the following hyperlink: **http://chid.nih.gov/detail/detail.html**. To find associations, use the drop boxes at the bottom of the search page where "You may refine your search by." For the publication date, select "All Years." Select your preferred language and the format option "Fact Sheet." Type "heart disease" (or synonyms) into the "For these words:" box. The following is a sample result:

- **Assisting Employees With Life - Threatening Illnesses**

  Contact: Bank of America National Trust and Savings Association, Bank America Corporation, Corporate Health, PO Box 37000, San Francisco, CA, 94137, (415) 241-3016.

  Summary: This statement, from a banking company, explains the policies and procedures that managers should follow in dealing with situations involving employees with life-threatening illnesses such as cancer, **heart disease,** and Human immunodeficiency virus (HIV) infection. The guidelines emphasize ill employees' rights to be treated consistently with other employees, so long as their conditions are not a threat to themselves or to others. Confidentiality issues, referrals, employee education, consultation, and employee and coworker transfers are discussed.

- **AIDSCOM: Reducing HIV Transmission, Lessons From the Past**

  Contact: Academy for Educational Development, 1255 23rd St NW, Washington, DC, 20037, (202) 884-8000.

  Summary: This report summarizes what is known about how education programs can reduce the transmission of Human immunodeficiency virus (HIV), which causes Acquired immunodeficiency syndrome (AIDS); identifies evaluation problems; and suggests areas for future research and investment. The report outlines early AIDS prevention programs and discusses possible correlations to reduced sexual transmission of HIV. It analyzes existing national AIDS health programs and lessons learned about HIV prevention. Finally, it compares HIV prevention efforts to those directed against other lifestyle diseases, including other Sexually transmitted diseases (STD's), cancer, **heart disease,** family planning and contraception, child survival, and smallpox survival. Major findings indicate that current evidence cannot prove a direct correlation between educational intervention and reduced HIV seroconversion, but there is a probable link between information and risk reduction. The report also states that personal loss seems to correlate with risk reduction, changes in knowledge are easier to produce than changes in behavior or attitude, information source credibility is significant in the success of behavior modification, and changes in drug use have been more difficult to produce than changes in sexual behavior. Effective education goes beyond information to include service delivery, counseling, needle exchange, and condom distribution, it

states, while external competition has reduced the effectiveness of positive education campaigns, and people selectively believe AIDS information for illogical reasons. Continuing programs are needed to sustain behavior modification. The report outlines principles of effective HIV prevention education: Programs must be based on factual, scientific information; prevention messages must be consistent, clear, and effective; programs must reflect the awareness spectrum; programs must enable people to modify their behavior and maintain those changes over time; and HIV prevention must build a community consensus.

- **AIDS Resource Manual: Workplace Policy #1. Workplace Policy #2**

  Contact: Vermont Department of Health, Division of Health Surveillance, PO Box 70, Burlington, VT, 05402-0070, (802) 863-7246, http://www.state.vt.us/health/_hs/aids/aids.htm.

  Summary: This statement presents two policy options for businesses to adopt in the workplace dealing with Human immunodeficiency virus (HIV) and Acquired immunodeficiency syndrome (AIDS). Both policies make the same general points, including approval for Persons with AIDS (PWA's) to work so long they meet performance standards and are no threat to themselves or others; the obligation of the company to provide a safe work environment for all; and the importance of education about serious illness. Policy #1 refers to serious illnesses such as cancer, **heart disease,** and HIV/AIDS, while policy #2 refers only to AIDS and AIDS-related complex (ARC).

- **Disability Statistics Report (14): Mobility Device Use in the United States**

  Source: Washington, DC: U.S. Department of Education, National Institute on Disability and Rehabilitation Research. 2000. 60 p.

  Contact: Available from U.S. Department of Education, OSERS, National Institute on Disability and Rehabilitation Research (NIDRR). Attn: David Keer. Switzer Building, Room 3431, Washington, DC 20202. (202) 205-5633. E-mail: david_keer@ed.gov. Website: www.ed.gov/offices/OSERS/NIDRR.

  Summary: This report provides health professionals, community service professionals, and people who have disabilities with information on mobility device use in the United States. The report provides data on the population using mobility devices, focusing on their age, gender, race and ethnicity, educational attainment, employment and labor force participation, family income, and area of residence. This is followed by data on health and disability status, including self reported health status, hospitalization history, perceived disability status, activity limitation, functional limitation, activities of daily living, and instrumental activities of daily living. The report also examines health conditions and impairments associated with mobility device use. The leading conditions associated with mobility device use among persons of all ages include osteoarthritis (OA) and allied disorders, cerebrovascular disease, orthopedic impairment of a lower extremity, orthopedic impairment of the back or neck, intervertebral disc disorders, senility without psychosis, **heart disease,** rheumatoid arthritis and other inflammatory polyarthropathies, orthopedic impairment of the hip or pelvis, and chronic injuries. OA is the top ranked condition responsible for disability among users of canes, walkers, and crutches. OA, the most prevalent main cause of disability among mobility device users of all ages, is also the primary cause of disability among working age adults and the elderly. The report concludes with data on accessibility features and problems both inside and outside the home and on health insurance coverage. 24 figures, 26 tables, and 5 references.

- **Medical Consequences**

  Source: in Seventh Special Report to the U.S. Congress on Alcohol and Health. Rockville, MD: National Institute on Alcohol Abuse and Alcoholism. 1990. p. 107-138.

  Contact: Available from National Clearinghouse for Alcohol and Drug Information. 6000 Executive Boulevard, P.O. Box 2345, Rockville, MD 20852. (800) 729-6686. PRICE: Single copy free.

  Summary: Alcohol affects almost every organ system in the body either directly or indirectly. This chapter, from a special report on alcohol and health, discusses the medical consequences of alcohol use and abuse. Topics include alcohol-induced liver disorders; effects of alcohol on the gastrointestinal tract; nutritional and metabolic disorders; effects on the cardiovascular system, including the heart, the vascular system, and coronary **heart disease;** effects on the immune system; alcohol and cancer; effects on endocrine and reproductive functions; and neurologic disorders. The authors conclude that new concepts and technological advances have great potential to accelerate progress in understanding the biomedical consequences of alcohol dependence and in developing improved methods to treat and prevent the consequences of alcohol dependence and alcohol abuse. 6 figures. 256 references.

- **Economics of Diabetes and Diabetes Care: A Report of the Diabetes Health Economics Study Group**

  Source: Brussels, Belgium: International Diabetes Federation. 1996. 136 p.

  Contact: Available from International Diabetes Federation. 1 Rue Defacqz B, 1000 Brussels, Belgium. Phone 32-2-538-55-11. Fax 32-2-538-51-14. E-mail: idf@idf.org. PRICE: Single copy free. ISBN: 2930229012.

  Summary: This report is designed for those who are assessing the economic impact of diabetes and the efficiency of various methods of providing care, those who are involved in the formulation of health policy, and those involved in training health care professionals. The authors note that the prevalence of diabetes, particularly noninsulin-dependent diabetes mellitus (NIDDM or Type II), is increasing dramatically, especially in developing countries. The World Health Organization (WHO) estimates that diabetes currently affects approximately 130 million people worldwide, and will affect about 300 million people by 2025. Seven sections provide information about diabetes, health economics, the impact of diabetes on the individual, regional health care systems, the global financial burden of diabetes, reducing the burden of diabetes, and methodology and research. According to the report, research priorities in diabetes health economics include the following: obtaining additional information on the costs of diabetes and the cost-effectiveness of treatment; standardizing methods of collecting and analyzing economic data; linking clinical and cost data; assessing the cost-effectiveness of various models of care; defining appropriate interventions; identifying and measuring outcomes; assessing the costs, benefits and cost-effectiveness of diabetes education; assessing the costs and benefit of preventing and treating **heart disease** in people with diabetes; and encouraging further collaboration between professional and lay people involved in diabetes care. An executive summary and a glossary are included in the report. 2 figures. 17 tables. 385 references. (AA-M).

- **Health Risks of Obesity: Special Report. 2nd ed**

  Source: Hettinger, ND: Obesity and Health, Healthy Living Institute. 1993. 190 p.

Contact: Available from Obesity and Health. 402 South 14th Street, Hettinger, ND 58639. (701) 567-2845. Fax (701) 567-2443. PRICE: $65.

Summary: This report sets forth current information on the health risks of obesity. The report is intended to help educators, policy makers, and health care providers deal more effectively with the complexities and dilemmas of obesity. Eleven chapters address the following topics: the health risks of obesity in the areas of **heart disease,** stroke, cancer, diabetes, and other related diseases; fat distribution; obesity in ethnic populations, notably the high risk of diabetes in the Native American population; early puberty; leanness and aging; the risks of losing weight; effectiveness of treatment; weight cycling; mortality and weight loss; treatment decisions; and challenges for the future. Numerous appendices present information about measuring and defining obesity and two NIH conferences.

- **The UnitedHealth Group state health rankings: Results, methodology and discussion**

  Source: Minneapolis, MN: UnitedHealth Group. 2000-. annual.

  Contact: Available from UnitedHealth Group, P.O. Box 1459, MR MN008-W109, Minneapolis, MN 55440-1459 / Web site: http://www.unitedhealthgroup.com. Available at no charge.

  Summary: This report for public officials, health care organizations, and consumer groups provides a snapshot of health indicators for each state and establishes a base line for monitoring changes over time by bringing together multiple measures and calculating an overall score and ranking. Health indicators include the components of lifestyle, access, occupational safety and disability, disease, and mortality. Additional information is provided in the report describing the scores and rankings; how components are selected, described, and weighted; the survey methodology; the state-by-state snapshots; and the component tables. The appendices include information on occupational fatalities and risk for **heart disease.** The report concludes with an index of tables and figures.

- **The oral-systemic health connection**

  Source: Bethesda, MD: National Institute of Dental and Craniofacial Research. 1999. 15 pp.

  Contact: Available from National Institute of Dental and Craniofacial Research, Building 45, Room 4AS19, 9000 Rockville Pike, Bethesda, MD 20892-6400. Telephone: (301) 496-4261 publications / fax: (301) 496- 9988 / Web site: http://www.nidcr.nih.gov. Available at no charge.

  Summary: This report describes the relationship between periodontal disease and other health problems such as chronic degenerative diseases, diabetes mellitus, heart diseases, low birth weight, AIDS, Sjogrens syndrome, and xerostomia. The report discusses current and projected research in the field.

- **Eliminating racial and ethnic disparities in health: A chartbook**

  Source: Washington, DC: Grantmakers in Health. 1998. 52 pp.

  Contact: Available from Grantmakers In Health, 1100 Connecticut Avenue, N.W., Suite 1200, Washington, DC 20036. Telephone: (202) 452-8331 / fax: (202) 452-8340 / e-mail: wcarr@gih.org / Web site: http://www.gih.org.

Summary: This chartbook presents data on racial and ethnic disparities in the six health areas identified by President Clinton in his February 1998 radio address: infant mortality, cancer screening and management, **heart disease,** diabetes, HIV / AIDS, and immunization. Data are also presented that set the context for considering these issues. In addition, data are presented on the disparities in risk factors that contribute to the incidence of diabetes, **heart disease,** cancer, and other health conditions. The chartbook concludes with sections on health care use, insurance coverage, and the health professions. The data are primarily drawn from federal sources.

- **Healthy lifestyles: Nutrition and physical activity**

  Source: Washington, DC: International Life Science Institute. 1998. 59 pp.

  Contact: Available from ILSI Press, 1126 16th Street, N.W, Washington, DC 20036-4810. Telephone: (202) 659-0074 / fax: (202) 659-8654.

  Summary: This report provides current information on the relationship between nutrition and a healthy lifestyle. It includes an overview of concepts that play a role in health status. Individual chapters based on research studies cover the following topics: dietary fat and energy balance; dietary fat and coronary **heart disease;** antioxidant nutrients; dietary fiber; fluid intake; alcohol consumption and health; physical activity; and oral health. A glossary of dietary and health terms is included.

- **Food, nutrition, and the prevention of cancer: A global perspective**

  Source: Washington, DC: American Institute for Cancer Research. 1997. 670 pp.

  Contact: Available from American Institute for Cancer Research, 1759 R Street, N.W., P.O. Box 97167, Washington, DC 20090. Telephone: (202) 328-7744. $35.00 includes shipping and handling.

  Summary: This report presents dietary guidelines for cancer prevention, public policy recommendations for cancer prevention, and a review of the science behind the findings reported. The report reviews the scientific and other expert literature linking foods, nutrition, food preparation, and dietary patterns and related factors with the risk of human cancers worldwide. A series of dietary and other recommendations designed as suitable for all societies and to reduce the risk of human cancers is presented. An evaluation of the degree of consistency between such recommendations and those proposed for the prevention of coronary **heart disease** and other diseases is discussed. And both the feasibility and the policy implications of the global implementation of these recommendations are considered.

- **In their own words: Adolescent girls discuss health and health care issues**

  Source: New York, NY: Commonwealth Fund. 1997. 70 pp.

  Contact: Available from Commonwealth Fund, One East 75 Street, New York, NY 10021-2692. Telephone: (888) 777-2744 or (212) 606-3840 / Web site: http://www.cmwf.org. Available at no charge.

  Summary: This report presents the issues addressed and the findings from the qualitative phase of a two-phase program of research undertaken to better understand adolescent girls' health. The overall objective of this phase was to gain a better understanding of the informational and health care needs of adolescent girls. Among the issues explored are: access to health care services and information inside and outside the school system; sources of health care support; confidentiality and other factors that

encourage or deter use of health care; ease or difficulty in communicating with health care professionals, mental health issues; adolescent girls' understanding of conditions such as **heart disease,** lung, breast, and cervical cancer, and osteoporosis; attitudes toward health behaviors; and knowledge, attitudes, and experiences related to reproductive health and sexuality. This report is intended to be read in conjunction with the findings of the survey, The health of adolescent girls.

- **Indian health focus: Women**

  Source: [Rockville, MD]: Program Statistics Team, Office of Public Health, Division of Community and Environmental Health, U.S. Indian Health Service. 1997. 43 pp.

  Contact: Available from U.S. Indian Health Service, Office of Public Health, Division of Community and Environmental Health, Program Statistics Team, Parklawn Building, Room 7B-114, 5600 Fishers Lane, Rockville, MD 20857. Telephone: (301) 443-4644.

  Summary: This report examines the health status of American Indian and Alaska Native women residing in the Indian Health Service (IHS) service area. Statistical topics covered include: low birthweight and high birthweight; population by age and sex; employment status; birth order; family planning visits; prenatal care; alcohol, tobacco, and other drugs; life expectancy; accident deaths, suicide deaths, and homicide deaths; **heart disease;** diabetes; cancer; and hospitalization. Also included in this publication is a glossary of ICD-9 codes, methods used to rank leading sites of cancer deaths, and a summary of sources and limitations of data for population statistics, vital event statistics, and patient care statistics.

- **For a healthy nation: Returns on investment in public health**

  Source: Washington, DC: Public Health Service, U.S. Department of Health and Human Services. ca. 1994. 49 pp.

  Contact: Available from Superintendent of Documents, U.S. Government Printing Office, P.O. Box 371954, Pittsburgh, PA 15250-7954. Telephone: (202) 512-1991 for public information (D.C. office) or (202) 512-1800 for ordering and publication information (D.C. office) / fax: (202) 512-1293 (public information); (202) 512-2250 (ordering) / Web site: http://www.access.gpo.gov. $4.00 includes shipping and handling; prepayment required.

  Summary: This report underscores the importance of public health services to the overall health of the nation. It examines factors that affect public health delivery: relationships between medical care and public health, the reduction in support for public health, and health care reform. The report reviews public health programs that achieved success by developing partnerships among national, state, and local public health agencies. Also included are case studies of programs on smoking, dental care, children's exposure to lead, vaccines for preventable diseases, reducing **heart disease** and stroke, preventing infant mortality, controlling the spread of sexually transmitted diseases, and reducing unintentional injuries. Developing priorities for providing future services and the role of public health in the changing health care system are discussed.

- **Lives in the balance: The health status of America's medically underserved populations**

  Source: Washington, DC: National Association of Community Health Centers. 1993. ca. 220 pp.

Contact: Available from National Association of Community Health Centers, 1330 New Hampshire Avenue, N.W., Suite 122, Washington, DC 20036. Telephone: (202) 659-8008 / fax: (202) 659-8519.

Summary: This report presents data on the health status of residents of medically underserved counties in all states. Its findings are based on an analysis of county-by-county health and demographic data from various federal sources. The measures of health status are infant mortality, low birthweight, immunizable disease cases, hepatitis cases, tuberculosis cases, deaths from **heart disease,** and deaths from pneumonia. Key findings are summarized, and various analyses of the data are presented in tables. An appendix includes a listing of all counties, their status as medically underserved, and the size of their underserved population.

- **Report of the expert panel on blood cholesterol levels in children and adolescents**

  Source: Bethesda, MD: National Heart, Lung, and Blood Institute, U.S. Department of Health and Human Services. 1991. 119 pp.

  Contact: Available from Information Center, National Heart, Lung, and Blood Institute, National Institutes of Health, 4733 Bethesda Avenue, Suite 530, Bethesda, MD 20814-4820. Telephone: (301) 951-3260. Available at no charge. (NIH 91-2732).

  Summary: This report includes a review of the significance of blood cholesterol levels in childhood and adolescence; nutrient recommendations and recommendations to groups that influence the eating patterns of children and adolescents; and an individualized approach to cholesterol lowering aimed at identifying and treating children and adolescents who are at the greatest risk of having high blood cholesterol as adults and an increased risk of coronary **heart disease.** Recommendations for screening, diet therapy, and drug therapy are included. A highlights volume is published separately.

- **Regional differences in Indian health**

  Source: Rockville, MD: Indian Health Service, U.S. Department of Health and Human Services. 1993-. annual.

  Contact: Available from Priscilla Sandoval, Division Secretary, U.S. Indian Health Service, 5600 Fishers Lane, Room 6A-55, Rockville, MD 20857. Telephone: (301) 443-1114 / fax: (301) 594-6213. Available at no charge.

  Summary: This publication presents tables and charts that describe the Indian Health Service program and the health status of American Indians and Alaska Natives for the period 1987-1989. Current regional differences are presented and comparisons to the general U.S. population are made. The information is grouped into four major categories: population statistics; natality and infant/maternal mortality statistics; general mortality statistics; and patient care statistics. Mortality rates are given for accidents, alcoholism, diabetes, homicide, suicide, tuberculosis, **heart disease,** and cancer. The tables provide detailed data while the charts show significant relationships; a glossary and sources of additional information are also included.

- **Nutrition and health campaign for women consumer kit**

  Source: Chicago, IL: American Dietetic Association. 1994. 16 items.

  Contact: Available from Customer Service, American Dietetic Association, 216 West Jackson Boulevard, Suite 800, Chicago, IL 60606-6995. Telephone: (312) 899-0040 or (800)

877-1600 or (800) 366-1655 or (800) 225-5267 / fax: (312) 899-1758 / Web site: http://www.eatright.org.

Summary: This campaign kit for women's health and nutrition represents a national effort to provide scientifically supported nutrition information to help women make decisions for good nutrition and healthy lifestyles. The contents of the kit include: 1) fact sheets about nutrition as it relates to four major diseases and conditions affecting women (breast cancer, **heart disease,** osteoporosis, and excess weight), 2) suggestions and tips for ADA members to bring the campaign to women in the community including activities in public education, talking points for a speaking engagement, working with the press, and a sample op-ed article, 3) tips on how to persuade investigators to include nutrition in their clinical research activities taking place in the community, 4) a brochure and poster for use at public forums and for distribution to clients, visitors, and others in the community, and 5) a reprint of a Glamour Magazine section profiling the dietary habits of three women and offering nutrition advice from a registered dietitian.

The NLM Gateway[16]

The NLM (National Library of Medicine) Gateway is a Web-based system that lets users search simultaneously in multiple retrieval systems at the U.S. National Library of Medicine (NLM). It allows users of NLM services to initiate searches from one Web interface, providing one-stop searching for many of NLM's information resources or databases.[17] To use the NLM Gateway, simply go to the search site at **http://gateway.nlm.nih.gov/gw/Cmd**. Type "heart disease" (or synonyms) into the search box and click "Search." The results will be presented in a tabular form, indicating the number of references in each database category.

Results Summary

Category	Items Found
Journal Articles	534467
Books / Periodicals / Audio Visual	4206
Consumer Health	2007
Meeting Abstracts	290
Other Collections	89
Total	541059

HSTAT[18]

HSTAT is a free, Web-based resource that provides access to full-text documents used in healthcare decision-making.[19] These documents include clinical practice guidelines, quick-reference guides for clinicians, consumer health brochures, evidence reports and technology

[16] Adapted from NLM: **http://gateway.nlm.nih.gov/gw/Cmd?Overview.x**.

[17] The NLM Gateway is currently being developed by the Lister Hill National Center for Biomedical Communications (LHNCBC) at the National Library of Medicine (NLM) of the National Institutes of Health (NIH).

[18] Adapted from HSTAT: **http://www.nlm.nih.gov/pubs/factsheets/hstat.html**.

[19] The HSTAT URL is **http://hstat.nlm.nih.gov/**.

assessments from the Agency for Healthcare Research and Quality (AHRQ), as well as AHRQ's Put Prevention Into Practice.[20] Simply search by "heart disease" (or synonyms) at the following Web site: **http://text.nlm.nih.gov**.

Coffee Break: Tutorials for Biologists[21]

Coffee Break is a general healthcare site that takes a scientific view of the news and covers recent breakthroughs in biology that may one day assist physicians in developing treatments. Here you will find a collection of short reports on recent biological discoveries. Each report incorporates interactive tutorials that demonstrate how bioinformatics tools are used as a part of the research process. Currently, all Coffee Breaks are written by NCBI staff.[22] Each report is about 400 words and is usually based on a discovery reported in one or more articles from recently published, peer-reviewed literature.[23] This site has new articles every few weeks, so it can be considered an online magazine of sorts. It is intended for general background information. You can access the Coffee Break Web site at the following hyperlink: **http://www.ncbi.nlm.nih.gov/Coffeebreak/**.

Other Commercial Databases

In addition to resources maintained by official agencies, other databases exist that are commercial ventures addressing medical professionals. Here are some examples that may interest you:

- **CliniWeb International:** Index and table of contents to selected clinical information on the Internet; see **http://www.ohsu.edu/cliniweb/**.
- **Medical World Search:** Searches full text from thousands of selected medical sites on the Internet; see **http://www.mwsearch.com/**.

The Genome Project and Heart Disease

In the following section, we will discuss databases and references which relate to the Genome Project and heart disease.

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[20] Other important documents in HSTAT include: the National Institutes of Health (NIH) Consensus Conference Reports and Technology Assessment Reports; the HIV/AIDS Treatment Information Service (ATIS) resource documents; the Substance Abuse and Mental Health Services Administration's Center for Substance Abuse Treatment (SAMHSA/CSAT) Treatment Improvement Protocols (TIP) and Center for Substance Abuse Prevention (SAMHSA/CSAP) Prevention Enhancement Protocols System (PEPS); the Public Health Service (PHS) Preventive Services Task Force's *Guide to Clinical Preventive Services*; the independent, nonfederal Task Force on Community Services' *Guide to Community Preventive Services*; and the Health Technology Advisory Committee (HTAC) of the Minnesota Health Care Commission (MHCC) health technology evaluations.

[21] Adapted from **http://www.ncbi.nlm.nih.gov/Coffeebreak/Archive/FAQ.html**.

[22] The figure that accompanies each article is frequently supplied by an expert external to NCBI, in which case the source of the figure is cited. The result is an interactive tutorial that tells a biological story.

[23] After a brief introduction that sets the work described into a broader context, the report focuses on how a molecular understanding can provide explanations of observed biology and lead to therapies for diseases. Each vignette is accompanied by a figure and hypertext links that lead to a series of pages that interactively show how NCBI tools and resources are used in the research process.

Online Mendelian Inheritance in Man (OMIM)

The Online Mendelian Inheritance in Man (OMIM) database is a catalog of human genes and genetic disorders authored and edited by Dr. Victor A. McKusick and his colleagues at Johns Hopkins and elsewhere. OMIM was developed for the World Wide Web by the National Center for Biotechnology Information (NCBI).[24] The database contains textual information, pictures, and reference information. It also contains copious links to NCBI's Entrez database of MEDLINE articles and sequence information.

To search the database, go to **http://www.ncbi.nlm.nih.gov/Omim/searchomim.html**. Type "heart disease" (or synonyms) into the search box, and click "Submit Search." If too many results appear, you can narrow the search by adding the word "clinical." Each report will have additional links to related research and databases. In particular, the option "Database Links" will search across technical databases that offer an abundance of information. The following is an example of the results you can obtain from the OMIM for heart disease:

- **Cleft Lip/palate with Characteristic Facies, Intestinal Malrotation, and Lethal Congenital Heart Disease**
  Web site: http://www.ncbi.nlm.nih.gov/htbin-post/Omim/dispmim?601165
- **Coronary Heart Disease, Susceptibility To, 1**
  Web site: http://www.ncbi.nlm.nih.gov/htbin-post/Omim/dispmim?607339
- **Craniostenosis, Sagittal, with Congenital Heart Disease, Mental Deficiency, and Mandibular Ankylosis**
  Web site: http://www.ncbi.nlm.nih.gov/htbin-post/Omim/dispmim?218450
- **Mental Retardation, Congenital Heart Disease, Blepharophimosis, Blepharoptosis, and Hypoplastic Teeth**
  Web site: http://www.ncbi.nlm.nih.gov/htbin-post/Omim/dispmim?249620
- **Microcephaly, Congenital Heart Disease, Unilateral Renal Agenesis, and Hyposegmented Lungs**
  Web site: http://www.ncbi.nlm.nih.gov/htbin-post/Omim/dispmim?601355
- **Pancreatic Hypoplasia, Congenital, with Diabetes Mellitus and Congenital Heart Disease**
  Web site: http://www.ncbi.nlm.nih.gov/htbin-post/Omim/dispmim?600001

Genes and Disease (NCBI - Map)

The Genes and Disease database is produced by the National Center for Biotechnology Information of the National Library of Medicine at the National Institutes of Health. This Web site categorizes each disorder by system of the body. Go to **http://www.ncbi.nlm.nih.gov/disease/**, and browse the system pages to have a full view of important conditions linked to human genes. Since this site is regularly updated, you may wish to revisit it from time to time. The following systems and associated disorders are addressed:

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[24] Adapted from **http://www.ncbi.nlm.nih.gov/**. Established in 1988 as a national resource for molecular biology information, NCBI creates public databases, conducts research in computational biology, develops software tools for analyzing genome data, and disseminates biomedical information--all for the better understanding of molecular processes affecting human health and disease.

- **Cancer:** Uncontrolled cell division.
  Examples: Breast and ovarian cancer, Burkitt lymphoma, chronic myeloid leukemia, colon cancer, lung cancer, malignant melanoma, multiple endocrine neoplasia, neurofibromatosis, p53 tumor suppressor, pancreatic cancer, prostate cancer, Ras oncogene, RB: retinoblastoma, von Hippel-Lindau syndrome.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Cancer.html**
- **Immune System:** Fights invaders.
  Examples: Asthma, autoimmune polyglandular syndrome, Crohn's disease, DiGeorge syndrome, familial Mediterranean fever, immunodeficiency with Hyper-IgM, severe combined immunodeficiency.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Immune.html**
- **Metabolism:** Food and energy.
  Examples: Adreno-leukodystrophy, atherosclerosis, Best disease, Gaucher disease, glucose galactose malabsorption, gyrate atrophy, juvenile-onset diabetes, obesity, paroxysmal nocturnal hemoglobinuria, phenylketonuria, Refsum disease, Tangier disease, Tay-Sachs disease.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Metabolism.html**
- **Muscle and Bone:** Movement and growth.
  Examples: Duchenne muscular dystrophy, Ellis-van Creveld syndrome, Marfan syndrome, myotonic dystrophy, spinal muscular atrophy.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Muscle.html**
- **Nervous System:** Mind and body.
  Examples: Alzheimer disease, amyotrophic lateral sclerosis, Angelman syndrome, Charcot-Marie-Tooth disease, epilepsy, essential tremor, fragile X syndrome, Friedreich's ataxia, Huntington disease, Niemann-Pick disease, Parkinson disease, Prader-Willi syndrome, Rett syndrome, spinocerebellar atrophy, Williams syndrome.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Brain.html**
- **Signals:** Cellular messages.
  Examples: Ataxia telangiectasia, Cockayne syndrome, glaucoma, male-patterned baldness, SRY: sex determination, tuberous sclerosis, Waardenburg syndrome, Werner syndrome.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Signals.html**
- **Transporters:** Pumps and channels.
  Examples: Cystic fibrosis, deafness, diastrophic dysplasia, Hemophilia A, long-QT syndrome, Menkes syndrome, Pendred syndrome, polycystic kidney disease, sickle cell anemia, Wilson's disease, Zellweger syndrome.
  Web site: **http://www.ncbi.nlm.nih.gov/disease/Transporters.html**

Entrez

Entrez is a search and retrieval system that integrates several linked databases at the National Center for Biotechnology Information (NCBI). These databases include nucleotide sequences, protein sequences, macromolecular structures, whole genomes, and MEDLINE through PubMed. Entrez provides access to the following databases:

- **3D Domains:** Domains from Entrez Structure,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=geo**

- **Books:** Online books,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=books**
- **Genome:** Complete genome assemblies,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Genome**
- **NCBI's Protein Sequence Information Survey Results:**
  Web site: **http://www.ncbi.nlm.nih.gov/About/proteinsurvey/**
- **Nucleotide Sequence Database (Genbank):**
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Nucleotide**
- **OMIM:** Online Mendelian Inheritance in Man,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=OMIM**
- **PopSet:** Population study data sets,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Popset**
- **ProbeSet:** Gene Expression Omnibus (GEO),
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=geo**
- **Protein Sequence Database:**
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Protein**
- **PubMed:** Biomedical literature (PubMed),
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=PubMed**
- **Structure:** Three-dimensional macromolecular structures,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Structure**
- **Taxonomy:** Organisms in GenBank,
  Web site: **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Taxonomy**

To access the Entrez system at the National Center for Biotechnology Information, go to **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=search&DB=genome**, and then select the database that you would like to search. The databases available are listed in the drop box next to "Search." Enter "heart disease" (or synonyms) into the search box and click "Go."

Jablonski's Multiple Congenital Anomaly/Mental Retardation (MCA/MR) Syndromes Database[25]

This online resource has been developed to facilitate the identification and differentiation of syndromic entities. Special attention is given to the type of information that is usually limited or completely omitted in existing reference sources due to space limitations of the printed form.

At **http://www.nlm.nih.gov/mesh/jablonski/syndrome_toc/toc_a.html**, you can search across syndromes using an alphabetical index**.** Search by keywords at **http://www.nlm.nih.gov/mesh/jablonski/syndrome_db.html**.

[25] Adapted from the National Library of Medicine:
http://www.nlm.nih.gov/mesh/jablonski/about_syndrome.html.

The Genome Database[26]

Established at Johns Hopkins University in Baltimore, Maryland in 1990, the Genome Database (GDB) is the official central repository for genomic mapping data resulting from the Human Genome Initiative. In the spring of 1999, the Bioinformatics Supercomputing Centre (BiSC) at the Hospital for Sick Children in Toronto, Ontario assumed the management of GDB. The Human Genome Initiative is a worldwide research effort focusing on structural analysis of human DNA to determine the location and sequence of the estimated 100,000 human genes. In support of this project, GDB stores and curates data generated by researchers worldwide who are engaged in the mapping effort of the Human Genome Project (HGP). GDB's mission is to provide scientists with an encyclopedia of the human genome which is continually revised and updated to reflect the current state of scientific knowledge. Although GDB has historically focused on gene mapping, its focus will broaden as the Genome Project moves from mapping to sequence, and finally, to functional analysis.

To access the GDB, simply go to the following hyperlink: **http://www.gdb.org/**. Search "All Biological Data" by "Keyword." Type "heart disease" (or synonyms) into the search box, and review the results. If more than one word is used in the search box, then separate each one with the word "and" or "or" (using "or" might be useful when using synonyms).

[26] Adapted from the Genome Database: **http://gdbwww.gdb.org/gdb/aboutGDB.html - mission**.

APPENDIX B. PATIENT RESOURCES

Overview

Official agencies, as well as federally funded institutions supported by national grants, frequently publish a variety of guidelines written with the patient in mind. These are typically called "Fact Sheets" or "Guidelines." They can take the form of a brochure, information kit, pamphlet, or flyer. Often they are only a few pages in length. Since new guidelines on heart disease can appear at any moment and be published by a number of sources, the best approach to finding guidelines is to systematically scan the Internet-based services that post them.

Patient Guideline Sources

The remainder of this chapter directs you to sources which either publish or can help you find additional guidelines on topics related to heart disease. Due to space limitations, these sources are listed in a concise manner. Do not hesitate to consult the following sources by either using the Internet hyperlink provided, or, in cases where the contact information is provided, contacting the publisher or author directly.

The National Institutes of Health

The NIH gateway to patients is located at **http://health.nih.gov/**. From this site, you can search across various sources and institutes, a number of which are summarized below.

Topic Pages: MEDLINEplus

The National Library of Medicine has created a vast and patient-oriented healthcare information portal called MEDLINEplus. Within this Internet-based system are "health topic pages" which list links to available materials relevant to heart disease. To access this system, log on to **http://www.nlm.nih.gov/medlineplus/healthtopics.html**. From there you can either search using the alphabetical index or browse by broad topic areas. Recently, MEDLINEplus listed the following when searched for "heart disease":

- Other guides

  Congenital Heart Disease
  http://www.nlm.nih.gov/medlineplus/congenitalheartdisease.html

  Coronary Disease
  http://www.nlm.nih.gov/medlineplus/coronarydisease.html

  Heart Attack
  http://www.nlm.nih.gov/medlineplus/heartattack.html

  Heart Diseases
  http://www.nlm.nih.gov/medlineplus/heartdiseases.html

  Heart Diseases--Prevention
  http://www.nlm.nih.gov/medlineplus/heartdiseasesprevention.html

Within the health topic page dedicated to heart disease, the following was listed:

- General/Overviews

  Heart Valve Disorders
  Source: Merck & Co., Inc.
  http://www.merck.com/mrkshared/mmanual_home/sec3/19.jsp

  Heart Valves
  Source: American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=4598

- Diagnosis/Symptoms

  Chest X-Rays: Helping Detect Heart and Lung Conditions
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=HB00019

  Echocardiogram
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=HB00012

  MR Angiography (MRA)
  Source: American College of Radiology, Radiological Society of North America
  http://www.radiologyinfo.org/content/mr-angiography.htm

  Tests to Diagnose Heart Disease
  Source: American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=4739

- Treatment

  Aortic Valve Replacement
  Source: Society of Thoracic Surgeons
  http://www.sts.org/doc/3620

  Heart Valve Replacement
  http://www.nlm.nih.gov/medlineplus/tutorials/heartvalvereplacementloader.html

- Specific Conditions/Aspects

  Aortic Regurgitation
  Source: American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=4448

  Aortic Valve Calcification (Aortic Valve Sclerosis)
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=HQ00245

  Aortic Valve Stenosis
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=DS00418

  Dental Care and Heart Disease
  Source: American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=4548

  Heart Murmurs
  Source: American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=4571

  Mitral Valve Regurgitation
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=DS00421

  Mitral Valve Stenosis
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=DS00420

  Rheumatic Heart Disease/Rheumatic Fever
  Source: American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=4709

- Children

  Heart Murmurs and Your Child
  Source: Nemours Foundation
  http://kidshealth.org/parent/medical/heart/murmurs.html

  Heart Murmurs: Can They Develop Later in Life?
  Source: Mayo Foundation for Medical Education and Research
  http://www.mayoclinic.com/invoke.cfm?id=AN00093

- Organizations

  American Heart Association
  http://www.americanheart.org/presenter.jhtml?identifier=1200000

  National Heart, Lung, and Blood Institute
  http://www.nhlbi.nih.gov/

  Society of Thoracic Surgeons
  http://www.sts.org/section/stspatientinfo/

You may also choose to use the search utility provided by MEDLINEplus at the following Web address: **http://www.nlm.nih.gov/medlineplus/**. Simply type a keyword into the search box and click "Search." This utility is similar to the NIH search utility, with the

exception that it only includes materials that are linked within the MEDLINEplus system (mostly patient-oriented information). It also has the disadvantage of generating unstructured results. We recommend, therefore, that you use this method only if you have a very targeted search.

The Combined Health Information Database (CHID)

CHID Online is a reference tool that maintains a database directory of thousands of journal articles and patient education guidelines on heart disease. CHID offers summaries that describe the guidelines available, including contact information and pricing. CHID's general Web site is **http://chid.nih.gov/**. To search this database, go to **http://chid.nih.gov/detail/detail.html**. In particular, you can use the advanced search options to look up pamphlets, reports, brochures, and information kits. The following was recently posted in this archive:

- **Steps for Controlling Diabetes and Heart Disease. [Pasos para Controlar la Diabetes y las Enfermedades del Corazon]**

  Source: San Bruno, CA: StayWell Company. 2000. 23 p.

  Contact: Available from StayWell Company. Order Department, 1100 Grundy Lane, San Bruno, CA 94066-9821. (800) 333-3032. Fax (650) 244-4512. E-mail: email@staywell.com. Website: www.staywell.com. PRICE: $1.50 plus shipping and handling; bulk copies available.

  Summary: This illustrated booklet, which is available in both English and Spanish, provides people who have diabetes with information on controlling diabetes and heart disease. People who have diabetes and do not control their blood glucose may develop narrowing in their coronary arteries. This problem may lead to a heart attack. Heart disease risk factors that people can control include lowering low density lipoprotein cholesterol and blood pressure, controlling blood glucose, losing excess weight, and quitting smoking. Regular checkups and laboratory tests help people know how well they are reducing their heart health risks. The booklet offers advice for planning healthy meals, including eating less fat and less cholesterol, consuming less salt, eating more fiber, limiting carbohydrates, and understanding serving sizes. In addition, the booklet provides tips for increasing physical activity; taking oral diabetes medications, insulin, and heart medications safely; testing blood glucose levels; quitting smoking; and managing stress. Other topics include the warning signs of a silent heart attack, self care following heart surgery, and special concerns of women who have diabetes. The booklet concludes with guidelines for making lifestyle changes.

- **Diabetes and Heart Disease: Bayer Care Health Facts**

  Source: Tarrytown, NY: Bayer Corporation. 2000. [7 p.].

  Contact: Available from Bayer Corporation. Diagnostics Division, 511 Benedict Avenue, Tarrytown, NY 10591-5097. (800) 445-5901. PRICE: Single copy free.

  Summary: This brochure uses a question and answer format to provide people who have diabetes with information on the relationship between diabetes and heart disease. Risk factors for heart disease include age, a family history of heart disease, smoking, high blood pressure, low high density lipoprotein, high low density lipoprotein, high triglycerides and cholesterol, physical inactivity, and diabetes. People who have diabetes have a two to four times greater risk of getting heart disease. Therefore, they

need to reduce as many risk factors for cardiovascular disease as possible. Lowering their cholesterol by modifying dietary fat intake is helpful. The brochure explains the differences between different kinds of fat, including dietary cholesterol and polyunsaturated, monounsaturated, saturated, and trans fats. In addition, the brochure offers tips to reduce dietary fat and outlines other healthy lifestyle choices people can make. 1 table.

- **Diabetes. Heart Disease. Stroke. Poor Circulation. What's the Connection? Can I Do Anything About It? [Diabetes and Heart Disease Booklet]**

  Source: Boston, MA: Joslin Diabetes Center. 1998. 5 p.

  Contact: Available from Joslin Diabetes Center. One Joslin Place, Boston, MA 02215. (800) 344-4501 or (508) 583-3240. Fax (617) 732-2562. Website: www.joslin.harvard.edu. PRICE: $12.50 for 1-25 packages of 10; plus shipping and handling. Order number JDC425.

  Summary: This booklet uses a question and answer format to provide people who have diabetes with information on the relationship between diabetes and cardiovascular problems. Cardiovascular complications are the most common long-term problems that can develop in people who have diabetes because high blood sugar levels damage blood vessels and people who have diabetes tend to have higher fat levels in their blood. Reducing the risk of cardiovascular complications involves quitting smoking, losing weight if overweight, keeping blood pressure in the proper range, exercising regularly, keeping blood fats and cholesterol levels in a healthy range, and keeping blood sugar levels down. Various tests should be conducted to maintain a healthy heart and blood vessels, including the hemoglobin A1c test.

- **Cholesterol and Heart Disease**

  Source: New York, NY: Pfizer Pratt Pharmaceuticals. May 1992. 4 p.

  Contact: Available from Pfizer Pratt Pharmaceuticals. Attn: Marketing, 235 East 42nd Street, New York, NY 10017. (212) 573-2551. PRICE: Single copy free. Order Number RGA025X91H.

  Summary: This chart-like brochure briefly presents information about cholesterol and heart disease. Topics include the risk factors for heart disease for people with diabetes; the role of cholesterol in heart problems; and lowering the risks for heart disease by controlling cholesterol, exercising, losing weight, and modifying one's diet. Simple line drawings on each page help to reiterate the concepts presented.

- **Living with High Blood Pressure and Heart Disease**

  Source: New York, NY: National Kidney Foundation, Inc. 1994. 6 p.

  Contact: Available from National Kidney Foundation. U.S. Materials Orders, 30 East 33rd Street, New York, NY 10016. (212) 889-2210. Fax (212) 689-9261. PRICE: $17.00 for 20 copies. Item number: 18-04.

  Summary: This booklet provides general information about high blood pressure (hypertension) and how readers can help prevent the complications of hypertension. Written in question and answer format, the booklet addresses topics including a definition of high blood pressure and how it is measured, how often blood pressure should be checked, why hypertension is a serious problem, how the heart is affected by hypertension, how to prevent hypertension-related heart disease, the risk factors for

developing heart disease (age, family history, gender, cigarette smoking, high blood pressure, and high blood cholesterol), what to do if heart disease is already present, how to choose the right type of blood pressure medication, the interplay of high blood pressure medications and heart medications, the role of dietary cholesterol, and self care suggestions. The brochure concludes with a list of other informational booklets available from the National Kidney Foundation. The booklet emphasizes that, if high blood pressure is diagnosed, treated, and kept under control, its damaging effects, including those on the heart, can be prevented or reduced.

- **Heart Disease and Impotence**

  Source: Augusta, GA: Geddings Osbon Sr. Foundation. 199x. 2 p.

  Contact: Available from Geddings Osbon Sr. Foundation. P.O. Box 1593, Augusta, GA 30903. (800) 433-4215. PRICE: Single copy free.

  Summary: This brochure provides information about heart disease and male impotence. Topics include the interrelationship of impotence and vascular disease; the anatomy of the penis; common questions that patients ask; and treatment options for impotence, including counseling or sex therapy, topical vasodilators, drug therapy, hormone replacement, external vacuum therapy, penile injection therapy, penile prostheses, and penile reconstruction and venous ligation. The brochure emphasizes that lifestyle changes that promote a healthy heart can also help reduce the chances of becoming impotent.

- **Congenital heart disease: Guidelines for care for children with special health care needs**

  Source: Minneapolis, MN: Services for Children with Handicaps, Minnesota Department of Health. 1990. 46 pp.

  Contact: Available from Minnesota Children with Special Health Care Needs, 717 Delaware Street, S.E., Box 9441, Minneapolis, MN 55440. Telephone: (612) 623-5150 or (800) 728-5420.

  Summary: This publication was developed for families and health professionals caring for children with congenital heart disease. The guidelines are aimed at helping families coordinate the health care needed for the optimal growth and development of their child. General information concerning congenital heart disease is provided along with an overview of the family centered health care team approach to treating a child or adolescent with this condition. The publication also outlines the child's needs at various stages of her life in terms of health care, development, school, and child care. A glossary and list of resources are also included.

- **Ask Your Periodontist About Periodontal Disease and Heart Disease**

  Source: Chicago, IL: American Academy of Periodontology. 1999. [5 p.].

  Contact: Available from American Academy of Periodontology. Suite 800, 737 North Michigan Avenue, Chicago, IL 60611-2690. Website: www.perio.org. PRICE: Single copy free.

  Summary: Periodontal disease (also known as gum disease) is a bacterial infection of the gums, bone and periodontal ligament (attachment fibers that support the teeth and hold them in the jaw). Recent research is showing a link between heart diseases and periodontal disease. This patient education brochure informs patients about these risks

and encourages them to take precautions before dental treatment. The brochure emphasizes that taking care of one's periodontal health may be an important step toward prevention of heart disease, along with controlling the other well known risk factors for cardiovascular disease. The brochure describes the precautions that should be taken before dental treatment, to help limit the entry of bacteria into the blood stream during dental procedures. The brochure includes an insert that requests patients to list specific drugs that they are taking; the insert can then be given to the dentist or periodontist, who will work with the patient to minimize any potentially negative effects of the dental treatment. 1 figure.

- **Heart Disease and Women: Be Physically Active**

  Source: Bethesda, MD: NHLBI, 1997.

  Contact: NHLBI Information Center, P.O. Box 30105, Bethesda, MD 20824-0105. (301) 251-1222. Fax (301) 251-1223.

  Summary: This factsheet describes why physical activity is important for women, and offers a 12-week walking program. The author says that exercise reduces the risk of heart disease, and reduces risk of other conditions, such as overweight and high blood pressure, that may contribute to heart disease. Suggestions to increase success at physical activity are offered, such as awareness of the body's limits, perseverance, and dressing for the weather. Ideas on making opportunities for exercise, such as lawn mowing and walking instead of snacking are included as well.

- **There's Good News for Women!: You Can Take Charge of Your Health, Achieve a Healthy Weight, and Help Prevent Heart Disease, Breast Cancer, and Osteoporosis**

  Source: Weight Watchers International, 8 p., N.D.

  Contact: Weight Watchers International, 360 Lexington Ave., 11th Floor, New York, NY 10017. (800) 651-6000.

  Summary: This brochure focuses on the small steps that women can take to improve their health, reduce their weight, and prevent certain diseases. According to the brochure, the key to good health is to reach and maintain a healthy weight. Excess weight increases the risk of heart disease, osteoporosis, and breast cancer. The brochure advises that women initiate a diet lower in fat and higher in carbohydrates, and to increase physical activity. The recommendations in this brochure are part of the principles of the Weight Watchers program, which include maintaining an active lifestyle; having a positive, healthy outlook; and belonging to a weight loss support group.

- **You Have Diabetes: But You Don't Have to Get Heart Disease Too**

  Source: American Family Physician. 62(12): 2645-2646. December 15, 2000.

  Contact: Available from American Academy of Family Physicians. 11400 Tomahawk Creek Parkway, Leawood, KS 66211-2672. (800) 274-2237. Website: www.aafp.org.

  Summary: This patient information sheet uses a question and answer format to provide people who have diabetes with information on the prevention of heart disease. The article advises people who have diabetes to reduce their risk of heart disease by keeping their blood sugar level under control, losing weight and maintaining weight loss, lowering their cholesterol level, increasing their physical activity, controlling their blood pressure, and quitting smoking. In addition, the information sheet identifies

organizations that can help people who have diabetes obtain diet and nutrition information.

- **What Is Your Risk of Heart Disease?**

  Source: Little Rock, AR: Arkansas Department of Health. 199x. [1 p.].

  Contact: Available from Arkansas Department of Health Diabetes Control Program. 4815 West Markham Street, Slot Number 3, Little Rock, AR 72205-3867. (501) 661-2627. Fax (501) 661-2009. PRICE: Single copy free.

  Summary: This master copy of a diabetes education sheet, which was developed by the Arkansas Diabetes Control Program in cooperation with the Education Subcommittee of the Diabetes Coalition, discusses heart disease risk among people who have diabetes. People who have type 2 diabetes are particularly prone to diseases of the large blood vessels. The education sheet identifies other risk factors, besides diabetes, that can lead to heart disease. In addition, the sheet recommends ways of reducing the risks: controlling blood sugar, improving lipids, quitting smoking, controlling blood pressure, increasing physical activity, losing weight, and eating less fat and more fiber.

The National Guideline Clearinghouse™

The National Guideline Clearinghouse™ offers hundreds of evidence-based clinical practice guidelines published in the United States and other countries. You can search this site located at **http://www.guideline.gov/** by using the keyword "heart disease" (or synonyms). The following was recently posted:

- **Antithrombotic therapy in valvular heart disease. In: Sixth ACCP Consensus Conference on Antithrombotic Therapy**

  Source: American College of Chest Physicians - Medical Specialty Society; 2001 January; 13 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=2727&nbr=1953&string=heart+AND+disease

- **Driving and heart disease**

  Source: European Society of Cardiology - Medical Specialty Society; 1998 August; 13 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=1930&nbr=1156&string=heart+AND+disease

- **Lipids and the primary prevention of coronary heart disease. A national clinical guideline**

  Source: Scottish Intercollegiate Guidelines Network - National Government Agency [Non-U.S.]; 1999 September; 60 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=2909&nbr=2135&string=heart+AND+disease

- **Management of patients with valvular heart disease**

  Source: American College of Cardiology Foundation - Medical Specialty Society; 1998 November 1; 96 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=1798&nbr=1024&string=heart+AND+disease

- **Nutritional strategies efficacious in the prevention or treatment of coronary heart disease (CHD)**

  Source: Nutrition Screening Initiative - Professional Association; 1998; 16 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=1418&nbr=591&string=heart+AND+disease

- **Prevention of coronary heart disease in clinical practice**

  Source: European Atherosclerosis Society - Professional Association; 1998; 70 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=1923&nbr=1149&string=heart+AND+disease

- **Secondary prevention of coronary heart disease following myocardial infarction. A national clinical guideline**

  Source: Scottish Intercollegiate Guidelines Network - National Government Agency [Non-U.S.]; 2000 January; 26 pages

  http://www.guideline.gov/summary/summary.aspx?doc_id=2303&nbr=1529&string=heart+AND+disease

Healthfinder™

Healthfinder™ is sponsored by the U.S. Department of Health and Human Services and offers links to hundreds of other sites that contain healthcare information. This Web site is located at **http://www.healthfinder.gov**. Again, keyword searches can be used to find guidelines. The following was recently found in this database:

- **An Ounce of Prevention: A Guide To Heart Health**

  Summary: A bilingual guide, this colorful photonovela contains five brief stories on how to prevent heart disease. It features tips on ways to make lifestyle changes to protect the heart.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=2794

- **Ask NOAH About: Heart Disease and Stroke**

  Summary: New York Online Access to Health (NOAH) provides full-text health information for consumers about cardiovascular diseases in both English and Spanish.

