SIXTH EDITION

OCCUPATIONAL AND ENVIRONMENTAL HEALTH

RECOGNIZING AND PREVENTING DISEASE AND INJURY

EDITED BY

BARRY S. LEVY | DAVID H. WEGMAN
SHERRY L. BARON | ROSEMARY K. SOKAS

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Occupational and Environmental Health

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Recognizing and Preventing Disease and Injury

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Edited by
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Dedicated to the memory of
Peggy Nelson Wegman,
who created an environment
in which work, play, love, and life can all thrive.

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Preface

Occupational and environmental health issues profoundly affect every person's health and well-being. Each of us has a responsibility to address the issues that affect us as individuals, as members of families and communities, and as citizens of the world. As we prepared the sixth edition of this textbook, we directed our attention to how health professionals can recognize and prevent occupational and environmental disease and injury—at both the individual and population levels. We developed this book to enable health professionals and students in the health professions to understand these issues and the contexts in which they occur.

Dramatic changes continue to impact both occupational health—ranging from the recognition of new workplace health hazards to the changing nature of work itself—and environmental health—ranging from climate change to how airborne contaminants adversely affect health. And dramatic changes continue to impact how we obtain, analyze, communicate, and use information for research, practice, and advocacy in this field. Moreover, relationships between occupational health and environmental health are increasingly recognized: Occupational health hazards can affect communities. Environmental health problems frequently originate in workplaces. And work-related hazards, environmental degradation, poverty, and social injustice are often interrelated. This textbook aims to reflect these developments and to enable readers to prepare themselves to recognize and prevent occupational and environmental disease and injury in a changing world.

We have updated chapters from the fifth edition, continuing to emphasize aspects of both occupational and environmental health. In addition, we have added several new chapters—on Occupational and Environmental Health Surveillance (Chapter 3), Occupational and Environmental Health Equity and Social Justice (Chapter 4), Food Safety (Chapter 9), Toxicology (Chapter 25), Risk Communication and Information Dissemination (Chapter 29), Protecting Disaster Rescue and Recovery Workers (Chapter 37), Implementing Programs and Policies for a Healthy Workforce (Chapter 38), and Addressing the Built Environment and Health (Chapter 39) —and an Appendix of Selected Non-governmental Organizations.

Although our focus is primarily on occupational and environmental health in the United States, this book is designed for use by practitioners and students in health and safety professions throughout the world. We have therefore included several authors and many specific examples from other countries.

We have organized the book into five sections. Section I provides an overview of occupational and environmental health. Section II focuses on hazardous exposures. Section III addresses adverse health effects, with emphasis on clinical features and prevention. Section IV focuses on recognition, assessment, and prevention. Section V offers an integrated approach to prevention, addressing a number of cross-cutting topics relevant to occupational and environmental health. The Appendix provides a list of illustrative non-governmental organizations that readers can contact to obtain additional information and resources on specific topics.

Information alone will not prevent occupational or environmental diseases and injuries. Prevention also depends, in part, on developing the popular and political will to support prevention and to implement specific measures. Our society woefully undervalues the importance of prevention. Informed health and safety professionals and students, through their values, vision, and leadership, can help develop the popular and political will to ensure that occupational and environmental diseases and injuries are recognized and prevented, and that occupational and environmental health is achieved and maintained.

The Editors
August 2010

Acknowledgments

We greatly appreciate the assistance and support of many people in the development of the sixth edition of *Occupational and Environmental Health.* We thank the many contributing authors, whose work is appropriately credited within the text. Their findings and conclusions do not necessarily represent the views of the agencies and organizations with which they are affiliated.

We acknowledge Heather Merrell for her excellent work in preparing the manuscript and communicating with editors, authors, and the production team.

We are grateful for the outstanding work and support of Regan Hofmann, Editor, Medicine, and Rachel Mayer, Production Editor, at Oxford University Press; and Viswanath Prasanna, Project Manager, at Glyph International.

The illustrative materials throughout the book are included to offer understanding and insights not easily gained from the text. We call special attention to the work of Earl Dotter, who provided many outstanding photographs to illustrate a wide range of occupational and environmental health issues, and Nick Thorkelson, who provided many creative drawings that convey concepts and perspectives that are difficult to capture in words and photographs. We are also grateful for the photographic contributions of Aaron Sussell and others.

We express our deep appreciation to our families for their ongoing support.

Finally, we express our appreciation to students, colleagues, workers, and community members who, over the years, have broadened—and continued to broaden—our understanding of occupational and environmental health.

—The Editors

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Frequently Used Abbreviations

ACGIH	American Conference of Governmental Industrial Hygienists
ANSI	American National Standards Institute
ATSDR	Agency for Toxic Substances and Disease Registry
BLL	blood lead level
BLS	Bureau of Labor Statistics
CDC	Centers for Disease Control and Prevention
CFOI	Census of Fatal Occupational Injuries
CT	computed tomogram
EPA	Environmental Protection Agency
FEMA	Federal Emergency Management Agency
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
IPCC	Intergovernmental Panel on Climate Change
ISO	International Organization for Standardization
MRI	magnetic resonance imaging
MSDS	material safety data sheet
MSHA	Mine Safety and Health Administration
NCEH	National Center for Environmental Health
NIEHS	National Institute of Environmental Health Sciences
NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
PEL	permissible exposure limit
ppb	parts per billion
ppm	parts per million
REL	recommended exposure limit
SARA	Superfund Amendments and Reauthorization Act
STEL	short-term exposure limit
TLV	threshold limit value
TWA	time-weighted average, usually averaged over an 8-hour work shift
WHO	World Health Organization

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SECTION I

WORK, ENVIRONMENT, AND HEALTH

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1

Occupational and Environmental Health: Twenty-First Century Challenges and Opportunities

Barry S. Levy, David H. Wegman, Sherry L. Baron, and Rosemary K. Sokas

Occupational and environmental health is the multidisciplinary approach to the recognition, diagnosis, treatment, and prevention of illnesses, injuries, and other adverse health conditions resulting from hazardous environmental exposures in the workplace, the home, and the community. It is a component of medical care and of public health—what we, as a society, do collectively to ensure that the conditions in which people live and work are healthy.

The twenty-first century presents many challenges and opportunities for occupational and environmental health, as illustrated by the following examples:

A 2-year-old girl, during a routine well-child checkup, is found to have an elevated blood lead level of 20 μg/dL. Could it be related to her father's work in a smelter or the water pipes in her home?

A pregnant woman works as a laboratory technician. Should she change her job because of the organic solvents to which she—and her fetus—are exposed? Is it safe for her eat fish with elevated levels of mercury?

A middle-aged man tells his orthopedic surgeon that he is totally disabled from chronic back pain. Could it be due to his many years of heavy lifting as a construction worker?

A long-distance truck driver has recently had a myocardial infarction. When will he be able to safely return to work, and what kinds of tasks will he be able to perform?

The board of directors of a chemical company approves its production of a carcinogenic pesticide that has recently been banned in the United States. Is it ethical for the company to export it for use in developing countries?

The wife of a former asbestos worker has developed a pleural mesothelioma, presumably as a result of having washed her husband's work clothes for many years. Can she or her family receive any compensation?

An oncologist observes an unusual cluster of bladder cancer cases in a small town. Should she ask the state health department to perform an investigation?

An elderly man suffers from emphysema due to his long history of cigarette smoking. Should he curtail his activities during air pollution alerts?

Several members of a family who live next to a hazardous waste site smell odors from the site and have

developed headaches, nausea, and other symptoms. What should they do?

An epidemiologic study has found a higher lung cancer mortality rate among workers at a chemical factory. What further research studies and preventive measures should be performed?

The vice president of a small tool and die company wants to promote health of company employees. What advice would you give her?

These are but a few of the many occupational and environmental health challenges facing health workers, all of whom need to recognize and help prevent occupational and environmental health problems.

Many hazardous exposures occur in both workplaces and the general environment, such as the following:

- Contamination of the ambient air and water near a chemical factory, where its workers are also exposed to hazardous substances
- Application by agricultural workers of pesticides that may contaminate surface and ground water
- Inadvertent transport of lead, asbestos, and other hazardous substances home on workers' clothes, shoes, skin, and hair
- Exposure of workers and community residents to hazardous wastes from an industrial facility

Whether the environment is a workplace, school, home, or community setting, the pathophysiology of specific hazards in humans is the same. However, the sociology and history of environmental health and occupational health have evolved along separate tracks, with differences of focus, scale, and the people involved.

Hippocrates recognized the importance of air quality for health, although he was concerned only with the few Greeks who were "citizens"—not for the slaves or the free workers who supported them. Pliny the Elder recognized the ill effects of lead on slaves who painted ships in the first century C.E., but the use of lead in making cookware, sweetening foods, and souring vintages persisted for more than 1,800 years. Occupational hazards were not addressed systematically until 1700, when Bernardino Ramazzini, an Italian physician, published *De Morbis Artificum Diatriba (On the Diseases of Workers)*. Starting in the 1920s, Alice Hamilton, a U.S. physician and colleague of the social reformer Jane Addams, pioneered occupational health as a specialty of public health and preventive medicine. In the 1960s, Rachel Carson, a U.S. biologist and ecologist, focused public attention on the wider impact of industrial pollution in her widely read book, *Silent Spring*. In the past 40 years, extraordinary developments in science, technology, legislation, public health, and social empowerment have led to much progress in occupational and environmental health.

Even though the nature of many occupational and environmental health problems is similar, workers tend to be exposed more intensively than community residents to various hazards, and, historically, have worked for many years in a given workplace—although this is less true today. As a result, the relationship between occupational exposures and adverse health effects has provided much of the information known about hazardous substances. Populations of community residents include not only workers, who are typically healthy, but also people who are very young, those who are very old, and those with chronic diseases and other health conditions that often make them vulnerable to hazardous exposures. Exposures of community residents are often continuous, although generally at lower levels than the exposures of workers. Environmental health focuses not only on hazardous substances emanating from industrial facilities but also on such fundamental issues as sanitation, safety of food and water, and control of pests.

While there are many similarities and overlapping issues between occupational health and environmental health, governmental regulatory agencies and various health and safety disciplines have evolved in ways that have separated occupational health and environmental health. For example, in the United States, there are separate federal regulatory agencies for occupational health—such as the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA)—and environmental health—such as

the Environmental Protection Agency (EPA). In addition, there are separate federal agencies for research in occupational health—the National Institute for Occupational Safety and Health (NIOSH), within the Centers for Disease Control and Prevention (CDC)—and environmental health—the National Institute for Environmental Health Sciences (NIEHS) within the National Institutes of Health (NIH), the Office of Research and Development within the EPA, and the National Center for Environmental Health (NCEH) and the Agency for Toxic Substances and Disease Registry (ATSDR) within the CDC. Similar separation exists within state and local government agencies, educational and research institutions, non-governmental organizations (NGOs), professional associations, and elsewhere.

Occupational and environmental safety and health hazards can be classified in many ways, including the following:

1. *Safety hazards*, which result in injuries through the uncontrolled transfer of energy to vulnerable recipients from sources such as electrical, thermal, kinetic, chemical, or radiation energy. Examples include unsafe playground equipment, loaded firearms in the home, motor-vehicle or bicycle crashes, unprotected electrical sources, work at heights without fall protection, work near unguarded moving machinery, and work in unshored trenches.
2. *Health hazards*, which result in environmental or occupational illnesses, including the following:
   a. *Chemical hazards*, including heavy metals, such as lead and mercury; pesticides; organic solvents, such as benzene and trichloroethylene; and many other chemicals. There are approximately 80,000 chemicals in commercial use, 15,000 of which are frequently produced or used. Approximately 1,000 new chemicals are added to commercial use annually.
   b. *Physical hazards*, such as excessive noise, vibration, extremes of temperature and pressure, and ionizing and nonionizing radiation.
   c. *Biomechanical hazards*, such as heavy lifting, repetitive or awkward or forceful movements that result in musculoskeletal disorders, like carpal tunnel syndrome and low back pain.
   d. *Biologic hazards*, such as human immunodeficiency virus (HIV), hepatitis B and hepatitis C viruses, the tubercle bacillus, and many other bacteria, viruses, and other microorganisms that may be transmitted through air, water, food, or direct contact.
   e. *Psychosocial hazards*, such as workplaces where there is high stress due to excessive demands on, and low control by, workers; stress and hostility resulting from urban congestion, such as "road rage"; and unemployment—a major stressor.

MAGNITUDE OF PROBLEMS

Estimates have been published concerning the occurrence of occupational injuries and illnesses in the United States.[1] In 2008, a total of 5,214 workers died from occupational injuries.[2] Another 49,000 annual deaths are attributed to work-related diseases each year.[3] In 2008, an estimated 3.7 million workers in private industry and 940,000 workers in state and local government had a nonfatal occupational injury or illness; approximately half of them were transferred, placed on work restrictions, or took time away from work.[4] In 2007 (the most recent year for which data are available), an estimated 3.4 million workers were treated in emergency departments for occupational injuries and illnesses; approximately 94,000 of these workers were hospitalized.[5] Work-related injuries and illnesses are costly. In 2006, employers spent almost $87.6 billion on workers' compensation insurance payments;[6] however, this amount represents only part of all work-related injury and illness costs borne by employers, workers, and society overall, largely because the cost of many injuries and most illnesses are shifted to other health insurance systems. In developing countries, the occurrence of occupational injuries and illnesses is much higher than in this country. On an average workday in the United States,

thousands of workers become temporarily or permanently disabled and 13 workers die from workplace injuries. The highest fatal occupational injury rates are in mining, construction, and agriculture (Chapter 36). In addition, an unknown number of workers die from occupational illnesses, which affect several organ systems (Table 1-1). Many workers are exposed to occupational health and safety hazards in the workplace as well as environmental health and safety hazards at home and elsewhere. Table 1-2 describes employed civilians in the United States by industry. There has been a declining percentage of workers in the United States in heavy industry (Figs. 1-1 and 1-2) and an increasing percentage in service industries (Fig. 1-3).

The scope of environmental health problems is broad, as reflected in the subjects of the environmental health objectives for the United States for the year 2010 (Table 1-3). (Environmental health objectives for the year 2020 were not available at the time of publication of this book. They can be accessed at http://www.healthypeople.gov.) Outdoor air pollution remains a widespread environmental and public health problem, causing chronic impairment of the respiratory and cardiovascular systems, cancer, and premature death (Fig. 1-4; see also Chapter 6). Approximately 113 million people in the United States reside in areas designated as "nonattainment areas" by the EPA for one or more of the six air pollutants for which the federal government has promulgated health-based standards (ozone, carbon monoxide, sulfur dioxide, lead, particulates, and nitrogen dioxide). Motor vehicles and electrical power plants account for much ambient air pollution in the United States. Water quality continues to be a problem from both point sources, such as industrial sites, and nonpoint sources, such as agricultural runoff (Fig. 1-5; see also Chapter 8). Toxic and hazardous substances, in addition to posing health problems for exposed workers, may also cause health problems to people exposed where they live and play. Children are at increased risk for many environmental health problems, including pesticide poisoning, because of *(a)* the developing state of their neurological and other organ systems, *(b)* their higher ratio of skin surface area to body mass, and *(c)* pesticides and other toxic substances may be improperly stored or applied in areas that are easily accessible to children.

Many additional environmental factors can adversely affect the health of people in their homes and communities. These include poor indoor air quality (Chapter 7), lead-based paint (Fig. 1-6) and lead-containing water pipes, household cleaning products, mold, radon, and electrical and fire hazards. Over 90% of poison exposures reported by the American Association of Poison Control Centers have occurred in the home environment.

There are fewer reliable data available for the occurrence of environmentally related, than for

Table 1-1. Major Categories of Occupational Illness, by Organ System

Musculoskeletal disorders
Respiratory disorders
Neurologic and psychiatric disorders, including hearing impairment
Skin disorders
Reproductive and developmental disorders
Cardiovascular disorders
Hematologic disorders
Hepatic disorders
Renal and urinary tract disorders

Table 1-2. Employees on Nonfarm Payrolls by Major Industry Sector, Seasonally Adjusted (September 2009)

Industry	Size of Workforce (in millions)	
Services		55.4
Professional and business services	17.6	
Educational services	3.2	
Health care and social assistance	16.0	
Leisure and hospitality	13.1	
Other services	5.5	
Government		23.0
Wholesale and retail trade		21.4
Manufacturing		13.1
Financial activities		8.0
Construction		7.1
Transportation and warehousing		4.5
Information		3.0
Mining and logging		0.8
Total		113.3

Source: From the Bureau of Labor Statistics, U.S. Department of Labor. Accessed on December 5, 2009, at: http://www.bls.gov.

Figure 1-1. Worker at a wheel stamping plant in Michigan. Manufacturing still represents an important part of the economy and a source of many occupational health and safety hazards. (Photograph by Earl Dotter.)

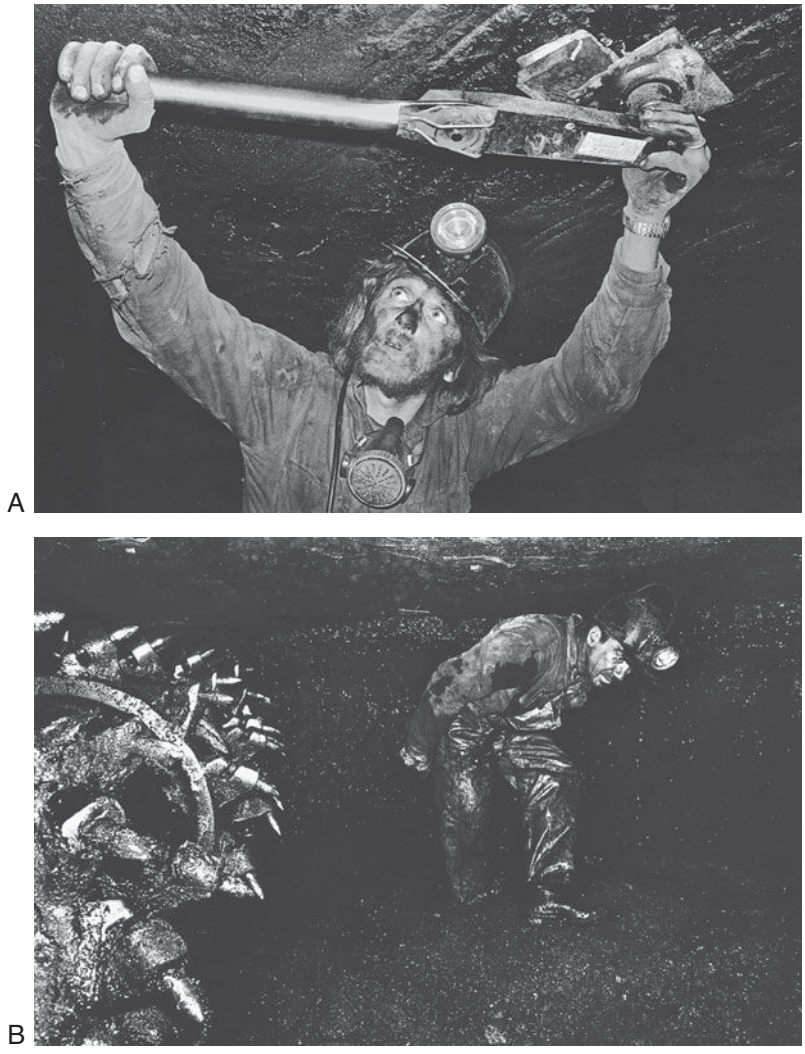

Figure 1-2. Coal miners face many occupational health and safety risks, including injuries and exposure to hazardous dusts, gases, and other substances. (A) Coal miner tests the roof support bolts in a mine. (B) Coal miner is exposed to ergonomic hazards from working in narrow mine passages. (Photographs by Earl Dotter.)

occupationally related, diseases and injuries. For some disorders, such as childhood lead poisoning, there are extensive data from screening programs, which, for example, show that 2.2% of children age 1 to 5 years had, in 2000, elevated blood lead levels (greater than 10 μg/dL). In contrast, data on pesticide poisoning are rather limited, and many cases go unreported because of the nonspecificity of symptoms. California, the state with the most extensive pesticide poisoning reporting system, found that 40% of the over 1,300 reported cases were due to nonoccupational exposures. As another example, there are extensive data on acute injuries in the home, on the road, and in other settings from various sources, ranging from vehicles to firearms. In the United States in 2000, about 30 million people were treated for injuries in emergency departments and almost 150,000 people were hospitalized. Motor-vehicle crashes are the leading cause of injury deaths, accounting for 30%. And while there are extensive data on ambient air pollution, there are only limited data on acute and chronic morbidity and

Figure 1-3. Health care workers, including these laundry workers in New York, face a number of occupational hazards, including human immunodeficiency virus, hepatitis B, hepatitis C, and other infections associated with needlestick injuries. These laundry workers found these sharp objects in soiled bed linens over the course of a year. (Photograph by Earl Dotter.)

mortality that are due to air pollution. The prevalence of asthma for the entire U.S. population between 2005 and 2007 was estimated to be 7.7%. There are a number of environmental causes of asthma, such as air pollution, environmental tobacco smoke (see Box 7-1 in Chapter 7), and other allergens. Firearms account for approximately 30,000 deaths in the United States each year.

Many occupational and environmental health problems escape detection for a variety of reasons. The difficulty in obtaining accurate estimates of the frequency of exposure-related diseases is due to several factors, as indicated below and in Figure 1-7:

1. Many problems do not come to the attention of health professionals, employers, and others, and therefore are not included in data collection systems. A worker or community resident may not recognize a medical problem as being occupationally or environmentally related, even when the connection is known. Educating workers and community residents about hazards, such as through community and workplace right-to-know campaigns, has been helpful.
2. Many occupational and environmental medical problems that do come to the attention of physicians, employers, and others are not recognized as occupationally and environmentally related. Recognition of occupational and environmental disorders is often difficult because of the long period between initial exposure and onset of symptoms (or time of diagnosis), making cause-and-effect relationships difficult to determine. It is also difficult because of the many and varied occupational and environmental hazards to which people are exposed over many years. The training of health professionals in occupational and environmental health has begun to improve health care providers' knowledge of these factors, resulting in increased recognition of occupational and environmental diseases and injuries.
3. Some health problems recognized by health professionals, employees, or others as occupationally or environmentally related are not reported because the association with the workplace or other environments is equivocal and because reporting requirements are not strict.

Table 1-3. Subjects of Environmental Health Objectives for the Year 2010, United States

Subjects	Subtopics
Outdoor Air Quality	Harmful air pollutants
	Alternative modes of transportation
	Cleaner alternative fuels
	Airborne toxins
Water Quality	Safe drinking water
	Waterborne disease outbreaks
	Water conservation
	Surface water health risks
	Beach closings
	Fish contamination
Toxics and Waste	Elevated blood lead levels in children
	Risks posed by hazardous waste sites
	Pesticide exposures
	Toxic pollutants
	Recycled municipal solid waste
Healthy Homes and Healthy Communities	Indoor allergens
	Office building air quality
	Homes tested for radon
	Radon-resistant new home construction
	School policies to protect against environmental hazards
	Disaster preparedness plans and protocols
	Lead-based paint testing
	Substandard housing
Infrastructure and Surveillance	Exposure to pesticides
	Exposure to heavy metals and other toxic chemicals
	Information systems used for environmental health
	Monitoring environmentally related diseases
	Local agencies using surveillance data for vector control
Global Environmental Health	Global burden of disease
	Water quality in the U.S.–Mexico border region

Source: http://www.healthypeople.gov/document/HTML/Volume1/08Environmental.htm. Accessed on August 10, 2010.

For example, there are only a few states where reporting of pesticide poisoning by physicians is mandatory. The initiation of occupational and environmental disease and injury surveillance activities by federal and state governments has begun to address this problem (see Chapter 3).

4. Because many occupational and environmental health problems are preventable, their very persistence implies that some individual, group, or organization is legally and economically responsible for creating or perpetuating them.

CONTEXT

Occupational and environmental health problems must be understood in social, economic, political, and historical contexts. In addition, the health and well-being of people exists in a broad ecological context. Health and safety professionals as well as many other "actors," operating in a political, economic, and social context, become involved in the recognition, assessment, and prevention and control of occupational and environmental health problems. These include the following:

- Workers, including members of labor unions
- Employers
- Representatives of business and industry associations
- Community residents
- Members of environmental non-governmental organizations (NGOs)
- Workers in the executive, legislative, and judicial branches of government at the federal, state, and local level
- Officials of international organizations

Figure 1-4. Ambient air pollution from a coal-cleaning plant in a rural area in Pennsylvania. (Photograph by Earl Dotter.)

Educators and trainers
Researchers
Print and broadcast journalists and other representatives of the news media
Officials of charitable organizations that provide financial support to programs and projects

These "actors" play different roles, rely on different sources of power and support, have different strengths and vulnerabilities, and interact with each other in multiple ways.

Partly because the treatment of occupational and environmental disorders and those not related to occupational and environmental exposures is the same, this book focuses on the recognition and prevention of these problems. Recognition focuses not only on detecting occupational and environmental illnesses and injuries in symptomatic and asymptomatic individuals (Chapter 2) but also on applying the principles of public health surveillance for detecting individual cases and overall trends of disease and injury occurrence in populations (Chapter 3).

Figure 1-5. Although nonpoint sources account for increasing amounts of water pollution in the United States, stationary point sources still account for a substantial amount of water pollution, such as with dioxin, a by-product of the manufacture of bleached white paper at this Mississippi plant. (Photograph by Earl Dotter.)

Figure 1-6. Lead-based paint in many older homes still represents a serious health hazard to many young children. This photograph depicts a lead abatement worker with personal protective equipment. Workers performing lead abatement must be trained and certified, and they must carefully adhere to safe practice standards. (Photograph by Earl Dotter.)

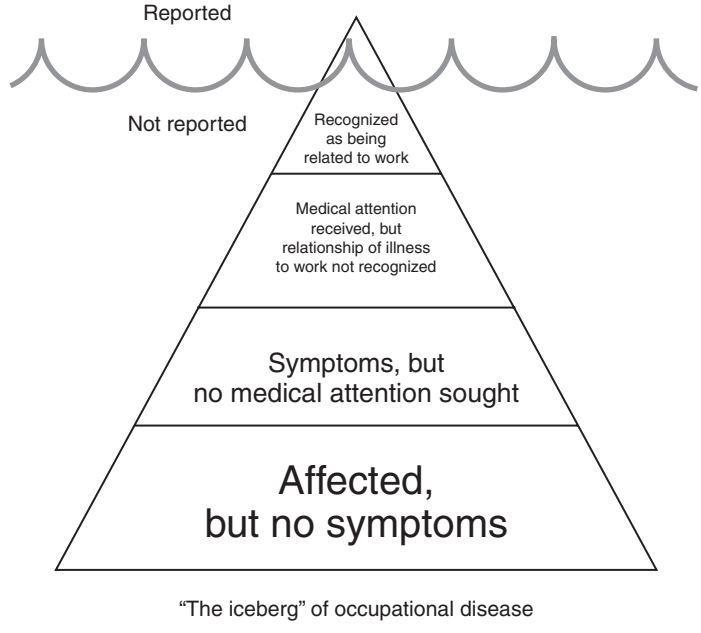

Figure 1-7. Most occupational and environmental disease is below the surface, as illustrated by the iceberg effect in this figure.

Public health principles have been applied to occupational and environmental health in preventing and controlling these adverse health effects (Chapter 2). *Primary prevention* focuses on diseases or injuries before they occur. *Secondary prevention* focuses on early identification and treatment of diseases to cure them or halt their progression. And *tertiary prevention* focuses on treatment and rehabilitation of individuals who have already developed diseases or injuries.

Another useful perspective on identifying opportunities for prevention and designing and implementing preventive measures is the traditional public health model of host, agent,

and environment. Many preventive measures focus on the host, such as the individual worker or community resident. These include education and labeling, screening programs, and, where other measures cannot be implemented, use of appropriate personal protective equipment. Other preventive measures focus more on the agent, such as insulation containing asbestos, and control measures are focused on restricting or banning production or use of the agent, or reducing human exposure to acceptable levels of risk. And some preventive measures focus on the environment. For example, designing and implementing engineering measures, such as local exhaust ventilation, can remove airborne hazards in the workplace, or installing sound-barrier walls alongside highways can reduce noise levels in adjacent residential neighborhoods. Other examples include urban planning to design more green space or bicycle routes.

ILLUSTRATIVE OCCUPATIONAL AND ENVIRONMENTAL HEALTH ISSUES

Legislation, social activism, educational activities, and other developments have contributed to increased interest in occupational and environmental health problems in recent years. Some of these developments are summarized next.

Changing Nature of Work and the Workforce

Enormous changes in work structure have taken place in recent decades, including mergers and, paradoxically, downsizing and outsourcing. For example, the production, packing, and distribution of meat in the United States is radically different now than it was 40 years ago. The number of poultry, beef, and pork producers has decreased while the size of the producers has grown. Family farms have given way to concentrated animal production operations, with large-scale production and mechanized processes, which have led to concerns about exploitation of workers, animal welfare, environmental contamination from concentrated waste, and production of greenhouse gases. Meat packaging and poultry processing plants have relocated near to large producers, and their workforce has been transformed from relatively highly paid, unionized, mostly white workers to one that is heavily comprised of immigrant Latino workers, who have low membership in labor unions, extremely high turnover, poor working conditions, and low pay (Fig. 1-8). In addition, one-third of those working in meat processing plants are contingent workers who work for subcontracting agencies and perform such tasks as cleaning and maintenance. Although these tasks often involve great hazard, workers'-compensation and OSHA requirements often fail to adequately address these contingent workers' needs. The hazards faced by undocumented immigrant workers who find themselves in informal work arrangements or day-labor settings have resulted in mortality rates for foreign-born Latino workers that are one-third higher than those of native-born citizens. Reliance on contingent and outsourced labor takes place throughout the economy, from health care to manufacturing to information technology. Other changes in the workforce over the past four decades include the integration of women into the workforce—although not in all work sectors—and the aging of the U.S. population as a whole as the "baby-boom" generation (born between 1946 and 1964) gets older. (See Chapter 4.)

Specific issues raised by these phenomena include the needs *(a)* to address the integration of family health with work schedules (Chapter 38), recognizing that work-related stresses extend into the home environment; and *(b)* to accommodate workers who have significant skills, but, for example, reduced physical capacity or visual acuity. In addition, advances in health care have increased the numbers of workers with severe impairments who nevertheless have the ability to contribute to society and the right to work, now recognized through the Americans with Disabilities Act. The careful development and implementation of redesigned community, home, and work spaces benefits all of us, in the same way that curb access has improved the lives of mobility-impaired individuals along with, for example, those of parents pushing strollers (see Chapter 39). All of these challenges can be met through concerted prevention activities, including development and implementation of employment policies, public health measures,

Figure 1-8. Workers processing chickens on an assembly line. Minority workers and women are overrepresented in entry-level jobs like this one, in which safety and health hazards are prevalent. Twenty-five workers in a similar chicken-processing plant died in 1991, when few workers were able to escape a fire that swept through the plant because the employer had locked most of the exit doors. (Photograph by Earl Dotter.)

engineering research, safety and health training, legislation and regulation, and the practice of clinical medicine.

Governmental Role

With the passage of the Federal Coal Mine Safety and Health Act in 1969 and legislation to establish OSHA and the EPA in 1970, the federal government began taking a more active role in the creation and enforcement of standards for a safe and healthful workplace and a safe and healthy ambient environment (Chapter 30). In addition, the passage of the Occupational Safety and Health Act in 1970 also established NIOSH, which *(a)* has greatly expanded epidemiologic and laboratory research into the causes of occupational diseases and injuries and the methods of preventing them; and *(b)* has strengthened the education and training of occupational health and safety professionals. In 1969, NIEHS was established as part of NIH, greatly expanding the funding for environmental health research, with an initial focus on toxicologic and etiologic work, which has expanded into community-based participatory research addressing environmental justice and other issues. The role of the U.S. government in funding scientific research, especially in the biomedical sciences, has remained strong over time. A similar sustained program to develop and implement public health measures, including surveillance tools and interventions, has never fully materialized, although interest has increased in the wake of the 9/11 terrorist attacks, the flooding following Hurricane Katrina, the emergence of the novel H1N1 influenza pandemic, numerous episodes of widespread food contamination and toy contamination, and other national public-health emergencies. Such a program would require strengthening of state and local government capacities through increased federal coordination and funding.

The roles of the federal government to set and enforce health and safety standards—for occupational or environmental contaminants, food safety, consumer protection, and many public health concerns—vary and remain controversial. After the initial attempts in 1969–1970 to bring standardization to all parts of the country

and to enact an initial series of environmental and occupational health laws—followed by promulgation of related standards, intense legal and political challenges slowed the setting of new standards to a crawl and Congressional budget cuts hampered enforcement of existing standards. Cooperative programs and educational outreach were given higher priority during an era of government downsizing and deregulation. The promotion of free trade without easing restrictions on the migration of workers caused increased immigration and growth in the undocumented and informal workforce that removed financial incentives for improved safety and health. Identifying and establishing an appropriate role for government in occupational and environmental health is a responsibility that all health and safety workers share.

Green Jobs and Green Production

Green jobs help improve the environment. Traditional jobs have changed, and new kinds of occupations have been created by energy efficiency and practices that are more environmentally friendly. However, with increased attention to green jobs and technological advances in industry, worker safety and health must not be overlooked.

Green production reduces toxic emissions by utilizing substances and processes that are more friendly to the environment. Some of these efforts have been facilitated by increasing concerns over the production of greenhouse gases and climate change. Others have been facilitated by concerns for environmental sustainability and reducing air, water, and soil pollution and improving workplace health and safety. However, the terms *green production* and *environmental protection* have been used so much that individuals and organizations need to confirm that these concepts are actually being implemented as products are produced and services provided. (See Box 2-1 in Chapter 2.)

Social and Ethical Questions

Serious social and ethical problems have arisen over such subjects as the allegiance of occupational and environmental physicians who are employed by management, worker and community "right to know" about occupational and environmental hazards, confidentiality of workers' medical records kept by employers, and the restriction of female workers of childbearing age from certain jobs. Some of the controversies on these subjects may eventually be settled by labor–management and community–company negotiations and by the deliberations of government—courts, legislatures, and executive bodies. For example, the U.S. Supreme Court has upheld a worker's right to refuse hazardous work, stating that a worker cannot be discharged or discriminated against for exercising a right not to work under conditions reasonably believed to be very dangerous (*Whirlpool Corp. v. Marshall*, 445 U.S. 1 [6th Cir. 1980]). (See also http://www.cwa-union.org/pages/Right_to_Refuse_Unsafe_Work.)

Environmental Justice

Disparities in environmental exposures between high-income and low-income communities partially account for differences in health status between those communities. The environmental justice movement is a network of people and organizations in low-income and minority communities who are fighting against placement in their communities of hazardous waste sites and polluting facilities. This movement has transformed the environmental movement from one supported primarily by the middle class and focused on ecological issues, to a grassroots struggle of poor and working-class communities who are concerned primarily with preserving the health of their families. Many environmental health professionals work with teams of urban sociologists, economists, community activists, and others to develop multidisciplinary prevention programs to decrease environmental health factors that contribute to health disparities. (See Chapters 4 and 33.)

Security and Terrorism Preparedness

The terrorist attack on the World Trade Center in 2001 followed by anthrax-tainted mail to Congressional offices and media companies led to increased awareness of the need for public health preparedness, which became a national priority. All of this highlighted the important role of occupational and environmental health.

Environmental contamination from the collapse of the World Trade Center caused respiratory and other disorders among community residents and rescue and recovery workers. Twenty-three people developed anthrax, five of whom died, as a result of their exposure to the contaminated mail. Environmental and occupational health workers played key roles in both of these situations—in identifying and measuring contaminants and in developing screening, treatment, and prevention programs. Subsequent investigations identified key vulnerabilities for potential future terrorist attacks, including the security of the food supply and chemical manufacturing facilities near heavily populated areas. These concerns are likely to continue to have an impact on the training and future roles of environmental and occupational health workers. (See Chapter 37.)

Liability

Some workers, barred from suing their employers under workers' compensation laws, have turned to "third-party," or product-liability, lawsuits as a means of redress for occupational disease; some community residents exposed to environmental hazards have also done so (Chapter 31). Fear of lawsuits has driven many employers to focus on preventive activities. Such lawsuits play an important role in directing attention to prevention of some diseases, although this approach can be cumbersome and outcomes may not be equitable. (In some jurisdictions, some of the most egregious health and safety offenders have been criminally prosecuted.) In recent years, plaintiffs and their attorneys have found it increasingly difficult to recover damages in such lawsuits for a variety of reasons, including federal and state court decisions that have disqualified testimony of experts.

Advances in Technology

Advances in technology continue to facilitate identification of workplace hazards and potential hazards, including increasing use of in vitro assays to determine the mutagenicity of substances—and therefore their possible carcinogenicity, improvements in ways of determining the presence and measuring the levels of hazardous exposures, and new methods of monitoring concentrations of hazardous substances in body fluids and the physiologic impairments they cause. In addition, technological breakthroughs have introduced new hazards into the workplace and ambient environment (see Box 26-2 in Chapter 26). The huge oil spill in the Gulf of Mexico in 2010 is an example of how advances in technology may introduce new hazards.

Promoting a Healthy Workforce

The overall health of the population is influenced by factors both inside and outside the workplace. Not only do workers experience stress and physical and chemical exposures at work and in the community, but these factors can also influence health behaviors such as diet, exercise, smoking, and alcohol use. The effects of these many factors cannot be artificially divided between "at work" and "non-work." Workplace conditions can affect health and well-being at home and in the community; exposures, activities, and conditions outside of working hours can substantially determine health, productivity, and responses to exposures during work. Recognizing this complexity, new health behavior theories have developed that incorporate the importance of both contextual environmental factors and personal and community empowerment in achieving and maintaining good health.

Careful study and understanding is required to evaluate health interventions to demonstrate which aspects succeed and which do not. These processes are often more time-consuming and expensive than traditional approaches that, for example, might rely on a pamphlet to encourage people to eat more fruits and vegetables. Instead, community-based participatory research has identified structural issues, such as the absence of stores selling fruits and vegetables in a given neighborhood, and personal and cultural factors, such as traditional cooking methods and tastes. Identification of these structural issues has led to projects that engage community members to develop, implement, and assess change. Similar projects addressing lead poisoning, triggers of asthma in the home and the community, and exercise recommendations for low-income populations are being implemented. Similarly, workers are providing input into development

of preventive measures to reduce work-related injuries. And integrative approaches that address both personal habits and occupational hazards are making smoking cessation programs for blue-collar workers more effective. (See Chapter 38.)

Economic Globalization

The growth of multinational corporations, reduction in trade barriers, and development of regional treaty arrangements, such as the North American Free Trade Agreement (NAFTA), and global organizations, such as the World Trade Organization (WTO), are often adversely impacting occupational and environmental health. In many developing countries, multinational corporations have exploited workers by employing them in jobs that have low wages and few benefits, offer little or no training or upward mobility, and exposure to serious health and safety hazards. (See Chapter 4.)

Additional Challenges in Developing Countries

In addition, developing countries—which comprise two-thirds of all countries and include the vast majority of people worldwide—face other challenges, which will be described next.

Export of Hazards

Developed countries often export their most hazardous industries, as well as hazardous materials (such as banned or restricted pesticides) and hazardous wastes, to developing countries, where laws and regulations concerning these substances are more lax or nonexistent and people may be less aware of these hazards (Fig. 1-9; See Box 4-4 in Chapter 4 and Box 20-1 in Chapter 20).

Inadequate Infrastructure and Human Resources

In developing countries, there are far fewer adequately trained personnel to recognize, diagnose, treat, and prevent and control occupational and environmental health problems. Governments and other sectors of society have fewer resources to devote to occupational and environmental health; and labor unions, facing other challenges such as low wages and high unemployment, often give little attention to occupational health and safety.

Figure 1-9. Agricultural workers are at high risk of poisoning from pesticides. (Photograph by Earl Dotter.)

Transnational Problems

Occupational and environmental health problems in developing countries often involve multiple countries in the same region, requiring transnational or regional approaches to problems, such as development and implementation of transnational standards.

Relationship between the Workplace and the Home Environment

In developing countries, where so many people work in or near their homes, the distinction between the workplace and the home environment is blurred. As a result, family members may often be exposed to workplace hazards.

Economic Development

Governments of developing countries often give high priority to economic development, sometimes even over the health of their people. In the context of economic development and accompanying rapid industrialization and urbanization, there is often pressure to overlook occupational and environmental health issues, given limited resources and the fear that attention to these issues may drive away potential investors or employers. Similarly, workers desperate for jobs in economies with high unemployment rates are unlikely to complain about occupational and environmental health and safety hazards once they are employed. In addition, many children are forced to leave school in order to work, often in hazardous jobs. (See Figs. 1-10 and 1-11 and Chapter 4.)

Occupational and Environmental Health Services and Primary Health Care

Given limited resources and infrastructure, many developing countries are exploring ways to integrate occupational and environmental health services with primary medical care and with a broader range of public health services. Although some successes have been achieved with this approach, there remains much untapped potential in fully achieving this kind of integration.

DISCIPLINES AND CAREERS IN OCCUPATIONAL AND ENVIRONMENTAL HEALTH SCIENCES

Identification and remediation of threats to the environment is a stewardship responsibility for

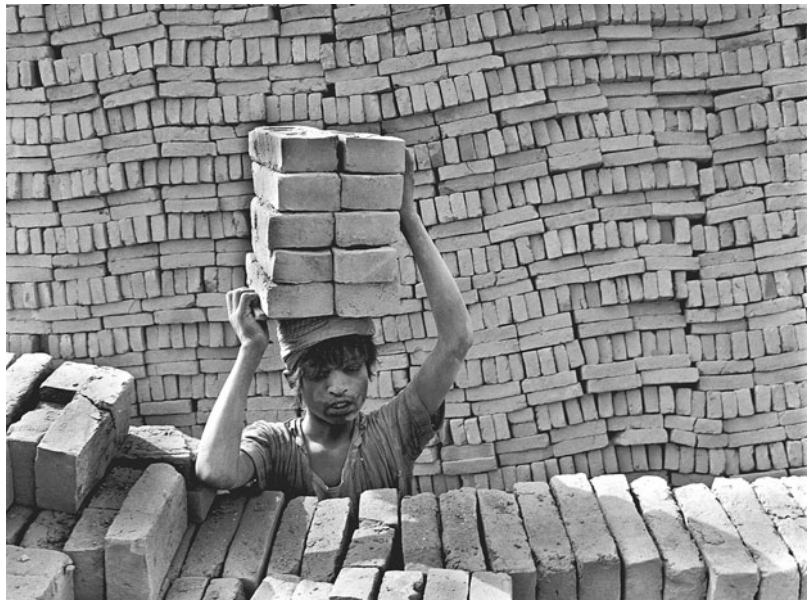

Figure 1-10. Young boy hauling fired bricks for storage in Nepal, 1993. Thousands of children are forced to work in brick kilns, rock quarries, or mines. (Photograph by David L. Parker.)

Figure 1-11. Migrant workers picking cotton. These workers face many challenges because of their minority status, poverty, inadequate education, and lack of information and control over the agrochemicals to which they are exposed. (Photograph by Earl Dotter.)

us all. For those who work in medical care or public health, there are a wide range of career options that span the physical, biologic, and social sciences as well as communications, policy making, and other fields. One of the most important challenges we face is the ability to communicate effectively across disciplines to develop the collaborative approaches needed to create safe, healthy, and sustainable environments for future generations.

Almost all health care providers encounter occupational and environmental health issues. The American College of Graduate Medical Education recognizes the specialty of preventive medicine, which includes three areas of expertise: public health and general preventive medicine, occupational medicine, and aerospace medicine. Physicians who choose to specialize in any of these areas may wish to become certified by the American Board of Preventive Medicine. (For criteria for certification, please access the American Board of Preventive Medicine Web site, http://www.abpm.org.) The American College of Occupational and Environmental Medicine is a primary professional association for physicians engaged in the practice of occupational and environmental medicine.

The field of nursing is similarly integrated with communication and prevention—key aspects of environmental and occupational health practice. For those who wish to specialize in the application of the science of occupational and environmental health in nursing practice, advanced practice degrees in nurse-practitioner programs and advanced master of science in nursing and doctoral programs are available. The American Association of Occupational Health Nurses is the primary professional association for occupational health nurses and represents nurses across the spectrum of practice.

Physicians' assistants are midlevel practice professionals who are trained typically in an applied master of science degree program. They have formed the practice core for several large occupational health programs in industry and in the Veterans Administration health system.

Other health care professions important to the field of environmental and occupational

health include audiology, physical therapy and rehabilitation, clinical psychology, clinical social work, and optometry.

A wide range of environmental health science programs are available at levels ranging from community colleges to postgraduate doctoral programs, with credentialing based on education, experience, and certifying examinations available for registered environmental health specialists, sanitarians, environmental health technicians, food-safety professionals, hazardous-substance professionals, and others.

Engineering and public health programs overlap in the training of industrial hygienists and environmental engineers, who provide primary prevention through exposure assessment as well as design and implementation of interventions. Radiation physicists and biologists address a specific aspect of environmental and occupational exposure assessment and prevention.

Safety professionals have education in engineering disciplines, often with additional management training. Bachelor, master, and doctoral programs are available. Public health practitioners are also trained through a variety of programs, although the core public health sciences—epidemiology, biostatistics, environmental health, health services administration, and health education/behavioral sciences—form the basis of the core professional degree, the Master of Public Health.

Occupational health psychologists apply psychology to improving the quality of work life and to protecting and promoting the safety, health, and well-being of workers.

Research into any of the occupational and environmental health sciences can form the basis for a doctoral program, which focuses on advancement of scientific knowledge. These sciences include toxicology, the study of the effects of foreign substances on living organisms; epidemiology, the science of the distribution and determinants of disease in populations; environmental chemistry, concerned with the fate and transport of pollutants in the environment; systems engineering, the study of processes and their improvement; and sociology, psychology, and anthropology, all of which are critical to the understanding of human behavior in relation to the environment. Communications science, including social marketing and journalism, represents an important related area of study and practice. Environmental law, economics, policy, urban planning, and environmental management are other important areas of work. Finally, the many fields of ecology, agronomy, chemistry, physics, and geology that do not directly address the human health impacts, but are nevertheless critical to our understanding of the external environment and our impact on it, provide additional career opportunities in occupational and environmental health.

CONCLUSION

Many health professionals will eventually work on occupational and environmental health and safety issues, and some will become occupational and environmental health and safety specialists. But almost all health professionals—in one way or another—will be involved with the recognition, diagnosis, treatment, or prevention and control of occupational and environmental illnesses and injuries.

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American Journal of Industrial Medicine, published monthly by Wiley-Liss, Inc.

American Journal of Public Health, published monthly by the American Public Health Association.

Environmental Health Perspectives, published monthly by the National Institute of Environmental Health Sciences.

International Journal of Occupational and Environmental Health, published quarterly by Hamilton Hardy Publishing, Inc.

Journal of Occupational and Environmental Medicine, the journal of the American College of Occupational and Environmental Medicine, published monthly by Wolters Kluwer Health/ Lippincott Williams & Wilkins.

New Solutions: A Journal of Occupational and Environmental Health Policy, published quarterly by the Baywood Publishing Company, Inc.

Occupational and Environmental Medicine, the journal of the Faculty of Occupational Medicine of the Royal College of Physicians of London, published monthly by the BMJ Publishing Group, Ltd.

Scandinavian Journal of Work, Environment & Health, published every other month by the Finnish Institute of Occupational Health, the Danish National Research Centre for the Working Environment, and the Norwegian National Institute of Occupational Health.

Occupational Health Nursing

American Association of Occupational Health Nurses Journal, published monthly by the American Association of Occupational Health Nurses.

Occupational and Environmental Hygiene

Journal of Occupational and Environmental Hygiene, published monthly by the American Industrial Hygiene Association and the American Conference of Governmental Industrial Hygienists.

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The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

2

Recognizing and Preventing Occupational and Environmental Disease and Injury

Rosemary K. Sokas, Barry S. Levy, David H. Wegman, and Sherry L. Baron

A woman complaining of headache and vomiting visited an urgent care center, where she was diagnosed with a viral syndrome and treated symptomatically. She and her husband and three children returned the following night with the same symptoms and were diagnosed with carbon monoxide poisoning. Inspection of their home revealed a faulty furnace.

A woman sought medical attention for a painful right knee. The physician prescribed diet and exercise with no improvement. At a follow-up visit, she announced that her problem had resolved after a co-worker showed her how to pad the knee-operated pedal on her sewing machine at work.

Orders for lead-containing chemical products from a factory increased when its competitor went out of business. As production increased, physicians found an increased number of workers with high blood lead levels (BLLs), removed them from work using the Occupational Safety and Health Administration (OSHA) Lead Standard, and returned them to work only after their BLLs declined. Several workers went through this cycle repeatedly. Some workers' children had elevated BLLs on routine testing by their pediatricians.

In the same factory, in response to an OSHA inspection, an overhead exhaust hood was installed in the room where workers opened bags of inorganic lead to feed into a hopper to be mixed into a final product. The purpose of the hood was to provide local exhaust ventilation to capture and remove lead dust from the workers' breathing zones. The workers had been using a table to hold the bags before opening them and dumping them; with the new hood, there was no room for the table, and bags were instead placed on and lifted from the floor. The number of back injuries resulting in lost work time increased.

In a mining town, occupational health investigations conducted in the 1980s revealed asbestos-related diseases and deaths among workers who mined and processed asbestos-containing vermiculite ore. These workplace deaths were not seen as sentinel health events. Vermiculite waste was not controlled until deaths from mesothelioma were reported in community residents in the 1990s.

