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Prevention of Leading Work-Related Diseases and Injuries

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Leading Work-Related Diseases and Injuries — United States

The National Institute for Occupational Safety and Health (NIOSH) has recently developed a suggested list of the 10 leading work-related diseases and injuries (Table 1). Three criteria were used to develop the list: the disease's or injury's frequency of occurrence, its severity in the individual case, and its amenability to prevention. The list is suggested with three purposes: 1) to encourage deliberation and debate among professionals about the major problems in this field of public health, 2) to assist in setting national priorities for efforts to prevent health problems related to work, and 3) to convey to a diverse audience the concerns of the leadership of NIOSH and the focus of the Institute's activities. The list is intended to be dynamic; it will be reviewed periodically for necessary updating as knowledge increases and as conditions change and are brought under better control.

Table 1. The 10 leading work-related diseases and injuries — United States, 1982*

1. Occupational lung diseases: asbestosis, byssinosis, silicosis, coal workers' pneumoconiosis, lung cancer, occupational asthma
2. Musculoskeletal injuries: disorders of the back, trunk, upper extremity, neck, lower extremity; traumatically induced Raynaud's phenomenon
3. Occupational cancers (other than lung): leukemia; mesothelioma; cancers of the bladder, nose, and liver
4. Severe occupational traumatic injuries: amputations, fractures, eye loss, lacerations, and traumatic deaths
5. Cardiovascular diseases: hypertension, coronary artery disease, acute myocardial infarction
6. Disorders of reproduction: infertility, spontaneous abortion, teratogenesis
7. Neurotoxic disorders: peripheral neuropathy, toxic encephalitis, psychoses, extreme personality changes (exposure-related)
8. Noise-induced loss of hearing
9. Dermatologic conditions: dermatoses, burns (scaldings), chemical burns, contusions (abrasions)
10. Psychologic disorders: neuroses, personality disorders, alcoholism, drug dependency

*The conditions listed under each category are to be viewed as *selected examples*, not comprehensive definitions of the category.

The following articles contain detailed discussions of the 10 leading work-related diseases and injuries.

OCCUPATIONAL LUNG DISEASES

The lung is both a target organ and a portal of entry for toxic substances. The likelihood of toxic exposure is high; for example, an estimated 1.2 million workers each year are potentially exposed to silica dust alone (*2*). The recognition of occupational lung diseases may be difficult, since the latent period for such diseases may be long—as long as 15 years for silicosis and 30 years or more for asbestos-related diseases. Other factors, such as cigarette smoking, may also contribute significantly to the disease process and hence obscure the association between work and the disease (*3*).

Six important components of occupational lung diseases are described below. Each is preventable, although years of effective control measures will be required to eliminate diseases of long latency. Because of the rapid rate at which new potentially toxic agents are introduced into the workplace, vigorous pre-market toxicologic testing of agents and effective disease surveillance are essential if epidemics of occupational lung diseases are to be avoided. The U.S. Public Health Service has established the following national objective for the prevention of occupational lung diseases: "by 1990, among workers newly exposed after 1985, there should be virtually no new cases of four preventable occupational diseases—asbestosis, byssinosis, silicosis, and coal workers' pneumoconiosis" (*4*). These diseases, as well as lung cancer and occupational asthma, are briefly discussed below.

Asbestosis: Asbestosis is characterized by diffuse, extensive scarring of the lung and progressive shortness of breath. Once established, the disease progresses even after exposure ends; there is no specific treatment. The latent period is 10-20 years. Smoking appears to increase the risk of death from asbestosis by a factor of two to three. Longitudinal studies of groups of asbestos insulation workers and shipyard workers have revealed that 10%-18% may be expected to die of asbestosis (*5*).

Byssinosis: This condition, characterized by both acute (reversible) and chronic lung disease, is associated with inhalation of the dusts of cotton, flax, or hemp. Symptoms include "chest tightness," cough, and obstruction of the small airways. Severely impaired lung function has disabled an estimated 35,000 current and retired textile workers (*6*). The specific causal agent(s) in the various dusts are yet to be identified (*7*).

Silicosis: Although the ill effects of exposure to free crystalline silica have been known for centuries, the prevalence of disabling silicosis remains high in certain groups of workers (*8*). Nearly 60,000 currently exposed workers in mines and foundries, in abrasive blasting operations, and in stone, clay, and glass manufacturing may be expected to suffer some degree of silicosis (*9*).

Coal workers' pneumoconiosis (CWP): The estimated prevalence of CWP among currently employed coal miners is about 4.5%. Approximately 0.2% of coal workers have been diagnosed as having progressive massive fibrosis, a potentially disabling form of CWP (*10*). In 1974, there were an estimated 19,400 cases of CWP. Some 4,000 deaths each year are attributed to legislatively defined "black lung disease" (*9*). Industrial bronchitis, another medical condition associated with exposure to coal dust, may lead to decreased ventilation capacity, but it is not well correlated with chest roentgenographic changes (*11*).

Lung cancer: The single most important cause of lung cancer is tobacco smoke (*12*). However, numerous occupational agents are associated with lung cancer, including arsenic, asbestos, chloroethers, chromates, ionizing radiation, nickel, and polynuclear aromatic hydrocarbon compounds (*13*). Tobacco smoke may interact synergistically with some of these agents (e.g., asbestos) to sharply increase the risk (*5*). Of special concern in this regard are workers currently or previously exposed to asbestos (estimated from 7.6 to 13.2 million) (*14, 15*); as many as 6,000 asbestos-related lung cancers may occur annually (*15*).

Occupational asthma: Hypersensitivity reactions to a wide variety of occupational organic and inorganic agents can cause asthma and hypersensitivity pneumonitis. The prevalence of occupational asthma varies from 10% to nearly 100% of workers in certain occupations (*16*). Many agents are incriminated as etiologic for occupational asthma, including grain dusts, flour, metals, inorganic chemicals, isocyanates, enzymes, and fungi. The list of agents associated with hypersensitivity pneumonitis is also long. If exposure continues, these conditions may result in progressive, irreversible pulmonary fibrosis.

Reported by Div of Surveillance, Hazard Evaluation, and Field Studies, Office of Director, NIOSH, CDC.

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MUSCULOSKELETAL INJURIES

In 1982, musculoskeletal injuries accounted for 580,000 (18%) of the estimated 3.2 million emergency-room-treated occupational injuries in the United States (*2*). Physical demands of many jobs make the musculoskeletal system highly vulnerable to a variety of occupational injuries and illnesses. Manual handling of materials, repetitive motions, and vibration are especially important etiologic factors in the development of these disorders.

Injuries associated with the manual handling of materials (e.g., unaided lifting and lowering): Low back injuries, often due to improper manual handling of materials, are the largest single subset of musculoskeletal injuries. The Bureau of Labor Statistics recently reported that approximately one million workers sustained back injuries in 1980 and that back injuries account for one of every five injuries and illnesses in the workplace. Approximately one-fourth of all workers' compensation indemnity expenditures in eight states were for back injuries (*3*).

Repetitive motion-associated trauma: Repetitive motion can cause "cumulative trauma disorders," including carpal tunnel syndrome, tendinitis, ganglionitis, tenosynovitis, bursitis, and epicondylitis. These disorders may be caused or aggravated by repeated twisting or awkward postures, particularly when combined with high force. The population at risk includes persons employed in such industries or occupations as construction, food preparation, clerical work, product fabrication, and mining.

Data from the National Occupational Hazard Survey suggest that 15%-20% of workers in these jobs are potentially at risk of cumulative trauma disorders (*4*). Data from the Bureau of Labor Statistics indicate that in 1980 approximately 23,200 occupational injuries were associated with repeated trauma (*5*).

Vibration-associated injuries: An estimated seven million workers in such occupations as vehicle operation are intermittently exposed to whole-body vibration, which significantly stresses the musculoskeletal system (*6*). Although the effects are poorly understood, preliminary data suggest that low back pain, vertebrogenic pain, and degenerative disk disease may be associated with whole-body vibration (*7,8*).

An estimated 1.2 million workers are exposed to "segmental" vibration, i.e., vibration principally of a part or parts of the body, of which the principal sources are handheld power tools, such as chain saws and jackhammers (*9*). These exposures are associated with "vibration syndrome," characterized by intermittent numbness and blanching of the fingers with reduced sensitivity to heat, cold, and pain (*10*). Vibration syndrome may affect up to 90% of workers in such occupations as chipping, grinding, and chain sawing (*11*).

Editorial Note: Musculoskeletal injuries can be prevented or reduced with such appropriate intervention measures as:

1. Substitution. Machines, such as hoists, cranes, and dollies, can substitute for workers in some aspects of the manual handling of materials.
2. Improved equipment design. Research has shown that improved design of some vibrating tools virtually eliminates hazardous vibration; suspension or isolation systems may be added to vehicles to greatly reduce whole-body vibration.
3. Task design. Manual tasks can be altered to minimize biomechanical stress to the worker (*12*).