  Source: NOAH: New York Online Access to Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=1996

- **Cardiovascular Disease Risk Assessment Tool**

  Summary: This risk assessment tool uses information from the Framingham Heart Study. This tool is designed for adults who do not have heart disease or diabetes.

  Source: American Heart Association

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7412

- **Cardiovascular Health, Assessment, Management, & Prevention**

  Summary: A booklet written to help lay people understand heart disease and help them make informed choices for adopting a

  Source: Educational Institution--Follow the Resource URL for More Information

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=4083

- **Check Your Cholesterol And Heart Disease I.Q.**

  Summary: Are you cholesterol smart? This quiz allows you to test your knowledge about high blood cholesterol.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=718

- **Check Your Healthy Heart I.Q.**

  Summary: Answer true or false to these questions to test your knowledge of heart disease and its risk factors. Answers and explanations are at the end of the page.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=721

- **Check Your Physical Activity and Heart Disease I.Q.**

  Summary: Test your knowledge about physical activity and its affects on the cardiovascular system. Mark each statement true or false. See how you did by checking the answers at the end of this page.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=244

- **Chelation Therapy: American Heart Association's Recommendations**

  Summary: This fact sheet contains recommendations from the American Heart Association regarding the use of chelation (E.D.T.A., ethylenediamine tetraacetic acid) in treating arteriosclerotic heart disease.

  Source: American Heart Association

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=3896

- **Congenital Heart Defects**

  Summary: This health education information fact sheet provides general information on congenital heart disease including causes, diagnosis, treatment and prevention.

  Source: March of Dimes Birth Defects Foundation

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=3262

- **Congenital Heart Disease Information and Resources**

  Summary: This web site provides resources for families of children with congenital and acquired heart disease, adults with CHD, and health professionals.

  Source: Children's Health Information Network

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=2895

- **Depression and Heart Disease**

  Summary: Depression can strike anyone.

  Source: National Institute of Mental Health, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6900

- **Depression Can Break Your Heart**

  Summary: Research over the past two decades has shown that depression and heart disease are common companions and, what is worse, each can lead to the other.

  Source: National Institute of Mental Health, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6614

- **Devices and Procedures: A Patients' Guide**

  Summary: Useful information for patients about angioplasty procedures and devices technologies, treatments for heart disease.

  Source: Nonprofit/Professional Entity--Follow the Resource URL for More Information

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=4479

- **Easy-to-Read English/Spanish Booklets on Heart Health**

  Summary: These set of eight booklets were written to help you make healthy changes in your life and reduce the risk of heart disease.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=1412

- **Fact Sheet American Indians and Alaska Natives and Tobacco**

  Summary: Tobacco use is a risk factor for heart disease, cancer, and stroke, all leading causes of death among American Indians and Alaska Natives.

  Source: American Lung Association

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6855

- **Facts About Coronary Heart Disease (CHD)**

  Summary: This consumer health education fact sheet contains information for the patient diagnosed with this form of heart disease, caused by a narrowing of the coronary arteries that feed the heart.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=2613

- **Facts About Heart Disease and Stroke Among American Indians and Alaska Natives**

  Summary: Heart disease and stroke, the principal causes of cardiovascular disease, are the first and fifth leading causes of death among American Indians and Alaska Natives (AI/AN).

  Source: CDC National Prevention Information Network

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6780

- **Facts About Heart Disease and Women - Be Physically Active**

  Summary: Regular physical activity can help women reduce the risk of coronary heart disease. This brochure provides details.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=88

- **Facts About Heart Disease and Women - Kicking the Smoking Habit**

  Summary: This brochure offers tips for women on getting ready to break the smoking habit and then following through. Includes statistics and suggested resources for additional information.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=89

- **Facts About Heart Disease and Women: Preventing and Controlling High Blood Pressure**

  Summary: If you have high blood pressure, you can control it with proper treatment. If you don't have high blood pressure now, you can take steps to prevent it from developing.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=242

- **Facts About Heart Failure**

  Summary: Heart failure is one of the most serious symptoms of heart disease. This fact sheet describes, the prevalence, causes, symptoms and treatment of this cardiovascular disorder.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=2010

- **Facts about Men and Heart Disease: Alaska**

  Summary: Men and Heart Disease: An Atlas of Racial and Ethnic Disparities in Mortality, a new publication from the Centers for Disease Control and Prevention and West Virginia University, features five heart

  Source: National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6790

- **Guide to Assessing Your Risks for Cardiovascular Disease and Stroke**

  Summary: A health risk awareness quiz that focuses on heart attack & stroke allows you to assess your risk for heart disease and stroke. Includes a stroke risk score card, and questions to ask your doctor.

  Source: American Heart Association

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=4109

- **healthfinder® just for you: Adults**

  Summary: healthfinder®'s just for you: Adults section features topics such as heart disease, high blood pressure, and physical activity.

  Source: U.S. Department of Health and Human Services

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7017

- **healthfinder® just for you: Men**

  Summary: healthfinder®'s just for you: Men section features topics such as fatherhood, heart disease, and prostate cancer.

  Source: U.S. Department of Health and Human Services

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7013

- **Healthy Heart Handbook for Women**

  Summary: This easy-to-use, easy-to-read handbook explains factors that place women at risk of heart disease and recommends steps they can take to protect their heart health.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=1401

- **Heart Disease - how treatments can help**

  Source: New South Wales Multicultural Health Communication Service

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7530

- **Heart disease - not just a man's problem**

  Source: New South Wales Multicultural Health Communication Service

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7528

- **Heart Disease Information for American Indian/Alaska Native Women**

  Summary: This brief fact sheet describes how heart disease affects minority women.

  Source: National Women's Health Information Center, U.S. Public Health Service's Office on Women's Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6936

- **Heart Truth**

  Summary: To make women more aware of the danger of heart disease, the National Heart, Lung, and Blood Institute (NHLBI) and partner organizations are sponsoring a national campaign called The Heart Truth.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7254

- **KidZone on PediHeart**

  Summary: A web site where children with heart disease can learn about the heart and some common congenital heart conditions, ask questions, send in ideas for the site and explore other similar web sites.

  Source: Nonprofit/Professional Entity--Follow the Resource URL for More Information

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=5809

- **Live Healthier, Live Longer: Lowering Cholesterol for the Person With Heart Disease (Interactive Web Site)**

  Summary: If you have heart disease or are at risk for heart disease, visit this interactive site and find out how you can lower your cholesterol.

  Source: National Heart, Lung, and Blood Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=2933

- **Men: Eat 9 A Day for Better Health**

  Summary: Black men are at high risk for many serious and potentially fatal diseases including many cancers, high blood pressure, diabetes, and heart disease.

  Source: National Cancer Institute, National Institutes of Health

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7470

- **National Heart Foundation**

  Summary: This document is AHAF's National Heart Foundation (NHF) program. It was established in 1976 to fund research on and educate the public about heart disease and stroke.

  Source: American Health Assistance Foundation

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=6687

- **PediHeart**

  Summary: This web site is designed for children with heart disease, their families, and the health care practitioners that care for them.

  Source: Educational Institution--Follow the Resource URL for More Information

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=4441

- **Public Health Action Plan to Prevent Heart Disease and Stroke**

  Summary: The purpose of the Public Health Action Plan to Prevent Heart Disease and Stroke is to chart a course for the Centers for Disease Control and Prevention (CDC) and collaborating public health agencies,

  Source: National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7445

- **Take Wellness to Heart -- American Heart Association Women's Web Site**

  Summary: This Web site is designed to give women of all ages the facts on women's heart disease and stroke.

  Source: American Heart Association

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=3893

- **Tips for Exercise Success**

  Summary: Lack of physical activity is a risk factor for heart disease. This online document advises the consumer about safety steps to take towards a successful and injury-free exercise program.

  Source: American Heart Association

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=2406

- **Triglycerides Test**

  Summary: Why get tested? To assess the risk of developing heart disease When to get tested? As part of a lipid profile during a regular medical exam or if you are being treated for high triglycerides

  Source: American Association for Clinical Chemistry

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=7105

- **Women and Heart Disease: An Atlas of Racial and Ethnic Disparities in Mortality**

  Summary: This publication shows heart disease death rates among women aged 35 years, by county, throughout the United States.

  Source: National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention

  http://www.healthfinder.gov/scripts/recordpass.asp?RecordType=0&RecordID=5230

The NIH Search Utility

The NIH search utility allows you to search for documents on over 100 selected Web sites that comprise the NIH-WEB-SPACE. Each of these servers is "crawled" and indexed on an ongoing basis. Your search will produce a list of various documents, all of which will relate in some way to heart disease. The drawbacks of this approach are that the information is not organized by theme and that the references are often a mix of information for professionals and patients. Nevertheless, a large number of the listed Web sites provide useful background information. We can only recommend this route, therefore, for relatively rare or specific disorders, or when using highly targeted searches. To use the NIH search utility, visit the following Web page: **http://search.nih.gov/index.html**.

Additional Web Sources

A number of Web sites are available to the public that often link to government sites. These can also point you in the direction of essential information. The following is a representative sample:

- AOL: **http://search.aol.com/cat.adp?id=168&layer=&from=subcats**
- Family Village: **http://www.familyvillage.wisc.edu/specific.htm**
- Google: **http://directory.google.com/Top/Health/Conditions_and_Diseases/**
- Med Help International: **http://www.medhelp.org/HealthTopics/A.html**

- Open Directory Project: **http://dmoz.org/Health/Conditions_and_Diseases/**
- Yahoo.com: **http://dir.yahoo.com/Health/Diseases_and_Conditions/**
- WebMD®Health: **http://my.webmd.com/health_topics**

Associations and Heart Disease

The following is a list of associations that provide information on and resources relating to heart disease:

- **American Health Assistance Foundation**

  Telephone: (301) 948-3244 Toll-free: (800) 437-2423

  Fax: (301) 258-9454

  Web Site: http://www.ahaf.org

  Background: The American Health Assistance Foundation (AHAF) is a national nonprofit health organization dedicated to raising funds for scientific research on age-related and degenerative diseases, educating the public about these diseases, and providing financial assistance to Alzheimer s disease patients and their caregivers. Some of the age-related and degenerative diseases with which AHAF is concerned include Alzheimer s disease, macular degeneration, glaucoma, **heart disease,** and stroke. AHAF is an umbrella organization comprised of five separate and distinct programs: Alzheimer s Disease Research, National Glaucoma Research, Macular Degeneration Research, National Heart Foundation, and the Alzheimer s Family Relief Program. Currently AHAF supports 22 investigations focusing on the causes of and potential cures for Alzheimer s disease, 16 research projects studying glaucoma, and nine studies of **heart disease** and stroke. Since its inception in 1973, AHAF has awarded more than $42 million in research grants. The Alzheimer s Family Relief Program has provided more than $1.4 million in emergency assistance grants since it began in 1988. The Foundation produces educational materials including brochures, pamphlets, booklets, and a regular newsletter.

  Relevant area(s) of interest: Heart Disease

- **American Heart Association**

  Telephone: (214) 373-6300 Toll-free: (800) 242-8721

  Fax: (214) 373-0268

  Email: inquire@heart.org

  Web Site: http://www.americanheart.org

  Background: The American Heart Association (AHA), a national not-for-profit voluntary health agency funded by private contributions, is dedicated to the reduction of death and disability from cardiovascular diseases including heart diseases and stroke. The Association consists of approximately 2,000 community organizations in all states, the District of Columbia, and Puerto Rico. More than 4 million people volunteer with the Association to fight cardiovascular diseases, the nation s number one cause of death and leading cause of disability. Preventing **heart disease** and stroke is the first priority of the American Heart Association. In support of this goal, the Association has contributed more than one billion dollars to cardiovascular research since 1949. The

Association also distributes a variety of educational materials and sponsors continuing medical education (CME) seminars and meetings.

Relevant area(s) of interest: Heart Disease

- **American Lyme Disease Foundation, Inc**

  Telephone: (914) 277-6970 Toll-free: (800) 876-5963

  Fax: (914) 277-6974

  Email: inquire@aldf.com

  Web Site: http://www.aldf.com

  Background: The American Lyme Disease Foundation, Inc. (ALDF) is a nonprofit organization that is dedicated to advancing treatment, research, prevention, and public awareness of Lyme disease and other tickborne illnesses. Lyme disease is an infectious tick-transmitted disease characterized by an early skin lesion and, subsequently, a growing red area on the skin (i.e., 'bull's eye' rash). In some cases, later symptoms may include joint, neurological, and/or heart problems. Established in 1990, the Foundation's work focuses on public and professional education and support of research. ALDF produces several brochures, including 'A Quick Guide to Lyme Disease,' 'Understanding Ticks and Lyme Disease (new in 2003, a 'Tick Identification Card' (which helps identify different types of ticks and proper removal of them)and a 'National Clinical Conference on Lyme Disease' supplement to the American Journal of Medicine (April 1995). Other educational materials include reports, videos, and a selection of audiovisual aids. The Foundation provides a toll-free informational number and a national physician referral service. The ALDF also supports peer-revied research into the prevention and control of tick-borne infections, and, in collaboration with the Dutchess County Department of Health and the Institute of Ecosystem Studies in Millbrook, NY, has been awarded a 3-year $300,000/yr. grant from the Centers for Disease Control and Prevention (CDC) to develop and implement innovative new programs to reduce tick habitat and populations at selected sites in the county to reduce disease risk.

- **Association Francophone Des Glycogenoses**

  Telephone: 02 47 87 83 0318

  Fax: 02 47 87 83 0318

  Email: ahugon@infonie.fr

  Web Site: http://www.infobiogen.fr/agora/associations/AFG/

  Background: The Association Francophone Des Glycogenoses (AFG) is a unique organization dedicated to assisting individuals and families affected by a rare group of genetic diseases that impair the body s ability to properly metabolize or break down glycogen (glycogenesis). Termed 'glycogen storage diseases,' these familial disorders are characterized by the abnormal accumulation of glycogen in various organs of the body. Symptoms vary greatly depending upon the specific organ systems that are affected and may include muscle weakness without loss of muscle mass (atrophy), abnormal enlargement of the heart (cardiomegaly), liver (hepatomegaly), and/or tongue (macroglossia), as well as heart and kidney problems. These diseases include (but are not limited to) Von Gierke Disease (Type 1), Cori Disease (Type III), Andersen Disease (Type IV), and Pompe Disease (Type II). Established in 1992, the Association is dedicated to identifying families affected by these diseases and providing appropriate

information about the different forms of the disease. In addition, the Association Francophone Des Glycogenoses is interested in stimulating ongoing research into the treatment and potential cure of these disorders.

- **Birth Defect Research for Children, Inc**

  Telephone: (407) 895-0802

  Fax: (407) 895-0824

  Email: abcd@birthdefects.org

  Web Site: http://www.birthdefects.org

  Background: Birth Defect Research for Children, Inc., (BDRC) formerly the Association of Birth Defect Children gives parents and expectant parents information about specific birth defects, their causes and treatments, support group referrals and parent-matching services. BDRC also provides information about environmental exposures that may be associated with birth defects. To study these exposures further, BDRC sponsors the National Birth Defect Registry, a research project designed to collect data on all kinds of birth defects and prenatal/preconceptual exposures of mothers and fathers.

- **Carcinoid Cancer Foundation, Inc**

  Telephone: (914) 683-1001 Toll-free: (888) 722-3132

  Fax: (914) 683-5919

  Email: mwarner@carcinoid.org

  Web Site: http://www.carcinoid.org

  Background: The Carcinoid Cancer Foundation, Inc. is a not-for-profit organization dedicated to encouraging and supporting research and education concerning carcinoid tumors and carcinoid syndrome. A carcinoid tumor is a rare, slow growing form of cancer characterized by overgrowth of certain cells that secrete serotonin, a naturally occurring derivative of the amino acid tryptophan. Serotonin has many functions including regulating activity of the intestinal tract. In some cases, individuals with carcinoid tumors may experience abnormally increased secretion of serotonin, resulting in carcinoid syndrome. In individuals with carcinoid syndrome, associated symptoms may include intense flushing of the face and upper body, wheezing, weight loss, severe diarrhea, and, in some cases, ulcer-like symptoms and/or eventual heart failure. The Carcinoid Cancer Foundation was established in 1968 to support research that will improve the understanding, diagnosis, and treatment of carcinoid tumor and carcinoid syndrome. The Foundation also provides understandable information to affected individuals and family members, engages in professional education, and offers networking services to affected families. In addition, the Carcinoid Cancer Foundation has a web site on the Internet that answers 'frequently asked questions' (FAQ) on carcinoid tumor and carcinoid syndrome; offers comprehensive information on diagnosis and treatment; provides dynamic linkage to support organizations, discussion groups, related web sites, and transportation assistance organizations; and offers information on current research and references.

- **Cardiomyopathy Association**

  Telephone: 0144(192) 324-9977

  Fax: 0144(192) 324-9987

Web Site: http://www.cardiomyopathy.org

Background: The Cardiomyopathy Association is an international not-for-profit self-help organization dedicated to providing information and support to individuals affected by cardiomyopathy, their family members, and medical professionals. Cardiomyopathy is a general diagnostic term indicating a primary noninflammatory disease of the heart muscle (myocardium) that is often due to unknown causes. Established in 1989 and consisting of 1,000 members throughout the world, the Cardiomyopathy Association publishes informational brochures on different types of cardiomyopathy. Titles include 'Dilated Cardiomyopathy,' 'Hypertrophic Cardiomyopathy,' and 'Arrhythmogenic Right Ventricular Cardiomyopathy.' The Association also offers videotapes on these topics. In addition to disseminating information, the Association also provides counseling to affected individuals and their family members and endeavors to open the lines of communication between affected individuals and families through an international networking program.

- **Children's Cardiomyopathy Foundation**

  Telephone: (201) 227-7016

  Fax: (201) 227-7016

  Email: info@childrenscardiomyopathy

  Web Site: www.childrenscardiomyopathy.org

  Background: The Children s Cardiomyopathy Foundation (C.C.F.) was established in 2002 by a parent who lost two young children to cardiomyopathy, a rare and underdiagnosed heart condition. C.C.F. is a non-profit, tax-exempt organization dedicated to accelerating the search for a cause and cure for pediatric cardiomyopathy. Run entirely by volunteers and guided by a medical advisory board, C.C.F. supports critical scientific and medical research into the genetic cause, early detection and effective treatment of this chronic disease. In addition, C.C.F. promotes physician education, public awareness, patient support, and advocacy for affected children and their families. C.C.F. provides a pamphlet, family directory and website with detailed information (medical information, coping and healing tips, resource links, and discussion forum) on the disease.

  Relevant area(s) of interest: Heart Disease

- **Children's Gaucher Research Fund**

  Telephone: (916) 797-3700

  Fax: (916) 797-3707

  Email: research@childrensgaucher.org

  Web Site: http://www.childrensgaucher.org

  Background: The Children s Gaucher Research Fund is a non-profit organization that raises money for medical research aimed at finding a cure for Gaucher disease Type 2 and Type 3. It also provides support to families of affected children and allows families who have lost a child to this disease to participate, in their child s honor, in fund-raising for medical research. Gaucher disease is a rare, inherited, metabolic disorder in which the body is unable to rid itself of worn-out red and white blood cells so that these cells accumulate in the liver, spleen, bone marrow, and, sometimes, heart and lungs. In Children s Gaucher disease, more commonly known as Types 2 and 3 Gaucher disease, all of the above-mentioned symptoms exist, but the disease is also characterized by

certain neurologic symptoms such as ocular motor apraxia, a defect of horizontal eye movements; breathing problems and, sometimes, speech or cognitive delay. Founded in 1999, the Children s Gaucher Research Fund has approximately 1900 members. It serves families in the United States and Canada.

- **Congenital Heart Anomalies, Support, Education, and Resources**

  Telephone: (419) 825-5575

  Fax: (419) 825-2880

  Email: myer106w@wonder.em.cdc.gov or chaser@compuserve.com

  Web Site: http://www.csun.edu/~hfmth006/chaser/

  Background: Congenital Heart Anomalies, Support, Education, and Resources (CHASER) is a not-for-profit voluntary health organization whose primary purpose is patient and family support for children with congenital **heart disease** and acquired heart disorders. Established in 1993, the organization seeks to increase public awareness about these disorders and provide educational materials to affected individuals, their families, and physicians. In addition, this self-help organization provides a networking program for both parents and young adults. Educational materials include a quarterly publication, newsletters, a variety of informational brochures, updates on medical research, and a directory of pediatric heart surgeons and treatment facilities. CHASER also maintains an international directory of individuals diagnosed with congenital and/or acquired heart defects.

  Relevant area(s) of interest: Heart Disease

- **Congenital Heart Information Network**

  Telephone: (215) 493-3068

  Fax: (215) 493-3068

  Email: mb@tchin.org

  Web Site: http://www.tchin.org

  Background: The Congenital Heart Information Network, a voluntary health organization, serves an international population, including people in Canada, Europe, the United Kingdom, Australia, Asia, and the Middle East, with educational materials, referrals, advocacy, networking, and support. Its materials and services are designed for patients and families, health professionals, other professionals such as teachers and lawyers, and the general public. It provides information and other services related to all forms of congenital heart defects and syndromes, as well as heart diseases, such as cardiomyopathy and Kawasaki disease, that begin in childhood.

- **Endocrine Society**

  Telephone: (301) 941-0200 Toll-free: (800) 467-6663

  Fax: (301) 941-0259

  Email: endostaff@endo-society.org

  Web Site: http://www.endo-society.org

  Background: The Endocrine Society is a not-for-profit organization dedicated to enhancing the understanding of hormonal communication at the molecular, cellular, and systems levels in order to promote improved prevention, diagnosis, and treatment

of endocrine disorders. The human body has 11 major endocrine glands, including the adrenal, thyroid, pancreas, pituitary, and thymus glands. The endocrine glands and tissues secrete hormones, the substances that regulate the body's reproduction, growth, and development; its response to the environment; and the provision of energy and nutrients required for cell function. The Society, which was established in 1916 and currently consists of approximately 9,000 members in over 70 countries, is the world's largest organization dedicated to the research, study, and clinical practice of endocrinology. The scientists, educators, clinicians, practicing physicians, nurses, and students who make up the organization's membership represent all basic, applied, and clinical interests in endocrinology. Endocrinologists conduct research; provide treatment for a wide range of functions and disorders of the human body, including infertility, metabolic disorders, **heart disease,** glandular cancers, short stature, genetic dysfunction, diabetes, and hormonal imbalances; and develop new drugs and treatments through biogenetic and synthetic techniques. The Endocrine Society is dedicated to fostering a greater understanding of endocrinology among the general public and practioners of complementary medical disciplines; serving as a primary advocate and integrative force for clinicians and investigators; functioning as the major provider of services and information to the endocrine community and the general public; and promoting the interests of all endocrinologists at the national scientific research and health policy levels of government. The Society offers continuing medical education (CME) meetings and courses to keep the scientific and medical communities current concerning the latest research findings and treatments in the field; publishes four medical journals and a variety of additional professional publications; and conducts an annual meeting during which scientific and clinical papers are presented.

- **Foundation for Sarcoidosis Research**

  Telephone: (773) 665-2400

  Fax: (773) 665-0805

  Email: fsr@fsrchicago.org

  Web Site: http://www.fsrchicago.org

  Background: The Foundation for Sarcoidosis Research (FSR) is a 501(c)3 organization dedicated to supporting research into the causes of sarcoidosis and to finding a cure. Sarcoidosis is a debilitating and potentially fatal disease causing massive tissue inflammation and damaging major organs, primarily the lungs. The disease can also attack the heart, eyes, central nervous system, liver, spleen, skin, joints, and bones. A chronic disease of unknown cause, it affects one of every 2,000 Americans, primarily African-Americans.

- **Heart and Stroke Foundation of Canada**

  Telephone: (613) 569-4361

  Fax: (613) 569-3278

  Email: itstaff@hsf.ca

  Web Site: www.heartandstroke.co; www.fmcoeur.ca

  Background: The Heart and Stroke Foundation is a national voluntary non-profit organization whose mission is to improve the health of Canadians by preventing and reducing disability and death from **heart disease** and stroke through research, health promotion and advocacy. The Heart and Stroke Foundation of Canada is a Federation of

10 independent Provincial Foundations and one national office, led and supported by a force of more than 250,000 volunteers. To fulfill its mission and objectives, the Foundation offers a variety of programs and services including sponsoring conferences and scientific meetings; offering scientific awards to recognize the merits of medical researchers' contributions; providing advocacy and representation for its provincial divisions; engaging in lobbying efforts; and providing position statements. The Heart and Stroke Foundation of Canada also regularly sponsors campaigns to promote public awareness, including 'Heart Month' and 'Health CheckTM' a food information program to help consumers make wise food choices when shopping for groceries. The Foundation provides regular news releases, health news and statistics, fact sheets on all aspects of stroke and **heart disease,** 'HeartSmartTM' cook books, a video course in cardiopulmonary resuscitation (CPR), and the 'Canadian Family Guide to Stroke.' The Heart and Stroke Foundation of Canada also has a web site that discusses its mission, goals, and services; provides information on its provincial divisions; posts news releases, fact sheets; and the Foundation's annual report.

Relevant area(s) of interest: Heart Disease

- **Heart Foundation of Australia**

  Telephone: 61 2 6269 2631

  Fax: 61 2 6282 5877

  Email: julia.trevena@heartfoundation.com.au

  Web Site: http://www.heartfoundation.com.au/

  Background: The Heart Foundation of Australia is an independent, not-for-profit organization dedicated to reducing disability and life-threatening complications from heart and blood vessel (cardiovascular) disease and improving the cardiovascular health of Australians. Established in 1959, the Foundation currently has divisional offices in every state and territory in Australia. The Heart Foundation of Australia works to fulfill its mission and objectives by supporting research concerning cardiovascular health and disease as well as offering educational and other programs directed to health professionals, patients, family members, and the general public that will help to promote cardiovascular health. The Foundation supports research programs in major Australian hospitals and universities and establishes links with leading research institutions in the United States and Europe. In addition, the Foundation offers a comprehensive series of publications on the prevention and treatment of cardiovascular disease. The Foundation provides such resource materials to affected individuals, family members, physicians, allied health professionals, teachers, employers, community groups, journalists, and the general public. In addition, the Heart Foundation of Australia has a web site on the Internet that discusses its mission, objectives, and services; provides divisional office contact information; and offers access to factsheets on cardiovascular disease, including those entitled 'The Heart,' 'Risk factors for **heart disease,**' Nutrition and cardiovascular disease,' and 'What is heart disease?'.

- **Hemochromatosis Foundation, Inc**

  Telephone: (518) 489-0972

  Fax: (518) 489-0227

  Web Site: http://www.hemochromatosis.org

Background: The Hemochromatosis Foundation is a national not-for-profit organization dedicated to finding a cure for Hemochromatosis. Hereditary Hemochromatosis (HH) is a metabolic disorder characterized by increased absorption of dietary iron. Without appropriate treatment, excessive iron may accumulate in the liver, heart, pancreas, and other organs, causing organ dysfunction and tissue damage. Established in 1971, the Foundation is committed to fostering understanding of the disease by supporting activities that encourage research; advising legislative and regulatory bodies at the local and federal levels; increasing general awareness of Hemochromatosis to help ensure early diagnosis and prompt treatment; and improving the quality of life for affected individuals and family members. The Foundation also establishes local chapters; sponsors discussion groups where people share their experiences; and provides physician referrals, general information, and local contacts. Educational materials produced by the Foundation include a brochure series.

- **International Center for Fabry Disease**

  Telephone: (212) 241-6944

  Fax: (212) 348-5811

  Email: fabry.disease@mssm.edu

  Web Site: http://www.mssm.edu/genetics/fabry

  Background: The International Center for Fabry Disease is a voluntary not-for-profit organization dedicated to aiding in the diagnosis, treatment, and management of Fabry Disease, a rare inherited disorder of lipid metabolism characterized by the abnormal accumulation of certain fatty substances in various organs of the body. Symptoms may include clusters of discolorations on the skin (angiokeratomas), abdominal pain, pain in the hands and feet, as well as intolerance to heat due to a lack of sweating. Later in the course of the disease, kidney failure, heart problems, and/or neurological symptoms may cause serious complications. Established in 1974, the International Center for Fabry Disease promotes and supports ongoing research on Fabry Disease and engages in patient and professional education. The Center also makes appropriate referrals and provides educational and support information to affected individuals, family members, health care professionals, and the general public through its database, reports, and brochures.

- **Iron Overload Diseases Association, Inc**

  Telephone: (561) 840-8512

  Fax: (561) 842-9881

  Email: iod@ironoverload.org

  Web Site: http://www.ironoverload.org

  Background: Iron Overload Diseases Association, Inc. is a voluntary not-for-profit organization dedicated to leading the search for individuals who have undiagnosed Hemochromatosis and preventing the health problems that may result. Hereditary Hemochromatosis is a metabolic disorder characterized by increased absorption of dietary iron. Without appropriate treatment, excessive iron may accumulate in the liver, heart, pancreas, and other organs, causing organ dysfunction and tissue damage. Established in 1981, the Association is also committed to providing information and support to affected individuals and their families; educating the general public; promoting and supporting research; and pressing for earlier diagnosis and more

effective treatment for Hemochromatosis. The Association acts as an international clearinghouse for affected individuals, family members, and physicians; provides telephone consultations; offers referrals to genetic counseling and support groups; promotes patient advocacy; and conducts an annual medical symposium as well as conference meetings between patients and health care professionals. The Iron Overload Diseases Association also provides a variety of educational and support materials including books, brochures, fact sheets, and a bimonthly newsletter entitled 'Ironic Blood.'.

- **Kawasaki Families' Network**

  Telephone: (808) 525-8053

  Fax: (808) 525-8055

  Email: kawasaki@compuserve.com

  Web Site: http://ourworld.compuserve.com/homepages/kawasaki

  Background: The Kawasaki Families' Network is a not-for-profit organization dedicated to providing information and support to individuals and families affected by Kawasaki Syndrome, an inflammatory disease of childhood characterized by fever, skin rash, swollen lymph nodes, and inflammation of the blood vessels. Inflammatory changes cause destructive lesions in blood vessels and may lead to complications involving the liver, gall bladder, and heart. Established in 1996 and currently consisting of approximately 70 members, the Network facilitates networking opportunities through the mail and the Internet and offers educational materials including an occasional newsletter entitled 'Heartlines.'.

- **Kids With Heart National Association for Children's Heart Disorders,Inc**

  Telephone: (920) 498-0058 Toll-free: (800) 538-5390

  Fax: (920) 498-0058

  Email: kdswhrt@execpc.com

  Web Site: http://www.execpc.com/~kdswhrt/

  Background: The Kids With Heart National Association for Children s Heart Disorders, Inc., is a not-for-profit corporation organized to provide support, education, and informational materials to families of children who have heart disorders, both congenital and acquired, to assist them in making decisions regarding their child s care. More than 25,000 infants are born with heart defects each year in the United States. Heart defects are among the most common birth defects, and are the leading cause of birth defect-related deaths. The association maintains a national database of families affected by congenital heart defects, which includes adult congenital heart defect survivors, families of children with congenital heart defects, and families who have lost a child to congenital heart defects, as well as a national database of local support groups who have registered with Kids with Heart NACHD.

- **Mainstream, Inc**

  Telephone: (301) 654-2400

  Fax: (301) 654-2403

  Email: mainstrm@aol.com

Background: Mainstream, Inc. is a nonprofit organization dedicated to improving competitive employment opportunities for persons with disabilities. Established in 1975, Mainstream was founded by Harold E. Krents, a graduate of Harvard Law School. His credentials and talents were seemingly useless in a business environment that judged him by his disability (blindness). He decided to change this for himself and all persons with disabilities by establishing Mainstream. Mainstream provides specialized services and acts as a bridge that links service providers, employers, and persons with disabilities. To achieve its goals, the organization provides its constituents with training, educational publications, and videos on disability employment issues. Educational materials include a magazine entitled 'Employment In the Mainstream' that reports on the latest trends and developments affecting the employment of Americans with disabilities. A brochure and audio-visual aids are also available. Programs include Project LINK, a model employment service program for persons with disabilities in Dallas, TX and Washington D.C., as well as the Disability Employment Network, a pilot program that provides counseling and referral services to job hunters in the New York City area.

Relevant area(s) of interest: Heart Disease

- **March of Dimes Birth Defects Foundation**

  Telephone: (914) 428-7100 Toll-free: (888) 663-4637

  Fax: (914) 997-4763

  Email: Askus@marchofdimes.com

  Web Site: http://www.marchofdimes.com

  Background: The March of Dimes Birth Defects Foundation is a national not-for-profit organization that was established in 1938. The mission of the Foundation is to improve the health of babies by preventing birth defects and infant mortality. The March of Dimes funds programs of research, community services, education, and advocacy. Educational programs that seek to prevent birth defects are important to the Foundation and to that end it also produces a wide variety of printed informational materials and videos. The Pregnancy and Newborn Health Education Center staffs trained health information specialists who provide researched information on pregnancy issues, complications and risks, newborn care, birth defects, genetic diseases and related topics as well as referrals to relevant organizations and support groups.

- **Mended Hearts, Inc**

  Telephone: (214) 706-1442 Toll-free: (800) 242-8721

  Fax: (214) 706-5231

  Email: dbonham@heart.org

  Web Site: http://www.mendedhearts.org

  Background: Mended Hearts, Inc. is a not-for-profit organization that is affiliated with the American Heart Association. Both organizations are dedicated to supporting one another's goals and objectives and are committed to offering help, support, encouragement, and information to individuals affected by **heart disease,** their families, and other caregivers. Established in 1955, Mended Hearts serves as an extended support group comprised of affected individuals, spouses, other family members and caregivers, health care professionals, and other interested persons. The organization's primary focus is helping individuals cope with emotional recovery from **heart disease.** Mended Hearts

currently includes local chapters in approximately 260 cities across the United States and in Sudbury, Ontario, Canada. The organization works to fulfill its mission and objectives by distributing educational information; cooperating with other organizations in educational and research activities; establishing and assisting **heart disease** rehabilitation programs for members and their families and caregivers; and establishing and maintaining a program of assistance for physicians, nurses, medical professionals, and health care organizations who work with **heart disease** patients, their families, and other caregivers. Mended Hearts is also committed to offering local programs in which members visit, with physician approval, and offer encouragement and support to **heart disease** patients and their families or other caregivers. Accredited visitors meet affected individuals and families in hospitals and homes as well as via the telephone or the Internet. Mended Hearts also conducts an annual national convention to educate and train members and chapter officers; offers regional workshops to train members and officers and to provide medical updates; publishes a regular journal entitled 'HEARTBEAT'; and maintains a web site.

- **Montgomery Heart Foundation for Cardiomyopathy**

  Telephone: (410) 254-6370

  Fax: (410) 254-6379

  Email: rmonty@welchlink.welch.jhu.edu

  Web Site: http://www.med.jhu.edu/cardiomyopathy

  Background: The Montgomery Heart Foundation for Cardiomyopathy is a not-for-profit organization dedicated to providing private funding to support genetic research for familial cardiomyopathy and raising awareness of the disease to help promote the advancement of a cure. Cardiomyopathy is a term used to describe a heterogeneous group of disorders causing primary heart muscle dysfunction in both men and women, often leading to heart failure or life-threatening complications. Established in 1993, the Foundation provides direct financial support for research studies focusing on the cause and treatment of cardiomyopathies; creates a constituency for cardiomyopathy and makes the needs and interests of this known to researchers, clinicians, and the general public; facilitates the flow of information between researchers, clinicians, and affected individuals about new developments and breakthroughs; informs the general public about this disease; and offers up-to-date information to affected individuals and their families and helps them to understand what it means to have this disease.

- **National Association for Pseudoxanthoma Elasticum (NAPE)**

  Telephone: (314) 963-9153

  Fax: (314) 977-3587

  Email: benham@SLU.edu

  Web Site: http://www.napxe.org

  Background: The National Association for Pseudoxanthoma Elasticum (NAPE) is a voluntary not-for-profit organization dedicated to disseminating information on Pseudoxanthoma Elasticum (PXE). PXE is a rare connective tissue disease that is inherited and progressive and affects the elastic tissues of the body; abnormal accumulations of calcium salts (calcifications) develop within elastic fibers of the skin, eyes, and cardiovascular system (i.e., heart and blood vessels). The Association promotes and supports ongoing medical research into possible new treatments,

prevention, and eventual cure for PXE. Established in 1988, the National Association for Pseudoxanthoma Elasticum promotes patient advocacy and provides referrals to support groups, genetic counseling, and other services. The National Association for Pseudoxanthoma Elasticum produces and distributes an educational brochure and quarterly newsletters to affected individuals, families, health care professionals, and the general public. NAPE also sponsors a matching Research Fund to be donated from time to time to a PXE research study in need of funding. NAPE has also established a program to help members purchase low vision aids that they might not be able to afford without some financial assistance.

- **National Fetal AntiConvulsant Syndrome Association**

  Telephone: 01 461 206 870

  Fax: 01 461 206 870

  Email: facsline3@aol.com

  Web Site: http://www.facsline.org

  Background: The National Fetal Anticonvulsant Syndrome Association provides advice and support to families and health workers involved with children who have been affected by anticonvulsant drugs prescribed for their mothers during pregnancy. The major problems encountered include spina bifida, cleft palate, congenital **heart disease,** kidney abnormalities, and limb defects. There is also an increased incidence of minor birth defects, including learning problems and speech delay. The Association works to increase awareness of these various problems, to put families in touch with each other, to encourage families in their quest for a firm diagnosis, to ensure that future mothers are made aware of risks, and to promote and participate in research.

- **National Hospice and Palliative Care Organization**

  Telephone: (703) 837-1500 Toll-free: (800) 658-8898

  Fax: (703) 837-1233

  Email: nhpco_info@nhpco.org

  Web Site: http://www.nhpco.org

  Background: The National Hospice and Palliative Care Organization is the oldest and largest nonprofit membership organization representing hospice and palliative care programs and professionals in the United States. The organization is committed to improving end-of-life care and expanding access to hospice care with the goal of profoundly enhancing quality of life for people dying in America and their loved ones. NHPCO collaborates with other end-of-life organizations. NHPCO offers memberships to hospices, palliative care programs, grief/bereavement centers, education and research organizations, foundations, home health agencies, companion services, medical supply companies, pharmaceutical organizations, staffing agencies, software vendors, and healthcare consultants. NHPCO has knowledge of 3,240 operational or planned hospices operating in all 50 states, the District of Columbia, the Virgin Islands, Puerto Rico, and Guam. NHPCO represents over 80 percent of hospices nationwide. NHPCO estimates that almost 800,000 patients were served by hospice in 2001. Over 90 percent of the patients served were provided hospice care by NHPCO members.

  Relevant area(s) of interest: Heart Disease

- **Progeria Research Foundation**

  Telephone: (978) 535-2594

  Fax: (978) 535-5849

  Email: progeria@netzero.net

  Web Site: http://www.progeriaresearch.org

  Background: The Progeria Research Foundation is a non-profit organization whose mission is to fund medical research to discover the cause, effective treatment, and cure for Hutchinson-Gilford Progeria Syndrome ('Progeria'), and to raise awareness about Progeria by educating the families, the public and health professionals. Progeria is a terminal, 'premature aging' syndrome that afflicts children. After the first year of life, severe developmental failure begins and the child's hair falls out. Over the next few years, the children are susceptible to strokes, arthritis, and osteoporosis. Their final average height is 3 feet, their final average weight is 33 pounds, and the average life expectancy is 13 years. Amazingly, the brain is unaffected; these remarkable children are recognized for their intelligence and lovable personalities throughout their shortened lives. Children with Progeria are genetically predisposed to premature, progressive cardiovascular disease. Death occurs almost exclusively due to widespread atherosclerosis (heart disease). Thus, finding a cure for Progeria may help the millions of people that suffer from **heart disease** and other conditions related to aging. PRF is always looking for volunteers to translate for the families that are located all over the world and speak over a dozen different languages; to donate or hold fundraisers to raise money; and to participate in many other ways to further the quest for a cure.

- **TAR Syndrome Association**

  Telephone: (609) 927-0418

  Fax: (609) 653-8639

  Email: spp212@aol.com

  Background: The TAR Syndrome Association, also known as TARSA, is an international not-for-profit self-help organization that was established in 1981. The group functions as a support system and educational resource for families affected by Thrombocytopenia Absent Radius (TAR) Syndrome. TAR Syndrome is a rare hereditary disorder characterized by abnormally low levels of circulating blood platelets and underdevelopment or absence of the radius, one of the bones of the forearm. In some cases, symptoms may include kidney abnormalities and congenital **heart disease.** Educational materials produced by the TAR Syndrome Association include informational brochures and an annual newsletter.

- **Vascular Disease Foundation**

  Telephone: (303) 949-8337 Toll-free: (866) 723-4636

  Fax: (303) 989-6522

  Email: info@vdf.org

  Web Site: http://www.vdf.org

  Background: The Vascular Disease Foundation was formed in 1998 to improve public recognition of the prevalence and seriousness of vascular disease, with an initial focus on increasing public awareness to improve prevention, diagnosis, and comprehensive treatment of peripheral arterial disease (PAD). The Foundation disseminates educational

information to the public, fosters vascular disease patient advocacy initiatives, and provides information about vascular disease to patients and to those at risk for development of common vascular disorders. PAD occurs because of blockages in the arteries in the legs, but it may also indicate that blockages are occurring in the arteries to the heart or brain. The Vascular Disease Foundation is increasing awareness and understanding of PAD and other vascular diseases for which early diagnosis and treatment may be very important.

Finding Associations

There are several Internet directories that provide lists of medical associations with information on or resources relating to heart disease. By consulting all of associations listed in this chapter, you will have nearly exhausted all sources for patient associations concerned with heart disease.

The National Health Information Center (NHIC)

The National Health Information Center (NHIC) offers a free referral service to help people find organizations that provide information about heart disease. For more information, see the NHIC's Web site at **http://www.health.gov/NHIC/** or contact an information specialist by calling 1-800-336-4797.

Directory of Health Organizations

The Directory of Health Organizations, provided by the National Library of Medicine Specialized Information Services, is a comprehensive source of information on associations. The Directory of Health Organizations database can be accessed via the Internet at **http://www.sis.nlm.nih.gov/Dir/DirMain.html**. It is composed of two parts: DIRLINE and Health Hotlines.

The DIRLINE database comprises some 10,000 records of organizations, research centers, and government institutes and associations that primarily focus on health and biomedicine. To access DIRLINE directly, go to the following Web site: **http://dirline.nlm.nih.gov/**. Simply type in "heart disease" (or a synonym), and you will receive information on all relevant organizations listed in the database.

Health Hotlines directs you to toll-free numbers to over 300 organizations. You can access this database directly at **http://www.sis.nlm.nih.gov/hotlines/**. On this page, you are given the option to search by keyword or by browsing the subject list. When you have received your search results, click on the name of the organization for its description and contact information.

The Combined Health Information Database

Another comprehensive source of information on healthcare associations is the Combined Health Information Database. Using the "Detailed Search" option, you will need to limit your search to "Organizations" and "heart disease". Type the following hyperlink into your

Web browser: **http://chid.nih.gov/detail/detail.html**. To find associations, use the drop boxes at the bottom of the search page where "You may refine your search by." For publication date, select "All Years." Then, select your preferred language and the format option "Organization Resource Sheet." Type "heart disease" (or synonyms) into the "For these words:" box. You should check back periodically with this database since it is updated every three months.

The National Organization for Rare Disorders, Inc.

The National Organization for Rare Disorders, Inc. has prepared a Web site that provides, at no charge, lists of associations organized by health topic. You can access this database at the following Web site: **http://www.rarediseases.org/search/orgsearch.html**. Type "heart disease" (or a synonym) into the search box, and click "Submit Query."

APPENDIX C. FINDING MEDICAL LIBRARIES

Overview

In this Appendix, we show you how to quickly find a medical library in your area.

Preparation

Your local public library and medical libraries have interlibrary loan programs with the National Library of Medicine (NLM), one of the largest medical collections in the world. According to the NLM, most of the literature in the general and historical collections of the National Library of Medicine is available on interlibrary loan to any library. If you would like to access NLM medical literature, then visit a library in your area that can request the publications for you.[27]

Finding a Local Medical Library

The quickest method to locate medical libraries is to use the Internet-based directory published by the National Network of Libraries of Medicine (NN/LM). This network includes 4626 members and affiliates that provide many services to librarians, health professionals, and the public. To find a library in your area, simply visit **http://nnlm.gov/members/adv.html** or call 1-800-338-7657.

Medical Libraries in the U.S. and Canada

In addition to the NN/LM, the National Library of Medicine (NLM) lists a number of libraries with reference facilities that are open to the public. The following is the NLM's list and includes hyperlinks to each library's Web site. These Web pages can provide information on hours of operation and other restrictions. The list below is a small sample of

[27] Adapted from the NLM: **http://www.nlm.nih.gov/psd/cas/interlibrary.html**.

libraries recommended by the National Library of Medicine (sorted alphabetically by name of the U.S. state or Canadian province where the library is located)[28]:

- **Alabama:** Health InfoNet of Jefferson County (Jefferson County Library Cooperative, Lister Hill Library of the Health Sciences), **http://www.uab.edu/infonet/**
- **Alabama:** Richard M. Scrushy Library (American Sports Medicine Institute)
- **Arizona:** Samaritan Regional Medical Center: The Learning Center (Samaritan Health System, Phoenix, Arizona), **http://www.samaritan.edu/library/bannerlibs.htm**
- **California:** Kris Kelly Health Information Center (St. Joseph Health System, Humboldt), **http://www.humboldt1.com/~kkhic/index.html**
- **California:** Community Health Library of Los Gatos, **http://www.healthlib.org/orgresources.html**
- **California:** Consumer Health Program and Services (CHIPS) (County of Los Angeles Public Library, Los Angeles County Harbor-UCLA Medical Center Library) - Carson, CA, **http://www.colapublib.org/services/chips.html**
- **California:** Gateway Health Library (Sutter Gould Medical Foundation)
- **California:** Health Library (Stanford University Medical Center), **http://www-med.stanford.edu/healthlibrary/**
- **California:** Patient Education Resource Center - Health Information and Resources (University of California, San Francisco), **http://sfghdean.ucsf.edu/barnett/PERC/default.asp**
- **California:** Redwood Health Library (Petaluma Health Care District), **http://www.phcd.org/rdwdlib.html**
- **California:** Los Gatos PlaneTree Health Library, **http://planetreesanjose.org/**
- **California:** Sutter Resource Library (Sutter Hospitals Foundation, Sacramento), **http://suttermedicalcenter.org/library/**
- **California:** Health Sciences Libraries (University of California, Davis), **http://www.lib.ucdavis.edu/healthsci/**
- **California:** ValleyCare Health Library & Ryan Comer Cancer Resource Center (ValleyCare Health System, Pleasanton), **http://gaelnet.stmarys-ca.edu/other.libs/gbal/east/vchl.html**
- **California:** Washington Community Health Resource Library (Fremont), **http://www.healthlibrary.org/**
- **Colorado:** William V. Gervasini Memorial Library (Exempla Healthcare), **http://www.saintjosephdenver.org/yourhealth/libraries/**
- **Connecticut:** Hartford Hospital Health Science Libraries (Hartford Hospital), **http://www.harthosp.org/library/**
- **Connecticut:** Healthnet: Connecticut Consumer Health Information Center (University of Connecticut Health Center, Lyman Maynard Stowe Library), **http://library.uchc.edu/departm/hnet/**

[28] Abstracted from **http://www.nlm.nih.gov/medlineplus/libraries.html**.