Recognizing an occupational or environmental illness and injury requires characterizing a specific health outcome, identifying a hazardous exposure, and determining a relationship between exposure and outcome. Once this relationship has been established, interventions can be developed that will interrupt the causal pathway, thereby preventing illness and injury—the goal of occupational and environmental health and safety. Since illnesses and injuries may be

difficult or impossible to treat or may result in complications, it is best to identify a problem early, when the affected person may be treated effectively and when other people at work and in the community can be protected.

The five examples at the start of this chapter demonstrate how occupational or environmental illnesses may fail to be recognized and opportunities for prevention may be missed. These examples highlight common themes, such as the need for better communication and feedback.

RECOGNIZING OCCUPATIONAL AND ENVIRONMENTAL DISEASES AND INJURIES

Occupational and environmental diseases and injuries can be recognized at the individual level, which depends on obtaining and assessing an occupational and environmental history, as discussed in the following section. In addition, occupational and environmental diseases and injuries can be recognized by surveillance at the population level, as discussed briefly later in this chapter and more thoroughly in Chapter 3.

The Occupational and Environmental History

Obtaining an occupational and environmental history helps a clinician to understand patients in the context of their lives; design anticipatory guidance; provide specific advice about work, community, or home exposures; and diagnose symptomatic individuals.

Consider the following five cases:

1. An emergency medicine physician diagnosed acute alcohol intoxication in a machinist who developed loss of balance at work.
2. A primary care physician diagnosed a garment worker's finger numbness and weakness as an exacerbation of her rheumatoid arthritis.
3. An internist diagnosed the worsening chronic cough of a man working at a bottle-making factory as a side effect of his antihypertensive medication.
4. A physician attributed a young boy's learning difficulties in school to borderline mental retardation.
5. A pediatrician concluded that a young girl's asthma exacerbation was caused by a viral infection.

In each of these cases, the facts fit together and resulted in a coherent story, leading each physician to recommend a specific therapeutic and preventive regimen. But, in each case, the physician made an inadequate or incorrect diagnosis because of a common oversight—failure to take an occupational and environmental history.

The first patient had acute central nervous system (CNS) intoxication caused by exposure to organic solvents at work.

The garment worker had carpal tunnel syndrome, possibly caused by some combination of her rheumatoid arthritis and the strenuous repetitive movements she performed with her hands and wrists hundreds of times an hour.

The man working in the bottle-making factory had worsening of his chronic cough and other respiratory tract symptoms as a result of occupational exposure to hydrochloric acid fumes.

The young boy had lead poisoning due to inhalation of dust from leaded paint in his home.

And the young girl had exacerbation of her asthma caused by allergy to mold growing in the basement of her home as a result of water damage.

The associations noted by the physicians may have contributed to causing the second and fifth cases. But without an occupational and environmental history, appropriate treatment and prevention could not have been performed. Although a physical examination and laboratory tests may raise suspicion or help confirm that a medical problem is related to occupational or environmental factors, identification of occupational and environmental health problems depends most importantly on the occupational and environmental history.

What Questions to Ask

The occupational and environmental history is outlined in Table 2-1. Most clinical situations do not require obtaining complete histories.

Table 2-1. Outline of the Occupational and Environmental History

Components	Specific Questions and Issues
Description of all jobs held	Obtain information on employers, details of jobs, and starting and ending dates of each job. Ask about second jobs, work in the home as a homemaker or parent, military service, and part-time and summer jobs. Ask worker to describe typical work shift. Ask worker to simulate performance of work tasks by demonstrating body movements associated with them. (Visiting the workplace may be necessary.) Obtain information on routine tasks as well as unusual and overtime tasks, such as cleaning out tanks or cleaning up spills.
Exposures	Ask about chemical, physical, biomechanical, biological, and psychosocial exposures at workplaces. Start with open-ended questions, such as "What have you worked with?" Follow with specific questions, such as "Were you ever exposed to lead or other heavy metals? To solvents? To asbestos?" Obtain material safety data sheets (MSDSs) for workplace chemicals. Ask about tasks performed in adjacent areas of the workplace that may contribute to a worker's exposure. Ask about unusual incidents, such as spills of hazardous materials, work in confined spaces (Fig. 2-1), use of new substances, and changed processes at work. Quantify exposures to the extent feasible, usually by estimating concentration and determining duration of exposure and route of entry. Check for the presence of protective engineering systems and devices, such as ventilation systems, and whether they seem to function adequately. Check for the use of personal protective equipment, such as gloves, work clothes, masks, respirators, and hearing protectors. Ask about eating, drinking, and smoking in the workplace (Fig. 2-2). Ask about hand washing and showering at work, changing of work clothes, and who cleans the work clothes.
Timing of symptoms	What is the time course of symptoms in relation to exposures? When do symptoms begin and end in relation to work shifts? Are symptoms present during weekends and vacation periods? Are symptoms related to certain processes, work tasks, or work exposures?
Symptoms among co-workers	Are there other workers at the same workplace or in similar jobs elsewhere who have the same symptoms or illnesses? If there are people similarly affected, find out what they may share in common.
Present and prior residences	List all the places where you have lived and the periods when you lived at each place. Have you ever lived near any of the following: *(a)* an industrial facility that may be polluting the air, surface or ground water, or the soil; *(b)* a hazardous waste site; and *(c)* a farm where pesticides or herbicides may have been applied.
Jobs of household members	Ask if workplace contaminants, such as lead, may have been brought into the home. Ask if children have been brought to the worksite, such as occurs frequently for farm work.
Environmental tobacco smoke	Do you share your home, car, or other environment with a smoker?
Lead exposure history	Have you ever lived in a home built before 1978? Have you known anyone who has had lead poisoning? (If yes, please provide additional information.) Is lead present in pipes in your home or supplying water to your home? Is there imported pottery in your home? Do you use folk remedies that may contain lead?
Home insulating, heating, and cooking	What type of fuel do you use for heating or cooking in your home? What type of insulation do you have in your home? Is your stove properly ventilated?
Household building materials	What type of materials is your home made of?
Home cleaning agents and other household products	What type of cleaning agents and other household products do you use in your home?

(*Continued*)

Table 2-1. Outline of the Occupational and Environmental History (Continued)

Components	Specific Questions and Issues
Presence of pests, mold, pets, dust in the home	Do you have dust mites or cockroaches in your home? Do you have growth of mold in your home? Is there evidence of water damage in your home? What type of carpeting do you have in your home? What pets do you have in your home?
Pesticide usage	What types of insecticides, herbicides, or other pesticides have you used in or near your home?
Water supply	What is the source of water for your home?
	If you have a private well, when was it last tested and what were the results?
Foodborne illness	What food was eaten in the time period just before onset of illness?
Renovation/remodeling	Has your home recently been renovated or remodeled?
Air contamination	Are you concerned about contamination or pollution of the air in or near your home? (If yes, please describe.)
Hobbies	What hobbies do you or other household members have?
	Do any of these hobbies cause contamination of the air or other hazards?
Recreational history	Have you been exposed to any hazards in recreational activities, such as swimming in polluted bodies of water?
Travel	Please describe any recent travel.

Figure 2-1. Many jobs require work in confined spaces, which requires specialized training and procedures, which are specified in the OSHA standard on Permit-required Confined Spaces (1910.146) (Photograph by Earl Dotter.)

Figure 2-2. Workers eating in the workplace may ingest toxic substances. (Photograph by Earl Dotter.)

But patients should always be asked what work they do. Health professionals need to exercise judgment in choosing which questions to ask. For a comprehensive medical examination, however, a question or two in the psychosocial section of the medical history is not enough; the clinician should obtain information, as deemed appropriate, on current and major past occupations of patients as well as information on residential and other environmental exposures. The extent of detail depends largely on the clinician's level of suspicion that occupational or environmental factors may have caused or contributed to the patient's illness. The occupational history should always contain sufficient detail to understand how patients spend their workdays and to determine safety and health hazards, including any potentially hazardous chemical, physical, biomechanical, biological, or psychological exposures.

Some hospitals and clinics have standardized forms for occupational and environmental histories, which can facilitate obtaining and recording this information. Ideally, such forms should include *(a)* a grid with column headings for employer, job title, primary job tasks, dates of starting and stopping the job, and major work exposures (Fig. 2-3); and *(b)* a series of questions on environmental exposures. It may be helpful to ask questions, from a list prepared in advance, about whether the patient has had any exposures to specific hazardous substances or physical factors, such as noise or ionizing radiation.

Further elaboration on each of the key parts of the occupational and environmental history may be helpful, especially when *(a)* the patient raises concerns about potential exposures, *(b)* the clinician needs to further evaluate exposures of concern, *(c)* organ systems that are commonly associated with exposure are adversely affected, or *(d)* the diagnosis remains unclear.

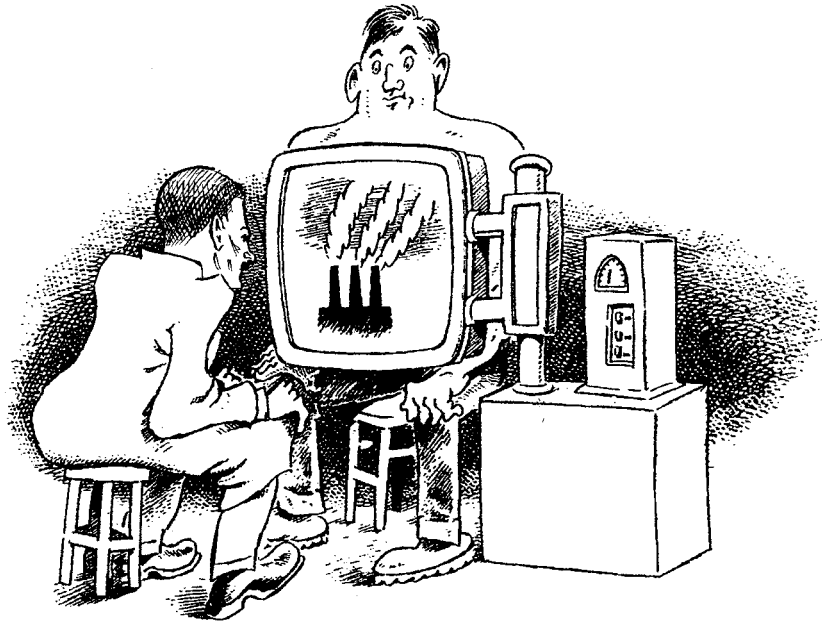
Sometimes there is an additive or synergistic relationship between occupational and/or environmental factors in causing disease. The clinician should ask whether the patient smokes cigarettes—or is exposed to environmental tobacco smoke, or drinks alcohol; if so, amount and duration should be quantified. For skin problems, questions should be asked regarding recent exposure to new soaps, new cosmetics, or new clothes.

One should determine whether the worker has had preplacement or periodic physical and laboratory examinations at work. For example, preplacement audiograms or pulmonary function tests may suggest the presence of occupational risk and may be helpful in determining whether hearing impairment or respiratory symptoms are work-related. Because OSHA regulations mandate periodic screening of workers with certain exposures, such as loud noise and asbestos, and because many employers voluntarily provide screening tests at work, such information may be available to a clinician, if the worker approves its release.

Finally, one should ask the worker if there is some reason to suspect that symptoms may be related to identified exposures, if the person

Employer	Job title	Primary job tasks	Start	End	Work exposures
ELECTRICIANS UNION	STEWARD	REPRESENT WORKERS	6/07	→	JOB STRESS
BRIGHT EXTERIORS	PAINTER	SPRAY PAINT, SCRAPE OFF OLD PAINT	12/99	4/07	PAINT, THINNERS, LEAD
XYZ FOUNDRY	LABORER	CAST METAL	11/98	10/99	LEAD, SILICA DUST, NOISE
TOWN OF FRANKIN	FIREFIGHTER	RESPOND TO FIRES, OTHER EMERG.	3/95	10/98	SMOKE, FUME, HEAT, FALLING OBJECTS
COUNTY MED. CENTER	HOSP. ORDERLY	MOVE + LIFT PATIENTS, EQUIP.	5/92	1/95	HEAVY LIFTING, SHARPS, BLOOD
ROADSIDE NURSERY	HELPER	SPRAY CHEMICALS, MOVE PLANTS	9/89	4/92	PESTICIDES, FERTILIZERS
U.S. ARMY	VEHICLE MECHANIC	SERVICE + REPAIR TRUCKS	7/87	6/89	MOTOR OIL, GASOLINE, SOLVENTS
GREEN'S GROCERY	STOCK CLERK	STOCK SHELVES	6/86	8/87	LIFTING HEAVY BOXES

Figure 2-3. Part of a sample occupational and environmental history, which also includes questions on environmental exposures, smoking history, timing of symptoms in relation to exposures, and symptoms or illness among co-workers and household contacts.



Physicians and other health professionals have a vital role in recognizing occupational and environmental disease. Contrary to the drawing above, there is no simple test. The suspicion and the determination of work-relatedness depend primarily on a carefully obtained occupational and environmental history. (Drawing by Nick Thorkelson.)

works with or encounters any known hazards, and if preventive measures are taken at work—and, if so, for what purpose.

When to Take Complete a Occupational and Environmental History

In the following situations, the clinician should have a strong suspicion of occupational and environmental factors or influences on the development of the problem and take a more detailed or complete occupational and environmental history. Many nonspecific symptoms arise from occupational and environmental exposures.

Respiratory Disease

Virtually any respiratory symptom can be related to occupational and environmental factors. It is easy to misdiagnose acute respiratory symptoms as acute tracheobronchitis or viral infection when the actual diagnosis is occupational asthma, or to attribute chronic respiratory symptoms to chronic obstructive pulmonary disease when the actual diagnosis is asbestosis. Viruses and cigarettes are often mistakenly assumed to be the sole agents responsible for respiratory disease. Environmental factors, including ambient and indoor air contaminants, account for a substantial portion of childhood asthma (Box 18-1). Adult-onset asthma is frequently work-related, but it is often not recognized as such. In addition, patients with preexisting asthma may have exacerbations of their otherwise quiescent condition when exposed to workplace sensitizers. Less commonly, pulmonary edema can be caused by workplace chemicals, such as phosgene or oxides of nitrogen. A detailed occupational and environmental history should be obtained for anyone with acute respiratory illness when no likely nonoccupational or nonenvironmental cause can be identified. (See Chapter 18.)

Skin Disorders

Many skin disorders are self-limited, but they can impact a person's daily activities, including work. The occupational and environmental history may identify the offending irritant, sensitizer, or other factor. Contact dermatitis, which accounts for about 90% of cases of work-related skin disorders, may be challenging to treat. Determining work-relatedness and the causative agent depends on a carefully obtained history. (See Chapter 22.)

Hearing Impairment

Many cases of hearing impairment are falsely attributed only to aging (presbycusis). Millions of U.S. workers have been exposed to hazardous noise at work, home, rock concerts, or elsewhere. A detailed occupational and environmental history should be obtained from anyone with hearing impairment. Recommendations for the prevention of future hearing loss should also be made. (See Chapter 21.)

Back and Joint Symptoms

Most back pain is at least partially work-related. But there are no tests or other procedures that can differentiate work-related from non–work-related back problems. Determination of work-relatedness depends on the occupational and environmental history. Many cases of arthritis and tenosynovitis are caused by awkward, forceful, or repetitive movements at work. Ergonomics—the study of the complex interactions among workers, their workplace environments, job demands, and work methods—can help prevent some of these problems. (See Chapters 16 and 27.)

Cancer

A number of occupational and environmental exposures are established causes of cancer. As new chemicals are introduced into commercial use and as epidemiologic and other studies increase our understanding of their potential hazards, more chemicals are being identified as carcinogens and probable carcinogens. Sometimes the initial suspicion that a substance may be carcinogenic comes from clinicians' reports, especially when a cluster of cases of a rare cancer occurs. Identification of occupational and environmental cancer would be facilitated if an occupational and environmental history was obtained from all patients with cancer. Exposure to a carcinogen may have begun many years before diagnosis of cancer and exposure need not have been continuous between onset of exposure and diagnosis. (See Chapter 17.)

Exacerbation of Coronary Artery Disease Symptoms

The frequency or severity of symptoms of coronary artery disease may increase due to chemicals in the workplace, workplace psychological stress, unemployment and underemployment, and environmental exposures, such as to lead, carbon monoxide, and fine airborne particulates. (See Chapters 3, 6, 11, 14, and 23.)

Liver Disease

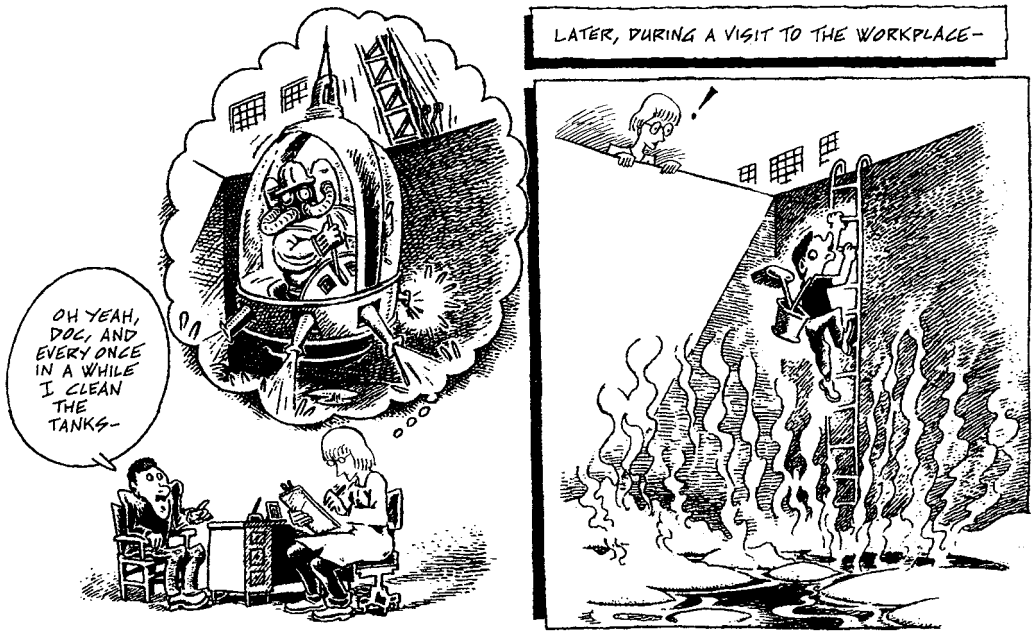
The liver is the major site for metabolizing chemicals in the body. The association between alcohol and liver disease may lead a clinician to overlook occupational or environmental causes of liver disease, such as hepatotoxins and hepatitis B virus. The occupational and environmental history should include questions on use of over-the-counter medications and traditional remedies that may adversely affect the liver. (See Chapters 11 and 13.)

Neuropsychiatric Problems

The possible association of neuropsychiatric problems to occupational and environmental factors is also often overlooked. Peripheral neuropathy is often attributed to diabetes, alcohol abuse, or "unknown etiology." Central nervous system depression is often attributed to substance abuse or a psychiatric disorder. And behavioral abnormalities—which may be the first sign of work-related stress or, in a child, lead poisoning—are often attributed to a psychosis, a personality disorder, or attention deficit hyperactivity disorder. More than 100 chemicals, including virtually all organic solvents, can cause CNS depression, and several neurotoxins—including arsenic, lead, mercury, and methyl n-butyl ketone—can produce peripheral neuropathy. Carbon disulfide exposure can cause symptoms that mimic a psychosis. And manganese can cause symptoms of parkinsonism. (See Chapters 14 and 19.)

Illnesses of Unknown Cause

A detailed, complete occupational and environmental history should be obtained in cases where the cause is unknown or uncertain or the diagnosis has not been established. The need to search carefully for an occupational and environmental source in such illnesses results from many factors, including the increases in informal work and self-employment and the use of alternative medicines and food supplements. Illnesses of unknown origin may also be due to exposure to hazardous wastes (Chapter 10) or



It is crucial to clearly understand working conditions and exposures. (Drawing by Nick Thorkelson.)

contamination of indoor air or water (Chapters 7 and 8).

Certain types of conditions and circumstances require the clinician to take a more in-depth approach. For example, a worker's back strain might require the clinician to play a role in designing or modifying work tasks to prevent a recurrence in the worker or prevent cases affecting other workers. (See Chapter 16 and 27.)

Proper diagnosis of illness or injury requires information from a variety of sources. Successful identification of an association with an occupational or environmental factor rarely results from a single laboratory test or diagnostic procedure, but rather depends on a comprehensive patient history that explores the relation of illness to occupational and environmental exposures and conditions. Health professionals play vital roles in recognizing occupational and environmental illness and injury. Contrary to the cartoon on page 28, there is no simple test.

Preventing Occupational and Environmental Illnesses and Injuries

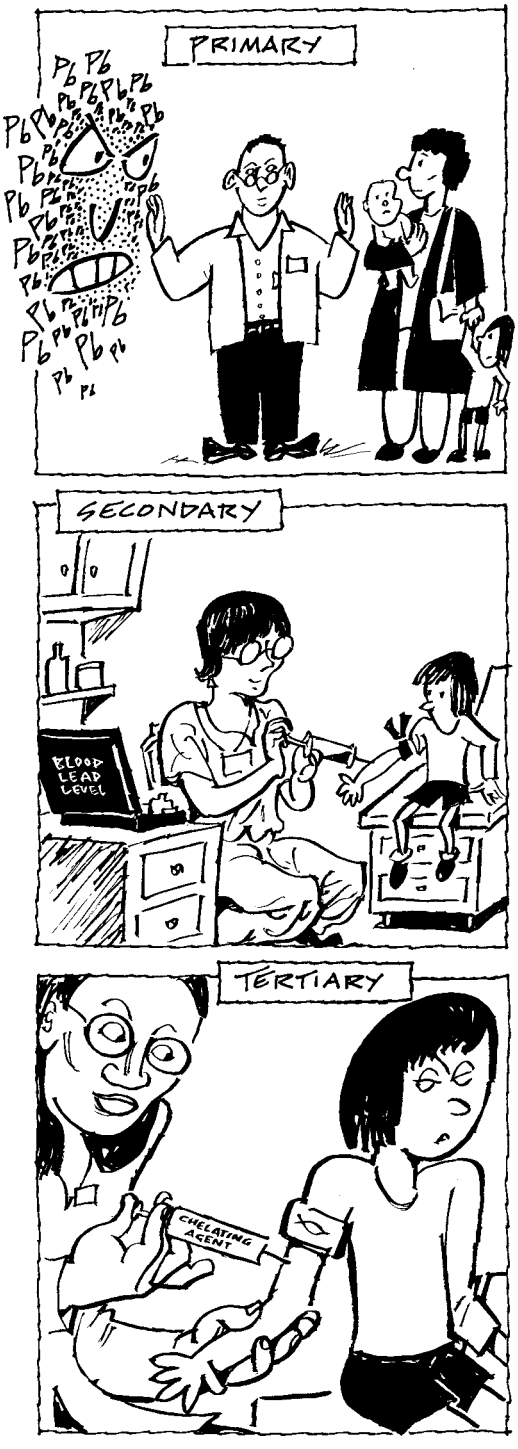
Prevention generally involves a sequence of measures, including the following:

1. Obtaining information about the causal relationship between exposures and outcomes by performing research, and disseminating this information
2. Identifying or diagnosing health problems in individuals and groups
3. Developing, communicating, and implementing preventive measures
4. Evaluating the process and outcome of implementing these measures

Information sharing can enable and empower individuals to recognize hazards and prevent illnesses and injuries. Examples include measures taken by *(a)* individuals, such as using seat belts and stopping smoking; *(b)* communities, such as constructing safe bicycle paths and speed bumps; and *(c)* workplace safety and health teams. Successful programs produce results, such as preventing dermatitis among printing workers and back injuries and needlesticks among health care workers.[1-3]

Health and safety professionals need to anticipate and recognize hazards in a systematic manner, and to design and implement preventive measures at all three levels of prevention:

1. *Primary prevention* to deter or avoid illnesses and injuries



Examples of primary, secondary, and tertiary prevention. (Drawing by Nick Thorkelson.)

2. *Secondary prevention* to identify and treat health problems as early as possible, often before symptoms have developed
3. *Tertiary prevention* to avoid complications of and disability from illnesses and injuries or to provide rehabilitative and palliative care

When properly planned and integrated, these approaches help to *(a)* control risks at the source, *(b)* identify new health problems as early as possible, *(c)* provide high-quality treatment and rehabilitation for those who are ill or injured, *(d)* prevent recurrent and new illnesses and injuries, *(e)* ensure appropriate economic compensation, and *(f)* discover new associations between occupational and environmental exposures and adverse health effects.

Identifying and Preventing Hazards

Hazard identification is performed by safety engineers, industrial hygienists, environmental and civil engineers, and others (see Chapters 26 and 27). By interacting with clinicians, these individuals can better identify lapses or gaps in prevention and clinicians can better understand and facilitate implementation of preventive measures.

Implementing Primary Prevention at an Organizational Level

The following paragraphs describe implementing primary prevention measures with substitution, engineering, and development of changes in job design, work practices, and work organization. Ideally, a public health approach to prevention aims to "move upstream" to address the primary sources of a health problem (Fig. 2-4).

Substitution of a Hazardous Process or Broad Approach with a Safer One

There are many ways in which substitution of a process or broad approach can be accomplished, as illustrated by the following four examples:

- Giving medications orally or, if possible, with transdermal patches can reduce needlestick injuries among health care workers.

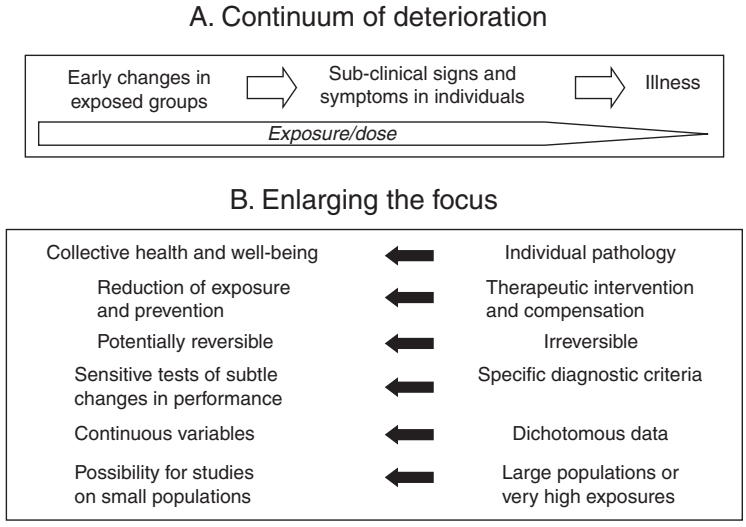


Figure 2-4. Moving upstream: Understanding the relationships of exposure to clinical illness offers the opportunity, in many different ways, to seek the earliest possible evidence of effects to prevent the chain of events and identify reversible changes.

- Implementing policies that promote mass transit—rather than automobile travel—can decrease air pollution and vehicle crashes, thereby reducing illnesses and injuries.
- Using wind power or solar energy can reduce air pollution from fossil fuels and safety risks from nuclear-power plants.
- Mixing chemicals in enclosed structures or bags can reduce dermal or inhalational exposures to hazardous substances.

Substitution of a Hazardous Substance with a Safer One

For example, synthetic vitreous fibers, such as fibrous glass, have been substituted for asbestos. Substitution carries certain risks because substitute materials may not have been adequately tested for adverse health effects and may be hazardous. Long ago, fire protection was enhanced by replacing flammable cleaning solvents with carbon tetrachloride, which was found to be hepatotoxic and, in turn, was replaced by chlorinated hydrocarbons. Now there is concern that use of chlorinated hydrocarbons should be reduced to protect health and the ambient environment. Therefore, substitution with a seemingly safer substance should be considered only a first step. The impact of substitution needs to be monitored on an ongoing basis to determine whether problems are recognized with increased use of the new material. Substitution is embodied in the broader concept of pollution prevention (see Box 2-1).

Installation of Engineering Controls and Devices

Often more feasible than substitution, this approach includes a wide range of options to reduce hazards, such as:

- Installing airbags in automobiles
- Installing ventilation exhaust systems to remove hazardous dusts (Fig. 2-5)
- Using jigs or fixtures that support pieces during machining or other work to reduce static muscle contractions while holding parts or tools
- Applying appropriately designed soundproofing materials to reduce loud noises that cannot be engineered out of a work process or an ambient environment
- Installing tools on overhead balancers to eliminate torque and vibration transmitted to the hand
- Constructing enclosures to isolate hazardous processes
- Installing hoists to eliminate manual lifting of containers or parts

Box 2-1. Avoiding the Transfer of Risk: Cleaner Production and Pollution Prevention

Rafael Moure-Eraso

Environmental sustainability describes the modern concern for social equity between generations by meeting the environmental and occupational needs of the present without compromising the ability of future generations to meet their needs. The European Union and the Environmental Protection Agency (EPA) have embraced cleaner production as a policy framework to achieve environmental sustainability.

Cleaner production (CP) is the continuous application of an integrated, preventive strategy applied to processes, products, and services in pursuit of economic, social, health, safety, and environmental benefits. It has been considered by the United Nations Environmental Programme as the basic strategy for sustainability.

Integral to a cleaner production strategy is pollution prevention (PP)—"the use of materials, processes or practices that reduce or eliminate the creation of pollutants or wastes at the source."[1] Only to the extent that pollution prevention cannot be achieved is it appropriate to implement pollution-control activities, such as treatment, disposal, and remediation. Waste management and control alone will not resolve environmental problems in the long run. A paradigm shift from *pollution control* to *pollution prevention* is necessary. Source reduction has been the tactic of choice to achieve pollution prevention. Since 1990, pollution prevention has evolved to become part of a more comprehensive policy framework of cleaner production. CP/PP provides a coordinated approach to primary prevention, eliminating the possibility of pollution-related health effects and superseding "end-of-pipe" interventions.

Since 1990, CP/PP has evolved in the United States through the voluntary activities of industry, while in the European Union it has evolved through more systematic approaches, going beyond voluntarism and involving specific regulatory regimes. In 1996, the EPA reported that more than 7,200 companies in the United States had established PP programs and predicted that the number would exceed 18,000 by 2000. Results were more modest between 2001 and 2009 due to the George W. Bush administration's opposition to environmental regulation and research. However, the American Chemistry Council (ACC), which formerly was the Manufacturers Chemical Association (MCA), reported in 2009 that 87% of its 142 affiliated companies had verifiable sustainability programs that included environmental management systems (including pollution prevention and cleaner production programs). Both environmental and occupational health are integrated in its sustainability efforts under its Responsible Care program. This program has been expanded to 53 countries, representing 80% of manufactured chemicals, but only 20% of chemical production sites. (See http://www.americanchemistry.com/s_responsiblecare/.) The Responsible Care program is voluntary – companies might choose at any time to start or stop participation. Historically, chemical companies have reduced costs during economic recessions. Programs that are voluntary and not required by regulations are early casualties of difficult economic times.

CP/PP has critical implications for environmental and occupational health. The important conceptual change from control of environmental exposures to their prevention through source reduction and changes in process methods allows the workplace to be seen as a separate source of pollution when undertaking a comprehensive and systematic evaluation of pollution sources. When industries that use chemicals as raw materials begin to use CP/PP to change materials and processes, there are opportunities to consider workplace exposures in choosing substitute materials.

In the past, control of workplace exposures has consisted of end-of-pipe interventions that did not systematically examine root causes. Therefore, it was not recognized that a preferred engineering control, such as local exhaust ventilation, shifted the hazard burden from the workplace to the ambient environment in the form of air pollution or, as a result of filters and other pollution collection media, solid hazardous waste. In a comprehensive CP/PP approach, environmental health and occupational health scientists need to work together to avoid shifting hazards from one medium to another, such as from workplace to community air, or from industrial waste to community water.

The six general PP (source-reduction) tactics that most directly affect environmental and occupational health are as follows: raw material substitution or reduced use, closed-loop recycling, process or equipment modification, improvement of maintenance, reformulation of products, and improvement of housekeeping and training. Some examples of pollution prevention interventions that incorporate concern for reduction or elimination of work exposures are the following:

1. Eliminating use of elemental mercury for switches in new car manufacturing, and substituting with traditional electrical switches, as the EPA has recommended.
2. Substituting water-based solvents for perchloroethylene in industrial textile dry-cleaning operations. This change eliminates exposures to a potential human carcinogen but also leads to improvement in dry-cleaning job organization and reduction in ergonomic risk factors.
3. Substituting the solvent with the lowest concentration of aliphatic organic chemicals for organic solvents in cleaning printing ink from metal surfaces in the offset lithographic industry.
4. Introducing an electrostatically delivered coating to replace a paint with resin-based epoxies in painting of small metal parts. Respiratory and skin hazards from epoxy are eliminated, and paint dispensers are made substantially lighter, avoiding an ergonomic hazard.

Occupational and environmental hygiene should strive to change its focus from secondary to primary prevention

(*Continued*)

Box 2-1. Avoiding the Transfer of Risk: Cleaner Production and Pollution Prevention (Continued)

by addressing workplace problems as comprehensive production problems that have impact inside and outside of the point of production. Workplace problems should not be compartmentalized from environmental problems. CP/PP approaches need to be integrated into occupational and environmental health.

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Further Reading

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This paper evaluated the impact of cleaner production alternatives on occupational health practices in 35 companies in Massachusetts.

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This site describes the sustainability programs (Responsible Care) of the ACC affiliates of the U.S. chemical industry. It also describes similar efforts of the chemical industry in 53 other countries.

Cleaner Production: Pollution Prevention and Occupational Health

Occupational Hygiene Approaches	Primary Prevention	Secondary Prevention
Anticipation	*Hazard surveillance*	———
Identification	*Hazard identification*	*Medical surveillance*
Evaluation	*Exposure assessment*	———
Control	*Exposure prevention*	*Control of generated exposures*
	1. Comprehensive approach	1. End-of-pipe interventions
	2. Source reduction	2. Engineering controls
	a) Materials changes:	a) Enclosure
	• Toxic use reduction	b) Local exhaust
	• Substitution	c) Wet methods
	b) Process changes:	d) General ventilation
	• Physical conditions	3. Administrative controls
	• Machinery	4. Personal protective equipment
	• Operations	
	Work organization	*Early therapeutic intervention*

Figure 2-5. Local exhaust ventilation used to protect a worker from asbestos dust generated in working with clutch plates. (Photograph by Earl Dotter.)

- Carefully maintaining process equipment to reduce or eliminate *(a)* fugitive emissions from processes designed as closed systems, or *(b)* the development of unwanted vibrations as equipment ages
- Using scrubbers or other mechanisms to reduce airborne pollutant emissions
- Maximizing fuel use through co-generation of hot water from the heat exhaust produced as a by-product of generating electricity
- Treating wastewater effluent before discharge

Although installation of engineering controls and devices can involve substantial initial capital expenditure, it often saves money by reducing use of materials, toxic and other material wastes, and costs of disease, injury, and lost productivity. Often, such approaches are not considered or implemented because of lack of awareness that such solutions are available.

Changes in Job Design, Work Practices, and Work Organization

Some changes can be introduced to reduce or eliminate risks in work processes, including job redesign, alternative work practices, and changes in work organization. Job redesign, which often combines engineering and administrative measures, aims to increase job content, make physical work less redundant or repetitive, and improve workers' individual or collective autonomy in decision making. (See Chapters 16 and 27.)

Work practices refer to the ways in which jobs are performed. Work practice alternatives can, through relatively limited changes, lead to important improvements in the workplace. For example, dust exposures can be significantly reduced by using vacuum cleaners to clean dusty surfaces in place of compressed air, and wet mops to clean floors in place of dry sweeping.

These preventive measures are generally more effective than those that rely primarily on behavior change.

Changes in work organization, often closely integrated with individual job redesign, are directed at elimination of undesirable features in the structure of work processes. For example, a change from piece-rate work with incentive wages to hourly-rate work reduces inappropriate physical and mental pressure on workers and musculoskeletal disorders.

The measures described in the next section potentially reduce the adverse effects of workplace and community hazards, without removing the source of the problem.

Primary Prevention at an Individual Level

The following paragraphs address opportunities for individuals for primary prevention of occupational and environmental health problems. (See Chapter 38 on Implementing Programs and Policies for a Healthy Workforce.)

Education and Training

Education and training concerning specific occupational and environmental hazards is an essential aspect of health and safety programs. Providing information about adverse effects of potential exposures conveyed in a user-friendly

manner has several benefits, including empowering workers and raising awareness of people outside the workplace, including children and their parents (Fig. 2-6). Health educators describe the various steps individuals use to understand a specific hazard, assess how they can reduce the hazard, and then choose to take action. Much scientific evidence supports specific approaches to encouraging healthy behaviors, such as using seat belts and bicycle helmets, checking home smoke detectors, and quitting smoking. Different approaches are needed for different activities.

Effective education often requires an understanding of basic principles of risk communication (Chapter 29). The Environmental Protection Agency (EPA) and other agencies and organizations have developed materials on risk communication that are useful for health and safety professionals who communicate with workers or community members about occupational and environmental hazards.[4] Its "seven cardinal rules" for effective risk communication are as follows:

1. Accept and involve members of the general public or workers as legitimate partners.
2. Listen.
3. Be honest, frank, and open.
4. Coordinate and collaborate with other credible sources.
5. Coordinate with the media.
6. Speak clearly and with compassion.
7. Plan carefully and evaluate performance.

Figure 2-6. Bee Safe is a community-based program that aims to raise awareness of parents and children about lead hazards in the home environment. (Photograph by Earl Dotter.)

Understanding how people perceive risk is important. People often dread some outcomes, such as cancer, more than others, such as heart disease. People distinguish between risks they perceive they control and risks controlled by others. Providing to people information that restores some control to them—such as information to follow up on a complaint—is often an important component of education and training.

Workers and community members should always be given full information about hazards to which they may be exposed and means of reducing their risk (Fig. 2-7). Many safety measures are based on changed behavior, which requires education or training. Those who are not aware of hazards will not take health and safety precautions necessary to protect themselves and others. Providing hazard and safety training should not replace other forms of hazard control, such as installing necessary workplace equipment or implementing pollution prevention measures. Effective training that builds on life experiences and empowers people to address and solve problems is a cornerstone for health and safety programs (see Box 2-2).

Personal Protective Equipment

Use of personal protective equipment (PPE), such as respirators, earplugs, gloves, and protective clothing (Figs. 2-8 and 2-9), or safety devices, such as helmets, seat belts, and child restraint systems, will continue to be necessary for *(a)* some workplaces, where PPE is the only available protective measure, and *(b)* most transportation safety.

Figure 2-7. Warning signs, as illustrated in this photograph, should be in multiple languages, if appropriate. (Photograph by Earl Dotter.)

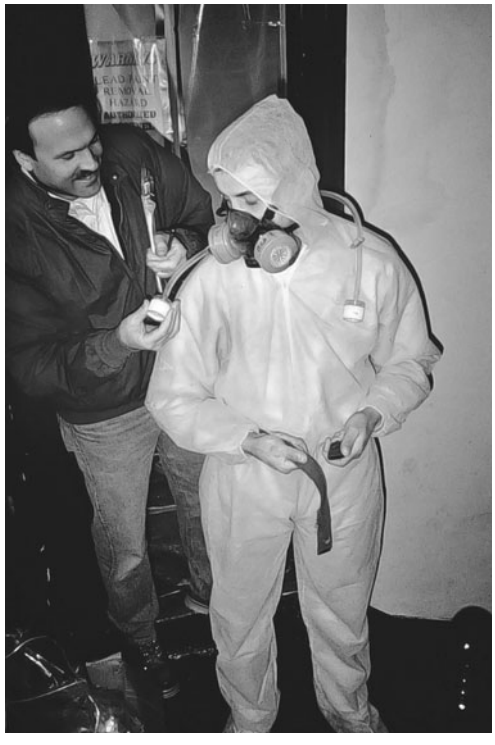


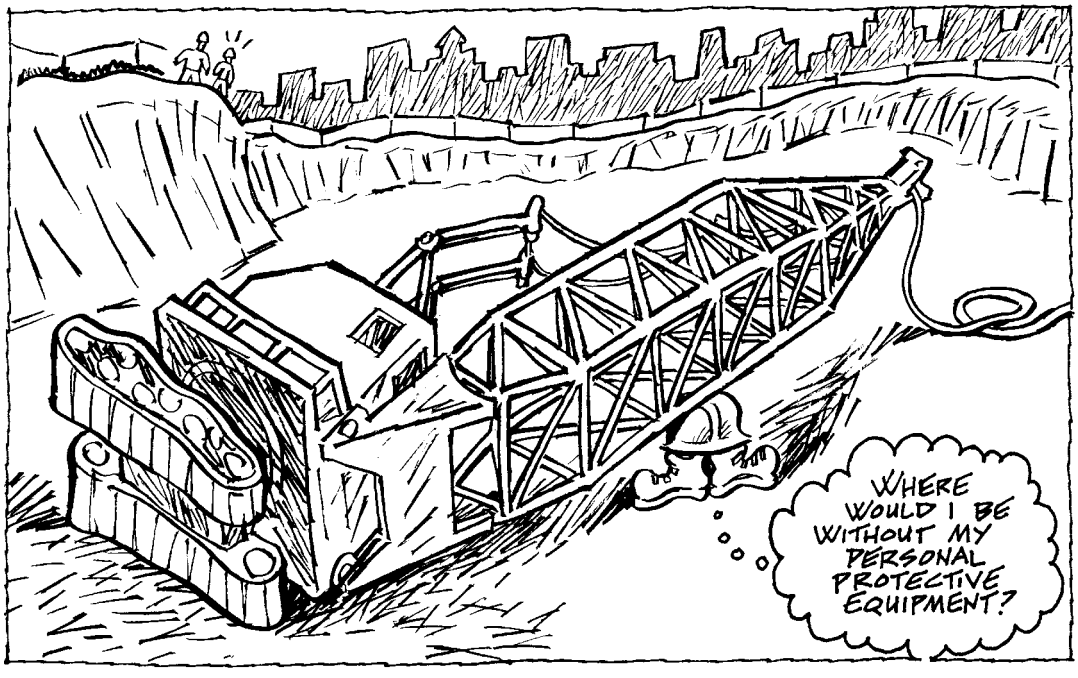
Figure 2-8. A National Institute for Occupational Safety and Health (NIOSH) industrial hygienist prepares to sample a worker's lead exposure during a residential lead-based paint abatement project. (Photograph by Aaron Sussell.)

However, using PPE to control a hazard has important limitations; for example, workers have difficulty wearing PPE because it is cumbersome or may limit visibility or communication with other workers. Since the experimentally determined effectiveness of PPE claimed by its manufacturer may not always be as effective in actual use, its effectiveness should be evaluated in "real-life" situations. OSHA has developed lists of acceptable PPE that can be helpful in proper selection and use of this equipment. OSHA and other agencies have emphasized the need and importance of developing a complete program for PPE—not merely a requirement for using it. Adequate programs include requirements for proper fitting of the equipment (especially respirators), education about proper use, and planning for maintenance, cleaning, and replacement of equipment or parts. The costs of an effective PPE program are significant, making it especially important to recognize that PPE should be used only when there is no alternative method to control a hazard.

Administrative Measures

Organizational measures taken by employers or community planners may offer some protection.

Figure 2-9. Workers with personal protective equipment. (Photograph by Earl Dotter.)



Personal protective equipment is generally not the best approach to prevention. (Drawing by Nick Thorkelson.)

For example, in communities, zoning ordinances that restrict certain types of industry in residential areas or set hours for noise restriction offer important protections. As another example, occupational exposure can be reduced somewhat by implementing work schedules so that workers spend carefully limited amounts of time in areas with potential hazardous exposure. Such measures require accurate environmental monitoring data to design appropriate schedules.

Box 2-2. Effectively Educating Workers and Communities

Margaret M. Quinn and Nancy Lessin

A prerequisite to effective health and safety programs is education. The most effective approach to teaching health and safety acknowledges that workers and community members are the ones most familiar with their jobs, homes, and communities. Workers can identify hazards—both apparent and hidden—that may be associated with their work. These include *(a)* physical hazards, such as noise, musculoskeletal strain, or the use of toxic chemicals; *(b)* psychosocial hazards, such as lack of social support, harassment, or discrimination; *(c)* work-organization hazards, such as understaffing, extended hours of work, and production pressures; and *(d)* social hazards, such as living in unsafe neighborhoods or near hazardous waste sites. Community members understand the specific cultural and political characteristics that will affect the success of a program.

Involvement of community members and workers in prioritizing educational needs and in designing and presenting training are major determinants of meaningful and useful programs. They should also be included in developing and implementing solutions to health and safety problems. Education regarding solutions should include a discussion of the traditional hierarchy of hazard controls that emphasizes hazard elimination, and it should not be limited to training on the use of personal protective equipment or actions that individuals can take to minimize the impact of hazards. In addition to worker and community involvement with needs assessment and program design, the following tasks serve as guidelines for successful educational programs:

1. Develop programs in trainees' literal and technical language. Understand the social context and psychosocial factors of a workplace or community that may affect a person's ability to participate in an educational program or to implement personal safety measures in response to potential hazards.
2. Define specific, clearly stated goals for each session based on a needs assessment that has involved representatives of workers or community residents to be trained. Begin each program with a concise overview. Reinforce the key issues that come up during the session.
3. Develop an evaluation mechanism that can easily be adapted to each program. Design the evaluation process to judge the effectiveness of the educational program in attaining the goals that have been set by the trainer and trainees.
4. Use participatory, popular education approaches, which draw on the experience that workers and community members have had, instead of a traditional lecture approach that imparts instructor-defined problems and solutions.

Participatory, popular-education approaches utilize adult learner–centered education methods; are designed to foster maximum participation and interaction; and empower participants to devise effective strategies for improving workplace and environmental health and safety. They constitute an approach to labor and community-based education that is based on the understanding that adults bring an enormous amount of experience to the classroom and that this experience should be used in the training program. In addition, adults learn more effectively by doing rather than listening passively. Learners' experiences are incorporated into the course material and are used to expand their grasp of new concepts and skills. Basing new knowledge on prior practical experience helps the learner solve problems and develop safe solutions to unforeseen hazards. Combining instructors' specialized knowledge and participants' direct experience leads to effective, long-lasting solutions to health and safety problems.

Participatory learning generally requires more trainer–trainee interaction than lecture-style presentations. Groups should be limited to approximately 20 participants, and these may be subdivided into groups of three to six for small-group exercises. Participatory teaching methods include a variety of techniques, including the following:

1. *Speakouts* (large-group discussions): Participants share their experiences in relation to a particular hazard or situation.
2. *Brainstorming sessions*: The instructor provides a particular question or problem; the participants call out their ideas, which are recorded on a flipchart so that patterns are identified and a collective work is created. In this activity, the trainer elicits information from the participants, rather than presenting it in a didactic manner.
3. *Buzz groups* (small-group discussions or exercises): Each group of three to six participants discusses a particular problem, situation, or question, and then records the answers or views of the group.
4. *Case studies* (small-group exercises): Participants apply new knowledge and skills in the exploration of solutions to a particular problem or situation.
5. *Discovery exercises*: Participants go back into the workplace or community to obtain certain items, such as OSHA-300 logs for their workplaces or Toxic Release Inventory records for their communities, or perform activities, such as interviewing co-workers regarding a particular hazard. This information is then brought back into the classroom for discussion.
6. *Hands-on training*: The participants practice skills, such as testing respirator fit, simulating asbestos removal or hazardous waste clean-up, using OSHA-300 logs to identify hazards in need of correction, or handling, and learning the uses and limitations of, industrial-hygiene or air-pollution monitoring equipment.
7. *Report-back sessions*: After buzz groups, the class reconvenes as a larger group, and a spokesperson for each buzz group reports the group's answers or views. Similarities and differences among groups are noted, and patterns may be discovered.
8. *Hazard mapping:* Participants create a map of their workplace or neighborhood, locating hazards and indicating their type, severity, and number of people affected. The hazards include psychosocial

(*Continued*)

Box 2-2. Effectively Educating Workers and Communities (Continued)

and work- organization hazards as well as physical and chemical hazards. If participants need technical assistance, they are referred to information by the course instructor and the participants actively seek it out. The map becomes a way for participants to visually integrate their existing knowledge with new knowledge. Participants then use the map to prioritize actions and resources for change. As improvements are made, the map can be used to show solutions and accomplishments. This technique can be especially useful for labor-management health and safety committees and community-based environmental organizations.

Participatory, popular-education approaches are well-established educational methods practiced in labor education programs, schools of education, national and international community education centers, labor unions, and Committees for Occupational Safety and Health (COSH) groups. These groups have demonstrated that it is possible to use participatory methods, even for educational programs that require conveyance of specific, technical knowledge. For example, the OSHA Hazard Communication Standard has worker training requirements for use of material safety data sheets (MSDSs), forms that contain brief information regarding chemical and physical hazards, health effects, proper handling, storage, and personal protection for a particular substance. Training on MSDSs should cover how to obtain the MSDSs, how to interpret them, and their uses and limitations, and should give the participants practice in each of these areas. Rather than presenting an MSDS in a lecture-style format, the information can be taught more effectively with a participatory exercise, such as the exercise that follows:

In the first part of the exercise, workers go back into their work areas, find a labeled chemical container, and seek an MSDS for that substance. This requires workers to become familiar with where MSDSs are located in their particular workplace and the process required to find one. It also serves to identify problems in the system that can be corrected, such as unlabeled containers, missing MSDSs, or locked offices to which no one on a given work shift has a key. In the second part of the exercise, the class is divided into small groups, which review sample MSDSs and collectively answer questions such as the following: "Is the substance flammable?" "What are the health effects associated with it?" "Does it require wearing of gloves?" and "What ventilation is required?" During the report-back session, the instructor asks for the answers from all of the groups and reviews how to read and interpret MSDSs in general. In the final part of the exercise, participants look up the chemicals covered in the sample MSDSs in other sources, such as the *NIOSH Pocket Guide to Chemical Hazards* (available at: http://www.cdc.gov/niosh/npg/). In some situations, more hazards—especially health hazards—are discovered when other sources are consulted. In this way, students learn about the uses and limitations of MSDSs, and practice using additional sources.