4. Worker education. Injuries due to musculoskeletal stresses may be reduced by preplacement strength testing, training in proper ways to do a task, and on-site programs of exercise and physical therapy.
5. Variation of work practices. Periodic rotation of workers into jobs with different physical demands may help reduce the sequelae of biomechanical stress.

Reported by Div of Surveillance, Hazard Evaluations, and Field Studies, Div of Safety Research, NIOSH, CDC

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OCCUPATIONAL CANCERS (OTHER THAN LUNG)

Cancer kills approximately 430,000 people in the United States annually; the American Cancer Society estimates that some form of cancer will develop in one-fourth of all Americans (*3*). It is the second leading cause of death and the second leading cause of lost years of potential life in this country (*4*). A high proportion of all cancers are thought to be caused by "extragenetic" factors, including behaviors (e.g., cigarette smoking, alcohol and drug use, and sexual activities) and toxic environmental exposures in the workplace and the community (*5*). Evidence for these relationships has been developed principally through epidemiologic and toxicologic studies. The main epidemiologic observations have included: differences in the incidence of cancer between groups with different exposures, changes in the incidence of cancer following migrations, changes in the incidence of cancer over time, etc. Toxicologic studies have led to the identification of specific agents that cause cancer in experimental animals (*5*).

A possible occupational origin for malignant disease was first recognized when an unusually high frequency of scrotal cancer was observed among London chimney sweeps in 1775 (*6*). Since then, several types of cancer have been associated with industrial agents or processes (Table 1) (*7*). Numerous other occupational agents—such as beryllium, cadmium, ethylene oxide, phenoxy-acetic acids, and chlorophenols—or processes—such as newsprint pressroom work—are suspected of being carcinogenic and are under investigation by NIOSH.

Although general agreement exists concerning the overall incidence of cancer, considerable controversy surrounds the proportion of cancer cases attributable to occupational exposures. Several characteristics of cancer contribute to the difficulty in making such estimates:

1. Latency in the development of cancer. Occupational cancer usually becomes evident long after initial exposure to the carcinogen; this interval may vary from 5 years to more than 40 years (*9*), making it difficult to characterize important exposures long past.
2. Influence of exposures to multiple carcinogens. Cancer victims may have been occupationally exposed to many carcinogens; interaction of these agents or interactions between them and other factors may greatly increase the risk of cancer (*10*).
3. Influence of behavioral factors. Cigarette smoking, alcohol drinking, and dietary habits also influence the development of cancer (*11*). Moreover, these factors—especially cigarette smoking—interact with chemical and physical agents in the working environment to increase the risk of cancer (*12*); e.g., exposure to asbestos interacts with cigarette smoking to greatly increase the risk of lung cancer.

In addition, problems with the documentation of cancer and the nature and extent of etiologic exposures obscure important epidemiologic associations:

1. Errors in diagnosis and classification of cancer. Unusual neoplasms are often misdiagnosed; even correct diagnoses may be improperly categorized according to the International Classification of Diseases (ICD); an example is mesothelioma (*10*).
2. Lack of meaningful occupational histories. In only a few states is information collected on the work histories of cancer victims; hence, for many cases, crucial associations with occupational carcinogens are not apparent.

3. Difficulty in assessing exposures quantitatively. Precise measurements of levels and duration of exposures have not generally been available (*13*). Consequently, the ability to delineate dose-response relationships has been very limited.
4. The frequency of specific types of cancers. The occupational etiology of a very rare cancer due to a specific agent (e.g., hemangiosarcoma of the liver due to vinyl chloride) is much more readily documented than the occupational etiology of a cancer type potentially caused by several factors (e.g., lung cancer associated with exposure to chromates).
5. The "dilution factor." Highly significant differences in the rates of cancer among small subgroups of a population may be overlooked because these rates affect the overall rate for cancer in the larger study population only slightly, if at all (*8*).

Despite these difficulties, various attempts have been made to estimate the proportion of cancers related to occupation. These estimates span a broad range, from less than 4% (*5, 14*) to more than 20% (*15*). While these estimates are obviously imprecise, little doubt remains that occupational factors are significantly related to an increased risk of cancer. Moreover, in

Table 1. Selected occupational cancers*

ICD-9†	Condition	Industry/occupation	Agent
155	Hemangiosarcoma of the liver	Vinyl chloride polymerization Industry vintners	Vinyl chloride monomer Arsenical pesticides
160.0	Malignant neoplasm of nasal cavities	Woodworkers, cabinet/furniture makers Boot and shoe producers Radium chemists, processors, dial painters Nickel smelting and refining	Hardwood dusts Unknown Radium Nickel
161	Malignant neoplasm of larynx	Asbestos industries and utilizers	Asbestos
158, 163	Mesothelioma (peritoneum) (pleura)	Asbestos industries and utilizers	Asbestos
170	Malignant neoplasm of bone	Radium chemists, processors, dial painters	Radium
187.7	Malignant neoplasm of scrotum	Automatic lathe operators, metalworkers Coke oven workers, petroleum refiners, tar distillers	Mineral/cutting oils Soots and tars, tar distillates
188	Malignant neoplasm of bladder	Rubber and dye workers	Benzidine, alpha and beta naphthylamine, auramine, magenta, 4-aminobiphenyl, 4-nitrophenyl
189	Malignant neoplasm of kidney, other, and unspecified urinary organs	Coke oven workers	Coke oven emissions
204	Lymphoid leukemia, acute	Rubber industry Radiologists	Unknown Ionizing radiation
205	Myeloid leukemia, acute	Occupations with exposure to benzene Radiologists	Benzene Ionizing radiation
207.0	Erythroleukemia	Occupations with exposure to benzene	Benzene

*Adapted from reference 7. †Modified International Classification of Diseases (ICD) rubric.

specific groups of workers exposed to specific carcinogens, the proportion who ultimately develop occupational cancer may be large. Of one group of workers distilling beta-naphthylamine who had more than 5 years of exposure, all reportedly developed tumors of the bladder (*17*); up to 11% of workers exposed to asbestos may ultimately develop mesothelial tumors (*16*).

Reported by Div of Surveillance, Hazard Evaluations, and Field Studies, NIOSH, CDC.

Editorial Note: Cancer caused by occupational agents, especially synthetic chemicals, is a problem of human origin, and should, therefore, be preventable. Substitution of noncarcinogens for carcinogens, enforcement of protective standards for exposure, design and application of engineering controls, and use of personal protective equipment by exposed workers are major modes of prevention.

Although it is difficult to predict a trend for the future incidence of occupational cancer, the increased volume and diversity of synthetic chemicals manufactured since World War II (*18*) raise serious concern about the risks from exposure to these substances. However, improved control technology, governmental regulatory activity to reduce exposures, surveillance of disease and risk factors, and vigilant use of preventive measures will ultimately reduce occupational cancer.

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*Additional references are available on request from the National Institute for Occupational Safety and Health, CDC.

SEVERE OCCUPATIONAL TRAUMATIC INJURIES

Severe occupational traumatic injuries usually occur suddenly on the job and are either fatal or require immediate medical care. Data on these events are available from several different sources, including: the National Electronic Injury Surveillance System (NEISS) of the Consumer Product Safety Commission (*4*); the Supplementary Data System (SDS) of the Bureau of Labor Statistics (BLS) (*5*); the Annual Survey of Occupational Injuries and Illnesses conducted by the BLS (*6*); and the National Safety Council (*7*). The National Safety Council and the Annual Survey of the BLS estimate occupational traumatic deaths.

These sources report different aspects of the problem because of differences in the scope of events that each system tries to reflect. NEISS reports cases of occupational trauma from a representative sample of U.S. hospital emergency rooms. SDS records information from Workers' Compensation claims filed in 33 states. As required by the Occupational Safety and Health Administration, the Annual Survey of the BLS reports traumatic events occurring in the private sector; thus, it does not include traumatic events in the public sector, on farms with 10 or fewer employees, and in firms regulated by other federal health and safety laws. The National Safety Council reports data from the National Health Survey (based on 41,000 annual interviews with heads of households) and data from several participating public and private organizations. The definition of "recordable injury" varies considerably among these systems.

Because of these differences, it is not easy to achieve a reliable national composite of severe occupational traumatic injuries. Within the limitations of these data sources, NIOSH estimates that at least 10,000,000 persons suffer traumatic injuries on the job each year. About 30% (at least 3,000,000) of these injuries are severe, and at least 10,000 are fatal.