- **Connecticut:** Waterbury Hospital Health Center Library (Waterbury Hospital, Waterbury), **http://www.waterburyhospital.com/library/consumer.shtml**
- **Delaware:** Consumer Health Library (Christiana Care Health System, Eugene du Pont Preventive Medicine & Rehabilitation Institute, Wilmington), **http://www.christianacare.org/health_guide/health_guide_pmri_health_info.cfm**
- **Delaware:** Lewis B. Flinn Library (Delaware Academy of Medicine, Wilmington), **http://www.delamed.org/chls.html**
- **Georgia:** Family Resource Library (Medical College of Georgia, Augusta), **http://cmc.mcg.edu/kids_families/fam_resources/fam_res_lib/frl.htm**
- **Georgia:** Health Resource Center (Medical Center of Central Georgia, Macon), **http://www.mccg.org/hrc/hrchome.asp**
- **Hawaii:** Hawaii Medical Library: Consumer Health Information Service (Hawaii Medical Library, Honolulu), **http://hml.org/CHIS/**
- **Idaho:** DeArmond Consumer Health Library (Kootenai Medical Center, Coeur d'Alene), **http://www.nicon.org/DeArmond/index.htm**
- **Illinois:** Health Learning Center of Northwestern Memorial Hospital (Chicago), **http://www.nmh.org/health_info/hlc.html**
- **Illinois:** Medical Library (OSF Saint Francis Medical Center, Peoria), **http://www.osfsaintfrancis.org/general/library/**
- **Kentucky:** Medical Library - Services for Patients, Families, Students & the Public (Central Baptist Hospital, Lexington), **http://www.centralbap.com/education/community/library.cfm**
- **Kentucky:** University of Kentucky - Health Information Library (Chandler Medical Center, Lexington), **http://www.mc.uky.edu/PatientEd/**
- **Louisiana:** Alton Ochsner Medical Foundation Library (Alton Ochsner Medical Foundation, New Orleans), **http://www.ochsner.org/library/**
- **Louisiana:** Louisiana State University Health Sciences Center Medical Library-Shreveport, **http://lib-sh.lsuhsc.edu/**
- **Maine:** Franklin Memorial Hospital Medical Library (Franklin Memorial Hospital, Farmington), **http://www.fchn.org/fmh/lib.htm**
- **Maine:** Gerrish-True Health Sciences Library (Central Maine Medical Center, Lewiston), **http://www.cmmc.org/library/library.html**
- **Maine:** Hadley Parrot Health Science Library (Eastern Maine Healthcare, Bangor), **http://www.emh.org/hll/hpl/guide.htm**
- **Maine:** Maine Medical Center Library (Maine Medical Center, Portland), **http://www.mmc.org/library/**
- **Maine:** Parkview Hospital (Brunswick), **http://www.parkviewhospital.org/**
- **Maine:** Southern Maine Medical Center Health Sciences Library (Southern Maine Medical Center, Biddeford), **http://www.smmc.org/services/service.php3?choice=10**
- **Maine:** Stephens Memorial Hospital's Health Information Library (Western Maine Health, Norway), **http://www.wmhcc.org/Library/**

- **Manitoba, Canada:** Consumer & Patient Health Information Service (University of Manitoba Libraries), **http://www.umanitoba.ca/libraries/units/health/reference/chis.html**
- **Manitoba, Canada:** J.W. Crane Memorial Library (Deer Lodge Centre, Winnipeg), **http://www.deerlodge.mb.ca/crane_library/about.asp**
- **Maryland:** Health Information Center at the Wheaton Regional Library (Montgomery County, Dept. of Public Libraries, Wheaton Regional Library), **http://www.mont.lib.md.us/healthinfo/hic.asp**
- **Massachusetts:** Baystate Medical Center Library (Baystate Health System), **http://www.baystatehealth.com/1024/**
- **Massachusetts:** Boston University Medical Center Alumni Medical Library (Boston University Medical Center), **http://med-libwww.bu.edu/library/lib.html**
- **Massachusetts:** Lowell General Hospital Health Sciences Library (Lowell General Hospital, Lowell), **http://www.lowellgeneral.org/library/HomePageLinks/WWW.htm**
- **Massachusetts:** Paul E. Woodard Health Sciences Library (New England Baptist Hospital, Boston), **http://www.nebh.org/health_lib.asp**
- **Massachusetts:** St. Luke's Hospital Health Sciences Library (St. Luke's Hospital, Southcoast Health System, New Bedford), **http://www.southcoast.org/library/**
- **Massachusetts:** Treadwell Library Consumer Health Reference Center (Massachusetts General Hospital), **http://www.mgh.harvard.edu/library/chrcindex.html**
- **Massachusetts:** UMass HealthNet (University of Massachusetts Medical School, Worchester), **http://healthnet.umassmed.edu/**
- **Michigan:** Botsford General Hospital Library - Consumer Health (Botsford General Hospital, Library & Internet Services), **http://www.botsfordlibrary.org/consumer.htm**
- **Michigan:** Helen DeRoy Medical Library (Providence Hospital and Medical Centers), **http://www.providence-hospital.org/library/**
- **Michigan:** Marquette General Hospital - Consumer Health Library (Marquette General Hospital, Health Information Center), **http://www.mgh.org/center.html**
- **Michigan:** Patient Education Resouce Center - University of Michigan Cancer Center (University of Michigan Comprehensive Cancer Center, Ann Arbor), **http://www.cancer.med.umich.edu/learn/leares.htm**
- **Michigan:** Sladen Library & Center for Health Information Resources - Consumer Health Information (Detroit), **http://www.henryford.com/body.cfm?id=39330**
- **Montana:** Center for Health Information (St. Patrick Hospital and Health Sciences Center, Missoula)
- **National:** Consumer Health Library Directory (Medical Library Association, Consumer and Patient Health Information Section), **http://caphis.mlanet.org/directory/index.html**
- **National:** National Network of Libraries of Medicine (National Library of Medicine) - provides library services for health professionals in the United States who do not have access to a medical library, **http://nnlm.gov/**
- **National:** NN/LM List of Libraries Serving the Public (National Network of Libraries of Medicine), **http://nnlm.gov/members/**

- **Nevada:** Health Science Library, West Charleston Library (Las Vegas-Clark County Library District, Las Vegas), **http://www.lvccld.org/special_collections/medical/index.htm**
- **New Hampshire:** Dartmouth Biomedical Libraries (Dartmouth College Library, Hanover), **http://www.dartmouth.edu/~biomed/resources.htmld/conshealth.htmld/**
- **New Jersey:** Consumer Health Library (Rahway Hospital, Rahway), **http://www.rahwayhospital.com/library.htm**
- **New Jersey:** Dr. Walter Phillips Health Sciences Library (Englewood Hospital and Medical Center, Englewood), **http://www.englewoodhospital.com/links/index.htm**
- **New Jersey:** Meland Foundation (Englewood Hospital and Medical Center, Englewood), **http://www.geocities.com/ResearchTriangle/9360/**
- **New York:** Choices in Health Information (New York Public Library) - NLM Consumer Pilot Project participant, **http://www.nypl.org/branch/health/links.html**
- **New York:** Health Information Center (Upstate Medical University, State University of New York, Syracuse), **http://www.upstate.edu/library/hic/**
- **New York:** Health Sciences Library (Long Island Jewish Medical Center, New Hyde Park), **http://www.lij.edu/library/library.html**
- **New York:** ViaHealth Medical Library (Rochester General Hospital), **http://www.nyam.org/library/**
- **Ohio:** Consumer Health Library (Akron General Medical Center, Medical & Consumer Health Library), **http://www.akrongeneral.org/hwlibrary.htm**
- **Oklahoma:** The Health Information Center at Saint Francis Hospital (Saint Francis Health System, Tulsa), **http://www.sfh-tulsa.com/services/healthinfo.asp**
- **Oregon:** Planetree Health Resource Center (Mid-Columbia Medical Center, The Dalles), **http://www.mcmc.net/phrc/**
- **Pennsylvania:** Community Health Information Library (Milton S. Hershey Medical Center, Hershey), **http://www.hmc.psu.edu/commhealth/**
- **Pennsylvania:** Community Health Resource Library (Geisinger Medical Center, Danville), **http://www.geisinger.edu/education/commlib.shtml**
- **Pennsylvania:** HealthInfo Library (Moses Taylor Hospital, Scranton), **http://www.mth.org/healthwellness.html**
- **Pennsylvania:** Hopwood Library (University of Pittsburgh, Health Sciences Library System, Pittsburgh), **http://www.hsls.pitt.edu/guides/chi/hopwood/index_html**
- **Pennsylvania:** Koop Community Health Information Center (College of Physicians of Philadelphia), **http://www.collphyphil.org/kooppg1.shtml**
- **Pennsylvania:** Learning Resources Center - Medical Library (Susquehanna Health System, Williamsport), **http://www.shscares.org/services/lrc/index.asp**
- **Pennsylvania:** Medical Library (UPMC Health System, Pittsburgh), **http://www.upmc.edu/passavant/library.htm**
- **Quebec, Canada:** Medical Library (Montreal General Hospital), **http://www.mghlib.mcgill.ca/**

- **South Dakota:** Rapid City Regional Hospital Medical Library (Rapid City Regional Hospital), **http://www.rcrh.org/Services/Library/Default.asp**
- **Texas:** Houston HealthWays (Houston Academy of Medicine-Texas Medical Center Library), **http://hhw.library.tmc.edu/**
- **Washington:** Community Health Library (Kittitas Valley Community Hospital), **http://www.kvch.com/**
- **Washington:** Southwest Washington Medical Center Library (Southwest Washington Medical Center, Vancouver), **http://www.swmedicalcenter.com/body.cfm?id=72**

ONLINE GLOSSARIES

The Internet provides access to a number of free-to-use medical dictionaries. The National Library of Medicine has compiled the following list of online dictionaries:

- ADAM Medical Encyclopedia (A.D.A.M., Inc.), comprehensive medical reference: **http://www.nlm.nih.gov/medlineplus/encyclopedia.html**
- MedicineNet.com Medical Dictionary (MedicineNet, Inc.): **http://www.medterms.com/Script/Main/hp.asp**
- Merriam-Webster Medical Dictionary (Inteli-Health, Inc.): **http://www.intelihealth.com/IH/**
- Multilingual Glossary of Technical and Popular Medical Terms in Eight European Languages (European Commission) - Danish, Dutch, English, French, German, Italian, Portuguese, and Spanish: **http://allserv.rug.ac.be/~rvdstich/eugloss/welcome.html**
- On-line Medical Dictionary (CancerWEB): **http://cancerweb.ncl.ac.uk/omd/**
- Rare Diseases Terms (Office of Rare Diseases): **http://ord.aspensys.com/asp/diseases/diseases.asp**
- Technology Glossary (National Library of Medicine) - Health Care Technology: **http://www.nlm.nih.gov/nichsr/ta101/ta10108.htm**

Beyond these, MEDLINEplus contains a very patient-friendly encyclopedia covering every aspect of medicine (licensed from A.D.A.M., Inc.). The ADAM Medical Encyclopedia can be accessed at **http://www.nlm.nih.gov/medlineplus/encyclopedia.html**. ADAM is also available on commercial Web sites such as drkoop.com (**http://www.drkoop.com/**) and Web MD (**http://my.webmd.com/adam/asset/adam_disease_articles/a_to_z/a**). The NIH suggests the following Web sites in the ADAM Medical Encyclopedia when searching for information on heart disease:

- **Basic Guidelines for Heart Disease**

  Heart disease
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/000147.htm

  Heart disease - resources
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002203.htm

  Heart disease and diet
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002436.htm

  Heart disease and vitamin E
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002100.htm

- **Signs & Symptoms for Heart Disease**

  Chest pain
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/003079.htm

High blood pressure
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/003082.htm

Muscle
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/003193.htm

Obesity
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/003101.htm

- **Nutrition for Heart Disease**

Carbohydrates
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002469.htm

Cholesterol
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002472.htm

Dietary cholesterol
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002472.htm

Fat
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002468.htm

Fats
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002468.htm

Fiber
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002470.htm

High-fat
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002468.htm

Low-fat
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002468.htm

Niacin
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002409.htm

Protein
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002467.htm

Riboflavin
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002411.htm

Saturated fat
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002468.htm

Vitamin E
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002406.htm

Vitamins
Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002399.htm

- **Background Topics for Heart Disease**

  HEART DISEASE
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/000147.htm

  Shellfish
  Web site: http://www.nlm.nih.gov/medlineplus/ency/article/002851.htm

Online Dictionary Directories

The following are additional online directories compiled by the National Library of Medicine, including a number of specialized medical dictionaries:

- Medical Dictionaries: Medical & Biological (World Health Organization): **http://www.who.int/hlt/virtuallibrary/English/diction.htm#Medical**
- MEL-Michigan Electronic Library List of Online Health and Medical Dictionaries (Michigan Electronic Library): **http://mel.lib.mi.us/health/health-dictionaries.html**
- Patient Education: Glossaries (DMOZ Open Directory Project): **http://dmoz.org/Health/Education/Patient_Education/Glossaries/**
- Web of Online Dictionaries (Bucknell University): **http://www.yourdictionary.com/diction5.html#medicine**

HEART DISEASE DICTIONARY

The definitions below are derived from official public sources, including the National Institutes of Health [NIH] and the European Union [EU].

5-hydroxyindoleacetic acid: 5HIAA. A break-down product of serotonin that is excreted in the urine. Serotonin is a hormone found in high levels in many body tissues. Serotonin and 5HIAA are produced in excess amounts by carcinoid tumors, and levels of these substances may be measured in the urine to test for carcinoid tumors. [NIH]

Abdomen: That portion of the body that lies between the thorax and the pelvis. [NIH]

Abdominal: Having to do with the abdomen, which is the part of the body between the chest and the hips that contains the pancreas, stomach, intestines, liver, gallbladder, and other organs. [NIH]

Abdominal Pain: Sensation of discomfort, distress, or agony in the abdominal region. [NIH]

Aberrant: Wandering or deviating from the usual or normal course. [EU]

Ablate: In surgery, is to remove. [NIH]

Ablation: The removal of an organ by surgery. [NIH]

Abortion: 1. The premature expulsion from the uterus of the products of conception - of the embryo, or of a nonviable fetus. The four classic symptoms, usually present in each type of abortion, are uterine contractions, uterine haemorrhage, softening and dilatation of the cervix, and presentation or expulsion of all or part of the products of conception. 2. Premature stoppage of a natural or a pathological process. [EU]

Abscess: Accumulation of purulent material in tissues, organs, or circumscribed spaces, usually associated with signs of infection. [NIH]

Absolute risk: The observed or calculated probability of an event in a population under study, as contrasted with the relative risk. [NIH]

Acceptor: A substance which, while normally not oxidized by oxygen or reduced by hydrogen, can be oxidized or reduced in presence of a substance which is itself undergoing oxidation or reduction. [NIH]

ACE: Angiotensin-coverting enzyme. A drug used to decrease pressure inside blood vessels. [NIH]

Acetylcholine: A neurotransmitter. Acetylcholine in vertebrates is the major transmitter at neuromuscular junctions, autonomic ganglia, parasympathetic effector junctions, a subset of sympathetic effector junctions, and at many sites in the central nervous system. It is generally not used as an administered drug because it is broken down very rapidly by cholinesterases, but it is useful in some ophthalmological applications. [NIH]

Acetylcysteine: The N-acetyl derivative of cysteine. It is used as a mucolytic agent to reduce the viscosity of mucous secretions. It has also been shown to have antiviral effects in patients with HIV due to inhibition of viral stimulation by reactive oxygen intermediates. [NIH]

Acetylgalactosamine: The N-acetyl derivative of galactosamine. [NIH]

Acetylglucosamine: The N-acetyl derivative of glucosamine. [NIH]

Acidity: The quality of being acid or sour; containing acid (hydrogen ions). [EU]

Acoustic: Having to do with sound or hearing. [NIH]

Acrylamide: A colorless, odorless, highly water soluble vinyl monomer formed from the

hydration of acrylonitrile. It is primarily used in research laboratories for electrophoresis, chromatography, and electron microscopy and in the sewage and wastewater treatment industries. [NIH]

Acrylonitrile: A highly poisonous compound used widely in the manufacture of plastics, adhesives and synthetic rubber. [NIH]

Actin: Essential component of the cell skeleton. [NIH]

Activities of Daily Living: The performance of the basic activities of self care, such as dressing, ambulation, eating, etc., in rehabilitation. [NIH]

Acute-Phase Proteins: Proteins that are secreted into the blood in increased or decreased quantities by hepatocytes in response to trauma, inflammation, or disease. These proteins can serve as inhibitors or mediators of the inflammatory processes. Certain acute-phase proteins have been used to diagnose and follow the course of diseases or as tumor markers. [NIH]

Adaptability: Ability to develop some form of tolerance to conditions extremely different from those under which a living organism evolved. [NIH]

Adaptation: 1. The adjustment of an organism to its environment, or the process by which it enhances such fitness. 2. The normal ability of the eye to adjust itself to variations in the intensity of light; the adjustment to such variations. 3. The decline in the frequency of firing of a neuron, particularly of a receptor, under conditions of constant stimulation. 4. In dentistry, (a) the proper fitting of a denture, (b) the degree of proximity and interlocking of restorative material to a tooth preparation, (c) the exact adjustment of bands to teeth. 5. In microbiology, the adjustment of bacterial physiology to a new environment. [EU]

Adenine: A purine base and a fundamental unit of adenine nucleotides. [NIH]

Adenosine: A nucleoside that is composed of adenine and d-ribose. Adenosine or adenosine derivatives play many important biological roles in addition to being components of DNA and RNA. Adenosine itself is a neurotransmitter. [NIH]

Adhesives: Substances that cause the adherence of two surfaces. They include glues (properly collagen-derived adhesives), mucilages, sticky pastes, gums, resins, or latex. [NIH]

Adipose Tissue: Connective tissue composed of fat cells lodged in the meshes of areolar tissue. [NIH]

Adjustment: The dynamic process wherein the thoughts, feelings, behavior, and biophysiological mechanisms of the individual continually change to adjust to the environment. [NIH]

Adolescence: The period of life beginning with the appearance of secondary sex characteristics and terminating with the cessation of somatic growth. The years usually referred to as adolescence lie between 13 and 18 years of age. [NIH]

Adrenal Cortex: The outer layer of the adrenal gland. It secretes mineralocorticoids, androgens, and glucocorticoids. [NIH]

Adrenal Glands: Paired glands situated in the retroperitoneal tissues at the superior pole of each kidney. [NIH]

Adrenal Medulla: The inner part of the adrenal gland; it synthesizes, stores and releases catecholamines. [NIH]

Adrenergic: Activated by, characteristic of, or secreting epinephrine or substances with similar activity; the term is applied to those nerve fibres that liberate norepinephrine at a synapse when a nerve impulse passes, i.e., the sympathetic fibres. [EU]

Adrenergic Agents: Drugs that act on adrenergic receptors or affect the life cycle of

adrenergic transmitters. Included here are adrenergic agonists and antagonists and agents that affect the synthesis, storage, uptake, metabolism, or release of adrenergic transmitters. [NIH]

Adverse Effect: An unwanted side effect of treatment. [NIH]

Aerobic: In biochemistry, reactions that need oxygen to happen or happen when oxygen is present. [NIH]

Aetiology: Study of the causes of disease. [EU]

Afferent: Concerned with the transmission of neural impulse toward the central part of the nervous system. [NIH]

Affinity: 1. Inherent likeness or relationship. 2. A special attraction for a specific element, organ, or structure. 3. Chemical affinity; the force that binds atoms in molecules; the tendency of substances to combine by chemical reaction. 4. The strength of noncovalent chemical binding between two substances as measured by the dissociation constant of the complex. 5. In immunology, a thermodynamic expression of the strength of interaction between a single antigen-binding site and a single antigenic determinant (and thus of the stereochemical compatibility between them), most accurately applied to interactions among simple, uniform antigenic determinants such as haptens. Expressed as the association constant (K litres mole -1), which, owing to the heterogeneity of affinities in a population of antibody molecules of a given specificity, actually represents an average value (mean intrinsic association constant). 6. The reciprocal of the dissociation constant. [EU]

Afterload: The tension produced by the heart muscle after contraction. [EU]

Agar: A complex sulfated polymer of galactose units, extracted from Gelidium cartilagineum, Gracilaria confervoides, and related red algae. It is used as a gel in the preparation of solid culture media for microorganisms, as a bulk laxative, in making emulsions, and as a supporting medium for immunodiffusion and immunoelectrophoresis. [NIH]

Age Groups: Persons classified by age from birth (infant, newborn) to octogenarians and older (aged, 80 and over). [NIH]

Age of Onset: The age or period of life at which a disease or the initial symptoms or manifestations of a disease appear in an individual. [NIH]

Age-Adjusted: Summary measures of rates of morbidity or mortality in a population using statistical procedures to remove the effect of age differences in populations that are being compared. Age is probably the most important and the most common variable in determining the risk of morbidity and mortality. [NIH]

Aged, 80 and Over: A person 80 years of age and older. [NIH]

Agonist: In anatomy, a prime mover. In pharmacology, a drug that has affinity for and stimulates physiologic activity at cell receptors normally stimulated by naturally occurring substances. [EU]

Airway: A device for securing unobstructed passage of air into and out of the lungs during general anesthesia. [NIH]

Albumin: 1. Any protein that is soluble in water and moderately concentrated salt solutions and is coagulable by heat. 2. Serum albumin; the major plasma protein (approximately 60 per cent of the total), which is responsible for much of the plasma colloidal osmotic pressure and serves as a transport protein carrying large organic anions, such as fatty acids, bilirubin, and many drugs, and also carrying certain hormones, such as cortisol and thyroxine, when their specific binding globulins are saturated. Albumin is synthesized in the liver. Low serum levels occur in protein malnutrition, active inflammation and serious hepatic and renal disease. [EU]

Alcohol Drinking: Behaviors associated with the ingesting of alcoholic beverages, including social drinking. [NIH]

Algorithms: A procedure consisting of a sequence of algebraic formulas and/or logical steps to calculate or determine a given task. [NIH]

Alimentary: Pertaining to food or nutritive material, or to the organs of digestion. [EU]

Alkaline: Having the reactions of an alkali. [EU]

Alkaloid: A member of a large group of chemicals that are made by plants and have nitrogen in them. Some alkaloids have been shown to work against cancer. [NIH]

Alleles: Mutually exclusive forms of the same gene, occupying the same locus on homologous chromosomes, and governing the same biochemical and developmental process. [NIH]

Allograft: An organ or tissue transplant between two humans. [NIH]

Alpha Particles: Positively charged particles composed of two protons and two neutrons, i.e., helium nuclei, emitted during disintegration of very heavy isotopes; a beam of alpha particles or an alpha ray has very strong ionizing power, but weak penetrability. [NIH]

Alprenolol: 1-((1-Methylethyl)amino)-3-(2-(2-propenyl)phenoxy)-2-propanol. Adrenergic beta-blocker used as an antihypertensive, anti-anginal, and anti-arrhythmic agent. [NIH]

Alternative medicine: Practices not generally recognized by the medical community as standard or conventional medical approaches and used instead of standard treatments. Alternative medicine includes the taking of dietary supplements, megadose vitamins, and herbal preparations; the drinking of special teas; and practices such as massage therapy, magnet therapy, spiritual healing, and meditation. [NIH]

Amenorrhea: Absence of menstruation. [NIH]

Amino acid: Any organic compound containing an amino (-NH2 and a carboxyl (- COOH) group. The 20 a-amino acids listed in the accompanying table are the amino acids from which proteins are synthesized by formation of peptide bonds during ribosomal translation of messenger RNA; all except glycine, which is not optically active, have the L configuration. Other amino acids occurring in proteins, such as hydroxyproline in collagen, are formed by posttranslational enzymatic modification of amino acids residues in polypeptide chains. There are also several important amino acids, such as the neurotransmitter y-aminobutyric acid, that have no relation to proteins. Abbreviated AA. [EU]

Amino Acid Sequence: The order of amino acids as they occur in a polypeptide chain. This is referred to as the primary structure of proteins. It is of fundamental importance in determining protein conformation. [NIH]

Amiodarone: An antianginal and antiarrhythmic drug. It increases the duration of ventricular and atrial muscle action by inhibiting Na,K-activated myocardial adenosine triphosphatase. There is a resulting decrease in heart rate and in vascular resistance. [NIH]

Ammonia: A colorless alkaline gas. It is formed in the body during decomposition of organic materials during a large number of metabolically important reactions. [NIH]

Amnestic: Nominal aphasia; a difficulty in finding the right name for an object. [NIH]

Amphetamines: Analogs or derivatives of amphetamine. Many are sympathomimetics and central nervous system stimulators causing excitation, vasopression, bronchodilation, and to varying degrees, anorexia, analepsis, nasal decongestion, and some smooth muscle relaxation. [NIH]

Amrinone: A positive inotropic cardiotonic agent with vasodilator properties, phosphodiesterase inhibitory activity, and the ability to stimulate calcium ion influx into the

cardiac cell. Its therapeutic use in congestive heart or left ventricular failure is associated with significant increases in the cardiac index, reductions in pulmonary capillary wedge pressure and systemic vascular resistance, and little or no change in mean arterial pressure. One of its more serious side effects is thrombocytopenia in some patients. [NIH]

Amyloid: A general term for a variety of different proteins that accumulate as extracellular fibrils of 7-10 nm and have common structural features, including a beta-pleated sheet conformation and the ability to bind such dyes as Congo red and thioflavine (Kandel, Schwartz, and Jessel, Principles of Neural Science, 3rd ed). [NIH]

Amyloidosis: A group of diseases in which protein is deposited in specific organs (localized amyloidosis) or throughout the body (systemic amyloidosis). Amyloidosis may be either primary (with no known cause) or secondary (caused by another disease, including some types of cancer). Generally, primary amyloidosis affects the nerves, skin, tongue, joints, heart, and liver; secondary amyloidosis often affects the spleen, kidneys, liver, and adrenal glands. [NIH]

Anaesthesia: Loss of feeling or sensation. Although the term is used for loss of tactile sensibility, or of any of the other senses, it is applied especially to loss of the sensation of pain, as it is induced to permit performance of surgery or other painful procedures. [EU]

Anal: Having to do with the anus, which is the posterior opening of the large bowel. [NIH]

Analysis of Variance: A statistical technique that isolates and assesses the contributions of categorical independent variables to variation in the mean of a continuous dependent variable. [NIH]

Anaphylatoxins: The family of peptides C3a, C4a, C5a, and C5a des-arginine produced in the serum during complement activation. They produce smooth muscle contraction, mast cell histamine release, affect platelet aggregation, and act as mediators of the local inflammatory process. The order of anaphylatoxin activity from strongest to weakest is C5a, C3a, C4a, and C5a des-arginine. The latter is the so-called "classical" anaphylatoxin but shows no spasmogenic activity though it contains some chemotactic ability. [NIH]

Anaplasia: Loss of structural differentiation and useful function of neoplastic cells. [NIH]

Anatomical: Pertaining to anatomy, or to the structure of the organism. [EU]

Anchorage: In dentistry, points of retention of fillings and artificial restorations and appliances. [NIH]

Anemia: A reduction in the number of circulating erythrocytes or in the quantity of hemoglobin. [NIH]

Anesthesia: A state characterized by loss of feeling or sensation. This depression of nerve function is usually the result of pharmacologic action and is induced to allow performance of surgery or other painful procedures. [NIH]

Aneuploidy: The chromosomal constitution of cells which deviate from the normal by the addition or subtraction of chromosomes or chromosome pairs. In a normally diploid cell the loss of a chromosome pair is termed nullisomy (symbol: 2N-2), the loss of a single chromosome is monosomy (symbol: 2N-1), the addition of a chromosome pair is tetrasomy (symbol: 2N+2), the addition of a single chromosome is trisomy (symbol: 2N+1). [NIH]

Aneurysm: A sac formed by the dilatation of the wall of an artery, a vein, or the heart. [NIH]

Angina: Chest pain that originates in the heart. [NIH]

Angina Pectoris: The symptom of paroxysmal pain consequent to myocardial ischemia usually of distinctive character, location and radiation, and provoked by a transient stressful situation during which the oxygen requirements of the myocardium exceed the capacity of the coronary circulation to supply it. [NIH]

Anginal: Pertaining to or characteristic of angina. [EU]

Angiocardiography: Radiography of the heart and great vessels after injection of a contrast medium. [NIH]

Angiogenesis: Blood vessel formation. Tumor angiogenesis is the growth of blood vessels from surrounding tissue to a solid tumor. This is caused by the release of chemicals by the tumor. [NIH]

Angiography: Radiography of blood vessels after injection of a contrast medium. [NIH]

Angioplasty: Endovascular reconstruction of an artery, which may include the removal of atheromatous plaque and/or the endothelial lining as well as simple dilatation. These are procedures performed by catheterization. When reconstruction of an artery is performed surgically, it is called endarterectomy. [NIH]

Angiotensin converting enzyme inhibitor: A drug used to decrease pressure inside blood vessels. [NIH]

Angiotensinogen: An alpha-globulin of which a fragment of 14 amino acids is converted by renin to angiotensin I, the inactive precursor of angiotensin II. It is a member of the serpin superfamily. [NIH]

Animal model: An animal with a disease either the same as or like a disease in humans. Animal models are used to study the development and progression of diseases and to test new treatments before they are given to humans. Animals with transplanted human cancers or other tissues are called xenograft models. [NIH]

Anions: Negatively charged atoms, radicals or groups of atoms which travel to the anode or positive pole during electrolysis. [NIH]

Annealing: The spontaneous alignment of two single DNA strands to form a double helix. [NIH]

Anomalies: Birth defects; abnormalities. [NIH]

Anterior chamber: The space in front of the iris and behind the cornea. [NIH]

Anthropometry: The technique that deals with the measurement of the size, weight, and proportions of the human or other primate body. [NIH]

Antianginal: Counteracting angina or anginal conditions. [EU]

Antiarrhythmic: An agent that prevents or alleviates cardiac arrhythmia. [EU]

Antibacterial: A substance that destroys bacteria or suppresses their growth or reproduction. [EU]

Antibiotic: A drug used to treat infections caused by bacteria and other microorganisms. [NIH]

Antibiotic Prophylaxis: Use of antibiotics before, during, or after a diagnostic, therapeutic, or surgical procedure to prevent infectious complications. [NIH]

Antibodies: Immunoglobulin molecules having a specific amino acid sequence by virtue of which they interact only with the antigen that induced their synthesis in cells of the lymphoid series (especially plasma cells), or with an antigen closely related to it. [NIH]

Antibody: A type of protein made by certain white blood cells in response to a foreign substance (antigen). Each antibody can bind to only a specific antigen. The purpose of this binding is to help destroy the antigen. Antibodies can work in several ways, depending on the nature of the antigen. Some antibodies destroy antigens directly. Others make it easier for white blood cells to destroy the antigen. [NIH]

Anticoagulant: A drug that helps prevent blood clots from forming. Also called a blood thinner. [NIH]

Anticonvulsant: An agent that prevents or relieves convulsions. [EU]

Antigen: Any substance which is capable, under appropriate conditions, of inducing a specific immune response and of reacting with the products of that response, that is, with specific antibody or specifically sensitized T-lymphocytes, or both. Antigens may be soluble substances, such as toxins and foreign proteins, or particulate, such as bacteria and tissue cells; however, only the portion of the protein or polysaccharide molecule known as the antigenic determinant (q.v.) combines with antibody or a specific receptor on a lymphocyte. Abbreviated Ag. [EU]

Antigen-Antibody Complex: The complex formed by the binding of antigen and antibody molecules. The deposition of large antigen-antibody complexes leading to tissue damage causes immune complex diseases. [NIH]

Antihypertensive: An agent that reduces high blood pressure. [EU]

Anti-infective: An agent that so acts. [EU]

Anti-inflammatory: Having to do with reducing inflammation. [NIH]

Anti-Inflammatory Agents: Substances that reduce or suppress inflammation. [NIH]

Antineoplastic: Inhibiting or preventing the development of neoplasms, checking the maturation and proliferation of malignant cells. [EU]

Antioxidant: A substance that prevents damage caused by free radicals. Free radicals are highly reactive chemicals that often contain oxygen. They are produced when molecules are split to give products that have unpaired electrons. This process is called oxidation. [NIH]

Antithrombotic: Preventing or interfering with the formation of thrombi; an agent that so acts. [EU]

Antiviral: Destroying viruses or suppressing their replication. [EU]

Anuria: Inability to form or excrete urine. [NIH]

Anus: The opening of the rectum to the outside of the body. [NIH]

Anxiety: Persistent feeling of dread, apprehension, and impending disaster. [NIH]

Anxiety Disorders: Disorders in which anxiety (persistent feelings of apprehension, tension, or uneasiness) is the predominant disturbance. [NIH]

Anxiolytic: An anxiolytic or antianxiety agent. [EU]

Aorta: The main trunk of the systemic arteries. [NIH]

Aortic Valve: The valve between the left ventricle and the ascending aorta which prevents backflow into the left ventricle. [NIH]

Apheresis: Components being separated out, as leukapheresis, plasmapheresis, plateletpheresis. [NIH]

Apnea: A transient absence of spontaneous respiration. [NIH]

Apolipoproteins: The protein components of lipoproteins which remain after the lipids to which the proteins are bound have been removed. They play an important role in lipid transport and metabolism. [NIH]

Apoptosis: One of the two mechanisms by which cell death occurs (the other being the pathological process of necrosis). Apoptosis is the mechanism responsible for the physiological deletion of cells and appears to be intrinsically programmed. It is characterized by distinctive morphologic changes in the nucleus and cytoplasm, chromatin cleavage at regularly spaced sites, and the endonucleolytic cleavage of genomic DNA (DNA fragmentation) at internucleosomal sites. This mode of cell death serves as a balance to mitosis in regulating the size of animal tissues and in mediating pathologic processes

associated with tumor growth. [NIH]

Applicability: A list of the commodities to which the candidate method can be applied as presented or with minor modifications. [NIH]

Apraxia: Loss of ability to perform purposeful movements, in the absence of paralysis or sensory disturbance, caused by lesions in the cortex. [NIH]

Aqueous: Having to do with water. [NIH]

Arachidonic Acid: An unsaturated, essential fatty acid. It is found in animal and human fat as well as in the liver, brain, and glandular organs, and is a constituent of animal phosphatides. It is formed by the synthesis from dietary linoleic acid and is a precursor in the biosynthesis of prostaglandins, thromboxanes, and leukotrienes. [NIH]

Arginine: An essential amino acid that is physiologically active in the L-form. [NIH]

Aromatic: Having a spicy odour. [EU]

Arrhythmia: Any variation from the normal rhythm or rate of the heart beat. [NIH]

Arrhythmogenic: Producing or promoting arrhythmia. [EU]

Arterial: Pertaining to an artery or to the arteries. [EU]

Arteries: The vessels carrying blood away from the heart. [NIH]

Arteriography: A procedure to x-ray arteries. The arteries can be seen because of an injection of a dye that outlines the vessels on an x-ray. [NIH]

Arteriolar: Pertaining to or resembling arterioles. [EU]

Arterioles: The smallest divisions of the arteries located between the muscular arteries and the capillaries. [NIH]

Arteriosclerosis: Thickening and loss of elasticity of arterial walls. Atherosclerosis is the most common form of arteriosclerosis and involves lipid deposition and thickening of the intimal cell layers within arteries. Additional forms of arteriosclerosis involve calcification of the media of muscular arteries (Monkeberg medial calcific sclerosis) and thickening of the walls of small arteries or arterioles due to cell proliferation or hyaline deposition (arteriolosclerosis). [NIH]

Arteriosus: Circle composed of anastomosing arteries derived from two long posterior ciliary and seven anterior ciliary arteries, located in the ciliary body about the root of the iris. [NIH]

Arteriovenous: Both arterial and venous; pertaining to or affecting an artery and a vein. [EU]

Artery: Vessel-carrying blood from the heart to various parts of the body. [NIH]

Articular: Of or pertaining to a joint. [EU]

Ascorbic Acid: A six carbon compound related to glucose. It is found naturally in citrus fruits and many vegetables. Ascorbic acid is an essential nutrient in human diets, and necessary to maintain connective tissue and bone. Its biologically active form, vitamin C, functions as a reducing agent and coenzyme in several metabolic pathways. Vitamin C is considered an antioxidant. [NIH]

Aspiration: The act of inhaling. [NIH]

Aspirin: A drug that reduces pain, fever, inflammation, and blood clotting. Aspirin belongs to the family of drugs called nonsteroidal anti-inflammatory agents. It is also being studied in cancer prevention. [NIH]

Assay: Determination of the amount of a particular constituent of a mixture, or of the biological or pharmacological potency of a drug. [EU]

Astringents: Agents, usually topical, that cause the contraction of tissues for the control of bleeding or secretions. [NIH]

Asymptomatic: Having no signs or symptoms of disease. [NIH]

Ataxia: Impairment of the ability to perform smoothly coordinated voluntary movements. This condition may affect the limbs, trunk, eyes, pharnyx, larnyx, and other structures. Ataxia may result from impaired sensory or motor function. Sensory ataxia may result from posterior column injury or peripheral nerve diseases. Motor ataxia may be associated with cerebellar diseases; cerebral cortex diseases; thalamic diseases; basal ganglia diseases; injury to the red nucleus; and other conditions. [NIH]

Atenolol: A cardioselective beta-adrenergic blocker possessing properties and potency similar to propranolol, but without a negative inotropic effect. [NIH]

ATP: ATP an abbreviation for adenosine triphosphate, a compound which serves as a carrier of energy for cells. [NIH]

Atresia: Lack of a normal opening from the esophagus, intestines, or anus. [NIH]

Atrial: Pertaining to an atrium. [EU]

Atrial Fibrillation: Disorder of cardiac rhythm characterized by rapid, irregular atrial impulses and ineffective atrial contractions. [NIH]

Atrial Flutter: Rapid, irregular atrial contractions due to an abnormality of atrial excitation. [NIH]

Atrioventricular: Pertaining to an atrium of the heart and to a ventricle. [EU]

Atrium: A chamber; used in anatomical nomenclature to designate a chamber affording entrance to another structure or organ. Usually used alone to designate an atrium of the heart. [EU]

Atrophy: Decrease in the size of a cell, tissue, organ, or multiple organs, associated with a variety of pathological conditions such as abnormal cellular changes, ischemia, malnutrition, or hormonal changes. [NIH]

Atypical: Irregular; not conformable to the type; in microbiology, applied specifically to strains of unusual type. [EU]

Audiovisual Aids: Auditory and visual instructional materials. [NIH]

Auscultation: Act of listening for sounds within the body. [NIH]

Autoantibodies: Antibodies that react with self-antigens (autoantigens) of the organism that produced them. [NIH]

Autoantigens: Endogenous tissue constituents that have the ability to interact with autoantibodies and cause an immune response. [NIH]

Autoimmune disease: A condition in which the body recognizes its own tissues as foreign and directs an immune response against them. [NIH]

Autoimmunity: Process whereby the immune system reacts against the body's own tissues. Autoimmunity may produce or be caused by autoimmune diseases. [NIH]

Autologous: Taken from an individual's own tissues, cells, or DNA. [NIH]

Autonomic: Self-controlling; functionally independent. [EU]

Autonomic Nervous System: The enteric, parasympathetic, and sympathetic nervous systems taken together. Generally speaking, the autonomic nervous system regulates the internal environment during both peaceful activity and physical or emotional stress. Autonomic activity is controlled and integrated by the central nervous system, especially the hypothalamus and the solitary nucleus, which receive information relayed from visceral

afferents; these and related central and sensory structures are sometimes (but not here) considered to be part of the autonomic nervous system itself. [NIH]

Autonomic Neuropathy: A disease of the nerves affecting mostly the internal organs such as the bladder muscles, the cardiovascular system, the digestive tract, and the genital organs. These nerves are not under a person's conscious control and function automatically. Also called visceral neuropathy. [NIH]

Autopsy: Postmortem examination of the body. [NIH]

Axillary: Pertaining to the armpit area, including the lymph nodes that are located there. [NIH]

Axillary Artery: The continuation of the subclavian artery; it distributes over the upper limb, axilla, chest and shoulder. [NIH]

Azithromycin: A semi-synthetic macrolide antibiotic structurally related to erythromycin. It has been used in the treatment of Mycobacterium avium intracellulare infections, toxoplasmosis, and cryptosporidiosis. [NIH]

Bacteria: Unicellular prokaryotic microorganisms which generally possess rigid cell walls, multiply by cell division, and exhibit three principal forms: round or coccal, rodlike or bacillary, and spiral or spirochetal. [NIH]

Bacterial Infections: Infections by bacteria, general or unspecified. [NIH]

Bacterial Physiology: Physiological processes and activities of bacteria. [NIH]

Bactericidal: Substance lethal to bacteria; substance capable of killing bacteria. [NIH]

Bacteriophage: A virus whose host is a bacterial cell; A virus that exclusively infects bacteria. It generally has a protein coat surrounding the genome (DNA or RNA). One of the coliphages most extensively studied is the lambda phage, which is also one of the most important. [NIH]

Bacteriuria: The presence of bacteria in the urine with or without consequent urinary tract infection. Since bacteriuria is a clinical entity, the term does not preclude the use of urine/microbiology for technical discussions on the isolation and segregation of bacteria in the urine. [NIH]

Baroreflex: A negative feedback system which buffers short-term changes in blood pressure. Increased pressure stretches blood vessels which activates pressoreceptors (baroreceptors) in the vessel walls. The net response of the central nervous system is a reduction of central sympathetic outflow. This reduces blood pressure both by decreasing peripheral vascular resistance and by lowering cardiac output. Because the baroreceptors are tonically active, the baroreflex can compensate rapidly for both increases and decreases in blood pressure. [NIH]

Basal Ganglia: Large subcortical nuclear masses derived from the telencephalon and located in the basal regions of the cerebral hemispheres. [NIH]

Basal Ganglia Diseases: Diseases of the basal ganglia including the putamen; globus pallidus; claustrum; amygdala; and caudate nucleus. Dyskinesias (most notably involuntary movements and alterations of the rate of movement) represent the primary clinical manifestations of these disorders. Common etiologies include cerebrovascular disease; neurodegenerative diseases; and craniocerebral trauma. [NIH]

Base: In chemistry, the nonacid part of a salt; a substance that combines with acids to form salts; a substance that dissociates to give hydroxide ions in aqueous solutions; a substance whose molecule or ion can combine with a proton (hydrogen ion); a substance capable of donating a pair of electrons (to an acid) for the formation of a coordinate covalent bond. [EU]

Basement Membrane: Ubiquitous supportive tissue adjacent to epithelium and around

smooth and striated muscle cells. This tissue contains intrinsic macromolecular components such as collagen, laminin, and sulfated proteoglycans. As seen by light microscopy one of its subdivisions is the basal (basement) lamina. [NIH]

Basophils: Granular leukocytes characterized by a relatively pale-staining, lobate nucleus and cytoplasm containing coarse dark-staining granules of variable size and stainable by basic dyes. [NIH]

Benham: A disk, half black and half white, with a number of concentric black arcs on the white sector, which, when rotated, elicits a variety of chromatic color sensations. [NIH]

Benign: Not cancerous; does not invade nearby tissue or spread to other parts of the body. [NIH]

Benign prostatic hyperplasia: A benign (noncancerous) condition in which an overgrowth of prostate tissue pushes against the urethra and the bladder, blocking the flow of urine. Also called benign prostatic hypertrophy or BPH. [NIH]

Benzene: Toxic, volatile, flammable liquid hydrocarbon biproduct of coal distillation. It is used as an industrial solvent in paints, varnishes, lacquer thinners, gasoline, etc. Benzene causes central nervous system damage acutely and bone marrow damage chronically and is carcinogenic. It was formerly used as parasiticide. [NIH]

Bereavement: Refers to the whole process of grieving and mourning and is associated with a deep sense of loss and sadness. [NIH]

Beta blocker: A drug used to slow the heart rate and reduce pressure inside blood vessels. It also can regulate heart rhythm. [NIH]

Beta-pleated: Particular three-dimensional pattern of amyloidoses. [NIH]

Beta-Thromboglobulin: A platelet-specific protein which is released when platelets aggregate. Elevated plasma levels have been reported after deep venous thrombosis, pre-eclampsia, myocardial infarction with mural thrombosis, and myeloproliferative disorders. Measurement of beta-thromboglobulin in biological fluids by radioimmunoassay is used for the diagnosis and assessment of progress of thromboembolic disorders. [NIH]

Bifida: A defect in development of the vertebral column in which there is a central deficiency of the vertebral lamina. [NIH]

Bilateral: Affecting both the right and left side of body. [NIH]

Bile: An emulsifying agent produced in the liver and secreted into the duodenum. Its composition includes bile acids and salts, cholesterol, and electrolytes. It aids digestion of fats in the duodenum. [NIH]

Bilirubin: A bile pigment that is a degradation product of heme. [NIH]

Binding Sites: The reactive parts of a macromolecule that directly participate in its specific combination with another molecule. [NIH]

Biochemical: Relating to biochemistry; characterized by, produced by, or involving chemical reactions in living organisms. [EU]

Bioengineering: The application of engineering principles to the solution of biological problems, for example, remote-handling devices, life-support systems, controls, and displays. [NIH]

Biological therapy: Treatment to stimulate or restore the ability of the immune system to fight infection and disease. Also used to lessen side effects that may be caused by some cancer treatments. Also known as immunotherapy, biotherapy, or biological response modifier (BRM) therapy. [NIH]

Biomarkers: Substances sometimes found in an increased amount in the blood, other body

fluids, or tissues and that may suggest the presence of some types of cancer. Biomarkers include CA 125 (ovarian cancer), CA 15-3 (breast cancer), CEA (ovarian, lung, breast, pancreas, and GI tract cancers), and PSA (prostate cancer). Also called tumor markers. [NIH]

Biopolymers: Polymers, such as proteins, DNA, RNA, or polysaccharides formed by any living organism. [NIH]

Bioprosthesis: Prosthesis, usually heart valve, composed of biological material and whose durability depends upon the stability of the material after pretreatment, rather than regeneration by host cell ingrowth. Durability is achieved 1) mechanically by the interposition of a cloth, usually polytetrafluoroethylene, between the host and the graft, and 2) chemically by stabilization of the tissue by intermolecular linking, usually with glutaraldehyde, after removal of antigenic components, or the use of reconstituted and restructured biopolymers. [NIH]

Biopsy: Removal and pathologic examination of specimens in the form of small pieces of tissue from the living body. [NIH]

Bioreactors: Tools or devices for generating products using the synthetic or chemical conversion capacity of a biological system. They can be classical fermentors, cell culture perfusion systems, or enzyme bioreactors. For production of proteins or enzymes, recombinant microorganisms such as bacteria, mammalian cells, or insect or plant cells are usually chosen. [NIH]

Biotechnology: Body of knowledge related to the use of organisms, cells or cell-derived constituents for the purpose of developing products which are technically, scientifically and clinically useful. Alteration of biologic function at the molecular level (i.e., genetic engineering) is a central focus; laboratory methods used include transfection and cloning technologies, sequence and structure analysis algorithms, computer databases, and gene and protein structure function analysis and prediction. [NIH]

Biotin: Hexahydro-2-oxo-1H-thieno(3,4-d)imidazole-4-pentanoic acid. Growth factor present in minute amounts in every living cell. It occurs mainly bound to proteins or polypeptides and is abundant in liver, kidney, pancreas, yeast, and milk.The biotin content of cancerous tissue is higher than that of normal tissue. [NIH]

Biotransformation: The chemical alteration of an exogenous substance by or in a biological system. The alteration may inactivate the compound or it may result in the production of an active metabolite of an inactive parent compound. The alteration may be either non-synthetic (oxidation-reduction, hydrolysis) or synthetic (glucuronide formation, sulfate conjugation, acetylation, methylation). This also includes metabolic detoxication and clearance. [NIH]

Birth Order: The sequence in which children are born into the family. [NIH]

Bladder: The organ that stores urine. [NIH]

Bloating: Fullness or swelling in the abdomen that often occurs after meals. [NIH]

Blood Cell Count: A count of the number of leukocytes and erythrocytes per unit volume in a sample of venous blood. A complete blood count (CBC) also includes measurement of the hemoglobin, hematocrit, and erythrocyte indices. [NIH]

Blood Coagulation: The process of the interaction of blood coagulation factors that results in an insoluble fibrin clot. [NIH]

Blood Glucose: Glucose in blood. [NIH]

Blood Platelets: Non-nucleated disk-shaped cells formed in the megakaryocyte and found in the blood of all mammals. They are mainly involved in blood coagulation. [NIH]

Blood pressure: The pressure of blood against the walls of a blood vessel or heart chamber.