Participatory, popular-education approaches not only make learning active, but they also value workers' and community residents' knowledge and experience. And they broaden the objectives of curricula to give workers and community residents the skills, support, context, framework, and strategic planning practice necessary for them to identify hazards and take action to improve health, safety, and environmental conditions.

Further Reading and Resources

The Labor Safety and Health Training Project at the National Labor College

A joint project of the National Labor College and the AFL-CIO, its goal is to increase involvement of workers and unions in improving workplace safety and health. It has developed extensive training materials and facilitator guides for educating workers and union members about workplace safety and health. It provides workshops on workplace safety and health for unions and labor-related organizations and offers week-long train-the-trainer courses. For more information, visit http://www.nlc.edu/educationalPrograms/laborSafetyHealth.html or contact Sharon Simon at the National Labor College (301-431-5414, or ssimon@nlc.edu).

The Highlander Research and Education Center, New Market, TN

A leader in adult, learner-centered, participatory education and a founding member of the North American Association of Popular and Adult Educators (NAAPAE), it promotes the understanding and practice of popular-education approaches among community groups and universities. Its library has an extensive collection of case studies, oral histories, and model curricula from adult education programs in the United States and elsewhere on worker literacy, community research, radio education, education for immigrants and indigenous peoples, and democratic facilitation. More information is available at: http://www.highlandercenter.org/r-popular-ed.asp.

Massachusetts Coalition for Occupational Safety and Health (MassCOSH)

MassCOSH brings together workers, unions, community groups, and health, safety, and environmental activists for training related to safe, secure jobs and healthy communities. Its Immigrant Worker Center uses community-based participatory methods to train immigrant workers More information is available at: http://tools.niehs.nih.gov/wetp/.

National Institute for Environmental Health Sciences maintains the National Clearinghouse for Worker Safety and Health Training at http://tools.niehs.nih.gov/wetp/

Its curricula catalog contains direct access to training curricula produced by awardees funded by the NIEHS Worker Education and Training Program (WETP) to help employers meet OSHA requirements under 29 CFR 1910.120, Hazardous Waste Operations & Emergency Response (HAZWOPER). It supports the development of model worker safety and health training development and delivery in hazardous waste worker training, minority worker training, brownfields worker training, Department of Energy nuclear weapons cleanup training, and hazmat disaster preparedness training. These model programs use innovative methods for training difficult-to-reach populations by addressing issues such as literacy, appropriate adult education techniques, training quality improvement, and other topics not addressed directly by the private sector.

Tools of the Trade—A Health and Safety Handbook for Action, Labor Occupational Health Program, University of California Berkeley.

Training for unions and community groups to assist them in integrating health and safety into every aspect of their work. More information is available at: http://www.lohp.org/Publications/publications.html

(*Continued*)

Box 2-2. Effectively Educating Workers and Communities (Continued)

Risk Mapping—A Group Method for Improving Workplace Health and Safety, Labor Occupational Safety and Health Program, University of California Los Angeles.
Describes how workers can develop risk maps of their worksite and then how these can be used to make health and safety improvements. Included are examples where risk mapping has been used, lesson plans, and other resources. More information is available at: http://www.losh.ucla.edu/resources-publications/index.htm.

Arnold R, Burke B, James C, et al. Educating for a change. Toronto, Ontario: Doris Marshall Institute for Education and Action/Between the Lines, 1991.
This classic text, which covers the theory, principles, and practice of popular education, is written for educators who are seeking to enhance their skills as facilitators of this type of educational philosophy and methods. It is not specific to any topic area or subject matter; rather, it relates to all types of education that support social change.

National Institute for Occupational Safety and Health. A model for research on training effectiveness. DHHS (NIOSH) Publication No. 99-142, October 1999.
Describes a systematic approach to evaluating all types of training. Available as 99-142.pdf at http://www.cdc.gov/niosh/99-142.html.

Since this approach may distribute hazardous exposure to more workers, it should not replace other preventive measures, such as engineering approaches, that may be safer and more effective. Another administrative measure is use of preplacement examinations to avoid assigning new workers to jobs in which their personal risk factors place them at higher risk for specific diseases or injuries. In the United States, the requirements of the Americans with Disabilities Act place a special responsibility on clinicians performing preplacement examinations.

Secondary Prevention

Screening and Surveillance

Screening and surveillance can identify the need for control measures to prevent further hazardous exposures. Both screening and surveillance are directed toward identification of health events or documentation of early evidence for adverse health effects that have already occurred. Screening is a clinical activity that seeks to identify disorders in an asymptomatic individual at a time when intervention can reduce the probability the individual will develop an adverse health outcome. Specific guidelines for appropriate screening include considerations of risks and benefits to the individual. Screening may identify individuals who need treatment or preventive intervention.

Surveillance is designed to obtain, analyze, and disseminate information on disorders that have already occurred (Chapter 3). Surveillance implies watching out or watching over, and it may consist of *(a)* watching out for single events (sentinel health events) that signal a breakdown in prevention; *(b)* reviewing grouped or aggregate data for subtle trends that may be significant for an entire population, but not necessarily meaningful for a specific individual—such as increases in liver enzymes that do not exceed population norms; or *(c)* reviewing grouped or aggregate data to discern trends in the occurrence of illnesses, injuries, or deaths. Surveillance can lead to primary prevention measures by identifying inadequate control measures and allowing them to be corrected so that others can be protected.

By recognizing potential or existing disease or injury, health professionals can initiate activities leading to one or more of these methods of prevention. They can play an active role in education by informing people about potentially hazardous exposures and ways of minimizing them. For example, they can help identify important residential exposures, such as radon and lead in the home environment, and they can facilitate appropriate measures to address these problems. They can advise workers on how to better protect themselves at work, such as by advocating for improved ventilation systems and, if necessary, using respirators or other PPE. They can also screen individuals and facilitate the screening of others who may be at high risk for specific diseases. Consultation with specialists in various disciplines may be necessary to facilitate these activities.

Prevention at the Systems Level

Preventive approaches to occupational and environmental health problems can occur at the level of the individual, workplace or local

environment, broader community, or entire society—all of which can potentially interact with one another. In the workplace, these approaches rely on management commitment, employee participation, hazard identification and control, training, medical surveillance, and program evaluation. Similar approaches have been developed to reduce environmental hazards. Community-based environmental organizations play important roles in advocating for better regulation and improved urban planning. The EPA's Pollution Prevention Initiative identifies alternatives that reduce the production of pollutants. The International Organization for Standardization management standard (ISO 14000) helps to reduce environmental pollution from industry. In order to be effective, these programs require external verification and constant engagement of vigilant civic groups and non-governmental organizations that empower community members. In workplace settings, labor-management safety committees can help implement the steps required to ensure that programs effectively promote safety and health (Fig. 2-10 and Box 2-3). It often takes a team effort of professionals in occupational and environmental health and safety, and others in medical care and public health, working together with workers, employers, community members, government officials, and others to prevent or control complex problems. Each member must accept responsibility to fully participate, communicate effectively, and engage others.

The following two examples of major improvements show how communication, focus, and persistence among individuals and policy makers can reduce exposure and improve lives—although in both of these examples much work remains to be done.

Lead Exposure

Lead has been widely used for thousands of years. Yet it continues to cause adverse health effects in some workers, young children exposed to lead-based paint, and others. From the 1920s to the 1970s, organic lead was used as an antiknock agent in gasoline, causing widespread population exposure. By the mid-1970s, the geometric mean BLL for the U.S. population was about 16 μg/dL. The removal of lead from

Figure 2-10. Joint labor-management health and safety committees help prevent occupational injuries and illnesses. This photograph depicts a worker, a safety specialist, and a management representative discussing a workplace problem. (Photograph by Earl Dotter.)

Box 2-3. Labor-Management Safety Committees

The benefits that accrue from seeking the participation of labor unions and workers in the development and implementation of occupational health and safety programs and research can be substantial. As a consequence of their experience and intimate knowledge of the actual work processes, workers and their unions often can add significantly to the understanding of a health or safety problem and determine the best approach to prevention of risks. Their participation also aids in understanding and explaining the nature and importance of programs and research efforts and in interpreting the impact and meaning of such work to individual workers.

One effective means for including workers and their labor unions in the development and improvement of approaches to prevention is joint labor-management health and safety committees in the workplace. These committees consist of representatives of workers and managers. They meet periodically to systematically review workplace health and safety hazards and their control and to respond to specific complaints concerning workplace health and safety. For these committees to function effectively, labor representatives must be truly representative of workers and not simply appointed by management.

Joint labor-management health and safety committees have been legally authorized and are more generally active in some countries, such as Canada. In the United States, they are less common and usually are established through collectively bargained agreements. Some legislative proposals to reform OSHA would require operation of health and safety committees in many more workplaces than at present.

Studies in Canada, where joint health and safety committees have been mandated, suggest that this particular form of involvement can be unusually effective. Reduction in work injuries and resolution of health and safety problems without the need for governmental intervention has been documented. Effective committees tend to have co-chairs and equal representation, readily available training and information, and well-established procedures. An important feature of successful committees is sufficient authority for action, either as a committee or on the part of the management representatives.

Typically, labor-management health and safety committees meet on a monthly basis for 1 to 2 hours. They review, evaluate, and respond to worker and manager complaints and concerns about working conditions and workplace hazards. They periodically walk through the workplace to observe and assess working conditions and possible health and safety hazards. In addition, they systematically evaluate work practices and procedures and materials used in the workplace in regard to their impacts on workplace health and safety. Labor-management health and safety committees are most effective when seen as one component of a more general prevention program that also relies on the development and enforcement of government regulations.

gasoline in the late 1970s was initiated by the introduction into automobile engines of catalytic converters, which cannot function effectively in the presence of lead. Research by scientists, advocacy by non-governmental organizations and professional associations, and regulatory measures by the EPA and other agencies facilitated the removal of lead from gasoline. Now the geometric mean BLL of the U.S. population is about 2 μg/dL. Additional measures to ban lead as a pigment in paint, in water-carrying pipes, and in solders used for canned foods have also had a positive impact, although lead exposure continues, mainly from lead paint in older homes. Only about 2% of U.S. children age 1 to 5 have BLLs above 10 μg/dL, the CDC level of concern.[4] (See Chapters 11, 19, and 20.)

Environmental Tobacco Smoke

Since the U.S. Surgeon General's seminal report in 1964 on the hazards of cigarette smoking, the tobacco industry has fought back, such as by attacking the science that demonstrated the adverse health effects of environmental tobacco smoke on nonsmokers. Public awareness has increased and the percentage of U.S. smokers has decreased due to smoking cessation programs, one-on-one counseling by primary care physicians, educational and advocacy projects by non-governmental organizations, legislation restricting smoking, and individual and state lawsuits. As a result, levels of cotinine (a biomarker for environmental tobacco smoke) among nonsmokers during the 1990s decreased 58% for children, 55% for adolescents, and 78% for adults. (See Box 7-1.)

Roles of the Clinician

Once a clinician has identified a probable case of occupational or environmental disease or injury, it is crucial to take preventive action while also providing appropriate treatment and

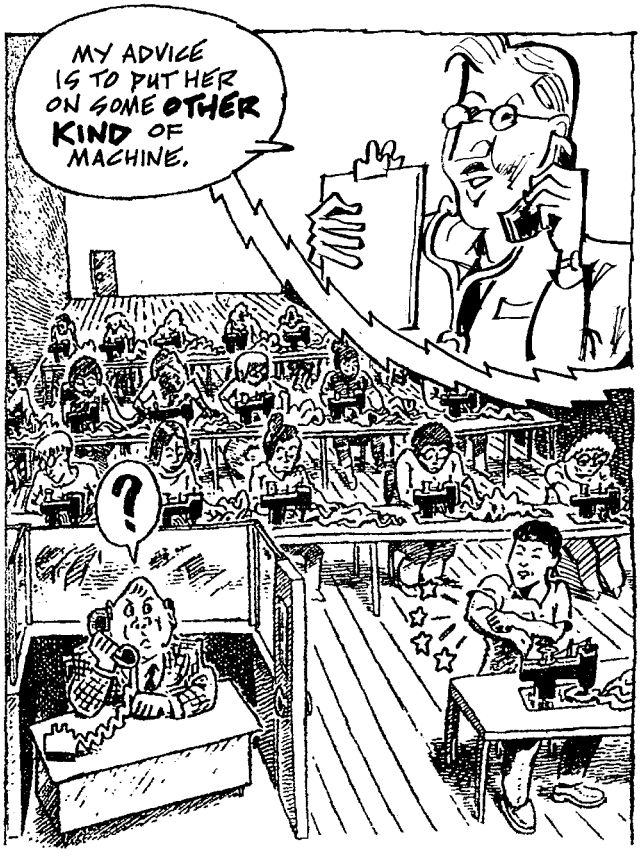
rehabilitation services. Failure to consider the prevention opportunities along with the necessary therapeutic measures may lead to recurrence or worsening of the disease or injury in the affected person and the continuation or new occurrence of similar cases among other workers or community members. A clinician has at least the following five opportunities for preventive action after identifying a case of occupational or environmental disease or injury:

- Advise the patient.
- Contact an appropriate labor or environmental organization
- With the consent of the patient, contact the responsible party, such as the patient's employer or landlord.
- Inform the appropriate governmental regulatory agency.
- Contact an appropriate research or expert group.

Often some combination of these approaches is undertaken.

Advise the Patient

The clinician should always advise the patient concerning the nature and prognosis of the condition; the possibility that there may be appropriate engineering controls to remove the hazard; the need, even if only temporarily, for PPE, or, in extreme circumstances, the necessity to change job or place of residence. The clinician should alert the patient to the need to file a workers' compensation report to protect the worker's rights to income replacement and both medical and rehabilitation services (Chapter 31). This report also may lead the employer to report the case and may lead its insurance company to provide consultative services to the employer to assess the problem and recommend appropriate control measures.



Advice to employees and employers should be practical. (Drawing by Nick Thorkelson.)

If a health problem results in a contested workers' compensation claim or the need for making a report or complaint with an appropriate government agency, the clinician may be asked to provide advice to the patient concerning legal remedies (see later discussion). Patients' options may be limited: Workers may not wish to file a workers' compensation claim or make a complaint to a government agency for fear of job loss or other punitive action. Patients may find it impossible to change residence for themselves and their families.

However, it is essential to inform a patient of potential hazards. It is not appropriate to withhold this information because of the possibility of upsetting the patient. A clinician cannot assume that even a large and relatively sophisticated employer has adequately educated its workers about workplace hazards. Once a patient is informed of the work-relatedness of a disease in writing, this may start the time clock on notification procedures and statutes of limitations for workers' compensation (Chapter 31).

Contact an Appropriate Labor or Environmental Organization

If it is agreeable to the affected patient, the clinician should inform the appropriate labor or environmental organization of the health hazards suspected to exist. The provision of this information may help to alert others to a potential hazard, facilitate investigation of the problem, identify additional similar cases, and eventually facilitate implementation of any necessary control measures. (Keep in mind, however, that only a small percentage of workers in the private sector in the United States belong to a labor union and that relatively few community members are represented by community-based environmental organizations.)

With the Consent of the Patient, Contact the Responsible Party, Such as the Patient's Employer or Landlord

The clinician, only with the patient's express consent, may choose to report the problem to the responsible party, such as the patient's employer or landlord. This can be effective in initiating preventive action. Many employers do not have the staff to deal with reported problems adequately, but they can obtain assistance from insurance carriers, government agencies, academic institutions, or private firms. In addition to triggering workplace-based prevention activity, discussions with the employer may lead to obtaining useful information concerning exposures and the possibility of similar cases among other workers. Depending on the circumstance, it can be particularly helpful for the clinician to arrange with an employer to visit a patient's work area. This presents the opportunity to observe the possibly hazardous environment firsthand and to establish the necessary rapport with managers to involve them in prevention. Environmental situations are often more complex; it may be unclear which responsible party should be contacted. In these situations, it is often most prudent to contact the state or local health department (see next section).

Although the law prohibits employers from firing workers for making complaints to OSHA, it does not prohibit them from firing workers who have a potentially work-related diagnosis. In the United States, only the OSHA lead and cotton dust standards mandate removal of workers from jobs that are making them sick. In addition, the OSHA lead standard (but not the cotton dust standard) provides that, during the period of removal, employers much maintain removed workers' earnings, seniority, and other employment rights and benefits as if the workers had not been removed. (See Chapters 11, 19, and 20 for more information on lead.)

Inform the Appropriate Governmental Regulatory Agency

If a case of occupational or environmental disease or injury appears to be serious or may be affecting others in the same workplace, company, industry, or community, it is wise for the affected person or the clinician to consider filing a complaint with the appropriate governmental regulatory agency. (See discussion in the next section and Boxes 2-4 through 2-6.) The clinician should always inform the patient before notifying a governmental agency. Although regulations of OSHA and the Mine Safety and Health Administration (MSHA) protect U.S. workers who file health and safety complaints against resultant discrimination by the employer (loss of job, earnings, or benefits), this protection is difficult to enforce, and workers' fears are

not unfounded. Clinicians should familiarize themselves with pertinent laws and regulations. For example, if the worker does not file an "11(c)" (anti-discrimination) complaint within 30 days of a discriminatory act, the worker's rights are lost. In the United States, health professionals and workers (or their union, if one exists) have the right, guaranteed by the Freedom of Information Act, to obtain the results of an OSHA inspection.

Contact An Appropriate Research or Expert Group

Occasionally, the health professional who is reporting a work-related or environmentally-mediated medical problem may undertake or assist in a research investigation of this problem. No matter who conducts the research, investigation of the workplace or the environment and the identification and analysis of additional cases often lead to new information. Publication of epidemiologic studies or case reports alerts others to newly discovered hazards and ways of controlling them. The health professional may also assist with research to evaluate the effectiveness of preventive approaches, such as the impact of OSHA or EPA regulations.

AVAILABLE RESOURCES

Identifying and using the broad range of available occupational, environmental, and public health resources enables health professionals to offer their patients and others a wide range of services to help recognize and prevent occupational and environmental disease and injury. Resources are available from federal, state, and local government agencies; international organizations; academic centers; professional organizations; and labor, environmental, and community organizations.

There is much information on the Web sites of the following agencies and organizations, but it is helpful to know the focus of each and their relative strengths and limitations. The major U.S. agencies addressing occupational and environmental health and safety are located in several different parts of the federal government. The Department of Labor includes OSHA, MSHA, and other agencies that administer programs on job training, child labor, wages and hours of workers, and obtaining, analyzing, and disseminating national data on work and on occupational injuries and illnesses. The Department of Health and Human Services includes the National Institute for Occupational Safety and Health (NIOSH); other agencies within the Centers for Disease Control and Prevention (CDC); the National Institutes of Health (NIH), within which the National Institute of Environmental Health Sciences (NIEHS) is located; the Agency for Toxic Substances and Disease Registry (ATSDR); and other agencies that address public health, food and drug safety, and research on health care quality. Other federal agencies that address occupational and environmental health include the Environmental Protection Agency (EPA) and the departments of Agriculture, Defense, Energy, Housing and Urban Development, the Interior, Transportation, and Veterans Affairs.

The roles of governmental agencies and international organizations are briefly described in the next section. Chapter 30 describes government regulation in the United States and the European Union. The Appendix at the back of this book provides a list of non-governmental organizations. Chapter 32 provides a list of the 10 largest international labor federations and labor unions. Chapter 33 provides a list of environmental non-governmental organizations.

U.S. Government Agencies and Programs in Occupational Health and Safety

The U.S. Department of Labor

Several agencies and other units within the Department, which are described below, profoundly affect work and occupational health and safety.

The Occupational Safety and Health Administration (OSHA) establishes and enforces standards for hazardous exposures in the workplace and undertakes inspections, both routinely and in response to complaints from workers, physicians, and others. In about half of the states in the United States, the program is implemented directly by OSHA; in the other states, a state agency—often the state department of labor—implements the program. Both OSHA and the state agency may investigate a workplace in response to a complaint. Most state agencies make recommendations to improve the situation, but

only those states with OSHA-delegated authority can order changes to improve health and safety in the workplace and impose fines if these changes are not made. Box 2-4 describes how to use OSHA. Additional information is available at http://www.osha.gov.

The Mine Safety and Health Administration (MSHA) develop and enforces regulations to protect the health and safety of miners in the United States. Box 2-5 describes MSHA in detail. Additional information is available at http://www.msha.gov.

Box 2-4. How to Use the Occupational Safety and Health Administration (OSHA)

Michael Silverstein

This box describes three ways to utilize the services of OSHA.

Getting a Workplace Inspected by OSHA

The Occupational Safety and Health Act (OSH Act) of 1970 states that "any employees or representative of employees who believe that a violation of a safety or health standard exists that threatens physical harm, or that an imminent danger exists, may request an inspection…" If, but only if, OSHA "determines there are reasonable grounds to believe that such violation or danger exists," an inspector will be sent to perform an on-site inspection. Although the decision about whether to file a complaint rests with an individual worker, health care providers can help ensure that OSHA inspections are performed appropriately.

One can file an official complaint in writing, such as by filling out OSHA's online complaint form at http://www.osha.gov/pls/osha7/ecomplaintform.html. Handwritten or typed complaints can also be submitted to any OSHA office. A list of the OSHA offices can be found at http://www.osha.gov/html/RAmap.html.

In nearly half of the states, a state agency—not federal OSHA—is responsible for workplace safety and health inspections. One can find out if one's state has a state agency at http://www.osha.gov/dcsp/osp/states.html.

Complaints can also be made anonymously or by telephone, e-mail, or fax. OSHA will review these but will consider them "informal." It is less certain that OSHA will perform a worksite inspection after an informal complaint than if the complaint is written and signed. OSHA is required to keep names confidential upon request.

Patients who do not have union representatives and do not want to file complaints directly may, in writing, designate health care providers or others as their representatives. The OSH Act directs OSHA to investigate complaints from employees or their representatives. OSHA will review all other complaints but will consider them "referrals," with less certainty that it will perform a worksite inspection.

One should provide as much specific information about the issues of concern as possible. The OSH Act requires unannounced, on-site inspections only when there are reasonable grounds to believe that there is an imminent danger at a workplace or that a violation of an OSHA regulation threatens physical harm.

Call the OSHA area office and talk to an inspector or supervisor. While this is not required, it will increase the likelihood that OSHA will respond quickly and seriously. If workers have a U.S. senator, Congressional representative, or state legislator call on their behalf, their complaints are more likely to be seriously considered.

Getting an On-site Consultation

Free safety and health consultation services are available to employers in every state. OSHA consultation services are funded primarily by federal OSHA, but delivered by the 50 state governments, most commonly through a state labor department or university. The list of consultation programs can be found at http://www.osha.gov/dcsp/smallbusiness/consult_directory.html. OSHA will not conduct a consultation visit without an invitation from the employer. If one feels that an employer might benefit from an OSHA consultation, one can try to convince the employer to seek assistance. One can contact the state consultation program office and suggest that a consultant call the employer. The consultation program will contact employers and offer its services, but a consultant will only enter the workplace if the employer responds positively and invites the consultant to enter.

Consultants provide advice about complying with OSHA regulations and otherwise controlling workplace hazards, but they do not enforce the OSH Act and do not issue citations and penalties. If they find violations of OSHA regulations, they inform employers and advise them about how to correct the problems. They will not pass this information on to OSHA inspectors, except in very special circumstances.

Protecting Against Discrimination

The OSH Act prohibits an employer from discriminating against any employee for having filed a complaint or exercising any rights afforded by the Act. Some examples of discrimination are firing, demotion, transfer, layoff, losing opportunity for overtime or promotion, assignment to an undesirable shift, denial of benefits (such as sick leave), blacklisting with other employers, and reducing pay or hours. Employees believing they have been discharged or otherwise discriminated against may file complaints with OSHA or state counterpart agencies within 30 days of alleged discrimination. Complaints can be telephoned, faxed, or mailed. OSHA conducts an interview with each complainant to determine the need for an investigation. OSHA or the state agency must then complete an investigation within 90 days of the complaint. If evidence supports a worker's claim, OSHA will ask the employer to restore the worker's job, earnings, and benefits. If the employer objects, OSHA may take the employer to court to seek relief for the worker. Instructions for filing a discrimination claim can be found at http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=11341.

Box 2-5. The Mine Safety and Health Administration (MSHA): Intensive Intervention in a Dangerous Industry

James L. Weeks

The Mine Safety and Health Administration (MSHA) in the Department of Labor writes and enforces regulations to protect the health and safety of the 200,000 miners in the United States. These miners work in underground and surface mines that produce coal, metal ore, and other nonmetal commodities (such as salt and trona) and in sand, stone, and gravel quarries. Mining is one of the most dangerous industries in the United States and worldwide, with high rates of injuries, coal workers' pneumoconiosis (CWP), silicosis, lung cancer, and noise-induced hearing loss.

MSHA represents intensive intervention in a dangerous industry It has demonstrated a concerted and multifaceted effort at controlling occupational hazards can succeed at reducing rates of injury, illness, and death. Its key components have included active enforcement, sufficient resources, surveillance, exposure monitoring, worker training, epidemiologic research, and engineering research and development—all of which have been supported by regulatory authority.

The Federal Coal Mine Health and Safety Act of 1969 was passed after a widespread miners strike for compensation for CWP and a disastrous mine explosion. It created agencies and programs for epidemiologic research, development of safe mining practices, and compensation for miners totally disabled by pneumoconiosis (the federal black lung program). This program established a series of presumptions, based on the miner's clinical status and work history, to facilitate decisions about eligibility when etiology is ambiguous. Since 1981, claims have been paid by operators who last employed miners or, when operators cannot be found, by a disability trust fund to which operators contribute.

The 1969 act was amended in 1977 by the Mine Safety and Health Act (Mine Act), which placed MSHA in the Department of Labor, extended authority to all mines and quarries, and required that miners receive training in health and safety when first hired and annually thereafter.

For the purpose of establishing regulations on exposure to hazardous substances, the legal and scientific requirements of MSHA and OSHA are essentially the same. But MSHA is significantly different from OSHA in its enforcement capabilities. Under MSHA, underground mines must be annually inspected four times and surface mines, twice; most OSHA inspections are discretionary. Under MSHA, inspectors are authorized to close all or part of a mine if there is imminent danger; OSHA inspectors must get a court order to close all or part of a workplace.

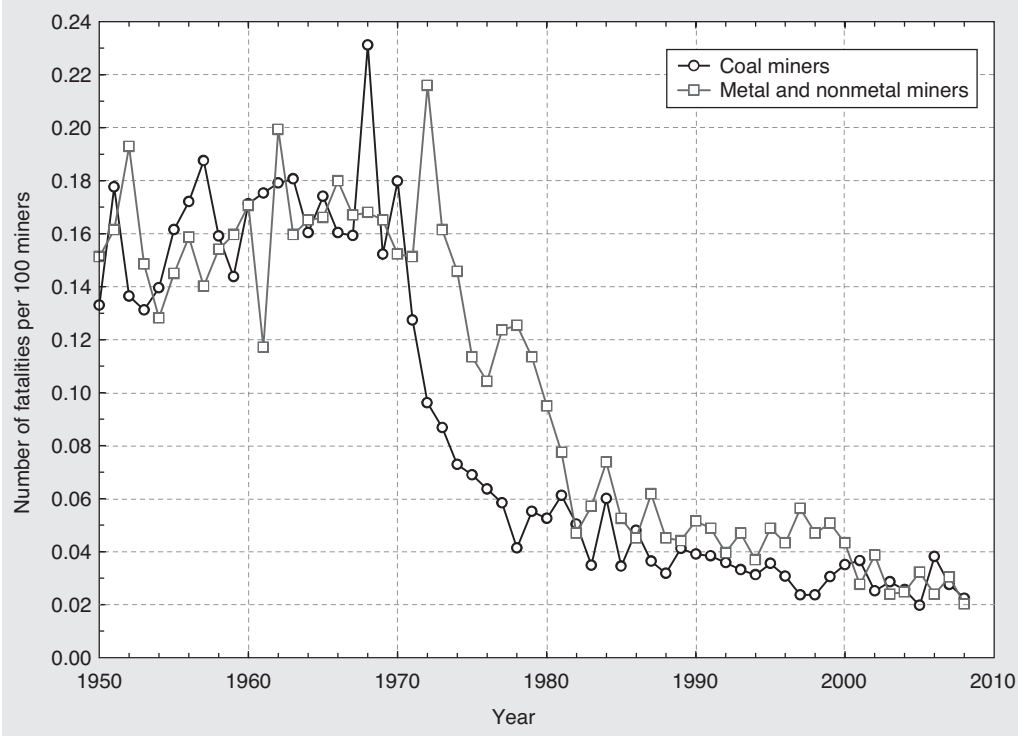


Figure 2-11. Rate of fatal injuries for coal and metal and nonmetal miners, United States, 1950–2008.

(*Continued*)

Box 2-5. The Mine Safety and Health Administration (MSHA): Intensive Intervention in a Dangerous Industry (Continued)

All mines are covered under MSHA; employers with 10 or fewer employees are exempt from OSHA general-schedule inspections. Mine operators must submit a mine plan and have it approved before they can produce; employers under OSHA's jurisdiction must obtain a permit only for certain confined-space conditions. MSHA has jurisdiction over less than 250,000 workers; OSHA, over about 100 million. Both agencies have about the same number of inspectors (including those in state plans), so the number of inspectors per worker under MSHA is approximately 400 times that under OSHA.

Mine-specific data on the number and rates of injuries, hours worked, and (coal) production are reported by mine operators to MSHA every quarter, and some of these data are available on the Internet. Surveillance data on exposure to dust, crystalline silica, other hazardous materials, and noise are also available from MSHA. In general, under OSHA, *estimates* of injury rates are available, by industrial category, based on an annual survey of a *sample* of employers; employers must post injury data annually, but they are not required to report it to OSHA. Neither MSHA nor OSHA guarantee the accuracy and reliability of surveillance data.

What has this regulatory intervention into the mining industry achieved? Before the passage of the 1969 act, the death rate of U.S. miners was about 0.25 per 100 workers per year—four times that of miners in Western European coal-mining countries. During the first 10 years after the Act was implemented, it declined to a level about the same as that in European mines. Since then, it has declined further to a death rate of approximately 0.03 per 100 workers (Fig. 2-11). Even so, it is the highest death rate of any major industry in the United States.

Regulation has also significantly reduced miners' exposure to respirable dust and the prevalence of CWP (Fig. 2-12). Respirable coal mine dust was measured at 6 to 8 mg/m^3 before the 1969 act, but, for the same job, declined to less than 3 mg/m^3 within 6 months. For continuous mining operators, the level is now regularly below 1 mg/m^3. This progress was achieved in spite of mine operators claiming, in 1969, that it was impossible to reduce exposure to the statutory limit of 2 mg/m^3. Exposure remains high at some mines and with some mining methods. Over the past decade, perhaps due to deregulation, CWP has nearly doubled among experienced miners and there are numerous "hot spots" with high prevalence of advanced CWP.

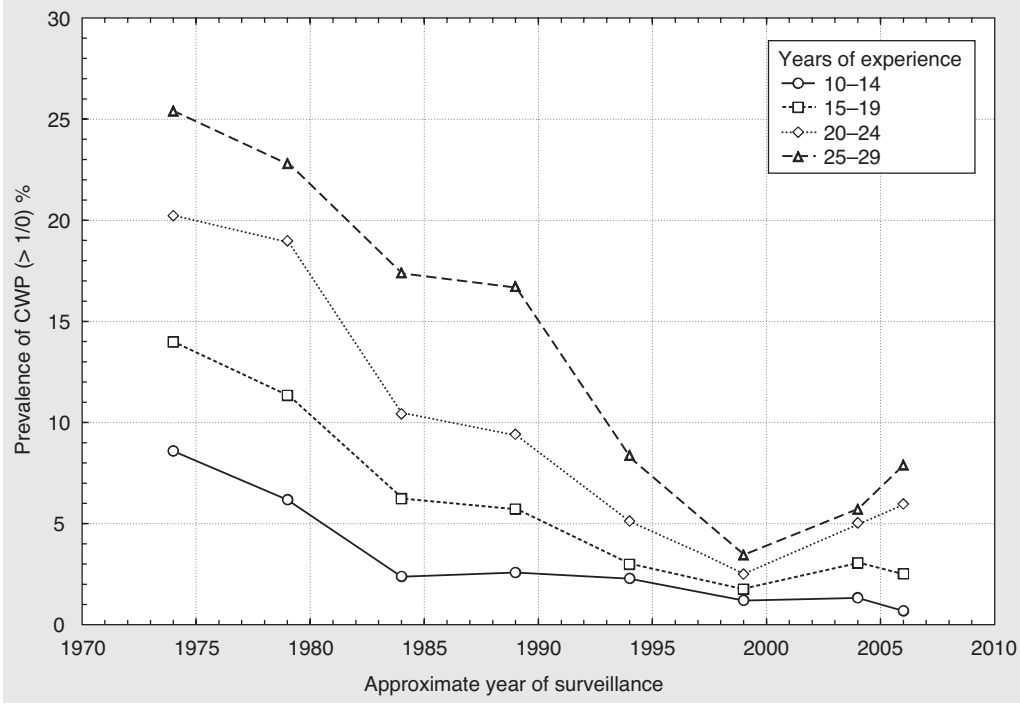


Figure 2-12. Prevalence of coal workers' pneumoconiosis (CWP) among coal miners, United States, 1974–2006, by miners' years of experience.

(*Continued*)

Box 2-5. The Mine Safety and Health Administration (MSHA): Intensive Intervention in a Dangerous Industry (Continued)

In addition, noise exposure remains high, exposure to crystalline silica is elevated, and underground miners are exposed to high levels of diesel exhaust.

From 2001 to 2005, preventable and predictable mine fires at four different coal mines killed nearly 30 miners. In 2007, elementary roof-control methods were neglected at a deep mine, resulting in a massive roof fall and the death of six miners and three rescue workers. These events prompted Congress to pass the Mine Improvement and New Emergency Response Act (the MINER Act) to improve emergency preparedness. In light of the failure of rescue efforts for recent disasters, these provisions are clearly needed, but stronger enforcement and disaster prevention—which the MINER Act did not address—could accomplish more. Indeed, as if to underscore the need, in early 2010, an explosion fueled (most likely) by methane and coal dust killed 29 miners at a mine in West Virginia. In sum, these disasters were the result of failure to control the best understood hazards of underground coal mining: fires, roof falls, and gas and dust explosions. Lack of knowledge about prevention methods did not contribute to these disasters; failure to use that knowledge did.

The Wage and Hour Division of the U.S. Department of Labor provides additional worker protection by enforcing federal minimum wage and overtime laws, minimum ages for various type of work, restrictions of young workers' exposure to specific hazardous tasks, provisions of the Family and Medical Leave Act, and field sanitation standards for migrant farmworkers.

The Department also administers workers' compensation programs for federal workers, longshoremen, and former energy workers, and provides benefits for miners who have developed coal workers' pneumoconiosis ("black lung").

The National Institute for Occupational Safety and Health (NIOSH)

Established by the OSH Act in 1970, NIOSH is part of the CDC, in the Department of Health and Human Services. It is responsible for conducting and supporting research to improve workplace safety and health, promoting and supporting training in occupational safety and health, providing technical assistance to employers and employees, and developing the scientific basis for standards and other policies.

One NIOSH program that is particularly relevant to health care practitioners of all types is the Health Hazard Evaluation (HHE) program, in which NIOSH responds to requests for investigations of workplace hazards. (See Chapter 34.) An HHE is a worksite study designed to evaluate potential workplace health hazards. HHEs can be requested by a management official, three current employees, or any officer of a labor union representing an employee. However, with an employee's consent, a health care professional can also contact NIOSH and speak with representatives of the HHE program. The program places a high priority on identifying and preventing emerging threats. The program generally will not conduct evaluations for known hazards, but will instead typically provide written information to the requestor. When an evaluation is conducted, NIOSH reports the results to the workers, the employer, and the U.S. Department of Labor, and makes recommendations for reduction or removal of the hazard. While the HHE program serves as a useful surveillance mechanism through which NIOSH is kept aware of emerging workplace concerns, NIOSH also conducts much additional surveillance to determine the number of workers exposed to specific hazards and in which industries and occupations they are at risk.

NIOSH supports research through *(a)* intramural programs that it conducts, *(b)* cooperative agreements that it initiates and in which it participates, and *(c)* research grants that extramural investigators initiate and conduct. In 1996, NIOSH established the National Occupational Research Agenda (NORA), a framework to guide occupational safety and health research—not only for NIOSH, but for the entire occupational health and safety community. Now, NORA organizes research by industry sector, although cross-cutting issues remain important. To disseminate research findings, NIOSH publishes reports and other materials that are designed to inform workers, employers, and occupational safety and health professionals of hazards and how to avoid them.

To further assist professionals and the public, NIOSH provides a toll-free information system.

It can be accessed by telephone at 1-800-CDC-INFO (1-800-232-4636). NIOSH specialists provide technical advice and information on subjects in occupational safety and health.

NIOSH supports comprehensive Occupational Safety and Health Educational Resource Centers (ERCs) and academic training programs that focus on occupational safety and health professional training and also provide continuing education and research training. They are a useful source of academic expertise and may be able to fund pilot research projects to permit preliminary investigation into new or emerging hazards.

U.S. Government Agencies and Programs in Environmental Health

The National Center for Environmental Health (NCEH)

NCEH, within CDC, coordinates a series of national programs to promote a healthy environment by targeting environmental exposures and provides technical assistance, on request, to state and local health departments. It helps to prevent disease and death resulting from interactions between people and their environment, including those due to chemicals. It also addresses hazards and impediments to walking and bicycling due to poor urban planning. NCEH has programs that address many environmental health problems and ways state and local health departments can improve their capabilities to address them. Its National Biomonitoring Program directly measures human exposure to toxic substances in the environment by measuring the substances or their metabolites. Based on the findings of this program, NCEH periodically publishes national reports to provide an overview of human exposure to environmental chemicals, a comprehensive assessment of the exposure of the U.S. population to chemicals in the environment.

The Agency for Toxic Substances and Disease Registry (ATSDR)

ATSDR, which is administratively part of CDC, was established to address health concerns arising from chemical pollution at Superfund sites. Its mission is to assess hazardous substances in the environment and mitigate their effect on public health. ATSDR performs public health assessments of waste sites and health consultations on specific hazardous substances. It also maintains disease registries, responds to emergency releases of hazardous substances, performs applied research in support of public health assessments, develops and disseminates information, and provides education and training on hazardous substances. ATSDR is obligated to formally respond to written citizen requests. (See Box 2-6.)

The National Institute of Environmental Health Sciences (NIEHS)

Part of the National Institutes of Health (NIH), NIEHS conducts and supports research to better understand how the environment influences development and progression of disease. It supports university-based resources; publishes Environmental Health Perspectives, a journal available in print and online; and produces documents on community-based participatory research and environmental justice and various research publications. NIEHS funds research and training in environmental health, including 22 university-based Environmental Health Core Science Centers, all of which have community outreach and educational components. NIEHS also funds worker education programs related to hazardous materials and environmental health education science curricula for grades K through 12.

The Environmental Protection Agency (EPA)

The mission of the EPA is to protect human health and to safeguard the natural environment on which it depends. By developing and enforcing regulations, it administers the Clean Air Act; the Clean Water Act; the Comprehensive Environmental Response, Compensation and Liability Act (Superfund); the Resource Conservation and Recovery Act; and the Toxic Substances Control Act (see Chapter 30). Although the EPA is primarily a regulatory agency, it also has research and laboratory facilities, training and outreach programs, and environmental justice initiatives that may provide expertise and technical assistance. It provides grants, studies environmental issues, sponsors partnerships, teaches people about the environment, and publishes information.

Box 2-6. How to Request Assistance from the Agency for Toxic Substances and Disease Registry (ATSDR)

Michelle Watters

ATSDR was established by the CERCLA (Superfund) legislation in 1980 to assist in evaluating public health impacts involving hazardous waste sites. A federal public health agency of the U.S. Department of Health and Human Services, it is administered together with the National Center for Environmental Health (NCEH), part of the Centers for Disease Control and Prevention (CDC). ATSDR does not have a regulatory role at hazardous waste sites, but it makes public health recommendations to the Environmental Protection Agency (EPA) and other government agencies concerning hazardous waste sites. ATSDR performs applied substance-specific research, exposure investigations and health studies, maintains registries, and provides information and health education on hazardous substances. ATSDR also receives requests from government agencies and citizens to investigate public health concerns from hazardous releases.

While public health practitioners and health care providers can access a variety of ATSDR educational materials and training opportunities on hazardous substances on the ATSDR Web site (http://www.atsdr.cdc.gov), more specific concerns or questions can be directed at any time to (800) 232-4636 or <cdcinfo@cdc.gov>, the regional ATSDR offices (http://www.atsdr.cdc.gov/dro/index.html), or the environmental divisions of state health departments. Information on chemical releases and permits from operating facilities can be obtained from the EPA Web site (http://www.epa.gov/enviro/index.html), EPA regional offices(http://www.epa.gov/epahome/postal.htm#regional), or state environmental agencies. Environmental emergencies that present sudden threats to public health should be reported to the National Response Center (800-424-8802).

By petitioning ATSDR in writing to perform a public health assessment, any person or group can request ATSDR to address health concerns related to an uncontrolled release of a hazardous substance from a waste site or former facility. The petition should include available relevant environmental data and the facility name, location, description of the release, and relevant health data.

ATSDR then gathers and reviews information about the site. Considerations are made regarding whether an assessment has already been performed that addresses the health concerns, whether a hazardous substance has been released, and whether a public health assessment is the most appropriate response. The petitioner is sent a written response as to whether the petition process will continue.

The ATSDR site team assembled includes environmental scientists, physicians, toxicologists, epidemiologists, and others. The site team gathers available environmental and exposure data from the site and identifies public health issues and the petitioner's concerns. A scoping visit may be made to the site to meet with the petitioner and other community members. A post-scoping debriefing session is held to determine whether there is a plausible relationship between possible human exposure and adverse health outcomes from a release given the location, concentration, and toxicity of the substance.

Petitioners are informed in writing of ATSDR's decision about the most appropriate public health response or if the petition has not been accepted. The public health response typically is a health consultation by ATSDR or a state public health agency that evaluates the available environmental exposure data and examines the exposure routes.

A *health consultation* is a written evaluation about the hazardous substance released at a site and the likelihood that human exposure can occur or has occurred, and if the level of exposure could result in harm. A *public health assessment* (PHA) is a more comprehensive document that examines multiple exposure pathways. Depending upon the complexity of the site, community advisory panels may be formed to act as a liaison between ATSDR and the community. If a public health hazard is determined to be present at the site, recommendations are made for reducing or eliminating the exposure and additional public health responses. ATSDR works with the EPA and other environmental and health agencies to ensure that the recommendations can be implemented. Before the PHA becomes final, there is a public release of the document for community review and comment. A public meeting may also be held to discuss the findings and public health action plan.

Its Web site includes tools to identify contaminant sources at the neighborhood level ("Enviromapper"), Toxic Release Inventory information, and real-time air pollution mapping, among other resources.

State and Local Government Agencies

Public health departments generally perform some environmental control and sanitation, and many have programs on childhood lead screening. Some address other housing issues, such as radon detection, window safety, and water incursion, although sometimes these programs are located in a department of housing. Control of vectors, including rats, mosquitoes, and other pests, is an additional responsibility of many health departments. State departments of environmental protection or environmental resources are tasked with many of the enforcement responsibilities required by EPA regulations,

although there is growing recognition of the need for regional collaboration among states for many of these responsibilities. These state-level departments or the EPA may provide information about certified laboratories that perform environmental testing. Some state health departments have strong units that address occupational safety and health. Some major cities have strong environmental health units within their public health departments. State, county, and municipal Web sites are useful sources of information. Government agencies are also listed in the blue pages of telephone books.

In those states with collaboration among different entities, such as departments of health, workers' compensation divisions, academic programs, and state-plan occupational safety and health programs, effective partnerships have been established that link surveillance to interventions so that hazards can be identified and reduced. Because of inadequate resources, many states do not have sufficient resources to address environmental and occupational health issues effectively.

International Organizations

The World Health Organization (WHO)

WHO is the directing and coordinating authority for health within the United Nations system. WHO aims to strengthen national systems to respond to the needs of working populations, establish basic levels of health protection for all workers, and ensure all workers access to preventive health services. The Occupational Health Group of WHO operates a network of Collaborating Centres that actively participate in its Global Plan of Action on Workers' Health. The Department of Public Health and Environment of WHO aims to promote a healthier environment, intensify primary prevention, and influence public policies in all sectors so as to address the root causes of environmental threats to health. The Pan American Health Organization (PAHO) is the regional organization of WHO for North and South America as well as the Caribbean.

The International Agency for Research on Cancer (IARC)

The mission of IARC, which is part of WHO, is to coordinate and conduct research on the causes of human cancer and the mechanisms of carcinogenesis, and to develop scientific strategies for cancer prevention and control. IARC is involved in both epidemiological and laboratory research and disseminates scientific information through publications, meetings, courses, and fellowships.

The International Labor Organization (ILO)

ILO is devoted to advancing opportunities for women and men to obtain decent and productive work in conditions of freedom, equity, security, and human dignity. Its main aims are to promote rights at work, encourage decent employment opportunities, enhance social protection, and strengthen dialogue in handling work-related issues. In promoting social justice and internationally recognized human and labor rights, ILO continues to pursue its founding mission that labor peace is essential to prosperity. ILO helps advance the creation of decent jobs and the kinds of economic and working conditions that give working people and business people a stake in lasting peace, prosperity, and progress.

The United Nations Environment Programme (UNEP)

The mission of UNEP is to provide leadership and encourage partnership in caring for the environment by inspiring, informing, and enabling nations and peoples to improve their quality of life without compromising that of future generations.

Intergovernmental Panel on Climate Change (IPCC)

IPCC is the leading body for the assessment of climate change. It was established by UNEP and the World Meterological Organization (WMO) to provide the world with a clear scientific view of the current state of climate change and its potential environmental and socioeconomic consequences.

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The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health, the Mine Safety and Health Administration, the Agency for Toxic Substances and Disease Registry, and the Chemical Safety and Hazard Investigation Board.

3

Occupational and Environmental Health Surveillance

Kerry Souza, Letitia Davis, and Jeffrey Shire

How and where are workers injured or made ill on the job?

How many workers are at risk of serious work-related health problems, and where do they work?

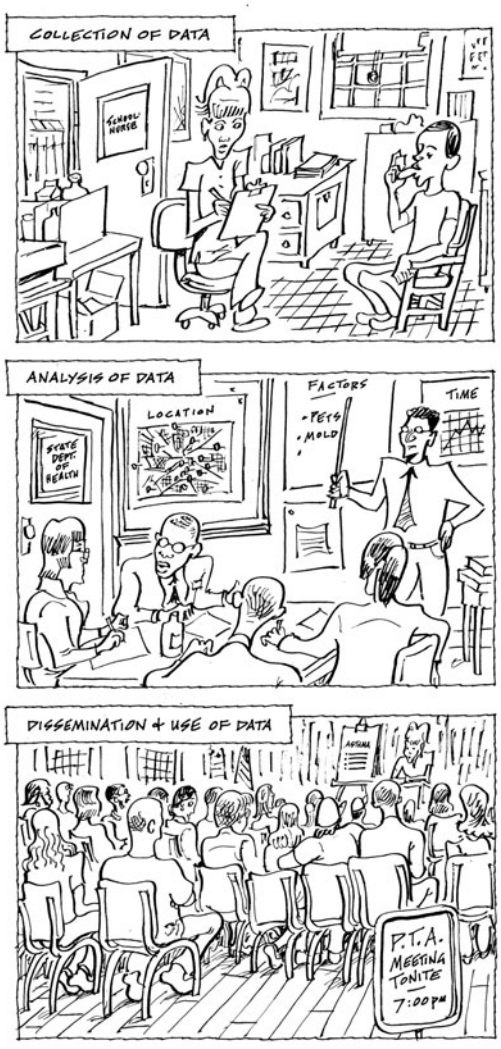
How are environmental hazards changing over time and space, and how might they be contributing to disease?

Public health surveillance, in response to these and many other questions, provides answers that ultimately lead to prevention. Surveillance is "the ongoing systematic collection, analysis, and interpretation of health data essential to the planning, implementation, and evaluation of public health practices, closely integrated with the timely dissemination of these data to those who need to know. The final link in the surveillance chain is the application of these data to prevention and control."[1]

The objectives of occupational and environmental health surveillance are the following:

- To characterize the most common types of injuries and illnesses related to occupational and environmental factors, their causes, and their risk factors
- To characterize affected populations
- To estimate the overall magnitude and severity of problems
- To identify geographic areas, industries and occupations, and specific workplaces and communities where interventions are most needed
- To identify new or previously unidentified risk factors that should be researched
- To characterize the distribution of occupational and environmental health hazards
- To evaluate the effectiveness of interventions
- To generate support for prevention activities

Surveillance is often referred to as the "cornerstone of public health practice," providing the foundation on which to build successful prevention programs. Broadly speaking, surveillance can be divided into surveillance for health outcomes (such as injuries, illnesses, and deaths) and surveillance for hazards or exposures. Ideally, surveillance is ongoing and continuous. Surveys that are performed repeatedly to monitor trends and changes in prevalence are generally regarded as surveillance, but cross-sectional studies and one-time surveys and data collections are generally not—although such activities are sometimes used to augment surveillance data.



The three phases of surveillance. (Drawing by Nick Thorkelson.)

Surveillance for environmental and occupational injuries and illnesses as well as health problems linked to the wide range of types of exposure in these different settings must utilize a wide range of approaches and data sources. Each method and data source will have its own advantages and disadvantages.

Surveillance for diseases caused by environmental exposures is especially challenging because the diseases of interest may have many potential causes. Therefore, the focus is often on hazards in the environment, rather than health outcomes. Surveillance for markers of exposure to hazards, such as blood lead levels, may also be performed. This chapter focuses on surveillance in the occupational context. An overview and select examples of environmental health surveillance are provided near the end of this chapter.