Traumatic Deaths: Each year, an estimated 10,000 persons are killed on the job. The major causes of these deaths are (1) highway motor-vehicle incidents, including to and from work and job-related travel (34%); (2) falls (13%); (3) nonhighway industrial-vehicle incidents (11%); (4) blows (other than by vehicles or equipment) (8%); and (5) electrocutions (7%) (Table 1). Industries with the highest estimated rates of fatal traumatic injury are (1) mining and quarrying, (2) agriculture (including forestry and fishing), and (3) construction (Table 2).

Amputations: Although amputations account for less than 1% of estimated injuries, they often impair a worker's skills. An estimated 21,000 workers suffered amputations in 1982. Based on NEISS data, approximately 93% of these amputations were of fingers (*8*); and 4%, of hands and toes. Amputations of fingers most frequently resulted from fingers being caught in machines or hand tools (11%) or cut by moving objects, such as saws or slicers (10%). Other important sources of amputations included presses (6%), belts (5%), powered hand tools (2%), and doors or gates (2%). Other specific sources accounted for less than 2% each of occupational amputations.

According to SDS data, amputations occurred in a wide range of industries and occupations. The largest single proportion of amputations (2%) occurred in the manufacture of miscellaneous plastic products, and machine operators had the largest proportion of occupational amputations (8%).*

Fractures: Falls and blows from falling objects produce many types of injuries, the less severe forms being contusions, abrasions, and sprains. During 1982, an estimated 400,000 work-related fractures occurred. SDS data for 1980 included approximately 208,000 compensation claims for fractures. The most frequently listed sources of fractures included floors (13%), the ground (10%), and metal items (7%), suggesting falls as the main cause of such injuries. Specifically, falls to a working surface accounted for 15% of the fractures; blows from unspecified or falling objects accounted for 31%. Fractures occurred most frequently among truck drivers (5%), miscellaneous laborers (4%), and construction laborers (3%).*

Eye Loss: Although it is difficult to measure the extent of eye loss or blindness among workers, NIOSH estimates (based on NEISS data) indicate that approximately 900,000 occupational eye injuries occurred in 1982. For 84% of these, the trauma was minor, caused mostly by foreign bodies (e.g., pieces of metal, wood, or glass) in the eyes. Burns and avulsions—44% of which were caused by chemicals or acids—accounted for nearly 15% of the estimated occupational eye injuries.*

Lacerations: An estimated 2,250,000 work-related lacerations occurred in 1982, representing 24% of all job-related injuries treated in hospital emergency rooms. Data from compensation claims described in SDS indicate that fingers (48%), arms (24%), legs (13%), and the head and neck (9%) were most likely to be seriously lacerated. These lacerations resulted primarily from being struck by an object (32%) or from striking against a stationary object (25%). The major sources of lacerations are knives (13%), other sharp metal items (13%), saws (6%), glass items (5%), nails (5%), and machines (3%). The settings in which workers incurred the largest proportion of lacerations were eating and drinking establishments (7%), grocery stores (4%), general building construction (2%), and meat packing (2%).*

*The remaining percentages are divided among a variety of specific categories, each accounting for less than the smallest percentage given.

Reported by Div of Safety Research, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Recent analyses of potential life lost due to various causes indicate that "accidents and adverse effects" are the leading cause of the loss of potential years of life in this country (*9*). Occupational injuries occur at a rate† twice that of injuries in the home or in public places (*7*), and severe traumatic injuries are an important component of all occupational injuries. Severe occupational trauma is second only to motor-vehicle incidents as a cause of unintentional death in the United States (*7*).

Despite the number of occupational injuries, effective prevention is practiced in many workplaces, and approximately 48% of all employment establishments report no recordable injuries in a given year (*6*). As with other occupational health hazards, the prevention of severe occupational traumatic injuries rests on the basic principles of control technology: engineering controls, work practices, personal protective equipment, and monitoring of the workplace for emerging hazards. Severe occupational traumatic injuries can be prevented by

TABLE 1. Distribution of occupational traumatic deaths, by cause—United States, 1980-1981*

Cause	Percentage
Highway motor-vehicles incidents	34.1
Falls	12.5
Industrial vehicles or equipment	11.4
Blows (other than by vehicles or equipment)	8.0
Electrocutions	6.8
Gun shots	4.5
Aircraft crashes	3.4
Entrapment	3.4
Fires	3.4
Plant machinery operations	3.4
Explosions	2.3
Gas inhalations	2.3
Other	4.5
Total	100.0

*Adapted from Bureau of Labor Statistics: Occupational Injuries and Illnesses in the United States by Industry, 1981. U.S. Department of Labor Bulletin 2164, January 1983. These revised statistics pertain to private-sector establishments (excluding nonmetal mining and railroads) with 11 or more employees.

such specific measures as physical barriers between the worker and the source of injury (e.g., machine guards, light curtains, worker-independent safety circuits, proximity sensors on robots); changes in the design of tools (e.g., knives and slicers) and tasks to reduce the hazard; use of personal protective equipment (e.g., seat belts, protective eye- and footwear, helmets, harnesses); training of workers in the safe performance of tasks; and repeated systematic inspection of the workplace for emerging or previously undetected hazards. A visible, serious, and persistent commitment to safety by both management and labor appears crucial for preventing severe occupational traumatic injuries.

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†Injuries per million exposure hours.

TABLE 2. Occupational traumatic death rates per 100,000 workers, by industry—United States, 1982*

Industry	Rate
Mining and quarrying	55
Agriculture	52
Construction	40
Transportation and public utilities	26
Government	10
Services	6
Manufacturing	6
Trade	5

*Adapted from (7) Accident Facts, National Safety Council, 1983 Edition. These rates are estimated by the National Safety Council based on data from the National Center for Health Statistics, state departments of health, state industrial commissions, and the Bureau of Labor Statistics. Agriculture includes forestry and fishing; services includes finance, insurance, and real estate; government includes federal, state, and local; trade includes wholesale and retail.

CARDIOVASCULAR DISEASES

Cardiovascular diseases, including hypertensive disease (International Classification of Diseases 9th Revision [ICD] codes 401-405), ischemic heart disease (ICD codes 410-414), other forms of heart disease (ICD codes 420-429), and cerebrovascular disease (ICD codes 430-438), are responsible for more deaths in the United States each year than any other category of disease (*5*). In 1980, cardiovascular diseases claimed over 960,000 lives, with ischemic heart disease responsible for over 565,000 of these deaths (*6*). Although the rates of death from cardiovascular diseases have declined gradually over the last decade, coronary atherosclerosis and acute myocardial infarction remain the leading causes of death in the United States.

The role of occupation as a factor in cardiovascular disease is far from clear (*7*). Most investigators believe that personal risk factors, such as cigarette smoking, blood pressure, diet, personality, and heredity, are more important than environmental factors (*8*). Specific data are sparse on the role of occupational factors. Nevertheless, some occupational factors are clearly associated with heart diseases, and evidence on other factors is accumulating (*9*). Because heart diseases are still so prevalent in the United States, identifying and preventing occupational factors that result in even a small increase in the relative risk of cardiovascular disease would involve large numbers of persons. Thus, preventing any occupational contribution to this problem would be an important public health measure.

In 1978, an ad hoc task force was formed by the American Heart Association to review the data regarding the environmental impact on cardiovascular disease (*8*). Its report, "The Impact of the Environment on Cardiovascular Disease," was published in 1981. The task force identified and reviewed six environmental factors that have potential impact on cardiovascular health: water hardness; trace elements; inhalant occupational exposures; carbon monoxide; noise and radiofrequency; and physical and psychosocial stress. The workplace is a specific source of potential exposure for all but the first.

Metals, Dusts, Trace Elements. The development of congestive heart failure that results from restrictive lung disease (cor pulmonale) has been observed in studies of occupational respiratory diseases, such as chronic beryllium disease and silicosis. Other metals, such as antimony, cobalt, and lead, have been implicated as possible causes of cardiovascular diseases.

Occupational Inhalants and Other Chemical Exposures. These include:

1. **Carbon monoxide**: Carbon monoxide decreases the oxygen-carrying capacity of hemoglobin and thus reduces the oxygen supply available to heart muscle and other tissues. In persons with preexisting coronary artery disease, occupational exposures to carbon monoxide may precipitate acute cardiovascular events, such as untoward changes in cardiac rhythm. In animal studies, life-threatening arrhythmias, such as ventricular tachycardia and ventricular fibrillation, have been observed in response to exposures to carbon monoxide that produced a carboxyhemoglobin concentration of 9% and above.