Unless there is reference to another location, such as the pulmonary artery or one of the heart chambers, it refers to the pressure in the systemic arteries, as measured, for example, in the forearm. [NIH]

Blood vessel: A tube in the body through which blood circulates. Blood vessels include a network of arteries, arterioles, capillaries, venules, and veins. [NIH]

Blood Viscosity: The internal resistance of the blood to shear forces. The in vitro measure of whole blood viscosity is of limited clinical utility because it bears little relationship to the actual viscosity within the circulation, but an increase in the viscosity of circulating blood can contribute to morbidity in patients suffering from disorders such as sickle cell anemia and polycythemia. [NIH]

Blood Volume: Volume of circulating blood. It is the sum of the plasma volume and erythrocyte volume. [NIH]

Blot: To transfer DNA, RNA, or proteins to an immobilizing matrix such as nitrocellulose. [NIH]

Body Composition: The relative amounts of various components in the body, such as percent body fat. [NIH]

Body Fluids: Liquid components of living organisms. [NIH]

Body Mass Index: One of the anthropometric measures of body mass; it has the highest correlation with skinfold thickness or body density. [NIH]

Bone Density: The amount of mineral per square centimeter of bone. This is the definition used in clinical practice. Actual bone density would be expressed in grams per milliliter. It is most frequently measured by photon absorptiometry or x-ray computed tomography. [NIH]

Bone Marrow: The soft tissue filling the cavities of bones. Bone marrow exists in two types, yellow and red. Yellow marrow is found in the large cavities of large bones and consists mostly of fat cells and a few primitive blood cells. Red marrow is a hematopoietic tissue and is the site of production of erythrocytes and granular leukocytes. Bone marrow is made up of a framework of connective tissue containing branching fibers with the frame being filled with marrow cells. [NIH]

Bone Marrow Cells: Cells contained in the bone marrow including fat cells, stromal cells, megakaryocytes, and the immediate precursors of most blood cells. [NIH]

Bone scan: A technique to create images of bones on a computer screen or on film. A small amount of radioactive material is injected into a blood vessel and travels through the bloodstream; it collects in the bones and is detected by a scanner. [NIH]

Bowel: The long tube-shaped organ in the abdomen that completes the process of digestion. There is both a small and a large bowel. Also called the intestine. [NIH]

Bowel Movement: Body wastes passed through the rectum and anus. [NIH]

Brachial: All the nerves from the arm are ripped from the spinal cord. [NIH]

Brachial Artery: The continuation of the axillary artery; it branches into the radial and ulnar arteries. [NIH]

Brachiocephalic Veins: Large veins on either side of the root of the neck formed by the junction of the internal jugular and subclavian veins. They drain blood from the head, neck, and upper extremities, and unite to form the superior vena cava. [NIH]

Bradykinin: A nonapeptide messenger that is enzymatically produced from kallidin in the blood where it is a potent but short-lived agent of arteriolar dilation and increased capillary permeability. Bradykinin is also released from mast cells during asthma attacks, from gut walls as a gastrointestinal vasodilator, from damaged tissues as a pain signal, and may be a

neurotransmitter. [NIH]

Branch: Most commonly used for branches of nerves, but applied also to other structures. [NIH]

Breakdown: A physical, metal, or nervous collapse. [NIH]

Breeding: The science or art of changing the constitution of a population of plants or animals through sexual reproduction. [NIH]

Bromine: A halogen with the atomic symbol Br, atomic number 36, and atomic weight 79.904. It is a volatile reddish-brown liquid that gives off suffocating vapors, is corrosive to the skin, and may cause severe gastroenteritis if ingested. [NIH]

Bromocriptine: A semisynthetic ergot alkaloid that is a dopamine D2 agonist. It suppresses prolactin secretion and is used to treat amenorrhea, galactorrhea, and female infertility, and has been proposed for Parkinson disease. [NIH]

Bronchi: The larger air passages of the lungs arising from the terminal bifurcation of the trachea. [NIH]

Bronchial: Pertaining to one or more bronchi. [EU]

Bronchitis: Inflammation (swelling and reddening) of the bronchi. [NIH]

Bronchoscopy: Endoscopic examination, therapy or surgery of the bronchi. [NIH]

Bronchus: A large air passage that leads from the trachea (windpipe) to the lung. [NIH]

Buccal: Pertaining to or directed toward the cheek. In dental anatomy, used to refer to the buccal surface of a tooth. [EU]

Buffers: A chemical system that functions to control the levels of specific ions in solution. When the level of hydrogen ion in solution is controlled the system is called a pH buffer. [NIH]

Bypass: A surgical procedure in which the doctor creates a new pathway for the flow of body fluids. [NIH]

Calcification: Deposits of calcium in the tissues of the breast. Calcification in the breast can be seen on a mammogram, but cannot be detected by touch. There are two types of breast calcification, macrocalcification and microcalcification. Macrocalcifications are large deposits and are usually not related to cancer. Microcalcifications are specks of calcium that may be found in an area of rapidly dividing cells. Many microcalcifications clustered together may be a sign of cancer. [NIH]

Calcineurin: A calcium- and calmodulin-binding protein present in highest concentrations in the central nervous system. Calcineurin is composed of two subunits. A catalytic subunit, calcineurin A, and a regulatory subunit, calcineurin B, with molecular weights of about 60 kD and 19 kD, respectively. Calcineurin has been shown to dephosphorylate a number of phosphoproteins including histones, myosin light chain, and the regulatory subunit of cAMP-dependent protein kinase. It is involved in the regulation of signal transduction and is the target of an important class of immunophilin-immunosuppressive drug complexes in T-lymphocytes that act by inhibiting T-cell activation. EC 3.1.3.-. [NIH]

Calcium: A basic element found in nearly all organized tissues. It is a member of the alkaline earth family of metals with the atomic symbol Ca, atomic number 20, and atomic weight 40. Calcium is the most abundant mineral in the body and combines with phosphorus to form calcium phosphate in the bones and teeth. It is essential for the normal functioning of nerves and muscles and plays a role in blood coagulation (as factor IV) and in many enzymatic processes. [NIH]

Calcium channel blocker: A drug used to relax the blood vessel and heart muscle, causing pressure inside blood vessels to drop. It also can regulate heart rhythm. [NIH]

Calcium Channel Blockers: A class of drugs that act by selective inhibition of calcium influx through cell membranes or on the release and binding of calcium in intracellular pools. Since they are inducers of vascular and other smooth muscle relaxation, they are used in the drug therapy of hypertension and cerebrovascular spasms, as myocardial protective agents, and in the relaxation of uterine spasms. [NIH]

Callus: A callosity or hard, thick skin; the bone-like reparative substance that is formed round the edges and fragments of broken bone. [NIH]

Calmodulin: A heat-stable, low-molecular-weight activator protein found mainly in the brain and heart. The binding of calcium ions to this protein allows this protein to bind to cyclic nucleotide phosphodiesterases and to adenyl cyclase with subsequent activation. Thereby this protein modulates cyclic AMP and cyclic GMP levels. [NIH]

Calsequestrin: Acidic protein found in sarcoplasmic reticulum that binds calcium to the extent of 700-900 nmoles/mg. It plays the role of sequestering calcium transported to the interior of the intracellular vesicle. [NIH]

Canonical: A particular nucleotide sequence in which each position represents the base more often found when many actual sequences of a given class of genetic elements are compared. [NIH]

Capillary: Any one of the minute vessels that connect the arterioles and venules, forming a network in nearly all parts of the body. Their walls act as semipermeable membranes for the interchange of various substances, including fluids, between the blood and tissue fluid; called also vas capillare. [EU]

Capillary Resistance: The resistance offered to the flow of blood through the capillary portion of the peripheral vascular bed. [NIH]

Carbohydrate: An aldehyde or ketone derivative of a polyhydric alcohol, particularly of the pentahydric and hexahydric alcohols. They are so named because the hydrogen and oxygen are usually in the proportion to form water, (CH2O)n. The most important carbohydrates are the starches, sugars, celluloses, and gums. They are classified into mono-, di-, tri-, poly- and heterosaccharides. [EU]

Carbon Dioxide: A colorless, odorless gas that can be formed by the body and is necessary for the respiration cycle of plants and animals. [NIH]

Carcinogenic: Producing carcinoma. [EU]

Carcinogens: Substances that increase the risk of neoplasms in humans or animals. Both genotoxic chemicals, which affect DNA directly, and nongenotoxic chemicals, which induce neoplasms by other mechanism, are included. [NIH]

Carcinoid: A type of tumor usually found in the gastrointestinal system (most often in the appendix), and sometimes in the lungs or other sites. Carcinoid tumors are usually benign. [NIH]

Carcinoma: Cancer that begins in the skin or in tissues that line or cover internal organs. [NIH]

Cardiac: Having to do with the heart. [NIH]

Cardiac arrest: A sudden stop of heart function. [NIH]

Cardiac catheterization: A procedure in which a thin, hollow tube is inserted into a blood vessel. The tube is then advanced through the vessel into the heart, enabling a physician to study the heart and its pumping activity. [NIH]

Cardiac Glycosides: Substances obtained from species of Digitalis, Strophanthus, and other plants that contain specific steroid glycosides or their semisynthetic derivatives and used in congestive heart failure. They increase the force of cardiac contraction without significantly

affecting other parameters, but are very toxic at larger doses. Their mechanism of action usually involves inhibition of the Na(+)-K(+)-exchanging ATPase and they are often used in cell biological studies for that purpose. [NIH]

Cardiac Output: The volume of blood passing through the heart per unit of time. It is usually expressed as liters (volume) per minute so as not to be confused with stroke volume (volume per beat). [NIH]

Cardiogenic: Originating in the heart; caused by abnormal function of the heart. [EU]

Cardiology: The study of the heart, its physiology, and its functions. [NIH]

Cardiomegaly: Hypertrophy or enlargement of the heart. [NIH]

Cardiomyopathy: A general diagnostic term designating primary myocardial disease, often of obscure or unknown etiology. [EU]

Cardiomyoplasty: A surgical procedure that involves detaching one end of a back muscle and attaching it to the heart. An electric stimulator causes the muscle to contract to pump blood from the heart. [NIH]

Cardiopathy: Any disorder or disease of the heart. In addition to heart disease of inflammatory origin, there are arteriosclerotic cardiopathy, due to arteriosclerosis; fatty cardiopathy, due to growth of fatty tissue; hypertensive cardiopathy, due to high blood pressure; nephropathic cardiopathy, due to kidney disease, thyrotoxic cardiopathy, due to thyroid intoxication; toxic cardiopathy, due to the effect of some toxin; and valvular cardiopathy, due to faulty valve action. [EU]

Cardiopulmonary: Having to do with the heart and lungs. [NIH]

Cardiopulmonary Bypass: Diversion of the flow of blood from the entrance of the right atrium directly to the aorta (or femoral artery) via an oxygenator thus bypassing both the heart and lungs. [NIH]

Cardiopulmonary Resuscitation: The artificial substitution of heart and lung action as indicated for heart arrest resulting from electric shock, drowning, respiratory arrest, or other causes. The two major components of cardiopulmonary resuscitation are artificial ventilation and closed-chest cardiac massage. [NIH]

Cardiorespiratory: Relating to the heart and lungs and their function. [EU]

Cardioselective: Having greater activity on heart tissue than on other tissue. [EU]

Cardiotonic: 1. Having a tonic effect on the heart. 2. An agent that has a tonic effect on the heart. [EU]

Cardiotoxic: Having a poisonous or deleterious effect upon the heart. [EU]

Cardiotoxicity: Toxicity that affects the heart. [NIH]

Cardiovascular: Having to do with the heart and blood vessels. [NIH]

Cardiovascular disease: Any abnormal condition characterized by dysfunction of the heart and blood vessels. CVD includes atherosclerosis (especially coronary heart disease, which can lead to heart attacks), cerebrovascular disease (e.g., stroke), and hypertension (high blood pressure). [NIH]

Cardiovascular System: The heart and the blood vessels by which blood is pumped and circulated through the body. [NIH]

Cardioversion: Electrical reversion of cardiac arrhythmias to normal sinus rhythm, formerly using alternatic current, but now employing direct current. [NIH]

Carotene: The general name for a group of pigments found in green, yellow, and leafy vegetables, and yellow fruits. The pigments are fat-soluble, unsaturated aliphatic

hydrocarbons functioning as provitamins and are converted to vitamin A through enzymatic processes in the intestinal wall. [NIH]

Carrier Proteins: Transport proteins that carry specific substances in the blood or across cell membranes. [NIH]

Case report: A detailed report of the diagnosis, treatment, and follow-up of an individual patient. Case reports also contain some demographic information about the patient (for example, age, gender, ethnic origin). [NIH]

Caspase: Enzyme released by the cell at a crucial stage in apoptosis in order to shred all cellular proteins. [NIH]

Catabolism: Any destructive metabolic process by which organisms convert substances into excreted compounds. [EU]

Cataracts: In medicine, an opacity of the crystalline lens of the eye obstructing partially or totally its transmission of light. [NIH]

Catecholamine: A group of chemical substances manufactured by the adrenal medulla and secreted during physiological stress. [NIH]

Catheter: A flexible tube used to deliver fluids into or withdraw fluids from the body. [NIH]

Catheter Ablation: Removal of tissue with electrical current delivered via electrodes positioned at the distal end of a catheter. Energy sources are commonly direct current (DC-shock) or alternating current at radiofrequencies (usually 750 kHz). The technique is used most often to ablate the AV junction and/or accessory pathways in order to interrupt AV conduction and produce AV block in the treatment of various tachyarrhythmias. [NIH]

Catheterization: Use or insertion of a tubular device into a duct, blood vessel, hollow organ, or body cavity for injecting or withdrawing fluids for diagnostic or therapeutic purposes. It differs from intubation in that the tube here is used to restore or maintain patency in obstructions. [NIH]

Cations: Postively charged atoms, radicals or groups of atoms which travel to the cathode or negative pole during electrolysis. [NIH]

Causal: Pertaining to a cause; directed against a cause. [EU]

Cause of Death: Factors which produce cessation of all vital bodily functions. They can be analyzed from an epidemiologic viewpoint. [NIH]

Cell: The individual unit that makes up all of the tissues of the body. All living things are made up of one or more cells. [NIH]

Cell Adhesion: Adherence of cells to surfaces or to other cells. [NIH]

Cell Adhesion Molecules: Surface ligands, usually glycoproteins, that mediate cell-to-cell adhesion. Their functions include the assembly and interconnection of various vertebrate systems, as well as maintenance of tissue integration, wound healing, morphogenic movements, cellular migrations, and metastasis. [NIH]

Cell Death: The termination of the cell's ability to carry out vital functions such as metabolism, growth, reproduction, responsiveness, and adaptability. [NIH]

Cell Division: The fission of a cell. [NIH]

Cell Fusion: Fusion of somatic cells in vitro or in vivo, which results in somatic cell hybridization. [NIH]

Cell membrane: Cell membrane = plasma membrane. The structure enveloping a cell, enclosing the cytoplasm, and forming a selective permeability barrier; it consists of lipids, proteins, and some carbohydrates, the lipids thought to form a bilayer in which integral proteins are embedded to varying degrees. [EU]

Cell motility: The ability of a cell to move. [NIH]

Cell Respiration: The metabolic process of all living cells (animal and plant) in which oxygen is used to provide a source of energy for the cell. [NIH]

Cell Size: The physical dimensions of a cell. It refers mainly to changes in dimensions correlated with physiological or pathological changes in cells. [NIH]

Cell Survival: The span of viability of a cell characterized by the capacity to perform certain functions such as metabolism, growth, reproduction, some form of responsiveness, and adaptability. [NIH]

Cellulose: A polysaccharide with glucose units linked as in cellobiose. It is the chief constituent of plant fibers, cotton being the purest natural form of the substance. As a raw material, it forms the basis for many derivatives used in chromatography, ion exchange materials, explosives manufacturing, and pharmaceutical preparations. [NIH]

Central Nervous System: The main information-processing organs of the nervous system, consisting of the brain, spinal cord, and meninges. [NIH]

Centrifugation: A method of separating organelles or large molecules that relies upon differential sedimentation through a preformed density gradient under the influence of a gravitational field generated in a centrifuge. [NIH]

Cerebellar: Pertaining to the cerebellum. [EU]

Cerebral: Of or pertaining of the cerebrum or the brain. [EU]

Cerebral Cortex: The thin layer of gray matter on the surface of the cerebral hemisphere that develops from the telencephalon and folds into gyri. It reaches its highest development in man and is responsible for intellectual faculties and higher mental functions. [NIH]

Cerebrospinal: Pertaining to the brain and spinal cord. [EU]

Cerebrospinal fluid: CSF. The fluid flowing around the brain and spinal cord. Cerebrospinal fluid is produced in the ventricles in the brain. [NIH]

Cerebrovascular: Pertaining to the blood vessels of the cerebrum, or brain. [EU]

Cerebrum: The largest part of the brain. It is divided into two hemispheres, or halves, called the cerebral hemispheres. The cerebrum controls muscle functions of the body and also controls speech, emotions, reading, writing, and learning. [NIH]

Cervical: Relating to the neck, or to the neck of any organ or structure. Cervical lymph nodes are located in the neck; cervical cancer refers to cancer of the uterine cervix, which is the lower, narrow end (the "neck") of the uterus. [NIH]

Cervix: The lower, narrow end of the uterus that forms a canal between the uterus and vagina. [NIH]

Chaos: Complex behavior that seems random but actually has some hidden order. [NIH]

Character: In current usage, approximately equivalent to personality. The sum of the relatively fixed personality traits and habitual modes of response of an individual. [NIH]

Chelation: Combination with a metal in complexes in which the metal is part of a ring. [EU]

Chelation Therapy: Therapy of heavy metal poisoning using agents which sequester the metal from organs or tissues and bind it firmly within the ring structure of a new compound which can be eliminated from the body. [NIH]

Chemotactic Factors: Chemical substances that attract or repel cells or organisms. The concept denotes especially those factors released as a result of tissue injury, invasion, or immunologic activity, that attract leukocytes, macrophages, or other cells to the site of infection or insult. [NIH]

Chest Pain: Pressure, burning, or numbness in the chest. [NIH]

Child Care: Care of children in the home or institution. [NIH]

Chin: The anatomical frontal portion of the mandible, also known as the mentum, that contains the line of fusion of the two separate halves of the mandible (symphysis menti). This line of fusion divides inferiorly to enclose a triangular area called the mental protuberance. On each side, inferior to the second premolar tooth, is the mental foramen for the passage of blood vessels and a nerve. [NIH]

Chlorine: A greenish-yellow, diatomic gas that is a member of the halogen family of elements. It has the atomic symbol Cl, atomic number 17, and atomic weight 70.906. It is a powerful irritant that can cause fatal pulmonary edema. Chlorine is used in manufacturing, as a reagent in synthetic chemistry, for water purification, and in the production of chlorinated lime, which is used in fabric bleaching. [NIH]

Cholesterol: The principal sterol of all higher animals, distributed in body tissues, especially the brain and spinal cord, and in animal fats and oils. [NIH]

Cholesterol Esters: Fatty acid esters of cholesterol which constitute about two-thirds of the cholesterol in the plasma. The accumulation of cholesterol esters in the arterial intima is a characteristic feature of atherosclerosis. [NIH]

Choline: A basic constituent of lecithin that is found in many plants and animal organs. It is important as a precursor of acetylcholine, as a methyl donor in various metabolic processes, and in lipid metabolism. [NIH]

Cholinergic: Resembling acetylcholine in pharmacological action; stimulated by or releasing acetylcholine or a related compound. [EU]

Chondrocytes: Polymorphic cells that form cartilage. [NIH]

Chondroitin sulfate: The major glycosaminoglycan (a type of sugar molecule) in cartilage. [NIH]

Chorea: Involuntary, forcible, rapid, jerky movements that may be subtle or become confluent, markedly altering normal patterns of movement. Hypotonia and pendular reflexes are often associated. Conditions which feature recurrent or persistent episodes of chorea as a primary manifestation of disease are referred to as choreatic disorders. Chorea is also a frequent manifestation of basal ganglia diseases. [NIH]

Choroid: The thin, highly vascular membrane covering most of the posterior of the eye between the retina and sclera. [NIH]

Chromatin: The material of chromosomes. It is a complex of DNA, histones, and nonhistone proteins (chromosomal proteins, non-histone) found within the nucleus of a cell. [NIH]

Chromium: A trace element that plays a role in glucose metabolism. It has the atomic symbol Cr, atomic number 24, and atomic weight 52. According to the Fourth Annual Report on Carcinogens (NTP85-002,1985), chromium and some of its compounds have been listed as known carcinogens. [NIH]

Chromosomal: Pertaining to chromosomes. [EU]

Chromosome: Part of a cell that contains genetic information. Except for sperm and eggs, all human cells contain 46 chromosomes. [NIH]

Chronic: A disease or condition that persists or progresses over a long period of time. [NIH]

Chronic Disease: Disease or ailment of long duration. [NIH]

Chronic renal: Slow and progressive loss of kidney function over several years, often resulting in end-stage renal disease. People with end-stage renal disease need dialysis or transplantation to replace the work of the kidneys. [NIH]

Chronotropic: Affecting the time or rate, as the rate of contraction of the heart. [EU]

Chylomicrons: A class of lipoproteins that carry dietary cholesterol and triglycerides from the small intestines to the tissues. [NIH]

Cirrhosis: A type of chronic, progressive liver disease. [NIH]

CIS: Cancer Information Service. The CIS is the National Cancer Institute's link to the public, interpreting and explaining research findings in a clear and understandable manner, and providing personalized responses to specific questions about cancer. Access the CIS by calling 1-800-4-CANCER, or by using the Web site at http://cis.nci.nih.gov. [NIH]

Citrus: Any tree or shrub of the Rue family or the fruit of these plants. [NIH]

Clamp: A u-shaped steel rod used with a pin or wire for skeletal traction in the treatment of certain fractures. [NIH]

Cleft Lip: Congenital defect in the upper lip where the maxillary prominence fails to merge with the merged medial nasal prominences. It is thought to be caused by faulty migration of the mesoderm in the head region. [NIH]

Cleft Palate: Congenital fissure of the soft and/or hard palate, due to faulty fusion. [NIH]

Clinical Clerkship: Undergraduate medical education programs for second-, third-, and fourth-year students in which the students receive clinical training and experience in teaching hospitals or affiliated health centers. [NIH]

Clinical Medicine: The study and practice of medicine by direct examination of the patient. [NIH]

Clinical study: A research study in which patients receive treatment in a clinic or other medical facility. Reports of clinical studies can contain results for single patients (case reports) or many patients (case series or clinical trials). [NIH]

Clinical trial: A research study that tests how well new medical treatments or other interventions work in people. Each study is designed to test new methods of screening, prevention, diagnosis, or treatment of a disease. [NIH]

Cloning: The production of a number of genetically identical individuals; in genetic engineering, a process for the efficient replication of a great number of identical DNA molecules. [NIH]

Coagulation: 1. The process of clot formation. 2. In colloid chemistry, the solidification of a sol into a gelatinous mass; an alteration of a disperse phase or of a dissolved solid which causes the separation of the system into a liquid phase and an insoluble mass called the clot or curd. Coagulation is usually irreversible. 3. In surgery, the disruption of tissue by physical means to form an amorphous residuum, as in electrocoagulation and photocoagulation. [EU]

Coca: Any of several South American shrubs of the Erythroxylon genus (and family) that yield cocaine; the leaves are chewed with alum for CNS stimulation. [NIH]

Cocaine: An alkaloid ester extracted from the leaves of plants including coca. It is a local anesthetic and vasoconstrictor and is clinically used for that purpose, particularly in the eye, ear, nose, and throat. It also has powerful central nervous system effects similar to the amphetamines and is a drug of abuse. Cocaine, like amphetamines, acts by multiple mechanisms on brain catecholaminergic neurons; the mechanism of its reinforcing effects is thought to involve inhibition of dopamine uptake. [NIH]

Codon: A set of three nucleotides in a protein coding sequence that specifies individual amino acids or a termination signal (codon, terminator). Most codons are universal, but some organisms do not produce the transfer RNAs (RNA, transfer) complementary to all codons. These codons are referred to as unassigned codons (codons, nonsense). [NIH]

Coenzyme: An organic nonprotein molecule, frequently a phosphorylated derivative of a water-soluble vitamin, that binds with the protein molecule (apoenzyme) to form the active enzyme (holoenzyme). [EU]

Cofactor: A substance, microorganism or environmental factor that activates or enhances the action of another entity such as a disease-causing agent. [NIH]

Cognition: Intellectual or mental process whereby an organism becomes aware of or obtains knowledge. [NIH]

Cognitive restructuring: A method of identifying and replacing fear-promoting, irrational beliefs with more realistic and functional ones. [NIH]

Cohort Studies: Studies in which subsets of a defined population are identified. These groups may or may not be exposed to factors hypothesized to influence the probability of the occurrence of a particular disease or other outcome. Cohorts are defined populations which, as a whole, are followed in an attempt to determine distinguishing subgroup characteristics. [NIH]

Colitis: Inflammation of the colon. [NIH]

Collagen: A polypeptide substance comprising about one third of the total protein in mammalian organisms. It is the main constituent of skin, connective tissue, and the organic substance of bones and teeth. Different forms of collagen are produced in the body but all consist of three alpha-polypeptide chains arranged in a triple helix. Collagen is differentiated from other fibrous proteins, such as elastin, by the content of proline, hydroxyproline, and hydroxylysine; by the absence of tryptophan; and particularly by the high content of polar groups which are responsible for its swelling properties. [NIH]

Collapse: 1. A state of extreme prostration and depression, with failure of circulation. 2. Abnormal falling in of the walls of any part of organ. [EU]

Collateral Circulation: Maintenance of blood flow to an organ despite obstruction of a principal vessel. Blood flow is maintained through small vessels. [NIH]

Colloidal: Of the nature of a colloid. [EU]

Colon: The long, coiled, tubelike organ that removes water from digested food. The remaining material, solid waste called stool, moves through the colon to the rectum and leaves the body through the anus. [NIH]

Combination Therapy: Association of 3 drugs to treat AIDS (AZT + DDC or DDI + protease inhibitor). [NIH]

Complement: A term originally used to refer to the heat-labile factor in serum that causes immune cytolysis, the lysis of antibody-coated cells, and now referring to the entire functionally related system comprising at least 20 distinct serum proteins that is the effector not only of immune cytolysis but also of other biologic functions. Complement activation occurs by two different sequences, the classic and alternative pathways. The proteins of the classic pathway are termed 'components of complement' and are designated by the symbols C1 through C9. C1 is a calcium-dependent complex of three distinct proteins C1q, C1r and C1s. The proteins of the alternative pathway (collectively referred to as the properdin system) and complement regulatory proteins are known by semisystematic or trivial names. Fragments resulting from proteolytic cleavage of complement proteins are designated with lower-case letter suffixes, e.g., C3a. Inactivated fragments may be designated with the suffix 'i', e.g. C3bi. Activated components or complexes with biological activity are designated by a bar over the symbol e.g. C1 or C4b,2a. The classic pathway is activated by the binding of C1 to classic pathway activators, primarily antigen-antibody complexes containing IgM, IgG1, IgG3; C1q binds to a single IgM molecule or two adjacent IgG molecules. The alternative pathway can be activated by IgA immune complexes and also by nonimmunologic materials

including bacterial endotoxins, microbial polysaccharides, and cell walls. Activation of the classic pathway triggers an enzymatic cascade involving C1, C4, C2 and C3; activation of the alternative pathway triggers a cascade involving C3 and factors B, D and P. Both result in the cleavage of C5 and the formation of the membrane attack complex. Complement activation also results in the formation of many biologically active complement fragments that act as anaphylatoxins, opsonins, or chemotactic factors. [EU]

Complementary and alternative medicine: CAM. Forms of treatment that are used in addition to (complementary) or instead of (alternative) standard treatments. These practices are not considered standard medical approaches. CAM includes dietary supplements, megadose vitamins, herbal preparations, special teas, massage therapy, magnet therapy, spiritual healing, and meditation. [NIH]

Complementary medicine: Practices not generally recognized by the medical community as standard or conventional medical approaches and used to enhance or complement the standard treatments. Complementary medicine includes the taking of dietary supplements, megadose vitamins, and herbal preparations; the drinking of special teas; and practices such as massage therapy, magnet therapy, spiritual healing, and meditation. [NIH]

Complementation: The production of a wild-type phenotype when two different mutations are combined in a diploid or a heterokaryon and tested in trans-configuration. [NIH]

Compliance: Distensibility measure of a chamber such as the lungs (lung compliance) or bladder. Compliance is expressed as a change in volume per unit change in pressure. [NIH]

Compress: A plug used to occludate an orifice in the control of bleeding, or to mop up secretions; an absorbent pad. [NIH]

Computational Biology: A field of biology concerned with the development of techniques for the collection and manipulation of biological data, and the use of such data to make biological discoveries or predictions. This field encompasses all computational methods and theories applicable to molecular biology and areas of computer-based techniques for solving biological problems including manipulation of models and datasets. [NIH]

Computed tomography: CT scan. A series of detailed pictures of areas inside the body, taken from different angles; the pictures are created by a computer linked to an x-ray machine. Also called computerized tomography and computerized axial tomography (CAT) scan. [NIH]

Computer Simulation: Computer-based representation of physical systems and phenomena such as chemical processes. [NIH]

Computerized tomography: A series of detailed pictures of areas inside the body, taken from different angles; the pictures are created by a computer linked to an x-ray machine. Also called computerized axial tomography (CAT) scan and computed tomography (CT scan). [NIH]

Concentric: Having a common center of curvature or symmetry. [NIH]

Conception: The onset of pregnancy, marked by implantation of the blastocyst; the formation of a viable zygote. [EU]

Concomitant: Accompanying; accessory; joined with another. [EU]

Conduction: The transfer of sound waves, heat, nervous impulses, or electricity. [EU]

Cones: One type of specialized light-sensitive cells (photoreceptors) in the retina that provide sharp central vision and color vision. [NIH]

Confounding: Extraneous variables resulting in outcome effects that obscure or exaggerate the "true" effect of an intervention. [NIH]

Congestion: Excessive or abnormal accumulation of blood in a part. [EU]

Congestive heart failure: Weakness of the heart muscle that leads to a buildup of fluid in body tissues. [NIH]

Conjugated: Acting or operating as if joined; simultaneous. [EU]

Conjunctiva: The mucous membrane that lines the inner surface of the eyelids and the anterior part of the sclera. [NIH]

Connective Tissue: Tissue that supports and binds other tissues. It consists of connective tissue cells embedded in a large amount of extracellular matrix. [NIH]

Connective Tissue: Tissue that supports and binds other tissues. It consists of connective tissue cells embedded in a large amount of extracellular matrix. [NIH]

Connective Tissue Cells: A group of cells that includes fibroblasts, cartilage cells, adipocytes, smooth muscle cells, and bone cells. [NIH]

Connexins: A group of homologous proteins which form the intermembrane channels of gap junctions. The connexins are the products of an identified gene family which has both highly conserved and highly divergent regions. The variety contributes to the wide range of functional properties of gap junctions. [NIH]

Consciousness: Sense of awareness of self and of the environment. [NIH]

Constriction: The act of constricting. [NIH]

Consultation: A deliberation between two or more physicians concerning the diagnosis and the proper method of treatment in a case. [NIH]

Consumption: Pulmonary tuberculosis. [NIH]

Contraception: Use of agents, devices, methods, or procedures which diminish the likelihood of or prevent conception. [NIH]

Contractility: Capacity for becoming short in response to a suitable stimulus. [EU]

Contraindications: Any factor or sign that it is unwise to pursue a certain kind of action or treatment, e. g. giving a general anesthetic to a person with pneumonia. [NIH]

Contrast medium: A substance that is introduced into or around a structure and, because of the difference in absorption of x-rays by the contrast medium and the surrounding tissues, allows radiographic visualization of the structure. [EU]

Control group: In a clinical trial, the group that does not receive the new treatment being studied. This group is compared to the group that receives the new treatment, to see if the new treatment works. [NIH]

Controlled clinical trial: A clinical study that includes a comparison (control) group. The comparison group receives a placebo, another treatment, or no treatment at all. [NIH]

Controlled study: An experiment or clinical trial that includes a comparison (control) group. [NIH]

Conus: A large, circular, white patch around the optic disk due to the exposing of the sclera as a result of degenerative change or congenital abnormality in the choroid and retina. [NIH]

Conventional therapy: A currently accepted and widely used treatment for a certain type of disease, based on the results of past research. Also called conventional treatment. [NIH]

Conventional treatment: A currently accepted and widely used treatment for a certain type of disease, based on the results of past research. Also called conventional therapy. [NIH]

Convulsions: A general term referring to sudden and often violent motor activity of cerebral or brainstem origin. Convulsions may also occur in the absence of an electrical cerebral discharge (e.g., in response to hypotension). [NIH]

Coordination: Muscular or motor regulation or the harmonious cooperation of muscles or groups of muscles, in a complex action or series of actions. [NIH]

Coronary: Encircling in the manner of a crown; a term applied to vessels; nerves, ligaments, etc. The term usually denotes the arteries that supply the heart muscle and, by extension, a pathologic involvement of them. [EU]

Coronary Angiography: Radiography of the vascular system of the heart muscle after injection of a contrast medium. [NIH]

Coronary Arteriosclerosis: Thickening and loss of elasticity of the coronary arteries. [NIH]

Coronary Artery Bypass: Surgical therapy of ischemic coronary artery disease achieved by grafting a section of saphenous vein, internal mammary artery, or other substitute between the aorta and the obstructed coronary artery distal to the obstructive lesion. [NIH]

Coronary Circulation: The circulation of blood through the coronary vessels of the heart. [NIH]

Coronary Disease: Disorder of cardiac function due to an imbalance between myocardial function and the capacity of the coronary vessels to supply sufficient flow for normal function. It is a form of myocardial ischemia (insufficient blood supply to the heart muscle) caused by a decreased capacity of the coronary vessels. [NIH]

Coronary heart disease: A type of heart disease caused by narrowing of the coronary arteries that feed the heart, which needs a constant supply of oxygen and nutrients carried by the blood in the coronary arteries. When the coronary arteries become narrowed or clogged by fat and cholesterol deposits and cannot supply enough blood to the heart, CHD results. [NIH]

Coronary Thrombosis: Presence of a thrombus in a coronary artery, often causing a myocardial infarction. [NIH]

Coronary Vessels: The veins and arteries of the heart. [NIH]

Corpus: The body of the uterus. [NIH]

Corpus Luteum: The yellow glandular mass formed in the ovary by an ovarian follicle that has ruptured and discharged its ovum. [NIH]

Cortex: The outer layer of an organ or other body structure, as distinguished from the internal substance. [EU]

Corticosteroid: Any of the steroids elaborated by the adrenal cortex (excluding the sex hormones of adrenal origin) in response to the release of corticotrophin (adrenocorticotropic hormone) by the pituitary gland, to any of the synthetic equivalents of these steroids, or to angiotensin II. They are divided, according to their predominant biological activity, into three major groups: glucocorticoids, chiefly influencing carbohydrate, fat, and protein metabolism; mineralocorticoids, affecting the regulation of electrolyte and water balance; and C19 androgens. Some corticosteroids exhibit both types of activity in varying degrees, and others exert only one type of effect. The corticosteroids are used clinically for hormonal replacement therapy, for suppression of ACTH secretion by the anterior pituitary, as antineoplastic, antiallergic, and anti-inflammatory agents, and to suppress the immune response. Called also adrenocortical hormone and corticoid. [EU]

Cortisol: A steroid hormone secreted by the adrenal cortex as part of the body's response to stress. [NIH]

Cosmic Radiation: High-energy radiation or particles from extraterrestrial space that strike the earth, its atmosphere, or spacecraft and may create secondary radiation as a result of collisions with the atmosphere or spacecraft. [NIH]

Cosmids: Plasmids containing at least one cos (cohesive-end site) of phage lambda. They are

used as cloning vehicles for the study of aberrant eukaryotic structural genes and also as genetic vectors for introducing the nucleic acid of transforming viruses into cultured cells. [NIH]

Counterpulsation: A technique for assisting the circulation by decreasing the afterload of the left ventricle and augmenting the diastolic pressure. It may be achieved by intra-aortic balloon, or by implanting a special pumping device in the chest, or externally by applying a negative pressure to the lower extremities during cardiac systole. [NIH]

Cranial: Pertaining to the cranium, or to the anterior (in animals) or superior (in humans) end of the body. [EU]

Creatinine: A compound that is excreted from the body in urine. Creatinine levels are measured to monitor kidney function. [NIH]

Critical Care: Health care provided to a critically ill patient during a medical emergency or crisis. [NIH]

Cross-Sectional Studies: Studies in which the presence or absence of disease or other health-related variables are determined in each member of the study population or in a representative sample at one particular time. This contrasts with longitudinal studies which are followed over a period of time. [NIH]

Cryptosporidiosis: Parasitic intestinal infection with severe diarrhea caused by a protozoan, Cryptosporidium. It occurs in both animals and humans. [NIH]

Cultured cells: Animal or human cells that are grown in the laboratory. [NIH]

Curative: Tending to overcome disease and promote recovery. [EU]

Cutaneous: Having to do with the skin. [NIH]

Cyclic: Pertaining to or occurring in a cycle or cycles; the term is applied to chemical compounds that contain a ring of atoms in the nucleus. [EU]

Cyst: A sac or capsule filled with fluid. [NIH]

Cystathionine beta-Synthase: A multifunctional pyridoxal phosphate enzyme. In the second stage of cysteine biosynthesis it catalyzes the reaction of homocysteine with serine to form cystathionine with the elimination of water. Deficiency of this enzyme leads to hyperhomocysteinemia and homocystinuria. EC 4.2.1.22. [NIH]

Cytochrome: Any electron transfer hemoprotein having a mode of action in which the transfer of a single electron is effected by a reversible valence change of the central iron atom of the heme prosthetic group between the +2 and +3 oxidation states; classified as cytochromes a in which the heme contains a formyl side chain, cytochromes b, which contain protoheme or a closely similar heme that is not covalently bound to the protein, cytochromes c in which protoheme or other heme is covalently bound to the protein, and cytochromes d in which the iron-tetrapyrrole has fewer conjugated double bonds than the hemes have. Well-known cytochromes have been numbered consecutively within groups and are designated by subscripts (beginning with no subscript), e.g. cytochromes c, c1, C2, . New cytochromes are named according to the wavelength in nanometres of the absorption maximum of the a-band of the iron (II) form in pyridine, e.g., c-555. [EU]

Cytokine: Small but highly potent protein that modulates the activity of many cell types, including T and B cells. [NIH]

Cytoplasm: The protoplasm of a cell exclusive of that of the nucleus; it consists of a continuous aqueous solution (cytosol) and the organelles and inclusions suspended in it (phaneroplasm), and is the site of most of the chemical activities of the cell. [EU]

Cytoskeleton: The network of filaments, tubules, and interconnecting filamentous bridges which give shape, structure, and organization to the cytoplasm. [NIH]

Cytotoxic: Cell-killing. [NIH]

Dairy Products: Raw and processed or manufactured milk and milk-derived products. These are usually from cows (bovine) but are also from goats, sheep, reindeer, and water buffalo. [NIH]

Data Collection: Systematic gathering of data for a particular purpose from various sources, including questionnaires, interviews, observation, existing records, and electronic devices. The process is usually preliminary to statistical analysis of the data. [NIH]

Daunorubicin: Very toxic anthracycline aminoglycoside antibiotic isolated from Streptomyces peucetius and others, used in treatment of leukemias and other neoplasms. [NIH]

Death Certificates: Official records of individual deaths including the cause of death certified by a physician, and any other required identifying information. [NIH]

Decarboxylation: The removal of a carboxyl group, usually in the form of carbon dioxide, from a chemical compound. [NIH]

Degenerative: Undergoing degeneration : tending to degenerate; having the character of or involving degeneration; causing or tending to cause degeneration. [EU]

Dehydration: The condition that results from excessive loss of body water. [NIH]

Dehydroepiandrosterone: DHEA. A substance that is being studied as a cancer prevention drug. It belongs to the family of drugs called steroids. [NIH]

Deletion: A genetic rearrangement through loss of segments of DNA (chromosomes), bringing sequences, which are normally separated, into close proximity. [NIH]

Delivery of Health Care: The concept concerned with all aspects of providing and distributing health services to a patient population. [NIH]

Delusions: A false belief regarding the self or persons or objects outside the self that persists despite the facts, and is not considered tenable by one's associates. [NIH]

Dementia: An acquired organic mental disorder with loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning. The dysfunction is multifaceted and involves memory, behavior, personality, judgment, attention, spatial relations, language, abstract thought, and other executive functions. The intellectual decline is usually progressive, and initially spares the level of consciousness. [NIH]

Denaturation: Rupture of the hydrogen bonds by heating a DNA solution and then cooling it rapidly causes the two complementary strands to separate. [NIH]

Dendrites: Extensions of the nerve cell body. They are short and branched and receive stimuli from other neurons. [NIH]

Density: The logarithm to the base 10 of the opacity of an exposed and processed film. [NIH]

Dental Care: The total of dental diagnostic, preventive, and restorative services provided to meet the needs of a patient (from Illustrated Dictionary of Dentistry, 1982). [NIH]

Dental Caries: Localized destruction of the tooth surface initiated by decalcification of the enamel followed by enzymatic lysis of organic structures and leading to cavity formation. If left unchecked, the cavity may penetrate the enamel and dentin and reach the pulp. The three most prominent theories used to explain the etiology of the disase are that acids produced by bacteria lead to decalcification; that micro-organisms destroy the enamel protein; or that keratolytic micro-organisms produce chelates that lead to decalcification. [NIH]

Dentists: Individuals licensed to practice dentistry. [NIH]

Depressive Disorder: An affective disorder manifested by either a dysphoric mood or loss

of interest or pleasure in usual activities. The mood disturbance is prominent and relatively persistent. [NIH]

Deprivation: Loss or absence of parts, organs, powers, or things that are needed. [EU]

Desquamation: The shedding of epithelial elements, chiefly of the skin, in scales or small sheets; exfoliation. [EU]

Detergents: Purifying or cleansing agents, usually salts of long-chain aliphatic bases or acids, that exert cleansing (oil-dissolving) and antimicrobial effects through a surface action that depends on possessing both hydrophilic and hydrophobic properties. [NIH]

Developing Countries: Countries in the process of change directed toward economic growth, that is, an increase in production, per capita consumption, and income. The process of economic growth involves better utilization of natural and human resources, which results in a change in the social, political, and economic structures. [NIH]

Developmental Biology: The field of biology which deals with the process of the growth and differentiation of an organism. [NIH]

Dextroamphetamine: The d-form of amphetamine. It is a central nervous system stimulant and a sympathomimetic. It has also been used in the treatment of narcolepsy and of attention deficit disorders and hyperactivity in children. Dextroamphetamine has multiple mechanisms of action including blocking uptake of adrenergics and dopamine, stimulating release of monamines, and inhibiting monoamine oxidase. It is also a drug of abuse and a psychotomimetic. [NIH]

Diabetes Mellitus: A heterogeneous group of disorders that share glucose intolerance in common. [NIH]

Diabetic Foot: Ulcers of the foot as a complication of diabetes. Diabetic foot, often with infection, is a common serious complication of diabetes and may require hospitalization and disfiguring surgery. The foot ulcers are probably secondary to neuropathies and vascular problems. [NIH]

Diagnostic procedure: A method used to identify a disease. [NIH]

Dialyzer: A part of the hemodialysis machine. (See hemodialysis under dialysis.) The dialyzer has two sections separated by a membrane. One section holds dialysate. The other holds the patient's blood. [NIH]

Diarrhea: Passage of excessively liquid or excessively frequent stools. [NIH]

Diastole: Period of relaxation of the heart, especially the ventricles. [NIH]

Diastolic: Of or pertaining to the diastole. [EU]

Diastolic blood pressure: The minimum pressure that remains within the artery when the heart is at rest. [NIH]

Diastolic pressure: The lowest pressure to which blood pressure falls between contractions of the ventricles. [NIH]

Diathesis: A constitution or condition of the body which makes the tissues react in special ways to certain extrinsic stimuli and thus tends to make the person more than usually susceptible to certain diseases. [EU]

Dietary Fats: Fats present in food, especially in animal products such as meat, meat products, butter, ghee. They are present in lower amounts in nuts, seeds, and avocados. [NIH]

Dietary Fiber: The remnants of plant cell walls that are resistant to digestion by the alimentary enzymes of man. It comprises various polysaccharides and lignins. [NIH]

Dietitian: An expert in nutrition who helps people plan what and how much food to eat.