CASE-BASED AND POPULATION-BASED SURVEILLANCE

Surveillance systems may provide detailed information on cases of injury or illness, generate incidence rates, or both. *Case-based surveillance* involves the ongoing and rapid identification of cases for purpose of follow-up investigation of—and possible intervention for—affected individuals. Case-based surveillance, which is generally used in conducting surveillance of communicable diseases, is based on the concept of a sentinel health event—a warning sign that prevention has failed and intervention is warranted. Follow-up may include interventions—such as to control spread of infectious disease in a community or reduce injury risks among co-workers—and collection of additional data to better understand the epidemiology of the disorder. Data from case-based surveillance may or may not be complete or representative.

Several states implement case-based surveillance for selected occupational disorders using a model developed by the National Institute for Occupational Safety and Health (NIOSH)—the Sentinel Event Notification System for Occupational Risks (SENSOR). Following the SENSOR approach, a state health agency identifies sentinel cases based on reports from health care providers and facilities and uses stringent case criteria to confirm cases. It sometimes obtains additional data from affected workers, health care providers, and employers. State health agencies also use administrative data, such as hospital discharge or workers' compensation records, to identify cases of illness and injury. Results of data analyses are used for prevention and intervention activities. Sometimes, data from several states are aggregated to gain a broader perspective. An example of a case-based surveillance system is the SENSOR asthma program, which uses case reports of work-related asthma from health care providers and other sources to target workplaces for follow-up

investigations and implement broader intervention activities (Box 3-1).[2]

In contrast, *population-based, or rate-based, surveillance* collects data that can be used to monitor trends in a population over time, locale, and population characteristics. It may involve collecting data on all cases—a census—or on a representative sample of cases. Population-based surveillance requires denominator information—such as the number of workers at risk for a specific injury or illness. The Childhood Lead Poisoning Prevention Programs and the Bureau of Labor Statistics (BLS) Survey of Occupational Injuries and Illnesses (SOII) are examples of population-based surveillance systems.

Case-based and population-based approaches to surveillance are not mutually exclusive; some of the best surveillance systems have attributes of both, identifying sentinel cases for follow-up and simultaneously generating representative summary data to guide broader-based prevention. To influence public health policy, a combination of case reports (stories) and summary data (statistics) is often most effective.

Box 3-1. Asthma Surveillance in California: Combining Environmental and Occupational Health Surveillance

Jennifer Flattery

Some conditions, such as asthma and disorders due to pesticides and lead, occur both in the community and in the workplace and offer opportunities to combine aspects of environmental and occupational surveillance to maximize yield of information and potential for prevention. The California Department of Public Health operates two parallel and complementary programs for asthma prevention, which include surveillance systems for environmental and occupational asthma. The programs collaborate to generate a statewide summary on asthma, profiles on the asthma burden and risk factors for each county in the state, and a statewide blueprint for asthma prevention.

An analysis of work-related asthma data indicated that cleaning chemicals in schools were associated with work-related asthma. Because schools were also a focus of the environmental asthma program for prevention of childhood asthma, a collaborative intervention project was initiated to promote "asthma-safe" cleaning methods in schools through training and technical assistance.

ACTIVE AND PASSIVE SURVEILLANCE

Surveillance systems can generally be characterized as active or passive. Passive surveillance relies on reports to a public health agency of injury or illness submitted by reporting individuals or facilities. Once reports are received, the agency will act on the information received. For example, an adult lead poisoning surveillance program receives reports of elevated blood lead levels (BLLs) from clinical laboratories, then analyzes and disseminates the data, and works with its community partners to develop interventions.

In contrast, active surveillance involves a more aggressive approach to case finding. For example, the Census of Fatal Occupational Injuries (CFOI) devotes much effort to educating potential reporters of work-related fatalities, such as medical examiners, and even uses newspaper searches to identify fatalities. Active surveillance, which is more costly and labor intensive, may be necessary when a passive approach is ineffective.

A surveillance system can incorporate aspects of both active and passive surveillance. For example, the SENSOR surveillance system for occupational asthma partially relies on physicians and nurses to report cases to state public health agencies. Since physicians and nurses may be unaware of reporting requirements or may find them burdensome, outreach and education are necessary to encourage them to report cases. Most surveillance systems require some degree of ongoing feedback and communication with those reporting cases to ensure continued success of the system.

HAZARD SURVEILLANCE

Surveillance for health hazards can be particularly valuable when disease latency periods are long. In such cases, identifying the communities, occupations, workplaces, and/or demographic groups exposed to a hazard can lead to primary prevention, even without data on affected persons. For example, surveillance for asbestos use can lead to mitigation of exposure, thereby preventing development of asbestosis. In contrast, surveillance for mesothelioma, a long-term

outcome of asbestos exposure, identifies cases that may not lead directly to prevention. Many workplaces, work tasks, and even industries that created asbestos exposures that caused current mesothelioma cases no longer exist.

Although there is no U.S. occupational hazard or exposure surveillance system, NIOSH has performed large-scale occupational exposure surveys. From 1981 to 1983 and again a decade later, NIOSH performed the National Occupational Exposure Survey (NOES) to collect data on potential occupational exposures to chemical, physical, and biological agents. The survey involved on-site visits to more than 4,000 workplaces in over 500 industries, with 1.8 million workers in almost 400 occupational categories. From survey data, NIOSH estimated the number of U.S. workers potentially exposed to thousands of hazardous substances, by occupation and industry.

The Occupational Health and Safety Administration (OSHA) also maintains data that can be used to identify possible hazards in a particular workplace. OSHA's Integrated Management Information System (IMIS) database includes information about hazardous exposures measured during OSHA's routine workplace inspections and its complaint- and incident-driven inspections.

Principles of hazard surveillance[3] have also been applied to monitoring occupational exposures in order to target opportunities for intervention and to track success in reducing or eliminating exposures. For example, OSHA has established a standard to eliminate exposure to ethylene oxide in hospitals to prevent risks to pregnant workers. Analysis of surveillance data has pointed to successes in reducing exposure and has identified workplaces where more efforts are needed to protect workers.

NATIONAL SURVEILLANCE SYSTEMS FOR OCCUPATIONAL INJURIES AND ILLNESSES

The primary national occupational health surveillance systems in the United States are CFOI and SOII, both of which are administered by the BLS, working in collaboration with the states.

CFOI is designed to count and describe all fatal work-related injuries in the United States (Fig. 3-1).[4] It gathers data from as many as 25 different sources, such as death certificates and newspaper clippings. CFOI collects information on the worker, and the types of work, industry,

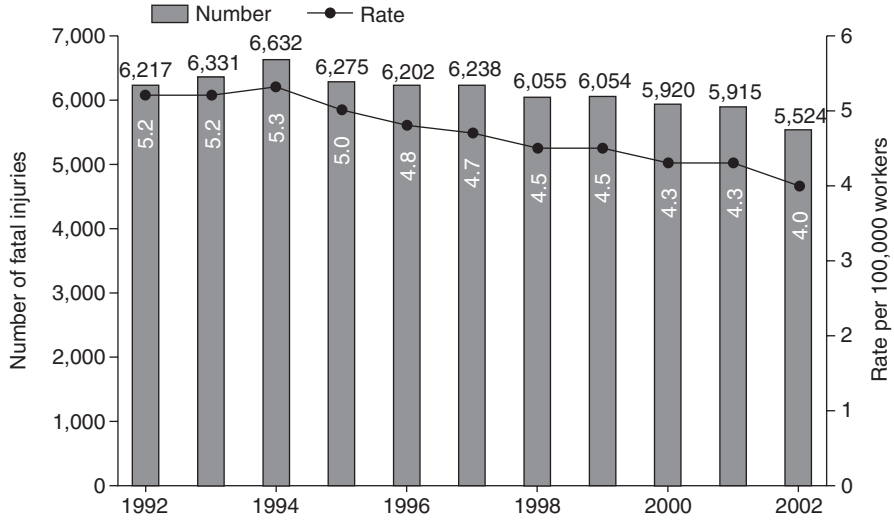


Figure 3-1. Numbers and rates of fatal occupational injuries, United States, 1992–2002. This graph depicts a decline in annual occupational fatality rates from 5.2 to 4.0 per 100,000 workers. (*Source*: Biddle EA. Is the fatal occupational injury experience in the United States really improving? Number and rate of fatal occupational injuries by year, 1992–2002. Washington, DC: Census of Fatal Occupational Injuries, Bureau of Labor Statistics, 2003. Available at: http://www.bls.gov/iif/oshwc/cfoi/biddle.pdf. Accessed on June 16, 2010.)

and workplace. It also collects information on the exposure that led to the injury, the source of injury, activity, and location of the worker at the time of the incident. The BLS provides training and resources for data collection by state agencies, usually state labor or health departments. State agencies transmit data to the BLS, which compiles a national CFOI data set. Each state agency seeks to collect case reports from sources that are specific to the state, facilitating local use of data for intervention.

SOII is the most comprehensive source of nonfatal occupational injury data for the United States, providing estimates of numbers and rates of occupational injuries nationally and for about 40 states, by a range of detailed worker and workplace characteristics. Unlike other major public health surveillance systems, SOII collects data from workplaces, rather than individuals or health care providers or facilities. This allows the BLS to collect information on the source of the injury, the event that caused the injury, and other detailed information about the workplace. Employers, unions, and others can use these data to compare their injury rates to industry averages. SOII relies on a nationwide sample of employers to report data from their OSHA-required records (OSHA-300 logs); farms with fewer than 11 employees, private households, federal government agencies, and self-employed workers are not included.[4]

Injuries and illnesses that are not recorded by employers on OSHA logs or not reported by workers to their employers are missed by SOII. For example, immigrant workers, who often perform the most hazardous tasks, may be reluctant to report their injuries due to possible fear of reprisal or job loss.[5] In addition, SOII is not a good system for tracking occupational illness, especially chronic diseases. Under-diagnosis of occupational illness by physicians, the long latency periods for some occupational diseases, and the multifactorial nature of many diseases contribute to difficulties in accomplishing surveillance for occupational disease through a workplace-based system such as SOII. Therefore, targeted surveillance systems combining data from select states have been developed for several specific occupational diseases, including adult lead poisoning, occupational pesticide poisoning, work-related asthma, mesothelioma, pneumoconiosis, and silicosis.

STATE-BASED OCCUPATIONAL HEALTH SURVEILLANCE

State health and labor departments carry out a variety of occupational health surveillance activities, much of which is funded by NIOSH. This surveillance can provide data on local variations in occupational injuries and illnesses, respond to state-specific needs, and facilitate local intervention activities. State-based surveillance can also fill information gaps at the national level by providing data poorly captured by national systems, such as information on occupational diseases. States rely on both existing data, such as data sets of hospital discharges, and data collected specifically for surveillance, such as case reports of occupational illness from physicians and medical care facilities.

As of 2009, NIOSH funded 15 states to implement occupational health surveillance programs. At a minimum, each of these states is encouraged to use data from existing systems to prepare 13 occupational safety and health indicators (Table 3-1).[6] Activities in these states include surveillance for occupational fatalities, pesticide poisoning, occupational asthma, silicosis, sharps injuries to health care workers, work-related burns, and serious work-related injuries to teenagers and trucking industry workers. Not all states track all outcomes. Additional states are funded by NIOSH to track adult lead poisoning through the Adult Blood Lead Epidemiology and Surveillance (ABLES) program.

Physician and Laboratory Reporting

Public health reporting laws have enabled state agencies to gather surveillance data, mainly on communicable diseases. In 1874, physician reporting of disease to public health agencies began when Massachusetts established a voluntary reporting program in which physicians mailed a postcard every week to the state health department listing "prevalent" diseases. In 1893, Michigan became the first state to require physician reporting of specific diseases. By 1901, reporting of smallpox, tuberculosis, and cholera

Table 3-1. Occupational Health Indicators

Indicator	Source of Data for Indicator
Nonfatal injuries and illnesses reported by employers	Bureau of Labor Statistics (BLS) Annual Survey of Occupational Injuries and Illnesses (SOII)
Work-related hospitalizations	State hospital discharge data
Fatal work-related injuries	Census of Fatal Occupational Injuries (CFOI)
Amputations reported by employers	SOII
Amputations identified in state workers' compensation systems	State workers' compensation data
Hospitalizations for work-related burns	State hospital discharge data
Musculoskeletal disorders reported by employers	SOII
Carpal tunnel syndrome cases identified in state workers' compensation system	State workers' compensation systems
Pneumoconiosis hospitalizations	State hospital discharge data
Pneumoconiosis mortality	State vital records
Acute work-related pesticide poisonings reported to poison control centers	American Association of Poison Control Centers
Incidence of malignant mesothelioma	State cancer registries
Elevated blood lead levels among adults	Adult Blood Lead Epidemiology Surveillance (ABLES) program
Workers employed in industries with high risk for occupational morbidity	Census Bureau County Business Patterns
Workers employed in occupations with high risk for occupational morbidity	Bureau of Labor Statistics Current Population Survey (CPS)
Workers in occupations and industries with high risk for occupational mortality	CPS
Occupational health and safety professionals	Current membership rosters of cited organizations.
Occupational Safety and Health Administration (OSHA) enforcement activities	OSHA Office of Statistics
Amount of workers' compensation awards paid	National Academy of Social Insurance

was legally required in all states. While communicable diseases still dominate the list of reportable conditions, 30 states also require health care providers to report selected occupational disorders, such as work-related asthma, to a state agency. (See Box 3-2.)

While not all cases are reported, these laws have been valuable in facilitating identification of cases of and risks for specific occupational diseases. For example, they have helped to identify health care workers as a group at risk of developing asthma from exposure to chemicals in cleaning products.

Mandatory reporting from laboratories provides the foundation for yet other surveillance programs, such as adult and child blood lead surveillance. As of 2009, there were 40 states participating in the ABLES program (Box 3-3). To participate in ABLES, a state must require clinical laboratories to report BLLs to a state public

Box 3-2. Occupational Health Reporting Requirements in New Jersey

In New Jersey, physicians, advanced practice nurses, and physician assistants are required, by law, to report the following diseases, injuries, and poisonings to the New Jersey Department of Health and Senior Services:

- Asbestosis, silicosis, and other pneumoconiosis
- Work-related asthma
- Extrinsic allergic alveolitis
- Lead, arsenic, mercury, and cadmium toxicity in adults
- Pesticide toxicity
- Work-related injuries in children under age 18
- Work-related fatal injuries
- Occupational dermatitis
- Work-related carpal tunnel syndrome
- Poisoning caused by known or suspected occupational exposure

Health care providers are also asked to report any other occupational disease that is "a threat to worker health."

Source: New Jersey Administrative Code 8:58-1.5, 1.6, and 1.7. Available at: http://www.nj.gov/health/ohs/rptrequirement.shtml. Accessed on October 6, 2009.

Box 3-3. Tracking Lead Exposure to Workers: The Massachusetts Blood Lead Registry

Richard Rabin

In 1990, the Massachusetts Legislature passed the Occupational Lead Poisoning Registry Law, which requires laboratories in the state to report blood lead levels (BLLs) of 15 μg/dl or higher in adults to the Massachusetts Occupational Lead Poisoning Registry in the Division of Occupational Safety (DOS). The Registry, which participates in ABLES, informs workers about the hazards of lead and how exposures can be controlled, provides employers with information and technical assistance to control lead exposure, and provides consultation and advice to health care providers on medical management of lead poisoning.

The Massachusetts Department of Public Health periodically analyzes Registry data and distributes reports to physicians, employers, unions, legislators, and other interested parties. These reports help to identify industries, occupations, and workplaces that present the greatest lead hazards and to target them for follow-up investigation and intervention. Data collection also permits the study of trends in the incidence of lead poisoning over time.

Upon receiving a report of an elevated BLL, the Registry contacts the physician who ordered the test to obtain further identifying information about the reported worker and lead exposure, and to provide medical guidelines to the physician. Its medical consultant is available to consult on individual cases. The Registry then calls the worker to gather more information on lead exposure and workplace conditions, and it sends the worker information on occupational lead exposure and workplace rights. When the employer is identified, the Division of Occupational Safety refers the case to the Occupational Safety and Health Administration (OSHA) and/or contacts the employer to discuss the problem and offer a worksite consultation.

Between 1999 and 2002, the Registry received reports of elevated BLLs—64, 63, and 48 μg/dl—in three immigrant Brazilian house painters who were not fluent in English and worked for the same painting company. According to the workers and their physicians, the employer had not complied with the OSHA lead standard. There had been no medical monitoring and no training or information provided on the health hazards of lead. The Division of Occupational Safety provided consultation to this company and continues to monitor its progress in protecting its employees from lead exposure.

health agency. State and local public health agencies rely on both physician and laboratory reporting to obtain information on children with elevated BLLs. As of 2009, 46 states reported data to the CDC Childhood Blood Lead Surveillance System.

THE USE OF ADMINISTRATIVE DATA FOR OCCUPATIONAL HEALTH SURVEILLANCE

Data collected for administrative purposes, such as workers' compensation, hospital discharge, and emergency department data, can contain information on injuries and illnesses not reported into employer-based surveillance systems.

Workers' Compensation Data

Workers' compensation data have been used extensively for research and surveillance. For example, the Massachusetts Teens at Work (TAW) program identifies an average of 400 cases annually of serious work-related injuries to teenagers by using workers' compensation data. TAW estimates rates of work-related injuries to teenagers and performs demographic and occupational analyses of data on injured teens.[7] TAW uses these analyses to plan interventions and also shares findings with community organizations, schools, employers, unions, and policy makers. Washington State's Safety and Health Assessment and Research for Prevention (SHARP) program stands out for its regular reporting on a variety of injuries and illnesses from its state workers'compensation system. Several other state programs access and utilize workers' compensation data as an important source of data on the occupational health of workers in their states.

Workers' compensation data have limitations for use in surveillance. Workers awarded compensation are not representative of all those with work-related injuries and illnesses. Not all worker groups are equally likely to receive benefits. In addition, differences in eligibility for workers' compensation among states make comparisons between states difficult.

Selected Other Occupational Health Surveillance Systems

The National Electronic Injury Surveillance System (NEISS)

The Consumer Project Safety Commission (CPSC) operates NEISS, which is based on a U.S. probability sample of hospital emergency departments (EDs). It collects information from

participating hospitals on ED visits involving nonfatal injuries associated with work or consumer products.

National Agricultural Workers Survey (NAWS)

Initiated in 1988, NAWS is a probability survey of a sample of U.S. hired crop workers, originally designed to collect demographic and employment data. NIOSH has incorporated occupational health questions into this survey to guide interventions among farmworkers.

National Healthcare Safety Network (NHSN)

NHSN is a voluntary, Internet-based surveillance system, managed by the CDC, that integrates and expands surveillance systems for the safety of patients and health care workers. It conducts surveillance for exposures to blood and body fluids and to influenza, and monitors vaccination of health care workers against influenza.

National Occupational Respiratory Mortality System (NORMS)

NORMS is an interactive data system that is based on mortality data provided annually from the National Center for Health Statistics. Information on deaths for which the underlying or contributing cause was pneumoconionis, malignant mesothelioma, or hypersensitivity pneumonitis is included. NORMS also determines annual industry- and occupation-specific death rates for many respiratory disorders.

Occupational Health Indicators

Health indicators are well-defined surveillance measures that allow states to uniformly collect and report on the health status of the population. States use both occupational and environmental health indicators to track health problems and to guide prevention and intervention measures (Tables 3-1 and 3-2).[8] Some of these indicators can also be used to compare rates of illnesses and injuries among states.

Table 3-2. Types and Examples of Environmental Health Indicators

Hazard Indicators (Potential for Exposure to Contaminants or Hazardous Conditions)
Criteria pollutants in ambient air
Hazardous or toxic substances released in ambient air
Residence in nonattainment areas (for criteria air pollutants)
Motor vehicle emissions
Tobacco smoke in homes with children
Residence in a flood plain
Pesticide use and patterns of use
Residual pesticide or toxic contaminants in foods
Ultraviolet light
Chemical spills
Monitored contaminants in ambient and drinking water
Point-source discharges into ambient water
Contaminants in shellfish and sport and commercial fish
Exposure Indicators (Biomarkers of Exposure)
Blood lead level (in children)
Health Effect Indicators
Carbon monoxide poisoning
Deaths attributed to extremes in ambient temperature
Lead poisoning (in children)
Noise-induced hearing loss (nonoccupational)
Pesticide-related poisoning and illness
Illness or condition with suspected or confirmed environmental contribution (a case or an unusual pattern)
Melanoma
Possible child poisoning (resulting in consultation or emergency department visit)
Outbreaks attributed to fish and shellfish
Outbreaks attributed to ambient or drinking water contaminants
Intervention indicators (programs or official policies addressing environmental hazards)
Programs that address motor vehicle emissions
Alternate fuel use in registered motor vehicles
Availability of mass transit
Policies that address indoor air hazards in schools
Laws pertaining to smoke-free indoor air
Indoor air inspections
Emergency preparedness, response, and mitigation training programs, plans, and protocols
Compliance with pesticide application standards (among pesticide workers)
Activity restrictions in ambient water (health-based restrictions)
Implementation of sanitary surveys
Compliance with operation and maintenance standards for drinking water systems
Advisories to boil water

Healthy People Objectives

The Healthy People objectives for the United States, which are developed for each decade, include a set of objectives specific to occupational health and safety and a set of objectives specific to environmental health.[9] These objectives are developed by staff members of federal and state agencies, academic and community-based

researchers, and others. Baseline surveillance data are necessary for a Healthy People objective to be established.

Healthy People 2020 objectives for occupational health and environmental health outcomes can be accessed at http://www.healthypeople.gov/hp2020/default.asp.

MEDICAL SURVEILLANCE AND MEDICAL SCREENING

Distinct from the population-based surveillance that is the focus of this chapter, *medical surveillance* is the ongoing medical monitoring of exposed individual workers in a company,

Box 3-4. National Childhood Blood Lead Surveillance

Lemuel Turner

Approximately 250,000 U.S. children 1 to 5 years of age have blood lead levels (BLLs) greater than 10 μg/dL, the level at which the Center for Disease Control and Prevention (CDC) recommends public health actions be initiated. Because young children are at highest risk for lead poisoning, the CDC recommends that screening programs focus on children under 6 years of age.

State and community health agencies are principal delivery points for childhood lead screening and case management. These agencies receive laboratory reports of children with elevated BLLs and collect demographic information and data on risk factors for lead poisoning during case investigations of these children. Many states do not have resources to independently develop lead surveillance systems that can systematically collect and maintain computerized records from laboratories on BLLs, and from environmental departments that conduct inspections and report on remediation activities. In 1992, the CDC began awarding cooperative agreements to state and local departments of health or departments of the environment for implementation of childhood blood lead surveillance. The CDC developed and provided to state and local Childhood Lead Poisoning Prevention Programs (CLPPPs) the Systematic Tracking of Elevated Lead Levels and Remediation (STELLAR) program. STELLAR allowed CLPPPs to conduct BLL surveillance, patient medical case management, and environmental investigation management, and to report surveillance data to the CDC. The CDC currently receives surveillance data quarterly from 42 comprehensive CLPPPs. Forty of these programs have been awarded cooperative agreements by the CDC. The surveillance data are a portion of the larger patient tracking system.

In 2000, the President's Task Force on Environmental Health Risks and Safety Risks to Children issued a new federal strategy entitled "Eliminating Childhood Lead Poisoning: A Federal Strategy Targeting Lead Paint," with recommendations to support state-based blood lead surveillance systems and capacity to use data linkage to monitor lead screening in the Medicaid population. This new strategy reinforces the 1991 Department of Health and Human Services "Strategic Plan for the Elimination of Childhood Lead Poisoning." The 1991 plan called for several strategies, including increased federal support for childhood lead poisoning prevention programs and national surveillance. The national and state systems are complementary.

The purposes of state surveillance and patient tracking are the following:

- To monitor case management of individual children with lead poisoning
- To evaluate the productivity and effectiveness of state and local programs
- To identify local program needs such as capacity building in inspection and abatement methods and laboratory services
- To identify clusters of cases to target preventive interventions
- To identify possible sources of lead and remove or reduce those exposures

The purposes of national surveillance and patient tracking are the following:

- To track national progress in eliminating childhood lead poisoning
- To track the number of children with lead poisoning to prioritize federal resources
- To evaluate the effectiveness of the CDC grant program
- To assess the effectiveness of state prevention activities to improve interventions
- To monitor national trends in lead sources exposing children

As screening activities become more effective at targeting high-risk children, surveillance data will more accurately represent the burden of childhood lead poisoning in the United States. In addition, information collected from surveillance programs will facilitate a comprehensive assessment of prevention effectiveness of childhood lead poisoning prevention activities. Trends can be tracked over time to assess the impact of childhood lead poisoning prevention activities on elimination of this disease, which will require removal and/or reduction of sources of lead in the environments of children. Documentation of all lead sources identified and actions taken to reduce the exposures will be important to track over time.

In 2008, the CDC began the development of the Healthy Housing and Lead Poisoning Surveillance System (HHLPSS) to replace STELLAR. HHLPSS is a Web-based system that vastly improves the ability of state and local CLPPPs to provide real-time services to their residents, greatly enhances the mission to eliminate childhood lead poisoning, and, for the first time, monitors housing risk factors other than lead that are associated with adverse health effects.

workplace, or other specific cohort. Individual companies may perform medical surveillance of their workers. Epidemiologists may conduct medical surveillance of a cohort of workers as part of a study. OSHA requires medical surveillance of workers exposed to some specific hazardous substances and exposures, including acrylonitrile, arsenic, asbestos, benzene, bloodborne pathogens, 1,3-butadiene, cadmium, suspect carcinogens, coke oven emissions, cotton dust, 2-dibromo-3-chloropropane, ethylene oxide, formaldehyde, lead, methylenedianiline, methylene chloride, noise, and vinyl chloride, as well as compressed air environments, hazardous waste, and hazardous chemicals in laboratories.[10] Government actions and legislative mandates also place certain cohorts of workers under medical surveillance. (See Chapter 2.)

An example of a medical surveillance program established by legislative mandate is the Former Worker Medical Surveillance Program of the U.S. Department of Energy (DOE). In 1993, Congress ordered the DOE to conduct medical surveillance for those who had worked with toxic and radioactive substances in U.S. nuclear weapons production. This program has provided medical screening and follow-up to thousands of former workers and has generated information about health problems in these workers.[11] For example, 1.3% of former Nevada Test Site workers who were screened had sensitization to beryllium, evidence of past exposure to this metal whose particles can cause berylliosis, an incurable lung disease.[12] In some individuals, a single exposure to beryllium can result in berylliosis.

ENVIRONMENTAL HEALTH SURVEILLANCE

Environmental health surveillance may include monitoring of environmental hazards, exposures to toxic environmental contaminants, or diseases caused by environmental factors.[13] Public health workers need to collect data regularly and systematically to determine, in a timely manner, whether levels of environmental contaminants are associated with illness in their communities. Environmental health surveillance typically utilizes a variety of data sources, each with its advantages and disadvantages.[14] National data sources for environmental health surveillance, such as in government agencies, have tended to be fragmented.[1]

Figure 3-2. Young children living in inner-city tenement buildings, as shown here, are at high risk for childhood lead poisoning. Surveillance programs can help identify children at high risk and lead to intervention and other preventive measures. (Photograph by Earl Dotter.)

Conducting surveillance on factors that could be used for prevention in the short term may be more valuable than focusing on diseases with long latency.[15] Environmental health surveillance systems in the United States focus on childhood lead exposure (Box 3-4 and Fig. 3-2), spills of hazardous substances and resultant health outcomes, carbon monoxide poisoning (Box 3-5), and combining fragmented sources of data into one readily accessible network. These disparate systems share the common purpose of using collected data and analyses to improve the public's health.

There is great interest from both the public and the public health community in understanding the distribution and risk factors for asthma. However, asthma, unlike many infectious diseases, is not reported to the Centers for Disease Control and Prevention (CDC). Surveillance for asthma is accomplished primarily through a national telephone survey called the Behavioral Risk Factor Surveillance System (BRFSS). CDC funding and support of the BRFSS have resulted in detailed collection of self-reported data on asthma from all 50 states. The National Environmental Public Health Tracking Network integrates and centralizes data from many sources for use by scientists, health professionals, policy makers, and members of the public (Box 3-6).

Much environmental health surveillance is also conducted at the state (or local) level, although it may be coordinated at the federal level. For example, state and local health departments

Box 3-5. Carbon Monoxide Poisoning Surveillance

Shahed Iqbal, Fuyuen Yip, Jacquelyn H. Clower, and Paul Garbe

Carbon monoxide (CO) is a colorless, odorless gas that is produced from the incomplete combustion of hydrocarbons. Major nonoccupational sources include poorly maintained and poorly ventilated home heating systems and cooking appliances, motor vehicle exhaust, and gasoline-powered or other fuel-powered equipment, such as portable generators and space heaters. Unintentional and non-fire-related carbon monoxide exposure results in nearly 450 deaths, more than 4,000 hospitalizations, and more than 20,000 emergency department visits annually in the United States.[1–3]

Symptoms of CO exposure range from minor flu-like symptoms, such as fatigue, headache, dizziness, nausea, vomiting, and confusion, to more severe effects, such as disorientation, collapse, coma, cardiac effects, and even death. Many affected people develop neurological sequelae, including impaired memory and executive functioning.

Because of its frequency, severity, and preventability—as well as the effectiveness of simple preventive measures, such as the installation of CO alarms, CO poisoning is a critical issue for public health surveillance.[4] Data for national surveillance of CO-related mortality and morbidity come from the National Vital Statistics System, the National Electronic Injury Surveillance System All Injury Program, and reports from hyperbaric oxygen treatment facilities. These data sources, however, are not designed primarily for CO poisoning surveillance. Data collected for purposes other than surveillance may suffer from limitations in timeliness, availability, completeness, data quality, and representativeness; therefore, there is need for a more comprehensive national surveillance system for CO poisoning.

Identification of more appropriate data sources and development of a national CO poisoning surveillance framework is ongoing. In addition to the existing sources, data for CO poisoning surveillance are now being drawn from the Nationwide Inpatient Sample and Nationwide Emergency Department Sample from the Hospitalization Cost and Utilization Project (HCUP). These nationally representative samples are drawn from the largest repository of hospital discharge data in the United States. For surveillance of CO exposures, the National Poison Data System, which is maintained by the American Association of Poison Control Centers, is also being utilized. This is the only national poisoning surveillance database that compiles exposure information from poison centers. Plans are being made to include CO-related health behavior questions, such as generator use and presence of working CO alarms in homes, in national surveys, such as the National Health Interview Survey, the American Housing Survey, and the Behavioral Risk Factor Surveillance System.

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Box 3-6. Environmental Public Health Tracking Network

The Environmental Public Health Tracking Network (EPHT), operated by the Center for Disease Control and Prevention (CDC), is a Web-based system initiated to track and report on environmental hazards and health outcomes that may be related to environmental factors. It presents health, exposure, and hazard information, and data from a variety of national, state, and city sources. It includes information on asthma, cancer, myocardial infraction, reproductive disorders and abnormal birth outcomes, childhood lead poisoning, and carbon monoxide poisoning.

Combining environmental and public health data, EPHT enables scientists, health professionals, policy makers, and members of the public to see where these hazards and health problems are present, to better understand the associations between the environment and its adverse effects on health, to assess unusual trends and events to determine which communities may be at risk, and to improve preventive measures. For example, parents can learn about asthma or air contaminants and take action to protect their children, such as by advocating for reducing the use of chemicals in school buildings. Elected officials can see air-quality trends in their communities to determine whether actions taken to reduce pollution levels are effective. Key features of EPHT are standardized environmental and health data for all contributing states, data organized by location, and easy-to-read maps, charts, and tables. Environmental data from EPHT include air quality related to ozone and particulate matter ($PM_{2.5}$), community water contaminants, and well water contaminants. The CDC is funding state and city health departments to build local tracking networks. Through states' participation in EPHT, data from many state surveillance activities will ultimately be available at the EPHT Web site.

The Tracking Network continues to grow as the CDC increases the types of data available and adds new capabilities. More information is available at: http://www.cdc.gov/ephtracking.

receive laboratory reports of children with elevated BLLs and collect demographic information and data on risk factors for lead poisoning during case investigations of these children. The CDC provides funds to state and local agencies to implement childhood blood lead surveillance. State and local activities focus on identifying individual children for follow-up, pointing out local sources of lead exposure, and identifying targets for intervention based on the occurrence of clusters of cases. In contrast, the role of the CDC includes tracking national

Box 3-7. Surveillance for Childhood Lead Poisoning Reveals Workplace Lead Problem

Workers exposed to lead on the job can transport lead dust home from a worksite through clothing, shoes, tools, or vehicles. In 2008, the Maine Childhood Lead Poisoning Prevention Program (MCLPPP) discovered the first reported cases of lead poisoning caused by elevated lead dust on child safety seats. During that year, MCLPPP identified 55 new cases of elevated venous blood lead levels (BLLs) (15 μg/dL or higher) among children under age 6 through mandated routine screening. (Screening requirements exist for children on Medicaid.) Children with venous BLLs 15 μg/dL and higher trigger an environmental investigation to determine the lead sources, and children are monitored until their venous BLLs are below 10 μg/dL.

Although 90% of childhood lead poisoning cases in Maine during 2003–2007 had been linked to lead hazards in the child's home, no lead-based paint, dust, or water with elevated lead levels were found inside the homes associated with six of the 2008 cases. When no lead dust was found within the children's homes, an expanded environmental investigation was conducted. In two of five homes, lead dust was detected in exterior areas where family members removed and kept work clothes, such as an entryway or laundry room. All family vehicles and all six child safety seats tested positive for lead dust.

The MCLPPP determined that the children were exposed to lead dust in the family vehicles and child safety seats. Among the five families, contacts included four persons who currently or recently worked in painting and paint removal, and one who was a self-employed metals recycler. The workers reported no lead-related occupational safety measures provided by their employers at worksites. A case of take-home lead poisoning was defined as: *(a)* a confirmed venous BLL 15 μg/dL or higher in a child under age 6 living in Maine; *(b)* a household contact in a high-risk lead-related occupation; and *(c)* environmental lead dust sampling of vehicle and child safety seat 40 $\mu g/ft^2$ or higher, with no detectable lead-based paint hazards present in the home.

During 2003–2004, 95% of reported elevated BLLs in adults were related to occupational exposures, especially in painting, the industry subsector which also had the highest number of lead-exposed workers. Both the Occupational Safety and Health Administration (OSHA) general-industry and construction lead standards require employers to provide washing, shower, and clothes-changing facilities for their employees who are exposed to lead above the permissible exposure limit (PEL). However, the parents and household contacts studied in Maine reported a lack of facilities available for washing, showering, and changing clothes before entering their personal vehicles.

Source: Adapted from: Centers for Disease Control and Prevention. Childhood lead poisoning associated with lead dust contamination of family vehicles and child safety seats—Maine. Mortality and Morbidity Weekly Report 2008; 58: 890-893.

Box 3-8. Infectious Disease Surveillance and Occupation

In the United States, surveillance systems for infectious disease usually do not include information on the individual's occupation or workplace. Pandemics, such as the novel H1N1 pandemic, point to the opportunity for surveillance systems to identify the workers most at risk for the illness, identify points of transmission to workers and other members of the public, and enable public health officials to quickly help workplaces institute preventive measures. During an influenza epidemic, health care workers are at high risk for illness, and resultant absenteeism can strain the health care delivery system. The inclusion of industry and occupation information in ongoing surveillance is helping to identify other groups of workers who may benefit most from interventions during future pandemics.

progress in eliminating childhood lead poisoning, tracking the magnitude of the problem nationally, and evaluating the effectiveness of both state and local surveillance activities.

Although occupational and environmental health surveillance systems are almost always separate, there can be value in including both occupational and environmental health within a more holistic approach to public health surveillance.[16] Occupational and environmental exposures are linked with common outcomes. Furthermore, many of the same populations, such as low-income, underserved populations, may suffer disproportionately from exposures *both* in the environment and in the workplace (Box 3-7). Even more broadly, infectious disease surveillance can sometimes benefit from an analysis by occupation, industry, or geography. Information on residence or workplace can lead to the identification of risk factors or clues to controlling the spread of transmission (Box 3-8).

EVALUATION OF SURVEILLANCE SYSTEMS

Surveillance systems should be periodically reviewed to determine whether they are serving an important public health function and whether they are operating well. It is important to consider whether a given injury or illness is important enough to remain under surveillance. An evaluation of a surveillance system includes evaluating the data collection methods (and their efficiency) and the attributes of the system, including representativeness, sensitivity, and timeliness.[17] Crucial to evaluation is an assessment of the system's usefulness by external stakeholders and partners who are responsible for prevention and intervention activities.

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The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health or the Centers for Disease Control and Prevention.

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Occupational and Environmental Health Equity and Social Justice

Sherry L. Baron and Sacoby Wilson

CASE 1

A 30-year-old man, who was a Mexican immigrant worker, began working for a temporary help agency because he was unable to find a permanent job. His work assignment changed every few months and, with each new assignment, he had to learn about a new manufacturing facility. He began working at a concrete casting company that made 8,000-pound concrete septic tanks, where he assisted a forklift driver in turning over the newly formed septic tanks. He initially received "on-the-job" training through a bilingual co-worker and communicated with the English-speaking forklift driver through hand signals. While he watched carefully to learn from others performing various tasks, he was afraid to ask too many questions because he did not want to lose his job.

One day, he was standing in the forklift driver's blind spot when a boom from the turning device fell and landed on his leg. He was taken to a hospital, where he received emergency care and was warned by physicians that, without extensive physical therapy, he would not likely regain full use of his leg. However, when he inquired with his employer about workers' compensation coverage for his medical expenses, he was informed that his employment agreement stated that he was hired as an independent contractor, which meant that he—and not the company—was responsible for any work-related medical costs. The social worker at the hospital also informed him that, since he was an undocumented immigrant, he would not qualify for any publicly funded medical assistance.

(Note: This case is based, in part, on the Massachusetts Fatal Accident and Control Evaluation [FACE] report, #02-MA-016-01.)

CASE 2

On a local television news program, a reporter provided an update about a recent health scare for rural residents living near a large industrial hog operation in eastern North Carolina. Several local physicians had noticed that many children who lived in a poor neighborhood near several industrial hog operations and attended a nearby elementary school were having diarrhea. Parents of some of the children stated that they too had been having gastrointestinal problems,

especially following recent heavy rains. Neighborhood residents had complained to the local health department and town officials about rainwater runoff from industrial hog operations and odors coming from confinement buildings that housed the hogs, but no action had been taken.

Initially, the local health department did not know why so many parents and children were sick. However, after testing local streams and residential water sources (primarily individual wells), it found high levels of *E. coli* and fecal coliforms in the well water; up to 1,000 times higher than maximum contaminant levels set by the Environmental Protection Agency (EPA). Using online mapping tools, some high school students mapped the industrial hog farms in the area and found that many were near poor and/or African American neighborhoods.

Health equity, the absence of systematic disparities in health between more- and less-advantaged groups, is a fundamental principle of justice and human rights.[1] Yet there is clear evidence of health disparities among racial, ethnic, and income groups in the United States and elsewhere. For example, the relative risk of premature death increases as family income decreases, so members of families that annually earn $20,000 to $30,000 have twice the risk of premature death compared to members of families that annually earn over $100,000. As another example, African Americans have a shorter life expectancy compared to whites, even when those with similar income levels are compared.[2] Recognizing both the importance and challenge of achieving health equity, the U.S. Department of Health and Human Services, in 2000, made the elimination of health disparities its second major goal for *Healthy People 2010*, its 10-year agenda.

Health disparities result, in part, because poor people and people of color are more likely to encounter hazards and stressors in their communities and at work.[2] Neighborhood environmental stressors include ambient air pollution, hazards from unhealthy uses of land (such as incinerators and landfills), and inadequate numbers of health-promoting facilities and resources, such as clinics, schools, and parks. Disparities in work-related exposures arise from disproportionate employment in hazardous jobs, compounded by workplace discrimination, ineffective training and safety communication due to low literacy and language barriers, and restructuring of jobs, which often creates a sense of instability and job stress.

WORKPLACE EXPOSURES AND HEALTH INEQUITIES

Over the last half of the twentieth century, the size and composition of the working population and the organization and content of work changed considerably.[3] In the United States, the workforce became more racially and ethnically diverse and older, and it gained proportionately more women. However, many permanent, full-time, often-unionized manufacturing jobs were replaced by service-sector jobs that were often temporary and often paid lower wages.

Today, almost one-third of U.S. workers have hourly wage rates so low that, even if they worked full time for a full year, their annual earnings would be below the poverty line for a family of four. Low-wage workers are more likely to be female, young, black or Hispanic, and working in an industry with a very high injury rate.[4] Although there have been many significant advances in civil rights in the United States, African Americans and members of other racial and ethnic minority groups remain disproportionately employed in hazardous jobs, while racism and other forms of discrimination—both in the community and the workplace—contribute to additional health risks.[5]

Working Women

Between 1950 and 2000, the proportion of women workers in the U.S. economy increased more than 150%.[3] Almost half of the U.S. workforce is now female, resulting in substantial new career opportunities for women. Among working women age 25 to 64, the proportion of college graduates more than tripled from 1970 to 2007. More than half of managers and professionals are now women. Women's earnings as a proportion of men's also have grown: In 1979,

the earnings of women working full time, on average, were 62% of men's; by 2007, this had increased to 80%. The remaining male-female earnings gap is due in large part to women's disproportionate employment in low-wage jobs. Of all low-wage workers, 59% are women. Compared to female workers with higher wages, low-wage female workers are more likely to be single mothers, have less than a high-school education, and be either Hispanic or African American.[6]

Overall, the rates of reported work-related injuries are lower among female than male workers, largely because women are less likely to work in the most hazardous industries, such as construction and mining (Fig. 4-1). However, in service-related occupations, women sustained 62% of nonfatal injuries while occupying only 57% of the jobs. Most nursing aides, orderlies, and attendants—who comprised the occupational group with the highest rate of work-related injuries and illnesses reported by the Bureau of Labor Statistics in 2008—are women. Assault-related injuries on the job are twice as frequent among women as men. Homicides are the second leading cause of work-related deaths among women. Those committing these homicides of women are 10 times more likely to be family members than perpetrators of work-related homicides of men.[7]

Figure 4-1. Women coal miners. (Photograph by Earl Dotter.)

CASE 3

The manager of a sausage factory reviewed the factory's annual injury logs and noted that female employees were more likely to develop musculoskeletal disorders (MSDs) than men. He recalled reading in a trade magazine that women are more likely to develop carpal tunnel syndrome, and he therefore attributed their relatively higher injury rate to biological factors. A union safety representative also reviewed these injury records and decided to investigate further. He inspected the sausage finishing station, where several of the injuries had occurred, and observed women lifting 40-pound racks of sausages onto a shelf that was designed for much taller workers. After a short discussion, he learned that these women had previously worked in evening-shift jobs that were less stressful ergonomically, but they had recently switched to day-shift jobs that were more stressful ergonomically in order to be home when their children returned from school.

This case illustrates one of the important questions for policy makers and researchers interested in women's health: What are the relative roles of biological factors and occupational exposures in explaining occupational health disparities between male and female workers?[8] For example, toxins that bioaccummulate in fat tissues could act differently in women and men, given gender differences in fat metabolism. However, the relative importance of these physiological differences, in comparison to differences in workplace exposure levels, has been inadequately studied. Most studies have not rigorously collected sufficient exposure information to adequately measure the differences in

exposure between genders, so misleading conclusions may have been drawn. As illustrated in Case 3, the design of a work station may be ergonomically optimal for the average male stature, but it may require significant reaching and awkward postures for short female workers, causing them to have more ergonomic stresses and increased risk of injury. In addition, female workers and their partners experience stress due to conflicts between work and family responsibilities. For low-wage female workers, many of whom are single mothers, the challenge of balancing their roles as wage earners and mothers is often especially stressful.

Racial and Ethnic Minority Workers

CASE 4

In 1930, a subsidiary of a large corporation contracted with a construction firm to dig a 3-mile tunnel through a stone mountain in West Virginia in order to divert the New River and build a hydroelectric energy plant. This project employed thousands of workers, at least 75% of whom were black, in a county whose population was 85% white. Many of these workers came from Alabama, Virginia, North Carolina, and South Carolina, where work was hard to find during the Great Depression and to whom the hourly wage of $0.30 to $0.60 seemed like good pay.

The rock through which they drilled had some of the highest known content of silica. To complete the job quickly, they chose to use minimal water to suppress dust levels. About 1 year after the project began, the local newspaper published a story commenting on "the unusually large number of deaths among the colored laborers. The deaths total about 37 in the past two weeks." Although the initial deaths were attributed to black workers' poor nutritional habits and unusual susceptibility to pneumonia, it soon became clear that they were dying of acute silicosis. As many as 581 of the 922 black workers who worked in the tunnels for at least 2 months of the 24-month project may have died.[9]

Although this disaster, known as the Gauley Bridge Disaster, occurred many years ago and such blatant discrimination against African American and other minority workers is far less common today, many economic and social disparities persist. (See Chapter 18 for a discussion of silicosis.) African Americans and Hispanic workers are more likely to be employed in occupations with higher injury rates (Fig. 4-2). For example, African American men are twice as likely as non-Hispanic white men to work in service and blue-collar occupations, such as laborers, fabricators, and operators, and they are half as likely to be in managerial or professional occupations. African American workers have higher rates of fatal and nonfatal occupational injuries compared to non-Hispanic whites.[10,11] For example, in Massachusetts, African American workers were found to be almost twice as likely as white workers to be hospitalized for a work-related amputation.[11]

In 2000, there were 36 million Hispanics in the United States, 58% more than in 1990, mainly due to increased immigration. During the 1990s, more immigrants entered the United States than during any other decade—about 1 million each year. Hispanic workers comprise about 13% of all U.S. workers, more than half of them immigrants, mostly from Mexico. Hispanics are more likely to work in blue-collar jobs in the service, construction, and other industries, and in farming, forestry, and fishing. Central American and Mexican immigrants, especially those who have been in the United States for less than 10 years, are most likely to work in these sectors.[12] The fatal occupational injury rate for Hispanic workers exceeds that of all other groups of workers—in 2006, it was 25% higher. In 2006, foreign-born Hispanic workers had a fatal occupational injury rate 70% higher than that of native-born Hispanic workers.[13] Hispanic workers also have higher rates of nonfatal occupational injuries than other workers. For example, male Hispanic workers in New Jersey were found to have been hospitalized more often than non-Hispanic workers for work-related falls, motor vehicle accidents, injuries from being struck by objects, and accidents related to machinery.[14]

Young Workers

In many parts of the world, child labor is widespread and children often work in dangerous

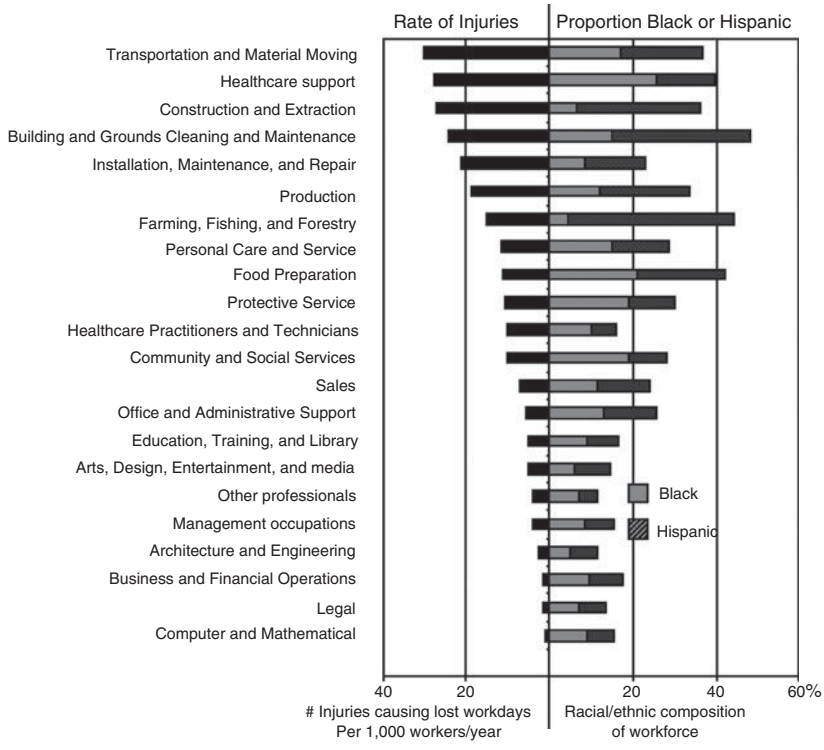


Figure 4-2. Racial and ethnic composition of occupations and job injury rates.

conditions (Box 4-1 and Fig. 4-3A). (See Special issue: Child labor and protecting young workers around the world. International Journal of Occupational and Environmental Health 2010; 16:103-237.) However, in the United States and other developed countries, since passage of strong federal child labor laws in the 1930s, exploitative child labor has been rare. However, youth employment is extremely common (Fig. 4-3B). An estimated 44% of 16- and 17-year-olds work sometime during the school year, and up to 80% of teenagers work sometime during their high school years.[15] Whether the exposure comes from the general environment or the workplace, children may face disproportionate risk (Box 4-2).

Box 4-1. Child Labor

Susan Gunn

In 2008, there were 305 million children under age 18 engaged in some form of work. For many of these children, work was age appropriate and in line with international law on minimum age (ILO Convention 138 concerning the Minimum Age for Admission to Employment). An estimated 215 million children (14% of all children in the 5-17 year age group), however, were engaged in *child labor*—the work they were doing or the conditions in which they worked posed a real danger to their physical, mental, and social health or development. Of particular concern is that between 2004 and 2008, the number of children age 15 to 17 engaged in hazardous work increased from 52 to 62 million.