   In one study of workers, short-term exposure to carbon monoxide at levels within the current Occupational Safety and Health Administration permissible exposure limit (50 ppm) was associated with decreased exercise tolerance and electrocardiographic evidence of myocardial ischemia. In another study among Finnish foundry workers exposed to carbon monoxide, the overall prevalence of angina pectoris was increased; this was most pronounced among workers who also smoked. Among British steelworkers, investigators found end-of-shift carboxyhemoglobin saturations substantially higher among blast furnace workers than among steelworkers in other jobs. This was observed for both smoking and nonsmoking employees.

2. **Carbon disulfide**. Carbon disulfide, a widely used solvent, has been shown to increase the risk of cardiovascular disorders, including coronary artery disease and hypertension, in both epidemiologic and experimental studies. It has also been shown to pose a significant risk for coronary death (*10*). The atherogenic potential of carbon disulfide involves both cerebrovascular and cardiovascular systems.
3. **Halogenated hydrocarbons**. Acute exposures to many common industrial solvents (e.g., chloroform, trichloroethylene) and fluorocarbon aerosol propellants have precipitated sudden death probably due to cardiac arrhythmias in workers exposed at high levels. Other common aerosols or solvents may be arrhythmogenic at concentrations permitted by current occupational exposure standards. A recent study of pathologists exposed to monochlorodifluoromethane (a fluorocarbon aerosol propellant) in hospitals showed an increased incidence of "palpitations" at levels of exposure far below the recommended standard.
4. **Nitroglycerin and nitrates**. Workers exposed to nitroglycerin and nitrates during the manufacture of explosives experienced increased risk of cardiac chest pain, myocardial infarction, and sudden death, particularly after a period of time away from exposure. The mechanism is thought to be "rebound vasospasm" as a consequence of withdrawal from exposure.

Noise. Tens of millions of workers are exposed to high levels of sustained and/or intermittent noise in the workplace. A number of studies have demonstrated that single exposures to noise cause transient increases in blood pressure. Chronic exposure to occupational noise has also been associated with sustained increases in blood pressure, particularly in workers with noise-induced hearing loss (*11,12*). Increases in serum cholesterol and changes in circulating hormones have been observed in humans in association with noise. In studies of animals, abnormalities in platelet aggregation have been documented following exposure to noise.

Psychosocial Stress. Stress has long been thought to adversely affect the cardiovascular system (*13*). A relationship between psychologic factors and cardiovascular disease is supported by the correlation between "Type A personality" and such disorders. A 1976 assessment suggested that "work-overload," role conflicts, and thwarted career goals were related to evidence of cardiovascular disease. A prospective evaluation of health changes among air traffic controllers, published in 1978, showed an increased prevalence of hypertension among controllers, attributed by the authors to difficulties in coping with working conditions.

An updated analysis of the Framingham heart study in 1980 (*14,15*), indicated that rates of coronary heart disease were nearly twofold greater among women employed in clerical jobs than among housewives. Significant predictors of the risk of coronary heart disease included a "nonsupportive supervisor" and decreased job mobility. Occupation may also affect the risk of cardiovascular disease in a spouse. Men whose wives worked in white collar jobs were observed to experience heart disease at a rate three times greater than men whose wives worked in clerical or blue collar jobs or were housewives. Similarly, men appeared to have a higher risk of cardiovascular disease if they had well-educated, working wives who reported nonsupportive supervisors or few opportunities for job promotion. These and similar results suggest that adjustments to the conflicting demands of job and family may be important factors in the risk of cardiovascular disease.

Recent evaluations of data from a large random sample of the Swedish working male population (*16*), and from other surveys, also suggest that certain working conditions, such as limited autonomy and heavy workloads, are associated with clinical indicators of coronary heart disease.

Epidemiologic studies are clearly needed to define the significance of these and other occupational stress factors in the etiology of cardiovascular diseases. Such physical stresses as noise, vibration, and heat also merit investigation for possible interaction with the psychologic risk factors of cardiovascular disease.

Reported by Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Because cardiovascular diseases are so prevalent, they clearly affect large numbers of workers in the United States. A proportion of these diseases are caused or aggravated by risk factors in the work environment. NIOSH is currently collecting epidemiologic data to properly evaluate the role of the workplace in these diseases.

Since some occupational exposures clearly contribute to the morbidity and mortality attributable to cardiovascular diseases, further epidemiologic research is essential to define the extent of their occupational role and to define etiologic mechanisms. In addition, since cardiovascular diseases cause so much mortality in the United States, preventing even a small increase in relative risk due to occupational exposures would have major consequences to the public health. That mortality from cardiovascular disease has declined markedly in recent years indicates that these diseases are preventable.

The workplace is an important focus for efforts to prevent cardiovascular disease because: (1) it is the source of some preventable environmental exposures and psychosocial stresses that adversely affect cardiovascular health; (2) it offers unique opportunities for health promotion activities that encourage workers to improve their personal health behaviors (e.g., smoking cessation, appropriate exercise, and sound nutrition) and; (3) it provides an advantageous setting for delivering preventive services, such as screening for hypertension.

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*Additional references are available on request from the National Institute for Occupational Safety and Health, CDC.

DISORDERS OF REPRODUCTION

Since antiquity, certain chemical and physical agents have been recognized as having detrimental effects on human reproduction. For example, the effect of industrial lead poisoning in inducing abortions was noted by the Romans and again in the first decade of this century (*6*). Evidence from more recent laboratory studies and clinical investigations indicates that a wide range of microbiologic, physical, and chemical agents, such as *Brucella*, rubella, ionizing and nonionizing radiation, heat and vibration, tobacco, alcohol, and certain drugs, can adversely affect reproductive outcomes. At least 50 chemicals—including heavy metals, such as lead and cadmium, glycol ethers, organohalide pesticides, organic solvents, and chemical intermediates, such as styrene and vinyl chloride—in widespread use in industry have been shown to produce impairment of reproductive functions in animals (*7*).

Until recently, the potential hazards to human reproduction posed by occupational exposures received little attention. However, adverse effects after thalidomide exposure in the 1960s and the occurrence in 1970 of methylmercury poisoning among residents of Minamata, Japan, dramatically demonstrated the teratogenic potential of chemical exposures. Those events and the increasing entry of women into the workforce focused greater attention on the potential hazards to female reproductive function of occupational exposures. In the late 1970s, the demonstration of sterility among male workers exposed to dibromochloropropane was described; this drew attention to the concomitant potential for hazards to male reproductive function (*8*).

Occupational exposures can produce a wide range of adverse effects on reproduction. The effects of parental exposure before conception to agents toxic to reproductive functions may be evident as reduced fertility, unsuccessful fertilization or implantation, or an abnormal fetus. Maternal exposure after conception may result in death of the fetus or structural and functional abnormalities in the newborn. Other possible adverse outcomes include spontaneous abortions (both early and late), major and minor birth defects, perinatal death, low birth weight, altered sex ratio, developmental or behavioral disabilities, and transplacental exposure to carcinogen (*9-11*).

Estimates of the prevalence of adverse reproductive outcomes indicate that these events occur with considerable frequency in the U.S. population. For example, an estimated 560,000 infant deaths, spontaneous abortions, and stillbirths occur each year. The March of Dimes estimates that 200,000 live infants with some type of birth defect—benign or disabling—are born in the United States each year (*9*).

The causes of most of these adverse outcomes are unknown. For example, 6%-30% of the infertile couples have no recognized anatomic or physiologic abnormalities to account for the infertility (*10*); neither the etiology of sperm abnormalities nor the cause of sister-chromatid exchange in spontaneous abortions has been established (*11,12*). The causes for as many as 65%-70% of the birth defects are not known (*13*).

Maternal Exposures. Studies of occupational reproductive hazards to date have consisted mainly of epidemiologic surveys of pregnancy outcomes following maternal exposures. Such studies have shown increased rates of spontaneous abortions among laboratory and chemical workers (*14,15*) and among workers exposed to lead (*16*), ethylene oxide (*17*), and anesthetic gases (*18,19*). Studies of adverse outcomes of pregnancy, however, are subject to several methodologic limitations. For example, the detection of rare outcomes, such as birth defects, requires the study of several thousand pregnancies, and retrospective studies are subject to problems of recall and misclassification, both of reproductive events and of exposures (*20,21*). The timing, duration, and frequency of exposure before and during pregnancy may critically

affect reproductive outcomes (*22*). For example, exposure to ionizing radiation during the first trimester may result in microcephaly and mental retardation, and exposure during the third trimester may produce low birth weight and neonatal death (*11*). Other studies have been limited by the selection of inadequate comparison groups or the failure to examine the influence of other factors, such as alcohol and tobacco consumption or maternal age, that affect reproductive outcomes.