[NIH]

Digestion: The process of breakdown of food for metabolism and use by the body. [NIH]

Digestive system: The organs that take in food and turn it into products that the body can use to stay healthy. Waste products the body cannot use leave the body through bowel movements. The digestive system includes the salivary glands, mouth, esophagus, stomach, liver, pancreas, gallbladder, small and large intestines, and rectum. [NIH]

Digestive tract: The organs through which food passes when food is eaten. These organs are the mouth, esophagus, stomach, small and large intestines, and rectum. [NIH]

Dihydrotestosterone: Anabolic agent. [NIH]

Dilatation: The act of dilating. [NIH]

Dilatation, Pathologic: The condition of an anatomical structure's being dilated beyond normal dimensions. [NIH]

Dilated cardiomyopathy: Heart muscle disease that leads to enlargement of the heart's chambers, robbing the heart of its pumping ability. [NIH]

Dilation: A process by which the pupil is temporarily enlarged with special eye drops (mydriatic); allows the eye care specialist to better view the inside of the eye. [NIH]

Dilator: A device used to stretch or enlarge an opening. [NIH]

Diploid: Having two sets of chromosomes. [NIH]

Direct: 1. Straight; in a straight line. 2. Performed immediately and without the intervention of subsidiary means. [EU]

Discrete: Made up of separate parts or characterized by lesions which do not become blended; not running together; separate. [NIH]

Disease Progression: The worsening of a disease over time. This concept is most often used for chronic and incurable diseases where the stage of the disease is an important determinant of therapy and prognosis. [NIH]

Disinfectant: An agent that disinfects; applied particularly to agents used on inanimate objects. [EU]

Disparity: Failure of the two retinal images of an object to fall on corresponding retinal points. [NIH]

Disposition: A tendency either physical or mental toward certain diseases. [EU]

Dissection: Cutting up of an organism for study. [NIH]

Distal: Remote; farther from any point of reference; opposed to proximal. In dentistry, used to designate a position on the dental arch farther from the median line of the jaw. [EU]

DNA Topoisomerase: An enzyme catalyzing ATP-independent breakage of single-stranded DNA, followed by passage and rejoining of another single-stranded DNA. This enzyme class brings about the conversion of one topological isomer of DNA into another, e.g., the relaxation of superhelical turns in DNA, the interconversion of simple and knotted rings of single-stranded DNA, and the intertwisting of single-stranded rings of complementary sequences. (From Enzyme Nomenclature, 1992) EC 5.99.1.2. [NIH]

Dobutamine: A beta-2 agonist catecholamine that has cardiac stimulant action without evoking vasoconstriction or tachycardia. It is proposed as a cardiotonic after myocardial infarction or open heart surgery. [NIH]

Dopamine: An endogenous catecholamine and prominent neurotransmitter in several systems of the brain. In the synthesis of catecholamines from tyrosine, it is the immediate precursor to norepinephrine and epinephrine. Dopamine is a major transmitter in the

extrapyramidal system of the brain, and important in regulating movement. A family of dopaminergic receptor subtypes mediate its action. Dopamine is used pharmacologically for its direct (beta adrenergic agonist) and indirect (adrenergic releasing) sympathomimetic effects including its actions as an inotropic agent and as a renal vasodilator. [NIH]

Dorsal: 1. Pertaining to the back or to any dorsum. 2. Denoting a position more toward the back surface than some other object of reference; same as posterior in human anatomy; superior in the anatomy of quadrupeds. [EU]

Double Outlet Right Ventricle: Incomplete transposition of the great vessels in which both the aorta and the pulmonary artery arise from the right ventricle, often associated with a subaortic ventricular septal defect. [NIH]

Double-blind: Pertaining to a clinical trial or other experiment in which neither the subject nor the person administering treatment knows which treatment any particular subject is receiving. [EU]

Doxorubicin: Antineoplastic antibiotic obtained from Streptomyces peucetics. It is a hydroxy derivative of daunorubicin and is used in treatment of both leukemia and solid tumors. [NIH]

Drive: A state of internal activity of an organism that is a necessary condition before a given stimulus will elicit a class of responses; e.g., a certain level of hunger (drive) must be present before food will elicit an eating response. [NIH]

Drug Tolerance: Progressive diminution of the susceptibility of a human or animal to the effects of a drug, resulting from its continued administration. It should be differentiated from drug resistance wherein an organism, disease, or tissue fails to respond to the intended effectiveness of a chemical or drug. It should also be differentiated from maximum tolerated dose and no-observed-adverse-effect level. [NIH]

Duct: A tube through which body fluids pass. [NIH]

Ductus Arteriosus: A fetal blood vessel connecting the pulmonary artery with the descending aorta. [NIH]

Dyes: Chemical substances that are used to stain and color other materials. The coloring may or may not be permanent. Dyes can also be used as therapeutic agents and test reagents in medicine and scientific research. [NIH]

Dyslipidemia: Disorders in the lipoprotein metabolism; classified as hypercholesterolemia, hypertriglyceridemia, combined hyperlipidemia, and low levels of high-density lipoprotein (HDL) cholesterol. All of the dyslipidemias can be primary or secondary. Both elevated levels of low-density lipoprotein (LDL) cholesterol and low levels of HDL cholesterol predispose to premature atherosclerosis. [NIH]

Dyspareunia: Painful sexual intercourse. [NIH]

Dysphoric: A feeling of unpleasantness and discomfort. [NIH]

Dysplasia: Cells that look abnormal under a microscope but are not cancer. [NIH]

Dystrophy: Any disorder arising from defective or faulty nutrition, especially the muscular dystrophies. [EU]

Echocardiography: Ultrasonic recording of the size, motion, and composition of the heart and surrounding tissues. The standard approach is transthoracic. [NIH]

Eclampsia: Onset of convulsions or coma in a previously diagnosed pre-eclamptic patient. [NIH]

Ectoderm: The outer of the three germ layers of the embryo. [NIH]

Ectopic: Pertaining to or characterized by ectopia. [EU]

Edema: Excessive amount of watery fluid accumulated in the intercellular spaces, most commonly present in subcutaneous tissue. [NIH]

Effector: It is often an enzyme that converts an inactive precursor molecule into an active second messenger. [NIH]

Effector cell: A cell that performs a specific function in response to a stimulus; usually used to describe cells in the immune system. [NIH]

Efficacy: The extent to which a specific intervention, procedure, regimen, or service produces a beneficial result under ideal conditions. Ideally, the determination of efficacy is based on the results of a randomized control trial. [NIH]

Effusion: The escape of fluid into a part or tissue, as an exudation or a transudation. [EU]

Eicosanoids: A class of oxygenated, endogenous, unsaturated fatty acids derived from arachidonic acid. They include prostaglandins, leukotrienes, thromboxanes, and hydroxyeicosatetraenoic acid compounds (HETE). They are hormone-like substances that act near the site of synthesis without altering functions throughout the body. [NIH]

Ejection fraction: A measure of ventricular contractility, equal to normally 65 8 per cent; lower values indicate ventricular dysfunction. [EU]

Elastic: Susceptible of resisting and recovering from stretching, compression or distortion applied by a force. [EU]

Elasticity: Resistance and recovery from distortion of shape. [NIH]

Elastin: The protein that gives flexibility to tissues. [NIH]

Elective: Subject to the choice or decision of the patient or physician; applied to procedures that are advantageous to the patient but not urgent. [EU]

Electric shock: A dangerous patho-physiological effect resulting from an electric current passing through the body of a human or animal. [NIH]

Electrocardiogram: Measurement of electrical activity during heartbeats. [NIH]

Electrocardiograph: Apparatus which, by means of currents produced by contractions of the cardiac muscle, records heart movements as electro-cardiograms. [NIH]

Electrocardiography: Recording of the moment-to-moment electromotive forces of the heart as projected onto various sites on the body's surface, delineated as a scalar function of time. [NIH]

Electrolyte: A substance that dissociates into ions when fused or in solution, and thus becomes capable of conducting electricity; an ionic solute. [EU]

Electrophoresis: An electrochemical process in which macromolecules or colloidal particles with a net electric charge migrate in a solution under the influence of an electric current. [NIH]

Electrophysiological: Pertaining to electrophysiology, that is a branch of physiology that is concerned with the electric phenomena associated with living bodies and involved in their functional activity. [EU]

Elementary Particles: Individual components of atoms, usually subatomic; subnuclear particles are usually detected only when the atomic nucleus decays and then only transiently, as most of them are unstable, often yielding pure energy without substance, i.e., radiation. [NIH]

Emboli: Bit of foreign matter which enters the blood stream at one point and is carried until it is lodged or impacted in an artery and obstructs it. It may be a blood clot, an air bubble, fat or other tissue, or clumps of bacteria. [NIH]

Embolism: Blocking of a blood vessel by a blood clot or foreign matter that has been

transported from a distant site by the blood stream. [NIH]

Embolization: The blocking of an artery by a clot or foreign material. Embolization can be done as treatment to block the flow of blood to a tumor. [NIH]

Embolus: Bit of foreign matter which enters the blood stream at one point and is carried until it is lodged or impacted in an artery and obstructs it. It may be a blood clot, an air bubble, fat or other tissue, or clumps of bacteria. [NIH]

Embryo: The prenatal stage of mammalian development characterized by rapid morphological changes and the differentiation of basic structures. [NIH]

Embryogenesis: The process of embryo or embryoid formation, whether by sexual (zygotic) or asexual means. In asexual embryogenesis embryoids arise directly from the explant or on intermediary callus tissue. In some cases they arise from individual cells (somatic cell embryoge). [NIH]

Embryology: The study of the development of an organism during the embryonic and fetal stages of life. [NIH]

Emollient: Softening or soothing; called also malactic. [EU]

Emphysema: A pathological accumulation of air in tissues or organs. [NIH]

Empirical: A treatment based on an assumed diagnosis, prior to receiving confirmatory laboratory test results. [NIH]

Enalapril: An angiotensin-converting enzyme inhibitor that is used to treat hypertension. [NIH]

Endarterectomy: Surgical excision, performed under general anesthesia, of the atheromatous tunica intima of an artery. When reconstruction of an artery is performed as an endovascular procedure through a catheter, it is called atherectomy. [NIH]

Endocarditis: Exudative and proliferative inflammatory alterations of the endocardium, characterized by the presence of vegetations on the surface of the endocardium or in the endocardium itself, and most commonly involving a heart valve, but sometimes affecting the inner lining of the cardiac chambers or the endocardium elsewhere. It may occur as a primary disorder or as a complication of or in association with another disease. [EU]

Endocardium: The innermost layer of the heart, comprised of endothelial cells. [NIH]

Endocrine Glands: Ductless glands that secrete substances which are released directly into the circulation and which influence metabolism and other body functions. [NIH]

Endocrine System: The system of glands that release their secretions (hormones) directly into the circulatory system. In addition to the endocrine glands, included are the chromaffin system and the neurosecretory systems. [NIH]

Endocrinology: A subspecialty of internal medicine concerned with the metabolism, physiology, and disorders of the endocrine system. [NIH]

Endoderm: The inner of the three germ layers of the embryo. [NIH]

Endodontics: A dental specialty concerned with the maintenance of the dental pulp in a state of health and the treatment of the pulp cavity (pulp chamber and pulp canal). [NIH]

Endogenous: Produced inside an organism or cell. The opposite is external (exogenous) production. [NIH]

Endopeptidases: A subclass of peptide hydrolases. They are classified primarily by their catalytic mechanism. Specificity is used only for identification of individual enzymes. They comprise the serine endopeptidases, EC 3.4.21; cysteine endopeptidases, EC 3.4.22; aspartic endopeptidases, EC 3.4.23, metalloendopeptidases, EC 3.4.24; and a group of enzymes yet to be assigned to any of the above sub-classes, EC 3.4.99. EC 3.4.-. [NIH]

Endoscopic: A technique where a lateral-view endoscope is passed orally to the duodenum for visualization of the ampulla of Vater. [NIH]

Endothelial cell: The main type of cell found in the inside lining of blood vessels, lymph vessels, and the heart. [NIH]

Endothelium: A layer of epithelium that lines the heart, blood vessels (endothelium, vascular), lymph vessels (endothelium, lymphatic), and the serous cavities of the body. [NIH]

Endothelium, Lymphatic: Unbroken cellular lining (intima) of the lymph vessels (e.g., the high endothelial lymphatic venules). It is more permeable than vascular endothelium, lacking selective absorption and functioning mainly to remove plasma proteins that have filtered through the capillaries into the tissue spaces. [NIH]

Endothelium, Vascular: Single pavement layer of cells which line the luminal surface of the entire vascular system and regulate the transport of macromolecules and blood components from interstitium to lumen; this function has been most intensively studied in the blood capillaries. [NIH]

Endothelium-derived: Small molecule that diffuses to the adjacent muscle layer and relaxes it. [NIH]

Endotoxic: Of, relating to, or acting as an endotoxin (= a heat-stable toxin, associated with the outer membranes of certain gram-negative bacteria. Endotoxins are not secreted and are released only when the cells are disrupted). [EU]

Endotoxin: Toxin from cell walls of bacteria. [NIH]

End-stage renal: Total chronic kidney failure. When the kidneys fail, the body retains fluid and harmful wastes build up. A person with ESRD needs treatment to replace the work of the failed kidneys. [NIH]

Energy balance: Energy is the capacity of a body or a physical system for doing work. Energy balance is the state in which the total energy intake equals total energy needs. [NIH]

Enterovirus: A genus of the family Picornaviridae whose members preferentially inhabit the intestinal tract of a variety of hosts. The genus contains many species. Newly described members of human enteroviruses are assigned continuous numbers with the species designated "human enterovirus". [NIH]

Environmental Exposure: The exposure to potentially harmful chemical, physical, or biological agents in the environment or to environmental factors that may include ionizing radiation, pathogenic organisms, or toxic chemicals. [NIH]

Environmental Health: The science of controlling or modifying those conditions, influences, or forces surrounding man which relate to promoting, establishing, and maintaining health. [NIH]

Enzymatic: Phase where enzyme cuts the precursor protein. [NIH]

Enzyme: A protein that speeds up chemical reactions in the body. [NIH]

Enzyme Inhibitors: Compounds or agents that combine with an enzyme in such a manner as to prevent the normal substrate-enzyme combination and the catalytic reaction. [NIH]

Eosinophils: Granular leukocytes with a nucleus that usually has two lobes connected by a slender thread of chromatin, and cytoplasm containing coarse, round granules that are uniform in size and stainable by eosin. [NIH]

Epidemic: Occurring suddenly in numbers clearly in excess of normal expectancy; said especially of infectious diseases but applied also to any disease, injury, or other health-related event occurring in such outbreaks. [EU]

Epidemiologic Studies: Studies designed to examine associations, commonly, hypothesized

causal relations. They are usually concerned with identifying or measuring the effects of risk factors or exposures. The common types of analytic study are case-control studies, cohort studies, and cross-sectional studies. [NIH]

Epidemiological: Relating to, or involving epidemiology. [EU]

Epidermis: Nonvascular layer of the skin. It is made up, from within outward, of five layers: 1) basal layer (stratum basale epidermidis); 2) spinous layer (stratum spinosum epidermidis); 3) granular layer (stratum granulosum epidermidis); 4) clear layer (stratum lucidum epidermidis); and 5) horny layer (stratum corneum epidermidis). [NIH]

Epigastric: Having to do with the upper middle area of the abdomen. [NIH]

Epinephrine: The active sympathomimetic hormone from the adrenal medulla in most species. It stimulates both the alpha- and beta- adrenergic systems, causes systemic vasoconstriction and gastrointestinal relaxation, stimulates the heart, and dilates bronchi and cerebral vessels. It is used in asthma and cardiac failure and to delay absorption of local anesthetics. [NIH]

Epithelial: Refers to the cells that line the internal and external surfaces of the body. [NIH]

Epithelial Cells: Cells that line the inner and outer surfaces of the body. [NIH]

Epithelium: One or more layers of epithelial cells, supported by the basal lamina, which covers the inner or outer surfaces of the body. [NIH]

Epitopes: Sites on an antigen that interact with specific antibodies. [NIH]

Erectile: The inability to get or maintain an erection for satisfactory sexual intercourse. Also called impotence. [NIH]

Erection: The condition of being made rigid and elevated; as erectile tissue when filled with blood. [EU]

Ergot: Cataract due to ergot poisoning caused by eating of rye cereals contaminated by a fungus. [NIH]

Erythema: Redness of the skin produced by congestion of the capillaries. This condition may result from a variety of causes. [NIH]

Erythrocyte Indices: Quantification of size and cell hemoglobin content or concentration of the erythrocyte, usually derived from erythrocyte count, blood hemoglobin concentration, and hematocrit. Includes the mean cell volume (MCV), mean cell hemoglobin (MCH), and mean cell hemoglobin concentration (MCHC). Use also for cell diameter and thickness. [NIH]

Erythrocyte Volume: Volume of circulating erythrocytes. It is usually measured by radioisotope dilution technique. [NIH]

Erythrocytes: Red blood cells. Mature erythrocytes are non-nucleated, biconcave disks containing hemoglobin whose function is to transport oxygen. [NIH]

Erythromycin: A bacteriostatic antibiotic substance produced by Streptomyces erythreus. Erythromycin A is considered its major active component. In sensitive organisms, it inhibits protein synthesis by binding to 50S ribosomal subunits. This binding process inhibits peptidyl transferase activity and interferes with translocation of amino acids during translation and assembly of proteins. [NIH]

Esophagus: The muscular tube through which food passes from the throat to the stomach. [NIH]

Essential Tremor: A rhythmic, involuntary, purposeless, oscillating movement resulting from the alternate contraction and relaxation of opposing groups of muscles. [NIH]

Estradiol: The most potent mammalian estrogenic hormone. It is produced in the ovary, placenta, testis, and possibly the adrenal cortex. [NIH]

Estrogen: One of the two female sex hormones. [NIH]

Estrogen receptor: ER. Protein found on some cancer cells to which estrogen will attach. [NIH]

Estrogen Receptor Modulators: Substances that possess antiestrogenic actions but can also produce estrogenic effects as well. They act as complete or partial agonist or as antagonist. They can be either steroidal or nonsteroidal in structure. [NIH]

Estrogen Replacement Therapy: The use of hormonal agents with estrogen-like activity in postmenopausal or other estrogen-deficient women to alleviate effects of hormone deficiency, such as vasomotor symptoms, dyspareunia, and progressive development of osteoporosis. This may also include the use of progestational agents in combination therapy. [NIH]

Ethanol: A clear, colorless liquid rapidly absorbed from the gastrointestinal tract and distributed throughout the body. It has bactericidal activity and is used often as a topical disinfectant. It is widely used as a solvent and preservative in pharmaceutical preparations as well as serving as the primary ingredient in alcoholic beverages. [NIH]

Ethnic Groups: A group of people with a common cultural heritage that sets them apart from others in a variety of social relationships. [NIH]

Eukaryotic Cells: Cells of the higher organisms, containing a true nucleus bounded by a nuclear membrane. [NIH]

Evoke: The electric response recorded from the cerebral cortex after stimulation of a peripheral sense organ. [NIH]

Excitation: An act of irritation or stimulation or of responding to a stimulus; the addition of energy, as the excitation of a molecule by absorption of photons. [EU]

Excrete: To get rid of waste from the body. [NIH]

Exercise Test: Controlled physical activity, more strenuous than at rest, which is performed in order to allow assessment of physiological functions, particularly cardiovascular and pulmonary, but also aerobic capacity. Maximal (most intense) exercise is usually required but submaximal exercise is also used. The intensity of exercise is often graded, using criteria such as rate of work done, oxygen consumption, and heart rate. Physiological data obtained from an exercise test may be used for diagnosis, prognosis, and evaluation of disease severity, and to evaluate therapy. Data may also be used in prescribing exercise by determining a person's exercise capacity. [NIH]

Exfoliation: A falling off in scales or layers. [EU]

Exocrine: Secreting outwardly, via a duct. [EU]

Exogenous: Developed or originating outside the organism, as exogenous disease. [EU]

Expiration: The act of breathing out, or expelling air from the lungs. [EU]

Extracellular: Outside a cell or cells. [EU]

Extracellular Matrix: A meshwork-like substance found within the extracellular space and in association with the basement membrane of the cell surface. It promotes cellular proliferation and provides a supporting structure to which cells or cell lysates in culture dishes adhere. [NIH]

Extracellular Matrix Proteins: Macromolecular organic compounds that contain carbon, hydrogen, oxygen, nitrogen, and usually, sulfur. These macromolecules (proteins) form an intricate meshwork in which cells are embedded to construct tissues. Variations in the relative types of macromolecules and their organization determine the type of extracellular matrix, each adapted to the functional requirements of the tissue. The two main classes of macromolecules that form the extracellular matrix are: glycosaminoglycans, usually linked

to proteins (proteoglycans), and fibrous proteins (e.g., collagen, elastin, fibronectins and laminin). [NIH]

Extracellular Space: Interstitial space between cells, occupied by fluid as well as amorphous and fibrous substances. [NIH]

Extracorporeal: Situated or occurring outside the body. [EU]

Extracorporeal Circulation: Diversion of blood flow through a circuit located outside the body but continuous with the bodily circulation. [NIH]

Extraction: The process or act of pulling or drawing out. [EU]

Extremity: A limb; an arm or leg (membrum); sometimes applied specifically to a hand or foot. [EU]

Eye Movements: Voluntary or reflex-controlled movements of the eye. [NIH]

Family Planning: Programs or services designed to assist the family in controlling reproduction by either improving or diminishing fertility. [NIH]

Fasciculation: A small local contraction of muscles, visible through the skin, representing a spontaneous discharge of a number of fibres innervated by a single motor nerve filament. [EU]

Fat: Total lipids including phospholipids. [NIH]

Fathers: Male parents, human or animal. [NIH]

Fatigue: The state of weariness following a period of exertion, mental or physical, characterized by a decreased capacity for work and reduced efficiency to respond to stimuli. [NIH]

Fatty acids: A major component of fats that are used by the body for energy and tissue development. [NIH]

Femoral: Pertaining to the femur, or to the thigh. [EU]

Femoral Artery: The main artery of the thigh, a continuation of the external iliac artery. [NIH]

Femur: The longest and largest bone of the skeleton, it is situated between the hip and the knee. [NIH]

Fentanyl: A narcotic opioid drug that is used in the treatment of pain. [NIH]

Fetal Blood: Blood of the fetus. Exchange of nutrients and waste between the fetal and maternal blood occurs via the placenta. The cord blood is blood contained in the umbilical vessels at the time of delivery. [NIH]

Fetal Heart: The heart of the fetus of any viviparous animal. It refers to the heart in the postembryonic period and is differentiated from the embryonic heart (heart/embryology) only on the basis of time. [NIH]

Fetus: The developing offspring from 7 to 8 weeks after conception until birth. [NIH]

Fibril: Most bacterial viruses have a hollow tail with specialized fibrils at its tip. The tail fibers attach to the cell wall of the host. [NIH]

Fibrillation: A small, local, involuntary contraction of muscle, invisible under the skin, resulting from spontaneous activation of single muscle cells or muscle fibres. [EU]

Fibrin: A protein derived from fibrinogen in the presence of thrombin, which forms part of the blood clot. [NIH]

Fibrinogen: Plasma glycoprotein clotted by thrombin, composed of a dimer of three non-identical pairs of polypeptide chains (alpha, beta, gamma) held together by disulfide bonds. Fibrinogen clotting is a sol-gel change involving complex molecular arrangements: whereas fibrinogen is cleaved by thrombin to form polypeptides A and B, the proteolytic action of

other enzymes yields different fibrinogen degradation products. [NIH]

Fibrinolysis: The natural enzymatic dissolution of fibrin. [NIH]

Fibrinolytic: Pertaining to, characterized by, or causing the dissolution of fibrin by enzymatic action [EU]

Fibroblast Growth Factor: Peptide isolated from the pituitary gland and from the brain. It is a potent mitogen which stimulates growth of a variety of mesodermal cells including chondrocytes, granulosa, and endothelial cells. The peptide may be active in wound healing and animal limb regeneration. [NIH]

Fibroblasts: Connective tissue cells which secrete an extracellular matrix rich in collagen and other macromolecules. [NIH]

Fibronectins: Glycoproteins found on the surfaces of cells, particularly in fibrillar structures. The proteins are lost or reduced when these cells undergo viral or chemical transformation. They are highly susceptible to proteolysis and are substrates for activated blood coagulation factor VIII. The forms present in plasma are called cold-insoluble globulins. [NIH]

Fibrosis: Any pathological condition where fibrous connective tissue invades any organ, usually as a consequence of inflammation or other injury. [NIH]

Fibula: The bone of the lower leg lateral to and smaller than the tibia. In proportion to its length, it is the most slender of the long bones. [NIH]

Finasteride: An orally active testosterone 5-alpha-reductase inhibitor. It is used as a surgical alternative for treatment of benign prostatic hyperplasia. [NIH]

Fish Oils: Oils high in unsaturated fats extracted from the bodies of fish or fish parts, especially the livers. Those from the liver are usually high in vitamin A. The oils are used as dietary supplements, in soaps and detergents, as protective coatings, and as a base for other food products such as vegetable shortenings. [NIH]

Fissure: Any cleft or groove, normal or otherwise; especially a deep fold in the cerebral cortex which involves the entire thickness of the brain wall. [EU]

Flatus: Gas passed through the rectum. [NIH]

Flecainide: A potent anti-arrhythmia agent, effective in a wide range of ventricular and atrial arrhythmias and tachycardias. Paradoxically, however, in myocardial infarct patients with either symptomatic or asymptomatic arrhythmia, flecainide exacerbates the arrhythmia and is not recommended for use in these patients. [NIH]

Flow Cytometry: Technique using an instrument system for making, processing, and displaying one or more measurements on individual cells obtained from a cell suspension. Cells are usually stained with one or more fluorescent dyes specific to cell components of interest, e.g., DNA, and fluorescence of each cell is measured as it rapidly transverses the excitation beam (laser or mercury arc lamp). Fluorescence provides a quantitative measure of various biochemical and biophysical properties of the cell, as well as a basis for cell sorting. Other measurable optical parameters include light absorption and light scattering, the latter being applicable to the measurement of cell size, shape, density, granularity, and stain uptake. [NIH]

Fluorescence: The property of emitting radiation while being irradiated. The radiation emitted is usually of longer wavelength than that incident or absorbed, e.g., a substance can be irradiated with invisible radiation and emit visible light. X-ray fluorescence is used in diagnosis. [NIH]

Fluorescent Dyes: Dyes that emit light when exposed to light. The wave length of the emitted light is usually longer than that of the incident light. Fluorochromes are substances that cause fluorescence in other substances, i.e., dyes used to mark or label other compounds

with fluorescent tags. They are used as markers in biochemistry and immunology. [NIH]

Fluorine: A nonmetallic, diatomic gas that is a trace element and member of the halogen family. It is used in dentistry as flouride to prevent dental caries. [NIH]

Flushing: A transient reddening of the face that may be due to fever, certain drugs, exertion, stress, or a disease process. [NIH]

Flutter: A rapid vibration or pulsation. [EU]

Focus Groups: A method of data collection and a qualitative research tool in which a small group of individuals are brought together and allowed to interact in a discussion of their opinions about topics, issues, or questions. [NIH]

Foetoplacental: Pertaining to the fetus and placenta. [EU]

Folate: A B-complex vitamin that is being studied as a cancer prevention agent. Also called folic acid. [NIH]

Fold: A plication or doubling of various parts of the body. [NIH]

Folic Acid: N-(4-(((2-Amino-1,4-dihydro-4-oxo-6-pteridinyl)methyl)amino)benzoyl)-L-glutamic acid. A member of the vitamin B family that stimulates the hematopoietic system. It is present in the liver and kidney and is found in mushrooms, spinach, yeast, green leaves, and grasses. Folic acid is used in the treatment and prevention of folate deficiencies and megaloblastic anemia. [NIH]

Foot Ulcer: Lesion on the surface of the skin of the foot, usually accompanied by inflammation. The lesion may become infected or necrotic and is frequently associated with diabetes or leprosy. [NIH]

Forearm: The part between the elbow and the wrist. [NIH]

Fossa: A cavity, depression, or pit. [NIH]

Free Radicals: Highly reactive molecules with an unsatisfied electron valence pair. Free radicals are produced in both normal and pathological processes. They are proven or suspected agents of tissue damage in a wide variety of circumstances including radiation, damage from environment chemicals, and aging. Natural and pharmacological prevention of free radical damage is being actively investigated. [NIH]

Fungi: A kingdom of eukaryotic, heterotrophic organisms that live as saprobes or parasites, including mushrooms, yeasts, smuts, molds, etc. They reproduce either sexually or asexually, and have life cycles that range from simple to complex. Filamentous fungi refer to those that grow as multicelluar colonies (mushrooms and molds). [NIH]

Fura-2: A fluorescent calcium chelating agent which is used to study intracellular calcium in many tissues. The fluorescent and chelating properties of Fura-2 aid in the quantitation of endothelial cell injury, in monitoring ATP-dependent calcium uptake by membrane vesicles, and in the determination of the relationship between cytoplasmic free calcium and oxidase activation in rat neutrophils. [NIH]

Gallbladder: The pear-shaped organ that sits below the liver. Bile is concentrated and stored in the gallbladder. [NIH]

Gallstones: The solid masses or stones made of cholesterol or bilirubin that form in the gallbladder or bile ducts. [NIH]

Gamma-Glutamyltransferase: An enzyme that catalyzes reversibly the transfer of a glutamyl group from a glutamyl-peptide and an amino acid to a peptide and a glutamyl-amino acid. EC 2.3.2.2. [NIH]

Gamma-interferon: Interferon produced by T-lymphocytes in response to various mitogens and antigens. Gamma interferon appears to have potent antineoplastic, immunoregulatory

and antiviral activity. [NIH]

Ganglia: Clusters of multipolar neurons surrounded by a capsule of loosely organized connective tissue located outside the central nervous system. [NIH]

Ganglion: 1. A knot, or knotlike mass. 2. A general term for a group of nerve cell bodies located outside the central nervous system; occasionally applied to certain nuclear groups within the brain or spinal cord, e.g. basal ganglia. 3. A benign cystic tumour occurring on a aponeurosis or tendon, as in the wrist or dorsum of the foot; it consists of a thin fibrous capsule enclosing a clear mucinous fluid. [EU]

Gap Junctions: Connections between cells which allow passage of small molecules and electric current. Gap junctions were first described anatomically as regions of close apposition between cells with a narrow (1-2 nm) gap between cell membranes. The variety in the properties of gap junctions is reflected in the number of connexins, the family of proteins which form the junctions. [NIH]

Gas: Air that comes from normal breakdown of food. The gases are passed out of the body through the rectum (flatus) or the mouth (burp). [NIH]

Gastric: Having to do with the stomach. [NIH]

Gastric Emptying: The evacuation of food from the stomach into the duodenum. [NIH]

Gastrin: A hormone released after eating. Gastrin causes the stomach to produce more acid. [NIH]

Gastroenteritis: An acute inflammation of the lining of the stomach and intestines, characterized by anorexia, nausea, diarrhoea, abdominal pain, and weakness, which has various causes, including food poisoning due to infection with such organisms as Escherichia coli, Staphylococcus aureus, and Salmonella species; consumption of irritating food or drink; or psychological factors such as anger, stress, and fear. Called also enterogastritis. [EU]

Gastrointestinal: Refers to the stomach and intestines. [NIH]

Gastrointestinal tract: The stomach and intestines. [NIH]

Gastroparesis: Nerve or muscle damage in the stomach. Causes slow digestion and emptying, vomiting, nausea, or bloating. Also called delayed gastric emptying. [NIH]

Gemfibrozil: A lipid-regulating agent that lowers elevated serum lipids primarily by decreasing serum triglycerides with a variable reduction in total cholesterol. These decreases occur primarily in the VLDL fraction and less frequently in the LDL fraction. Gemfibrozil increases HDL subfractions HDL2 and HDL3 as well as apolipoproteins A-I and A-II. Its mechanism of action has not been definitely established. [NIH]

Gene: The functional and physical unit of heredity passed from parent to offspring. Genes are pieces of DNA, and most genes contain the information for making a specific protein. [NIH]

Gene Expression: The phenotypic manifestation of a gene or genes by the processes of gene action. [NIH]

Gene Targeting: The integration of exogenous DNA into the genome of an organism at sites where its expression can be suitably controlled. This integration occurs as a result of homologous recombination. [NIH]

Gene Therapy: The introduction of new genes into cells for the purpose of treating disease by restoring or adding gene expression. Techniques include insertion of retroviral vectors, transfection, homologous recombination, and injection of new genes into the nuclei of single cell embryos. The entire gene therapy process may consist of multiple steps. The new genes may be introduced into proliferating cells in vivo (e.g., bone marrow) or in vitro (e.g.,

fibroblast cultures) and the modified cells transferred to the site where the gene expression is required. Gene therapy may be particularly useful for treating enzyme deficiency diseases, hemoglobinopathies, and leukemias and may also prove useful in restoring drug sensitivity, particularly for leukemia. [NIH]

General practitioner: A medical practitioner who does not specialize in a particular branch of medicine or limit his practice to a specific class of diseases. [NIH]

Generator: Any system incorporating a fixed parent radionuclide from which is produced a daughter radionuclide which is to be removed by elution or by any other method and used in a radiopharmaceutical. [NIH]

Genetic Code: The specifications for how information, stored in nucleic acid sequence (base sequence), is translated into protein sequence (amino acid sequence). The start, stop, and order of amino acids of a protein is specified by consecutive triplets of nucleotides called codons (codon). [NIH]

Genetic Counseling: Advising families of the risks involved pertaining to birth defects, in order that they may make an informed decision on current or future pregnancies. [NIH]

Genetic Engineering: Directed modification of the gene complement of a living organism by such techniques as altering the DNA, substituting genetic material by means of a virus, transplanting whole nuclei, transplanting cell hybrids, etc. [NIH]

Genetic Markers: A phenotypically recognizable genetic trait which can be used to identify a genetic locus, a linkage group, or a recombination event. [NIH]

Genetic testing: Analyzing DNA to look for a genetic alteration that may indicate an increased risk for developing a specific disease or disorder. [NIH]

Genetic Vectors: Any DNA molecule capable of autonomous replication within a host cell and into which other DNA sequences can be inserted and thus amplified. Many are derived from plasmids, bacteriophages or viruses. They are used for transporting foreign genes into recipient cells. Genetic vectors possess a functional replicator site and contain genetic markers to facilitate their selective recognition. [NIH]

Genetics: The biological science that deals with the phenomena and mechanisms of heredity. [NIH]

Genistein: An isoflavonoid derived from soy products. It inhibits protein-tyrosine kinase and topoisomerase-ii (dna topoisomerase (atp-hydrolysing)) activity and is used as an antineoplastic and antitumor agent. Experimentally, it has been shown to induce G2 phase arrest in human and murine cell lines. [NIH]

Genital: Pertaining to the genitalia. [EU]

Genomics: The systematic study of the complete DNA sequences (genome) of organisms. [NIH]

Genotype: The genetic constitution of the individual; the characterization of the genes. [NIH]

Germ Layers: The three layers of cells comprising the early embryo. [NIH]

Germline mutation: A gene change in the body's reproductive cells (egg or sperm) that becomes incorporated into the DNA of every cell in the body of offspring; germline mutations are passed on from parents to offspring. Also called hereditary mutation. [NIH]

Gestation: The period of development of the young in viviparous animals, from the time of fertilization of the ovum until birth. [EU]

Giant Cells: Multinucleated masses produced by the fusion of many cells; often associated with viral infections. In AIDS, they are induced when the envelope glycoprotein of the HIV virus binds to the CD4 antigen of uninfected neighboring T4 cells. The resulting syncytium

leads to cell death and thus may account for the cytopathic effect of the virus. [NIH]

Gingivitis: Inflammation of the gingivae. Gingivitis associated with bony changes is referred to as periodontitis. Called also oulitis and ulitis. [EU]

Ginseng: An araliaceous genus of plants that contains a number of pharmacologically active agents used as stimulants, sedatives, and tonics, especially in traditional medicine. [NIH]

Gland: An organ that produces and releases one or more substances for use in the body. Some glands produce fluids that affect tissues or organs. Others produce hormones or participate in blood production. [NIH]

Gluconeogenesis: The process by which glucose is formed from a non-carbohydrate source. [NIH]

Glucose: D-Glucose. A primary source of energy for living organisms. It is naturally occurring and is found in fruits and other parts of plants in its free state. It is used therapeutically in fluid and nutrient replacement. [NIH]

Glucose Intolerance: A pathological state in which the fasting plasma glucose level is less than 140 mg per deciliter and the 30-, 60-, or 90-minute plasma glucose concentration following a glucose tolerance test exceeds 200 mg per deciliter. This condition is seen frequently in diabetes mellitus but also occurs with other diseases. [NIH]

Glucose tolerance: The power of the normal liver to absorb and store large quantities of glucose and the effectiveness of intestinal absorption of glucose. The glucose tolerance test is a metabolic test of carbohydrate tolerance that measures active insulin, a hepatic function based on the ability of the liver to absorb glucose. The test consists of ingesting 100 grams of glucose into a fasting stomach; blood sugar should return to normal in 2 to 21 hours after ingestion. [NIH]

Glucose Tolerance Test: Determination of whole blood or plasma sugar in a fasting state before and at prescribed intervals (usually 1/2 hr, 1 hr, 3 hr, 4 hr) after taking a specified amount (usually 100 gm orally) of glucose. [NIH]

Glucuronic Acid: Derivatives of uronic acid found throughout the plant and animal kingdoms. They detoxify drugs and toxins by conjugating with them to form glucuronides in the liver which are more water-soluble metabolites that can be easily eliminated from the body. [NIH]

Glutamic Acid: A non-essential amino acid naturally occurring in the L-form. Glutamic acid (glutamate) is the most common excitatory neurotransmitter in the central nervous system. [NIH]

Glutamine: A non-essential amino acid present abundantly throught the body and is involved in many metabolic processes. It is synthesized from glutamic acid and ammonia. It is the principal carrier of nitrogen in the body and is an important energy source for many cells. [NIH]

Glutathione Peroxidase: An enzyme catalyzing the oxidation of 2 moles of glutathione in the presence of hydrogen peroxide to yield oxidized glutathione and water. EC 1.11.1.9. [NIH]

Glycerol: A trihydroxy sugar alcohol that is an intermediate in carbohydrate and lipid metabolism. It is used as a solvent, emollient, pharmaceutical agent, and sweetening agent. [NIH]

Glycerophospholipids: Derivatives of phosphatidic acid in which the hydrophobic regions are composed of two fatty acids and a polar alcohol is joined to the C-3 position of glycerol through a phosphodiester bond. They are named according to their polar head groups, such as phosphatidylcholine and phosphatidylethanolamine. [NIH]

Glycine: A non-essential amino acid. It is found primarily in gelatin and silk fibroin and

used therapeutically as a nutrient. It is also a fast inhibitory neurotransmitter. [NIH]

Glycogen: A sugar stored in the liver and muscles. It releases glucose into the blood when cells need it for energy. Glycogen is the chief source of stored fuel in the body. [NIH]

Glycogen Storage Disease: A group of inherited metabolic disorders involving the enzymes responsible for the synthesis and degradation of glycogen. In some patients, prominent liver involvement is presented. In others, more generalized storage of glycogen occurs, sometimes with prominent cardiac involvement. [NIH]

Glycolysis: The pathway by which glucose is catabolized into two molecules of pyruvic acid with the generation of ATP. [NIH]

Glycoprotein: A protein that has sugar molecules attached to it. [NIH]

Glycosaminoglycan: A type of long, unbranched polysaccharide molecule. Glycosaminoglycans are major structural components of cartilage and are also found in the cornea of the eye. [NIH]

Goats: Any of numerous agile, hollow-horned ruminants of the genus Capra, closely related to the sheep. [NIH]

Gonadal: Pertaining to a gonad. [EU]

Governing Board: The group in which legal authority is vested for the control of health-related institutions and organizations. [NIH]

Grade: The grade of a tumor depends on how abnormal the cancer cells look under a microscope and how quickly the tumor is likely to grow and spread. Grading systems are different for each type of cancer. [NIH]

Grading: A system for classifying cancer cells in terms of how abnormal they appear when examined under a microscope. The objective of a grading system is to provide information about the probable growth rate of the tumor and its tendency to spread. The systems used to grade tumors vary with each type of cancer. Grading plays a role in treatment decisions. [NIH]

Graft: Healthy skin, bone, or other tissue taken from one part of the body and used to replace diseased or injured tissue removed from another part of the body. [NIH]

Grafting: The operation of transfer of tissue from one site to another. [NIH]

Gram-negative: Losing the stain or decolorized by alcohol in Gram's method of staining, a primary characteristic of bacteria having a cell wall composed of a thin layer of peptidoglycan covered by an outer membrane of lipoprotein and lipopolysaccharide. [EU]

Gram-positive: Retaining the stain or resisting decolorization by alcohol in Gram's method of staining, a primary characteristic of bacteria whose cell wall is composed of a thick layer of peptidologlycan with attached teichoic acids. [EU]

Granulocytes: Leukocytes with abundant granules in the cytoplasm. They are divided into three groups: neutrophils, eosinophils, and basophils. [NIH]

Grasses: A large family, Gramineae, of narrow-leaved herbaceous monocots. Many grasses produce highly allergenic pollens and are hosts to cattle parasites and toxic fungi. [NIH]

Gravidity: Pregnancy; the condition of being pregnant, without regard to the outcome. [EU]

Growth: The progressive development of a living being or part of an organism from its earliest stage to maturity. [NIH]

Growth factors: Substances made by the body that function to regulate cell division and cell survival. Some growth factors are also produced in the laboratory and used in biological therapy. [NIH]

Guanylate Cyclase: An enzyme that catalyzes the conversion of GTP to 3',5'-cyclic GMP and pyrophosphate. It also acts on ITP and dGTP. (From Enzyme Nomenclature, 1992) EC 4.6.1.2. [NIH]

Guideline Adherence: Conformity in fulfilling or following official, recognized, or institutional requirements, guidelines, recommendations, protocols, pathways, or other standards. [NIH]

Habitat: An area considered in terms of its environment, particularly as this determines the type and quality of the vegetation the area can carry. [NIH]

Habitual: Of the nature of a habit; according to habit; established by or repeated by force of habit, customary. [EU]

Haematuria: Blood in the urine. [EU]

Haemophilia: A haemorrhagic diathesis occurring in two main forms: 1. Haemophilia A (classic haemophilia, factor VIII deficiency), an X-linked disorder due to deficiency of coagulation factor VIII; 2. Haemophilia B (factor IX deficiency, Christmas disease), also X-linked, due to deficiency of coagulation factor IX. Both forms are determined by a mutant gene near the telomere of the long arm of the X chromosome (Xq), but a different loci, and are characterized by subcutaneous and intramuscular haemorrhages; bleeding from the mouth, gums, lips, and tongue; haematuria; and haemarthroses. [EU]

Half-Life: The time it takes for a substance (drug, radioactive nuclide, or other) to lose half of its pharmacologic, physiologic, or radiologic activity. [NIH]

Haploid: An organism with one basic chromosome set, symbolized by n; the normal condition of gametes in diploids. [NIH]

Haplotypes: The genetic constitution of individuals with respect to one member of a pair of allelic genes, or sets of genes that are closely linked and tend to be inherited together such as those of the major histocompatibility complex. [NIH]

Headache: Pain in the cranial region that may occur as an isolated and benign symptom or as a manifestation of a wide variety of conditions including subarachnoid hemorrhage; craniocerebral trauma; central nervous system infections; intracranial hypertension; and other disorders. In general, recurrent headaches that are not associated with a primary disease process are referred to as headache disorders (e.g., migraine). [NIH]

Health Behavior: Behaviors expressed by individuals to protect, maintain or promote their health status. For example, proper diet, and appropriate exercise are activities perceived to influence health status. Life style is closely associated with health behavior and factors influencing life style are socioeconomic, educational, and cultural. [NIH]

Health Care Costs: The actual costs of providing services related to the delivery of health care, including the costs of procedures, therapies, and medications. It is differentiated from health expenditures, which refers to the amount of money paid for the services, and from fees, which refers to the amount charged, regardless of cost. [NIH]

Health Care Reform: Innovation and improvement of the health care system by reappraisal, amendment of services, and removal of faults and abuses in providing and distributing health services to patients. It includes a re-alignment of health services and health insurance to maximum demographic elements (the unemployed, indigent, uninsured, elderly, inner cities, rural areas) with reference to coverage, hospitalization, pricing and cost containment, insurers' and employers' costs, pre-existing medical conditions, prescribed drugs, equipment, and services. [NIH]

Health Education: Education that increases the awareness and favorably influences the attitudes and knowledge relating to the improvement of health on a personal or community

basis. [NIH]

Health Expenditures: The amounts spent by individuals, groups, nations, or private or public organizations for total health care and/or its various components. These amounts may or may not be equivalent to the actual costs (health care costs) and may or may not be shared among the patient, insurers, and/or employers. [NIH]

Health Policy: Decisions, usually developed by government policymakers, for determining present and future objectives pertaining to the health care system. [NIH]

Health Promotion: Encouraging consumer behaviors most likely to optimize health potentials (physical and psychosocial) through health information, preventive programs, and access to medical care. [NIH]

Health Services: Services for the diagnosis and treatment of disease and the maintenance of health. [NIH]

Health Status: The level of health of the individual, group, or population as subjectively assessed by the individual or by more objective measures. [NIH]

Heart Arrest: Sudden and usually momentary cessation of the heart beat. This sudden cessation may, but not usually, lead to death, sudden, cardiac. [NIH]

Heart attack: A seizure of weak or abnormal functioning of the heart. [NIH]

Heart Catheterization: Procedure which includes placement of catheter, recording of intracardiac and intravascular pressure, obtaining blood samples for chemical analysis, and cardiac output measurement, etc. Specific angiographic injection techniques are also involved. [NIH]

Heart failure: Loss of pumping ability by the heart, often accompanied by fatigue, breathlessness, and excess fluid accumulation in body tissues. [NIH]

Heart Murmurs: Abnormal heart sounds heard during auscultation caused by alterations in the flow of blood into a chamber, through a valve, or by a valve opening or closing abnormally. They are classified by the time of occurrence during the cardiac cycle, the duration, and the intensity of the sound on a scale of I to V. [NIH]

Heart Sounds: The sounds heard over the cardiac region produced by the functioning of the heart. There are four distinct sounds: the first occurs at the beginning of systole and is heard as a "lubb" sound; the second is produced by the closing of the aortic and pulmonary valves and is heard as a "dupp" sound; the third is produced by vibrations of the ventricular walls when suddenly distended by the rush of blood from the atria; and the fourth is produced by atrial contraction and ventricular filling but is rarely audible in the normal heart. The physiological concept of heart sounds is differentiated from the pathological heart murmurs. [NIH]

Heart Transplantation: The transference of a heart from one human or animal to another. [NIH]

Heart Ventricle: The lower right and left chambers of the heart. The right pumps venous blood into the lungs and the left pumps oxygenated blood into the systemic arterial circulation. [NIH]

Heartbeat: One complete contraction of the heart. [NIH]

Hematocrit: Measurement of the volume of packed red cells in a blood specimen by centrifugation. The procedure is performed using a tube with graduated markings or with automated blood cell counters. It is used as an indicator of erythrocyte status in disease. For example, anemia shows a low hematocrit, polycythemia, high values. [NIH]

Hematoma: An extravasation of blood localized in an organ, space, or tissue. [NIH]

Heme: The color-furnishing portion of hemoglobin. It is found free in tissues and as the

prosthetic group in many hemeproteins. [NIH]

Hemochromatosis: A disease that occurs when the body absorbs too much iron. The body stores the excess iron in the liver, pancreas, and other organs. May cause cirrhosis of the liver. Also called iron overload disease. [NIH]

Hemodialysis: The use of a machine to clean wastes from the blood after the kidneys have failed. The blood travels through tubes to a dialyzer, which removes wastes and extra fluid. The cleaned blood then flows through another set of tubes back into the body. [NIH]

Hemodynamics: The movements of the blood and the forces involved in systemic or regional blood circulation. [NIH]

Hemoglobin: One of the fractions of glycosylated hemoglobin A1c. Glycosylated hemoglobin is formed when linkages of glucose and related monosaccharides bind to hemoglobin A and its concentration represents the average blood glucose level over the previous several weeks. HbA1c levels are used as a measure of long-term control of plasma glucose (normal, 4 to 6 percent). In controlled diabetes mellitus, the concentration of glycosylated hemoglobin A is within the normal range, but in uncontrolled cases the level may be 3 to 4 times the normal conentration. Generally, complications are substantially lower among patients with Hb levels of 7 percent or less than in patients with HbA1c levels of 9 percent or more. [NIH]

Hemoglobinopathies: A group of inherited disorders characterized by structural alterations within the hemoglobin molecule. [NIH]

Hemoglobinuria: The presence of free hemoglobin in the urine. [NIH]

Hemorrhage: Bleeding or escape of blood from a vessel. [NIH]

Hemorrhagic stroke: A disorder involving bleeding within ischemic brain tissue. Hemorrhagic stroke occurs when blood vessels that are damaged or dead from lack of blood supply (infarcted), located within an area of infarcted brain tissue, rupture and transform an "ischemic" stroke into a hemorrhagic stroke. Ischemia is inadequate tissue oxygenation caused by reduced blood flow; infarction is tissue death resulting from ischemia. Bleeding irritates the brain tissues, causing swelling (cerebral edema). Blood collects into a mass (hematoma). Both swelling and hematoma will compress and displace brain tissue. [NIH]

Hemostasis: The process which spontaneously arrests the flow of blood from vessels carrying blood under pressure. It is accomplished by contraction of the vessels, adhesion and aggregation of formed blood elements, and the process of blood or plasma coagulation. [NIH]

Heparin: Heparinic acid. A highly acidic mucopolysaccharide formed of equal parts of sulfated D-glucosamine and D-glucuronic acid with sulfaminic bridges. The molecular weight ranges from six to twenty thousand. Heparin occurs in and is obtained from liver, lung, mast cells, etc., of vertebrates. Its function is unknown, but it is used to prevent blood clotting in vivo and vitro, in the form of many different salts. [NIH]

Hepatic: Refers to the liver. [NIH]

Hepatitis: Inflammation of the liver and liver disease involving degenerative or necrotic alterations of hepatocytes. [NIH]

Hepatitis C: A form of hepatitis, similar to type B post-transfusion hepatitis, but caused by a virus which is serologically distinct from the agents of hepatitis A, B, and E, and which may persist in the blood of chronic asymptomatic carriers. Hepatitis C is parenterally transmitted and associated with transfusions and drug abuse. [NIH]

Hepatocyte: A liver cell. [NIH]

Hepatocyte Growth Factor: Multifunctional growth factor which regulates both cell growth

and cell motility. It exerts a strong mitogenic effect on hepatocytes and primary epithelial cells. Its receptor is proto-oncogene protein C-met. [NIH]

Hepatomegaly: Enlargement of the liver. [NIH]

Hereditary: Of, relating to, or denoting factors that can be transmitted genetically from one generation to another. [NIH]

Hereditary mutation: A gene change in the body's reproductive cells (egg or sperm) that becomes incorporated into the DNA of every cell in the body of offspring; hereditary mutations are passed on from parents to offspring. Also called germline mutation. [NIH]

Heredity: 1. The genetic transmission of a particular quality or trait from parent to offspring. 2. The genetic constitution of an individual. [EU]

Heterogeneity: The property of one or more samples or populations which implies that they are not identical in respect of some or all of their parameters, e. g. heterogeneity of variance. [NIH]

High blood cholesterol: Cholesterol is the most abundant steroid in animal tissues, especially in bile and gallstones. The relationship between the intake of cholesterol and its manufacture by the body to its utilization, sequestration, or excretion from the body is called the cholesterol balance. When cholesterol accumulates, the balance is positive; when it declines, the balance is negative. In 1993, the NHLBI National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults issued an updated set of recommendations for monitoring and treatment of blood cholesterol levels. The NCEP guidelines recommended that total cholesterol levels and subfractions of high-density lipoprotein (HDL) cholesterol be measured beginning at age 20 in all adults, with subsequent periodic screenings as needed. Even in the group of patients at lowest risk for coronary heart disease (total cholesterol 200 mg/dL and HDL 35 mg/dL), the NCEP recommended that rescreening take place at least once every 5 years or upon physical examination. [NIH]

Histology: The study of tissues and cells under a microscope. [NIH]

Histone Deacetylase: Hydrolyzes N-acetyl groups on histones. [NIH]

Homeobox: Distinctive sequence of DNA bases. [NIH]

Homicide: The killing of one person by another. [NIH]

Homodimer: Protein-binding "activation domains" always combine with identical proteins. [NIH]

Homologous: Corresponding in structure, position, origin, etc., as (a) the feathers of a bird and the scales of a fish, (b) antigen and its specific antibody, (c) allelic chromosomes. [EU]

Hormonal: Pertaining to or of the nature of a hormone. [EU]

Hormone: A substance in the body that regulates certain organs. Hormones such as gastrin help in breaking down food. Some hormones come from cells in the stomach and small intestine. [NIH]

Hormone Replacement Therapy: Therapeutic use of hormones to alleviate the effects of hormone deficiency. [NIH]

Hormone therapy: Treatment of cancer by removing, blocking, or adding hormones. Also called endocrine therapy. [NIH]

Hospice: Institution dedicated to caring for the terminally ill. [NIH]

Hospital Records: Compilations of data on hospital activities and programs; excludes patient medical records. [NIH]

Host: Any animal that receives a transplanted graft. [NIH]

Humoral: Of, relating to, proceeding from, or involving a bodily humour - now often used of endocrine factors as opposed to neural or somatic. [EU]

Humour: 1. A normal functioning fluid or semifluid of the body (as the blood, lymph or bile) especially of vertebrates. 2. A secretion that is itself an excitant of activity (as certain hormones). [EU]

Hybrid: Cross fertilization between two varieties or, more usually, two species of vines, see also crossing. [NIH]

Hybridization: The genetic process of crossbreeding to produce a hybrid. Hybrid nucleic acids can be formed by nucleic acid hybridization of DNA and RNA molecules. Protein hybridization allows for hybrid proteins to be formed from polypeptide chains. [NIH]

Hybridomas: Cells artificially created by fusion of activated lymphocytes with neoplastic cells. The resulting hybrid cells are cloned and produce pure or "monoclonal" antibodies or T-cell products, identical to those produced by the immunologically competent parent, and continually grow and divide as the neoplastic parent. [NIH]

Hydration: Combining with water. [NIH]

Hydrogen: The first chemical element in the periodic table. It has the atomic symbol H, atomic number 1, and atomic weight 1. It exists, under normal conditions, as a colorless, odorless, tasteless, diatomic gas. Hydrogen ions are protons. Besides the common H1 isotope, hydrogen exists as the stable isotope deuterium and the unstable, radioactive isotope tritium. [NIH]

Hydrogen Peroxide: A strong oxidizing agent used in aqueous solution as a ripening agent, bleach, and topical anti-infective. It is relatively unstable and solutions deteriorate over time unless stabilized by the addition of acetanilide or similar organic materials. [NIH]

Hydrolysis: The process of cleaving a chemical compound by the addition of a molecule of water. [NIH]

Hydrophobic: Not readily absorbing water, or being adversely affected by water, as a hydrophobic colloid. [EU]

Hydroxylysine: A hydroxylated derivative of the amino acid lysine that is present in certain collagens. [NIH]

Hydroxyproline: A hydroxylated form of the imino acid proline. A deficiency in ascorbic acid can result in impaired hydroxyproline formation. [NIH]

Hypercholesterolemia: Abnormally high levels of cholesterol in the blood. [NIH]

Hyperglycemia: Abnormally high blood sugar. [NIH]

Hyperhomocysteinemia: An inborn error of methionone metabolism which produces an excess of homocysteine in the blood. It is often caused by a deficiency of cystathionine beta-synthase and is a risk factor for coronary vascular disease. [NIH]

Hyperlipidemia: An excess of lipids in the blood. [NIH]

Hyperplasia: An increase in the number of cells in a tissue or organ, not due to tumor formation. It differs from hypertrophy, which is an increase in bulk without an increase in the number of cells. [NIH]