Almost all countries have child labor, most often in the informal economy, such as in small workshops, eating places, and family farms where workers are not unionized and which labor inspectors seldom visit. The largest proportion of working children age 5 to 17 (60%) is in agricultural work. Twenty-six percent is in services—mainly children working as domestic servants.

Children are at higher risk of occupational injuries and illnesses than adults because they have proportionately more skin surface area, deeper breathing, less mature nervous and reproductive systems, and less developed judgment. In 2007 in the United States, 38 children under age 18 died of work-related injuries and more than 150,000 suffered from work-related injuries or illnesses. Data for developing countries are less reliable, but rates of work-related injuries and illnesses in children in these countries

(*Continued*)

Box 4-1. Child Labor (Continued)

are probably much higher than in the United States and other developed countries.

Throughout the world, the adverse health effects of child labor can be very serious. For example, many of the estimated 1 million children who work in mining and quarrying suffer neurological problems from handling mercury (used to extract gold from rock) and musculoskeletal disorders from carrying heavy loads of rocks and stones. In small auto repair shops, children work under precariously balanced cars and handle lead-based compounds or organic solvents. In agriculture, children are injured by farm equipment and exposed to pesticides. As domestic servants, children may work 16 or more hours a day, suffering not only from fatigue but also from isolation, beatings, and sexual abuse.

The emotional impacts on children of job stress caused by the speed of production, repetitive work, violence, and intimidation are of as much concern as physical impacts (see Chapter 14). For example, an ILO study in Cambodia on the adverse effect of work on children's health found that each additional hour of work per week increased the probability of injury or illness by about 0.3%. The psychosocial impacts of work on children's health, however, remain inadequately researched. There need to be better methods for population-based research on such issues.

Child labor is not only a danger to children's health and education. It also locks families into an ongoing cycle of poverty, and it hinders achievement of national development goals. Concerned about the persistence of child labor and the emergence of new forms, the International Labor Organization (ILO) launched a major campaign to eliminate the worst forms of child labor by 2016. Over 80 countries have now established programs to address child labor, and public attitudes have markedly shifted from indifference and denial to active concern.

Health workers can help to maintain this concern and address child labor issues by doing the following:

1. Documenting the problem by being alert to work-related injuries and illnesses in children. For example, Brazil has pioneered an innovative occupational safety and health surveillance system, training thousands of primary health care workers to recognize and report occupational injuries and illnesses among children.
2. Testing practical ways of reducing risks so that children of legal working age can work safely. These young people need to receive training on health and safety, and employers need to be made aware of how vulnerable young people are to occupational injury and illness.
3. Serving as a resource to schools and vocational training programs in disseminating information on occupational hazards and ways of reducing them, as well as on young workers' rights.
4. Participating in processes to establish, legally mandate, and regularly revise lists of hazardous work prohibited for children under age 18. These lists are required of countries that have ratified ILO Convention 182 (Convention Concerning the Prohibition and Immediate Action for the Elimination of the Worst Forms of Child Labor). Almost all countries have ratified the Convention. These processes provide excellent opportunities for health workers to collaborate with trade unions, employers, and labor inspection services in working toward the common goal of protecting children from abusive work.

Further Reading

The following Web sites of the ILO accessed on August 6, 2010, offer much useful information:

Action against child labour: IPEC Highlights 2008. Available at: http://www.ilo.org/public/libdoc/ilo/P/09322/09322(2008)highlights.pdf

Combating trafficking in children for labour exploitation: A resource kit for policy makers and practitioners. Available at: http://www.ilo.org/ipecinfo/product/viewProduct.do?productId=9130

Modern policy and legislative responses to child labour. Available at: http://www.ilo.org/ipecinfo/product/viewProduct.do?productId=8192

Child labour: A textbook for university students. Available at: http://www.ilo.org/ipecinfo/product/viewProduct.do?productId=174

Eliminating the worst forms of child labour: a practical guide to ILO Convention no. 182. Available at: http://www.ilo.org/ipecinfo/product/viewProduct.do?productId=1200

Accelerating action against child labour: Global report. Rights at Work. Geneva, Switzerland: ILO, 2010. Available at: http://www.ilo.org/wcmsp5/groups/public/---dgreports/---dcomm/documents/publication/wcms_126752.pdf

Safe Work for Youth kit (Packet for Administrators; Packet for Employers: Keep Them Safe; Packet for Youth: Stay Safe) http://www.ilo.org/ipecinfo/product/searchProduct.do?userType=3&type=normal&title=safe%20work%20for%20youth&selectedSortById=4&createdMonthFrom=-1

In 2007 in the United States, approximately 2.6 million adolescents age 15 to 17 worked, most commonly in food services (37%) and retail trade (24%). While employment provides many benefits to youth, including increased self-confidence, job skills, and income, it also poses potential hazards. In 2007 in the United States, 38 young workers under age 18 died from occupational injuries.[16] In 2006, an estimated 4.2 injuries occurred per 100 full-time workers age 15 to 17.[17] The total societal cost of these injuries may have been considerable, given the potential for long-term impairment, high medical expenses, school absences, and parents' lost workdays.

Figure 4-3. In both developing and developed countries, young-child and adolescent workers face multiple hazards: (A) Young girl working as a carpet weaver in India. (Photograph by David L. Parker.) (B) Teenage short-order cook in the United States. (Photograph by Earl Dotter.)

Box 4-2. Children as a Special Population at Risk for Environmental Hazards

Adam Spanier

One summer afternoon, a frantic mother brought her 5-year-old son to an emergency department for evaluation. "He was just out playing in the barn," she told a physician there. "When I went to check on him, he was sweating, confused, vomiting, having difficulty breathing, and had wet himself." The physician noted a decreased heart rate, decreased blood pressure, and excessive tearing of his eyes. From a detailed environmental history, he learned that the child lived on a farm and was exposed to an organophosphate pesticide. He removed the child's clothing to decrease any continued exposure, asked the nurse while wearing gloves to bathe the child, and treated him with pralidoxime (2-PAM) and atropine.

Children are not just small adults. There are many reasons why a child's risk of environmental exposure differs greatly from that of an adult. Children may be particularly vulnerable to a specific chemical. For example, in this case the likelihood of unintentional exposure to pesticides is higher in children than adults, and the dose needed to produce equivalent symptoms is lower in children than adults. Each of the stages of child development holds unique health risks from various environmental exposures.

Anything that may interfere with development of the fetus, which is undergoing rapid growth and organogenesis, can cause serious long-term effects. Low-molecular-weight compounds (such as carbon monoxide), fat-soluble compounds (such as ethanol and polycyclic aromatic hydrocarbons, or PAHs), and some heavy metals (such as lead and mercury) can cross the placental barrier. During fetal development there are specific periods of elevated risk during which organs are developing. During these periods, some environmental exposures, some medicines, and use of tobacco, alcohol, and recreational drugs can lead to devastating results. For example, thalidomide can cause severe birth defects of the limbs, ethanol can impair brain development, and diethylstilbestrol (DES) can later cause vaginal cancer and other reproductive system defects.

Children may face numerous other environmental risks. Breastfed children may be exposed to toxic chemicals in milk, such as pesticides, lead, mercury, nicotine, and polychlorinated biphenyls (PCBs)—and their metabolites as well as medicines that a nursing mother has taken. In addition, infant formula mixed with tap water may contain toxic contaminants from the formula or the tap water.

Toddlers, who have increasing mobility and persistent mouthing behaviors, may ingest toxins in their environment, such as pesticides, lead (in house dust), and arsenic (in treated wood). Since they are close to the ground, they are more likely to breathe heavier airborne particles,

(*Continued*)

Box 4-2. Children as a Special Population at Risk for Environmental Hazards (Continued)

such as some airborne allergens and mercury. In addition, children generally have higher respiratory rates than adults and are therefore exposed to more airborne toxins, such as environmental tobacco smoke. Since children ingest, per body weight, more water and food than adults, they are at increased risk of ingesting contaminants of water and food, such as pesticides.

Children also have larger body surface-to-mass ratios than adults, so dermal exposures to hazardous substances that are absorbed through the skin, such as organophosphate pesticides, may pose proportionately more risks for children than for adults.

Children are at increased risk of physical injury from a variety of hazards, including open windows, swimming pools, stairs, roads, and pots of boiling liquid on stovetops.

School may present new hazards for children. For example, some schools are built on property that is less than desirable. In Cincinnati, a school was built on a former shooting range and the schoolyard was found to have elevated soil lead levels, likely due to use of lead shot. As another example, most schools use pesticides, most of which have not been tested for adverse neurodevelopmental effects.

Adolescents often take more risks than adults, so they face threats such as motor-vehicle, gun-related, and other injuries, and exposure to environmental toxins, such as cigarette smoke. Adolescents who work are at increased risk of occupational injuries.

The Association of Occupational and Environmental Clinics (http://www.aoec.org) has established a network of Pediatric Environmental Health Specialty Units throughout North America to provide education and consultation for health professionals concerning the impact of the environment on children's health.

CASE 5

A 16-year-old boy was anxious, but excited, as he began his first job at a neighborhood hamburger restaurant. His job was to clear tables, wash dishes, and clean the counter. He had hoped that, by the end of the school year, he would have learned enough to become a "fry cook" and earn an extra $1 per hour. One Saturday, a cook had to leave early, and he eagerly volunteered to help close the grill. He had watched the cook do the necessary tasks for months and felt confident that he knew what to do. One task was to empty the grease from the deep fryer. He grabbed a container—not realizing it was the refuse container for the meat scraps and would melt when filled with hot grease—and emptied the hot grease from the fryer into it. As he walked out to the dumpster, the hot grease burned a hole in the bottom of the container and fell onto his legs, causing severe burns.

This case demonstrates some characteristics of young workers that raise concerns about their safety and health. Like all new workers, young workers are at increased risk for injury. Since the level of physical and cognitive development varies among teenagers, developmental characteristics may also place them at risk. Shorter teens may have difficulty reaching machines and may not have the physical strength required for certain tasks. Even when young workers have reached adult stature, their psychological and cognitive maturity may lag behind in conventional wisdom or ability. Employers may assign them tasks to which they are not yet cognitively prepared. Their enthusiasm and desire to do well—positive attributes—may make them uncomfortable asking questions or expressing concerns about their ability to perform a challenging task.

In addition to the specific hazards young workers may face, there may be unintended consequences affecting their ability to function and succeed in their school and social lives. Using a relatively arbitrary cutoff, policy makers and researchers divide youth labor into high-intensity labor (those who work more than 20 hours per week) and low-intensity labor. Low-intensity labor is associated with future postsecondary education, but high-intensity labor is associated with substance abuse, inadequate sleep, and less eventual educational attainment.[15] Between 1997 and 2003, 23% of high school freshmen and 75% of high school seniors in the United States worked at some time during the school year, and about 25% of working freshmen and 56% of working seniors worked 21 or more hours, on average, per week.

The Fair Labor Standards Act of 1938 includes protective legislation for young workers. The Act empowers the U.S. Department of Labor to establish *(a)* specific rules pertaining to child

labor, which include limits on the hours of work for children under age 16, and *(b)* documents called *hazardous orders* that identify certain tasks, such as operating power-driven woodworking equipment, that cannot be performed by youth under age 18 in nonagricultural work (or under age 16 in agricultural work). However, violations of these rules are common. A nationwide survey of teen workers found that over one-third of workers under age 16 worked past 7 p.m. on school nights, which is prohibited by law. More than 50% of male and 43% of female teen workers reported having performed one or more of five hazardous tasks that are prohibited by the federal government.[18]

Older Workers

The average age of U.S. workers is increasing due to 80 million "baby boomers," who were born between 1946 and 1964, and a decreasing U.S. fertility rate. Between 1990 and 2000, the number of U.S. workers age 25 to 44 did not change, but the number age 45 to 64 increased by more than 12 million. People age 55 and older account for almost 19% of the workforce—the highest share held by this age group since the Department of Labor started reporting labor-force statistics in 1948.

Older workers, on average, are retiring at later ages. About 16% of people age 65 and older were in the U.S. workforce in 2008, compared with 14% in 2003.[19] This trend may be due to *(a)* changing policies regarding Social Security and the restructuring of many pension programs, which has caused workers to delay retirement; and *(b)* the return of some retired people to part-time employment, for financial and/or social needs. The economic recession, which began in 2007, reduced the value of many retirement funds, leading older workers to delay retirement. Those workers who are forced to delay retirement because of financial necessity appear more likely to have depression than those who continue working for personal fulfillment.[20]

As health researchers and policy experts explore the impact of the increasing age of the workforce, two major concerns are the effects of aging on health and working capacity, and the effect of working on the aging process. Although these effects may be partially dependent on a worker's job, researchers are examining the relative importance of *(a)* physiological and cognitive deterioration associated with aging, and *(b)* positive attributes of older workers' experience and expertise.[21] (See Chapter 38.)

Nonfatal occupational injury rates decrease with age, possibly due to job selection factors, improved vigilance and work experience, and/or changes in injury reporting patterns. However, when injuries occur in older workers, they are more severe than in younger workers, as measured by lost workdays. In addition, the fatal occupational injury rate in older workers is higher than in younger workers.

One specific work-related injury—falls on the same level—provides an example of how age can affect the severity of work-related injuries. About 14% of work-related injuries result from these types of falls. For most workers, these falls usually result in mild bruises, sprains, or strains, but, for older workers, 30% of these falls result in a fracture and these falls are five times more likely to be fatal—usually due to head trauma—than in younger workers.[22]

CAUSES OF WORK-RELATED HEALTH INEQUITIES

Disproportionate Exposures

Inequities in work-related injuries and illnesses result from inequities in workplace exposures. Minority and low-income workers and others are disproportionately employed in occupations known to have more work hazards, and they therefore suffer disproportionately higher rates of adverse health outcomes. Disparities in rates of reportable work-related injuries for African American and Hispanic workers, as compared to white workers, are largely due to their different employment patterns.[23] Differences in exposure to physical and psychosocial stressors have explained apparent disparities in injury rates between hospital workers with the highest incomes (such as administrators and professionals) and those with the lowest incomes (semiskilled workers).[24]

Disproportionate employment, however, does not explain all occupational health disparities. For example, Hispanic construction workers have a higher risk of dying from a work-related

injury than non-Hispanic workers in the same job category.[25] Similarly, African American workers in the South have a higher work-related fatality rate compared to African American workers living elsewhere in the United States, even after accounting for differences in regional employment patterns.[26] Therefore, well-documented disparities in work-related injuries and illnesses by race, ethnicity, gender, and social class arise not only from inequities in job opportunities but also from coexisting social, political, and economic factors.[2] Several of these are further described in the next section.

Workplace Injustice

Workplace injustice, including abuse, mistreatment, discrimination, and harassment, has been linked to mental and physical health problems; and it accounts for some work-related health inequities. For example, workplace discrimination, based on race, gender, age, or sexual preference, can occur in many forms, including preferential hiring, firing, or job placement, as well as co-worker or supervisor hostility—all of which can cause job stress and chronic physical and mental health problems. (See Chapter 14.) One manifestation of such discrimination is the wage gap between white and black workers. In 2005, the median hourly wage for black men in low-status jobs was $10.23, compared to $13.08 for white men—a gap that persisted even after accounting for worker, job, and employer differences.[27] In 2004, 39% of African Americans stated that race and gender discrimination was widely practiced at their workplaces.[28]

Beyond its psychological toll, workplace discrimination may lead to differential exposure to workplace physical or chemical agents. Racial attitudes can also interfere with important worker-to-worker communication of safety advice, especially for new employees. Discrimination in job placement can mean that less-favored workers are assigned to more hazardous work tasks. For example, a study in North Carolina of immigrant poultry workers from Mexico and Central America found an association between retaliatory behavior by supervisors and a 10%–30% increase in adverse health outcomes. Workers reported that native-born workers were given the easier and cleaner jobs and that undocumented immigrants were more frequently

Figure 4-4. Worker in a commercial laundry. (Photograph by Earl Dotter.)

asked to work unpaid overtime or, if they refused, were assigned unpleasant tasks.[29]

A population-based study of workers in the United Kingdom examined the association between experience of workplace discrimination and mental health problems in six ethnic populations as compared to a white, nonethnic population. It found that experiences of racial insults (both at and outside of work) and unfair treatment at work were associated with common mental health disorders.[30] When workers experience discrimination in one job, the psychological effects can follow them into future jobs as they develop heightened "vigilance" and anxiety because of their previous experiences of discrimination.[31] The psychological toll of workplace discrimination can also have a negative impact on workers' family members, who experience greater psychological distress.[31]

Some workers also commonly experience other forms of injustice in the workplace. For example, more than two-thirds of 1,200 unionized low-wage workers in various workplaces reported workplace abuse, most commonly "being screamed or yelled at," perpetrated by co-workers or supervisors. Racial discrimination was reported by 58% of workers of color compared to 37% of white workers.[32] These experiences of workplace injustice contribute to disparities in injuries and illnesses. In a study of British civil servants, some of the gradient in cardiovascular disease between high- and low-status workers was due to differences in indicators of workplace justice, such as being unfairly criticized or receiving inconsistent or insufficient information from supervisors.[33]

Another form of discrimination at work is workplace segregation, in which one group of workers is disproportionately working—and sometimes feeling stuck—in certain jobs. This segregation is most apparent in many lower-status jobs (Fig. 4-4). For example, in 2008, 42% of bus drivers and security guards and 48% of nursing and home-health aides were African American or Hispanic. African Americans, especially those in the middle class, who perceive that they are in a "black job" experience greater psychological distress.[34] African American and white workers, who worked in jobs having more than 20% African American workers were found to report poor or fair overall health more often.[35] Workplace segregation also affects others. For example, 90% of health care support workers are women, but over 90% of construction workers are men. When women take jobs in traditionally male occupations, they can face discrimination and harassment (Box 4-3).

Globalization and the Rise of Insecure Work

Globalization, the worldwide movement of goods and services, capital, technology, and labor, has profoundly changed the character of work everywhere. Corporations have reorganized both industrial production and provision of services so that they now extend across multiple national borders. This restructuring of the global economy has had significant impacts on

Box 4-3. Women Construction Workers: An Example of Sexual Harassment in the Workplace

Sexual harassment of women workers, in the form of gender stereotyping, sexist jokes, and demeaning behavior remains a problem, and it has been associated with both mental health problems, such as depression and anxiety, and physical health problems, such as high blood pressure. Although present in many sectors of the economy, some very clear examples have been documented in traditionally male-dominated occupations, such as construction work. Researchers found the following comments in focus groups with women construction workers.

Regarding personal protective equipment:

"They gave me a welding leather jacket that was a foot longer than my hand…and they said they couldn't order anything smaller. They gave me gloves, humongous, I couldn't even pick anything up."

Regarding the need to prove themselves:

"…a lot of times, I feel like I've got to do this because I'm a girl because if I don't they're going to say, 'See, whad I tell ya, she's a girl, she can't lift it."

Regarding issues related to misperceptions about sexual interactions:

"(The foreman) hired her very quickly. Until the wife showed up. And then it changes…she got every dirty job that was there. He more or less forced her to quit."

Adapted from Goldenhar L, Sweeney MH. Tradewomen's perspectives on occupational safety and health: a qualitative investigation. American Journal of Industrial Medicine 1996; 29: 516–520.

the organization of work that can affect workers' safety and health.[36] To compete more effectively, many companies have reorganized by downsizing workforces, increasing reliance on temporary and contractor-supplied workers, and adopting more flexible and "lean-production" technologies.[37] Globalization has also resulted in the export of hazardous substances from developed to developing countries (Box 4-4).

Even before the global economic recession that began in 2007, workers increasingly experienced downsizing and corporate reorganizations that created a fear of layoffs—job insecurity.[38] In the United States, employment in the manufacturing sector has steadily declined—from 22% of jobs in 1979, to less than 10% in 2008. Many of these manufacturing jobs, such as in the automobile industry, were well-paid jobs, with good

Box 4-4. The Export of Hazard

Barry S. Levy

The export of hazardous substances from developed to developing countries continues with relatively few restrictions. Factors in developing countries that enable this practice include their limited financial resources, limited numbers of experts in occupational and environmental health and related fields, limited enforcement of lax regulations, workers' desperation for jobs, and government's desperation for economic development. Major categories of exported hazards include pesticides and other chemicals, asbestos, tobacco products, hazardous waste, and potentially hazardous medications—as well as hazardous industries.

The use of many pesticides—herbicides, insecticides, fungicides, and rodenticides—is banned or restricted in many developed countries, including France, Germany, the United States, Great Britain, and Switzerland. Nevertheless, these are the leading countries that produce and export pesticides.

Despite various national and international attempts to ban or significantly reduce the export of pesticides, it has continued. As examples, the United States banned DDT from export in 1972, but export continued for another 20 years, and it banned ethylene dibromide (EDB) from export in 1982, but export continued for another 14 years. In 1989, two United Nations agencies, the Food and Agricultural Organization and the United Nations Environmental Program, adopted a policy of prior informed consent by the importing country; however, compliance has been voluntary under this policy.

In 1994, $700 million worth of pesticides banned in the United States were sold to other countries. Overall, the United States annually exported 683 million pounds of pesticides in 1992–1996 and 803 million pounds in 1997–2000. Between 1997 and 2000, the United States reduced its export of "severely restricted" pesticides from 7.6 million pounds in 1997 to 4.7 million pounds in 2000. However, during the same period, it increased its annual export of "never registered" pesticides from 10.5 million to 11.2 million pounds. As specific examples, during this period it increased its annual export of aldicarb from 4.7 million to 8.9 million pounds, and its annual export of paraquat from 1.1 million to 2.7 million pounds. During the same period, the United States totally eliminated its export of methyl parathion, Lindane, captafol, and mirex. Many of the banned or restricted pesticides are carcinogens, teratogens, endocrine disruptors, and other types of toxins.[1]

During the 2001–2003 period, U.S. export of banned and restricted pesticides decreased. However, exports still included more than 27 million pounds of pesticides that are banned for use in the United States. These exports included more than 500,000 pounds of known and suspected carcinogens, most of which were exported to developing countries.[2]

The following recommendations have been made to reduce the export of banned and restricted pesticides:

1. "Aggressive efforts should be made to implement alternatives to chemical-intensive agriculture.
2. Exporting countries should assume [a] proactive, precautionary stance in regard to pesticides.
3. The quality and quantity of information regarding pesticide production, trade, and use must be improved.
4. Hazardous pesticides should be phased out when safer alternatives exist."[2]

The Stockholm Convention on Persistent Organic Pollutants (the Global POPs Treaty) initially targets the elimination of 12 high-priority chemicals (mostly pesticides), including dioxins, polychlorinated biphenyls, DDT, Lindane, paraquat, pentachlorophenol, and aldicarb.[3] This treaty was signed in 2001 by more than 100 countries; as of May 2009, a total of 162 countries and the European Union, but not the United States, were parties to the Convention.

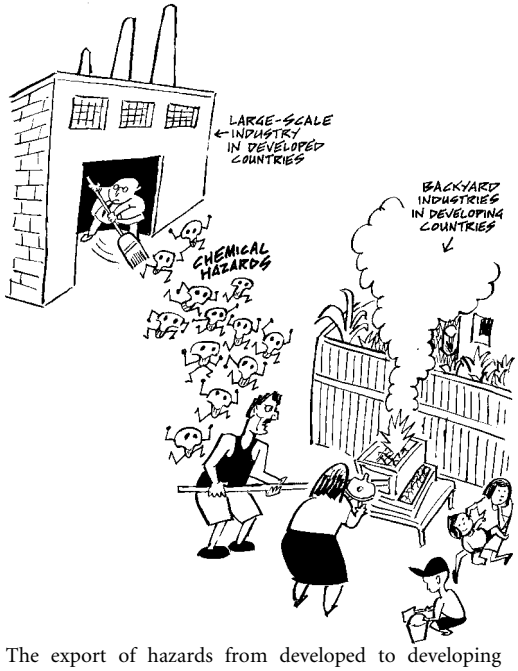
The export of asbestos and asbestos products to developing countries remains a major problem.[4] The leading asbestos producers, including Russia, China, Kazakhstan, Canada, and Brazil account for most of this export. Together, these five countries account for approximately 90% of asbestos production worldwide. The United States ceased production of asbestos in 2003, but it continues to export almost 400 million of asbestos products annually.

For most of the twentieth century, the United States accounted for huge amounts of export of tetraethyl lead. This chemical was developed in 1928 to boost octane levels in gasoline. Starting in 1973, worldwide use of this chemical decreased, with the phase-out of leaded gasoline. Current production of tetraethyl lead is still 50 million pounds annually, but use is declining by about 20% per year as countries phase out or reduce the content of lead in gasoline.[5]

(*Continued*)

Box 4-4. The Export of Hazard (Continued)

The export of tobacco products from the United States and other developed countries to less developed countries, especially China, Middle Eastern countries, and countries in Central and Eastern Europe is a major problem.[6] Various measures have been taken to reduce demand for tobacco products, including price and tax measures, protection from exposure to tobacco smoke, regulation of contents and disclosures, packaging and labeling, education and awareness raising, and smoking cessation activities. In addition, measures have been taken to reduce the supply of tobacco, including reduction of illicit trade, prevention of tobacco sales to and by minors, and support for economically viable alternative activities. The WHO Framework Convention on Tobacco Control, which was the first treaty negotiated under the auspices of WHO, reaffirms the right of all people to the highest standard of health and asserts the importance of strategies to reduce both the demand for tobacco products and supply of these products.[7] As of January 2010, a total of 168 countries, not including the United States had signed the Framework.



The export of hazards from developed to developing countries causes multiple problems. (Drawing by Nick Thorkelson.)

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benefits and pensions. In 1984, about one-half of all 40–45-year-old workers in the United States had worked for the same employer for at least 10 years, but by 2008 this had decreased to one-third.

To maintain productivity levels and compete more effectively, employers have implemented new methods of organizing workflow. For example, "just-in-time" warehousing and production methods take advantage of improved communication, transportation, and inventory systems to speed the flow of parts and supplies—intensifying the pace of work.[39]

Employers have also increased the use of *temporary and contract workers*, also known as *contingent or precarious labor*.[38,40] Corporations, by employing temporary workers, drive down labor costs by reducing employment during periods of low production. In this way, they reduce costs of paying for workers' benefits, such as health insurance and pensions.[40] Temporary employment arrangements also allow companies to recruit and screen new employees by contracting with temporary-help companies for short-term assignments, during which they can identify the most desirable candidates for longer term jobs.

Yet all of these attributes that make temporary workers attractive to employers can also make temporary work more hazardous.[40,41] Increased injury and illness rates have been attributed to increased workloads, longer working hours, decreased training, and breakdown in workplace communication. For example, temporary workers have little input into their working conditions and some, especially those working for temporary-services agencies, may not even know their employers.[37] Pressure to maximize output

and minimize time can lead temporary workers to cut corners and take greater risks. A study found that contingent workers have less knowledge of their work environment and less job training, and they believe that it is difficult to criticize working conditions and to get their views heard by management.[42]

Contingent workers are those who are not employed full time and long term by a single employer. In 2005 in the United States, there were about 43 million contingent workers—about 31% of all workers.[41] Contingent workers span all economic strata and include some who choose to work in more flexible or part-time arrangements. Contingent workers are twice as likely as standard full-time workers to have annual family incomes below $20,000, and more than 80% less likely to have employer-provided health insurance. Contingent workers are also more likely to be young, female, and African American or Hispanic.[43] Many are not protected by key workforce protection laws that are designed to ensure proper pay and safe, healthful, and nondiscriminatory workplaces.[41] The trend toward use of contract labor is not limited to the private sector. Between 2000 and 2006, the number of federal contract workers in the United States increased from 1.4 to 2.0 million; by 2006, 43% of employees who performed work for the federal government were employed by *private* businesses.[44]

Temporary workers have higher rates of mental health problems, especially depression; musculoskeletal disorders; and both fatal and nonfatal work-related injuries.[38] In Washington State, temporary workers have had higher rates of work-related injury and illness claims than those employed in standard work arrangements.[45] In Spain, temporary workers have experienced an almost three-fold greater rate of nonfatal work-related injuries than permanent workers.[46] Official government records of occupational injuries and illnesses are likely to underestimate the actual numbers among temporary workers because of barriers to recognizing, reporting, and recording them by workers, employers, and physicians (see Fig. 1-7 in Chapter 1). These barriers are likely to disproportionately affect low-status and temporary workers because of their job insecurity, job mobility, and lack of health insurance.[47]

Informal employment, also called the *underground economy*, is another—and perhaps the most extreme—form of contingent employment. The International Labor Organization (ILO) defined informal employment in the 1970s as "the activities of the working poor who were working very hard but who were not recognized, recorded, protected or regulated by the public authorities." Each day, over 100,000 day laborers in the United States wait on street corners or at hiring centers, seeking very temporary employment in construction, landscaping, and moving and hauling. Like other contingent workers, informal workers have high rates of work-related injuries. There is a strong association in many countries between the proportion of jobs in the informal sector and rate of disability-adjusted life years (DALYs) lost due to all diseases.[48]

Permanent, full-time employees who remain in the workforce when companies downsize experience fears of job insecurity. Increased work-related injuries, musculoskeletal disorders, psychological distress, and cardiovascular disease have been attributed to increased workloads, longer working hours, decreased training, and breakdown in workplace communication.[38,40] Job insecurity may also lead workers to postpone necessary treatment for work-related injuries, resulting in more severe problems. A study of low-wage, African American, mainly female, poultry workers living in a rural community reported that, when one woman was advised to seek follow-up medical care for a possible musculoskeletal disorder, she was reluctant to do so because "there are 300 people in line behind me for my job."[49]

As globalization and economic restructuring have increased, the proportion of workers in unions has decreased. In the private sector in the United States, the proportion of workers in unions decreased from a high of 35% in 1950 to less than 8% in 2008. Even in workplaces that are unionized, temporary and contract workers are frequently not covered by union contracts. Unions promote workplace safety through training programs, union-management safety and health committees, and provision of protection against retaliations when workers speak out about unsafe conditions. When a union is not present, workplace safety may suffer. A study found that those states with low density of

unions and low rates of labor grievances and those states with *right-to-work laws*, which allow workers to opt out of union membership, had higher work-related fatality rates, after accounting for differences in industrial structure.[50]

International Trade Agreements

Increasing globalization of markets has emphasized the need for multinational trade agreements. Countries govern their international trade through international financial institutions and trade agreements. The contemporary system of international trade agreements began in the aftermath of World War II, when the Bretton Woods Accords were negotiated to stimulate economic growth in Japan and European countries. These accords led to the creation of the International Monetary Fund, the World Bank, and the General Agreement of Tariffs and Trade (GATT). With acceleration of global trade, the World Trade Organization (WTO) in 1994 replaced the GATT. The WTO is a more-formalized organization overseeing international trade, including more than 150 member countries and representing more than 90% of world trade. Many of its agreements are *free-trade agreements*, which remove both tariff and nontariff barriers to trade. Tariffs include taxes and other financial disincentives on imported goods that protect national industries against international competition. Nontariff barriers include rules and regulations that could limit trade, such as regulations to control environmental contamination and promote workplace safety. For example, a Canadian corporation that manufactured the gasoline additive methyl tertiary butyl ether (MTBE) filed a $1 billion lawsuit against the United States when California enacted regulations that limited use in gasoline of MBTE because of its carcinogenicity, claiming these regulations represented a nontariff trade barrier. Although the WTO permits "measures necessary to protect human, animal or plant life or health," these exceptions are difficult to implement.[51]

The North American Free Trade Agreement (NAFTA), which took effect in 1994, included special "side agreements" to address concerns about workplace and environmental protections. Trinational bodies, representing Mexico, United States, and Canada, were established to monitor progress. By 2004, seven formal complaints had been filed concerning gaps in protection of workplace safety and health. These complaints against all three countries, included claims of lack of government enforcement of workplace safety regulations, such as in factories owned by multinational corporations in Mexico and by companies employing immigrant workers in the United States. Comprehensive investigations in most of these cases identified gaps in enforcement. However, because "government-to-government consultations" either did not have or did not exercise their power to enforce changes, no actions were taken to improve workers' health and safety.[52] A recent assessment by the U.S. Government Accounting Office (GAO) of four other free-trade agreements with Singapore, Morocco, Chile, and Jordon found similar results. The GAO found that "with respect to the labor obligations the responsible U.S. agencies have made little or no effort, or a belated effort, to identify labor compliance concerns after NAFTA enactment, and engagement with these partners on labor issues has been a low priority most of the time."[53]

Migrant Labor

As international financial institutions, national governments, and corporations embraced free trade and introduced new forms of work, tens of millions of peasants and millions of workers, in search of work, began to migrate both within their own countries and also abroad. The mass migration of workers, as a result of globalization, has brought a whole new series of political economic and social challenges. The ILO estimated in 2003 that globally there were 120 million international migrant workers and family members.

In 2007, almost 13% of U.S. residents were immigrants—a 22% increase since 2000. The two largest immigrant groups are Mexicans, accounting for 31% of all immigrants, and South and East Asians, accounting for 24%. Of the 45 million Hispanics in the United States, 39% are foreign born; of the 13 million Asians and Pacific Islanders, more than 66% are foreign born. Employment profiles of immigrants vary substantially by region of birth. South and East Asian workers are overrepresented in science, engineering, and health-related occupations.

Mexican immigrants are overrepresented in many high-hazard occupations, such as jobs in construction, agriculture, food processing, food services, and cleaning and maintenance.[12]

CASE 6

A young man, in search of a job, crossed the border from his native Mexico to the United States. He had a cousin living in Los Angeles, who told him that construction jobs were easy to obtain. Once he arrived, he found a job working as a sandblaster for a small construction company, which did not ask for any official documents, and paid him "under the table." Although sandblasting creates much dust, his employer gave him no respiratory protection. To avoid breathing too much dust, he tied a bandana around his face, as farmworkers in his small home town in rural Mexico had done when they sprayed pesticides. He earned a good income and regularly sent money back to his family in Mexico. However, after a few years doing this job, he began to cough and wheeze. When he barely had enough energy to make it through the work day, he saw a doctor who diagnosed him with advanced silicosis. Unable to work and without medical insurance, he returned to Mexico and died a few years later.

Since the demand for work visas in the United States each year far exceeds the quota set by the federal government, millions immigrate for work, although they have no legal documentation. These unauthorized immigrants may be at especially high risk for work-related injuries and illnesses, as their immigration status and economic desperation drive them to take hazardous jobs that others have refused because of low pay and unsafe working conditions. The combination of language barriers, lack of familiarity with programs to protect workers, and fear of "speaking up" may compound the inherent risk in these jobs.[54] Unauthorized immigrants are two to five times more likely to be employed as agricultural or construction laborers, building maintenance workers, groundskeepers, and food preparation workers. Low educational levels and limited specialty skills further limit job opportunities. In the United States, over half of immigrant adults and three-fourths of Mexican immigrants do not speak English well. Among unauthorized immigrants age 25 to 64, almost half have less than a high school education.

A survey in five community clinics in Boston of 1,500 patients, two-thirds of whom were born in other countries, illustrates the barriers that immigrants face.[55] Three-fourths of foreign-born respondents had never heard of the Occupational Safety and Health Administration (OSHA), and more than half had never heard of workers' compensation—despite having resided in the United States for an average of 12 years. Fewer than half had received written information or training about workplace safety; for the one-third who received training or written materials, it was in a language they did not understand well. Even when safety-training materials are translated into workers' native languages, they may not effectively communicate safety messages, especially if the terms, images, and formats are not consistent with the literacy levels and cultural backgrounds of workers.

Immigrant workers' high job mobility and their desire to remain invisible make it difficult to determine their work-related injury and illness rates. The only government data on work-related injuries that include nativity are those for work-related fatalities. Between 1997 and 2001, foreign-born workers were 18% more likely to die at work than native-born workers.[56]

Unequal Access to Medical Care and Sick Leave

There are great inequities in access to medical care, especially for the working poor, who are disproportionately uninsured or underinsured, which may contribute to the severity of work-related injuries and illnesses. In 2008 in the United States, about 22% of employed adults age 18 to 64 were uninsured for at least part of the year, and an additional 14% were underinsured. Minority workers and workers employed in service, blue-collar, and agricultural jobs were those who were least likely to be insured.[57] Between 1999 and 2009, health insurance coverage decreased in the United States, and this trend

disproportionately impacted Hispanic workers, especially Hispanic blue-collar workers.[58] The proportion of contingent workers receiving health insurance is smaller than the proportion of standard full-time workers. An estimated 13% of contingent workers received health insurance through their employers in 2005, compared to 72% of standard full-time workers.[41] (It remains to be seen to what extent health reform legislation in the United States will improve these problems.)

Disparities in insurance coverage are compounded by additional inequities in access to occupational health services and workers' compensation coverage. While all workers face significant barriers to obtaining adequate coverage for workers' compensation (Chapter 31), racial and ethnic minority workers are disproportionately impacted.[59] For example, among low-wage garment workers in New York City, African American, Asian, and Hispanic workers have been more likely to be denied workers' compensation payments for carpal tunnel syndrome compared to their white co-workers.[60] Among workers who filed workers' compensation claims for low back pain, African American workers and workers of low socioeconomic status had, after settling their claims, higher levels of pain intensity, psychological distress, disability, and financial struggle.[61]

Job insecurity and fears of retaliation, such as being labeled a "careless" employee, may mean that many low-wage workers do not report work-related injuries.[47] For example, a study of hotel room cleaners in Las Vegas found that almost 20% did not report work-related injuries, many claiming that they were either "afraid" or that it was "too much trouble."[62] In some cases, medical costs are shifted to private insurance, but often workers absorb the costs themselves. For example, Hispanic construction workers were half as likely as non-Hispanic white construction workers to have a work-related injury covered by workers' compensation and four times more likely to pay out-of-pocket expenses—on average, almost $2,000.[63]

Another way that low wages influence health is through the availability of paid sick leave. When sick workers do not stay at home—a phenomenon called *presenteeism*—they and their co-workers can develop adverse health effects. And when workers cannot take time off to care for sick children, the health of their children and others in the children's schools and day care centers can be adversely affected. Low-income workers with family incomes below 200% of the federal poverty level who have children at home are less likely to have paid sick leave and less likely to take time off from work to care for themselves or sick family members compared to higher wage workers.[64]

ENVIRONMENTAL EXPOSURES AND HEALTH INEQUITIES

CASE 7

A father from a small community on the outskirts of a city testified in court about how, for the previous 15 years, a landfill near his property has adversely affected his health, his family members' health, and the quality of life in his neighborhood. He described odors from the landfill and, when the wind blew in his direction, headaches, a bad cough, and burning of his eyes, nose, and throat. He described the noise from trucks bringing garbage to the landfill and seeing rats in the woods near the landfill and buzzards flying overhead. He stated that he did not understand why more was not being done to monitor the landfill. He noticed that family members and many neighbors were sick. For 15 years, they complained to the local health department and the state environmental protection agency. They finally contacted the EPA and found out that tests of local well water 20 years before found that the groundwater was not safe for consumption because it contained high levels of metals and other contaminants that cause cancer, birth defects, and neurological disorders. The EPA stated that anyone who lived within 2 miles of the landfill should not drink well water and should tap into the closest publicly regulated drinking water system or drink only bottled water.

During testimony from town officials, the man learned that the city knew about this contamination and provided alternate water sources to people living in affluent neighborhoods near the landfill, but not to poor people, immigrants, or people of color.

When the judge questioned town officials about their actions, they stated that they disseminated public notices and held stakeholder meetings, but nobody from the man's neighborhood had responded.

This case is not unique. For over 20 years, researchers have demonstrated that many low-income populations, communities of color, immigrant communities, underserved populations, and marginalized and disenfranchised groups live in neighborhoods that experience disproportionate risks from the burden of, and exposure to, environmental hazards. These hazards include many noxious land uses, such as landfills, incinerators, publicly owned treatment works (POTWs, such as sewer and water treatment plants), industrial animal operations, Superfund sites, facilities reporting releases of priority chemicals to the EPA's Toxic Release Inventory (TRI) program, energy production facilities, chemical plants, heavily trafficked roadways, and other locally unwanted land uses (LULUs).[65-75] This disproportionate burden results in increases in exposure to adverse environmental conditions, low environmental quality, and high levels of pollution. The cumulative impact of environmental injustice, due to the spatial concentration of environmental hazards, factories, and noxious land uses, leads to increases in adverse health outcomes and community stress as well as lower quality of life and community sustainability.

In the 1980s, the environmental justice (EJ) movement emerged to address the disproportionate burden of environmental exposures on low-income and minority communities.[65,66] Concerned communities raised awareness of the many environmental and health issues that they faced and asked the federal government to respond. Two groundbreaking studies provided the initial set of evidence that supported the claims of grassroots activists who had been fighting against environmental injustice in places like Warren County, North Carolina; Oakland, California; "Cancer Alley" (a heavily industrial area along the Mississippi River in Louisiana); and Native American reservations and territories in the Great Plains, the Southwest, the South, and Alaska. The first study by the GAO in 1983, *Siting of Hazardous Waste Landfills and Their Correlation with Racial and Economic Status of Surrounding Communities*, which examined the distribution of landfills in EPA Region IV (eight southeastern states), found that 75% of communities containing large hazardous waste landfills were mainly African American. In addition, the study found that African Americans were overrepresented in communities with waste sites.[76] The second study, by the Commission for Racial Justice of the United Church of Christ in 1987 (under the leadership of Charles Lee, director of the EPA Office of Environmental Justice), *Toxic Waste and Race in America,* demonstrated that ZIP codes without a toxic facility had a population with less than 12% persons of color, those with one toxic facility had 24% persons of color, and those with multiple toxic facilities or one of the five largest landfills had 38% persons of color.[77] The major conclusion of this study was that 60% of African Americans and Hispanic Americans lived in communities with toxic waste sites.[77] In 1990, at the Conference on Race and the Incidence of Environmental Hazards, one of the first national environmental justice conferences, researchers presented results that documented and supported the conclusions of the studies cited earlier.[75]

Published in 2007, the *Toxic Wastes and Race at Twenty* report, a follow-up to the 1987 study, provided additional evidence about the disproportionate burden of environmental hazards, industrial facilities, and noxious land uses on disadvantaged populations.[71] The report demonstrated that, nationally, people of color are approximately three times more likely to live in neighborhoods that host a commercial hazardous waste facility than whites.[71] The study found that *(a)* proportionately more African Americans, Hispanics, and Asians reside in neighborhoods that host toxic facilities than in "non-host" neighborhoods, and *(b)* in metropolitan areas, proportionately more poor people live in "host" neighborhoods than "non-host" neighborhoods. There is now a large body of literature on environmental justice, which has documented the disproportionate burden on poor populations, people of color, and other disadvantaged groups of environmental hazards, unhealthy land uses, and other built-environment

problems, such as hazardous waste sites, landfills, refineries, petrochemical plants, industrial facilities, and large highways.[65,66,68,78,79] (See also Chapters 10, 33, and 38.)

Environmental Injustice

CASE 8

At a local community meeting in a poor segregated neighborhood, its primarily Latino, African American, and Asian residents discussed government plans to build another highway in the neighborhood. As a result of highways built earlier, motor vehicle traffic and air pollution increased in the community, causing respiratory problems. During summers, many "ozone-alert" days made children and elderly residents stay inside, and heat waves caused many hospitalizations for exhaustion and heat stroke. Residents complained of dirty, black diesel smoke from trucks that drove through the neighborhood and transit and school buses that idled throughout the day.

A Department of Transportation (DOT) official at the meeting stated that an environmental impact assessment of the planned highway showed that it would not increase air pollution. Town officials stated that the new highway could help promote economic development and bring in new industries, businesses, and consumer traffic. A local physician reported that many of his young patients had asthma and many of his adult patients, especially those who lived near the bus stops and highway exit ramps, were having respiratory and cardiovascular problems. Some residents, who lived near an incinerator (which was also near a middle school) that released pollutants into the atmosphere, observed that the building of highways in the neighborhood had been accompanied by the construction of polluting factories.

Asthma, a prime example of a health disparity resulting from environmental injustice,[68] is more prevalent among people of color than white people. In 2005 in the United States, the following disparities were present:

- Puerto Ricans had a prevalence of asthma 125% higher than non-Hispanic white people and 80% higher than non-Hispanic black people.[68]
- American Indians, Alaska Natives, and blacks had a 25% higher prevalence than whites.
- The asthma hospitalization rate for blacks was 240% higher than it was for whites.
- Blacks had an asthma mortality rate twice that of whites.

Several pollutants in ambient air appear to contribute to asthma attacks, including particles with a diameter less than or equal to 2.5 microns ($PM_{2.5}$), particles with a diameter less than or equal to 10 microns (PM_{10}), ozone, oxides of nitrogen, and sulfur dioxide (see Chapter 6). Exposure to one pollutant may exacerbate the adverse effects of another. In the ambient air, these pollutants may act synergistically to increase respiratory health disorders among exposed vulnerable populations.

There are likely multiple explanations for asthma disparities. Disadvantaged communities tend to live in areas with higher rates of exposure to environmental toxicants. Racial minorities are more likely to live in counties that exceed the 24-hour air quality standard of 65 $\mu g/m^3$ for $PM_{2.5}$. In addition, asthma is influenced by social factors. Minority and low-income communities encounter a higher burden of social stressors, including unstable employment and community violence,[80] which can exacerbate asthma and increase its prevalence—possibly with synergistic effects of environmental exposures. Disadvantaged populations also have limited access to quality medical care, including proper treatment for asthma (see Box 18-1 in Chapter 18).

Environmental factors can contribute to adverse pregnancy outcomes. Residential proximity to environmental hazards increases the risks not only for preterm birth, low birthweight, and birth defects but also for childhood cancer and autism.[81,82] Many studies that have examined the relationship between toxic exposures and birth outcomes have revealed how environmental disparities contribute to adverse pregnancy outcomes and disorders of children, including the adverse effects of place of residence on health. (See Chapter 20.)

Residential Segregation

Residential segregation leads to disproportionate exposure to environmental risk factors—physical, social, and economic—that adversely affect health and lead to health disparities in both urban and rural areas.[65-68] In many urban areas, social, economic, and political forces along with historical patterns of community development, disinvestment, industrialization, and zoning and planning (including for highway development and expansion) have segregated populations of color in impoverished communities that have few resources and increased environmental risks.[65,66] *Redlining* (the practice of denying, or increasing the cost of, services such as banking and insurance) and institutional discrimination have also contributed to segregation of disadvantaged populations.[67,69,70] In these communities, relatively few municipal services are available, infrastructure has deteriorated, and the physical and natural environments have been eroded.[80] Many segregated populations are exposed to high levels of *criteria air pollutants*, such as carbon monoxide, particulate matter, sulfur dioxide, and oxides of nitrogen, released from vehicles and factories in or near these neighborhoods.[68] (See Chapter 6.) Exposure to these pollutants can cause lung cancer or nonmalignant respiratory disorders, such as asthma.[65,66,68] For example, black-white segregation has been correlated with increased levels of sulfur dioxide, PM_{10}, and ozone in metropolitan areas.[2,66] In addition, segregation is associated with *(a)* greater exposure of populations of color to hazardous air pollutants (HAPs) and *(b)* increased risk of cancer, even after controlling for socioeconomic status (Fig. 4-5).

Segregated communities are characterized by concentrated poverty, limited economic infrastructure, and low-quality social services and medical care. These factors act synergistically to raise levels of stress, increase vulnerability, and limit capacity of burdened populations to overcome disease and increase health status.[65-68] The spatial distribution of unhealthy land uses in disadvantaged and marginalized areas, such as hazardous waste facilities, chemical plants, landfills, incinerators, sewage treatment plants, coal-fired power plants, and heavily trafficked roads, are important contributors to unhealthy environmental conditions to which segregated populations are exposed.

Figure 4-5. Children's play area near an industrial facility. (Photograph by Earl Dotter.)

Community Planning and Development

Many factors contributed to inequitable development in urban, suburban, and rural areas in the United States, including suburbanization (population movement from within cities to the rural-urban fringe, which leads to urban sprawl), discriminatory housing policies, segregation, massive highway construction, deindustrialization, and poor zoning and planning.[67,69] As a result, many areas have been divided by race, ethnicity, and socioeconomic status, creating environmental injustice. The segregation and spatial variation in planning and development in communities with different racial, ethnic, and socioeconomic composition have arisen from conditions and policies in different time periods. These conditions and policies have included *Jim Crow policies* in the South—state and local laws in the United States enacted between 1876 and 1965 that mandated racial segregation in all public facilities with a supposedly "separate but equal" status for African Americans. They have also limited access for non-whites to low-interest home loans after World War II, exclusionary zoning, racial covenants, and redlining.[69] The uneven nature of community planning, zoning, and development has led to *fragmentation* (the division of metropolitan areas into multiple smaller municipal districts), *gentrification* (the restoration of run-down urban areas by the middle class, resulting in the displacement of low-income residents), and sprawl and the spatial concentration of environmental hazards and unhealthy land uses in communities affected by environmental injustice. Spatial fragmentation and gentrification have limited sustainable economic development which, in turn, has adversely affected the quality of schools, housing, transportation, civic engagement, and social climate.

Although zoning and planning are sometimes perceived as objective processes, they are, in reality, highly political, class-conscious practices. Early in the twentieth century, zoning became widespread in the United States because it effectively regulated land use, making it difficult or impossible for less-affluent people to cross community boundaries. For example, in New York City, zoning was a social and political process, in which much of the boroughs of Bronx, Brooklyn, and Queens was zoned as unrestricted, which promoted—for economic reasons—development of hazardous industrial facilities in poor and working-class areas.[83] Zoning and race were closely related. For example, the Bronx had the highest concentration of poor and minority residents as well as large increases in areas zoned for manufacturing; in contrast, more affluent Manhattan had the greatest decrease in manufacturing.[83] Land zoned for manufacturing in the Bronx exposed nearby residents to disproportionate amounts of environmental toxins. This zoning pattern occurred in other U.S. cities, including Chicago, Atlanta, Detroit, and Los Angeles.