Paternal Exposures. Since azoospermia (absence of living spermatoza in the semen) and oligospermia (subnormal concentration of spermatoza) were reported in 1977 among workers exposed to dibromochloropropane (*8*), at least 14 studies have examined the quality of semen in workers exposed to lead, carbon disulfide, anesthetic gases, ionizing radiation, toluenediamine, dinitrotoluene, carbaryl, and several other pesticides (*10*). Adverse effects on the quality of semen were reported in workers exposed to lead or ionizing radiation. In other studies (e.g., of exposures to ethylene dibromide) results were inconclusive because of problems in design of the study or inadequate numbers of participants (*10*). CDC recently used data collected by the Metropolitan Atlanta Congenital Defects Program to examine the risk of serious structural birth defects among the children of male Vietnam veterans; no statistically excessive risks were noted (*23*). In general, relatively few studies have been conducted of reproductive outcomes associated with paternal exposures (*9*).

Extent of potential exposures. Estimates have been made of the number of workers potentially exposed to selected agents known or suspected to be toxic to reproductive function (Table 1). NIOSH estimates that approximately 200,000 workers are potentially exposed to various glycol ethers (*24*), several of which exhibit marked testicular toxicity in animals (*25*).

TABLE 1. Estimated numbers of workers potentially exposed to selected substances known or suspected to cause adverse reproductive outcomes

Agent*	Estimated no. workers
Dibromochloropropane	11,362
Cadmium	157,383
Chloroprene	343,596
Ethylene glycol	2,060,470
Ethylene oxide	139,000
Formaldehyde	1,658,151
Lead (inorganic)	1,401,831
Radiofrequency/microwave radiation	9,000,000
Waste anesthetic gases	50,000

*Examples of agents have been selected on the basis of positive animal and/or human data; inclusion or exclusion of agents does not constitute an evaluation of their potential reproductive toxicity in humans.

An estimated 9 million workers are exposed to radiofrequency/microwave radiation (*26*), which has been shown to cause embryonic death and impaired fertility in animals but which has yet to be studied adequately in humans. NIOSH has estimated that approximately 50,000 personnel in hospital operating rooms are potentially exposed to waste anesthetic gases, and 139,000 hospital and other industrial workers may be exposed to ethylene oxide (*24*); both agents have been linked to an increased risk of spontaneous abortions in humans.

Reported by Industrywide Studies Br, Surveillance Br, Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note: The extent to which occupational exposures in American workers produce adverse reproductive outcomes is largely unknown. However, the information presented here

suggests that the problem is both widespread and serious. Epidemiologic and toxicologic research into the reproductive effects of occupational exposures is in its infancy. There is a continuing effort to elucidate the etiology of adverse reproductive outcomes, such as fetal chromosomal abnormalities or abnormal spermatogenesis and to develop improved animal models for screening agents for possible mutagenic and toxic effects related to human reproduction. Registries for the surveillance of outcomes of reproduction, such as CDC's Birth Defects Monitoring Program (*9*), and improved methodologies developed to evaluate such parameters as quality of semen (*12*) and outcomes of pregnancy (*20*), will permit further identification of specific occupational hazards to reproduction. When such hazards are identified and controlled in the workplace, the prevention of reproductive disorders in the population as a whole will be substantially improved.

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NEUROTOXIC DISORDERS

Background. Diseases of the nervous system resulting from toxic exposures in the workplace were known as early as the first century A.D., when Pliny identified palsy as a manifestation of lead poisoning among workers exposed to lead dust (*7*). In 1557, Jean Fernel linked gingival pigmentation, tremor, and behavioral changes to occupational mercury poisoning (*8*); in the nineteenth century, Delpech recognized rubber processing as the cause of the bizarre psychoses occurring among French workers who manufactured condoms and balloons in small cottage industries. Later, carbon disulfide was implicated as the specific neurotoxic agent (*9*).

Industrial hygiene practices have improved in the twentieth century, and some animal models of neurotoxic disease have been developed. In addition, workers who become ill often draw attention to outbreaks of neurotoxic diseases. Despite the prior identification of acrylamide as neurotoxic in animals, its neurotoxicity in humans was first recognized in the 1950s, when several Japanese workers involved in a pilot production project developed peripheral neuropathy (*10*). During the 1960s and early 1970s, dozens of cases of neuropathy occurred among Japanese and Italian workers exposed to solutions containing *n*-hexane during the manufacture of shoes (*11*). Subsequently, high doses of *n*-hexane were found to be neurotoxic in exposed animals. In the past 15 years alone, outbreaks of serious human neurotoxicity occurred among workers exposed to three substances not previously known to be neurotoxic: the chlorinated hydrocarbon, chlordecone, which caused opsoclonus, tremor, disturbances of gait, and changes in personality (*12*); and two hexacarbons, methyl-*n*-butyl ketone and 2-t-butylazo-2-hydroxy-5-methylhexane, both of which caused a predominantly peripheral neuropathy (*13,14*).

Nature of Neurotoxic Disorders. Neurotoxic disorders are on the NIOSH list of Ten Leading Work-Related Diseases and Injuries (*1*) because of their potential severity—as exemplified by the neurotoxicity of chlordecone—and because of the large number of workers potentially at risk. A conservative estimate of the workers exposed full time to one or more neurotoxic agents is 7.7 million (*15*). The number of potentially neurotoxic chemicals found in the workplace exceeds 850; an abbreviated list of the more commonly used of these chemicals is shown in Table **1** (*16*).

Clinically, symptoms and signs of neurotoxicity can be diverse. Depending on the intensity of exposure, the molecular configuration of the agent, and the mechanism of toxicity, either central or peripheral neurologic effects may predominate. Most neurotoxic chemicals, however, affect both the central and peripheral nervous systems. Because the symptoms of peripheral neuropathy are more specific and the nerves themselves more directly accessible to precise diagnostic examinations, the effects of neurotoxic agents on the peripheral nervous system are usually more easily identified than effects on the central nervous system (CNS). Early symptoms of peripheral neuropathy may include numbness, tingling, or pain in the feet or hands. As the disease progresses, clumsiness or incoordination due to both sensory and motor changes may develop. Production workers may find their ability to do usual work partially or fully impaired. Chemicals used extensively in industry, which cause peripheral neuropathy when present in sufficiently high and persistent concentrations, include: lead, *n*-hexane, acrylamide, carbon disulfide, mercury, and methyl bromide (*17,18*) (Table **2**). Several chemicals are known to cause selective impairment of cranial-nerve function, including dysfunction of the fifth cranial nerve (trichloroethylene) (*18*).

The effects of neurotoxic agents on the CNS present a far wider range of disturbances (*16,18,19*) (Table **3**). At times, the most striking effects are changes in mood and personality (*20*). High levels of exposure to manganese or carbon disulfide produce psychoses and suicidal tendencies. Delusions and hallucinations may result from exposure to high concentrations of solvents, such as methylene chloride. Manifestations of cognitive dysfunction, such as reduced attention span, lack of alertness, and memory loss, are prominent neurotoxic effects that may occur in addition to personality changes after exposure to many solvents and to asphyxiants, such as carbon monoxide. Other neurologic effects occur under certain restricted conditions of exposure to unique chemical substances (Table **4**).

Although research into the neurobehavioral effects of industrial chemicals is relatively new, early results suggest that occupational neurotoxicity may be a larger problem than previously suspected. Sensitive methods for evaluating subtle losses in cognitive function have only recently been applied to the evaluation of exposed workers. Because of the complexity of the nervous system and the variety of potentially neurotoxic exposures, the true scope of this health hazard in the workplace is unknown.

Reported by Div of Biomedical and Behavioral Science, Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Studies of the neurotoxicity of workplace chemicals demonstrate the problems encountered in recognizing occupational disease in general. Despite occasional large and dramatic outbreaks of neurotoxic disorders, such as those mentioned above, more often small numbers of workers in many workplaces are chronically exposed to neurotoxic agents

TABLE 1. Commonly used industrial chemicals recognized as neurotoxic

Acetyl ethyl tetramethyl tetralin	Cobalt	Methyl *n*-butyl ketone
Acetyl pyridine	Cuprizone	Nickel (carbonyl)
Acrylamide	Cyanide	Nitrogen trichloride
Adiponitrile	2,4-Dichlorophenoxy acetic acid (2,4-D)	Organochlorine insecticides
Alkyl phosphates	Dichlorodiphenyl tri chloroethane (DDT)	Organophosphate esters
Aluminum	Diethyl ether	Organotins (triethyltin)
Aniline	Diisopropyl fluorophosphate (DFP)	Paraquat
Arsenic, inorganic	Dimethyl sulphate	Phenol
Arsine	Ethylene dichloride	Phenyl mercury
Aryl phosphates	Hexachlorophene	Phthalate esters
Azide	*n*-Hexane	Polybrominated biphenyls (PBB's)
Barium	Hydroquinone	Selenium
Benzene	Lead	Styrene
Boron	Lead, tetraethyl	Sulfur dioxide
p-Bromophenyl acetylurea	Leptophos	Tetrachlorobiphenyl
Cadmium	Malonitrile	Thallium
Carbon disulfide	Manganese	Toluene
Carbon monoxide	Mercury	Trichloroethylene
Carbon tetrachloride	Methanol	Triorthocresylphosphate (TOCP)
Chlordane	Methyl bromide	Vanadium, inorganic salt
Chlordecone	Methyl chloride	Zinc
Chloroprene		Zinc pyridinethione

that subtly and slowly alter nervous-system functions. Several neurotoxic syndromes mimic diseases of nonoccupational and "idiopathic" etiology, e.g., the toxic axonopathy associated with exposure to various metals and solvents, the parkinsonian syndrome of chronic intoxication with manganese, and the organic brain syndrome of chronic solvent intoxication. Because of these similarities to other nonoccupational diseases, such cases are frequently not identified as occupational in origin. In addition, many physicians are not trained to take an adequate occupational medical history (*21*). For these reasons, the prevalence of occupational neurologic disease is unknown, and important causal relationships between chemicals and disease remain obscure.