Hypersensitivity: Altered reactivity to an antigen, which can result in pathologic reactions upon subsequent exposure to that particular antigen. [NIH]

Hypertension: Persistently high arterial blood pressure. Currently accepted threshold levels are 140 mm Hg systolic and 90 mm Hg diastolic pressure. [NIH]

Hyperthyroidism: Excessive functional activity of the thyroid gland. [NIH]

Hypertriglyceridemia: Condition of elevated triglyceride concentration in the blood; an inherited form occurs in familial hyperlipoproteinemia IIb and hyperlipoproteinemia type IV. It has been linked to higher risk of heart disease and arteriosclerosis. [NIH]

Hypertrophic cardiomyopathy: Heart muscle disease that leads to thickening of the heart walls, interfering with the heart's ability to fill with and pump blood. [NIH]

Hypertrophy: General increase in bulk of a part or organ, not due to tumor formation, nor to an increase in the number of cells. [NIH]

Hypnotic: A drug that acts to induce sleep. [EU]

Hypoglycemia: Abnormally low blood sugar [NIH]

Hypoplasia: Incomplete development or underdevelopment of an organ or tissue. [EU]

Hypoplastic Left Heart Syndrome: A condition characterized by underdevelopment of the left cardiac chambers, atresia or stenosis of the aorta or mitral valve or both, and hypoplasia of the aorta. These anomalies are a common cause of heart failure in early infancy. [NIH]

Hypothalamic: Of or involving the hypothalamus. [EU]

Hypothalamus: Ventral part of the diencephalon extending from the region of the optic chiasm to the caudal border of the mammillary bodies and forming the inferior and lateral walls of the third ventricle. [NIH]

Hypoxia: Reduction of oxygen supply to tissue below physiological levels despite adequate perfusion of the tissue by blood. [EU]

Id: The part of the personality structure which harbors the unconscious instinctive desires and strivings of the individual. [NIH]

Idiopathic: Describes a disease of unknown cause. [NIH]

Ileum: The lower end of the small intestine. [NIH]

Imidazole: C3H4N2. The ring is present in polybenzimidazoles. [NIH]

Immune function: Production and action of cells that fight disease or infection. [NIH]

Immune response: The activity of the immune system against foreign substances (antigens). [NIH]

Immune Sera: Serum that contains antibodies. It is obtained from an animal that has been immunized either by antigen injection or infection with microorganisms containing the antigen. [NIH]

Immune system: The organs, cells, and molecules responsible for the recognition and disposal of foreign ("non-self") material which enters the body. [NIH]

Immunity: Nonsusceptibility to the invasive or pathogenic effects of foreign microorganisms or to the toxic effect of antigenic substances. [NIH]

Immunization: Deliberate stimulation of the host's immune response. Active immunization involves administration of antigens or immunologic adjuvants. Passive immunization involves administration of immune sera or lymphocytes or their extracts (e.g., transfer factor, immune RNA) or transplantation of immunocompetent cell producing tissue (thymus or bone marrow). [NIH]

Immunoassay: Immunochemical assay or detection of a substance by serologic or immunologic methods. Usually the substance being studied serves as antigen both in antibody production and in measurement of antibody by the test substance. [NIH]

Immunodeficiency: The decreased ability of the body to fight infection and disease. [NIH]

Immunodeficiency syndrome: The inability of the body to produce an immune response. [NIH]

Immunogenic: Producing immunity; evoking an immune response. [EU]

Immunoglobulin: A protein that acts as an antibody. [NIH]

Immunohistochemistry: Histochemical localization of immunoreactive substances using labeled antibodies as reagents. [NIH]

Immunologic: The ability of the antibody-forming system to recall a previous experience with an antigen and to respond to a second exposure with the prompt production of large amounts of antibody. [NIH]

Immunology: The study of the body's immune system. [NIH]

Immunophilin: A drug for the treatment of Parkinson's disease. [NIH]

Immunosuppressive: Describes the ability to lower immune system responses. [NIH]

Immunosuppressive Agents: Agents that suppress immune function by one of several mechanisms of action. Classical cytotoxic immunosuppressants act by inhibiting DNA synthesis. Others may act through activation of suppressor T-cell populations or by inhibiting the activation of helper cells. While immunosuppression has been brought about in the past primarily to prevent rejection of transplanted organs, new applications involving mediation of the effects of interleukins and other cytokines are emerging. [NIH]

Impairment: In the context of health experience, an impairment is any loss or abnormality of psychological, physiological, or anatomical structure or function. [NIH]

Implantation: The insertion or grafting into the body of biological, living, inert, or radioactive material. [EU]

Impotence: The inability to perform sexual intercourse. [NIH]

Impotent: Unable to have an erection adequate for sexual intercourse. [NIH]

In situ: In the natural or normal place; confined to the site of origin without invasion of neighbouring tissues. [EU]

In Situ Hybridization: A technique that localizes specific nucleic acid sequences within intact chromosomes, eukaryotic cells, or bacterial cells through the use of specific nucleic acid-labeled probes. [NIH]

In vitro: In the laboratory (outside the body). The opposite of in vivo (in the body). [NIH]

In vivo: In the body. The opposite of in vitro (outside the body or in the laboratory). [NIH]

Incision: A cut made in the body during surgery. [NIH]

Incontinence: Inability to control the flow of urine from the bladder (urinary incontinence) or the escape of stool from the rectum (fecal incontinence). [NIH]

Indicative: That indicates; that points out more or less exactly; that reveals fairly clearly. [EU]

Induction: The act or process of inducing or causing to occur, especially the production of a specific morphogenetic effect in the developing embryo through the influence of evocators or organizers, or the production of anaesthesia or unconsciousness by use of appropriate agents. [EU]

Infancy: The period of complete dependency prior to the acquisition of competence in walking, talking, and self-feeding. [NIH]

Infant Mortality: Perinatal, neonatal, and infant deaths in a given population. [NIH]

Infant, Newborn: An infant during the first month after birth. [NIH]

Infarction: A pathological process consisting of a sudden insufficient blood supply to an area, which results in necrosis of that area. It is usually caused by a thrombus, an embolus, or a vascular torsion. [NIH]

Infection: 1. Invasion and multiplication of microorganisms in body tissues, which may be clinically unapparent or result in local cellular injury due to competitive metabolism, toxins, intracellular replication, or antigen-antibody response. The infection may remain localized, subclinical, and temporary if the body's defensive mechanisms are effective. A local infection may persist and spread by extension to become an acute, subacute, or chronic clinical infection or disease state. A local infection may also become systemic when the microorganisms gain access to the lymphatic or vascular system. 2. An infectious disease. [EU]

Infertility: The diminished or absent ability to conceive or produce an offspring while sterility is the complete inability to conceive or produce an offspring. [NIH]

Inflammation: A pathological process characterized by injury or destruction of tissues caused by a variety of cytologic and chemical reactions. It is usually manifested by typical signs of pain, heat, redness, swelling, and loss of function. [NIH]

Influenza: An acute viral infection involving the respiratory tract. It is marked by inflammation of the nasal mucosa, the pharynx, and conjunctiva, and by headache and severe, often generalized, myalgia. [NIH]

Infusion: A method of putting fluids, including drugs, into the bloodstream. Also called intravenous infusion. [NIH]

Ingestion: Taking into the body by mouth [NIH]

Inhalation: The drawing of air or other substances into the lungs. [EU]

Initiation: Mutation induced by a chemical reactive substance causing cell changes; being a step in a carcinogenic process. [NIH]

Inotropic: Affecting the force or energy of muscular contractions. [EU]

Insight: The capacity to understand one's own motives, to be aware of one's own psychodynamics, to appreciate the meaning of symbolic behavior. [NIH]

Institutionalization: The caring for individuals in institutions and their adaptation to routines characteristic of the institutional environment, and/or their loss of adaptation to life outside the institution. [NIH]

Insulin: A protein hormone secreted by beta cells of the pancreas. Insulin plays a major role in the regulation of glucose metabolism, generally promoting the cellular utilization of glucose. It is also an important regulator of protein and lipid metabolism. Insulin is used as a drug to control insulin-dependent diabetes mellitus. [NIH]

Insulin-dependent diabetes mellitus: A disease characterized by high levels of blood glucose resulting from defects in insulin secretion, insulin action, or both. Autoimmune, genetic, and environmental factors are involved in the development of type I diabetes. [NIH]

Insulin-like: Muscular growth factor. [NIH]

Intensive Care: Advanced and highly specialized care provided to medical or surgical patients whose conditions are life-threatening and require comprehensive care and constant monitoring. It is usually administered in specially equipped units of a health care facility. [NIH]

Intercellular Adhesion Molecule-1: A cell-surface ligand with a role in leukocyte adhesion and inflammation. Its production is induced by gamma-interferon and it is required for neutrophil migration into inflamed tissue. [NIH]

Interleukin-1: A soluble factor produced by monocytes, macrophages, and other cells which activates T-lymphocytes and potentiates their response to mitogens or antigens. IL-1 consists of two distinct forms, IL-1 alpha and IL-1 beta which perform the same functions but are distinct proteins. The biological effects of IL-1 include the ability to replace macrophage

requirements for T-cell activation. The factor is distinct from interleukin-2. [NIH]

Interleukin-2: Chemical mediator produced by activated T lymphocytes and which regulates the proliferation of T cells, as well as playing a role in the regulation of NK cell activity. [NIH]

Interleukin-6: Factor that stimulates the growth and differentiation of human B-cells and is also a growth factor for hybridomas and plasmacytomas. It is produced by many different cells including T-cells, monocytes, and fibroblasts. [NIH]

Interleukin-8: A cytokine that activates neutrophils and attracts neutrophils and T-lymphocytes. It is released by several cell types including monocytes, macrophages, T-lymphocytes, fibroblasts, endothelial cells, and keratinocytes by an inflammatory stimulus. IL-8 is a member of the beta-thromboglobulin superfamily and structurally related to platelet factor 4. [NIH]

Interleukins: Soluble factors which stimulate growth-related activities of leukocytes as well as other cell types. They enhance cell proliferation and differentiation, DNA synthesis, secretion of other biologically active molecules and responses to immune and inflammatory stimuli. [NIH]

Intermittent: Occurring at separated intervals; having periods of cessation of activity. [EU]

Internal Medicine: A medical specialty concerned with the diagnosis and treatment of diseases of the internal organ systems of adults. [NIH]

Interstitial: Pertaining to or situated between parts or in the interspaces of a tissue. [EU]

Intervertebral: Situated between two contiguous vertebrae. [EU]

Intestinal: Having to do with the intestines. [NIH]

Intestines: The section of the alimentary canal from the stomach to the anus. It includes the large intestine and small intestine. [NIH]

Intoxication: Poisoning, the state of being poisoned. [EU]

Intracellular: Inside a cell. [NIH]

Intramuscular: IM. Within or into muscle. [NIH]

Intraocular: Within the eye. [EU]

Intraocular pressure: Pressure of the fluid inside the eye; normal IOP varies among individuals. [NIH]

Intravascular: Within a vessel or vessels. [EU]

Intravenous: IV. Into a vein. [NIH]

Intrinsic: Situated entirely within or pertaining exclusively to a part. [EU]

Intubation: Introduction of a tube into a hollow organ to restore or maintain patency if obstructed. It is differentiated from catheterization in that the insertion of a catheter is usually performed for the introducing or withdrawing of fluids from the body. [NIH]

Invasive: 1. Having the quality of invasiveness. 2. Involving puncture or incision of the skin or insertion of an instrument or foreign material into the body; said of diagnostic techniques. [EU]

Involuntary: Reaction occurring without intention or volition. [NIH]

Iodine: A nonmetallic element of the halogen group that is represented by the atomic symbol I, atomic number 53, and atomic weight of 126.90. It is a nutritionally essential element, especially important in thyroid hormone synthesis. In solution, it has anti-infective properties and is used topically. [NIH]

Ion Channels: Gated, ion-selective glycoproteins that traverse membranes. The stimulus for channel gating can be a membrane potential, drug, transmitter, cytoplasmic messenger, or a mechanical deformation. Ion channels which are integral parts of ionotropic neurotransmitter receptors are not included. [NIH]

Ionizing: Radiation comprising charged particles, e. g. electrons, protons, alpha-particles, etc., having sufficient kinetic energy to produce ionization by collision. [NIH]

Ions: An atom or group of atoms that have a positive or negative electric charge due to a gain (negative charge) or loss (positive charge) of one or more electrons. Atoms with a positive charge are known as cations; those with a negative charge are anions. [NIH]

Iris: The most anterior portion of the uveal layer, separating the anterior chamber from the posterior. It consists of two layers - the stroma and the pigmented epithelium. Color of the iris depends on the amount of melanin in the stroma on reflection from the pigmented epithelium. [NIH]

Ischemia: Deficiency of blood in a part, due to functional constriction or actual obstruction of a blood vessel. [EU]

Ischemic stroke: A condition in which the blood supply to part of the brain is cut off. Also called "plug-type" strokes. Blocked arteries starve areas of the brain controlling sight, speech, sensation, and movement so that these functions are partially or completely lost. Ischemic stroke is the most common type of stroke, accounting for 80 percent of all strokes. Most ischemic strokes are caused by a blood clot called a thrombus, which blocks blood flow in the arteries feeding the brain, usually the carotid artery in the neck, the major vessel bringing blood to the brain. When it becomes blocked, the risk of stroke is very high. [NIH]

Isoflavones: 3-Phenylchromones. Isomeric form of flavones in which the benzene group is attached to the 3 position of the benzopyran ring instead of the 2 position. [NIH]

Isoflurane: A stable, non-explosive inhalation anesthetic, relatively free from significant side effects. [NIH]

Isosorbide: 1,4:3,6-Dianhydro D-glucitol. Chemically inert osmotic diuretic used mainly to treat hydrocephalus; also used in glaucoma. [NIH]

Isosorbide Dinitrate: A vasodilator used in the treatment of angina. Its actions are similar to nitroglycerin but with a slower onset of action. [NIH]

Joint: The point of contact between elements of an animal skeleton with the parts that surround and support it. [NIH]

Kb: A measure of the length of DNA fragments, 1 Kb = 1000 base pairs. The largest DNA fragments are up to 50 kilobases long. [NIH]

Keratinocytes: Epidermal cells which synthesize keratin and undergo characteristic changes as they move upward from the basal layers of the epidermis to the cornified (horny) layer of the skin. Successive stages of differentiation of the keratinocytes forming the epidermal layers are basal cell, spinous or prickle cell, and the granular cell. [NIH]

Kidney Disease: Any one of several chronic conditions that are caused by damage to the cells of the kidney. People who have had diabetes for a long time may have kidney damage. Also called nephropathy. [NIH]

Kidney Failure: The inability of a kidney to excrete metabolites at normal plasma levels under conditions of normal loading, or the inability to retain electrolytes under conditions of normal intake. In the acute form (kidney failure, acute), it is marked by uremia and usually by oliguria or anuria, with hyperkalemia and pulmonary edema. The chronic form (kidney failure, chronic) is irreversible and requires hemodialysis. [NIH]

Kidney Failure, Acute: A clinical syndrome characterized by a sudden decrease in

glomerular filtration rate, often to values of less than 1 to 2 ml per minute. It is usually associated with oliguria (urine volumes of less than 400 ml per day) and is always associated with biochemical consequences of the reduction in glomerular filtration rate such as a rise in blood urea nitrogen (BUN) and serum creatinine concentrations. [NIH]

Kidney Failure, Chronic: An irreversible and usually progressive reduction in renal function in which both kidneys have been damaged by a variety of diseases to the extent that they are unable to adequately remove the metabolic products from the blood and regulate the body's electrolyte composition and acid-base balance. Chronic kidney failure requires hemodialysis or surgery, usually kidney transplantation. [NIH]

Kinetics: The study of rate dynamics in chemical or physical systems. [NIH]

Labile: 1. Gliding; moving from point to point over the surface; unstable; fluctuating. 2. Chemically unstable. [EU]

Lactation: The period of the secretion of milk. [EU]

Laminin: Large, noncollagenous glycoprotein with antigenic properties. It is localized in the basement membrane lamina lucida and functions to bind epithelial cells to the basement membrane. Evidence suggests that the protein plays a role in tumor invasion. [NIH]

Large Intestine: The part of the intestine that goes from the cecum to the rectum. The large intestine absorbs water from stool and changes it from a liquid to a solid form. The large intestine is 5 feet long and includes the appendix, cecum, colon, and rectum. Also called colon. [NIH]

Latent: Phoria which occurs at one distance or another and which usually has no troublesome effect. [NIH]

Lectin: A complex molecule that has both protein and sugars. Lectins are able to bind to the outside of a cell and cause biochemical changes in it. Lectins are made by both animals and plants. [NIH]

Left ventricular assist device: A mechanical device used to increase the heart's pumping ability. [NIH]

Lenses: Pieces of glass or other transparent materials used for magnification or increased visual acuity. [NIH]

Lethal: Deadly, fatal. [EU]

Leukapheresis: The preparation of leukocyte concentrates with the return of red cells and leukocyte-poor plasma to the donor. [NIH]

Leukemia: Cancer of blood-forming tissue. [NIH]

Leukocytes: White blood cells. These include granular leukocytes (basophils, eosinophils, and neutrophils) as well as non-granular leukocytes (lymphocytes and monocytes). [NIH]

Leukotrienes: A family of biologically active compounds derived from arachidonic acid by oxidative metabolism through the 5-lipoxygenase pathway. They participate in host defense reactions and pathophysiological conditions such as immediate hypersensitivity and inflammation. They have potent actions on many essential organs and systems, including the cardiovascular, pulmonary, and central nervous system as well as the gastrointestinal tract and the immune system. [NIH]

Library Services: Services offered to the library user. They include reference and circulation. [NIH]

Life cycle: The successive stages through which an organism passes from fertilized ovum or spore to the fertilized ovum or spore of the next generation. [NIH]

Life Expectancy: A figure representing the number of years, based on known statistics, to

which any person of a given age may reasonably expect to live. [NIH]

Ligament: A band of fibrous tissue that connects bones or cartilages, serving to support and strengthen joints. [EU]

Ligands: A RNA simulation method developed by the MIT. [NIH]

Ligation: Application of a ligature to tie a vessel or strangulate a part. [NIH]

Linkage: The tendency of two or more genes in the same chromosome to remain together from one generation to the next more frequently than expected according to the law of independent assortment. [NIH]

Lip: Either of the two fleshy, full-blooded margins of the mouth. [NIH]

Lipaemia: The presence of an excess of fats or lipids in the blood. [NIH]

Lipase: An enzyme of the hydrolase class that catalyzes the reaction of triacylglycerol and water to yield diacylglycerol and a fatty acid anion. It is produced by glands on the tongue and by the pancreas and initiates the digestion of dietary fats. (From Dorland, 27th ed) EC 3.1.1.3. [NIH]

Lipid: Fat. [NIH]

Lipid A: Lipid A is the biologically active component of lipopolysaccharides. It shows strong endotoxic activity and exhibits immunogenic properties. [NIH]

Lipid Peroxidation: Peroxidase catalyzed oxidation of lipids using hydrogen peroxide as an electron acceptor. [NIH]

Lipolysis: The hydrolysis of lipids. [NIH]

Lipopolysaccharide: Substance consisting of polysaccaride and lipid. [NIH]

Lipoprotein: Any of the lipid-protein complexes in which lipids are transported in the blood; lipoprotein particles consist of a spherical hydrophobic core of triglycerides or cholesterol esters surrounded by an amphipathic monolayer of phospholipids, cholesterol, and apolipoproteins; the four principal classes are high-density, low-density, and very-low-density lipoproteins and chylomicrons. [EU]

Lipoprotein Lipase: An enzyme of the hydrolase class that catalyzes the reaction of triacylglycerol and water to yield diacylglycerol and a fatty acid anion. The enzyme hydrolyzes triacylglycerols in chylomicrons, very-low-density lipoproteins, low-density lipoproteins, and diacylglycerols. It occurs on capillary endothelial surfaces, especially in mammary, muscle, and adipose tissue. Genetic deficiency of the enzyme causes familial hyperlipoproteinemia Type I. (Dorland, 27th ed) EC 3.1.1.34. [NIH]

Lipoprotein(a): A family of lipoprotein particles varying in density and size depending on the protein-lipid ratio and the protein composition. These particles consist of apolipoprotein B-100 covalently linked to apolipoprotein-a by one or two disulfide bonds. There is a correlation between high plasma levels of this lipoprotein and increased risk for atherosclerotic cardiovascular disease. [NIH]

Lithium: An element in the alkali metals family. It has the atomic symbol Li, atomic number 3, and atomic weight 6.94. Salts of lithium are used in treating manic-depressive disorders. [NIH]

Liver: A large, glandular organ located in the upper abdomen. The liver cleanses the blood and aids in digestion by secreting bile. [NIH]

Liver scan: An image of the liver created on a computer screen or on film. A radioactive substance is injected into a blood vessel and travels through the bloodstream. It collects in the liver, especially in abnormal areas, and can be detected by the scanner. [NIH]

Localization: The process of determining or marking the location or site of a lesion or

disease. May also refer to the process of keeping a lesion or disease in a specific location or site. [NIH]

Localized: Cancer which has not metastasized yet. [NIH]

Locomotion: Movement or the ability to move from one place or another. It can refer to humans, vertebrate or invertebrate animals, and microorganisms. [NIH]

Longitudinal Studies: Studies in which variables relating to an individual or group of individuals are assessed over a period of time. [NIH]

Longitudinal study: Also referred to as a "cohort study" or "prospective study"; the analytic method of epidemiologic study in which subsets of a defined population can be identified who are, have been, or in the future may be exposed or not exposed, or exposed in different degrees, to a factor or factors hypothesized to influence the probability of occurrence of a given disease or other outcome. The main feature of this type of study is to observe large numbers of subjects over an extended time, with comparisons of incidence rates in groups that differ in exposure levels. [NIH]

Long-Term Care: Care over an extended period, usually for a chronic condition or disability, requiring periodic, intermittent, or continuous care. [NIH]

Lovastatin: A fungal metabolite isolated from cultures of Aspergillus terreus. The compound is a potent anticholesteremic agent. It inhibits 3-hydroxy-3-methylglutaryl coenzyme A reductase (hydroxymethylglutaryl CoA reductases), which is the rate-limiting enzyme in cholesterol biosynthesis. It also stimulates the production of low-density lipoprotein receptors in the liver. [NIH]

Low vision: Visual loss that cannot be corrected with eyeglasses or contact lenses and interferes with daily living activities. [NIH]

Low-density lipoprotein: Lipoprotein that contains most of the cholesterol in the blood. LDL carries cholesterol to the tissues of the body, including the arteries. A high level of LDL increases the risk of heart disease. LDL typically contains 60 to 70 percent of the total serum cholesterol and both are directly correlated with CHD risk. [NIH]

Lupus: A form of cutaneous tuberculosis. It is seen predominantly in women and typically involves the nasal, buccal, and conjunctival mucosa. [NIH]

Lymph: The almost colorless fluid that travels through the lymphatic system and carries cells that help fight infection and disease. [NIH]

Lymph node: A rounded mass of lymphatic tissue that is surrounded by a capsule of connective tissue. Also known as a lymph gland. Lymph nodes are spread out along lymphatic vessels and contain many lymphocytes, which filter the lymphatic fluid (lymph). [NIH]

Lymphadenopathy: Disease or swelling of the lymph nodes. [NIH]

Lymphatic: The tissues and organs, including the bone marrow, spleen, thymus, and lymph nodes, that produce and store cells that fight infection and disease. [NIH]

Lymphatic system: The tissues and organs that produce, store, and carry white blood cells that fight infection and other diseases. This system includes the bone marrow, spleen, thymus, lymph nodes and a network of thin tubes that carry lymph and white blood cells. These tubes branch, like blood vessels, into all the tissues of the body. [NIH]

Lymphocyte: A white blood cell. Lymphocytes have a number of roles in the immune system, including the production of antibodies and other substances that fight infection and diseases. [NIH]

Lymphoid: Referring to lymphocytes, a type of white blood cell. Also refers to tissue in which lymphocytes develop. [NIH]

Lymphoma: A general term for various neoplastic diseases of the lymphoid tissue. [NIH]

Lysine: An essential amino acid. It is often added to animal feed. [NIH]

Macronutrients: Nutrients in the diet that are the key sources of energy, namely protein, fat, and carbohydrates. [NIH]

Macrophage: A type of white blood cell that surrounds and kills microorganisms, removes dead cells, and stimulates the action of other immune system cells. [NIH]

Macula: A stain, spot, or thickening. Often used alone to refer to the macula retinae. [EU]

Macula Lutea: An oval area in the retina, 3 to 5 mm in diameter, usually located temporal to the superior pole of the eye and slightly below the level of the optic disk. [NIH]

Macular Degeneration: Degenerative changes in the macula lutea of the retina. [NIH]

Magnetic Resonance Imaging: Non-invasive method of demonstrating internal anatomy based on the principle that atomic nuclei in a strong magnetic field absorb pulses of radiofrequency energy and emit them as radiowaves which can be reconstructed into computerized images. The concept includes proton spin tomographic techniques. [NIH]

Magnetic Resonance Spectroscopy: Spectroscopic method of measuring the magnetic moment of elementary particles such as atomic nuclei, protons or electrons. It is employed in clinical applications such as NMR Tomography (magnetic resonance imaging). [NIH]

Major Histocompatibility Complex: The genetic region which contains the loci of genes which determine the structure of the serologically defined (SD) and lymphocyte-defined (LD) transplantation antigens, genes which control the structure of the immune response-associated (Ia) antigens, the immune response (Ir) genes which control the ability of an animal to respond immunologically to antigenic stimuli, and genes which determine the structure and/or level of the first four components of complement. [NIH]

Malabsorption: Impaired intestinal absorption of nutrients. [EU]

Malformation: A morphologic defect resulting from an intrinsically abnormal developmental process. [EU]

Malignant: Cancerous; a growth with a tendency to invade and destroy nearby tissue and spread to other parts of the body. [NIH]

Malignant tumor: A tumor capable of metastasizing. [NIH]

Malnutrition: A condition caused by not eating enough food or not eating a balanced diet. [NIH]

Mammary: Pertaining to the mamma, or breast. [EU]

Mammogram: An x-ray of the breast. [NIH]

Manic: Affected with mania. [EU]

Manic-depressive psychosis: One of a group of psychotic reactions, fundamentally marked by severe mood swings and a tendency to remission and recurrence. [NIH]

Manifest: Being the part or aspect of a phenomenon that is directly observable : concretely expressed in behaviour. [EU]

Man-made: Ionizing radiation emitted by artificial or concentrated natural, radioactive material or resulting from the operation of high voltage apparatus, such as X-ray apparatus or particle accelerators, of nuclear reactors, or from nuclear explosions. [NIH]

Marital Status: A demographic parameter indicating a person's status with respect to marriage, divorce, widowhood, singleness, etc. [NIH]

Mass Media: Instruments or technological means of communication that reach large numbers of people with a common message: press, radio, television, etc. [NIH]

Maternal Mortality: Maternal deaths resulting from complications of pregnancy and childbirth in a given population. [NIH]

Maxillary: Pertaining to the maxilla : the irregularly shaped bone that with its fellow forms the upper jaw. [EU]

Meat: The edible portions of any animal used for food including domestic mammals (the major ones being cattle, swine, and sheep) along with poultry, fish, shellfish, and game. [NIH]

Meat Products: Articles of food which are derived by a process of manufacture from any portion of carcasses of any animal used for food (e.g., head cheese, sausage, scrapple). [NIH]

Medial: Lying near the midsaggital plane of the body; opposed to lateral. [NIH]

Mediate: Indirect; accomplished by the aid of an intervening medium. [EU]

Mediator: An object or substance by which something is mediated, such as (1) a structure of the nervous system that transmits impulses eliciting a specific response; (2) a chemical substance (transmitter substance) that induces activity in an excitable tissue, such as nerve or muscle; or (3) a substance released from cells as the result of the interaction of antigen with antibody or by the action of antigen with a sensitized lymphocyte. [EU]

Medical Records: Recording of pertinent information concerning patient's illness or illnesses. [NIH]

MEDLINE: An online database of MEDLARS, the computerized bibliographic Medical Literature Analysis and Retrieval System of the National Library of Medicine. [NIH]

Megakaryocytes: Very large bone marrow cells which release mature blood platelets. [NIH]

Megaloblastic: A large abnormal red blood cell appearing in the blood in pernicious anaemia. [EU]

Melanin: The substance that gives the skin its color. [NIH]

Melanocytes: Epidermal dendritic pigment cells which control long-term morphological color changes by alteration in their number or in the amount of pigment they produce and store in the pigment containing organelles called melanosomes. Melanophores are larger cells which do not exist in mammals. [NIH]

Melanoma: A form of skin cancer that arises in melanocytes, the cells that produce pigment. Melanoma usually begins in a mole. [NIH]

Membrane: A very thin layer of tissue that covers a surface. [NIH]

Membrane Lipids: Lipids, predominantly phospholipids, cholesterol and small amounts of glycolipids found in membranes including cellular and intracellular membranes. These lipids may be arranged in bilayers in the membranes with integral proteins between the layers and peripheral proteins attached to the outside. Membrane lipids are required for active transport, several enzymatic activities and membrane formation. [NIH]

Memory: Complex mental function having four distinct phases: (1) memorizing or learning, (2) retention, (3) recall, and (4) recognition. Clinically, it is usually subdivided into immediate, recent, and remote memory. [NIH]

Meninges: The three membranes that cover and protect the brain and spinal cord. [NIH]

Menopause: Permanent cessation of menstruation. [NIH]

Menstrual Cycle: The period of the regularly recurring physiologic changes in the endometrium occurring during the reproductive period in human females and some primates and culminating in partial sloughing of the endometrium (menstruation). [NIH]

Menstruation: The normal physiologic discharge through the vagina of blood and mucosal

tissues from the nonpregnant uterus. [NIH]

Mental: Pertaining to the mind; psychic. 2. (L. mentum chin) pertaining to the chin. [EU]

Mental Disorders: Psychiatric illness or diseases manifested by breakdowns in the adaptational process expressed primarily as abnormalities of thought, feeling, and behavior producing either distress or impairment of function. [NIH]

Mental Health: The state wherein the person is well adjusted. [NIH]

Mental Retardation: Refers to sub-average general intellectual functioning which originated during the developmental period and is associated with impairment in adaptive behavior. [NIH]

Mentors: Senior professionals who provide guidance, direction and support to those persons desirous of improvement in academic positions, administrative positions or other career development situations. [NIH]

Mercury: A silver metallic element that exists as a liquid at room temperature. It has the atomic symbol Hg (from hydrargyrum, liquid silver), atomic number 80, and atomic weight 200.59. Mercury is used in many industrial applications and its salts have been employed therapeutically as purgatives, antisyphilitics, disinfectants, and astringents. It can be absorbed through the skin and mucous membranes which leads to mercury poisoning. Because of its toxicity, the clinical use of mercury and mercurials is diminishing. [NIH]

Mesenchymal: Refers to cells that develop into connective tissue, blood vessels, and lymphatic tissue. [NIH]

Mesoderm: The middle germ layer of the embryo. [NIH]

Meta-Analysis: A quantitative method of combining the results of independent studies (usually drawn from the published literature) and synthesizing summaries and conclusions which may be used to evaluate therapeutic effectiveness, plan new studies, etc., with application chiefly in the areas of research and medicine. [NIH]

Metabolic disorder: A condition in which normal metabolic processes are disrupted, usually because of a missing enzyme. [NIH]

Metabolite: Any substance produced by metabolism or by a metabolic process. [EU]

Metastasis: The spread of cancer from one part of the body to another. Tumors formed from cells that have spread are called "secondary tumors" and contain cells that are like those in the original (primary) tumor. The plural is metastases. [NIH]

Methamphetamine: A central nervous system stimulant and sympathomimetic with actions and uses similar to dextroamphetamine. The smokable form is a drug of abuse and is referred to as crank, crystal, crystal meth, ice, and speed. [NIH]

Methylprednisolone: (6 alpha,11 beta)-11,17,21-Trihydroxy-6-methylpregna-1,4-diene-3,2-dione. A prednisolone derivative which has pharmacological actions similar to prednisolone. [NIH]

Methyltransferase: A drug-metabolizing enzyme. [NIH]

Metoprolol: Adrenergic beta-1-blocking agent with no stimulatory action. It is less bound to plasma albumin than alprenolol and may be useful in angina pectoris, hypertension, or cardiac arrhythmias. [NIH]

MI: Myocardial infarction. Gross necrosis of the myocardium as a result of interruption of the blood supply to the area; it is almost always caused by atherosclerosis of the coronary arteries, upon which coronary thrombosis is usually superimposed. [NIH]

Mice Minute Virus: The type species of parvovirus prevalent in mouse colonies and found as a contaminant of many transplanted tumors or leukemias. [NIH]

Microbe: An organism which cannot be observed with the naked eye; e. g. unicellular animals, lower algae, lower fungi, bacteria. [NIH]

Microbiology: The study of microorganisms such as fungi, bacteria, algae, archaea, and viruses. [NIH]

Microcalcifications: Tiny deposits of calcium in the breast that cannot be felt but can be detected on a mammogram. A cluster of these very small specks of calcium may indicate that cancer is present. [NIH]

Microcirculation: The vascular network lying between the arterioles and venules; includes capillaries, metarterioles and arteriovenous anastomoses. Also, the flow of blood through this network. [NIH]

Micronutrients: Essential dietary elements or organic compounds that are required in only small quantities for normal physiologic processes to occur. [NIH]

Microorganism: An organism that can be seen only through a microscope. Microorganisms include bacteria, protozoa, algae, and fungi. Although viruses are not considered living organisms, they are sometimes classified as microorganisms. [NIH]

Microscopy: The application of microscope magnification to the study of materials that cannot be properly seen by the unaided eye. [NIH]

Microsomal: Of or pertaining to microsomes : vesicular fragments of endoplasmic reticulum formed after disruption and centrifugation of cells. [EU]

Midaxillary line: An imaginary vertical line that passes midway between the anterior and posterior axillary (armpit) folds. [NIH]

Midazolam: A short-acting compound, water-soluble at pH less than 4 and lipid-soluble at physiological pH. It is a hypnotic-sedative drug with anxiolytic and amnestic properties. It is used for sedation in dentistry, cardiac surgery, endoscopic procedures, as preanesthetic medication, and as an adjunct to local anesthesia. Because of its short duration and cardiorespiratory stability, it is particularly useful in poor-risk, elderly, and cardiac patients. [NIH]

Migration: The systematic movement of genes between populations of the same species, geographic race, or variety. [NIH]

Milliliter: A measure of volume for a liquid. A milliliter is approximately 950-times smaller than a quart and 30-times smaller than a fluid ounce. A milliliter of liquid and a cubic centimeter (cc) of liquid are the same. [NIH]

Millimeter: A measure of length. A millimeter is approximately 26-times smaller than an inch. [NIH]

Milrinone: A positive inotropic cardiotonic agent with vasodilator properties. It inhibits cAMP phosphodiesterase activity in myocardium and vascular smooth muscle. Milrinone is a derivative of amrinone and has 20-30 times the ionotropic potency of amrinone. [NIH]

Mineralization: The action of mineralizing; the state of being mineralized. [EU]

Mitosis: A method of indirect cell division by means of which the two daughter nuclei normally receive identical complements of the number of chromosomes of the somatic cells of the species. [NIH]

Mitral Valve: The valve between the left atrium and left ventricle of the heart. [NIH]

Mobility: Capability of movement, of being moved, or of flowing freely. [EU]

Mobilization: The process of making a fixed part or stored substance mobile, as by separating a part from surrounding structures to make it accessible for an operative procedure or by causing release into the circulation for body use of a substance stored in the

body. [EU]

Modeling: A treatment procedure whereby the therapist presents the target behavior which the learner is to imitate and make part of his repertoire. [NIH]

Modification: A change in an organism, or in a process in an organism, that is acquired from its own activity or environment. [NIH]

Modulator: A specific inductor that brings out characteristics peculiar to a definite region. [EU]

Molecular: Of, pertaining to, or composed of molecules : a very small mass of matter. [EU]

Molecular mass: The sum of the atomic masses of all atoms in a molecule, based on a scale in which the atomic masses of hydrogen, carbon, nitrogen, and oxygen are 1, 12, 14, and 16, respectively. For example, the molecular mass of water, which has two atoms of hydrogen and one atom of oxygen, is 18 (i.e., 2 + 16). [NIH]

Molecular Structure: The location of the atoms, groups or ions relative to one another in a molecule, as well as the number, type and location of covalent bonds. [NIH]

Molecule: A chemical made up of two or more atoms. The atoms in a molecule can be the same (an oxygen molecule has two oxygen atoms) or different (a water molecule has two hydrogen atoms and one oxygen atom). Biological molecules, such as proteins and DNA, can be made up of many thousands of atoms. [NIH]

Monitor: An apparatus which automatically records such physiological signs as respiration, pulse, and blood pressure in an anesthetized patient or one undergoing surgical or other procedures. [NIH]

Monoclonal: An antibody produced by culturing a single type of cell. It therefore consists of a single species of immunoglobulin molecules. [NIH]

Monoclonal antibodies: Laboratory-produced substances that can locate and bind to cancer cells wherever they are in the body. Many monoclonal antibodies are used in cancer detection or therapy; each one recognizes a different protein on certain cancer cells. Monoclonal antibodies can be used alone, or they can be used to deliver drugs, toxins, or radioactive material directly to a tumor. [NIH]

Monocyte: A type of white blood cell. [NIH]

Monogenic: A human disease caused by a mutation in a single gene. [NIH]

Mononuclear: A cell with one nucleus. [NIH]

Monosomy: The condition in which one chromosome of a pair is missing. In a normally diploid cell it is represented symbolically as 2N-1. [NIH]

Mood Disorders: Those disorders that have a disturbance in mood as their predominant feature. [NIH]

Morphogenesis: The development of the form of an organ, part of the body, or organism. [NIH]

Morphological: Relating to the configuration or the structure of live organs. [NIH]

Morphology: The science of the form and structure of organisms (plants, animals, and other forms of life). [NIH]

Motility: The ability to move spontaneously. [EU]

Mucolytic: Destroying or dissolving mucin; an agent that so acts : a mucopolysaccharide or glycoprotein, the chief constituent of mucus. [EU]

Mucosa: A mucous membrane, or tunica mucosa. [EU]

Multicenter Studies: Controlled studies which are planned and carried out by several

cooperating institutions to assess certain variables and outcomes in specific patient populations, for example, a multicenter study of congenital anomalies in children. [NIH]

Multicenter study: A clinical trial that is carried out at more than one medical institution. [NIH]

Multidose: Occurring in, or using multiple doses. [EU]

Multivariate Analysis: A set of techniques used when variation in several variables has to be studied simultaneously. In statistics, multivariate analysis is interpreted as any analytic method that allows simultaneous study of two or more dependent variables. [NIH]

Muscle Contraction: A process leading to shortening and/or development of tension in muscle tissue. Muscle contraction occurs by a sliding filament mechanism whereby actin filaments slide inward among the myosin filaments. [NIH]

Muscle Fibers: Large single cells, either cylindrical or prismatic in shape, that form the basic unit of muscle tissue. They consist of a soft contractile substance enclosed in a tubular sheath. [NIH]

Muscle Hypertonia: Abnormal increase in skeletal or smooth muscle tone. Skeletal muscle hypertonicity may be associated with pyramidal tract lesions or basal ganglia diseases. [NIH]

Muscular Atrophy: Derangement in size and number of muscle fibers occurring with aging, reduction in blood supply, or following immobilization, prolonged weightlessness, malnutrition, and particularly in denervation. [NIH]

Muscular Dystrophies: A general term for a group of inherited disorders which are characterized by progressive degeneration of skeletal muscles. [NIH]

Myalgia: Pain in a muscle or muscles. [EU]

Mydriatic: 1. Dilating the pupil. 2. Any drug that dilates the pupil. [EU]

Myocardial infarction: Gross necrosis of the myocardium as a result of interruption of the blood supply to the area; it is almost always caused by atherosclerosis of the coronary arteries, upon which coronary thrombosis is usually superimposed. [NIH]

Myocardial Ischemia: A disorder of cardiac function caused by insufficient blood flow to the muscle tissue of the heart. The decreased blood flow may be due to narrowing of the coronary arteries (coronary arteriosclerosis), to obstruction by a thrombus (coronary thrombosis), or less commonly, to diffuse narrowing of arterioles and other small vessels within the heart. Severe interruption of the blood supply to the myocardial tissue may result in necrosis of cardiac muscle (myocardial infarction). [NIH]

Myocardial Reperfusion: Generally, restoration of blood supply to heart tissue which is ischemic due to decrease in normal blood supply. The decrease may result from any source including atherosclerotic obstruction, narrowing of the artery, or surgical clamping. Reperfusion can be induced to treat ischemia. Methods include chemical dissolution of an occluding thrombus, administration of vasodilator drugs, angioplasty, catheterization, and artery bypass graft surgery. However, it is thought that reperfusion can itself further damage the ischemic tissue, causing myocardial reperfusion injury. [NIH]

Myocardial Reperfusion Injury: Functional, metabolic, or structural changes in ischemic heart muscle thought to result from reperfusion to the ischemic areas. Changes can be fatal to muscle cells and may include edema with explosive cell swelling and disintegration, sarcolemma disruption, fragmentation of mitochondria, contraction band necrosis, enzyme washout, and calcium overload. Other damage may include hemorrhage and ventricular arrhythmias. One possible mechanism of damage is thought to be oxygen free radicals. Treatment currently includes the introduction of scavengers of oxygen free radicals, and injury is thought to be prevented by warm blood cardioplegic infusion prior to reperfusion. [NIH]

Myocarditis: Inflammation of the myocardium; inflammation of the muscular walls of the heart. [EU]

Myocardium: The muscle tissue of the heart composed of striated, involuntary muscle known as cardiac muscle. [NIH]

Myosin: Chief protein in muscle and the main constituent of the thick filaments of muscle fibers. In conjunction with actin, it is responsible for the contraction and relaxation of muscles. [NIH]

Myotonic Dystrophy: A condition presenting muscle weakness and wasting which may be progressive. [NIH]

Nadolol: A non-selective beta-adrenergic antagonist with a long half-life, used in cardiovascular disease to treat arrhythmias, angina pectoris, and hypertension. Nadolol is also used for migraine and for tremor. [NIH]

Narcotic: 1. Pertaining to or producing narcosis. 2. An agent that produces insensibility or stupor, applied especially to the opioids, i.e. to any natural or synthetic drug that has morphine-like actions. [EU]

Nasal Mucosa: The mucous membrane lining the nasal cavity. [NIH]

Nausea: An unpleasant sensation in the stomach usually accompanied by the urge to vomit. Common causes are early pregnancy, sea and motion sickness, emotional stress, intense pain, food poisoning, and various enteroviruses. [NIH]

NCI: National Cancer Institute. NCI, part of the National Institutes of Health of the United States Department of Health and Human Services, is the federal government's principal agency for cancer research. NCI conducts, coordinates, and funds cancer research, training, health information dissemination, and other programs with respect to the cause, diagnosis, prevention, and treatment of cancer. Access the NCI Web site at http://cancer.gov. [NIH]

Need: A state of tension or dissatisfaction felt by an individual that impels him to action toward a goal he believes will satisfy the impulse. [NIH]

Neonatal: Pertaining to the first four weeks after birth. [EU]

Neoplasia: Abnormal and uncontrolled cell growth. [NIH]

Neoplasms: New abnormal growth of tissue. Malignant neoplasms show a greater degree of anaplasia and have the properties of invasion and metastasis, compared to benign neoplasms. [NIH]

Neoplastic: Pertaining to or like a neoplasm (= any new and abnormal growth); pertaining to neoplasia (= the formation of a neoplasm). [EU]

Nephropathy: Disease of the kidneys. [EU]

Nerve: A cordlike structure of nervous tissue that connects parts of the nervous system with other tissues of the body and conveys nervous impulses to, or away from, these tissues. [NIH]

Nervous System: The entire nerve apparatus composed of the brain, spinal cord, nerves and ganglia. [NIH]

Networks: Pertaining to a nerve or to the nerves, a meshlike structure of interlocking fibers or strands. [NIH]

Neural: 1. Pertaining to a nerve or to the nerves. 2. Situated in the region of the spinal axis, as the neutral arch. [EU]

Neural Crest: A strip of specialized ectoderm flanking each side of the embryonal neural plate, which after the closure of the neural tube, forms a column of isolated cells along the dorsal aspect of the neural tube. Most of the cranial and all of the spinal sensory ganglion cells arise by differentiation of neural crest cells. [NIH]

Neurologic: Having to do with nerves or the nervous system. [NIH]

Neurologist: A doctor who specializes in the diagnosis and treatment of disorders of the nervous system. [NIH]

Neuromuscular: Pertaining to muscles and nerves. [EU]

Neuromuscular Diseases: A general term encompassing lower motor neuron disease; peripheral nervous system diseases; and certain muscular diseases. Manifestations include muscle weakness; fasciculation; muscle atrophy; spasm; myokymia; muscle hypertonia, myalgias, and musclehypotonia. [NIH]

Neuronal: Pertaining to a neuron or neurons (= conducting cells of the nervous system). [EU]

Neurons: The basic cellular units of nervous tissue. Each neuron consists of a body, an axon, and dendrites. Their purpose is to receive, conduct, and transmit impulses in the nervous system. [NIH]

Neuropathy: A problem in any part of the nervous system except the brain and spinal cord. Neuropathies can be caused by infection, toxic substances, or disease. [NIH]

Neurotransmitter: Any of a group of substances that are released on excitation from the axon terminal of a presynaptic neuron of the central or peripheral nervous system and travel across the synaptic cleft to either excite or inhibit the target cell. Among the many substances that have the properties of a neurotransmitter are acetylcholine, norepinephrine, epinephrine, dopamine, glycine, y-aminobutyrate, glutamic acid, substance P, enkephalins, endorphins, and serotonin. [EU]

Neutrons: Electrically neutral elementary particles found in all atomic nuclei except light hydrogen; the mass is equal to that of the proton and electron combined and they are unstable when isolated from the nucleus, undergoing beta decay. Slow, thermal, epithermal, and fast neutrons refer to the energy levels with which the neutrons are ejected from heavier nuclei during their decay. [NIH]

Neutrophil: A type of white blood cell. [NIH]

Niacin: Water-soluble vitamin of the B complex occurring in various animal and plant tissues. Required by the body for the formation of coenzymes NAD and NADP. Has pellagra-curative, vasodilating, and antilipemic properties. [NIH]

Nicotine: Nicotine is highly toxic alkaloid. It is the prototypical agonist at nicotinic cholinergic receptors where it dramatically stimulates neurons and ultimately blocks synaptic transmission. Nicotine is also important medically because of its presence in tobacco smoke. [NIH]

Nitric Oxide: A free radical gas produced endogenously by a variety of mammalian cells. It is synthesized from arginine by a complex reaction, catalyzed by nitric oxide synthase. Nitric oxide is endothelium-derived relaxing factor. It is released by the vascular endothelium and mediates the relaxation induced by some vasodilators such as acetylcholine and bradykinin. It also inhibits platelet aggregation, induces disaggregation of aggregated platelets, and inhibits platelet adhesion to the vascular endothelium. Nitric oxide activates cytosolic guanylate cyclase and thus elevates intracellular levels of cyclic GMP. [NIH]

Nitrogen: An element with the atomic symbol N, atomic number 7, and atomic weight 14. Nitrogen exists as a diatomic gas and makes up about 78% of the earth's atmosphere by volume. It is a constituent of proteins and nucleic acids and found in all living cells. [NIH]

Nitroglycerin: A highly volatile organic nitrate that acts as a dilator of arterial and venous smooth muscle and is used in the treatment of angina. It provides relief through improvement of the balance between myocardial oxygen supply and demand. Although

total coronary blood flow is not increased, there is redistribution of blood flow in the heart when partial occlusion of coronary circulation is effected. [NIH]

Norepinephrine: Precursor of epinephrine that is secreted by the adrenal medulla and is a widespread central and autonomic neurotransmitter. Norepinephrine is the principal transmitter of most postganglionic sympathetic fibers and of the diffuse projection system in the brain arising from the locus ceruleus. It is also found in plants and is used pharmacologically as a sympathomimetic. [NIH]

Normotensive: 1. Characterized by normal tone, tension, or pressure, as by normal blood pressure. 2. A person with normal blood pressure. [EU]

Nortriptyline: A metabolite of amitryptyline that is also used as an antidepressive agent. Nortriptyline is used in major depression, dysthymia, and atypical depressions. [NIH]

Nuclear: A test of the structure, blood flow, and function of the kidneys. The doctor injects a mildly radioactive solution into an arm vein and uses x-rays to monitor its progress through the kidneys. [NIH]

Nuclear Medicine: A specialty field of radiology concerned with diagnostic, therapeutic, and investigative use of radioactive compounds in a pharmaceutical form. [NIH]

Nuclei: A body of specialized protoplasm found in nearly all cells and containing the chromosomes. [NIH]

Nucleic acid: Either of two types of macromolecule (DNA or RNA) formed by polymerization of nucleotides. Nucleic acids are found in all living cells and contain the information (genetic code) for the transfer of genetic information from one generation to the next. [NIH]

Nucleic Acid Hybridization: The process whereby two single-stranded polynucleotides form a double-stranded molecule, with hydrogen bonding between the complementary bases in the two strains. [NIH]

Nucleus: A body of specialized protoplasm found in nearly all cells and containing the chromosomes. [NIH]