New movements in planning and community development, including *new urbanism* (an urban design movement that focuses on the development of walkable communities) and *smart growth* (an urban planning approach that focuses on concentrated growth, mixed-use development, compact, walkable, pedestrian-friendly, transit-oriented neighborhoods to reduce sprawl and improve neighborhood sustainability), have been adopted by planners, local government officials, architects, and environmental organizations to improve health, sustainability, and quality of life in neighborhoods, towns, and cities. Unfortunately, these movements have not gone far enough in addressing environmental injustice and social inequalities, and they may lead to more segregation, gentrification, and uneven planning, zoning, and development.[67,69] For example, the adverse social, economic, environmental, and health impacts of urban revitalization on disadvantaged populations are evident in the destruction of core urban neighborhoods in large cities and displacement of underserved and disadvantaged residents. Therefore, economically advantaged populations, who benefited disproportionately from the suburbanization movement, may benefit disproportionately from new revitalization efforts, while historically disadvantaged populations may be adversely affected.[69] Without equity-based policies, the elimination of environmental injustice and health disparities in disadvantaged communities through new forms of planning and community development may not occur.[69]

Publications on environmental justice have recognized how inequitable zoning and planning

and community development contribute to lack of access to basic amenities, such as sewer and water infrastructure, good housing stock, parks, green space, recreational facilities, and pedestrian-friendly residential environments in rural areas and small towns.[67,69,70,84] The problems of unjust transportation planning and urban sprawl have been studied in Atlanta and Southern California,[79,85] revealing how transportation inequities can contribute to environmental injustice and public health problems. There is also a high concentration of *pathogenic infrastructure*, such as fast-food restaurants, liquor stores, and check-cashing facilities, in poor neighborhoods and communities of color in Southern states, such as North Carolina, and large cities, such as Detroit.[86,87]

Many low-income populations and populations of color live in neighborhoods that are differentially burdened, due to discriminatory and exclusionary zoning, by toxic land uses and polluting industrial facilities, such as landfills, hazardous waste sites, incinerators, sewer treatment plants, TRI facilities, petrochemical plants, and large highways.[65–75,78] The higher burden of noxious land uses and pathogenic infrastructure in disadvantaged and underserved communities leads to higher exposure to unhealthy physical environments, increased health risks, poor health behaviors (such as less leisure-time physical activity and poor diets), and adverse health outcomes and health disparities for asthma, cancer, obesity, diabetes, and cardiovascular disease. Exposure to such noxious conditions has been linked to the exacerbation of preexisting health problems, asthma-related morbidity, premature adult mortality, infant mortality, low birthweight, psychological stress, and higher body burdens of toxic chemicals, such as lead.[65,67]

The Built Environment

CASE 9

A mother of three children attended a parent–teacher association meeting at a local junior high school to find out more information about its new garden. Her children came home after school a few weeks before excited about a new school program in which students would have physical activity and eat organic produce from the school's garden or the local farmers' market. At the meeting, the mother was shocked to learn that the program was established because of high rates of obesity and diabetes among students. Two of her children were overweight and one had been diagnosed with diabetes at age 10. A local professor stated that her neighborhood was a *food desert*, with no supermarkets or grocery stores and fresh fruits and vegetables available only at a gas station's convenience store. The professor stated that the neighborhood had poor access to mass transit, preventing residents from having access to supermarkets in suburban locations, and had 10 times the average number of fast-food restaurants. The mother recalled how often she bought her children hamburgers and french fries from a nearby fast-food restaurant.

In response to the professor's assertions, a community leader stated that the neighborhood was not a *food desert*, but rather that it had been impacted by environmental injustice and *food apartheid*. She said she had been working for 20 years to try to bring about better community development and more supermarkets, but that politicians countered that the neighborhood could not support a supermarket or even a medium-sized grocery store. However, she noted that some progress had been made in turning empty lots into community gardens and cleaning up many of the parks.

The lack of positive and health-promoting features in the built and social environments, which contributes to health inequalities, is a major concern for communities affected by environmental injustice.[67] For example, low-income neighborhoods, urban neighborhoods, and neighborhoods that are predominately African American have less access to supermarkets than wealthier neighborhoods, suburban neighborhoods, and those that are predominantly white.[86] The presence of supermarkets is associated with better diets and lower rates of overweight, obesity, and hypertension.[86] In many segregated and fragmented areas, the lack of health-promoting food resources creates a *food desert*, which is made worse by limited transportation opportunities for local residents. Many of

these poor segregated communities do not have access to personal vehicles or reliable public transit, which limits access to distant supermarkets. These environmental restraints and overabundance of food outlets in convenience stores and gas stations adversely affect diet, lifestyle, and risks for obesity, cardiovascular disease, and diabetes.[67,69] (See Chapter 39.)

Poor and minority neighborhoods impacted by environmental injustice are also less likely to have access to opportunities for physical activity, including green space, parks, and recreational facilities.[67,87] Even when there are facilities, other factors, such as poor neighborhood aesthetics and safety, limit physical activity in these neighborhoods. Limited access to medical care and lower quality of care adversely affect health and increase disparities in disadvantaged neighborhoods.[80] Being both disadvantaged and medically underserved means disadvantaged populations may have higher rates of chronic conditions, drug abuse, emotional problems, poor health behaviors, lower childhood immunization rates, and more hospitalizations for preventable diseases than other populations. In addition, poor and minority communities impacted by environmental injustice are also overburdened by health-restricting infrastructure with *environmental pathogens.*[67] Poor and minority communities have more retail access to fast food, alcohol, and tobacco, and are more frequently targeted by advertisements for fast food, alcohol, and tobacco.[67]

The local environment in disadvantaged communities, especially those affected by environmental injustice, has adverse impacts on quality of life, lifestyles, and behaviors. Taken together, the differential burden of increased exposure to *environmental pathogens* and decreased access to health-promoting resources have important implications for promoting public health and addressing environmental health disparities in these communities.[67] The presence of environmental pathogens in a community can limit the ability of agencies to promote public health because these pathogens may create community stress or promote negative health behaviors. In addition, these pathogens may act as sources of pollution. And, because these communities have little or no access to health-promoting infrastructure, such as parks, open space, and health care facilities, policies to reduce environmental health disparities may be unsuccessful.

COMMUNITY EMPOWERMENT: ONE APPROACH TO ADDRESS HEALTH INEQUITIES

Health inequities resulting from environmental and occupational injustice are challenging to eliminate, especially given the complex social, political, and economic forces that have created and sustained them. New approaches to public health interventions should recognize these complexities and should develop comprehensive and more effective public health prevention programs. One especially promising approach has emerged: community-driven research, also known as *community-based participatory research* (CBPR).[88–92]

With CBPR, community groups utilize their grassroots activism, resources, and expert local knowledge and collaborate with university partners to develop a framework to address environmental and occupational issues at the local level.[88–92] This approach allows for the research process to be more action-oriented, thereby increasing and sustaining the community's capacity to address justice and health issues, and increasing civic engagement by minority and low-income stakeholders.[91,92] By creating a shared responsibility for research, this approach brings equality to the relationships between local experts and academic experts, and ensures that the research is locally relevant.[88–92] Many CBPR projects also emphasize the role and participation of community youth, which creates an intergenerational pipeline of community leaders knowledgeable about these issues. The use of community-driven research methods has helped to empower communities; raise awareness about environmental and occupational justice issues at the local, state, and national level; increase environmental health literacy; and enhance building local capacity to develop sustainable prevention programs.[91,92] As described in the book *Street Science*,[90] El Puente and the Watchperson Project, two community-based organizations in Brooklyn, engaged in CBPR to address asthma and health risks from consuming subsistence diets of locally caught fish. Each organization

built its capacity to collect locally relevant data, working in partnership with scientists and receiving training in data collection methods. Similarly, the West End Revitalization Association, a community-based environmental justice organization in Mebane, North Carolina, developed a community–university partnership with researchers and students, primarily from the University of North Carolina at Chapel Hill. It developed its own research framework and received training on environmental health issues and data collection methods to build community capacity to address environmental health issues associated with infrastructure disparities at the local level.[91,92]

The success of this community-driven approach has also been demonstrated in an evaluation of Partnerships for Communication, a long-term initiative in which the NIEHS, NIOSH, and EPA funded 54 environmental justice and occupational justice projects that addressed exposures in urban and rural communities. Each project required collaboration among a research organization, a community-based organization, and an organization of health care providers. Some projects addressed environmental justice concerns, including exposures from hazardous waste sites, industrial animal operations, water and air pollution, uranium, and pesticides. Others addressed occupational justice concerns, including exposures to lead, organic solvents, pesticides, and other chemicals, among various types of workers, including day laborers, nail-salon workers, floor refinishers, farmworkers, and domestic workers. The initiative also created programs to address workers' rights and language barriers for Asian and Hispanic immigrant workers in meatpacking, agricultural production, and restaurant work.

The evaluation of Partnerships for Communication projects found that they were remarkably successful at developing community training and education programs, creating sustainable community leadership, and producing many new and innovative mass-media campaigns.[93] Many positive public health and public policy impacts were documented, including reductions in community exposures through changes in laws and regulations, changes in government planning and zoning, and adoption of new work practices by employers. For example, some projects led to legislation to control diesel emissions from idling school buses, to prohibit plating operations that used hexavalent chromium from locating in residential or mixed-use neighborhoods, to stop permitting for landfills, and to close a medical waste incinerator. Other projects created safer work environments by successfully promoting the substitution of safer chemicals, by collaborating with a manufacturer to market blueberry rakes that were ergonomically sound, and by developing linguistically and culturally appropriate worker training programs that were adopted by employers.

CONCLUSION

The ambitious goal of eliminating racial and ethnic health disparities, set by the U.S. Department of Health and Human Services in 2000, has yet to be achieved. However, the attention given to these disparities by researchers, public health workers, and communities has led to a clearer understanding of the complex and deeply rooted social and economic factors that sustain these inequities, including those resulting in disproportionate occupational and environmental exposures. Eliminating these disparities will require the commitment not only of public health workers but also of policy makers and actively engaged community members to create a more just society.

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The John D. and Catherine T. MacArthur Foundation Research Network on Socioeconomic Status and Health. Reaching for a healthier life: Facts on socioeconomic status and health in the U.S., 2007. Available at: http://www.macses.ucsf.edu.
This report provides a clear and succinct overview of the broad range of social and economic determinants that contribute to health inequities.

Benach J, Muntaner C, Santana V. Employment conditions and health inequalities: final report to the WHO Commission on Social Determinants of Health, Employment Conditions Knowledge Network, 2007. World Health Organization. Available at: http://www.who.int/social_determinants/resources/articles/emconet_who_report.pdf.
This comprehensive report provides a global overview of the contribution of working conditions to worldwide health inequalities.

Morello-Frosch R, Lopez R. The riskscape and the color line: examining the role of segregation in environmental health disparities. Environmental Research 2006; 102: 181–196.

This paper provides an excellent example of research demonstrating how segregation concentrates economic disadvantage and environmental risks. The authors examine links between racial residential segregation and estimated ambient air exposures to toxic substances and their associated cancer risks, using modeled concentration estimates from the EPA.

Wilson SM, Heaney CD, Cooper J, Wilson OR. Built environment issues in unserved and underserved African-American neighborhoods in North Carolina. Environmental Justice 2008; 1: 63–72.
This article describes built-environment issues that burden unserved and underserved communities of color in North Carolina. The authors use a case study from Mebane, North Carolina, to describe how neighborhoods of color in this small town have been impacted by environmental injustice through the denial of basic amenities, especially sewer and water services, and overburdened by unhealthy land uses through inequities in the use of extraterritorial jurisdiction and annexation statutes.

Bullard RD, Mohai P, Saha R, Wright B. Toxic wastes and race at twenty, 1987–2007: grassroots struggles to dismantle environmental racism in the United States. Cleveland, OH: United Church of Christ, 2007.
This report is essential reading for those interested in learning more about environmental justice in the United States. It discusses exposure disparities at the regional, state, and metropolitan level, using data on hazardous waste sites. The authors discuss various tools that can be used to assess disparities in exposure to and body burden of toxic substances among demographic groups.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health or the International Labor Organization.

5

Global Environmental Hazards

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Global environmental changes are large-scale changes to the world's natural environment that result from human actions. Only in recent decades has the combined size of the human population and the intensity of human economic activities been sufficiently great to begin to change and disrupt the natural environment systemically. The aggregate environmental impact of humankind is so great that it is beginning to alter the Earth system on a planetary scale. Global environmental changes demonstrate that we have begun to live beyond the biosphere's capacity to supply, absorb, and replenish.

Most of the environmental health hazards that have attracted our attention recently have entailed human exposures to chemical and physical contaminants in workplaces and communities. In contrast, global environmental changes act predominantly by exacerbating existing adverse environmental influences on health. For example, global climate change increases thermal stresses, facilitates the spread of various infectious diseases, and increases psychological stresses on farmers, coastal dwellers, and other vulnerable populations.

The "global" category of human-induced environmental change is defined by both scale and its systemic character (causing alteration to basic life-supporting systems). Such change to the structure and function of large natural biophysical and ecological systems diminishes the capacity of the natural environment to supply "services," such as replenishing resources, and absorbing and recycling waste products of humans and domesticated animals.

The Earth system, comprising physical, chemical, biological, and human components, is self-regulating. Global environmental changes alter the "forcings" (drivers) and feedbacks that comprise the internal dynamics of this system. In addition, global dynamics are characterized by critical thresholds and abrupt changes. The Earth system has operated in various states during the past half million years, during which time abrupt transitions—within a decade or less—have sometimes occurred. Our understanding of global dynamics has greatly advanced in recent years, enabling more confident assessment of the consequences of human-induced change, including the possibility that human activities could inadvertently trigger abrupt changes with severe consequences.

We live in a world that is undergoing widespread and rapid globalization. This process encompasses the extension and intensification of various social, economic, cultural, technological, and political interrelationships worldwide. Economic globalization, characterized by increasingly integrated and deregulated ("liberalized") systems of markets, capital flows, and

trading in recent decades, has adversely affected many aspects of the natural global environment. Beginning in 2008, it has also been seen as seriously defective as a means of achieving sustainable economic progress, financial security, and fairer distribution of resources between low-income and high-income countries and between low-income and high-income groups within countries. Globalization and global environmental change, although strongly associated now, need not be so. Globalization could continue in the future, yet be managed in an environmentally sustainable and more equitable manner.

Global environmental changes derive from multiple point sources of human economic activity. Each change is "global" in the sense of either *(a)* being integrated—therefore, becoming a "systemic" change to a global process, such as changes to the global climate system and to global cycles of elements; or *(b)* occurring by the worldwide aggregation of local changes, such as land degradation. The best known and most intensively studied of global environmental changes that are hazardous to health are those resulting from the following two major changes to the atmosphere:

1. *Depletion of stratospheric ozone by various anthropogenic gases, primarily halocarbons.* The resultant increased flux of solar ultraviolet radiation (UVR) and associated health risks are generally well understood. The clear-cut and physical nature of this additional exposure to UVR has made assessing its associated risks relatively easy.
2. *Amplification of the natural greenhouse effect by anthropogenic emissions of carbon dioxide and other greenhouse gases.* This process, which increases the heat-trapping capacity of the lower atmosphere, is the cause of anthropogenic climate change over the past 40 years. Compared to the human health risks of ozone depletion, those of climate change are much more complex and diverse and less easily defined.

Other global environmental changes also pose serious health risks, including the following:

1. Biodiversity losses, which are often associated with impoverishment and destruction of major ecosystems and adverse effects on human health
2. Overfishing, which together with climatic influences, diminishes fish stocks and the viability of some fishing areas
3. Land degradation, which impairs food yield and food quality
4. Disruption of other major elemental cycles, such as those of nitrogen, sulfur, phosphorus, and carbon
5. Depletion of freshwater supplies and water stress, which result from excessive demand and mismanagement of freshwater resources
6. Global dissemination of persistent pollutants

These global environmental changes have significance for public health in three major ways. First, the health of a population is increasingly being influenced by changes originating beyond the boundaries of that population's immediate living space. Second, these major changes to the biosphere's life-support system, which may be irreversible, increase the likelihood of long-term, and possibly escalating, adverse health impacts on future generations. Third, prevention strategies to address these global environmental changes will need to depend largely on an integrated, systems-based approach that aims to change energy use, modes of transportation, food production, water-shed management, contact with microorganisms, use of industrial chemicals, and other modes of living (Fig. 5-1).

Periods of social and environmental upheaval have often been accompanied by infectious disease outbreaks, which, in turn, have often generated social and political changes. Since 1976, approximately 30 infectious diseases have emerged, demonstrating that such infectious diseases can arise suddenly and spread rapidly, adversely affecting health as well as employment, trade, travel, tourism, and other aspects of human life.[1] Emergence of these infectious diseases may constitute the fourth* and largest—of

* The first three transitions were as follows: *(a)* early agrarian-based settlements enabled microorganisms to begin contact with *Homo sapiens*; *(b)* early Eurasian civilizations, such as the Greek and Roman empires, China, and South Asia, came into military and commercial contact about 2,000 to 3,000 years ago, exchanging their dominant infections; and *(c)* European expansionism during the past 500 years spread infectious diseases that were often fatal across the Atlantic Ocean.[2]

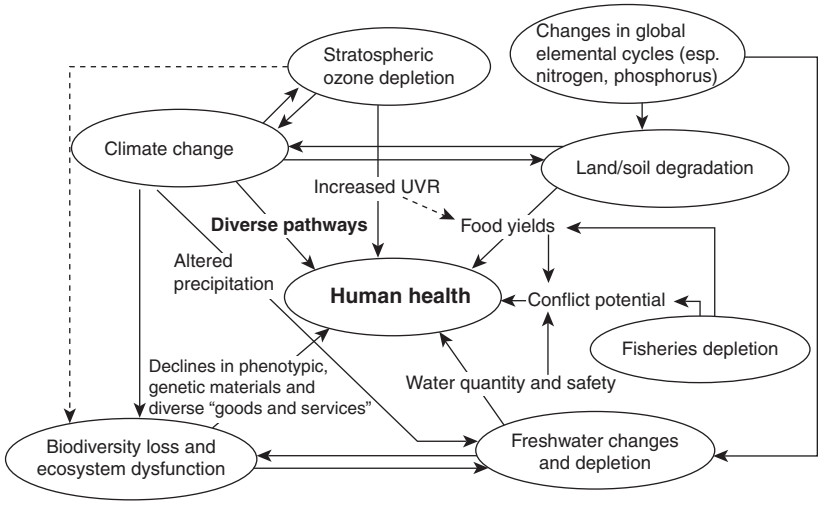


Figure 5-1. Interrelationships among major types of global environmental change, including climate change. Note that all of them impinge on human health and—although not shown here explicitly—there are various interactive effects between jointly acting environmental stresses. (*Source*: McMichael AJ, Campbell-Lendrum DH, Corvalan CF, et al. eds. Climate change and human health: Risks and responses. Geneva: World Health Organization, 2003.)

the great historical transitions in the relationship between microbes and humans.[2]

Sometimes a single infectious disease arrives at a time of particular population vulnerability, with devastating consequences, as occurred with the bubonic plague in the fourteenth century, influenza at the end of World War I, and, since the 1980s, the HIV/AIDS epidemic, especially in sub-Saharan Africa. Sometimes several epidemics of infectious diseases arrive or intensify as a group—"syndemics," as occurred with the urban epidemics of tuberculosis, smallpox, and cholera in England as it became industrialized in the early nineteenth century.

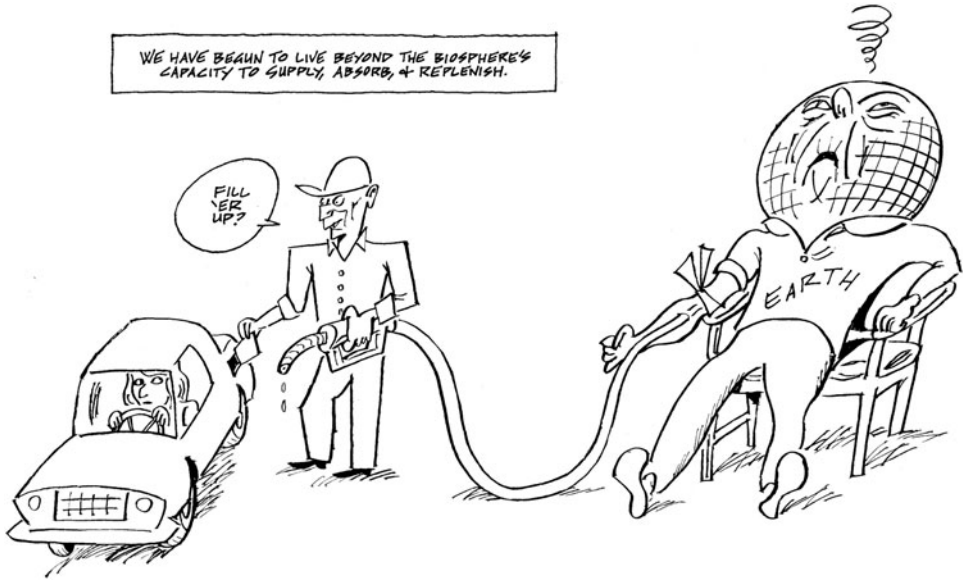
GLOBAL CLIMATE CHANGE

Compared to other planets in the solar system, Earth has a distinctive atmosphere. Its high concentration of oxygen is a direct consequence of photosynthesis. Various trace gases in the atmosphere, especially carbon dioxide, produce a natural greenhouse effect, which warms the Earth by 30°C and keeps it comfortably above freezing point.

Many countries have developed their economies in ways that have increased atmospheric concentrations that enhance the heat-trapping properties of the atmosphere. These greenhouse gases, primarily carbon dioxide, methane, nitrous oxide, and various humanmade halocarbons, increase the atmospheric absorption of infrared radiation reflecting off the Earth's surface. Therefore, more heat energy accumulates in the lower atmosphere and the surface of the Earth warms. In its *Fourth Assessment Report* in 2007, the United Nations Intergovernmental Panel on Climate Change (IPCC 2007) stated: "Warming of the climate system is unequivocal, as is now evident from observations of increases in global average air and ocean temperatures, widespread melting of snow and ice, and rising global average sea level."[3]

During the twentieth century, the average surface temperature of the Earth increased by approximately 0.74°C. Climate scientists use global circulation models to assess future changes in climate (Fig. 5-2). Based on these models, global average surface temperature is projected to increase by 1.8°C to 4.0°C during this century.[3] This estimate is uncertain because we do not know *(a)* how the climate system will respond to continuing change in atmospheric composition, and *(b)* what social, technological, demographic, and behavioral changes will occur in human societies.

Climate models project that temperature increases will be greater on land (than at sea),



(Drawing by Nick Thorkelson.)

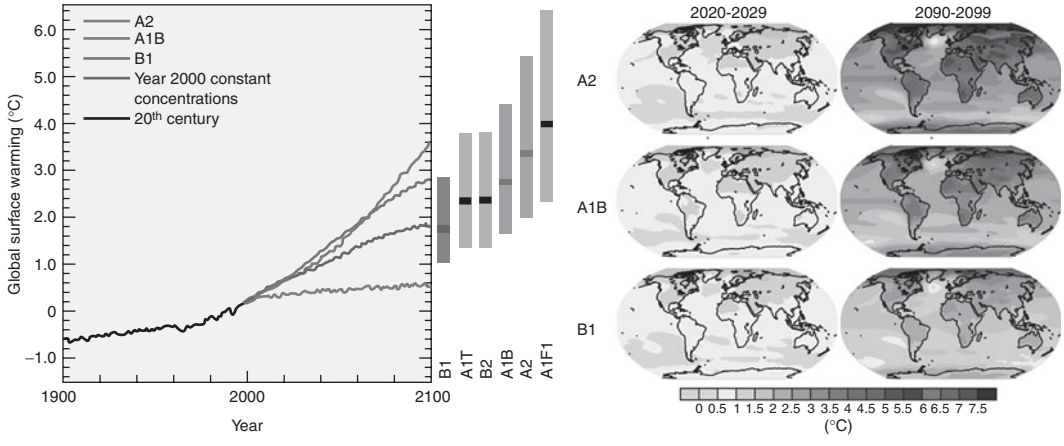


Figure 5-2. Changes in global average surface temperature projected under scenarios of global climate change. *Left:* Solid lines are multimodel global averages of surface warming (relative to 1980–1999) for the Special Report on Emissions Scenarios (SRES) A2, A1B, and B1, shown as continuations of the twentieth-century simulations. A line is for the experiment where concentrations were held constant at year 2000 values. The bars in the middle of the figure indicate the best estimate (solid line within each bar) and the likely range assessed for the six SRES marker scenarios at 2090–2099 relative to 1980–1999. The assessment of the best estimate and likely ranges in the bars includes the Atmosphere-Ocean General Circulation Models (AOGCMs) in the left part of the figure, as well as results from a hierarchy of independent models and observational constraints. *Right:* Projected surface temperature changes for the early and late twenty-first century compared to 1980–1999. The panels show the multi-AOGCM average projections for the A2 (top), A1B (middle), and B1 (bottom) SRES scenarios averaged over 2020–2029 (left) and 2090–2099 (right). (*Source*: Intergovernmental Panel on Climate Change. IPCC Fourth Assessment Report. Geneva: Intergovernmental Panel on Climate Change, 2007.) A color version of this figure is available at: http://www.ipcc.ch/graphics/syr/fig3-2.jpg.

at night, and at higher latitudes. Rainfall is projected to increase over the oceans but decrease over much of the land surface. Extreme rainfall events (both floods and droughts) will intensify. Climate variability and the frequency of extreme weather events have already increased in several regions, consistent with model projections.

Surprise events may occur. For example, there has been unexpectedly rapid loss of summertime sea ice in the Arctic and acceleration in the melting of glaciers.[4] Loss of ice uncovers relatively dark surfaces, such as rock or sea water, increasing, in turn, the amount of solar radiation absorbed at the surface, surface temperature, and melting. Loss of major land-based ice masses would eventually, over several centuries, raise sea level by at least several meters and could become irreversible. In addition, warming of the Arctic may release large amounts of greenhouse gases (carbon dioxide and methane) trapped in layers of permafrost. Warming also reduces net uptake of carbon dioxide by land and oceans, another potentially serious consequence.

Potential Health Impacts of Climate Change

Global climate change is adversely affecting the functioning of many ecosystems and the health of plants and animals. If climate change occurs as forecasted, one-third of terrestrial plant and animal species are likely to become extinct by 2050.[5] On the other hand, climate change could have beneficial health impacts for some human populations. For example, in temperate regions, milder winters could reduce the frequent wintertime peak in deaths, and, in torrid regions, a further increase in temperature might reduce transmission of some vectorborne diseases. Overall, however, almost all health impacts of climate change will be adverse (Fig. 5-3).

Direct health impacts of climate change will result from the following:

1. Changes in exposure to weather extremes, such as heat waves and winter cold (see Box 5-1 concerning the impact of climate change on workers' health)

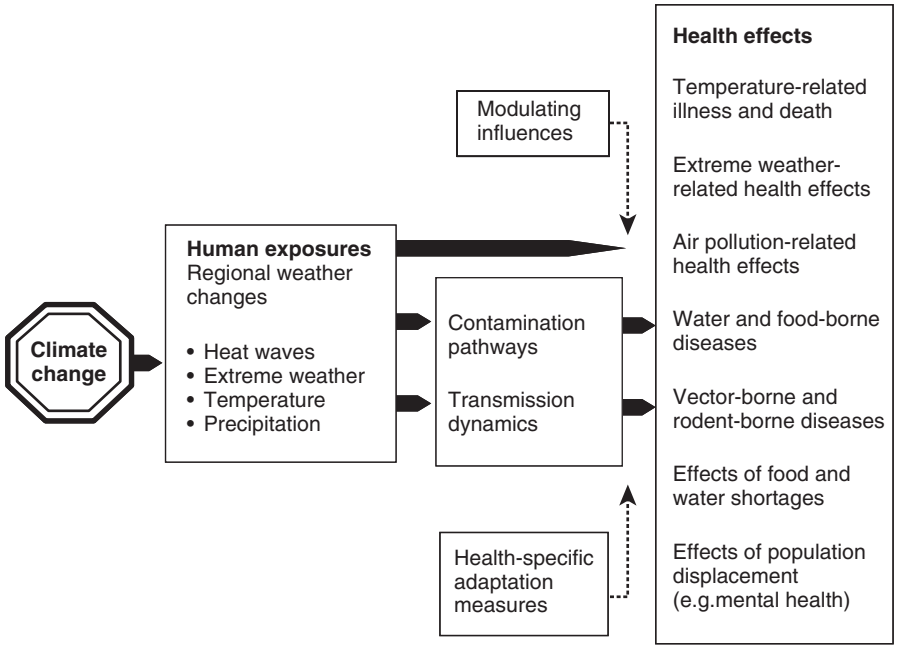


Figure 5-3. The main pathways by which climate change causes direct and indirect impacts on human health. The impact-modifying role of modulating factors and specifically adaptation measures is also shown. (*Source*: McMichael AJ, Campbell-Lendrum DH, Corvalan CF, et al. eds. Climate change and human health: Risks and responses. Geneva: World Health Organization, 2003.)

Box 5-1. Climate Change, Workplace Heat, and Health

Tord Kjellstrom

Global climate change is making already hot seasons in hot parts of the world even hotter.[1] Since 1980 many populated places, especially cities, with hot temperatures (regularly above 35°C or 95°F), have already recorded a 1°C–2°C increase in average temperature.[1,2] An additional 2°C–4°C average increase can be expected in these places during this century.[1] In urban areas with rapid development of buildings, roads, and other major structures, the temperature increase is likely to proceed faster and higher due to the "urban heat-island effect."[3,4] Increased heat exposure for workers due to climate change will vary by location. Places that already have many hot days will experience extremely hot days, when physical labor becomes virtually impossible. Extreme heat load is occurring in the southern United States for about 1 month each year and strong heat load for 2 months—similar to the situation in most tropical areas.[5] By 2050, the duration of extreme heat load will likely increase by 1 month in the southern United States.[5] Outdoor work and indoor work without air conditioning during the hottest part of days will become increasingly difficult.

A range of health impacts related to climate and climate change have been identified. These include heat exhaustion, heat stroke, kidney disease, effects of additional air pollution, injuries and mental stress from extreme weather events, vectorborne diseases, diarrhea, and malnutrition. While many of these impacts can create even higher health risks among workers, heat exhaustion and heat stroke are of special importance to them.

Workers who perform substantial physical activity produce internal heat, which, in combination with excessive external heat exposure, can create substantial health risks.[6,7] Acclimatization partially reduces this risk.[8,9] Outdoor work in the sun during the hottest season can kill workers if adequate preventive measures are not implemented. For example, the annual heat-related mortality rate among crop workers in the United States has been reported as 3.9 deaths per million workers.[10] During the heat wave in France in 2003 more than 500 additional deaths occurred among those age 35 to 54, with a much greater increase among men than women.[11] This was the only age group in which more men than women died as a result of the heat wave. No analysis was done to determine whether high occupational heat exposure was a factor.

Many more workers are affected by nonfatal heat stroke, heat exhaustion, and other heat-related disorders.[6,12] High-risk occupations are those that require heavy physical activity and are performed either outdoors or indoors without air conditioning. To prevent effects, the wet-bulb globe temperature (WBGT) is used as an exposure index by the National Institute for Occupational Safety and Health (NIOSH), the American Conference of Governmental Industrial Hygienists (ACGIH), and the International Standards Organization (ISO).[13] The international standard includes recommendations for length of rest breaks during each work hour during exposure to high heat, taking into account acclimatization, work intensity, and workers' clothing.[13] If employers and workers comply with the standard, overheating will be avoided in most workers; however, productivity for repetitive work is reduced when rest breaks are lengthened.[14]

As occupational heat exposure increases, the risk of heat-related disorders increases if sufficient prevention is not applied. In contrast, productivity decreases if rest breaks are allowed. Therefore, there is tension between prevention and economic performance, which becomes even more important with global warming.[15]

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2. Increases in other extreme weather events, such as floods, cyclones, and droughts
3. Increased exposure to certain air pollutants and aeroallergens, such as spores and mold

In addition, indirect impacts of climate change will result from the following:

1. Increases in transmission of many infectious diseases, especially those that are waterborne, foodborne, or vectorborne
2. Decreases in regional food yields, especially those of cereal grains

In the long term, these indirect health effects of climate change are likely to be greater than direct impacts.

Concerning vectorborne infectious diseases, the distribution, abundance, and seasonal activity of vectors, such as mosquitoes and ticks, are affected by *(a)* various physical factors, such as temperature, precipitation, humidity, surface water, and wind; and *(b)* biotic factors, such as vegetation, abundance of host species, predators, competitors, and human interventions. Increased replication of vectors and pathogens (such as protozoa, bacteria, and viruses) under warmer, wetter conditions often increases transmission of many vectorborne diseases, such as malaria and dengue fever (transmitted by mosquitoes), leishmaniasis (transmitted by sandflies), and schistosomiasis (transmitted by water snails). In some cases, climate change may have mixed effects on transmission of vectorborne diseases. For example, schistosomiasis may decrease in some regions because warmer water adversely affects snail survival, but it may increase in others as ice-free areas during winter expand.[6] As a result of global climate change, the geographic range of potential transmission for many vectorborne diseases will increase.

Climate change during the next 50 years will probably slightly decrease global yields of grain, which globally account for two-thirds of food energy worldwide, especially in food-insecure regions in South Asia, Africa, and Central America. Such a decrease would increase the number of malnourished people by tens of millions.

Climate change over the past 25 years probably has had some adverse incremental impacts on health.[7] However, detection of these effects and their attribution to climate change are functions of statistical power and reasonable judgment. Statistical power depends on numbers of observations and the extent of divergence between observed and expected rates or magnitudes of health outcomes. Attribution depends, in part, on pattern recognition. If, for example, a particular infectious disease changes in occurrence in multiple geographic locations, each associated with local climate changes, then we can be much more confident that there is a climatic influence than if we were to see such a change in occurrence in just one location.

A study of attributable deaths and disabling diseases and injuries due to climate change found that approximately 155,000 extra deaths were attributable to climate change—mainly in poor countries—from malnutrition, diarrheal disease, malaria, and flooding. This study needs to be updated and extended, covering a wider spectrum of health impacts possibly due to climate change.[8]

STRATOSPHERIC OZONE DEPLETION

Ozone first appeared in the Earth's atmosphere about 2 billion years ago. Oxygen produced by photosynthesis in water-based plants spilled over into the atmosphere and, in the upper atmosphere, was chemically converted by incoming solar UVR† to ozone. This stratospheric ozone chemically filtered out the harmful short wavelengths of UVR, eventually facilitating evolution from aqueous to land-based life.

Surface-level ambient UVR consists of *(a)* most of the incident solar UVA, which almost completely penetrates the atmosphere; *(b)* less than 10% of incoming solar UVB, most of which is filtered out by stratospheric ozone; and *(c)* no short-wave UVC, which is completely absorbed in the atmosphere (Fig. 5-4). Adverse health effects of stratospheric ozone depletion are thus

† Ultraviolet radiation is that part of the electromagnetic spectrum with wavelengths just shorter than the violet component of visible light. It comprises longer wavelength UVA, intermediate wavelength UVB, and shorter wavelength UVC. In general, the shorter the wavelength, the greater the potential that the radiation is more biologically damaging (see Chapter 11C).

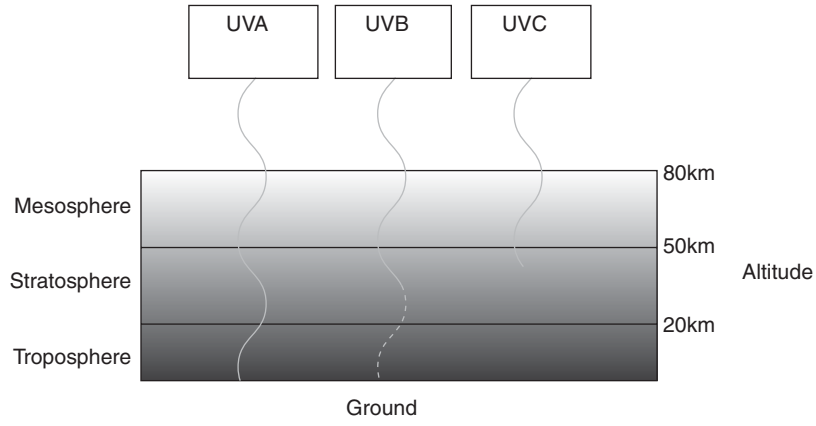

Figure 5-4. Penetration of incoming solar ultraviolet (UV) radiation to the Earth's surface. (*Source*: Lucas RM, Ponsonby AL. Ultraviolet radiation and health: friend and foe. Medical Journal of Australia 2002; 177: 594–598.)

largely confined to those associated with increases in UVB radiation specifically, rather than with UVR in general.

Ninety percent of the Earth's ozone is in the stratosphere; the remaining 10% is in the troposphere. Dobson units (DUs) are a measure of the number of ozone molecules between the top of the atmosphere and the Earth's surface—that is, the "thickness" of the ozone layer. Total column ozone is least at the equator (less than 300 DUs) and increases at higher latitude, with greater overall column amounts at high latitude in the Northern Hemisphere than in the Southern Hemisphere. There are seasonal and annual fluctuations in total column ozone due to wind transport and stratospheric circulation of ozone.

Chlorofluorocarbons (CFCs) were developed in the 1920s as safe, nontoxic, nonflammable replacements for toxic refrigerants then in use, such as ammonia. With later use in the automotive industry and as propellants for aerosol cans, CFCs eventually entered the atmosphere.

In 1974, scientists theorized that free chlorine atoms released from atmospheric CFCs, by reaction with UVR at low atmospheric temperatures, might catalytically destroy stratospheric ozone (Box 5-2). Within 10 years, international action was taken in the United States and Europe to decrease the use of CFCs, especially in aerosols. The Vienna Convention for the Protection of the Ozone Layer was signed in March 1985 by 20 nations, preceding by 2 months the first published reports of measurable ozone loss (less than 220 DUs) in Antarctica—subsequently called the ozone hole. An international response rapidly ensued, with the Montreal Protocol in 1987 and its subsequent amendments providing global phase-out schedules for CFCs and their less damaging substitutes, the halons and hydrochlorofluorocarbons (HCFCs).

Chlorofluorocarbons have long half-lives in the atmosphere, so that, despite the relatively prompt international action to limit CFC production and consumption, atmospheric CFCs have continued to accumulate—with increasing destruction of stratospheric ozone. In 2007, the United Nations Environment Program (UNEP) reported that the total column ozone at midlatitudes reached a minimum in the late 1990s, may have been increasing since then, but remains below levels found in the 1980s. UVB irradiance

Box 5-2. Chemical Reactions in the Destruction of Ozone

Ozone "absorbs" UVR in the UVB band when UVB breaks an ozone molecule into an oxygen molecule and an oxygen atom. This atom then combines with another oxygen molecule to regenerate ozone. The result of these reactions is the conversion of solar UVB into heat energy.

Ozone formation: $O_2 + UVC \rightarrow O + O$

$O_2 + O \rightarrow O_3$

Absorption of UVB: $O_3 + UVB \rightarrow O_2 + O + heat$

$O_2 + O \rightarrow O_3$

Although the reaction of ozone with UVR results in the regeneration of ozone molecules, as a catalyst, free chlorine radicals are regenerated post reaction to destroy further ozone molecules.

$Cl + O_3 \rightarrow ClO + O_2$

$ClO + O \rightarrow Cl + O_2$

increased during the period of ozone depletion, but it may now be decreasing. At high latitudes, in the Arctic and in Antarctica, ozone levels have been highly variable. In the spring of 2007 the ozone hole over Antarctica was smaller than in 2006, at around the average size for the past 15 years. Antarctic ozone is heavily influenced by the polar stratospheric temperature; despite decreasing stratospheric halocarbon levels, there was a large 2006 ozone hole, due to unusually low temperatures. Nevertheless, atmospheric levels of CFCs appear to have peaked and stratospheric concentrations of most ozone-depleting halocarbons are now decreasing. Stratospheric ozone at mid-latitudes is predicted to return to levels found in the 1980s by around 2050, and ozone in polar regions, 10 to 20 years later. However, atmospheric changes induced by climate change may considerably delay recovery of stratospheric ozone. In addition, climate change may directly affect UVB levels at the Earth's surface through changes in cloudiness, atmospheric aerosols, and surface reflectivity.[9] (See Box 5-3.)

Health Effects

There are three important determinants of the dose of UVR received by an individual:

- *The ambient UVR level and its relative wavelength constitution.* This is determined by season, latitude, and the level of stratospheric ozone. Ground-level measurements of UVR differ from satellite measurements

Box 5-3. Interactions between Climate Change and Stratospheric Ozone Depletion

Global climate change and stratospheric ozone depletion are essentially different processes—in both cause and effect. They are not, however, isolated processes, and they will interact.

Positive interactions (worsening the effects of stratospheric ozone depletion or climate change):

- Behaviorally, warmer temperatures as a result of global climate change may result in increased exposure to ultraviolet radiation (UVR) due to wearing fewer clothes and an increase in the time when the weather is suitable for outdoor activities.
- Stratospheric cooling due to increased lower atmospheric carbon dioxide may delay recovery of the ozone layer, although modeling study results vary.[1] Estimates of skin cancer incidence under different scenarios of chlorofluorocarbon and halon restrictions indicate that, under full compliance with the Montreal Protocol and its amendments, there would be a peak excess incidence of 9% above baseline (1980–1990 levels) in 2055.[2] If ozone layer recovery is delayed by climate change, the excess peak incidence may increase to 15% above baseline in 2065.
- Animal studies suggest that warmer temperatures may enhance the carcinogenicity of UVR, thus accentuating increases in skin cancer associated with ozone depletion. This temperature effect has yet to be confirmed in human populations.
- UVB-induced immunosuppression may worsen the effects of those diseases predicted to increase under climate change scenarios, such as malaria.
- Increased levels of UVB may decrease the uptake capacity of marine carbon sinks. Global warming and acid precipitation may improve UVB penetration into aquatic environments, thus enhancing UVB effects on aquatic ecosystems.

Negative interaction (decreasing the effects of stratospheric ozone depletion or climate change):

- Aerosols (including ozone) responsible for lower atmospheric pollution will decrease ambient UVR for any stratospheric ozone level.
- Warmer temperatures may decrease the amount of time spent outdoors during the part of the day when there is peak UVB radiation.
- Increased levels of UVB may decrease enhanced plant growth seen under high carbon dioxide conditions.[4]
- Although in some areas frosts will be more infrequent under climate change scenarios, greater UVB levels may increase frost sensitivity of plants.[4]

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of ambient UVR due to variations in cloud cover and lower atmospheric pollution.

- *Culture and behavior.* Although the usual outdoor exposure may be only 5% to 10% of daily erythemal UVR, for any ground-level measurement of UVR at any location, there may be a 100-fold difference in individual exposure due to differences in sun exposure habits. Even in high-latitude countries, such as Sweden, most adults and adolescents report sunburn from outdoor tanning.
- *Skin pigmentation.* Deeply pigmented skin has a natural sun protection factor of approximately 13; the dose of erythemally weighted UVR required to produce barely discernable erythema in people with deeply pigmented skin is 33 times that in people with lightly pigmented skin.[10] For any level of UVR exposure, the biologically damaging effect will be much greater on fair skin than more deeply pigmented skin.

UVB does not penetrate tissues as deeply as UVA, so that its main deleterious effects on human health are on superficial tissues: the skin and the eye. UVB is absorbed by DNA, causing characteristic chemical changes (pyrimidine dimers) that, as mutations, may be critical in the initiation of carcinogenesis. UVB exposure also has beneficial health effects. The main source of vitamin D is from UVB-induced conversion of precursors in the skin. This essential vitamin is important for skeletal health, especially the prevention of rickets, osteomalacia, and osteoporosis. There is now evidence that vitamin D insufficiency plays a role in a wide range of chronic disorders, including autoimmune diseases (such as type 1 diabetes and multiple sclerosis), hypertension, malignancies (including breast cancer, colorectal cancer, prostate cancer, and non-Hodgkin lymphoma), and mental health disorders (including schizophrenia and depression).

Adverse Effects of UVB on the Eyes, Skin, and Immune System

The eye is the only part of the human body not shielded from harmful UVB radiation by the protective layer of the skin. The vulnerability of the eye to environmental hazards is the price we pay for being able to see. Acute high-dose UVB exposure results in acute inflammation of the cornea and conjunctiva (photokeratitis and photoconjunctivitis, or snow blindness). Chronic UVB exposure is one risk factor for the development of pterygium (a fleshy wing-shaped growth on the surface of the eye) and for squamous cell carcinoma of the cornea and conjunctiva.

There is a causal relationship between "senile" cataract and UVR, especially UVB, exposure. Cataracts are extremely common, especially in older people, and may cause visual impairment, including complete blindness. Exposure to UVR causes cortical and nuclear cataracts, and it might also cause posterior subcapsular cataract.[11]

Under normal circumstances, the anterior parts of the eye and the vitreous humor filter out most UVB radiation. However, a retinal "sunburn" (phototoxic retinopathy, solar retinopathy, or eclipse retinopathy) can occur if the sun is viewed directly, such as by sun-gazing or looking at the sun during a solar eclipse. The acute vision loss usually resolves over weeks or months, but it occasionally progresses to permanent visual impairment.

Sunburn is the immediate result of acute overexposure of the skin to sunlight. UVB is three to four times as effective as UVA in causing erythema (skin redness) in humans. Chronic or repeated UVR exposure is the strongest risk factor for the development of cutaneous malignant melanoma (CMM), squamous cell carcinoma (SCC), and basal cell carcinoma (BCC).[11] These cancers are particularly common in regions where pale-skinned populations are exposed to high levels of UVR, such as at lower latitudes. Cumulative lifetime UVR exposure appears to be most important in the development of SCC; both BCC and CMM may be more closely associated with a pattern of intermittent high-level exposure and there may be critical ages of exposure.[12] Although UVA may also be important in the development of CMM, the DNA damage associated with UVB absorption is particularly implicated in SCC and BCC. Modeling suggests that there will be a 5% to 10% excess incidence of skin cancers occurring between 2025 and 2050 attributable to stratospheric ozone depletion.[13]

Exposure to UVR causes local and systemic immunosuppression, with both deleterious and beneficial consequences.[14] It appears to impair the activity of T helper 1 lymphocytes, which are important in the body's reaction to simple chemicals, intracellular infections (such as those caused by viruses), and tumor growth (including skin cancers). However, suppression of the T helper 1 lymphocytes may lower the risk of autoimmune disorders, such as type 1 diabetes and multiple sclerosis.[15] There appears to be either little effect on or enhancement of T helper 2 lymphocytes, which are important in the immune response to extracellular infections, such as those caused by most bacteria.[10]

Ecological Effects

Increased UVB can cause adverse effects on terrestrial and aquatic ecosystems, which may indirectly affect human health.[9] Because species differ in their sensitivity to UVB-induced damage, there may be changes in species composition and biodiversity of plants as well as bacteria and fungi growing on plants. Enhanced UVB irradiation may decrease root mass and, through changes in soil microbial communities, induce complex changes in processing of mineral nutrients in soil.[9] In aquatic ecosystems, increased solar UVB may reduce productivity and impair reproduction and development. It may also increase the mutation rate in phytoplankton, fish eggs and larvae, and in zooplankton, as well as primary and secondary consumers of these life forms. Stratospheric ozone depletion and resultant increased UVB radiation also affects biogeochemical cycles, including those of oceanic carbon, nitrogen availability to plants, and tropospheric ozone, by producing halogen-containing compounds from decomposition of plant matter.[9]

BIODIVERSITY LOSS

Biodiversity underpins the resilience of the ecosystems on which human societies depend. Biodiversity loss is now occurring at least as rapidly as in any of the five great extinctions that have occurred over the past 500 million years—since the advent of multicellular organisms. This loss is being driven by a range of adverse factors: overexploitation and fragmentation of ecosystems, land use changes, climate change, chemical pollution, introduced species, and biotechnology.[16] Loss of biodiversity is an increasing threat to ecosystem services that are vital to human health and well-being, including provision of food, freshwater, fuel, and fiber; cycling of nutrients; processing of waste; flood and storm protection; and climate stability.

Causal pathways linking biodiversity loss and health are complex and strongly contingent upon local circumstances. Therefore, despite the fundamental importance of ecosystem services for human health, links between biodiversity loss and human health are difficult to demonstrate epidemiologically.[17]

Local social conditions can and do modulate the effects of ecosystem disruption.[18] Human societies have adapted to natural fluctuations in ecosystem services and, especially in rich countries, have implemented efficient methods of buffering communities, such as systems of trade, agriculture, and water storage. In countries dominated by market economies, these adaptations have historically been designed to minimize short-term, local ecological changes, while maximizing profits. As a result, large-scale unintended consequences of human economic activity tend to be displaced geographically (such as the costs of overconsumption in rich countries) or postponed into the future (such as the long-term consequences of climate change or desertification). Historically, loss of productive ecosystem services has led to the collapse of whole civilizations. For example, the Mayan Empire collapsed around 1,000 years ago largely as a result of soil erosion, silting of rivers, and prolonged drought conditions—collectively leading to agro-ecosystem failure.[16,19]

The health of human populations depends crucially upon the services of food-producing ecosystems. This is most obvious in poor countries, especially in rural areas where food is derived mainly from local sources. Human dependence on ecosystems for nutrition is less apparent—but ultimately no less fundamental—in urban communities.

Undernutrition remains a dominant health problem globally, with poverty a strong determinant of undernutrition everywhere.[20] About 25% of the global burden of disease in the poorest

countries is attributable to childhood and maternal undernutrition. Worldwide, undernutrition accounts for nearly 10% of disability adjusted life-years (DALYs).[20] In developed countries, diet-related risks—mainly overnutrition—in combination with physical inactivity, accounts for one-third of the burden of disease.