The prevention of neurotoxicity among workers will require strategies such as those suggested in the 1990 objectives for improving the nation's health (*22*), developed by the U.S. Public Health Service: (1) analyses of structural analogues of known neurotoxic agents in an effort to predict the neurotoxicity of untested chemicals; (2) continuing search for animal models of disease; (3) ongoing research in establishing an acceptable human exposure level for identified neurotoxic agents; (4) epidemiologic evaluations of suspected neurotoxicity; (5) development of simple screening tools for use on asymptomatic populations exposed to known neurotoxic agents; and (6) premanufacture and premarket testing of new chemicals as required by the Toxic Substances Control Act (*23*). As in the prevention of other work-related diseases, however, the most direct and effective method for preventing neurotoxic illness will continue to be the environmental control of exposures to neurologic chemicals. Such efforts as the substitution of less toxic substances where possible, engineering controls, teaching appropriate work practices, and educating workers about the potential neurotoxicity of chemicals will aid a comprehensive prevention effort.

TABLE 2. Examples of peripheral neuropathies associated with occupational toxins

Type of neuropathy	Toxin	Comments
Motor	Lead	Wrist extensors primarily involved; wrist and ankle drop are rare.
Mixed sensorimotor	Acrylamide	Ataxia; desquamation of hands and soles; sweating of palms.
	Arsenic	Early distal paresthesias; pain in limbs, especially calves; hyperpathia of feet; weakness prominent in legs.
	Carbon disulfide	Peripheral neuropathy rather mild; CNS effects more important.
	Carbon monoxide	After severe intoxication.
	DDT	Only with ingestion.
	n-Hexane, methyl *n*-Butyl ketone	Distal paresthesias, motor weakness; weight loss, fatigue, and muscle cramps.
	Mercury	Predominantly distal sensory involvement; more common with alkyl mercury exposure.

TABLE 3. Clinical manifestations and causes of central nervous system (CNS) conditions

Condition	Symptoms	Signs	Latency	Prognosis following exposure cessation	Major neurotoxic substances
Acute conditions					
Acute intoxication	Dizziness, lightheadedness, balance and gait impairment, incoordination, feeling "high"	Ataxia, slow psychomotor function	Minutes-hours	Reversible	Organic solvents, inhalation anesthetics
Acute toxic encephalopathy	Obtundation, coma, seizures, potentially fatal	Signs of diffuse CNS depression, reflex slowing, EEG slowing	Hours-days	Persistent deficits common	Solvents, lead, pesticides
Chronic syndromes					
Symptoms only	Mood changes (irritability, depression), sleep disorders, difficulty concentrating, memory complaints, symptoms are more noticeable to relatives than to patient	No objective signs	Weeks-months	Reversible	Carbon disulfide, lead, organic solvents
Mild chronic toxic encephalopathy					
Organic personality or mood disorders	Similar to those noted above but of greater frequency and severity	Alterations in mood or personality	Weeks-months	Incomplete reversibility possible but uncommon	Lead, organic solvents
Neurobehavioral impairment	Symptoms as above	Reduced motor speed, reduced vigilance and reaction time, plus reduced performance on memory (short-term) testing and other tests of cognitive function (i.e., visuospatial ability)	Weeks-months	Potentially reversible, partial or complete	Carbon disulfide, lead, organic solvents, possibly carbon monoxide
Severe chronic toxic encephalopathy (dementia)	Significant loss of ability to perform activities of daily living, difficulty in comprehension, profound memory loss, reduced verbal fluency	Testing compatible with severe neurologic damage and neuropsychologic impairment as seen in dementia	Unknown	Poorly reversible	Lead, organic solvents

TABLE 4. Other neurologic effects

Health effects	Agents
Motor and gait disorders	
Ataxia	Acrylamide Chlordane Chlordecone DDT *n*-Hexane Manganese Mercury (especially methyl mercury) Methyl *n*-butyl ketone Methyl chloride Toluene
Myoclonus	Benzene hexachloride Mercury
Paraplegia	Organotin compounds
Parkinsonism	Carbon disulfide Carbon monoxide Manganese
Seizures	Lead Organic mercurials Organochlorine insecticides Organotin compounds
Tremor	Carbon disulfide Chlordecone DDT Manganese Mercury
Visual-system effects	
Nystagmus	Mercury
Opsoclonus	Chlordecone
Constricted visual field	Mercury
Impaired visual acuity	*n*-Hexane Mercury Methanol

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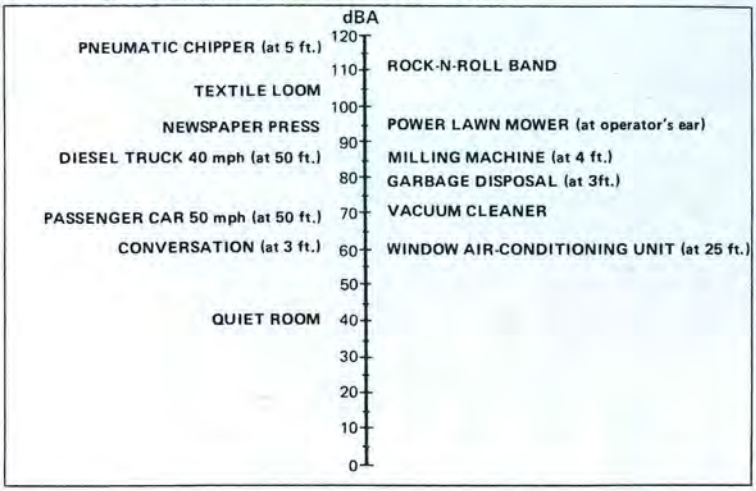
NOISE-INDUCED LOSS OF HEARING

Occupational deafness was first documented among metalworkers in the sixteenth century (*8*). Since then, workers have experienced excessive hearing loss in many occupations associated with noise. Typical occupational and nonoccupational noise levels are shown in Figure 1. Noise-induced loss of hearing is an irreversible, sensorineural condition that progresses with exposure. Although hearing ability declines with age (presbycusis) in all populations, exposure

FIGURE 1. Typical A-weighted noise levels in decibels (dBA)*



to noise produces hearing loss higher than that resulting from the natural aging process; this is caused by damage to nerve cells of the inner ear (cochlea) and, unlike some conductive hearing disorders, cannot be treated medically.

While loss of hearing may result from a single exposure to a very brief impulse noise or explosion, such traumatic losses are rare. In most cases, noise-induced hearing loss is insidious. Typically, it begins to develop at 4,000 hertz (Hz, or cycles per second) in the hearing range of 20 Hz to 20,000 Hz and spreads to lower and higher frequencies. Often, material impairment has occurred before the condition is clearly recognized.

Such impairment is usually severe enough to permanently affect a person's ability to hear and understand speech under everyday conditions. Although the primary frequencies of human speech range from 200 Hz to 2,000 Hz, research has shown that the consonant sounds, which enable people to distinguish words such as "fish" from "fist," have still higher frequency components. As a result, an average hearing threshold (lowest audible sound level) at separate frequencies of 1,000 Hz, 2,000 Hz, and 3,000 Hz is used widely to define material impairment caused by noise (*10,11*).

*The decibel is a logarithmic measure of sound intensity; the "A-weighted scale" is used to weight the various frequency components of the noise to approximate the response of the human ear.