Nutritional Status: State of the body in relation to the consumption and utilization of nutrients. [NIH]

Observational study: An epidemiologic study that does not involve any intervention, experimental or otherwise. Such a study may be one in which nature is allowed to take its course, with changes in one characteristic being studied in relation to changes in other characteristics. Analytical epidemiologic methods, such as case-control and cohort study designs, are properly called observational epidemiology because the investigator is observing without intervention other than to record, classify, count, and statistically analyze results. [NIH]

Occupational Exposure: The exposure to potentially harmful chemical, physical, or biological agents that occurs as a result of one's occupation. [NIH]

Ocular: 1. Of, pertaining to, or affecting the eye. 2. Eyepiece. [EU]

Odds Ratio: The ratio of two odds. The exposure-odds ratio for case control data is the ratio of the odds in favor of exposure among cases to the odds in favor of exposure among noncases. The disease-odds ratio for a cohort or cross section is the ratio of the odds in favor of disease among the exposed to the odds in favor of disease among the unexposed. The prevalence-odds ratio refers to an odds ratio derived cross-sectionally from studies of prevalent cases. [NIH]

Oestradiol: Growth hormone. [NIH]

Oestrogen: A generic term for oestrus-producing steroid compounds; the female sex hormones. In humans, oestrogen is formed in the ovary, possibly the adrenal cortex, the testis, and the foetoplacental unit; it has various functions in both sexes. It is responsible for the development of the female secondary sex characteristics, and during the menstrual cycle it acts on the female genitalia to produce an environment suitable for the fertilization, implantation, and nutrition of the early embryo. Oestrogen is used in oral contraceptives and as a palliative in cancer of the breast after menopause and cancer of the prostate; other uses include the relief of the discomforts of menopause, inhibition of lactation, and treatment of osteoporosis, threatened abortion, and various functional ovarian disorders. [EU]

Oliguria: Clinical manifestation of the urinary system consisting of a decrease in the amount of urine secreted. [NIH]

Omega-3 fatty acid: A type of fat obtained in the diet and involved in immunity. [NIH]

Oncogene: A gene that normally directs cell growth. If altered, an oncogene can promote or allow the uncontrolled growth of cancer. Alterations can be inherited or caused by an environmental exposure to carcinogens. [NIH]

On-line: A sexually-reproducing population derived from a common parentage. [NIH]

Opacity: Degree of density (area most dense taken for reading). [NIH]

Operon: The genetic unit consisting of a feedback system under the control of an operator gene, in which a structural gene transcribes its message in the form of mRNA upon blockade of a repressor produced by a regulator gene. Included here is the attenuator site of bacterial operons where transcription termination is regulated. [NIH]

Ophthalmoscope: A lighted instrument used to examine the inside of the eye, including the retina and the optic nerve. [NIH]

Opiate: A remedy containing or derived from opium; also any drug that induces sleep. [EU]

Opium: The air-dried exudate from the unripe seed capsule of the opium poppy, Papaver somniferum, or its variant, P. album. It contains a number of alkaloids, but only a few - morphine, codeine, and papaverine - have clinical significance. Opium has been used as an analgesic, antitussive, antidiarrheal, and antispasmodic. [NIH]

Opsin: A protein formed, together with retinene, by the chemical breakdown of meta-rhodopsin. [NIH]

Optic Nerve: The 2nd cranial nerve. The optic nerve conveys visual information from the retina to the brain. The nerve carries the axons of the retinal ganglion cells which sort at the optic chiasm and continue via the optic tracts to the brain. The largest projection is to the lateral geniculate nuclei; other important targets include the superior colliculi and the suprachiasmatic nuclei. Though known as the second cranial nerve, it is considered part of the central nervous system. [NIH]

Oral Health: The optimal state of the mouth and normal functioning of the organs of the mouth without evidence of disease. [NIH]

Organogenesis: Clonal propagation which involves culturing explants from roots, leaves, or stems to form undifferentiated callus tissue; after the cells form shoots, they are separated and rooted. Alternatively, if the callus is put in liquid culture, somatic embryos form. [NIH]

Osmotic: Pertaining to or of the nature of osmosis (= the passage of pure solvent from a solution of lesser to one of greater solute concentration when the two solutions are separated by a membrane which selectively prevents the passage of solute molecules, but is permeable to the solvent). [EU]

Osteoarthritis: A progressive, degenerative joint disease, the most common form of arthritis,

especially in older persons. The disease is thought to result not from the aging process but from biochemical changes and biomechanical stresses affecting articular cartilage. In the foreign literature it is often called osteoarthrosis deformans. [NIH]

Osteoporosis: Reduction of bone mass without alteration in the composition of bone, leading to fractures. Primary osteoporosis can be of two major types: postmenopausal osteoporosis and age-related (or senile) osteoporosis. [NIH]

Outpatient: A patient who is not an inmate of a hospital but receives diagnosis or treatment in a clinic or dispensary connected with the hospital. [NIH]

Ovary: Either of the paired glands in the female that produce the female germ cells and secrete some of the female sex hormones. [NIH]

Overexpress: An excess of a particular protein on the surface of a cell. [NIH]

Overweight: An excess of body weight but not necessarily body fat; a body mass index of 25 to 29.9 kg/m2. [NIH]

Ovum: A female germ cell extruded from the ovary at ovulation. [NIH]

Oxidation: The act of oxidizing or state of being oxidized. Chemically it consists in the increase of positive charges on an atom or the loss of negative charges. Most biological oxidations are accomplished by the removal of a pair of hydrogen atoms (dehydrogenation) from a molecule. Such oxidations must be accompanied by reduction of an acceptor molecule. Univalent o. indicates loss of one electron; divalent o., the loss of two electrons. [EU]

Oxidative Phosphorylation: Electron transfer through the cytochrome system liberating free energy which is transformed into high-energy phosphate bonds. [NIH]

Oxidative Stress: A disturbance in the prooxidant-antioxidant balance in favor of the former, leading to potential damage. Indicators of oxidative stress include damaged DNA bases, protein oxidation products, and lipid peroxidation products (Sies, Oxidative Stress, 1991, pxv-xvi). [NIH]

Oximetry: The determination of oxygen-hemoglobin saturation of blood either by withdrawing a sample and passing it through a classical photoelectric oximeter or by electrodes attached to some translucent part of the body like finger, earlobe, or skin fold. It includes non-invasive oxygen monitoring by pulse oximetry. [NIH]

Oxygen Consumption: The oxygen consumption is determined by calculating the difference between the amount of oxygen inhaled and exhaled. [NIH]

Oxygenase: Enzyme which breaks down heme, the iron-containing oxygen-carrying constituent of the red blood cells. [NIH]

Oxygenation: The process of supplying, treating, or mixing with oxygen. No:1245 - oxygenation the process of supplying, treating, or mixing with oxygen. [EU]

Oxygenator: An apparatus by which oxygen is introduced into the blood during circulation outside the body, as during open heart surgery. [NIH]

Pacemaker: An object or substance that influences the rate at which a certain phenomenon occurs; often used alone to indicate the natural cardiac pacemaker or an artificial cardiac pacemaker. In biochemistry, a substance whose rate of reaction sets the pace for a series of interrelated reactions. [EU]

Palate: The structure that forms the roof of the mouth. It consists of the anterior hard palate and the posterior soft palate. [NIH]

Palliative: 1. Affording relief, but not cure. 2. An alleviating medicine. [EU]

Palpation: Application of fingers with light pressure to the surface of the body to determine

consistence of parts beneath in physical diagnosis; includes palpation for determining the outlines of organs. [NIH]

Pancreas: A mixed exocrine and endocrine gland situated transversely across the posterior abdominal wall in the epigastric and hypochondriac regions. The endocrine portion is comprised of the Islets of Langerhans, while the exocrine portion is a compound acinar gland that secretes digestive enzymes. [NIH]

Pancreatic: Having to do with the pancreas. [NIH]

Pancreatic cancer: Cancer of the pancreas, a salivary gland of the abdomen. [NIH]

Papilla: A small nipple-shaped elevation. [NIH]

Papillary: Pertaining to or resembling papilla, or nipple. [EU]

Paralysis: Loss of ability to move all or part of the body. [NIH]

Parasite: An animal or a plant that lives on or in an organism of another species and gets at least some of its nutrition from that other organism. [NIH]

Parasitic: Having to do with or being a parasite. A parasite is an animal or a plant that lives on or in an organism of another species and gets at least some of its nutrients from it. [NIH]

Parenteral: Not through the alimentary canal but rather by injection through some other route, as subcutaneous, intramuscular, intraorbital, intracapsular, intraspinal, intrasternal, intravenous, etc. [EU]

Parity: The number of offspring a female has borne. It is contrasted with gravidity, which refers to the number of pregnancies, regardless of outcome. [NIH]

Parotid: The space that contains the parotid gland, the facial nerve, the external carotid artery, and the retromandibular vein. [NIH]

Paroxetine: A serotonin uptake inhibitor that is effective in the treatment of depression. [NIH]

Paroxysmal: Recurring in paroxysms (= spasms or seizures). [EU]

Particle Accelerators: Devices which accelerate electrically charged atomic or subatomic particles, such as electrons, protons or ions, to high velocities so they have high kinetic energy. [NIH]

Parvovirus: A genus of the family Parvoviridae, subfamily Parvovirinae, infecting a variety of vertebrates including humans. Parvoviruses are responsible for a number of important diseases but also can be non-pathogenic in certain hosts. The type species is mice minute virus. [NIH]

Patch: A piece of material used to cover or protect a wound, an injured part, etc.: a patch over the eye. [NIH]

Patent ductus arteriosus: Abnormal persistence of the opening in the arterial duct that connects the pulmonary artery to the descending aorta; this opening normally closes within 24 hours of birth. [NIH]

Pathogenesis: The cellular events and reactions that occur in the development of disease. [NIH]

Pathologic: 1. Indicative of or caused by a morbid condition. 2. Pertaining to pathology (= branch of medicine that treats the essential nature of the disease, especially the structural and functional changes in tissues and organs of the body caused by the disease). [EU]

Pathologic Processes: The abnormal mechanisms and forms involved in the dysfunctions of tissues and organs. [NIH]

Pathologies: The study of abnormality, especially the study of diseases. [NIH]

Pathophysiology: Altered functions in an individual or an organ due to disease. [NIH]

Patient Advocacy: Promotion and protection of the rights of patients, frequently through a legal process. [NIH]

Patient Education: The teaching or training of patients concerning their own health needs. [NIH]

Pediatrics: A medical specialty concerned with maintaining health and providing medical care to children from birth to adolescence. [NIH]

Pelvic: Pertaining to the pelvis. [EU]

Pelvis: The lower part of the abdomen, located between the hip bones. [NIH]

Penis: The external reproductive organ of males. It is composed of a mass of erectile tissue enclosed in three cylindrical fibrous compartments. Two of the three compartments, the corpus cavernosa, are placed side-by-side along the upper part of the organ. The third compartment below, the corpus spongiosum, houses the urethra. [NIH]

Peptide: Any compound consisting of two or more amino acids, the building blocks of proteins. Peptides are combined to make proteins. [NIH]

Perceived risk: Estimate or evaluation of risk as observed through personal experience or personal study, and personal evaluation of consequences. [NIH]

Perception: The ability quickly and accurately to recognize similarities and differences among presented objects, whether these be pairs of words, pairs of number series, or multiple sets of these or other symbols such as geometric figures. [NIH]

Percutaneous: Performed through the skin, as injection of radiopacque material in radiological examination, or the removal of tissue for biopsy accomplished by a needle. [EU]

Perforation: 1. The act of boring or piercing through a part. 2. A hole made through a part or substance. [EU]

Perfusion: Bathing an organ or tissue with a fluid. In regional perfusion, a specific area of the body (usually an arm or a leg) receives high doses of anticancer drugs through a blood vessel. Such a procedure is performed to treat cancer that has not spread. [NIH]

Pergolide: A long-acting dopamine agonist which is effective in the treatment of Parkinson's disease and hyperprolactinemia. It has also been observed to have antihypertensive effects. [NIH]

Pericardiectomy: Surgical excision (total or partial) of a portion of the pericardium. Pericardiotomy refers to incision of the pericardium. [NIH]

Pericarditis: Inflammation of the pericardium. [EU]

Pericardium: The fibroserous sac surrounding the heart and the roots of the great vessels. [NIH]

Perinatal: Pertaining to or occurring in the period shortly before and after birth; variously defined as beginning with completion of the twentieth to twenty-eighth week of gestation and ending 7 to 28 days after birth. [EU]

Perineal: Pertaining to the perineum. [EU]

Perineum: The area between the anus and the sex organs. [NIH]

Periodontal disease: Disease involving the supporting structures of the teeth (as the gums and periodontal membranes). [NIH]

Periodontal disease: Disease involving the supporting structures of the teeth (as the gums and periodontal membranes). [NIH]

Periodontal Ligament: Fibrous connective tissue surrounding the root of a tooth that

separates it from and attaches it to the alveolar bone. [NIH]

Periodontics: A dental specialty concerned with the histology, physiology, and pathology of the tissues that support, attach, and surround the teeth, and of the treatment and prevention of disease affecting these tissues. [NIH]

Periodontist: A specialist in the treatment of diseases of the gums. [NIH]

Periodontitis: Inflammation of the periodontal membrane; also called periodontitis simplex. [NIH]

Perioperative: Around the time of surgery; usually lasts from the time of going into the hospital or doctor's office for surgery until the time the patient goes home. [NIH]

Peripheral Nervous System: The nervous system outside of the brain and spinal cord. The peripheral nervous system has autonomic and somatic divisions. The autonomic nervous system includes the enteric, parasympathetic, and sympathetic subdivisions. The somatic nervous system includes the cranial and spinal nerves and their ganglia and the peripheral sensory receptors. [NIH]

Peripheral Nervous System Diseases: Diseases of the peripheral nerves external to the brain and spinal cord, which includes diseases of the nerve roots, ganglia, plexi, autonomic nerves, sensory nerves, and motor nerves. [NIH]

Peripheral Neuropathy: Nerve damage, usually affecting the feet and legs; causing pain, numbness, or a tingling feeling. Also called "somatic neuropathy" or "distal sensory polyneuropathy." [NIH]

Peripheral Vascular Disease: Disease in the large blood vessels of the arms, legs, and feet. People who have had diabetes for a long time may get this because major blood vessels in their arms, legs, and feet are blocked and these limbs do not receive enough blood. The signs of PVD are aching pains in the arms, legs, and feet (especially when walking) and foot sores that heal slowly. Although people with diabetes cannot always avoid PVD, doctors say they have a better chance of avoiding it if they take good care of their feet, do not smoke, and keep both their blood pressure and diabetes under good control. [NIH]

Peritoneal: Having to do with the peritoneum (the tissue that lines the abdominal wall and covers most of the organs in the abdomen). [NIH]

Peritoneal Cavity: The space enclosed by the peritoneum. It is divided into two portions, the greater sac and the lesser sac or omental bursa, which lies behind the stomach. The two sacs are connected by the foramen of Winslow, or epiploic foramen. [NIH]

Peritoneal Dialysis: Dialysis fluid being introduced into and removed from the peritoneal cavity as either a continuous or an intermittent procedure. [NIH]

Peritoneum: Endothelial lining of the abdominal cavity, the parietal peritoneum covering the inside of the abdominal wall and the visceral peritoneum covering the bowel, the mesentery, and certain of the organs. The portion that covers the bowel becomes the serosal layer of the bowel wall. [NIH]

PH: The symbol relating the hydrogen ion (H+) concentration or activity of a solution to that of a given standard solution. Numerically the pH is approximately equal to the negative logarithm of H+ concentration expressed in molarity. pH 7 is neutral; above it alkalinity increases and below it acidity increases. [EU]

Pharmacogenetics: A branch of genetics which deals with the genetic components of variability in individual responses to and metabolism (biotransformation) of drugs. [NIH]

Pharmacokinetic: The mathematical analysis of the time courses of absorption, distribution, and elimination of drugs. [NIH]

Pharmacologic: Pertaining to pharmacology or to the properties and reactions of drugs. [EU]

Pharmacotherapy: A regimen of using appetite suppressant medications to manage obesity by decreasing appetite or increasing the feeling of satiety. These medications decrease appetite by increasing serotonin or catecholamine—two brain chemicals that affect mood and appetite. [NIH]

Pharynx: The hollow tube about 5 inches long that starts behind the nose and ends at the top of the trachea (windpipe) and esophagus (the tube that goes to the stomach). [NIH]

Phenotype: The outward appearance of the individual. It is the product of interactions between genes and between the genotype and the environment. This includes the killer phenotype, characteristic of yeasts. [NIH]

Phenyl: Ingredient used in cold and flu remedies. [NIH]

Phenylalanine: An aromatic amino acid that is essential in the animal diet. It is a precursor of melanin, dopamine, noradrenalin, and thyroxine. [NIH]

Phosphodiesterase: Effector enzyme that regulates the levels of a second messenger, the cyclic GMP. [NIH]

Phospholipids: Lipids containing one or more phosphate groups, particularly those derived from either glycerol (phosphoglycerides; glycerophospholipids) or sphingosine (sphingolipids). They are polar lipids that are of great importance for the structure and function of cell membranes and are the most abundant of membrane lipids, although not stored in large amounts in the system. [NIH]

Phosphorus: A non-metallic element that is found in the blood, muscles, nevers, bones, and teeth, and is a component of adenosine triphosphate (ATP; the primary energy source for the body's cells.) [NIH]

Phosphorylated: Attached to a phosphate group. [NIH]

Phosphorylation: The introduction of a phosphoryl group into a compound through the formation of an ester bond between the compound and a phosphorus moiety. [NIH]

Physical Examination: Systematic and thorough inspection of the patient for physical signs of disease or abnormality. [NIH]

Physical Fitness: A state of well-being in which performance is optimal, often as a result of physical conditioning which may be prescribed for disease therapy. [NIH]

Physiologic: Having to do with the functions of the body. When used in the phrase "physiologic age," it refers to an age assigned by general health, as opposed to calendar age. [NIH]

Physiology: The science that deals with the life processes and functions of organismus, their cells, tissues, and organs. [NIH]

Pigments: Any normal or abnormal coloring matter in plants, animals, or micro-organisms. [NIH]

Pilot study: The initial study examining a new method or treatment. [NIH]

Pituitary Gland: A small, unpaired gland situated in the sella turcica tissue. It is connected to the hypothalamus by a short stalk. [NIH]

Placenta: A highly vascular fetal organ through which the fetus absorbs oxygen and other nutrients and excretes carbon dioxide and other wastes. It begins to form about the eighth day of gestation when the blastocyst adheres to the decidua. [NIH]

Plant sterols: Plant-based compounds that can compete with dietary cholesterol to be absorbed by the intestines. This results in lower blood cholesterol levels. They may have some effect in cancer prevention. Also known as phytosterols. [NIH]

Plants: Multicellular, eukaryotic life forms of the kingdom Plantae. They are characterized

by a mainly photosynthetic mode of nutrition; essentially unlimited growth at localized regions of cell divisions (meristems); cellulose within cells providing rigidity; the absence of organs of locomotion; absense of nervous and sensory systems; and an alteration of haploid and diploid generations. [NIH]

Plaque: A clear zone in a bacterial culture grown on an agar plate caused by localized destruction of bacterial cells by a bacteriophage. The concentration of infective virus in a fluid can be estimated by applying the fluid to a culture and counting the number of. [NIH]

Plasma: The clear, yellowish, fluid part of the blood that carries the blood cells. The proteins that form blood clots are in plasma. [NIH]

Plasma cells: A type of white blood cell that produces antibodies. [NIH]

Plasma protein: One of the hundreds of different proteins present in blood plasma, including carrier proteins (such albumin, transferrin, and haptoglobin), fibrinogen and other coagulation factors, complement components, immunoglobulins, enzyme inhibitors, precursors of substances such as angiotension and bradykinin, and many other types of proteins. [EU]

Plasmapheresis: Procedure whereby plasma is separated and extracted from anticoagulated whole blood and the red cells retransfused to the donor. Plasmapheresis is also employed for therapeutic use. [NIH]

Plasmin: A product of the lysis of plasminogen (profibrinolysin) by plasminogen activators. It is composed of two polypeptide chains, light (B) and heavy (A), with a molecular weight of 75,000. It is the major proteolytic enzyme involved in blood clot retraction or the lysis of fibrin and quickly inactivated by antiplasmins. EC 3.4.21.7. [NIH]

Plasminogen: Precursor of fibrinolysin (plasmin). It is a single-chain beta-globulin of molecular weight 80-90,000 found mostly in association with fibrinogen in plasma; plasminogen activators change it to fibrinolysin. It is used in wound debriding and has been investigated as a thrombolytic agent. [NIH]

Plasminogen Activators: A heterogeneous group of proteolytic enzymes that convert plasminogen to plasmin. They are concentrated in the lysosomes of most cells and in the vascular endothelium, particularly in the vessels of the microcirculation. EC 3.4.21.-. [NIH]

Platelet Aggregation: The attachment of platelets to one another. This clumping together can be induced by a number of agents (e.g., thrombin, collagen) and is part of the mechanism leading to the formation of a thrombus. [NIH]

Plateletpheresis: The preparation of platelet concentrates with the return of red cells and platelet-poor plasma to the donor. [NIH]

Platelets: A type of blood cell that helps prevent bleeding by causing blood clots to form. Also called thrombocytes. [NIH]

Pneumonia: Inflammation of the lungs. [NIH]

Point Mutation: A mutation caused by the substitution of one nucleotide for another. This results in the DNA molecule having a change in a single base pair. [NIH]

Poisoning: A condition or physical state produced by the ingestion, injection or inhalation of, or exposure to a deleterious agent. [NIH]

Polycystic: An inherited disorder characterized by many grape-like clusters of fluid-filled cysts that make both kidneys larger over time. These cysts take over and destroy working kidney tissue. PKD may cause chronic renal failure and end-stage renal disease. [NIH]

Polymerase: An enzyme which catalyses the synthesis of DNA using a single DNA strand as a template. The polymerase copies the template in the 5'-3'direction provided that

sufficient quantities of free nucleotides, dATP and dTTP are present. [NIH]

Polymerase Chain Reaction: In vitro method for producing large amounts of specific DNA or RNA fragments of defined length and sequence from small amounts of short oligonucleotide flanking sequences (primers). The essential steps include thermal denaturation of the double-stranded target molecules, annealing of the primers to their complementary sequences, and extension of the annealed primers by enzymatic synthesis with DNA polymerase. The reaction is efficient, specific, and extremely sensitive. Uses for the reaction include disease diagnosis, detection of difficult-to-isolate pathogens, mutation analysis, genetic testing, DNA sequencing, and analyzing evolutionary relationships. [NIH]

Polymorphic: Occurring in several or many forms; appearing in different forms at different stages of development. [EU]

Polymorphism: The occurrence together of two or more distinct forms in the same population. [NIH]

Polytetrafluoroethylene: Homopolymer of tetrafluoroethylene. Nonflammable, tough, inert plastic tubing or sheeting; used to line vessels, insulate, protect or lubricate apparatus; also as filter, coating for surgical implants or as prosthetic material. Synonyms: Fluoroflex; Fluoroplast; Ftoroplast; Halon; Polyfene; PTFE; Tetron. [NIH]

Polyunsaturated fat: An unsaturated fat found in greatest amounts in foods derived from plants, including safflower, sunflower, corn, and soybean oils. [NIH]

Popliteal: Compression of the nerve at the neck of the fibula. [NIH]

Postal Service: The functions and activities carried out by the U.S. Postal Service, foreign postal services, and private postal services such as Federal Express. [NIH]

Posterior: Situated in back of, or in the back part of, or affecting the back or dorsal surface of the body. In lower animals, it refers to the caudal end of the body. [EU]

Postmenopausal: Refers to the time after menopause. Menopause is the time in a woman's life when menstrual periods stop permanently; also called "change of life." [NIH]

Postnatal: Occurring after birth, with reference to the newborn. [EU]

Postoperative: After surgery. [NIH]

Postoperative Period: The period following a surgical operation. [NIH]

Postprandial: Occurring after dinner, or after a meal; postcibal. [EU]

Post-translational: The cleavage of signal sequence that directs the passage of the protein through a cell or organelle membrane. [NIH]

Postural: Pertaining to posture or position. [EU]

Potassium: An element that is in the alkali group of metals. It has an atomic symbol K, atomic number 19, and atomic weight 39.10. It is the chief cation in the intracellular fluid of muscle and other cells. Potassium ion is a strong electrolyte and it plays a significant role in the regulation of fluid volume and maintenance of the water-electrolyte balance. [NIH]

Potentiates: A degree of synergism which causes the exposure of the organism to a harmful substance to worsen a disease already contracted. [NIH]

Practice Guidelines: Directions or principles presenting current or future rules of policy for the health care practitioner to assist him in patient care decisions regarding diagnosis, therapy, or related clinical circumstances. The guidelines may be developed by government agencies at any level, institutions, professional societies, governing boards, or by the convening of expert panels. The guidelines form a basis for the evaluation of all aspects of health care and delivery. [NIH]

Pravastatin: An antilipemic fungal metabolite isolated from cultures of Nocardia

autotrophica. It acts as a competitive inhibitor of HMG CoA reductase (hydroxymethylglutaryl CoA reductases). [NIH]

Precancerous: A term used to describe a condition that may (or is likely to) become cancer. Also called premalignant. [NIH]

Preclinical: Before a disease becomes clinically recognizable. [EU]

Precursor: Something that precedes. In biological processes, a substance from which another, usually more active or mature substance is formed. In clinical medicine, a sign or symptom that heralds another. [EU]

Predisposition: A latent susceptibility to disease which may be activated under certain conditions, as by stress. [EU]

Prednisolone: A glucocorticoid with the general properties of the corticosteroids. It is the drug of choice for all conditions in which routine systemic corticosteroid therapy is indicated, except adrenal deficiency states. [NIH]

Preeclampsia: A toxaemia of late pregnancy characterized by hypertension, edema, and proteinuria, when convulsions and coma are associated, it is called eclampsia. [EU]

Prekallikrein: A plasma protein which is the precursor of kallikrein. Plasma that is deficient in prekallikrein has been found to be abnormal in thromboplastin formation, kinin generation, evolution of a permeability globulin, and plasmin formation. The absence of prekallikrein in plasma leads to Fletcher factor deficiency, a congenital disease. [NIH]

Premalignant: A term used to describe a condition that may (or is likely to) become cancer. Also called precancerous. [NIH]

Premenopausal: Refers to the time before menopause. Menopause is the time of life when a women's menstrual periods stop permanently; also called "change of life." [NIH]

Prenatal: Existing or occurring before birth, with reference to the fetus. [EU]

Prenatal Care: Care provided the pregnant woman in order to prevent complications, and decrease the incidence of maternal and prenatal mortality. [NIH]

Preoperative: Preceding an operation. [EU]

Pressoreceptors: Receptors in the vascular system, particularly the aorta and carotid sinus, which are sensitive to stretch of the vessel walls. [NIH]

Prevalence: The total number of cases of a given disease in a specified population at a designated time. It is differentiated from incidence, which refers to the number of new cases in the population at a given time. [NIH]

Primary endpoint: The main result that is measured at the end of a study to see if a given treatment worked (e.g., the number of deaths or the difference in survival between the treatment group and the control group). What the primary endpoint will be is decided before the study begins. [NIH]

Primary Prevention: Prevention of disease or mental disorders in susceptible individuals or populations through promotion of health, including mental health, and specific protection, as in immunization, as distinguished from the prevention of complications or after-effects of existing disease. [NIH]

Probe: An instrument used in exploring cavities, or in the detection and dilatation of strictures, or in demonstrating the potency of channels; an elongated instrument for exploring or sounding body cavities. [NIH]

Problem Solving: A learning situation involving more than one alternative from which a selection is made in order to attain a specific goal. [NIH]

Progeny: The offspring produced in any generation. [NIH]

Progeria: An abnormal congenital condition characterized by premature aging in children, where all the changes of cell senescence occur. It is manifested by premature greying, hair loss, hearing loss, cataracts, arthritis,osteoporosis, diabetes mellitus, atrophy of subcutaneous fat, skeletal hypoplasia, and accelerated atherosclerosis. Many affected individuals develop malignant tumors, especially sarcomas. [NIH]

Progesterone: Pregn-4-ene-3,20-dione. The principal progestational hormone of the body, secreted by the corpus luteum, adrenal cortex, and placenta. Its chief function is to prepare the uterus for the reception and development of the fertilized ovum. It acts as an antiovulatory agent when administered on days 5-25 of the menstrual cycle. [NIH]

Progression: Increase in the size of a tumor or spread of cancer in the body. [NIH]

Progressive: Advancing; going forward; going from bad to worse; increasing in scope or severity. [EU]

Proinsulin: The substance made first in the pancreas that is then made into insulin. When insulin is purified from the pancreas of pork or beef, all the proinsulin is not fully removed. When some people use these insulins, the proinsulin can cause the body to react with a rash, to resist the insulin, or even to make dents or lumps in the skin at the place where the insulin is injected. The purified insulins have less proinsulin and other impurities than the other types of insulins. [NIH]

Projection: A defense mechanism, operating unconsciously, whereby that which is emotionally unacceptable in the self is rejected and attributed (projected) to others. [NIH]

Prolactin: Pituitary lactogenic hormone. A polypeptide hormone with a molecular weight of about 23,000. It is essential in the induction of lactation in mammals at parturition and is synergistic with estrogen. The hormone also brings about the release of progesterone from lutein cells, which renders the uterine mucosa suited for the embedding of the ovum should fertilization occur. [NIH]

Proline: A non-essential amino acid that is synthesized from glutamic acid. It is an essential component of collagen and is important for proper functioning of joints and tendons. [NIH]

Promoter: A chemical substance that increases the activity of a carcinogenic process. [NIH]

Prone: Having the front portion of the body downwards. [NIH]

Prophylaxis: An attempt to prevent disease. [NIH]

Propranolol: A widely used non-cardioselective beta-adrenergic antagonist. Propranolol is used in the treatment or prevention of many disorders including acute myocardial infarction, arrhythmias, angina pectoris, hypertension, hypertensive emergencies, hyperthyroidism, migraine, pheochromocytoma, menopause, and anxiety. [NIH]

Propulsive: Tending or having power to propel; driving onward or forward; impelling to action or motion. [EU]

Prospective Studies: Observation of a population for a sufficient number of persons over a sufficient number of years to generate incidence or mortality rates subsequent to the selection of the study group. [NIH]

Prospective study: An epidemiologic study in which a group of individuals (a cohort), all free of a particular disease and varying in their exposure to a possible risk factor, is followed over a specific amount of time to determine the incidence rates of the disease in the exposed and unexposed groups. [NIH]

Prostaglandin: Any of a group of components derived from unsaturated 20-carbon fatty acids, primarily arachidonic acid, via the cyclooxygenase pathway that are extremely potent mediators of a diverse group of physiologic processes. The abbreviation for prostaglandin is PG; specific compounds are designated by adding one of the letters A through I to indicate

the type of substituents found on the hydrocarbon skeleton and a subscript (1, 2 or 3) to indicate the number of double bonds in the hydrocarbon skeleton e.g., PGE2. The predominant naturally occurring prostaglandins all have two double bonds and are synthesized from arachidonic acid (5,8,11,14-eicosatetraenoic acid) by the pathway shown in the illustration. The 1 series and 3 series are produced by the same pathway with fatty acids having one fewer double bond (8,11,14-eicosatrienoic acid or one more double bond (5,8,11,14,17-eicosapentaenoic acid) than arachidonic acid. The subscript a or ß indicates the configuration at C-9 (a denotes a substituent below the plane of the ring, ß, above the plane). The naturally occurring PGF's have the a configuration, e.g., PGF2a. All of the prostaglandins act by binding to specific cell-surface receptors causing an increase in the level of the intracellular second messenger cyclic AMP (and in some cases cyclic GMP also). The effect produced by the cyclic AMP increase depends on the specific cell type. In some cases there is also a positive feedback effect. Increased cyclic AMP increases prostaglandin synthesis leading to further increases in cyclic AMP. [EU]

Prostaglandins A: (13E,15S)-15-Hydroxy-9-oxoprosta-10,13-dien-1-oic acid (PGA(1)); (5Z,13E,15S)-15-hydroxy-9-oxoprosta-5,10,13-trien-1-oic acid (PGA(2)); (5Z,13E,15S,17Z)-15-hydroxy-9-oxoprosta-5,10,13,17-tetraen-1-oic acid (PGA(3)). A group of naturally occurring secondary prostaglandins derived from PGE. PGA(1) and PGA(2) as well as their 19-hydroxy derivatives are found in many organs and tissues. [NIH]

Prostate: A gland in males that surrounds the neck of the bladder and the urethra. It secretes a substance that liquifies coagulated semen. It is situated in the pelvic cavity behind the lower part of the pubic symphysis, above the deep layer of the triangular ligament, and rests upon the rectum. [NIH]

Prostatic Hyperplasia: Enlargement or overgrowth of the prostate gland as a result of an increase in the number of its constituent cells. [NIH]

Protease: Proteinase (= any enzyme that catalyses the splitting of interior peptide bonds in a protein). [EU]

Protease Inhibitors: Compounds which inhibit or antagonize biosynthesis or actions of proteases (endopeptidases). [NIH]

Protective Agents: Synthetic or natural substances which are given to prevent a disease or disorder or are used in the process of treating a disease or injury due to a poisonous agent. [NIH]

Protein C: A vitamin-K dependent zymogen present in the blood, which, upon activation by thrombin and thrombomodulin exerts anticoagulant properties by inactivating factors Va and VIIIa at the rate-limiting steps of thrombin formation. [NIH]

Protein S: The vitamin K-dependent cofactor of activated protein C. Together with protein C, it inhibits the action of factors VIIIa and Va. A deficiency in protein S can lead to recurrent venous and arterial thrombosis. [NIH]

Proteins: Polymers of amino acids linked by peptide bonds. The specific sequence of amino acids determines the shape and function of the protein. [NIH]

Protein-Tyrosine Kinase: An enzyme that catalyzes the phosphorylation of tyrosine residues in proteins with ATP or other nucleotides as phosphate donors. EC 2.7.1.112. [NIH]

Proteinuria: The presence of protein in the urine, indicating that the kidneys are not working properly. [NIH]

Proteoglycans: Glycoproteins which have a very high polysaccharide content. [NIH]

Proteolytic: 1. Pertaining to, characterized by, or promoting proteolysis. 2. An enzyme that promotes proteolysis (= the splitting of proteins by hydrolysis of the peptide bonds with formation of smaller polypeptides). [EU]

Protocol: The detailed plan for a clinical trial that states the trial's rationale, purpose, drug or vaccine dosages, length of study, routes of administration, who may participate, and other aspects of trial design. [NIH]

Protons: Stable elementary particles having the smallest known positive charge, found in the nuclei of all elements. The proton mass is less than that of a neutron. A proton is the nucleus of the light hydrogen atom, i.e., the hydrogen ion. [NIH]

Protozoa: A subkingdom consisting of unicellular organisms that are the simplest in the animal kingdom. Most are free living. They range in size from submicroscopic to macroscopic. Protozoa are divided into seven phyla: Sarcomastigophora, Labyrinthomorpha, Apicomplexa, Microspora, Ascetospora, Myxozoa, and Ciliophora. [NIH]

Proximal: Nearest; closer to any point of reference; opposed to distal. [EU]

Psychiatric: Pertaining to or within the purview of psychiatry. [EU]

Psychiatry: The medical science that deals with the origin, diagnosis, prevention, and treatment of mental disorders. [NIH]

Psychic: Pertaining to the psyche or to the mind; mental. [EU]

Psychological Adaptation: The alteration of the selective response of a neural unit due to the received signals. [NIH]

Psychopathology: The study of significant causes and processes in the development of mental illness. [NIH]

Psychosis: A mental disorder characterized by gross impairment in reality testing as evidenced by delusions, hallucinations, markedly incoherent speech, or disorganized and agitated behaviour without apparent awareness on the part of the patient of the incomprehensibility of his behaviour; the term is also used in a more general sense to refer to mental disorders in which mental functioning is sufficiently impaired as to interfere grossly with the patient's capacity to meet the ordinary demands of life. Historically, the term has been applied to many conditions, e.g. manic-depressive psychosis, that were first described in psychotic patients, although many patients with the disorder are not judged psychotic. [EU]

Psyllium: Dried, ripe seeds of Plantago psyllium, P. indica, and P. ovata (Plantaginaceae). Plantain seeds swell in water and are used as demulcents and bulk laxatives. [NIH]

Puberty: The period during which the secondary sex characteristics begin to develop and the capability of sexual reproduction is attained. [EU]

Public Health: Branch of medicine concerned with the prevention and control of disease and disability, and the promotion of physical and mental health of the population on the international, national, state, or municipal level. [NIH]

Public Policy: A course or method of action selected, usually by a government, from among alternatives to guide and determine present and future decisions. [NIH]

Pulmonary: Relating to the lungs. [NIH]

Pulmonary Artery: The short wide vessel arising from the conus arteriosus of the right ventricle and conveying unaerated blood to the lungs. [NIH]

Pulmonary Edema: An accumulation of an excessive amount of watery fluid in the lungs, may be caused by acute exposure to dangerous concentrations of irritant gasses. [NIH]

Pulmonary hypertension: Abnormally high blood pressure in the arteries of the lungs. [NIH]

Pulmonary Valve: A valve situated at the entrance to the pulmonary trunk from the right ventricle. [NIH]

Pulsation: A throb or rhythmical beat, as of the heart. [EU]

Pulse: The rhythmical expansion and contraction of an artery produced by waves of pressure caused by the ejection of blood from the left ventricle of the heart as it contracts. [NIH]

Pupil: The aperture in the iris through which light passes. [NIH]

Purified Insulins: Insulins with much less of the impure proinsulin. It is thought that the use of purified insulins may help avoid or reduce some of the problems of people with diabetes such as allergic reactions. [NIH]

Purpura: Purplish or brownish red discoloration, easily visible through the epidermis, caused by hemorrhage into the tissues. [NIH]

Purulent: Consisting of or containing pus; associated with the formation of or caused by pus. [EU]

Pyridoxal: 3-Hydroxy-5-(hydroxymethyl)-2-methyl-4- pyridinecarboxaldehyde. [NIH]

Pyruvate Dehydrogenase Complex: An organized assembly of three kinds of enzymes; catalyzes the oxidative decarboxylation of pyruvate. [NIH]

Quality of Life: A generic concept reflecting concern with the modification and enhancement of life attributes, e.g., physical, political, moral and social environment. [NIH]

Quiescent: Marked by a state of inactivity or repose. [EU]

Race: A population within a species which exhibits general similarities within itself, but is both discontinuous and distinct from other populations of that species, though not sufficiently so as to achieve the status of a taxon. [NIH]

Radiation: Emission or propagation of electromagnetic energy (waves/rays), or the waves/rays themselves; a stream of electromagnetic particles (electrons, neutrons, protons, alpha particles) or a mixture of these. The most common source is the sun. [NIH]

Radiation Hybrid Mapping: A method for ordering genetic loci along chromosomes. The method involves fusing irradiated donor cells with host cells from another species. Following cell fusion, fragments of DNA from the irradiated cells become integrated into the chromosomes of the host cells. Molecular probing of DNA obtained from the fused cells is used to determine if two or more genetic loci are located within the same fragment of donor cell DNA. [NIH]

Radioactive: Giving off radiation. [NIH]

Radiofrequency ablation: The use of electrical current to destroy tissue. [NIH]

Radiography: Examination of any part of the body for diagnostic purposes by means of roentgen rays, recording the image on a sensitized surface (such as photographic film). [NIH]

Radioisotope: An unstable element that releases radiation as it breaks down. Radioisotopes can be used in imaging tests or as a treatment for cancer. [NIH]

Radiolabeled: Any compound that has been joined with a radioactive substance. [NIH]

Radiological: Pertaining to radiodiagnostic and radiotherapeutic procedures, and interventional radiology or other planning and guiding medical radiology. [NIH]

Radiology: A specialty concerned with the use of x-ray and other forms of radiant energy in the diagnosis and treatment of disease. [NIH]

Radionuclide Imaging: Process whereby a radionuclide is injected or measured (through tissue) from an external source, and a display is obtained from any one of several rectilinear scanner or gamma camera systems. The image obtained from a moving detector is called a scan, while the image obtained from a stationary camera device is called a scintiphotograph. [NIH]

Radiopharmaceutical: Any medicinal product which, when ready for use, contains one or

more radionuclides (radioactive isotopes) included for a medicinal purpose. [NIH]

Radium: A radioactive element of the alkaline earth series of metals. It has the atomic symbol Ra, atomic number 88, and atomic weight 226. Radium is the product of the disintegration of uranium and is present in pitchblende and all ores containing uranium. It is used clinically as a source of beta and gamma-rays in radiotherapy, particularly brachytherapy. [NIH]

Radius: The lateral bone of the forearm. [NIH]

Radon: A naturally radioactive element with atomic symbol Rn, atomic number 86, and atomic weight 222. It is a member of the noble gas family and released during the decay of radium and found in soil. There is a link between exposure to radon and lung cancer. [NIH]

Raloxifene: A second generation selective estrogen receptor modulator (SERM) used to prevent osteoporosis in postmenopausal women. It has estrogen agonist effects on bone and cholesterol metabolism but behaves as a complete estrogen antagonist on mammary gland and uterine tissue. [NIH]

Randomized: Describes an experiment or clinical trial in which animal or human subjects are assigned by chance to separate groups that compare different treatments. [NIH]

Randomized clinical trial: A study in which the participants are assigned by chance to separate groups that compare different treatments; neither the researchers nor the participants can choose which group. Using chance to assign people to groups means that the groups will be similar and that the treatments they receive can be compared objectively. At the time of the trial, it is not known which treatment is best. It is the patient's choice to be in a randomized trial. [NIH]

Randomized Controlled Trials: Clinical trials that involve at least one test treatment and one control treatment, concurrent enrollment and follow-up of the test- and control-treated groups, and in which the treatments to be administered are selected by a random process, such as the use of a random-numbers table. Treatment allocations using coin flips, odd-even numbers, patient social security numbers, days of the week, medical record numbers, or other such pseudo- or quasi-random processes, are not truly randomized and trials employing any of these techniques for patient assignment are designated simply controlled clinical trials. [NIH]

Reactive Oxygen Species: Reactive intermediate oxygen species including both radicals and non-radicals. These substances are constantly formed in the human body and have been shown to kill bacteria and inactivate proteins, and have been implicated in a number of diseases. Scientific data exist that link the reactive oxygen species produced by inflammatory phagocytes to cancer development. [NIH]

Reagent: A substance employed to produce a chemical reaction so as to detect, measure, produce, etc., other substances. [EU]

Reality Testing: The individual's objective evaluation of the external world and the ability to differentiate adequately between it and the internal world; considered to be a primary ego function. [NIH]

Receptor: A molecule inside or on the surface of a cell that binds to a specific substance and causes a specific physiologic effect in the cell. [NIH]

Receptors, Serotonin: Cell-surface proteins that bind serotonin and trigger intracellular changes which influence the behavior of cells. Several types of serotonin receptors have been recognized which differ in their pharmacology, molecular biology, and mode of action. [NIH]

Recombinant: A cell or an individual with a new combination of genes not found together in either parent; usually applied to linked genes. [EU]

Recombination: The formation of new combinations of genes as a result of segregation in crosses between genetically different parents; also the rearrangement of linked genes due to crossing-over. [NIH]

Rectum: The last 8 to 10 inches of the large intestine. [NIH]

Recurrence: The return of a sign, symptom, or disease after a remission. [NIH]

Red blood cells: RBCs. Cells that carry oxygen to all parts of the body. Also called erythrocytes. [NIH]

Red Nucleus: A pinkish-yellow portion of the midbrain situated in the rostral mesencephalic tegmentum. It receives a large projection from the contralateral half of the cerebellum via the superior cerebellar peduncle and a projection from the ipsilateral motor cortex. [NIH]

Reductase: Enzyme converting testosterone to dihydrotestosterone. [NIH]

Reentry: Reexcitation caused by continuous propagation of the same impulse for one or more cycles. [NIH]

Refer: To send or direct for treatment, aid, information, de decision. [NIH]

Reflex: An involuntary movement or exercise of function in a part, excited in response to a stimulus applied to the periphery and transmitted to the brain or spinal cord. [NIH]

Refraction: A test to determine the best eyeglasses or contact lenses to correct a refractive error (myopia, hyperopia, or astigmatism). [NIH]

Regeneration: The natural renewal of a structure, as of a lost tissue or part. [EU]

Regimen: A treatment plan that specifies the dosage, the schedule, and the duration of treatment. [NIH]

Registries: The systems and processes involved in the establishment, support, management, and operation of registers, e.g., disease registers. [NIH]

Regurgitation: A backward flowing, as the casting up of undigested food, or the backward flowing of blood into the heart, or between the chambers of the heart when a valve is incompetent. [EU]

Relative risk: The ratio of the incidence rate of a disease among individuals exposed to a specific risk factor to the incidence rate among unexposed individuals; synonymous with risk ratio. Alternatively, the ratio of the cumulative incidence rate in the exposed to the cumulative incidence rate in the unexposed (cumulative incidence ratio). The term relative risk has also been used synonymously with odds ratio. This is because the odds ratio and relative risk approach each other if the disease is rare (5 percent of population) and the number of subjects is large. [NIH]

Reliability: Used technically, in a statistical sense, of consistency of a test with itself, i. e. the extent to which we can assume that it will yield the same result if repeated a second time. [NIH]

Remission: A decrease in or disappearance of signs and symptoms of cancer. In partial remission, some, but not all, signs and symptoms of cancer have disappeared. In complete remission, all signs and symptoms of cancer have disappeared, although there still may be cancer in the body. [NIH]

Renin: An enzyme which is secreted by the kidney and is formed from prorenin in plasma and kidney. The enzyme cleaves the Leu-Leu bond in angiotensinogen to generate angiotensin I. EC 3.4.23.15. (Formerly EC 3.4.99.19). [NIH]

Renin-Angiotensin System: A system consisting of renin, angiotensin-converting enzyme, and angiotensin II. Renin, an enzyme produced in the kidney, acts on angiotensinogen, an

alpha-2 globulin produced by the liver, forming angiotensin I. The converting enzyme contained in the lung acts on angiotensin I in the plasma converting it to angiotensin II, the most powerful directly pressor substance known. It causes contraction of the arteriolar smooth muscle and has other indirect actions mediated through the adrenal cortex. [NIH]

Reperfusion: Restoration of blood supply to tissue which is ischemic due to decrease in normal blood supply. The decrease may result from any source including atherosclerotic obstruction, narrowing of the artery, or surgical clamping. It is primarily a procedure for treating infarction or other ischemia, by enabling viable ischemic tissue to recover, thus limiting further necrosis. However, it is thought that reperfusion can itself further damage the ischemic tissue, causing reperfusion injury. [NIH]

Reperfusion Injury: Functional, metabolic, or structural changes, including necrosis, in ischemic tissues thought to result from reperfusion to ischemic areas of the tissue. The most common instance is myocardial reperfusion injury. [NIH]

Repressor: Any of the specific allosteric protein molecules, products of regulator genes, which bind to the operator of operons and prevent RNA polymerase from proceeding into the operon to transcribe messenger RNA. [NIH]

Reproductive cells: Egg and sperm cells. Each mature reproductive cell carries a single set of 23 chromosomes. [NIH]

Research Design: A plan for collecting and utilizing data so that desired information can be obtained with sufficient precision or so that an hypothesis can be tested properly. [NIH]

Research Support: Financial support of research activities. [NIH]

Respiration: The act of breathing with the lungs, consisting of inspiration, or the taking into the lungs of the ambient air, and of expiration, or the expelling of the modified air which contains more carbon dioxide than the air taken in (Blakiston's Gould Medical Dictionary, 4th ed.). This does not include tissue respiration (= oxygen consumption) or cell respiration (= cell respiration). [NIH]

Respiratory distress syndrome: A lung disease that occurs primarily in premature infants; the newborn must struggle for each breath and blueing of its skin reflects the baby's inability to get enough oxygen. [NIH]

Resting metabolic rate: RMR accounts for 65 to 75 percent of daily energy expenditure and represents the minimum energy needed to maintain all physiological cell functions in the resting state. The principal determinant of RMR is lean body mass (LBM). Obese subjects have a higher RMR in absolute terms than lean individuals, an equivalent RMR when corrected for LBM and per unit surface area, and a lower RMR when expressed per kilogram of body weight. Obese persons require more energy for any given activity because of a larger mass, but they tend to be more sedentary than lean subjects. [NIH]

Retina: The ten-layered nervous tissue membrane of the eye. It is continuous with the optic nerve and receives images of external objects and transmits visual impulses to the brain. Its outer surface is in contact with the choroid and the inner surface with the vitreous body. The outer-most layer is pigmented, whereas the inner nine layers are transparent. [NIH]

Retinal: 1. Pertaining to the retina. 2. The aldehyde of retinol, derived by the oxidative enzymatic splitting of absorbed dietary carotene, and having vitamin A activity. In the retina, retinal combines with opsins to form visual pigments. One isomer, 11-cis retinal combines with opsin in the rods (scotopsin) to form rhodopsin, or visual purple. Another, all-trans retinal (trans-r.); visual yellow; xanthopsin) results from the bleaching of rhodopsin by light, in which the 11-cis form is converted to the all-trans form. Retinal also combines with opsins in the cones (photopsins) to form the three pigments responsible for colour vision. Called also retinal, and retinene1. [EU]