The major cause of undernutrition globally is not lack of food production, but lack of equitable food distribution. Global food production has, historically, been sufficient to meet the needs of all, but the global financial crisis and increases in food prices have led to increases in food insecurity and undernourishment. Globally, an estimated 1 billion people are now undernourished—while hundreds of millions are overfed.[21]

The present global inequality in access to food is driven primarily by political and economic factors—although ecological problems, including climate change and water scarcity, threaten to play an increasingly important role in limiting food production in the future. Agricultural production has tripled in the past four decades, mainly through growth in yield. However, food production has not kept pace with population increase in many countries, and improvements in yield appear to have slowed.[16,22] In poor countries, population increases mean that the number of people per hectare of arable land increased from three in 1961–1963 to five in 1997–1999.[22]

Vulnerable poor communities are often forced to settle on marginal, drought-prone lands, with poor soil fertility. At least 1 billion people live in areas where the land is becoming degraded through soil erosion, water logging, or salinity. Providing sufficient food sustainably for an expected human population of 8 to 9 billion people by 2050 will require profound changes in both *(a)* production methods and technologies, and *(b)* the distribution of resources (wealth, knowledge, and power).[8,23]

In many countries, agricultural production is increasingly dependent on irrigation. This situation is likely to lead to armed conflict where there are existing tensions over access to freshwater supplies. Many river systems with scarce water resources are shared uneasily among neighboring countries in unstable regions of the Nile, the Ganges, the Mekong, the Jordan, and the Tigris and Euphrates Rivers. "Water wars" have therefore been postulated as increasingly likely in the future, as population pressures and demands increase, including among countries in the Middle East, and between Ethiopia and Egypt, Lesotho and South Africa, and India and Bangladesh.[24]

Freshwater

About 1.1 billion people lack access to safe water, and about 2.4 billion lack adequate sanitation. Lack of improved water and sanitation is strongly associated with poverty, although this relationship varies among regions.[20] Along with sanitation, water availability and water quality are well recognized as important risk factors for infectious diarrheal diseases and other potentially serious diseases, especially in children.[25]

Freshwater, which is used for growing food, drinking, washing, cooking, and recycling of wastes, is a key resource for human health. Almost 4% of the global burden of disease is attributable to unsafe water and poor sanitation and hygiene.[25] In this century, water resources will be strongly affected by trends in population growth, land use, and management of freshwater ecosystems. Increasing demand for food will worsen water scarcity. By 2025, an estimated 50% of people will live in river basins where water is scarce, and 70% of readily available water supplies will be exploited.[26] Water scarcity can lead to use of poorer-quality sources of freshwater, which are more likely to be contaminated and more likely to cause water-related diseases.

Fuel

Globally, most people have limited or no access to electricity. More than 2 billion people rely on biomass—wood, dung, and agricultural residues—for heating and cooking.[27] Energy consumption per capita is about 25 times higher in rich countries than in poor countries.[28] Lack of clean, safe power contributes to many adverse impacts on health.

Ambient air pollution, resulting from combustion of fossil fuels for transport, power generation, and industrial production, aggravates heart and lung disease (Chapter 6). Indoor air

pollution causes much respiratory disease in adults and children (Chapter 7). About 50% of people still use solid fuel for cooking. Almost 3% of the global burden of disease is attributed to indoor air pollution from solid fuel. Urban air pollution has accounted for a further 1% of the global burden of disease.[20]

Clean, reliable energy supplies are a fundamental requirement for sustainable development. The need to spend considerable time collecting fuel can preclude proper education, with indirect adverse health effects due to illiteracy, lost work opportunities, and large family size. In addition, energy use is indirectly linked to adverse health effects due to climate change, as noted earlier.[29]

Nutrient Management and Waste Management, Processing, and Detoxification

Well-functioning ecosystems absorb and remove chemical and biological contaminants. For example, wetlands can remove excess nutrients from runoff, preventing damage to downstream ecosystems. Inadequate sanitation (management of solid waste) increases human exposure to infectious disease agents—such as by fecal contamination of water or disease transmission by rats—leading to diarrheal illness and other infectious diseases.[30] Human sewage waste can be safely used as a fertilizer provided that appropriate precautions are taken to avoid contamination of produce.[31] Excessive use of fertilizer leads to accumulation of nitrogen and phosphorus in surface waters and coastal sea areas. The resulting overgrowth of bacteria, phytoplankton, and algae can lead, in turn, to increases in waterborne diseases and poisoning from harmful algal blooms.[32]

Climate Regulation

Climate regulation is an important property of the Earth's natural systems. Many vital ecological services are affected by climate change. For example, as a result of climate change, the number of people affected by water stress may increase by 500 million by 2025. Water-related disasters, due to droughts and floods, will have increasingly severe health impacts. The frequency of heavy rainfall events is likely to increase, leading to an increase in the magnitude and frequency of floods and a decrease in low-river flows.

Healthy ecosystems provide a buffer against the damaging effects of climate extremes. For example, forests absorb rainfall and provide a buffer against increases in runoff, thereby reducing flooding and soil erosion. A combination of deforestation and increased heavy rainfall events could have much more severe ecological and health consequences than either would alone. Healthy coral reefs and mangroves stabilize coastlines, limiting the damaging effect of storm surges. A combination of overfishing, local pollution, sea temperature increase, and sea level rise could damage coral reefs and, in turn, increase the vulnerability of small island communities to extreme weather events. For example, the flooding of the Yangtze River Basin in China in 1998 was attributed to a complex web of factors, including heavy rain associated with an El Niño event, deforestation that increased water runoff, and more intensive cultivation of lakes and wetlands in the river basin, which reduced their "sponge" function.

Heavy rainfall can adversely affect water quality by increasing chemical and biological pollutants flushed into rivers and overloading sewers and waste storage facilities. Increases in temperature tend to worsen water quality by increasing the growth of microorganisms and decreasing dissolved oxygen. In some parts of the world, climate change also may increase requirements for irrigation water because of increased evaporation.

Urbanization

Since the early nineteenth century, the proportion of people living in cities or large towns has increased from approximately 5% to 50%.[33] This radical transformation of human ecology continues apace, entailing changes in social organization, family relations, housing conditions, transport choices, dietary patterns, occupational environments, access to educational and health care services, and transmission of infectious disease agents.[34]

Some health risks are obvious, such as hospitalizations for asthma during air pollution crises and road traffic injuries. Others are more subtle, such as sustained exposure to environmental

lead, which causes adverse effects on the central nervous system and other organ systems (Chapters 11 and 19). Physical aspects of the urban environment affect seasonal patterns of morbidity and mortality. For example, quality of housing, including dampness and internal temperature, may contribute to early-life exposure to fungal spores and house dust mites, both of which can cause asthma in children.

As modern cities expand, transport systems become increasingly important. Car ownership and travel has escalated over the past 50 years in much of the world. In addition to problems related to exhaust gases—which cause local air pollution and contribute to acid rain and greenhouse gas emissions—major public health impacts of car-based systems include injuries, reduced physical activity (and resultant obesity), disruption of neighborhoods, and increased noise. (See Chapter 39.)

Urban air pollution has become a worldwide public health problem. The earlier industrial and domestic air pollution from coal burning, characteristic of much of nineteenth-century Europe and North America, has been largely replaced by pollutants from motorized transport. These form photochemical smog in summer and a heavy haze of particulates and nitrogen oxides in winter. (See Chapter 6.)

Cities have increasingly large "ecological footprints." There are ecological benefits of urbanism, including economies of scale, shared use of resources, and opportunities for reuse and recycling. But there are also great "externalities." Urban populations depend on food grown elsewhere, on raw materials and energy sources (especially fossil fuels) extracted elsewhere, and on disposal of their voluminous wastes elsewhere. For example, the estimated consumption of wood, paper, fiber, and food by 29 cities of the Baltic Sea Region requires a total area many hundred times greater than the combined area of the 29 cities there.[35] Urban populations—often with little awareness—are therefore a major and growing source of pressure on the biosphere.

Environmental Conflict and Security

As human populations have expanded over millennia, exploitation of natural resources and increasing territorial expansion have increased, often leading to armed conflict between rival groups. Although the causes of such conflicts have been multifactorial, complex, and contentious, competition for natural resources has been a key factor.[36] Nations have often fought to assert or resist control over raw materials, energy supplies, and land.[37] The Persian Gulf War of 1991 is a recent example of major conflict triggered by concern over an environmental resource: oil. Other recent, but lesser known, resource-associated conflicts include those in India, the Philippines, Mauritania, and Senegal.[38] The risk of conflict may significantly increase in the near future because of increased scarcity of natural resources, much of it due to declining environmental capacities.

Even if the "sustainability transition"[39] gains momentum, it is still likely that the per-capita availability of water, arable land, and other critical environmental resources will decline. Spectacular technological improvements in the exploration and recovery of oil have not relieved concerns that the end of cheap oil is likely in this century. Therefore, oil wars are also possible.

More speculatively, climate change may interact with natural resource stresses (such as water scarcity) and expanding human populations to increase the possibility of armed conflict. Many parts of Africa already experience a less-than-favorable agricultural climate, which will probably deteriorate further in the second half of this century, increasing the likelihood of armed conflict.[40]

Global warming may intensify the El Niño southern oscillation (ENSO).[41] Stronger, more frequent El Niños and La Niñas would increase adverse social, economic, and health consequences in various regions.[42] These, in turn, would tend to increase the risk of conflict in resource-scarce areas, such as by increasing regional food scarcity through intensified droughts.

Loss of biodiversity may not so obviously appear to potentiate conflict. The loss of genetic diversity will reduce the isolation of useful chemicals and the discovery of potentially useful biological compounds, but it is unlikely to lead to war. However, reduced ecosystem function, due to biodiversity loss, may interact with climate change to cause further deforestation

and ecosystem collapse, such as by the loss of "keystone species," leading to reduction in food supplies and loss of other ecosystem services.[43] These changes could exacerbate local and regional tensions.

There are numerous other mechanisms by which damaged ecosystems that otherwise provide essential "goods and services" may cause economic harm and increase the risk of armed conflict. Several worst-case scenarios could even lead to global conflict, including *(a)* runaway global warming; *(b)* food scarcity, leading to nuclear war involving South Asia or China; and *(c)* slowing of the Gulf Stream, which would disrupt European agriculture and greatly increase European energy needs.[44]

GLOBAL TRADE AND DEVELOPMENT

Income is a strong predictor of health status, especially among low-income populations. In the past 50 years, coincident with economic growth, there have been widespread increases in life expectancy and decreases in fertility rates.[39] In recent years, economic gains have been greatest in Western Europe, North America, Oceania, and some countries in Asia.[45] Yet during this period, income inequality has increased both within and among countries. The ratio of income earned in countries with the richest fifth of the population, compared to the poorest fifth, widened from 30:1 in 1960, to 60:1 in 1990, to 74:1 in 1997.[46]

There is a strong coupling of the political and economic processes driving global economic inequality and ecologically unsustainable resource use. International trade and development policies have contributed substantially to the present global social and ecological predicaments (Box 5-4). For example, "liberalized" trading structures and practices have contributed to the emergence and spread of infectious diseases. Overall, globalization of the food market has unavoidably accentuated the movement of pathogens from one region to another—and has also amplified the redistribution of microbial-resistance genes.

A primary stimulus for the great increase in migration internationally is the urge to enter the cash economy, with its demand for both skilled and unskilled workers in a globalizing marketplace. Rapid urbanization, often characterized by informal housing and periurban slums, tends to increase the occurrence of "old" infectious diseases, such as childhood pneumonia, diarrhea, tuberculosis, and dengue. Urbanization can also facilitate the spread of some emerging infectious diseases. For example, poor-quality, high-rise housing creates new risks, as occurred with severe acute respiratory syndrome (SARS) in Hong Kong in 2003. The SARS virus was

Box 5-4. Examples of Health Risks Arising from Global Trade Processes

1. Perpetuation and exacerbation of income differentials, both within and among countries, thereby creating and maintaining the basic poverty-associated conditions for poor health
2. Fragmentation and weakening of labor markets as internationally mobile capital acquires greater relative power. The resultant job insecurity, substandard wages, and "lowest common denominator" approach to occupational and environmental conditions and safety can jeopardize the health of workers and their families.
3. The consequences of global environmental changes, including changes in atmospheric composition, land degradation, depletion of biodiversity, spread of "invasive" species, and dispersal of persistent organic pollutants

Other, more specific, examples of risks to health include:

- Spread of smoking-related diseases as the tobacco industry globalizes its markets
- Diseases of dietary excess as food production and food processing become intensified and as urban consumer preferences are shaped increasingly by globally promoted images
- Diverse public health consequences of the proliferation of private car ownership, as car manufacturers extend their marketing
- Continued widespread rise of urban obesity as daily living patterns (eating, physical activity) evolve
- Expansion of the international drug trade, exploiting the inner-urban underclass
- Increasing prevalence of depression and mental health disorders in aging and socially fragmented urban populations
- Infectious diseases that now spread more easily because of increased worldwide travel

Source: McMichael AJ, Beaglehole R. The changing global context of public health. Lancet 2000; 356: 495–499.

transmitted via aerosol backflows from faulty sewage systems. Overcrowded, poor-quality housing may be associated with family breakdown, drug abuse, and antisocial behavior, which may increase transmission of human immunodeficiency virus (HIV) and other infectious agents by unsafe sex and intravenous drug use.[47]

The West Nile virus, a recently emerged infectious agent in North America, illustrates the epidemiological impact of long-distance trade and travel. It originated in Africa and had been detected sporadically in the Middle East and parts of Europe. It was unknown in North America until it arrived in New York in mid-1999, probably via an infected mosquito on an airplane. Birds were first affected, then humans. The apparently favorable conditions for viral propagation within New York City included the following:

- Early season rain and summer drought, providing ideal conditions for *Culex* mosquitoes
- The warmest July on record
- Suburban/urban ecosystems supporting large populations of selected avian host and mosquito vector species adapted to those conditions
- Large populations of susceptible bird species, especially crows
- Suburban/urban ecosystems conducive to close interaction of mosquitoes, birds, and humans

The West Nile virus then spread rapidly across the United States and has now established itself as an endemic virus, harbored by animals, including birds and horses, and transmitted via mosquitoes. The virus could spread more rapidly in Central and South America than in North America because countries there have warmer climates, large bird populations, and year-round mosquito breeding.

RISK ASSESSMENT AND RISK MANAGEMENT

The risk to human health from global environmental change can be addressed at the individual, community, regional, and global levels. The risk is a function of *(a)* the level of exposure to risk; *(b)* vulnerability (sensitivity and coping capacity); and *(c)* adaptation responses.

Exposure to risk from global environmental changes depends on the underlying weather and climate characteristics of the geographic region and the characteristics, rate, and magnitude of any changes. Sensitivity and coping capacity depend on characteristics of the individual, the population, and the region, while developmental status and demographic structure moderate the exposure response profile of the population.[48]

Coping capacity is a measure of the current ability of an individual or population to manage adverse exposure. Adaptive responses include the ability to adjust to potential harm, to take advantage of opportunities, and to cope with the medium- to- long-term consequences of environmental changes. In human societies, the ability to adapt effectively varies with wealth, access to technology, education and information, levels of skill, societal infrastructure, access to resources, management capabilities, and developmental status. There will be large disparities between the ability of rich and poor countries to adapt to global environmental change.

Risk assessment aims to identify and quantify the risk of a particular exposure to human health and well-being. By assessing the exposure and knowing the likely health impact (a function of vulnerability) in a particular population, one can examine the magnitude and frequency of risk and the risks of various groups or populations. An assessment can be made of the current coping capacity, especially to deal with risks that gradually increase (such as shrinking water supplies), and of future adaptive capacity. Risk communication then becomes important for increasing the awareness and tolerance of risk at the local, regional, and national level. Using a common metric for "risk" may help determine risk management priorities: For whom, how quickly, to what extent, and in which order should and could risks be reduced? Recent estimates (2002) by the World Health Organization (WHO) of the environmental burden of disease exemplify quantitative risk assessment, using DALYs to measure both current and projected health risks from environmental exposures. Risk assessment

should include ongoing monitoring and evaluation of the effectiveness of any risk-decreasing interventions.[48]

Classically, risk management integrates the information derived from risk assessment with other information, including socioeconomic and political concerns, to formulate public health actions to decrease or eliminate risk.[49] In the context of global environmental changes that are now unavoidable, the focus of the public health must be to minimize—rather than eliminate—risk. Risk management thus comprises *(a)* mitigation, to decrease the level of future hazardous environmental exposures; and *(b)* adaptation, to reduce the adverse effects of exposure to the hazard. For example, because of the momentum and time delays in the climate change process, immediate cessation of excess greenhouse gas emissions (mitigation) cannot preclude some level of climate change—that is, past and current emissions have already committed us to future global climate change, entailing warming of approximately 0.1°C per decade.

Adaptation versus Mitigation

Mitigation strategies to halt and reverse global environmental change require participation by the global community. Although the first industrialized countries have largely driven global environmental change, mitigation cannot be fully effective unless all countries are prepared to make the necessary changes to decrease the production of greenhouse gases, explore different energy sources, and conserve water and other renewable resources. Adaptation may be country specific, but wealth dependent, with poorer countries less able to adapt to the consequences of global environmental change. Adaptation can be responsive (to particular and immediate risks) or anticipatory (actions taken in advance of climate change effects). Although both adaptation and mitigation decrease risk, mitigation decreases risk exposure, while adaptation alters the exposure–response relationship.

Potential for Mitigation

Mitigation has been an effective strategy in reversing the effects of CFC accumulation and stratospheric ozone depletion. Mitigation, also an essential element of climate change strategies, has already been successful in reducing greenhouse gas emissions in some sectors, despite overall growth in global emissions. Carbon cycle models indicate that to stabilize atmospheric carbon dioxide concentration at 450 ppm, anthropogenic carbon dioxide emissions would need to drop below 1990 levels within a few decades. (Before the nineteenth century, atmospheric carbon dioxide concentration was stable for many centuries, at around 280 ppm.) Under the seriously inadequate mitigation strategies of the early twenty-first century, it seems inevitable that greenhouse gas emissions will continue to increase over the next few decades. Reduction of carbon dioxide emissions to 1920s levels—about one-third of current emissions—may eventually be required to prevent serious damage to ecological and other biophysical systems.[50]

Climate change mitigation has major implications at the economic, political, institutional, social, and technological levels—and for individuals, communities, and countries. A broad range of effective mitigation strategies have been described, but all require political will for effective implementation at the required scale.

Potential for Adaptation

Based on information from risk assessment, adaptive responses include the development of strategies, policies, and technological approaches to allow populations to plan and cope better with adverse health risks of environmental exposures that cannot be prevented. Global environmental changes with a single exposure route or a single outcome present a relatively straightforward situation for adaptation. Thus, adaptations to stratospheric ozone depletion through behaviors to avoid excess UVR exposure, such as sunscreen, clothing, and avoiding sunburn, are easier to advocate and implement than adaptations to the widespread and somewhat ill-defined effects of climate change due to greenhouse gas accumulation. In addition, the potential for adaptation is greater for human systems than natural systems and for rich countries compared to poor countries.

Adaptation can be considered in terms of primary, secondary, and tertiary prevention of

(Drawing by Nick Thorkelson.)

adverse health effects. Examples of primary prevention (avoiding the exposure or removing the hazardous element of the exposure) include early warning systems for extreme events. Secondary prevention or reactive adaptation includes rapid response to disasters.[7] Tertiary prevention includes better treatment of an established disease, such as malaria.

Adaptation builds on current coping capacity, including baseline strategies for dealing with risk exposure. In addition to being reactive or anticipatory, adaptation can be autonomous (actions of individuals) or planned (for whole populations) through policy decisions.

CONCLUSION

Global environmental changes—both systemic and worldwide—are now occurring. The aggregate environmental impact of humankind is now so great that it is beginning to change conditions of life on Earth. There is clear evidence of rising global temperatures, loss of stratospheric ozone, loss of biodiversity, and depletion of freshwater supplies. These changes pose new and escalating risks to human population health.

Climate change and ozone depletion are the best known examples of global environmental change. Other categories of change, less appreciated but no less important, include urbanization, ecological disruption, land degradation, disruption of elemental cycles, depletion of freshwater supplies, and global dissemination of persistent organic pollutants. There are important, complex, interactions between many of these categories of global environmental change. For example, biodiversity loss is driven by a combination of factors, including overexploitation of productive ecosystems, other land use

changes, and climate change. In turn, biodiversity loss threatens vital ecosystem services, including the provision of food, fuel, fiber, and freshwater; processing of waste; protection from floods; and stabilization of climate.

Remedying stratospheric ozone depletion is a global cooperation "success" story, although recovery of the ozone layer will take several decades. Greenhouse gas emissions, disruption of elemental cycles, global dissemination of persistent organic pollutants, and the problems associated with biodiversity loss are more complex environmental change processes to study and much more challenging, both economically and politically.

There is considerable overlap in the political and economic processes that drive global economic instability, social inequality, and ecologically unsustainable resource use.[51] The risk of conflict is likely to increase because of regional resource scarcity, arising in part from damage to environmental "goods and services." Therefore, it is in the self-interest of all people, including those in powerful nations, to reduce these large-scale environmental changes and their attendant risks to human social health, well-being, and survival.

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FURTHER READING

McMichael AJ. Human frontiers, environments and disease: past patterns, uncertain futures. Cambridge, England: Cambridge University Press, 2001.

The expansion of human frontiers—geographic, climatic, cultural, and technological—has encountered frequent setbacks from disease, famine, and dwindling resources. However, recognition of how environmental change can limit health and survival has been slow. Over many millennia, disease and longevity profiles in populations have reflected changes in environmental conditions and, often, exceedances of carrying capacity. Today, population growth and the aggregated pressures of consumption and emissions are beginning to impair various global environmental systems. The research tasks in detecting, attributing, and projecting the resultant health effects are complex. Have recent health gains, in part, depended on depleting natural environmental capital? A summary of this book is available as: McMichael AJ. Population, environment, disease, and survival: past patterns, uncertain futures. Lancet 2002; 359: 1145–1148. Available at: http://www.thelancet.com/journal/vol359/iss9312/full/llan.359.9312.editorial_and_review.20512.1

WHO/WMO/UNEP 2003: Climate change and human health—risks and responses. Summary available at: http://www.who.int/globalchange/publications/cchhsummary/en/

Climate change poses a major, and largely unfamiliar, challenge. This publication describes the process of global climate change, its current and future impacts on human health, and how our societies can lessen those adverse impacts, via adaptation strategies and by reducing greenhouse gas emissions.

Intergovernmental Panel on Climate Change. IPCC Fourth Assessment Report. Geneva: Intergovernmental Panel on Climate Change, 2007. Available at: http://www.ipcc.ch/

IPCC assessments attempt to answer such general questions as: Has the Earth's climate changed as a result of human activities? In what ways is climate projected to change in the future? How vulnerable are agriculture, water supply, ecosystems, coastal infrastructure, and human health to different levels of change in climate and sea level? What is the technical, economic, and market potential of options to adapt to climate change or reduce emissions of the gases that influence climate?

Environmental Health Criteria 160. Available at: www.who.int/uv/publications/EHC160/en/

Environmental Health Criteria 160 is a comprehensive review of the effects of ultraviolet radiation on human health and the environment. Although it is now 10 years old, it provides an excellent basis for understanding the range of health effects associated with excessive UVR exposure.

Norval M, Cullen AP, de Gruijl FR, et al. The effects on human health from stratospheric ozone depletion and its interactions with climate change. Photochemistry and Photobiological Science 2007; 6: 232–251.

This is the most recent report of the UNEP on the health effects of stratospheric ozone depletion and is an excellent review of the evidence.

SECTION II

HAZARDOUS EXPOSURES

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6

Outdoor Air Pollution

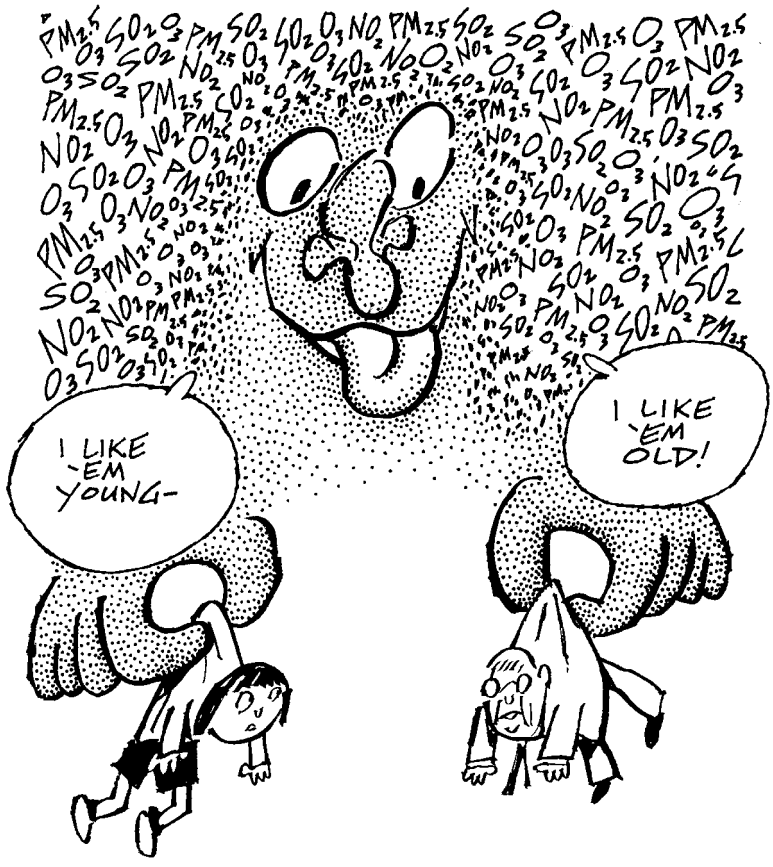
Isabelle Romieu, Mauricio Hernández-Ávila, and Fernando Holguin

In the mid-twentieth century, dramatic episodes of outdoor (ambient) air pollution in developed countries showed that air pollution could cause excess deaths. For example, during the London Fog of 1952, which was due mainly to smoke from coal-burning household stoves, several thousand excess deaths occurred. Infants and young children as well as older people were at especially increased risk, and the proportion of deaths attributed to respiratory causes was increased.[1] Ambient air pollution has now been examined as a risk factor for respiratory and cardiovascular morbidity and mortality in numerous epidemiologic studies.[2–5] Although pollutant levels have decreased in developed countries, the epidemiologic evidence demonstrates adverse health effects at levels, which are frequently reached in many urban areas, that were previously considered to be safe.[6,7] The World Health Organization (WHO) has estimated the burden of disease related to urban air pollution to be 6.4 million DALY (disability-adjusted life-years, accounting for both years of life lost to premature mortality and years of life lived with disability due to disease).[8]

NATURE AND SOURCES OF AMBIENT AIR POLLUTION

In both developed and developing countries, ambient air pollutants are derived mainly from fuel combustion (Fig. 6-1). They include *(a)* primary pollutants, such as sulfur dioxide, oxides of nitrogen, and particulate matter; *(b)* secondary acidic aerosols and other particles; and *(c)* oxidant pollutants (primarily ozone) that are produced by photochemical reactions involving hydrocarbons and oxides of nitrogen. In most areas where photochemical reactions are present, the emissions of oxides of nitrogen and hydrocarbons reflect urban sprawl (Chapter 39), with heavy motor vehicle traffic that is often associated with high levels of particulates—especially in the large cities of developing countries.[3,7,9] In cities and some rural areas of developing countries, residential space-heating and cooking with solid fuels (biomass and coal) can also contribute significantly to ambient air pollution (see Box 7-2 in Chapter 7). Industrial processes also emit contaminants, such as volatile organic compounds, that may adversely affect health.

To understand relevant health effects, one should have a basic understanding of the sources



(Drawing by Nick Thorkelson.)

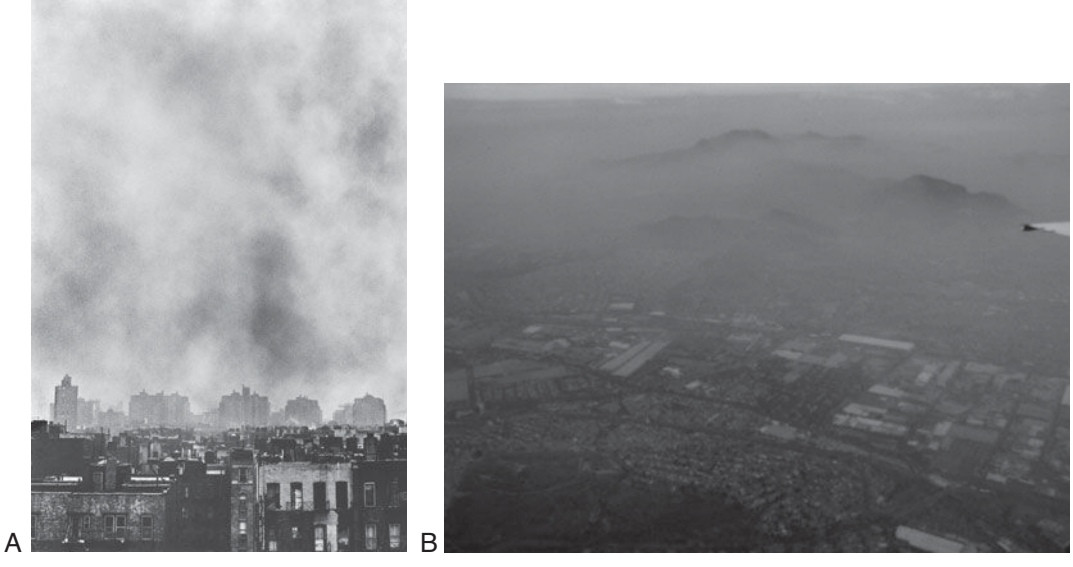


Figure 6-1. Outdoor air pollution has been a major problem in urban areas of both developed and developing countries. (A) Air pollution in New York City in 1968. (Photograph by Earl Dotter.) (B) Air pollution in Mexico City in 2000. (Photograph by Fernando Holguin.)

and properties of major ambient air pollutants, including sulfur dioxide, particulates, oxides of nitrogen, ozone, volatile organic compounds, carbon monoxide, persistent organic pollutants, and lead.

Sulfur Dioxide

Sulfur dioxide (SO_2) is a water-soluble gas formed from the oxidation of sulfur, which contaminates coal and petroleum fuels. Consequently, sulfur dioxide is emitted by coal- and oil-fired power plants and by industrial processes involving fossil fuel combustion. Sulfur dioxide and particulate pollution are typically emitted together by combustion sources and exist as components of a complex mixture.[3] However, depending on the source, the proportion of particulates to sulfur dioxide varies greatly. For example, in areas where low-sulfur fuel is used, the ambient sulfur dioxide level is low. In contrast, in areas where high-sulfur fuel is used or where much coal is burned, such as China, the ambient level of sulfur dioxide is high.

Particulates

Particulate air pollution refers to the mixture of solid and liquid particles suspended in the air that form an aerosol. The particles in air vary in shape, size, composition, and origin. Typically particles are classified according to their size (Fig. 6-2).[10] Particle size affects deposition in the respiratory tract and, consequently, the potential to cause adverse health effects. Particles less than 10 μm in diameter (PM_{10}) comprise the "inhalable" fraction of airborne particles. Particles between 2.5 and 10 μm, the "coarse" fraction, include mainly soil material, such as suspended road dust and windblown dust, and particles generated by handling, crushing, and grinding operations. Particles less than 2.5 μm ($PM_{2.5}$), the "respirable" or "fine" fraction, comprise all particles capable of entering the alveoli. They are produced from fuel and biomass combustion and the atmospheric reaction of gases. A subset of $PM_{2.5}$, "ultrafine" particles smaller than 0.1 μm, are formed by combustion exhaust.[10]

Oxides of Nitrogen

Like sulfur dioxide, nitrogen dioxide (NO_2) and other oxides of nitrogen (NOx) are produced by high-temperature combustion processes and contribute to the formation of acid aerosols. Outdoors, oxides of nitrogen are nearly always present together with other combustion pollutants. Initially, almost all oxides of nitrogen emissions are in the form of nitric oxide (NO), which

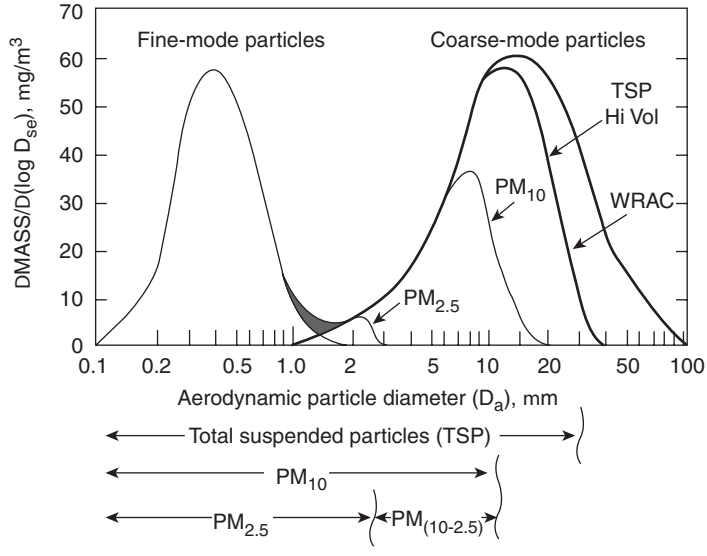


Figure 6-2. Example of a mass distribution of ambient particulate matter (PM) as a function aerodynamic particle diameter. (*Source*: EPA. Air quality criteria for particulate matter. National Center for Environmental Assessment, 1996.)

is then oxidized in air to form nitrogen dioxide, a more toxic compound and a major precursor of photochemical smog.[10]

Ozone

Ozone (O_3) is a colorless gas that occurs naturally in the stratosphere, where it filters out ultraviolet (UV) radiation. At ground level in cities and many rural areas, ozone is the prime oxidant ingredient of smog, along with other oxidant species and fine particles. Ozone is a secondary pollutant formed as the product of the atmospheric photochemical reaction of primary emissions, such as oxides of nitrogen and volatile organic compounds, in the presence of sunlight and accelerated at high temperature.[2] This photochemical pollution is especially prevalent in the many large cities with heavy vehicle traffic, especially those located in sunny regions and/or at high altitude, such as Mexico City.[7]

Volatile Organic Compounds

Volatile organic compounds (VOCs) are present in the atmosphere, mainly as gases. They include a variety of hydrocarbons, such as alkenes, aldehydes, and aromatic hydrocarbons (including benzene and toluene). Some VOCs are chlorinated compounds. The sources of VOCs include evaporation and combustion of fossil fuels, use of solvents, and industrial processes. Benzene, a VOC that has received much attention because of its carcinogenicity (see Chapter 17), is present in gasoline. Population exposure in urban areas to benzene depends on its concentration in the gasoline used in the area.[10]

Carbon Monoxide

Carbon monoxide (CO) is produced by the incomplete combustion of fossil fuels, mainly derived from mobile sources. Most of the carbon in automotive fuel is oxidized to carbon dioxide, with only a small fraction incompletely oxidized to carbon monoxide.[11]

Persistent Organic Pollutants

Persistent organic pollutants (POPs) are a subclass of air toxicants that persist for long periods in the environment. A recent international treaty aims to eliminate 12 of these compounds from the environment, including eight pesticides (aldrin, chlordane, dieldrin, endrin, heptachlor, mirex, toxaphene, and DDT); an industrial chemical (hexachlorobenzene) and a group of industrial chemicals (polychlorinated biphenyls [PCBs]); and two types of combustion byproducts (dioxin and furans).[10] Because they are volatile, POPs travel great distances in the atmosphere, settling out in colder regions, where they become incorporated into the food chain. Exposure is primarily via ingestion.

Lead

Population exposure to lead, as a gasoline additive, is decreasing as leaded gasoline is being phased out in many countries. Leaded gasoline, however, is still used in many developing countries. The primary air pollutant is lead oxide, a product of gasoline combustion.

In the United States, removal of lead from gasoline lowered the average blood lead level from 13 to 3 μg/dL.[12] In Mexico City, control measures implemented from 1988 to 1998 to phase out lead from gasoline lowered the annual ambient lead level from 1.2 to 0.2 μg/m^3.[13] Simultaneously, an estimated 7.6 μg/dL average decline in blood lead level was observed in children living in Mexico City.[6] (See Chapters 11 and 19.)

AMBIENT AIR QUALITY STANDARDS AND GUIDELINES

In the past 30 years, much progress has been made in many countries to control ambient air pollution and thereby reduce adverse impacts on human health and the environment. In the United States, the Clean Air Act of 1970 mandated that the federal government develop and promulgate national ambient air quality standards (NAAQSs), specifying uniform nationwide limits for certain major air pollutants ("criteria air pollutants"): carbon monoxide, lead, nitrogen dioxide, ozone, particulate matter, and sulfur dioxide. The Act has been amended several times, most recently in 1990.[14] (See Chapter 30.)

Under the Act, the Environmental Protection Agency (EPA) must identify pollutants that "may reasonably be anticipated to endanger public health or welfare" and issue air quality criteria for them—"primary" and "secondary" NAAQSs for these pollutants. Primary standards set limits to protect public health, including the health of sensitive populations, such as people with asthma, children, and older people. Secondary standards protect against other effects, such as decreased visibility and damage to animals, crops, vegetation, and buildings. Standards are set for two types of averaging time periods: long-term (such as annual average) and short-term (such as 24 hours or less) (Table 6-1). The Act requires that NAAQSs be reviewed periodically and, if appropriate, revised. In 1997, the NAAQS for ozone was revised and the NAAQS for $PM_{2.5}$ was added, which the EPA is frequently revising.

The World Health Organization (WHO) has developed air-quality guidelines for international use, which can be accessed at: http://www.who.ch/pll/dsa. These guidelines, which consist of concentration limits of air pollutants for certain averaging times that were recommended by international experts, are intended for consideration by national and international authorities in promulgating air quality standards.[14]

EXPOSURE ASSESSMENT

Individuals within a population differ considerably in their exposure to air pollutants. However, nearly all routine monitoring and regulation of air pollution are based upon measurements that are conducted at fixed locations. Assessment of individual and population exposure to air pollution should consider variations of sources of exposure among individuals.[10] Personal exposure assessments encompass *(a)* identification of key sources of selected pollutants, *(b)* their emission rates, *(c)* their concentration in outdoor and indoor air; and *(d)* the duration of contact with the pollutants (Fig. 6-3).[15] Knowledge of where people are and what they do during the course of a typical day is essential for determining personal exposure.

People living in North America spend, on average, approximately 87% of their time indoors.

Table 6-1. National Ambient Air Quality Standards, United States*

	Primary Standards		
Pollutants	Level	Averaging Time	Secondary Standards
Carbon monoxide	9 ppm (10 mg/m^3)	8-hour	None
	35 ppm (40 mg/m^3)	1-hour	None
Lead	0.15 μg/m^3	Rolling 3-month avg.	Same as primary
	1.5 μg/m^3	Quarterly avg.	Same as primary
Nitrogen dioxide	53 ppb	Annual (arithmetic avg.)	Same as primary
	100 ppb	1-hour	None
Particulate matter (PM_{10})	150 μg/m^3	24-hour	Same as primary
Particulate matter ($PM_{2.5}$)	15.0 μg/m^3	Annual (arithmetic avg.)	Same as primary
	35 μg/m^3	24-hour	Same as primary
Ozone	0.075 ppm (2008 std.)	8-hour	Same as primary
	0.08 ppm (1997 std.)	8-hour	Same as primary
Sulfur dioxide	0.03 ppm**	Annual (arithmetic avg.)	0.5 ppm 3-hour
	0.14 ppm**	24-hour	0.5 ppm 3-hour
	75 ppb	1-hour	None

*For detailed information on scientific bases and policy considerations underlying decisions establishing the NAAQS listed here, see the air quality criteria, staff papers, and NAAQS promulgation notices cited in text. Such information can also be obtained from several Web sites, such as http://www.epa.gov/air/criteria.html, http://www.epa.gov/oar/oaqps/publicat.html, and http://www.epa.gov/ncea/biblio.htm. ppm, parts per million; ppb, parts per billion; avg., average; std., standard.
**Revocation anticipated.

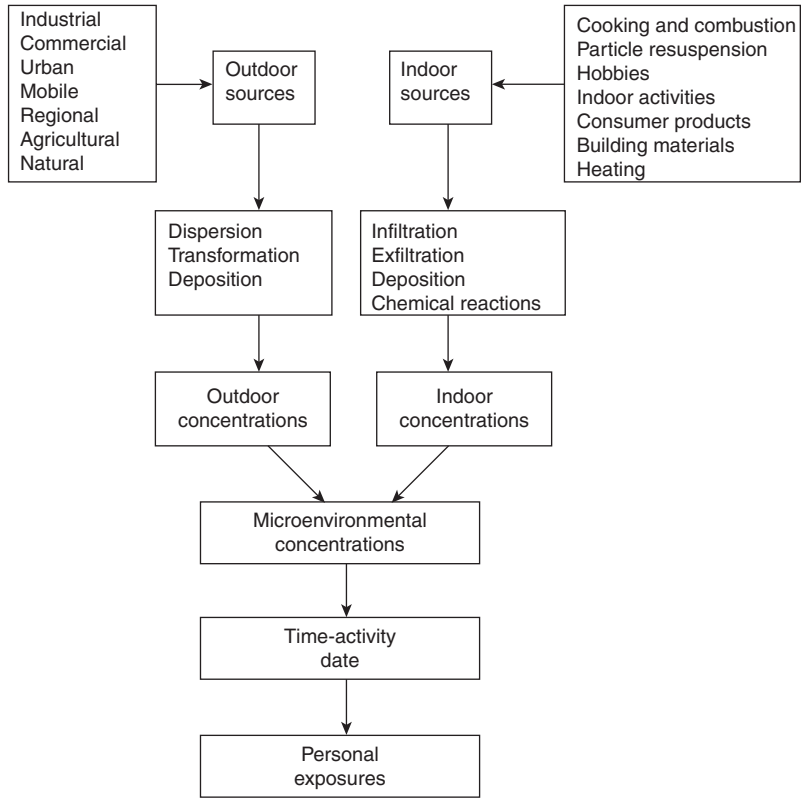


Figure 6-3. Sources and factors influencing indoor, outdoor, and personal exposures to air pollutants. (*Source*: Ozkaynak H. Exposure assessment. In: Holgate ST, Koren HS, Samet JM, Maynard RL [eds.]. Air pollution and health. London: Academic Press, 1999, pp. 149-162.)

People living in urban areas of developing countries also spend most of their time indoors. When indoors, individuals are exposed to outdoor air pollutants that penetrate inside as well as to pollutants that are generated inside. (See Chapter 7.) If particles as small as 1 μm are considered, the correlation between indoor and outdoor air concentrations is very high. Penetration of outdoor air pollutants to indoor air is a function of the exchange rate, which is determined by type of construction and use of air conditioning. Carbon dioxide, sulfur dioxide, and nitrogen dioxide penetrate from outdoor to indoor air with great efficiency.[10] Ozone exposure is directly related to the amount of time spent outdoors.[10] An estimated 70% of fine particles ($PM_{2.5}$) from outdoors penetrates indoors, in the absence of air conditioning.

Three factors govern the risk of toxic injury from pollutants and their metabolites: *(a)* their chemical and physical properties, *(b)* the dose that reaches critical tissues, and *(c)* the responsiveness of these sites to the pollutants and their metabolites. The physical form and properties, such as the solubility of airborne contaminants, influences distribution in the atmosphere and body tissues—and, therefore, the dose delivered to the target site (critical tissues). Dose is very difficult, if not impossible, to determine in epidemiological studies; therefore, surrogate measures are used, ranging from atmospheric concentration of pollutants to concentrations of biomarkers. For some pollutants, mathematical models of the relationship between exposure and dose can be used to develop surrogate measures. The interaction of pollutants with

biological receptors can trigger mechanisms of toxic response, by direct stimulation or a cascade of molecular and cellular events that ultimately damages tissues.[3,16] Different pathways of pollutant sources—from exposure to inhalation to toxic effects—are shown in Figure 6-4.

GLOBAL CONCENTRATION PATTERNS OF AMBIENT AIR POLLUTION

During the past 25 years, in developed countries, the generally measured indicators of urban air quality have tended to improve. In contrast, in many developing countries, higher levels of ambient air pollution have resulted from rapid growth of urban population, development of industry, intensification of traffic, limited availability to clean fuel, and lack of effective control programs.[7,17] The Air Management Information System (AMIS) of WHO[17] provides comparative data from cities in more than 60 countries for major air pollutant levels (see http://www.cepis.ops-oms.org/enwww/aire/amis.html). Figure 6-5 presents data on the global distribution of PM_{10} concentrations and cumulative percentage of

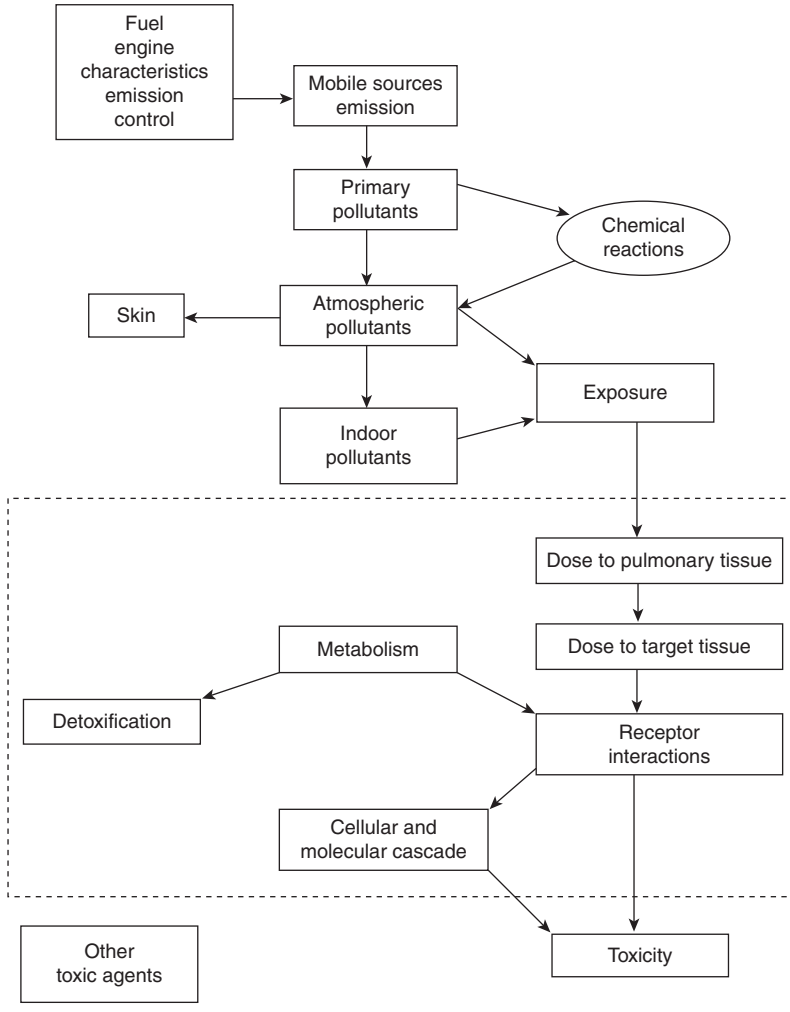


Figure 6-4. Pathway from motor vehicle pollutant sources to toxic effects in humans by exposure through inhalation. (*Source*: Watson AY, Bates RR, Kennedy D [eds.]. Air pollution, the automobile, and public health. Sponsored by the Health Effects Institute. Washington, DC: National Academy Press, 1988, p. 21.)