Recent estimates by the Occupational Safety and Health Administration (OSHA) indicate that about 9,400,000 U.S. production workers (7,900,000 active and 1,500,000 retired) either now work or have worked in industrial locations where noise-exposure levels are 80 decibels (dBA) or higher. This estimate includes most noisy workplaces in the United States, except agricultural, mining, construction, transportation, and government (Table 1) (*11*). At exposure levels below 80 decibels (weighted to the approximate response of the human ear, dBA), an increased risk of hearing loss caused by occupational noise has not been found. Based on the average hearing threshold level at 1,000 Hz, 2,000 Hz, and 3,000 Hz, OSHA estimated that 1,624,000 (17%) production workers have at least mild hearing loss resulting from their occupational noise exposures; 1,060,000 (11%) have material hearing impairment; and 473,000 (5%) have moderate to severe impairment (Table 2) (*11*). These estimates generally agree with NIOSH survey findings, which indicate that one-fourth of persons 55 years of age or older who have been exposed over their working lifetime to an average of about 90 dBA have developed a material hearing impairment caused by occupational noise exposure (*10,12*). An estimated $835 million will be paid in workers' compensation claims for occupational hearing impairment for the 10-year period 1978-1987 (*13*).

Reported by Physical Agents Effects Br, Div of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Occupational noise-induced loss of hearing is preventable. In its 1990 objectives for the nation, the U.S. Public Health Service set an objective that "By 1990, the prevalence of occupational noise-induced hearing loss should be reduced to 415,000 cases" (*14*). This objective relates to the number of cases of hearing loss that result in moderate to severe impairment (Table 2). However, it is important to note that if the number of moderate to severe impairments is reduced, the number of mild hearing loss and of material impairments would be reduced proportionately. OSHA has estimated that within 10 years, the number of cases of noise-induced hearing impairment can be reduced by 20% if all workers exposed to noise levels higher than 85 dBA wear personal hearing protectors (earplugs or muffs) and receive on the average 15 dBA noise reduction (*11*). However, this estimate hinges on effective use of hearing protectors to an extent that has not yet been demonstrated for all workers. NIOSH field investigations of industrial workers who routinely use earplugs indicate average noise reduction ranging from 7 dBA to 20 dBA, depending on the type of plug used (*15*).

TABLE 1. Distribution of 9,368,000 production workers who had noise-exposure levels of 80 dBA or greater* — United States

Noise-exposure level (dBA)	No. workers
80-85	3,305,000
86-90	2,656,000
91-95	1,936,000
96-100	965,000
>100	506,000

*From the 1981 OSHA Final Regulatory Analysis for the Hearing Conservation Amendment.

A noise-control/hearing-conservation program is the most important step in eliminating occupational hearing loss. Such a program must include:

1. Reduction of noise through engineering controls and the purchase of new, noise-engineered equipment.
2. Proper fit of personal hearing-protection devices.
3. Education of workers and managers about certain characteristics of noise-induced loss of hearing (e.g., irreversible, subtle in onset, psychologically distressing).
4. Proper periodic audiometric testing and notification of workers who are developing hearing loss.
5. Visible commitment of management and workers to the program.

The joint efforts of management, labor, and health-care providers are needed to establish effective hearing-conservation programs in industry. All interested groups must work together to achieve the goal of protecting workers' hearing.

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TABLE 2. Hearing levels (dB) of 9,368,000 production workers who had noise-exposure levels of 80 dBA or greater* — United States

Hearing threshold level (1,000, 2,000, and 3,000 Hz)	Cumulative cases (%)	Expected cases† (%)	Excess cases§ (%)
>15 dB (mild hearing loss)	3,735,000 (40%)	2,111,000 (23%)	1,624,000 (17%)
>25 dB (material hearing impairment)	2,025,000 (22%)	965,000 (10%)	1,060,000 (11%)
>40 dB (moderate to severe hearing impairment)	718,000 (8%)	245,000 (3%)	473,000 (5%)

*From the OSHA 1981 Final Regulatory Analysis for the Hearing Conservation Amendment.
†Based on hearing levels of a nationwide sample of adults in U.S. Public Health Service hearing surveys.
§Cumulative cases minus expected cases.

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DERMATOLOGIC CONDITIONS

Background. A worker's skin is directly exposed to the occupational environment and is susceptible to a large number of dermatologic injuries and other conditions (Table 1). Complete data on the extent and cost of dermatologic injuries are not available; however, dermatologic conditions other than injuries accounted for 37% of the 106,100 occupational illnesses recorded in 1983 in the Bureau of Labor Statistics (BLS) Annual Survey of Occupational Injuries and Illnesses[†] (*1*). Results from the BLS Annual Survey for 1972-1976[§] indicated that 20%-25% of all occupational dermatologic conditions resulted in lost time from work (average 10-12 lost work days) (*2*). Similar data based on workers' compensation claims have been reported from California and South Carolina (*3,4*). Assuming that only 2%-10% of cases are actually reported, the annual cost of occupational dermatologic conditions resulting from lost worker productivity, medical care, and disability payments may range between $222 million and $1 billion (*5,6*).

Because 10%-15% of requests that NIOSH receives for health hazard evaluations involve skin complaints, and because the economic impact of work-related dermatologic conditions is substantial, NIOSH has included dermatologic conditions on its list of 10 leading work-related diseases and injuries in the United States (*7*).

Dermatologic Injuries. Dermatologic injuries are usually described as the immediate adverse effects on skin that result from instantaneous trauma or brief exposure to toxic agents involving a single incident in the work environment (*1*). Skin injuries may constitute 23%-35% of all injuries (*8,9*). Thus, based on 4,748,000 injuries of all types, and a full-time worker population of 74,750,000 for 1983 (*1*), an estimated 1,070,000-1,650,000 dermatologic injuries may occur yearly, with an estimated annual rate of skin injury of 1.4-2.2 per 100 full-time workers. The highest percentage of skin injuries are due to lacerations/punctures (82%), followed by burns (chemical and other) (14%) (*8*) (Table 2).

TABLE 1. Selected examples of occupational dermatologic conditions

Skin disorders	Affected skin structures
Contact dermatitis	Epidermis
Infection	Epidermis and dermis
Trauma	Connective tissue
Cancer	Squamous and basal cells, melanocytes
Vitiligo	Melanocytes
Urticaria	Blood vessels and mast cells
Chloracne	Sebaceous glands

*References to the previous articles are given in the most recent article (*MMWR* 1986;35:185-8).

†The BLS Annual Survey provides yearly national estimates of incidence rates of occupational illness based on a randomly selected national sample of private-sector U.S. businesses from all industrial classifications. The survey records all new illnesses recognized during the reporting year (incidence) but does not measure continuing conditions from previous years (prevalence).

§Since 1978, the Annual Survey has not tabulated lost workday statistics separately by type of occupational illness.

Other Dermatologic Conditions. Other dermatologic conditions ("illnesses of the skin") may also result from exposure to environmental factors or toxic agents associated with employment. However, they usually result from more sustained or cumulative exposures and involve longer intervals between exposure and occurrence of disease. These conditions include contact dermatitis, infection, acne, and skin cancer. Workers' compensation claims data from California suggest that 95% of these occupational skin conditions are either contact dermatitis (90%) or infections (5%) (*3*). Field investigations in the 1950s showed that at least 80% of occupational contact dermatitis cases may be caused by the irritating direct cytotoxic effects of causal agents rather than immunologically mediated allergic reactions (*10*).

The highest number of other occupational skin conditions (23,017) in 1984 occurred in the manufacturing sector; the highest incidence rate (28.5/10,000 full-time workers) involved the combined agriculture/forestry/fishing division (Table 3).

The clinical course for occupational contact dermatitis is relatively poor. In three studies, complete resolution occurred in 25% of workers affected; 50% improved but had periodic recurrences; and 25% developed persistent dermatitis as severe as or worse than the original condition (*11-13*). Contact dermatitis often necessitates job changes or modifications. Despite these, however, complete resolution may occur in only a limited proportion of cases.

Prevention of Work-Related Dermatologic Disorders. The most effective prevention measures are engineering controls that eliminate exposures of the skin to chemical, physical, or mechanical agents through isolation, containment, or redesign of industrial processes. Substitution of less toxic substances through chemical engineering may also be effective (*14*). Protective clothing should be selected on the basis of resistance to both chemical and physical hazards, as well as on the relative permeabilities to specific chemical exposures. Effective cleaning of skin and clothing is important, but workers should not wash vigorously or excessively with harsh soaps and detergents (*15*). Barrier creams have been suggested as alternatives, although their effectiveness has not yet been established (*16*). Prevention strategies should always include education of workers and management.

Expanded activities concerning occupational dermatologic conditions include improved methods for surveillance of occupational skin disease and vigorous research in dermatotoxicology to identify preventable risk factors and facilitate effective interventions at early stages.