Retinoblastoma: An eye cancer that most often occurs in children younger than 5 years. It occurs in hereditary and nonhereditary (sporadic) forms. [NIH]

Retinoids: Derivatives of vitamin A. Used clinically in the treatment of severe cystic acne, psoriasis, and other disorders of keratinization. Their possible use in the prophylaxis and treatment of cancer is being actively explored. [NIH]

Retinol: Vitamin A. It is essential for proper vision and healthy skin and mucous membranes. Retinol is being studied for cancer prevention; it belongs to the family of drugs called retinoids. [NIH]

Retinopathy: 1. Retinitis (= inflammation of the retina). 2. Retinosis (= degenerative, noninflammatory condition of the retina). [EU]

Retrospective: Looking back at events that have already taken place. [NIH]

Retrospective Studies: Studies used to test etiologic hypotheses in which inferences about an exposure to putative causal factors are derived from data relating to characteristics of persons under study or to events or experiences in their past. The essential feature is that some of the persons under study have the disease or outcome of interest and their characteristics are compared with those of unaffected persons. [NIH]

Retroviral vector: RNA from a virus that is used to insert genetic material into cells. [NIH]

Retrovirus: A member of a group of RNA viruses, the RNA of which is copied during viral replication into DNA by reverse transcriptase. The viral DNA is then able to be integrated into the host chromosomal DNA. [NIH]

Reversion: A return to the original condition, e. g. the reappearance of the normal or wild type in previously mutated cells, tissues, or organisms. [NIH]

Rheology: The study of the deformation and flow of matter, usually liquids or fluids, and of the plastic flow of solids. The concept covers consistency, dilatancy, liquefaction, resistance to flow, shearing, thixotrophy, and viscosity. [NIH]

Rheumatism: A group of disorders marked by inflammation or pain in the connective tissue structures of the body. These structures include bone, cartilage, and fat. [NIH]

Rheumatoid: Resembling rheumatism. [EU]

Rheumatoid arthritis: A form of arthritis, the cause of which is unknown, although infection, hypersensitivity, hormone imbalance and psychologic stress have been suggested as possible causes. [NIH]

Rhodopsin: A photoreceptor protein found in retinal rods. It is a complex formed by the binding of retinal, the oxidized form of retinol, to the protein opsin and undergoes a series of complex reactions in response to visible light resulting in the transmission of nerve impulses to the brain. [NIH]

Ribose: A pentose active in biological systems usually in its D-form. [NIH]

Ribosome: A granule of protein and RNA, synthesized in the nucleolus and found in the cytoplasm of cells. Ribosomes are the main sites of protein synthesis. Messenger RNA attaches to them and there receives molecules of transfer RNA bearing amino acids. [NIH]

Rickettsiae: One of a group of obligate intracellular parasitic microorganisms, once regarded as intermediate in their properties between bacteria and viruses but now classified as bacteria in the order Rickettsiales, which includes 17 genera and 3 families: Rickettsiace. [NIH]

Rigidity: Stiffness or inflexibility, chiefly that which is abnormal or morbid; rigor. [EU]

Risk factor: A habit, trait, condition, or genetic alteration that increases a person's chance of developing a disease. [NIH]

Risk patient: Patient who is at risk, because of his/her behaviour or because of the type of person he/she is. [EU]

Ristocetin: An antibiotic mixture of two components, A and B, obtained from Nocardia lurida (or the same substance produced by any other means). It is no longer used clinically because of its toxicity. It causes platelet agglutination and blood coagulation and is used to assay those functions in vitro. [NIH]

Rod: A reception for vision, located in the retina. [NIH]

Root Canal Therapy: A treatment modality in endodontics concerned with the therapy of diseases of the dental pulp. For preparatory procedures, root canal preparation is available. [NIH]

Ryanodine: Insecticidal alkaloid isolated from Ryania speciosa; proposed as a myocardial depressant. [NIH]

Salivary: The duct that convey saliva to the mouth. [NIH]

Salivary glands: Glands in the mouth that produce saliva. [NIH]

Saphenous: Applied to certain structures in the leg, e. g. nerve vein. [NIH]

Saphenous Vein: The vein which drains the foot and leg. [NIH]

Saponins: Sapogenin glycosides. A type of glycoside widely distributed in plants. Each consists of a sapogenin as the aglycon moiety, and a sugar. The sapogenin may be a steroid or a triterpene and the sugar may be glucose, galactose, a pentose, or a methylpentose. Sapogenins are poisonous towards the lower forms of life and are powerful hemolytics when injected into the blood stream able to dissolve red blood cells at even extreme dilutions. [NIH]

Sarcoidosis: An idiopathic systemic inflammatory granulomatous disorder comprised of epithelioid and multinucleated giant cells with little necrosis. It usually invades the lungs with fibrosis and may also involve lymph nodes, skin, liver, spleen, eyes, phalangeal bones, and parotid glands. [NIH]

Sarcolemma: The plasma membrane of a smooth, striated, or cardiac muscle fiber. [NIH]

Sarcomere: The repeating structural unit of a striated muscle fiber. [NIH]

Sarcoplasmic Reticulum: A network of tubules and sacs in the cytoplasm of skeletal muscles that assist with muscle contraction and relaxation by releasing and storing calcium ions. [NIH]

Saturated fat: A type of fat found in greatest amounts in foods from animals, such as fatty cuts of meat, poultry with the skin, whole-milk dairy products, lard, and in some vegetable oils, including coconut, palm kernel, and palm oils. Saturated fat raises blood cholesterol more than anything else eaten. On a Step I Diet, no more than 8 to 10 percent of total calories should come from saturated fat, and in the Step II Diet, less than 7 percent of the day's total calories should come from saturated fat. [NIH]

Scans: Pictures of structures inside the body. Scans often used in diagnosing, staging, and monitoring disease include liver scans, bone scans, and computed tomography (CT) or computerized axial tomography (CAT) scans and magnetic resonance imaging (MRI) scans. In liver scanning and bone scanning, radioactive substances that are injected into the bloodstream collect in these organs. A scanner that detects the radiation is used to create pictures. In CT scanning, an x-ray machine linked to a computer is used to produce detailed pictures of organs inside the body. MRI scans use a large magnet connected to a computer to create pictures of areas inside the body. [NIH]

Scatter: The extent to which relative success and failure are divergently manifested in qualitatively different tests. [NIH]

Schizophrenia: A mental disorder characterized by a special type of disintegration of the personality. [NIH]

Sclerosis: A pathological process consisting of hardening or fibrosis of an anatomical structure, often a vessel or a nerve. [NIH]

Screening: Checking for disease when there are no symptoms. [NIH]

Scurvy: A deficiency disease due to lack of vitamin C in the diet. [NIH]

Second Messenger Systems: Systems in which an intracellular signal is generated in response to an intercellular primary messenger such as a hormone or neurotransmitter. They are intermediate signals in cellular processes such as metabolism, secretion, contraction, phototransduction, and cell growth. Examples of second messenger systems are the adenyl cyclase-cyclic AMP system, the phosphatidylinositol diphosphate-inositol triphosphate system, and the cyclic GMP system. [NIH]

Secretion: 1. The process of elaborating a specific product as a result of the activity of a gland; this activity may range from separating a specific substance of the blood to the elaboration of a new chemical substance. 2. Any substance produced by secretion. [EU]

Secular trends: A relatively long-term trend in a community or country. [NIH]

Secundum: The second atrial septum to appear in the embryonic heart. [NIH]

Sedative: 1. Allaying activity and excitement. 2. An agent that allays excitement. [EU]

Sedentary: 1. Sitting habitually; of inactive habits. 2. Pertaining to a sitting posture. [EU]

Sediment: A precipitate, especially one that is formed spontaneously. [EU]

Sedimentation: The act of causing the deposit of sediment, especially by the use of a centrifugal machine. [EU]

Segmental: Describing or pertaining to a structure which is repeated in similar form in successive segments of an organism, or which is undergoing segmentation. [NIH]

Segmentation: The process by which muscles in the intestines move food and wastes through the body. [NIH]

Seizures: Clinical or subclinical disturbances of cortical function due to a sudden, abnormal, excessive, and disorganized discharge of brain cells. Clinical manifestations include abnormal motor, sensory and psychic phenomena. Recurrent seizures are usually referred to as epilepsy or "seizure disorder." [NIH]

Selective estrogen receptor modulator: SERM. A drug that acts like estrogen on some tissues, but blocks the effect of estrogen on other tissues. Tamoxifen and raloxifene are SERMs. [NIH]

Selenium: An element with the atomic symbol Se, atomic number 34, and atomic weight 78.96. It is an essential micronutrient for mammals and other animals but is toxic in large amounts. Selenium protects intracellular structures against oxidative damage. It is an essential component of glutathione peroxidase. [NIH]

Self Care: Performance of activities or tasks traditionally performed by professional health care providers. The concept includes care of oneself or one's family and friends. [NIH]

Semen: The thick, yellowish-white, viscid fluid secretion of male reproductive organs discharged upon ejaculation. In addition to reproductive organ secretions, it contains spermatozoa and their nutrient plasma. [NIH]

Semisynthetic: Produced by chemical manipulation of naturally occurring substances. [EU]

Senescence: The bodily and mental state associated with advancing age. [NIH]

Senile: Relating or belonging to old age; characteristic of old age; resulting from infirmity of

old age. [NIH]

Senility: Old age; the physical and mental deterioration associated with old age. [EU]

Sensor: A device designed to respond to physical stimuli such as temperature, light, magnetism or movement and transmit resulting impulses for interpretation, recording, movement, or operating control. [NIH]

Sepsis: The presence of bacteria in the bloodstream. [NIH]

Septal: An abscess occurring at the root of the tooth on the proximal surface. [NIH]

Septum: A dividing wall or partition; a general term for such a structure. The term is often used alone to refer to the septal area or to the septum pellucidum. [EU]

Septum Pellucidum: A triangular double membrane separating the anterior horns of the lateral ventricles of the brain. It is situated in the median plane and bounded by the corpus callosum and the body and columns of the fornix. [NIH]

Sequela: Any lesion or affection following or caused by an attack of disease. [EU]

Sequencing: The determination of the order of nucleotides in a DNA or RNA chain. [NIH]

Sequester: A portion of dead bone which has become detached from the healthy bone tissue, as occurs in necrosis. [NIH]

Seroconversion: The change of a serologic test from negative to positive, indicating the development of antibodies in response to infection or immunization. [EU]

Serologic: Analysis of a person's serum, especially specific immune or lytic serums. [NIH]

Serology: The study of serum, especially of antigen-antibody reactions in vitro. [NIH]

Serotonin: A biochemical messenger and regulator, synthesized from the essential amino acid L-tryptophan. In humans it is found primarily in the central nervous system, gastrointestinal tract, and blood platelets. Serotonin mediates several important physiological functions including neurotransmission, gastrointestinal motility, hemostasis, and cardiovascular integrity. Multiple receptor families (receptors, serotonin) explain the broad physiological actions and distribution of this biochemical mediator. [NIH]

Serous: Having to do with serum, the clear liquid part of blood. [NIH]

Serum: The clear liquid part of the blood that remains after blood cells and clotting proteins have been removed. [NIH]

Sex Characteristics: Those characteristics that distinguish one sex from the other. The primary sex characteristics are the ovaries and testes and their related hormones. Secondary sex characteristics are those which are masculine or feminine but not directly related to reproduction. [NIH]

Sex Determination: The biological characteristics which distinguish human beings as female or male. [NIH]

Sex Ratio: The number of males per 100 females. [NIH]

Sexually Transmitted Diseases: Diseases due to or propagated by sexual contact. [NIH]

Shedding: Release of infectious particles (e. g., bacteria, viruses) into the environment, for example by sneezing, by fecal excretion, or from an open lesion. [NIH]

Shock: The general bodily disturbance following a severe injury; an emotional or moral upset occasioned by some disturbing or unexpected experience; disruption of the circulation, which can upset all body functions: sometimes referred to as circulatory shock. [NIH]

Shunt: A surgically created diversion of fluid (e.g., blood or cerebrospinal fluid) from one area of the body to another area of the body. [NIH]

Side effect: A consequence other than the one(s) for which an agent or measure is used, as the adverse effects produced by a drug, especially on a tissue or organ system other than the one sought to be benefited by its administration. [EU]

Signal Transduction: The intercellular or intracellular transfer of information (biological activation/inhibition) through a signal pathway. In each signal transduction system, an activation/inhibition signal from a biologically active molecule (hormone, neurotransmitter) is mediated via the coupling of a receptor/enzyme to a second messenger system or to an ion channel. Signal transduction plays an important role in activating cellular functions, cell differentiation, and cell proliferation. Examples of signal transduction systems are the GABA-postsynaptic receptor-calcium ion channel system, the receptor-mediated T-cell activation pathway, and the receptor-mediated activation of phospholipases. Those coupled to membrane depolarization or intracellular release of calcium include the receptor-mediated activation of cytotoxic functions in granulocytes and the synaptic potentiation of protein kinase activation. Some signal transduction pathways may be part of larger signal transduction pathways; for example, protein kinase activation is part of the platelet activation signal pathway. [NIH]

Signs and Symptoms: Clinical manifestations that can be either objective when observed by a physician, or subjective when perceived by the patient. [NIH]

Simvastatin: A derivative of lovastatin and potent competitive inhibitor of 3-hydroxy-3-methylglutaryl coenzyme A reductase (hydroxymethylglutaryl CoA reductases), which is the rate-limiting enzyme in cholesterol biosynthesis. It may also interfere with steroid hormone production. Due to the induction of hepatic LDL receptors, it increases breakdown of LDL-cholesterol (lipoproteins, LDL cholesterol). [NIH]

Skeletal: Having to do with the skeleton (boney part of the body). [NIH]

Skeleton: The framework that supports the soft tissues of vertebrate animals and protects many of their internal organs. The skeletons of vertebrates are made of bone and/or cartilage. [NIH]

Skull: The skeleton of the head including the bones of the face and the bones enclosing the brain. [NIH]

Sleep apnea: A serious, potentially life-threatening breathing disorder characterized by repeated cessation of breathing due to either collapse of the upper airway during sleep or absence of respiratory effort. [NIH]

Small intestine: The part of the digestive tract that is located between the stomach and the large intestine. [NIH]

Smallpox: A generalized virus infection with a vesicular rash. [NIH]

Smoking Cessation: Discontinuation of the habit of smoking, the inhaling and exhaling of tobacco smoke. [NIH]

Smooth muscle: Muscle that performs automatic tasks, such as constricting blood vessels. [NIH]

Sneezing: Sudden, forceful, involuntary expulsion of air from the nose and mouth caused by irritation to the mucous membranes of the upper respiratory tract. [NIH]

Snoring: Rough, noisy breathing during sleep, due to vibration of the uvula and soft palate. [NIH]

Soaps: Sodium or potassium salts of long chain fatty acids. These detergent substances are obtained by boiling natural oils or fats with caustic alkali. Sodium soaps are harder and are used as topical anti-infectives and vehicles in pills and liniments; potassium soaps are soft, used as vehicles for ointments and also as topical antimicrobials. [NIH]

Social Change: Social process whereby the values, attitudes, or institutions of society, such as education, family, religion, and industry become modified. It includes both the natural process and action programs initiated by members of the community. [NIH]

Social Environment: The aggregate of social and cultural institutions, forms, patterns, and processes that influence the life of an individual or community. [NIH]

Social Security: Government sponsored social insurance programs. [NIH]

Social Support: Support systems that provide assistance and encouragement to individuals with physical or emotional disabilities in order that they may better cope. Informal social support is usually provided by friends, relatives, or peers, while formal assistance is provided by churches, groups, etc. [NIH]

Socialism: A system of government in which means of production and distribution of goods are controlled by the state. [NIH]

Socioeconomic Factors: Social and economic factors that characterize the individual or group within the social structure. [NIH]

Sodium: An element that is a member of the alkali group of metals. It has the atomic symbol Na, atomic number 11, and atomic weight 23. With a valence of 1, it has a strong affinity for oxygen and other nonmetallic elements. Sodium provides the chief cation of the extracellular body fluids. Its salts are the most widely used in medicine. (From Dorland, 27th ed) Physiologically the sodium ion plays a major role in blood pressure regulation, maintenance of fluid volume, and electrolyte balance. [NIH]

Soft tissue: Refers to muscle, fat, fibrous tissue, blood vessels, or other supporting tissue of the body. [NIH]

Solid tumor: Cancer of body tissues other than blood, bone marrow, or the lymphatic system. [NIH]

Solitary Nucleus: Gray matter located in the dorsomedial part of the medulla oblongata associated with the solitary tract. The solitary nucleus receives inputs from most organ systems including the terminations of the facial, glossopharyngeal, and vagus nerves. It is a major coordinator of autonomic nervous system regulation of cardiovascular, respiratory, gustatory, gastrointestinal, and chemoreceptive aspects of homeostasis. The solitary nucleus is also notable for the large number of neurotransmitters which are found therein. [NIH]

Solvent: 1. Dissolving; effecting a solution. 2. A liquid that dissolves or that is capable of dissolving; the component of a solution that is present in greater amount. [EU]

Soma: The body as distinct from the mind; all the body tissue except the germ cells; all the axial body. [NIH]

Somatic: 1. Pertaining to or characteristic of the soma or body. 2. Pertaining to the body wall in contrast to the viscera. [EU]

Sotalol: An adrenergic beta-antagonist that is used in the treatment of life-threatening arrhythmias. [NIH]

Sound wave: An alteration of properties of an elastic medium, such as pressure, particle displacement, or density, that propagates through the medium, or a superposition of such alterations. [NIH]

Soy Proteins: Proteins which are present in or isolated from soybeans. [NIH]

Soybean Oil: Oil from soybean or soybean plant. [NIH]

Spasm: An involuntary contraction of a muscle or group of muscles. Spasms may involve skeletal muscle or smooth muscle. [NIH]

Specialist: In medicine, one who concentrates on 1 special branch of medical science. [NIH]

Species: A taxonomic category subordinate to a genus (or subgenus) and superior to a subspecies or variety, composed of individuals possessing common characters distinguishing them from other categories of individuals of the same taxonomic level. In taxonomic nomenclature, species are designated by the genus name followed by a Latin or Latinized adjective or noun. [EU]

Specificity: Degree of selectivity shown by an antibody with respect to the number and types of antigens with which the antibody combines, as well as with respect to the rates and the extents of these reactions. [NIH]

Spectrum: A charted band of wavelengths of electromagnetic vibrations obtained by refraction and diffraction. By extension, a measurable range of activity, such as the range of bacteria affected by an antibiotic (antibacterial s.) or the complete range of manifestations of a disease. [EU]

Sperm: The fecundating fluid of the male. [NIH]

Spices: The dried seeds, bark, root, stems, buds, leaves, or fruit of aromatic plants used to season food. [NIH]

Spina bifida: A defect in development of the vertebral column in which there is a central deficiency of the vertebral lamina. [NIH]

Spinal cord: The main trunk or bundle of nerves running down the spine through holes in the spinal bone (the vertebrae) from the brain to the level of the lower back. [NIH]

Spleen: An organ that is part of the lymphatic system. The spleen produces lymphocytes, filters the blood, stores blood cells, and destroys old blood cells. It is located on the left side of the abdomen near the stomach. [NIH]

Sporadic: Neither endemic nor epidemic; occurring occasionally in a random or isolated manner. [EU]

Stabilization: The creation of a stable state. [EU]

Staging: Performing exams and tests to learn the extent of the cancer within the body, especially whether the disease has spread from the original site to other parts of the body. [NIH]

Steel: A tough, malleable, iron-based alloy containing up to, but no more than, two percent carbon and often other metals. It is used in medicine and dentistry in implants and instrumentation. [NIH]

Stem Cells: Relatively undifferentiated cells of the same lineage (family type) that retain the ability to divide and cycle throughout postnatal life to provide cells that can become specialized and take the place of those that die or are lost. [NIH]

Stenosis: Narrowing or stricture of a duct or canal. [EU]

Stent: A device placed in a body structure (such as a blood vessel or the gastrointestinal tract) to provide support and keep the structure open. [NIH]

Sterility: 1. The inability to produce offspring, i.e., the inability to conceive (female s.) or to induce conception (male s.). 2. The state of being aseptic, or free from microorganisms. [EU]

Steroid: A group name for lipids that contain a hydrogenated cyclopentanoperhydrophenanthrene ring system. Some of the substances included in this group are progesterone, adrenocortical hormones, the gonadal hormones, cardiac aglycones, bile acids, sterols (such as cholesterol), toad poisons, saponins, and some of the carcinogenic hydrocarbons. [EU]

Steroid therapy: Treatment with corticosteroid drugs to reduce swelling, pain, and other symptoms of inflammation. [NIH]

Stimulant: 1. Producing stimulation; especially producing stimulation by causing tension on muscle fibre through the nervous tissue. 2. An agent or remedy that produces stimulation. [EU]

Stimulus: That which can elicit or evoke action (response) in a muscle, nerve, gland or other excitable issue, or cause an augmenting action upon any function or metabolic process. [NIH]

Stomach: An organ of digestion situated in the left upper quadrant of the abdomen between the termination of the esophagus and the beginning of the duodenum. [NIH]

Stool: The waste matter discharged in a bowel movement; feces. [NIH]

Strand: DNA normally exists in the bacterial nucleus in a helix, in which two strands are coiled together. [NIH]

Streptococcal: Caused by infection due to any species of streptococcus. [NIH]

Streptococcus: A genus of gram-positive, coccoid bacteria whose organisms occur in pairs or chains. No endospores are produced. Many species exist as commensals or parasites on man or animals with some being highly pathogenic. A few species are saprophytes and occur in the natural environment. [NIH]

Stress: Forcibly exerted influence; pressure. Any condition or situation that causes strain or tension. Stress may be either physical or psychologic, or both. [NIH]

Stress management: A set of techniques used to help an individual cope more effectively with difficult situations in order to feel better emotionally, improve behavioral skills, and often to enhance feelings of control. Stress management may include relaxation exercises, assertiveness training, cognitive restructuring, time management, and social support. It can be delivered either on a one-to-one basis or in a group format. [NIH]

Striatum: A higher brain's domain thus called because of its stripes. [NIH]

Stricture: The abnormal narrowing of a body opening. Also called stenosis. [NIH]

Stroke: Sudden loss of function of part of the brain because of loss of blood flow. Stroke may be caused by a clot (thrombosis) or rupture (hemorrhage) of a blood vessel to the brain. [NIH]

Stroma: The middle, thickest layer of tissue in the cornea. [NIH]

Subacute: Somewhat acute; between acute and chronic. [EU]

Subclinical: Without clinical manifestations; said of the early stage(s) of an infection or other disease or abnormality before symptoms and signs become apparent or detectable by clinical examination or laboratory tests, or of a very mild form of an infection or other disease or abnormality. [EU]

Subcutaneous: Beneath the skin. [NIH]

Sublingual: Located beneath the tongue. [EU]

Subspecies: A category intermediate in rank between species and variety, based on a smaller number of correlated characters than are used to differentiate species and generally conditioned by geographical and/or ecological occurrence. [NIH]

Substance P: An eleven-amino acid neurotransmitter that appears in both the central and peripheral nervous systems. It is involved in transmission of pain, causes rapid contractions of the gastrointestinal smooth muscle, and modulates inflammatory and immune responses. [NIH]

Sudden cardiac death: Cardiac arrest caused by an irregular heartbeat. [NIH]

Sudden death: Cardiac arrest caused by an irregular heartbeat. The term "death" is somewhat misleading, because some patients survive. [NIH]

Sulfur: An element that is a member of the chalcogen family. It has an atomic symbol S,

atomic number 16, and atomic weight 32.066. It is found in the amino acids cysteine and methionine. [NIH]

Superior vena cava: Vein which returns blood from the head and neck, upper limbs, and thorax. It is formed by the union of the two brachiocephalic veins. [NIH]

Superoxide: Derivative of molecular oxygen that can damage cells. [NIH]

Superoxide Dismutase: An oxidoreductase that catalyzes the reaction between superoxide anions and hydrogen to yield molecular oxygen and hydrogen peroxide. The enzyme protects the cell against dangerous levels of superoxide. EC 1.15.1.1. [NIH]

Supplementation: Adding nutrients to the diet. [NIH]

Support group: A group of people with similar disease who meet to discuss how better to cope with their cancer and treatment. [NIH]

Sympathetic Nervous System: The thoracolumbar division of the autonomic nervous system. Sympathetic preganglionic fibers originate in neurons of the intermediolateral column of the spinal cord and project to the paravertebral and prevertebral ganglia, which in turn project to target organs. The sympathetic nervous system mediates the body's response to stressful situations, i.e., the fight or flight reactions. It often acts reciprocally to the parasympathetic system. [NIH]

Sympathomimetic: 1. Mimicking the effects of impulses conveyed by adrenergic postganglionic fibres of the sympathetic nervous system. 2. An agent that produces effects similar to those of impulses conveyed by adrenergic postganglionic fibres of the sympathetic nervous system. Called also adrenergic. [EU]

Symphysis: A secondary cartilaginous joint. [NIH]

Symptomatic: Having to do with symptoms, which are signs of a condition or disease. [NIH]

Symptomatology: 1. That branch of medicine with treats of symptoms; the systematic discussion of symptoms. 2. The combined symptoms of a disease. [EU]

Synapse: The region where the processes of two neurons come into close contiguity, and the nervous impulse passes from one to the other; the fibers of the two are intermeshed, but, according to the general view, there is no direct contiguity. [NIH]

Synaptic: Pertaining to or affecting a synapse (= site of functional apposition between neurons, at which an impulse is transmitted from one neuron to another by electrical or chemical means); pertaining to synapsis (= pairing off in point-for-point association of homologous chromosomes from the male and female pronuclei during the early prophase of meiosis). [EU]

Synaptic Transmission: The communication from a neuron to a target (neuron, muscle, or secretory cell) across a synapse. In chemical synaptic transmission, the presynaptic neuron releases a neurotransmitter that diffuses across the synaptic cleft and binds to specific synaptic receptors. These activated receptors modulate ion channels and/or second-messenger systems to influence the postsynaptic cell. Electrical transmission is less common in the nervous system, and, as in other tissues, is mediated by gap junctions. [NIH]

Syncope: A temporary suspension of consciousness due to generalized cerebral schemia, a faint or swoon. [EU]

Synergistic: Acting together; enhancing the effect of another force or agent. [EU]

Systemic: Affecting the entire body. [NIH]

Systemic lupus erythematosus: SLE. A chronic inflammatory connective tissue disease marked by skin rashes, joint pain and swelling, inflammation of the kidneys, inflammation of the fibrous tissue surrounding the heart (i.e., the pericardium), as well as other problems.

Not all affected individuals display all of these problems. May be referred to as lupus. [NIH]

Systole: Period of contraction of the heart, especially of the ventricles. [NIH]

Systolic: Indicating the maximum arterial pressure during contraction of the left ventricle of the heart. [EU]

Systolic blood pressure: The maximum pressure in the artery produced as the heart contracts and blood begins to flow. [NIH]

Tachyarrhythmia: Tachycardia associated with an irregularity in the normal heart rhythm. [EU]

Tachycardia: Excessive rapidity in the action of the heart, usually with a heart rate above 100 beats per minute. [NIH]

Technetium: The first artificially produced element and a radioactive fission product of uranium. The stablest isotope has a mass number 99 and is used diagnostically as a radioactive imaging agent. Technetium has the atomic symbol Tc, atomic number 43, and atomic weight 98.91. [NIH]

Telangiectasia: The permanent enlargement of blood vessels, causing redness in the skin or mucous membranes. [NIH]

Telencephalon: Paired anteriolateral evaginations of the prosencephalon plus the lamina terminalis. The cerebral hemispheres are derived from it. Many authors consider cerebrum a synonymous term to telencephalon, though a minority include diencephalon as part of the cerebrum (Anthoney, 1994). [NIH]

Telomere: A terminal section of a chromosome which has a specialized structure and which is involved in chromosomal replication and stability. Its length is believed to be a few hundred base pairs. [NIH]

Temporal: One of the two irregular bones forming part of the lateral surfaces and base of the skull, and containing the organs of hearing. [NIH]

Teratogen: A substance which, through immediate, prolonged or repeated contact with the skin may involve a risk of subsequent non-hereditable birth defects in offspring. [NIH]

Terminator: A DNA sequence sited at the end of a transcriptional unit that signals the end of transcription. [NIH]

Testis: Either of the paired male reproductive glands that produce the male germ cells and the male hormones. [NIH]

Testosterone: A hormone that promotes the development and maintenance of male sex characteristics. [NIH]

Thalamic: Cell that reaches the lateral nucleus of amygdala. [NIH]

Thalamic Diseases: Disorders of the centrally located thalamus, which integrates a wide range of cortical and subcortical information. Manifestations include sensory loss, movement disorders; ataxia, pain syndromes, visual disorders, a variety of neuropsychological conditions, and coma. Relatively common etiologies include cerebrovascular disorders; craniocerebral trauma; brain neoplasms; brain hypoxia; intracranial hemorrhages; and infectious processes. [NIH]

Therapeutics: The branch of medicine which is concerned with the treatment of diseases, palliative or curative. [NIH]

Thermal: Pertaining to or characterized by heat. [EU]

Thigh: A leg; in anatomy, any elongated process or part of a structure more or less comparable to a leg. [NIH]

Thoracic: Having to do with the chest. [NIH]

Thorax: A part of the trunk between the neck and the abdomen; the chest. [NIH]

Threshold: For a specified sensory modality (e. g. light, sound, vibration), the lowest level (absolute threshold) or smallest difference (difference threshold, difference limen) or intensity of the stimulus discernible in prescribed conditions of stimulation. [NIH]

Thrombin: An enzyme formed from prothrombin that converts fibrinogen to fibrin. (Dorland, 27th ed) EC 3.4.21.5. [NIH]

Thrombocytes: Blood cells that help prevent bleeding by causing blood clots to form. Also called platelets. [NIH]

Thrombocytopenia: A decrease in the number of blood platelets. [NIH]

Thrombolytic: 1. Dissolving or splitting up a thrombus. 2. A thrombolytic agent. [EU]

Thrombomodulin: A cell surface glycoprotein of endothelial cells that binds thrombin and serves as a cofactor in the activation of protein C and its regulation of blood coagulation. [NIH]

Thrombophilia: A disorder of hemostasis in which there is a tendency for the occurrence of thrombosis. [NIH]

Thromboplastin: Constituent composed of protein and phospholipid that is widely distributed in many tissues. It serves as a cofactor with factor VIIa to activate factor X in the extrinsic pathway of blood coagulation. [NIH]

Thrombosis: The formation or presence of a blood clot inside a blood vessel. [NIH]

Thromboxanes: Physiologically active compounds found in many organs of the body. They are formed in vivo from the prostaglandin endoperoxides and cause platelet aggregation, contraction of arteries, and other biological effects. Thromboxanes are important mediators of the actions of polyunsaturated fatty acids transformed by cyclooxygenase. [NIH]

Thrombus: An aggregation of blood factors, primarily platelets and fibrin with entrapment of cellular elements, frequently causing vascular obstruction at the point of its formation. Some authorities thus differentiate thrombus formation from simple coagulation or clot formation. [EU]

Thymus: An organ that is part of the lymphatic system, in which T lymphocytes grow and multiply. The thymus is in the chest behind the breastbone. [NIH]

Thyroid: A gland located near the windpipe (trachea) that produces thyroid hormone, which helps regulate growth and metabolism. [NIH]

Thyroxine: An amino acid of the thyroid gland which exerts a stimulating effect on thyroid metabolism. [NIH]

Ticks: Blood-sucking arachnids of the order Acarina. [NIH]

Time Management: Planning and control of time to improve efficiency and effectiveness. [NIH]

Tin: A trace element that is required in bone formation. It has the atomic symbol Sn, atomic number 50, and atomic weight 118.71. [NIH]

Tissue: A group or layer of cells that are alike in type and work together to perform a specific function. [NIH]

Tolerance: 1. The ability to endure unusually large doses of a drug or toxin. 2. Acquired drug tolerance; a decreasing response to repeated constant doses of a drug or the need for increasing doses to maintain a constant response. [EU]

Tomography: Imaging methods that result in sharp images of objects located on a chosen

plane and blurred images located above or below the plane. [NIH]

Tone: 1. The normal degree of vigour and tension; in muscle, the resistance to passive elongation or stretch; tonus. 2. A particular quality of sound or of voice. 3. To make permanent, or to change, the colour of silver stain by chemical treatment, usually with a heavy metal. [EU]

Tonometry: The standard to determine the fluid pressure inside the eye (intraocular pressure). [NIH]

Tonus: A state of slight tension usually present in muscles even when they are not undergoing active contraction. [NIH]

Tooth Loss: The failure to retain teeth as a result of disease or injury. [NIH]

Tooth Preparation: Procedures carried out with regard to the teeth or tooth structures preparatory to specified dental therapeutic and surgical measures. [NIH]

Topical: On the surface of the body. [NIH]

Torsion: A twisting or rotation of a bodily part or member on its axis. [NIH]

Toxaemia: 1. The condition resulting from the spread of bacterial products (toxins) by the bloodstream. 2. A condition resulting from metabolic disturbances, e.g. toxaemia of pregnancy. [EU]

Toxic: Having to do with poison or something harmful to the body. Toxic substances usually cause unwanted side effects. [NIH]

Toxicity: The quality of being poisonous, especially the degree of virulence of a toxic microbe or of a poison. [EU]

Toxicologic: Pertaining to toxicology. [EU]

Toxicology: The science concerned with the detection, chemical composition, and pharmacologic action of toxic substances or poisons and the treatment and prevention of toxic manifestations. [NIH]

Toxin: A poison; frequently used to refer specifically to a protein produced by some higher plants, certain animals, and pathogenic bacteria, which is highly toxic for other living organisms. Such substances are differentiated from the simple chemical poisons and the vegetable alkaloids by their high molecular weight and antigenicity. [EU]

Toxoplasmosis: The acquired form of infection by Toxoplasma gondii in animals and man. [NIH]

Trace element: Substance or element essential to plant or animal life, but present in extremely small amounts. [NIH]

Tracer: A substance (such as a radioisotope) used in imaging procedures. [NIH]

Trachea: The cartilaginous and membranous tube descending from the larynx and branching into the right and left main bronchi. [NIH]

Traction: The act of pulling. [NIH]

Transcriptase: An enzyme which catalyses the synthesis of a complementary mRNA molecule from a DNA template in the presence of a mixture of the four ribonucleotides (ATP, UTP, GTP and CTP). [NIH]

Transcription Factors: Endogenous substances, usually proteins, which are effective in the initiation, stimulation, or termination of the genetic transcription process. [NIH]

Transfection: The uptake of naked or purified DNA into cells, usually eukaryotic. It is analogous to bacterial transformation. [NIH]

Transfer Factor: Factor derived from leukocyte lysates of immune donors which can transfer

both local and systemic cellular immunity to nonimmune recipients. [NIH]

Transforming Growth Factor beta: A factor synthesized in a wide variety of tissues. It acts synergistically with TGF-alpha in inducing phenotypic transformation and can also act as a negative autocrine growth factor. TGF-beta has a potential role in embryonal development, cellular differentiation, hormone secretion, and immune function. TGF-beta is found mostly as homodimer forms of separate gene products TGF-beta1, TGF-beta2 or TGF-beta3. Heterodimers composed of TGF-beta1 and 2 (TGF-beta1.2) or of TGF-beta2 and 3 (TGF-beta2.3) have been isolated. The TGF-beta proteins are synthesized as precursor proteins. [NIH]

Transfusion: The infusion of components of blood or whole blood into the bloodstream. The blood may be donated from another person, or it may have been taken from the person earlier and stored until needed. [NIH]

Transgenes: Genes that are introduced into an organism using gene transfer techniques. [NIH]

Translation: The process whereby the genetic information present in the linear sequence of ribonucleotides in mRNA is converted into a corresponding sequence of amino acids in a protein. It occurs on the ribosome and is unidirectional. [NIH]

Translational: The cleavage of signal sequence that directs the passage of the protein through a cell or organelle membrane. [NIH]

Transmitter: A chemical substance which effects the passage of nerve impulses from one cell to the other at the synapse. [NIH]

Transplantation: Transference of a tissue or organ, alive or dead, within an individual, between individuals of the same species, or between individuals of different species. [NIH]

Trauma: Any injury, wound, or shock, must frequently physical or structural shock, producing a disturbance. [NIH]

Tremor: Cyclical movement of a body part that can represent either a physiologic process or a manifestation of disease. Intention or action tremor, a common manifestation of cerebellar diseases, is aggravated by movement. In contrast, resting tremor is maximal when there is no attempt at voluntary movement, and occurs as a relatively frequent manifestation of Parkinson disease. [NIH]

Trichloroethylene: A highly volatile inhalation anesthetic used mainly in short surgical procedures where light anesthesia with good analgesia is required. It is also used as an industrial solvent. Prolonged exposure to high concentrations of the vapor can lead to cardiotoxicity and neurological impairment. [NIH]

Tricyclic: Containing three fused rings or closed chains in the molecular structure. [EU]

Triglyceride: A lipid carried through the blood stream to tissues. Most of the body's fat tissue is in the form of triglycerides, stored for use as energy. Triglycerides are obtained primarily from fat in foods. [NIH]

Trimetazidine: A vasodilator used in angina of effort or ischemic heart disease. [NIH]

Trisomy: The possession of a third chromosome of any one type in an otherwise diploid cell. [NIH]

Tropomyosin: A protein found in the thin filaments of muscle fibers. It inhibits contraction of the muscle unless its position is modified by troponin. [NIH]

Troponin: One of the minor protein components of skeletal muscle. Its function is to serve as the calcium-binding component in the troponin-tropomyosin B-actin-myosin complex by conferring calcium sensitivity to the cross-linked actin and myosin filaments. [NIH]

Troponin C: One of the three polypeptide chains that make up the troponin complex of

skeletal muscle. It is a calcium-binding protein. [NIH]

Troponin I: One of the three polypeptide chains that make up the troponin complex. It inhibits F-actin-myosin interactions. [NIH]

Tryptophan: An essential amino acid that is necessary for normal growth in infants and for nitrogen balance in adults. It is a precursor serotonin and niacin. [NIH]

Tuberous Sclerosis: A rare congenital disease in which the essential pathology is the appearance of multiple tumors in the cerebrum and in other organs, such as the heart or kidneys. [NIH]

Tumor marker: A substance sometimes found in an increased amount in the blood, other body fluids, or tissues and which may mean that a certain type of cancer is in the body. Examples of tumor markers include CA 125 (ovarian cancer), CA 15-3 (breast cancer), CEA (ovarian, lung, breast, pancreas, and gastrointestinal tract cancers), and PSA (prostate cancer). Also called biomarker. [NIH]

Tumor Necrosis Factor: Serum glycoprotein produced by activated macrophages and other mammalian mononuclear leukocytes which has necrotizing activity against tumor cell lines and increases ability to reject tumor transplants. It mimics the action of endotoxin but differs from it. It has a molecular weight of less than 70,000 kDa. [NIH]

Tunica: A rather vague term to denote the lining coat of hollow organs, tubes, or cavities. [NIH]

Type 2 diabetes: Usually characterized by a gradual onset with minimal or no symptoms of metabolic disturbance and no requirement for exogenous insulin. The peak age of onset is 50 to 60 years. Obesity and possibly a genetic factor are usually present. [NIH]

Tyrosine: A non-essential amino acid. In animals it is synthesized from phenylalanine. It is also the precursor of epinephrine, thyroid hormones, and melanin. [NIH]

Ulcer: A localized necrotic lesion of the skin or a mucous surface. [NIH]

Ulceration: 1. The formation or development of an ulcer. 2. An ulcer. [EU]

Ultrasonography: The visualization of deep structures of the body by recording the reflections of echoes of pulses of ultrasonic waves directed into the tissues. Use of ultrasound for imaging or diagnostic purposes employs frequencies ranging from 1.6 to 10 megahertz. [NIH]

Unconscious: Experience which was once conscious, but was subsequently rejected, as the "personal unconscious". [NIH]

Unsaturated Fats: A type of fat. [NIH]

Uranium: A radioactive element of the actinide series of metals. It has an atomic symbol U, atomic number 92, and atomic weight 238.03. U-235 is used as the fissionable fuel in nuclear weapons and as fuel in nuclear power reactors. [NIH]

Uremia: The illness associated with the buildup of urea in the blood because the kidneys are not working effectively. Symptoms include nausea, vomiting, loss of appetite, weakness, and mental confusion. [NIH]

Urethra: The tube through which urine leaves the body. It empties urine from the bladder. [NIH]

Urinalysis: Examination of urine by chemical, physical, or microscopic means. Routine urinalysis usually includes performing chemical screening tests, determining specific gravity, observing any unusual color or odor, screening for bacteriuria, and examining the sediment microscopically. [NIH]

Urinary: Having to do with urine or the organs of the body that produce and get rid of

urine. [NIH]

Urinate: To release urine from the bladder to the outside. [NIH]

Urine: Fluid containing water and waste products. Urine is made by the kidneys, stored in the bladder, and leaves the body through the urethra. [NIH]

Uterus: The small, hollow, pear-shaped organ in a woman's pelvis. This is the organ in which a fetus develops. Also called the womb. [NIH]

Uvula: Uvula palatinae; specifically, the tongue-like process which projects from the middle of the posterior edge of the soft palate. [NIH]

Vaccine: A substance or group of substances meant to cause the immune system to respond to a tumor or to microorganisms, such as bacteria or viruses. [NIH]

Vagal: Pertaining to the vagus nerve. [EU]

Vagus Nerve: The 10th cranial nerve. The vagus is a mixed nerve which contains somatic afferents (from skin in back of the ear and the external auditory meatus), visceral afferents (from the pharynx, larynx, thorax, and abdomen), parasympathetic efferents (to the thorax and abdomen), and efferents to striated muscle (of the larynx and pharynx). [NIH]

Valves: Flap-like structures that control the direction of blood flow through the heart. [NIH]

Vascular: Pertaining to blood vessels or indicative of a copious blood supply. [EU]

Vascular endothelial growth factor: VEGF. A substance made by cells that stimulates new blood vessel formation. [NIH]

Vascular Resistance: An expression of the resistance offered by the systemic arterioles, and to a lesser extent by the capillaries, to the flow of blood. [NIH]

Vasculitis: Inflammation of a blood vessel. [NIH]

Vasoconstriction: Narrowing of the blood vessels without anatomic change, for which constriction, pathologic is used. [NIH]

Vasodilation: Physiological dilation of the blood vessels without anatomic change. For dilation with anatomic change, dilatation, pathologic or aneurysm (or specific aneurysm) is used. [NIH]

Vasodilator: An agent that widens blood vessels. [NIH]

Vasomotor: 1. Affecting the calibre of a vessel, especially of a blood vessel. 2. Any element or agent that effects the calibre of a blood vessel. [EU]

VE: The total volume of gas either inspired or expired in one minute. [NIH]

Vector: Plasmid or other self-replicating DNA molecule that transfers DNA between cells in nature or in recombinant DNA technology. [NIH]

Vein: Vessel-carrying blood from various parts of the body to the heart. [NIH]

Vena: A vessel conducting blood from the capillary bed to the heart. [NIH]

Venous: Of or pertaining to the veins. [EU]

Venous blood: Blood that has given up its oxygen to the tissues and carries carbon dioxide back for gas exchange. [NIH]

Venter: Belly. [NIH]

Ventilation: 1. In respiratory physiology, the process of exchange of air between the lungs and the ambient air. Pulmonary ventilation (usually measured in litres per minute) refers to the total exchange, whereas alveolar ventilation refers to the effective ventilation of the alveoli, in which gas exchange with the blood takes place. 2. In psychiatry, verbalization of one's emotional problems. [EU]

Ventral: 1. Pertaining to the belly or to any venter. 2. Denoting a position more toward the belly surface than some other object of reference; same as anterior in human anatomy. [EU]

Ventricle: One of the two pumping chambers of the heart. The right ventricle receives oxygen-poor blood from the right atrium and pumps it to the lungs through the pulmonary artery. The left ventricle receives oxygen-rich blood from the left atrium and pumps it to the body through the aorta. [NIH]

Ventricular: Pertaining to a ventricle. [EU]

Ventricular Dysfunction: A condition in which the ventricles of the heart exhibit a decreased functionality. [NIH]

Ventricular Function: The hemodynamic and electrophysiological action of the ventricles. [NIH]

Ventricular Function, Left: The hemodynamic and electrophysiological action of the left ventricle. Its measurement is an important aspect of the clinical evaluation of patients with heart disease to determine the effects of the disease on cardiac performance. [NIH]

Ventricular Pressure: The pressure within a cardiac ventricle. Ventricular pressure waveforms can be measured in the beating heart by catheterization or estimated using imaging techniques (e.g., Doppler echocardiography). The information is useful in evaluating the function of the myocardium, cardiac valves, and pericardium, particularly with simultaneous measurement of other (e.g., aortic or atrial) pressures. [NIH]

Ventricular Remodeling: The geometric and structural changes that the ventricle undergoes, usually following myocardial infarction. It comprises expansion of the infarct and dilatation of the healthy ventricle segments. While most prevalent in the left ventricle, it can also occur in the right ventricle. [NIH]

Venules: The minute vessels that collect blood from the capillary plexuses and join together to form veins. [NIH]

Vertebrae: A bony unit of the segmented spinal column. [NIH]

Vertebral: Of or pertaining to a vertebra. [EU]

Very low-density lipoprotein: The lipoprotein particles that initially leave the liver, carrying cholesterol and lipid. VLDLs contain 10 to 15 percent of the total serum cholesterol along with most of the triglycerides in the fasting serum; VLDLs are precursors of LDL, and some forms of VLDL, particularly VLDL remnants, appear to be atherogenic. [NIH]

Vesicular: 1. Composed of or relating to small, saclike bodies. 2. Pertaining to or made up of vesicles on the skin. [EU]

Veterinary Medicine: The medical science concerned with the prevention, diagnosis, and treatment of diseases in animals. [NIH]

Viral: Pertaining to, caused by, or of the nature of virus. [EU]

Viral vector: A type of virus used in cancer therapy. The virus is changed in the laboratory and cannot cause disease. Viral vectors produce tumor antigens (proteins found on a tumor cell) and can stimulate an antitumor immune response in the body. Viral vectors may also be used to carry genes that can change cancer cells back to normal cells. [NIH]

Virulence: The degree of pathogenicity within a group or species of microorganisms or viruses as indicated by case fatality rates and/or the ability of the organism to invade the tissues of the host. [NIH]

Virus: Submicroscopic organism that causes infectious disease. In cancer therapy, some viruses may be made into vaccines that help the body build an immune response to, and kill, tumor cells. [NIH]

Visceral: , from viscus a viscus) pertaining to a viscus. [EU]

Visceral Afferents: The sensory fibers innervating the viscera. [NIH]

Viscosity: A physical property of fluids that determines the internal resistance to shear forces. [EU]

Vitamin A: A substance used in cancer prevention; it belongs to the family of drugs called retinoids. [NIH]

Vitamin K: A substance that promotes the clotting of blood. [NIH]

Vitreous: Glasslike or hyaline; often used alone to designate the vitreous body of the eye (corpus vitreum). [EU]

Vitreous Body: The transparent, semigelatinous substance that fills the cavity behind the crystalline lens of the eye and in front of the retina. It is contained in a thin hyoid membrane and forms about four fifths of the optic globe. [NIH]

Vitro: Descriptive of an event or enzyme reaction under experimental investigation occurring outside a living organism. Parts of an organism or microorganism are used together with artificial substrates and/or conditions. [NIH]

Vivo: Outside of or removed from the body of a living organism. [NIH]

Void: To urinate, empty the bladder. [NIH]

Waist circumference: To define the level at which the waist circumference is measured, a bony landmark is first located and marked. The subject stands, and the technician, positioned to the right of the subject, palpates the upper hip bone to locate the right ileum. Just above the uppermost lateral border of the right ileum, a horizontal mark is drawn and then crossed with a vertical mark on the midaxillary line. The measuring tape is then placed around the trunk, at the level of the mark on the right side, making sure that it is on a level horizontal plane on all sides. The tape is then tightened slightly without compressing the skin and underlying subcutaneous tissues. The measure is recorded in centimeters to the nearest millimeter. [NIH]

Walkers: Walking aids generally having two handgrips and four legs. [NIH]

Weight Gain: Increase in body weight over existing weight. [NIH]

Wheezing: Breathing with a rasp or whistling sound; a sign of airway constriction or obstruction. [NIH]

White blood cell: A type of cell in the immune system that helps the body fight infection and disease. White blood cells include lymphocytes, granulocytes, macrophages, and others. [NIH]

Windpipe: A rigid tube, 10 cm long, extending from the cricoid cartilage to the upper border of the fifth thoracic vertebra. [NIH]

Wound Healing: Restoration of integrity to traumatized tissue. [NIH]

Xenograft: The cells of one species transplanted to another species. [NIH]

Xerostomia: Decreased salivary flow. [NIH]

X-ray: High-energy radiation used in low doses to diagnose diseases and in high doses to treat cancer. [NIH]

Yeasts: A general term for single-celled rounded fungi that reproduce by budding. Brewers' and bakers' yeasts are Saccharomyces cerevisiae; therapeutic dried yeast is dried yeast. [NIH]

Zebrafish: A species of North American fishes of the family Cyprinidae. They are used in embryological studies and to study the effects of certain chemicals on development. [NIH]

Zymogen: Inactive form of an enzyme which can then be converted to the active form,

usually by excision of a polypeptide, e. g. trypsinogen is the zymogen of trypsin. [NIH]

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