TABLE 2. Occupational dermatologic injuries* — United States, 1983

Type of injury	No.	(%)
Lacerations and punctures	253,141	(82.3)
Burns (nonchemical)	36,477	(11.9)
Abrasions	10,576	(3.4)
Burns (chemical)	6,828	(2.2)
Cold injuries	566	(0.2)
Radiation injuries	135	(0.04)
Total	**307,723**	**(100.0)**

*Reported by the Supplementary Data System of the Bureau of Labor Statistics from 29 participating states.

Reported by Div of Periodic Surveys and Supplementary Data Systems, Office of Occupational Health and Safety Statistics, Bureau of Labor Statistics, US Dept of Labor; Occupational Dermatology Activity, Industrywide Studies Br, Surveillance Br, Div of Surveillance, Hazard Evaluations, and Field Studies, Data Analysis Section, Div of Safety Research, National Institute for Occupational Safety and Health, CDC.

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TABLE 3. Cases and incidence rate of occupational dermatologic conditions, in a segment of workers, by major industrial divisions — United States, 1984*

Industrial division	No.	Incidence rate†
Agriculture/forestry/fishing	2,233	28.5
Manufacturing	23,017	12.3
Construction	2,456	6.6
Services	7,973	5.0
Transportation/utilities	2,114	4.3
Mining	393	4.0
Wholesale/retail trade	3,770	2.1
Finance/insurance/real estate	563	1.1

*Bureau of Labor Statistics Annual Survey.

†Per 10,000 full-time workers (2,000 employment hours/full-time worker/year).

PSYCHOLOGICAL DISORDERS

There is increasing evidence that an unsatisfactory work environment may contribute to psychological disorders. Studies have shown that factors contributing to an unsatisfactory work environment may include work overload, lack of control over one's work, nonsupportive supervisors or co-workers, limited job opportunities, role ambiguity or conflict, rotating shift-work, and machine-paced work (*1-4*). Psychological disorders that can result from such factors may be classified as a) affective disturbances (e.g., anxiety, irritability), b) behavioral problems (e.g., substance abuse, sleep difficulties), c) psychiatric disorders (e.g., neuroses), and d) somatic complaints (e.g., headache, gastrointestinal symptoms). In addition to psychological disorders, stressful working conditions may have a systemic influence, possibly affecting the etiology and/or prognosis of other disease states, as suggested by recent studies of stress-related immunologic suppression (*5*).

Although data bases currently available for determining the extent of work-related psychological disorders are limited, several indicators suggest that these problems impose substantial health and financial costs in the United States. A recent study in California showed that claims for the development of "work-related neuroses" more than doubled during 1980-1982; claims for all other disabling work-related injuries during the same period actually decreased by about one-tenth (*6*). A study of representative medical claims throughout the country showed that during 1980-1982 claims for "mental stress" that developed gradually (i.e., a chronic problem unrelated to a single traumatic incident or to any physical work-related disorder) accounted for about 11% of all occupational disease claims (*7*). Average medical costs and indemnity payments in 1981-1982 for these forms of mental stress actually surpassed the average amounts for other occupational diseases (*7*). The American Psychiatric Association now lists occupational stress in its *Diagnostic and Statistical Manual* as a subcategory of the major diagnostic axis of "psychosocial stress" (*8*).

There are increasing data on the relationship between specific working conditions and psychological disorders. For example, in a questionnaire survey of over 2,000 workers in 23 different occupations, strong occupational differences were found in psychosocial job stressors and in somatic and affective complaints (*1*). Ratings of boring, repetitive job tasks and role ambiguity were more prominent among several classes of blue-collar workers (e.g., assembly-line workers, fork-lift truck drivers, and machine operators) than among white-collar professionals (e.g., professors and family physicians). The most satisfied occupational groups were physicians, professors, and white-collar supervisors. Groups experiencing the highest levels of job stressors and their resultant ill effects were assemblers and relief workers on machine-paced assembly lines.

NIOSH investigators ranked 130 occupations by rate of admission to community mental health centers in Tennessee to determine the relative risk of psychological or stress-related disorders by occupation (*9*). Heading the list were jobs in health care, service occupations, and blue-collar factory work—which tend to be characterized by stress-producing conditions such as a lack of control over the job by the worker, repetitive work, shift work, and a responsibility for others.† In other studies, workers on night and rotating shifts (including the health-care occupations) reported more disturbances of sleep; altered eating habits; and higher rates of visits to clinics, absences due to sickness, and on-the-job injuries than did those on fixed day shifts (*10-12*).

*References to the previous articles are given in the most recent article: MMWR 1986;35:561.

†These results represent findings in only one state (Tennessee), and occupational groups may differ considerably in their patterns of use of community mental health centers.

Work environments characterized by technological innovation have also been investigated; a major focus has been on office work influenced by the introduction of computers (*13,14*). "Adverse working conditions" (e.g., poorer physical environment, reduced job control and social support) tend to be reported more frequently by workers using new-technology office equipment such as video display terminals. Some of these conditions have been linked to chronic stress-related disorders (*4,15*).

Worksite studies by NIOSH have revealed that job stresses may contribute to acute disturbances among groups of workers, including those termed "mass psychogenic illness" (*16*). The sudden appearance of symptoms, usually in response to some "trigger factor" such as a strange odor, may result in spread of the apparent "illness" throughout the plant, with symptoms such as headaches, dizziness, and nausea. Investigations often fail to detect specific physical or chemical causative agents. However, factors such as heavy work load, strained labor/management relations, and physical discomfort at work may be present and related to the reporting of symptoms.

Emerging trends in technology, the economy, and demographic characteristics of the work force may lead to increased risk for psychological disorders. For example, a 26% increase is projected for employment in the health services, an area that may be associated with elevated risk (*9, 17*). Computers and robots are expected to affect seven million factory jobs and 39 million office jobs (*18*). According to some forecasters (*18*), possible consequences may include job displacement, reduced skill requirements, and lower-paying jobs. It has been projected that in the next decade, nine of every 10 new jobs will be in the service sector (*19*). Routine service jobs may not provide the compensation and benefits associated with the more traditional industrial and manufacturing jobs (*18*). Six of 10 new jobs in the next decade will be filled by women (*19*), and dual job/home role demands and constrained occupational opportunities for women may result in an adverse impact on their mental health.

Reported by Div of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, CDC.

Editorial Note: A prevention strategy for psychological disorders should take into account both the causal mechanisms and the factors that perpetuate these disorders. Work-related psychological disturbances are known to be influenced by both the physical and psychosocial characteristics of given job situations. Moreover, these factors operate in concert with factors unrelated to the job—such as life events; familial demands and support; and the traits, capacities, and needs of the workers themselves (e.g., personality, age, sex, experience/learning). The interaction of these variables is complex, and the relative influence of each is not thoroughly understood. Nevertheless, approaches to prevent work-related psychological disorders should still be taken using the information currently available.

Stress-reduction techniques (e.g., meditation, biofeedback, muscle relaxation, cognitive restructuring, and anxiety management) have been taught to both blue- and white-collar workers in worksite training sessions. Follow-up studies have shown decreases in psychophysiologic activity (e.g., muscle tension and blood pressure levels) and reductions in subjective reports of anxiety, sleep disturbances, and other health complaints with each technique (*20*). However, improvement in all these parameters persisted less than 3 months after training ended.

Stress management treats only the symptoms of the problem—not the cause. Therefore, efforts to control risk factors at the worksite are also important. Some previously described suggestions for controlling worksite risk factors for psychological disorders are listed below (*21*). These suggestions appear to have merit for reducing work-related psychological disorders, but further evaluation and study are needed for a complete understanding of their impact.

- **Work schedule**. Design work schedules to avoid conflict with demands and responsibilities unrelated to the job. Schedules for rotating shifts should be stable and predictable, with rotation in a forward (day-to-night) direction.
- **Participation/control**. Allow workers to provide input for decisions or actions affecting their jobs.
- **Workload**. Ensure assignments are compatible with the capabilities and resources of the worker, and allow for recovery from especially demanding physical or mental tasks.
- **Content**. Design tasks to provide meaning, stimulation, a sense of completeness, and an opportunity to use skills.
- **Roles**. Define work roles and responsibilities clearly.
- **Social environment**. Provide opportunities for social interaction, including emotional support and help directly related to one's job.
- **Future**. Avoid ambiguity in matters of job security and career development.

In addition to evaluation of these suggested actions, efforts are needed to advance the understanding of work-related psychological disorders and of methods appropriate for their control, including:

1. Improving the systems for surveillance of psychological disorders in the workforce as related to working conditions.
2. Improving research techniques for investigating stressful working conditions and their health consequences.
3. Improving training of occupational health professionals and workers in recognizing stressful workplace conditions and signs of worker stress and in effecting remedial measures.
4. Furthering the development of mental health components in occupational health and safety programs.

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