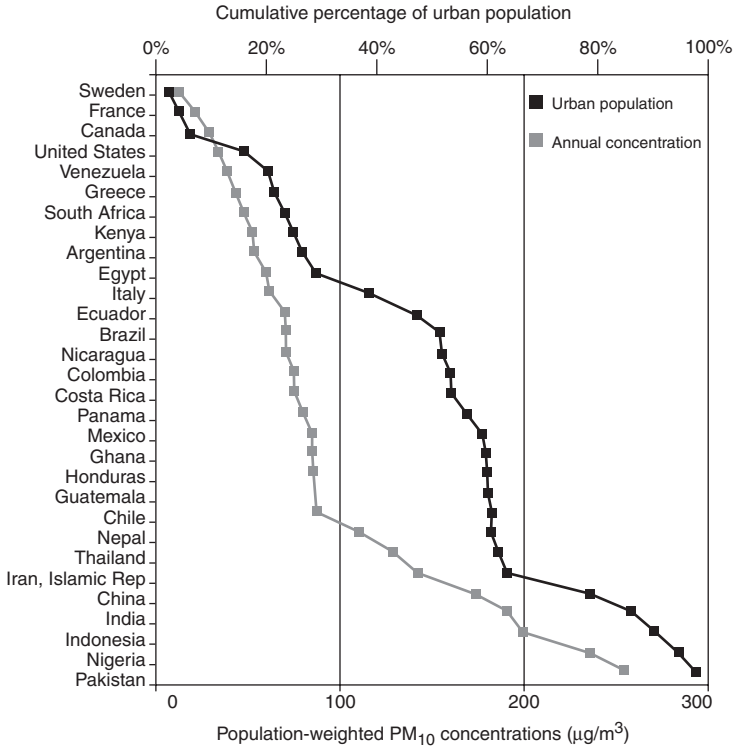


Figure 6-5. Cumulative percentage of PM_{10} (particulate matter less than 10 μm in aerodynamic diameter) and average annual concentration in urban populations, by country. Population-weighted concentrations are averages that take into account both air pollution levels and the number of people exposed in each country. For example, the average annual concentration of PM_{10} in cities in Pakistan is approximately 260 μg/m^3, and approximately all of the world's urban population experiences less air pollution than do urban residents in Pakistan. Similarly, the average annual concentration of PM_{10} in cities in India is approximately 190 μg/m^3, and approximately 90% of the world's urban population experiences less air pollution than do urban dwellers in India. (*Source*: Holdren JP, Smith KR. Energy, the environment and health. In: Goldenburg J, [ed.]. World energy assessment: energy and the challenge of sustainability. New York: United Nations Publication, 2000, pp. 61-110. Reprinted with the permission of the publisher.)

urban population exposed to these levels, by country.[18]

ADVERSE HEALTH EFFECTS OF AMBIENT AIR POLLUTANTS

Adverse health effects ascribed to exposure to ambient air pollution include excess cardiorespiratory mortality, exacerbation of asthma, increased respiratory symptoms and illnesses, decreased lung function, and reduced host defense (Table 6-2).[2] The evidence linking these effects to air pollution comes from studies based on animal toxicology, human clinical exposure, field exposure, and epidemiology. Some of the outcomes listed in Table 6-2, such as increased deaths and hospitalizations, are clearly adverse, while others, such as elevated levels of biomarkers (including inflammatory mediators in bronchoalveolar lavage [BAL] fluid), have uncertain clinical significance.[2]

Levels of ambient air pollutants usually correlate with one another, either because *(a)* emission sources are common to different pollutants (vehicles emit particles, oxides of nitrogen, and carbon monoxide); or *(b)* pollutants interact in the atmosphere (as is the case of ozone and the secondary aerosols that are part of $PM_{2.5}$). Although the health effects of specific pollutants have been studied separately, and are regulated and controlled separately, mixtures of specific

Table 6-2. Health Effects and Biologic Markers of Response Associated with Air Pollution

Excess cardiorespiratory mortality
Excess deaths from heart or lung disease
Increased health care utilization
Increased hospitalization, physician visits, and emergency department visits
Asthma exacerbations
Increased physician visits, and medication use
Decreased peak-flow measurements
Increased respiratory illness
Increased upper and lower respiratory infections
Increased physician visits, and episodic symptoms
Increased respiratory symptoms
Wheezing
Cough/phlegm
Chest tightness
Decreased lung function
Acute reduction
Chronic reduction
Increased airways reactivity
Altered response to challenge with methacholine, carbachol, histamine, and cold air
Increased lung inflammation
Influx of inflammatory cells, mediators, proteins
Decreased heart-rate variability
Increased systemic inflammatory markers
Fibrinogen
C-reactive protein
Increased plasma viscosity
Altered host defense
Altered mucociliary clearance, macrophage function, and immune response
Eye, nose, and throat irritation

pollutants commonly occur and may be responsible for observed effects.[2] Such mixtures may lead to difficulties in interpreting epidemiological data. Correct interpretation of data on chronic human exposures often depends on *(a)* comparing results from different locations, and *(b)* considering results of acute human exposures and animal experiments as indications of the adverse health effects of the primary pollutant.[10]

In the next section, we will consider only the adverse health effects of the *criteria air pollutants* regulated by the NAAQS, except for lead, which is discussed in Chapters 11, 19, and 20. (See Table 6-3.)

Particulate Matter and Sulfur Dioxide

The health effects of particulate matter and sulfur dioxide are presented together because they are both products of fossil fuel combustion and are often, but not always, present together in complex mixtures. Epidemiological studies suggest *(a)* an increase in mortality and morbidity associated with levels of airborne particles below the current standards, and *(b)* approximately twice the previously reported effect of fine particles (smaller than 2.5 μm), which appear to contain more of the reactive substances linked to adverse health effects.[19,20]

Experimental Studies

Sulfur dioxide may cause bronchitis-like pathology in animals exposed to levels far above ambient air concentrations. In asthmatics, exposure to 0.25 to 0.5 ppm elicits acute bronchoconstriction associated with increased airway resistance and decreased air flow rates. Sulfur dioxide can also reduce various aspects of pulmonary defense.[21]

Fine particulates, especially ultrafine particles (<100 nm), are toxic to the lungs due to their small size, particle surface area, number, particle surface chemistry, oxidative stress, and interstitialization of particles (their potential for becoming internalized from the airway lumen into the interstitial space).[22] In addition, transition metals (those with the capacity of generating free radicals) contained in particles may cause oxidative stress, augmented by the release of reactive oxygen species by the inflammatory cell influx that results from the primary interaction between the lung and particles. Short-term exposure to high levels of concentrated ambient particles (200 $\mu g/m^3$ of $PM_{2.5}$) can induce a transient, mild pulmonary inflammatory reaction[22] and changes in both blood indices and heart-rate variability.[23] Figure 6-6 summarizes the pathways by which deposition of particles in the airways can induce effects, both in the airways and systematically, that may lead to adverse health effects.[24] In addition, combustion products may modulate the immune system, impairing inflammatory and host-defense functions of the lung, and acting in synergy with allergens to enhance allergen-specific IgE production, initiate a TH_2 cytokine environment, and promote primary allergic sensitization.[25] (A TH_2 cytokine environment is one in which there is a phenotypical immune response, characterized by a subpopulation of lymphocytes that produce

Table 6-3. Health Effects of Air Pollutants and Populations at Greatest Risk

Agent	Susceptible Population	Clinical Consequences	Other Information
Particles (PM_{10} and $PM_{2.5}$)	Children People with chronic heart and lung disease, including asthma	Increased acute cardiovascular and respiratory mortality Increased cardiovascular mortality with chronic exposure Increased hospital admissions for respiratory and cardiac conditions Increased respiratory symptoms Decreased lung function Increased asthma exacerbations Increased prevalence of chronic bronchitis Increased risk of lung cancer Increased blood fibrinogen Increased inflammatory markers Reduced heart-rate variability	Effects seen alone or in combination with sulfur dioxide Probable effects: • Acute respiratory infections in children • Decreased rate in lung function growth • Low birth rate • Postneonatal mortality
Sulfur dioxide	Healthy adults and COPD patients People with asthma	Increased respiratory symptoms Increased respiratory mortality and increase hospital visits for respiratory disease Acute bronchoconstriction in people with asthma	Highly soluble gas with little penetration to distal airways Observations related to short-term exposures
Acid aerosols	Healthy adults Children People with asthma Others	Increased respiratory illness Decreased lung function Increased hospitalizations	Currently not a criteria pollutant; no NAAQS established Effects seen in combination with ozone and particles
Ozone	Athletes Outdoor workers People with asthma and other respiratory illnesses Children	Increased hospital admissions for acute respiratory illnesses Aggravation of asthma Increased bronchial responsiveness Decreased lung function Lung inflammation Increased respiratory symptoms Decreased exercise capacity Increased hospitalizations	Effects found at or below current NAAQS; effects increased with exercise Effects seen in combination with acid aerosols and particles Probable effects: • Increase of mortality • Aggravation of acute respiratory infections • Chronic bronchiolitis with repetitive exposure
Nitrogen dioxide	Children with asthma Young children	Increased respiratory morbidity Increased airway reactivity Decreased lung function Increased respiratory symptoms Increased respiratory illnesses	Effects occur at levels found indoors with unvented sources of combustion
Carbon monoxide	Healthy adults Patients with ischemic heart disease	Increased cardiac ischemia Decreased exercise capacity	Effects increase with anemia or chronic lung disease Possible effects: • Low birthweight • Preterm birth

COPD, chronic obstructive pulmonary disease; NAAQS, National Ambient Air Quality Standards.

cytokines involved in allergic inflammation, such as IL-5 and IL-13.)

Population-Based Studies of Mortality

Acute exposure to airborne particulates increases mortality. Occurrence of deaths is related to daily changes in air pollution levels.[3,19] A study in 20 U.S. cities found, per 10 μg/m^3 increase of PM_{10}, increases in total mortality (0.51%) and cardiovascular mortality (0.68%).[26,27] The relationship appears to be linear down to the lowest levels, without any threshold. People who otherwise

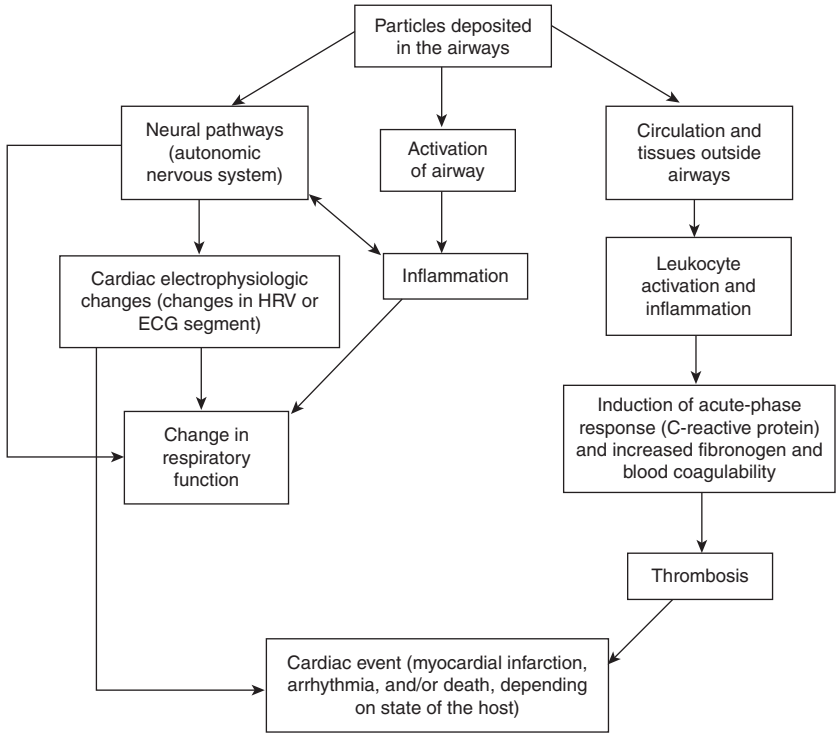


Figure 6-6. Adverse effects of particles on airways and the cardiovascular system: Possible pathways. ECG, electrocardiogram; HRV, heart rate variability. (*Source*: Adapted from Health Effects Institute. Understanding the health effects of components of the particulate matter mix: progress and next steps. Boston, MA: Health Effects Institute, 2002.)

might have survived for a long time are among those who died. Daily mortality appears to be more strongly associated with concentrations of $PM_{2.5}$ than with concentrations of larger particles[20]—an important finding for urban areas of developing countries, where vehicular traffic with poorly maintained engines and extensive use of diesel fuel is a major source of particulate pollution. Increased infant mortality in these countries has been partially linked to exposure to airborne particulates.[7,9]

It has been estimated, based on these data, that each 10 ug/m^3 increase in PM_{10} causes an increase of 1% in nonaccidental deaths in young children (less than 5 years of age).[28] Chronic exposure to fine particulates also increases mortality.[29] Fine particulate pollution seem to be associated with mortality from all causes combined, cardiopulmonary diseases, and lung cancer. Each 10 μg/m^3 elevation in fine-particulate air pollution has been associated with a 4% increase in all-cause mortality, a 6% increase in cardiopulmonary mortality, and an 8% increase in lung cancer mortality.[30] Cardiopulmonary mortality has also been associated with residing near major roads.[31]

Population-Based Studies of Morbidity

Acute exposure to high concentrations of sulfur dioxide can cause bronchoconstriction, chemical bronchitis, and tracheitis. Asthmatics appear to be more sensitive. There is a linear exposure–response relationship.[3]

Acute exposure to particulates has been associated with morbidity in children and older people. Among children, particulates have been associated with emergency department visits and hospital admissions, increased respiratory illnesses (including upper respiratory infection and pneumonia), respiratory symptoms, and decrease in lung function.[3,32] Among older people, ambient levels of PM_{10} have been associated with increased hospital admissions for respiratory illnesses (including chronic obstructive pulmonary disease and pneumonia) and

cardiovascular disorders (including ischemic heart disease).[26] Most studies show an increase of 1% to 2% in illness occurrence with each 10 μg/m^3 increase in PM_{10}. Ambient levels of particulates have also been associated with increases in systemic inflammatory markers, such as fibrinogen, C-reactive protein, and plasma viscosity, and decreases in neural control of heart function, such as decreases in heart rate and heart-rate variability.[24] People with asthma appear to be more susceptible to the impact of PM_{10}, with increases in respiratory symptoms and decreases in lung function.[2,19] In addition, diesel particulates increase allergic response and might lead to the development of allergy and asthma.[5,6] Long-term exposure to sulfur dioxide has been related to chronic bronchitis, especially in cigarette smokers.[3]

Long-term exposure to particulate air pollution has been associated with chronic cough, bronchitis, and chest illness—a 10 μg/m^3 increase in PM_{10} seems to be associated with a 5% to 25% increase in bronchitis or chronic cough, and a 1% to 3% decrease in lung function.[4] Exposure to particulates may lead to a reduction in maximum attained lung function, which occurs early in adult life and, ultimately, to an increased risk of chronic respiratory illness.[32,33]

Adverse effects of particulates and sulfur dioxide on fetal growth, preterm birth, birthweight, and other pregnancy outcomes have been reported,[6,33] but further studies are needed to verify these findings.

Ozone

Ozone is a powerful oxidant that can react with a wide range of cellular components and biological material. Its biological effects are likely caused by intermediates, such as free radicals, lipid hydroperoxides, aldehydes, and hydrogen peroxide. The primary target organ for ozone is the lung, where exposure produces cellular and structural changes that reduce lung function.

Experimental Studies

Ozone causes oxidation or peroxidation of biomolecules via free radical reaction. Ozone reacts rapidly with substrates in the lung lining fluid, preferentially with antioxidants; however, low content of lung lining fluid (antioxidant defense) or high ozone exposure leads to the oxidation product responsible for adverse health effects. Lipoperoxidation of polyunsaturated fatty acids (PUFAs) of lung cells can release arachidonic acid, which subsequently is converted into protaglandine E_2 (PGE_2) and prostaglandine F_{2alpha}, which act on neuroreceptors in airways and induce an inflammatory response.[34]

Ozone damages the respiratory tract, increasing permeability and inflammation, which causes morphological, biochemical, and functional changes and decreases host defenses. Ozone exposure causes a major lesion in the centriacinar area, affecting the efficiency of gas exchange. It causes fibrotic changes in animals,[35] which are believed to cause airflow obstruction.

In humans, ozone induces an increase in the constituents of the bronchoalveolar lavage fluid. Levels of ozone frequently measured in urban areas in the United States reduce lung function. Decrement in lung function and physical performance, aggravation of respiratory tract symptoms, increased airway reactivity, and acute inflammation have been observed at exposure levels as low as 0.08 ppm. Acute reversible reductions in lung function have been observed in exercising children exposed to 0.12 ppm.[3,34] After repeated exposure to ozone, decrement in lung function observed after single exposures attenuated, suggesting adaptation, but airway inflammation persisted despite attenuation of some markers of inflammation.

These studies, taken together, show wide, but reproducible, variability among individuals' sensitivity to ozone, and they suggest that adults and children who engage in prolonged exercise or work outdoors may be at risk of adverse health effects of ozone concentrations near the ambient standard.[36]

Population-Based Studies of Mortality

Ozone is related to daily mortality counts when levels of ozone in the air are high. A pooling of 15 time-series studies found that an increase of 200 μg/m^3 (100 ppb) in ozone was associated with a 3.6% increase in mortality.[37]

Population-Based Studies of Morbidity

Exposure of healthy individuals, including children, to relatively low concentrations of ozone can cause lung inflammation, acutely decreased

lung function, and respiratory impairment—increases in emergency department visits and hospital admissions due to respiratory diseases, respiratory symptoms (such as cough, throat dryness, and chest discomfort and pain) as well as headache and eye discomfort, and temporary lung function decrements. The combined relative risk estimate for respiratory-disease hospital admission from major time-series studies for all ages has been estimated at 1.18 per 100 ppb increase in daily 1-hour maximum ozone concentrations.[36] People with asthma appear to be more sensitive to ozone exposure; increases in respiratory-related emergency department visits and symptoms and decreases in lung function have been reported.[3,6] In clinical studies, ozone potentiates the effect of allergen exposure in sensitive people with asthma, perhaps as a consequence of increased penetrability of the respiratory epithelium from ozone exposure.[34]

Because of the acute health effects associated with short-term ozone exposure and the available studies on long-term animal exposure to ozone, there is concern that long-term exposure may have a cumulative adverse impact on human health. Long-term ozone-exposure studies suggest both a decrease in baseline pulmonary function and induction of new cases of asthma.[36] Ozone exposure has been linked to the incidence of asthma in children participating in heavy-exercise activities in communities with high-ozone concentration.[33]

Nitrogen Dioxide

Nitrogen dioxide is highly reactive and causes bronchitis and pneumonia and increases susceptibility to respiratory infections. Much exposure to nitrogen dioxide takes place indoors, where sources include cooking stoves and space heaters. Brief exposure to concentrations as high as 0.5 ppm may be experienced while cooking with gas stoves or driving in traffic.[37] In ambient air, nitrogen dioxide does not generally occur alone, but as part of a complex mixture of primary and secondary pollutants. Consequently, characterizing the effects of nitrogen dioxide in ambient air has proved difficult. The contribution of nitrogen dioxide to secondary particles and its role in the formation of ozone may be more relevant to public health than any of its direct effects.

Experimental Studies

Animal experiments show that exposure to nitrogen dioxide concentrations of an order of magnitude greater than those generally found in ambient urban air can impair both the cellular and humoral immunological mechanisms of the lung.[38]

Population-Based Studies of Mortality

There is a significant positive association between daily deaths and nitrogen dioxide. Increases of 50 $\mu g/m^3$ (26.5 ppb) in nitrogen dioxide have been associated with a 1.3% increase in daily deaths. Nitrogen dioxide has also been associated with daily mortality in children under 5 years of age and intrauterine mortality.[6]

Population-Based Studies of Morbidity

Studies of short-term effects among children and adults related to outdoor exposure have been inconsistent. One study indicated an increase in asthma admissions related to ambient levels of nitrogen dioxide.[39] In certain occupations, workers are intermittently exposed to high concentrations of oxides of nitrogen. The spectrum of pathological effects in the lung resulting from occupational exposure to oxides of nitrogen range from mild inflammatory response in the tracheobronchial mucosa at low concentrations to bronchitis, bronchopneumonia, and acute pulmonary edema at high concentrations.[3]

Long-term exposure to outdoor nitrogen dioxide has been associated with increased chronic respiratory symptoms and infections among children and possibly to a decrease in lung function. A meta-analysis of 11 epidemiological studies reported an increase in respiratory illness in children under age 12, associated with long-term exposure to high concentrations of nitrogen dioxide from gas stoves, as compared with low concentrations.[40] One study showed a significant deficit in lung growth related to nitrogen dioxide and fine particulate exposure, and more frequent chronic cough and phlegm among children with asthma in communities with higher nitrogen dioxide exposure.[33] Among adults, chronic nitrogen dioxide exposure has been associated with increased respiratory symptoms and reduced lung function.

Carbon Monoxide

High exposure to carbon monoxide occurs primarily in certain occupations (such as firefighting), in suicide, and in unintended poisoning (such as due to defective or improperly used combustion devices). High exposure can cause acute poisoning, resulting in coma and death. Most fatal carbon monoxide poisoning occurs in confined spaces, such as inside garages, automobiles, weather-sealed houses, and ice skating rinks. However, episodes of fatal carbon monoxide poisoning have been documented outdoors, such as among those using recreational houseboats near generators and other gasoline-powered motor exhaust, which has prompted hazard warnings and boat redesign. Although outdoor exposures to carbon monoxide in urban settings are generally several orders of magnitude lower than those associated with intoxication or poisoning, some exposures during urban activities may adversely affect the heart and the brain, the most oxygen-sensitive organs.[3] People who suffer from cardiovascular disease, particularly those with angina or peripheral vascular disease, are much more susceptible to the adverse health effects of carbon monoxide.

In the lungs, carbon monoxide is rapidly absorbed into the blood, where it binds to hemoglobin (Hb) and forms carboxyhemoglobin (COHb), which impairs the oxygen-carrying capacity of the blood. The dissociation of oxyhemoglobin is also reduced due to the presence of COHb in the blood, thereby further impairing the oxygen supply to tissues. (The affinity of Hb for carbon monoxide is about 240 times that of oxygen.) The main factors influencing the uptake of carbon monoxide are the intensity of physical activity, body size, the condition of the lung, and barometric pressure. Table 6-4 shows expected COHb levels after exposure to carbon monoxide concentrations between 10 and 100 ppm (11.5 and 115 μg/m^3) during different types of physical activity. In the absence of carbon monoxide exposure, the COHb level is approximately 0.5%; people who smoke one pack per day of cigarettes may have COHb levels of 4% to 7% (Table 6-4).[3]

Experimental Studies

Carbon monoxide leads to a decreased oxygen-uptake capacity, with decreased work capacity under maximum exercise conditions. A blood COHb concentration of approximately 5% is required to decrease oxygen-uptake capacity. An impairment in the ability to judge correctly slight differences in successive short time intervals has been observed at COHb levels of 3.2% to 4.2%. Headaches and dizziness occur at COHb levels between 10% and 30%. At COHb levels higher than about 30%, severe headaches, cardiovascular symptoms, and malaise occur. Above COHb levels of about 40%, there is considerable risk of coma and death (Table 6-5).[3]

People with previous cardiovascular disease are very sensitive to carbon monoxide exposure. Exposure to carbon monoxide sufficient to raise the concentration of COHb to 2% may produce adverse effects during exercise in patients with coronary artery disease. The length of time to onset of angina was reduced by 4.2% at a COHb level of 2%, and by 7.1% at a COHb level of 3.9%. Similar results have been observed for intermittent claudication due to peripheral vascular disease.[3]

Population-Based Studies

Daily increases in carbon monoxide levels have been associated with increases in premature

Table 6-4. Predicted Carboxyhemoglobin Levels for People Engaged in Different Types of Work in Different Concentrations of Carbon Monoxide

CO Concentration		Exposure Time	Predicted COHb Level for Those Engaged in:		
in ppm	in mg/m^3		Sedentary Work	Light Work	Heavy Work
100	115	15 minutes	1.2	2.0	2.8
50	57	30 minutes	1.1	1.9	2.6
25	29	1 hour	1.1	1.7	2.2
10	11.5	8 hours	1.5	1.7	1.7

Table 6-5. Human Health Effects Associated with Low-Level Carbon Monoxide Exposure

Carboxyhemoglobin Concentrations (%)	Lowest Observed Effect Level (LOEL)
2.3–4.3	Statistically significant decrease (3%–7%) in the relation between work time and exhaustion in exercising young healthy men
2.9–4.5	Statistically significant decrease in exercise capacity (shortened duration of exercise before onset of pain) in patients with angina pectoris and increase in duration of angina attacks
5–5.5	Statistically significant decrease in maximal oxygen consumption and exercise time in young healthy men during strenuous exercise
<5.0	No statistically significant vigilance decrements after exposure to carbon monoxide
5–7.6	Statistically significant impairment of vigilance tasks in healthy experimental subjects
5–17	Statistically significant diminution of visual perception, manual dexterity, ability to learn, or performance in complex sensorimotor tasks (such as driving)
7–20	Statistically significant decrease in maximal oxygen consumption during strenuous exercise in young healthy men

mortality and hospitalizations from congestive heart failure.[3] However, epidemiologic studies relating carbon monoxide with daily counts of mortality or hospital admissions need to be interpreted with caution. In contrast with other pollutants, carbon monoxide measurements from fixed monitors used for air surveillance correlate poorly with personal carbon monoxide measurements; therefore, carbon monoxide may be a proxy for other pollutants, such as fine particles.[3]

Carbon monoxide exposure may also affect the fetus directly through oxygen deficit, without elevation of COHb level in fetal blood. During exposure to high carbon monoxide levels, the mother's hemoglobin gives up oxygen less readily, with a consequent lowering of the oxygen pressure in the placenta and in fetal blood. In animals, carbon monoxide causes low birthweight and developmental effects.[2] In humans, research has mainly focused on the effect of cigarette smoking during pregnancy, including decreased birthweight and retarded postnatal development.[3] Ambient carbon monoxide exposure during pregnancy has been associated with adverse outcomes, including intrauterine death and low birthweight.[6]

Susceptibility Factors

Susceptibility is a concern in the regulation of ambient air pollution. Susceptibility may include *(a)* intrinsic factors, such as age, gender, race, preexisting health impairment, and genetic factors and *(b)* extrinsic factors, such as the profile of exposures to pollutants, concomitant exposure to other toxic living conditions, nutritional status, and lifestyle factors.[41]

Intrinsic Factors

Certain subgroups are more susceptible to the impact of air pollution, including children, older people, people with certain diseases, and those with certain genetic factors. Several factors are responsible for the high susceptibility of children to ambient air pollutants. Children spend more time outdoors than do most adults and are often engaged in vigorous play. They also have higher respiratory rates than adults; therefore, they may receive higher doses of pollutants in proportion to body weight. Intensive growth and development processes create windows of great vulnerability to environmental toxicants. Older people are more likely to suffer from cardiovascular disease and impairment of immune response, both of which increase their susceptibility to air pollutants, especially to fine particles. In general, people with asthma are more responsive to short-term exposure to inhaled agents, especially particulates and ozone. People with preexisting chronic obstructive pulmonary disease or cardiovascular disease appear to be more susceptible to exposure to particulates.

Genetic factors can play a major role in responsiveness to air pollutants, especially to ozone and particulate.[2,42] Certain genetic polymorphisms affect synthesis of enzymes involved in the response to oxidative stress,

such as glutathione-S-transferase, which could increase the susceptibility to ozone and enhance the allergic response to diesel exposure. These genetic factors might explain part of the large variability among individuals in response to ozone exposure.

Extrinsic Factors

Air pollutants appear to have both short-term and long-term effects, but there are no clear data on how patterns of exposure might influence the development of health effects. Most exposures are to air pollutants in complex mixtures. Failure to consider the presence of multiple pollutants may confuse interpretation of observed effects. Within vehicular exhaust emission, it has been difficult to assess the effect of individual pollutants, particularly fine particles and nitrogen dioxide. Some studies have reported a synergistic effect of ambient particulates and ozone, and others a synergistic effect of ozone and diesel exhaust in increasing susceptibility to allergens.

Dietary antioxidants modulate the response to photo-oxidant exposure in animals and humans. Water-soluble antioxidants, such as ascorbate, urate, and reduced glutathione, are abundantly present in lung fluid and provide protection against damaging oxidation reactions in the extracellular components of this compartment. Within the cell, alpha-tocopherol and glutathione peroxidase may act to prevent the propagation of lipid peroxidation reactions. Vitamin E may prevent ozone-induced peroxidation, especially in vitamin E–deficient animals. Vitamin E, vitamin C, and betacarotene may protect against the adverse health effect of ozone on lung function.[43,44] Other micronutrients, such as omega-3 fatty acids, may decrease the adverse cardiovascular response to particulate exposure. Deficiency of these micronutrients could increase susceptibility to particulates and photo-oxidants, especially where populations are chronically exposed to high ambient air levels of pollutants.

Low socioeconomic status increases the association between air pollution and adverse health effects. Several factors, such as poor living conditions, poor nutrition, concomitant exposure, and limited access to health care, likely interact to increase the vulnerability to air pollutants.[45]

Traffic-Related Emissions

Although outdoor air pollutants have been linked to many adverse respiratory and cardiovascular outcomes, this risk is not uniformly distributed and may vary in relation to the proximity to emission sources. Therefore, people who reside or work close to major roads or highways may have a greater risk of suffering adverse outcomes compared to similar people who reside or work further away.[46] This new area of environmental research relies heavily on exposure metrics other than pollutant levels; it utilizes information from geographical information systems (GIS), which gather data on the geospatial location of study subjects and their relation to traffic exposure sources, such as distance to major roads or highways and traffic and road density measures.[47] GIS may also incorporate variables from the terrain (elevation and use of land), population density, and pollutant levels. These data can be used to construct a land-based regression model, which can predict the spread of pollutant levels throughout an area.[48] This technique may be useful in identifying "hot spots" of higher concentration for ecological studies or in evaluating rates of chronic health outcomes in relation to exposures. Land-based regression models are less useful when studies involve repeated exposures throughout a period of time, such as a panel or cohort study that evaluates how traffic-related emissions associate with changes in exhaled nitric oxide levels and lung function over time. For these types of studies, multilevel modeling, which incorporates GIS as well as exposure levels, offers a unique alternative.[49]

Traffic-related emissions have been associated with more serious respiratory health outcomes, including increased severity and frequency of respiratory symptoms, higher rates of nonscheduled medical visits and hospitalizations for asthma exacerbations, increased airway inflammation, and reduced lung volumes.[46] Traffic-related emissions are also associated with increased risk of cardiovascular morbidity and mortality. An association has been found between exposure to traffic and onset of a myocardial infarction within 1 hour afterward.[50] Living near a major road has been shown to increase the risk of deep venous thrombosis, a

risk factor for pulmonary embolism.[51] Traffic exposure is associated with chronic cardiovascular health outcomes. Subjects residing within 50 meters of a major highway have more evidence of atherosclerosis,[52] which may explain why close proximity to major roads is associated with higher blood pressure and pulse pressure and increased left ventricular mass.[53]

"Black smoke," a surrogate for traffic emissions, has been associated with increased risk for cerebrovascular disease and heart failure mortality, and traffic density has been associated with increased risk for ischemic heart disease.[54]

The physical and chemical properties of the air pollution mix near vehicular traffic may explain why this exposure causes more health problems. Near vehicular emissions there is a much higher burden of fine and ultrafine particles with greater content of organic and elemental carbon[55]—highly toxic particles that are more likely to penetrate into the distal airways and the systemic circulation. High carbon monoxide levels may also play a role in increasing risk for cardiovascular disease.

There are inconsistencies among studies, with some reporting no association between traffic emission and health effects.[56–58] In addition, residual confounding is a significant concern in cross-sectional and ecologic studies of road proximity and health outcomes. Given that populations with lower socioeconomic status are more likely to reside close to major roads or highways, factors other than environmental exposures could be biasing the results.[59]

Many unanswered questions remain: What constitutes proximity to a road? Is there a threshold, either based on distance to the exposure or traffic density, that could offer some degree of protection to an exposed population? Should people with severe asthma or congestive heart failure minimize their exposure to traffic, such as by residing and working further from traffic?

With more future studies standardizing their approach and controlling for bias, more information will be available to translate results into effective health policy and public health interventions that reduce morbidity and mortality due to traffic emissions.

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The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the Instituto Nacional de Salud Pública of Mexico or the Ministry of Health of Mexico.

7

Indoor Air Quality

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The focus of occupational health has been transformed in many ways by the increasing proportion of the workforce employed in offices and other kinds of public facilities, merging in many respects with the concerns of environmental health. Once considered safe by crude comparison with industrial settings such as construction, mining, and agriculture, experience has proven that these indoor environments are not free of significant health hazards. Moreover, the workers engaged in these sectors are neither experienced with environmental risks, nor as well prepared in general to think about hazards of work as their industrial counterparts were even long before the modern regulatory era. Because almost all previous attention has focused on the kinds of conditions and hazards that arise in more traditionally dangerous settings, the regulatory framework has not evolved forms of controls that ensure, at least in law, that work will be safe.

This chapter is divided into two sections. The first deals with the spectrum of problems that occur indoors in nonindustrial buildings, focusing on common features of implicated facilities. The second deals with the spectrum of clinical complaints related to low-dose chemical exposures (relative to doses that occur in industry), which have received increasing attention. Although these problems of chemical sensitivity most often occur in association with indoor nonindustrial environments, they may also be seen in a range of other work settings as well as in the non-work environment. Their distinguishing feature is the occurrence of symptoms or other clinical problems at levels that are far below those at which knowledge of toxicology would predict effects, and typically far below accepted standards in industry for human exposures. These somewhat vexing problems have challenged many of the cherished paradigms of occupational and environmental health about what is safe and what is not, and they pose a special challenge for physicians whose patients may complain about chemicals at levels deemed "safe."

BUILDING-RELATED CONDITIONS

Nonspecific Building-Related Illness

Since the 1970s, office workers worldwide have frequently complained of mucous membrane irritation, fatigue, and headache when working in specific buildings, with improvement within minutes to an hour of leaving the building. This constellation of symptoms, with tight temporal association to building occupancy, is called sick building syndrome, or, more recently, nonspecific building-related illness. It is the most

frequent of the building-associated health complaints in industrialized countries, which also include diseases caused by infection, allergic hypersensitivity, and specific toxins. Up to 30% of office workers surveyed may report symptoms attributed to poor air quality, and workers in buildings not known to have indoor air-quality problems have many complaints attributed to the indoor work environment.

Despite the impacts on productivity and employee morale when many workers in a building have building-related symptoms, the causes of these symptoms are incompletely understood. Early investigations of this phenomenon sometimes concluded that symptoms were caused by mass psychogenic illness because no specific contaminants were measured in concentrations that could account for symptoms. However, the endemic nature of complaints in specific buildings and the consistency of complaints from workers in tight buildings across the world did not satisfy diagnostic criteria for mass psychogenic illness. Fortunately, such attribution to psychological cause is no longer common or acceptable, although work stress is associated with reporting of symptoms among occupants of specific buildings (see Chapters 14 and 19). Occupants of buildings with high levels of complaints are often angry and fearful, in no small part due to resistance of managers to investigation of the cause(s) of their problems, inconclusive results of investigations that are conducted, or ineffectual remediation.

The recognition of building-related complaints by public health workers in the United States followed an energy crisis in the 1970s, during which ventilation standards were lowered to supplying 5 cubic feet of outdoor air per person per minute. This observation led to the hypothesis that building-related symptoms were attributable to lower rates of ventilation in relation to indoor contaminant sources. In some studies, ventilation rates are related to the prevalence of nonspecific building-related complaints, especially for ventilation supplying outdoor air at less than 30 cubic feet per person per minute. Indoor air-quality consultants commonly measure carbon dioxide levels as a marker for inadequate fresh air in buildings with high complaint rates. However, carbon dioxide level is not predictive of nonspecific building-related complaints.

The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) publishes consensus standards for ventilation of various types of buildings that are frequently adopted into building codes. These standards are not health based, and they are not performance standards for operating ventilation systems. Rather, they stipulate ventilation rates for design purposes and are based on occupant comfort. The latest ASHRAE Standard (62.1-2007) recommends 5 cubic feet per minute of outdoor air per occupant and an additional 0.06 cubic feet per minute for every square foot in office buildings, in the absence of cigarette smoking. Measuring effective ventilation is technically difficult, expensive, and rarely done apart from research settings. Indoor air consultants examine ventilation systems for possible entrainment of contaminants in the outdoor air source; design and operation of air flow; filter condition and maintenance schedules; cleanliness of the cooling coils and drip pans, which commonly support microbial growth because of moisture and dirt; condition of the duct lining, which commonly supports microbial growth if wet; and postdesign changes in occupancy, activities, and layout that may impact air quality.[1]

Studies suggest that certain building features and occupant characteristics are related to symptom prevalence. Variation in prevalence of building-related complaints among buildings suggests remediable causes.[2] Occupants of buildings with air conditioning have higher rates of building-related symptoms than occupants of naturally ventilated buildings or buildings with mechanical ventilation that does not alter air temperature or humidity, suggesting that the ventilation system may be the source of poor air quality in some buildings. In one study, ultraviolet germicidal irradiation in office ventilation systems reduced microbial contamination of cooling coils and drip pans, and also reduced work-related respiratory and mucosal symptoms.[3] Building dampness, associated with bioaerosols, is also frequently accompanied by nonspecific building-related illness. Measurable indices of bioaerosols are being studied as correlates of building-related illness; some evidence implicates endotoxin, β-1,3-glucan, ergosterol, and culturable hydrophilic fungi in dust samples. Other environmental correlates include carpeting,

high occupancy, and video display terminal use. Personal factors associated with building-related symptoms include female gender, allergies, and job stress or dissatisfaction.

Health care providers faced with the challenge of responding to indoor air-quality complaints must proceed without the benefit of a complete scientific understanding of what may be a multifactorial syndrome.[4] No single measurement can determine the adequacy of indoor air quality; determination of the acceptability of indoor air quality rests with the occupants—not a laboratory. In response to complaints about indoor air quality, a multidisciplinary approach allows attention to design and maintenance of air-conditioning systems, exclusion of obvious contaminant sources or water damage in the occupied space, and reassurance of occupants that nonspecific building-related symptoms (unlike building-related respiratory symptoms, described later) are self-limited. Indoor air-quality investigations customarily assess the ventilation in relation to occupant load by *(a)* measuring carbon dioxide, *(b)* identifying remediable deficiencies in ventilation system maintenance and cleanliness, *(c)* assessing water damage and moisture incursion, and *(d)* examining smoking policies. Health care providers, on a multidisciplinary team alongside industrial hygienists and ventilation engineers, have an important role to play in ruling out the possibility of less common, but more serious, building-related diseases, such as asthma and hypersensitivity pneumonitis, that frequently occur with a background of nonspecific building-related complaints among other workers.

Building-Related Allergic Disease

A 48-year-old social services eligibility technician began working in an office building in October. She had a history of sinus symptoms and 15 pack-year history of cigarette smoking, having been an ex-smoker for 10 years. Three months later, in January, she began to have insidious onset of dry cough, which, in March, was diagnosed as asthma. Skin-prick tests were negative to common aeroallergens. She was referred to an occupational medicine clinic in August, when she noted worsening symptoms during the workday (requiring use of inhaled bronchodilators) and recovery in the evenings and on weekends (when she did not need to use them). Her asthma became much worse when she handled dusty records while her desk was being moved. Self-monitoring of peak-flow showed reproducible, striking air-flow limitation shortly after entering the building, with partial recovery during lunch breaks outside the building and full recovery on weekends (Fig. 7-1). Methacholine challenge testing in September and November, before a 16-day vacation, found the provocative concentrations (PC20) for a 20% decrement in forced expiratory volume in 1 second (FEV_1) to be 0.29, and after the vacation to be 0.47 mg/mL (normal PC20 >15 mg/mL). These results confirmed a diagnosis of asthma

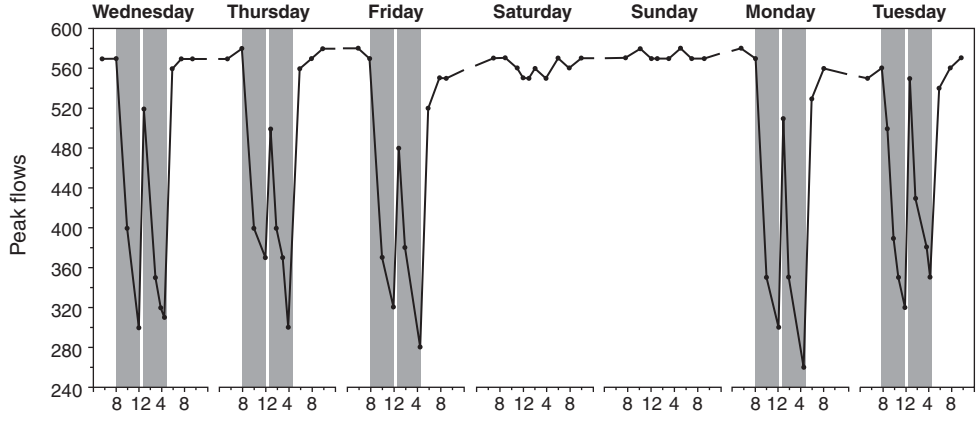

Figure 7-1. Peak-expiratory flow measurements by hour and day in a case of office building–related asthma. Stippled columns indicate time at work.

and suggested slight improvement in airway hyperreactivity with a short absence from work. Although she had notified her employer, her relocation to another building was delayed until late February, after her third course of prednisone treatment. After this relocation, her symptoms cleared, her work-related airflow limitation (documented by peak-flow measurements) resolved, and her need for asthma medications ended. Three months after her relocation, her PC20 normalized to above 25 mg/mL.

Nine months later, she was moved back to the original building into a set of offices that shared no ventilation system with the offices that she had previously occupied. Over the next 6 weeks, she experienced increasing symptoms and airflow limitation, once again requiring daily medication, and her PC20 fell to 0.22 mg/mL. She was medically restricted from the implicated building, with resolution of her work-related decrements in peak flow, decrease of her medication requirements, and increase in her PC20 to 5.19 mg/mL over the following 6 weeks. She has had no further difficulty with clinical asthma since then.

This building was built into an earthen bank, and workers reported musty odors and visible mold growth on the interior wall that abutted the bank. Aspergillus species of fungi were detected in the interior air but not in simultaneous measurements of outdoor air, suggesting amplification and dissemination of this fungus indoors. The presumed source of the woman's asthma was fungal bioaerosols associated with moisture coming in from the earthen bank.

Building-related asthma is infrequently recognized by physicians, although, unlike nonspecific building-related illness, it can lead to chronic irreversible illness. Early recognition and removal from the building, as in this woman's case, can result in cure of asthma. Permanent asthma can result when recognition of occupational etiology is delayed and asthma becomes severe before the patient leaves the implicated exposure. Such sentinel cases of asthma imply risk for other workers. In this case, public health investigation after two sentinel cases showed that co-workers had nearly five times the prevalence of physician-diagnosed asthma with onset or exacerbation since building occupancy, compared with workers in another social service agency.[5]

Building-related asthma occurs in water-damaged buildings and in relation to microbially contaminated humidifiers used in them.[6] Biologic aerosols containing mold spores and possibly bacteria are the sensitizing and irritant agents. No air measurement of viable fungi or spore count has been shown to predict hazard in the nonindustrial environment. An indoor source of microbial amplification and dissemination can be inferred from looking at the rank order of mold species concentrations indoors compared to outdoors, but no quantitative standards exist or are likely to be developed based on air exposure–response studies. Visible mold (Fig. 7-2) and moldy smells should be remediated without demonstrating specific mold air levels by culture or air sampling. Despite the difficulty in characterizing the exposure, the affected individual's symptom history and peak-flow measurements can be valuable in documenting the occupational nature of building-related asthma. Cases of building-related asthma may occur along with cases of hypersensitivity pneumonitis in water-damaged buildings.

A 46-year-old pediatrician had been followed by an allergist for 10 years for upper respiratory and chest complaints after moving into an office suite. At first, he complained of sinus drainage and a sore feeling in his nose and throat. Over the years, he had acquired achiness in his chest associated with fever, productive cough, chest tightness, wheezing, fatigue to exhaustion, and shortness of breath on exertion. His forced vital capacity (FVC) fell within 3 years of building occupancy, consistent with a restrictive pattern. Without ever receiving a diagnosis, he had been treated with nasal cromolyn, inhaled steroids, bronchodilators, theophylline, antibiotics, and intermittent oral corticosteroids. A year before his referral to an occupational medicine specialist, he had noted exacerbation of his chest symptoms when he returned to his office suite after a week away from work. He then began to suspect an office-related cause to his symptoms, with increased cough, chest tightness, and achiness when he entered his suite, and resolution within hours after leaving and improvement on weekends. He noted a musty smell and fungal discoloration of wall board in the suite bathroom, which resulted from leaking pipes.

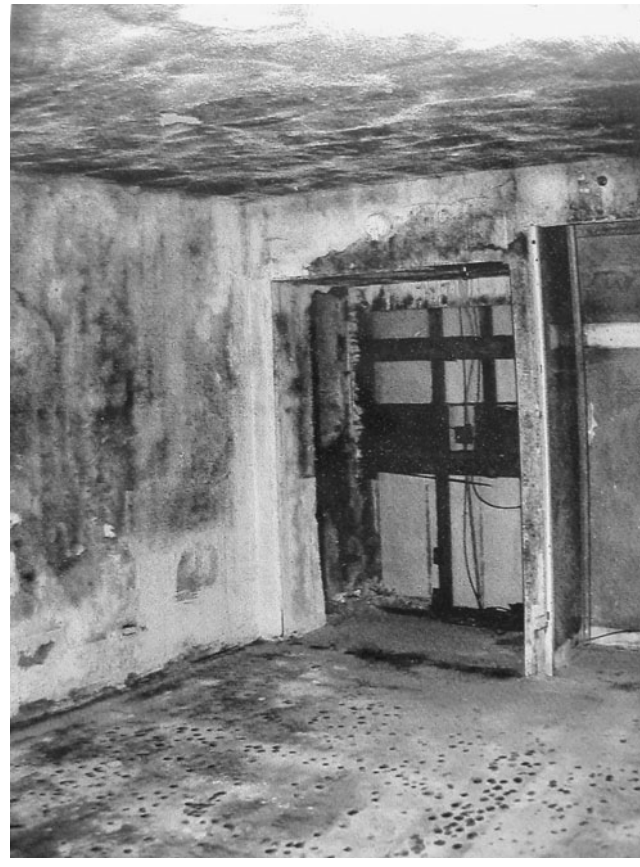

Figure 7-2. Extensive mold growth in a room of a former hotel in New Orleans, years before Hurricane Katrina struck New Orleans in 2005. (Photograph courtesy of Michael R. Gray.)

On referral, he was found to have basilar rales, bronchial hyperreactivity on histamine challenge testing, and reduced exercise tolerance with an excessive respiratory rate at rest and excessive minute ventilation for oxygen consumption. Chest X-ray was normal, but a high-resolution computed tomography (CT) scan showed fine centrilobular nodules. Bronchoalveolar lavage showed a lymphocytic alveolitis compatible with hypersensitivity pneumonitis. A transbronchial lung biopsy showed a mild, patchy lymphocytic interstitial pneumonitis. His symptoms resolved with prednisone and removal from the office suite.

However, 2 months later, chest aching, exertional shortness of breath, profound fatigue, and chilly feelings recurred within 45 minutes of using a musty restaurant bathroom that had been water-damaged from recurrent roof leaks. He had a prolonged recovery time, requiring systemic steroids for 7 months. A year after this acute exacerbation, he again had a recurrence of chest symptoms, within hours of handling medical records from his previous office suite that had become wet while stored in his basement (because of a hot water heater leak). He again required months of prednisone use and did not fully recover his health until 1 year later.

This case of building-related hypersensitivity pneumonitis illustrates the typical medical delay in suspecting and diagnosing a building-related etiology for symptoms. Few physicians are aware that office settings can be associated with diseases related to organic antigens. In contrast to building-related asthma, however, there are many published case reports and epidemic investigations of hypersensitivity pneumonitis and humidifier fever. Typically, people with hypersensitivity lung diseases may not be able to reoccupy a building in which they were sensitized to biologic aerosols from humidifiers, ventilation systems, or water-damaged materials on which fungal growth

has occurred. Even after remediation of the conditions that led to sensitization and disease, low levels of exposure can trigger recurrent symptoms. Because hypersensitivity pneumonitis can lead to irreversible lung fibrosis after recurrent acute episodes or prolonged exposure, early recognition and restriction of affected people from the implicated building are the best measures for preventing progression. Remediation is warranted to prevent cases in co-workers who are not yet sensitized. Occupational medicine physicians can encourage specialists to proceed with diagnostic tests before the affected individuals develop classic late-stage abnormalities, such as those evident on chest X-rays. The aforementioned case suggests that this pediatrician was sensitized to an antigen that was not unique to his water-damaged office setting.

Cases of hypersensitivity pneumonitis are often accompanied by systemic symptoms of myalgia, fever, and profound fatigue. These symptoms are not usually present in asthma, although both diseases commonly share chest symptoms, such as cough, chest tightness, and wheezing. In contrast to asthma and hypersensitivity pneumonitis, sick building syndrome alone is not accompanied by chest symptoms. When indoor air-quality complaints exist, health care providers should evaluate occupants for building-related asthma and hypersensitivity pneumonitis. The occurrence of building-related chest disease dictates evaluation for sources of fungal and bacterial growth and means of dissemination from areas of water damage or from the ventilation system.[7] The presence of chest disease also requires more aggressive medical restriction from the building to prevent irreversibility of the condition.

Many patients report that they have building-related nose and sinus symptoms. Allergic rhinosinusitis can occur, in a way analogous to the response of airways and lung tissue to building-related antigen exposure. Little research has been done on this common clinical complaint to determine its epidemiology, to distinguish it from non-immunologic mucous membrane complaints in sick building syndrome, or to link it to exposures in implicated buildings.[7] Unfortunately, there are no practical ways of measuring antigens related to indoor microbial bioaerosols, although research is underway on antigen identification, measurement, and size differentiation. However, rhinitis may precede or exacerbate asthma. If the temporal association suggests that the nasal or sinus symptoms are building related, the same attention to identifying and removing sources of water damage and attending to the maintenance of the heating, ventilation, and air-conditioning system is needed as for building-related chest diseases.

In residential environments, allergic disease commonly occurs in relation to indoor allergens, which are more diverse than those in office settings. Antigens from dust mites, cockroaches, and animal danders are implicated in asthma beginning in childhood. Environmental intervention to lower these antigen exposures, such as by using antigen-impermeable mattress covers, vacuum cleaners equipped with high-efficiency particulate air (HEPA) filters, HEPA air purifiers, and professional pest control, can reduce childhood asthma morbidity when it is tailored to the sensitizers affecting an asthmatic child.

Building-Related Infection

In 1976, a total of 182 cases of a mysterious pneumonia occurred among members of the American Legion attending a convention in Philadelphia. After months of laboratory investigation, a newly discovered bacterial organism, *Legionella pneumophila*, was found to be the responsible agent. We now know that, in the absence of vigorous attempts to eradicate it, this common environmental organism frequently grows in the warm water of building cooling towers. When contaminated cooling tower mists are entrained in air intakes of large buildings, cases of infection with this organism (legionellosis) can occur. Outbreaks have also been recognized as a result of contaminated industrial water sprays, hospital shower heads, and hot tubs. Legionella pneumonia is not spread by person-to-person transmission.

When legionellosis occurs, molecular biology techniques are now used to identify specific strains by DNA fingerprinting. Possible sources can be tested for the same strain in environmental reservoirs. This matching of aerosol source with clinical cases can help prioritize environmental controls through disinfection of hot water systems and avoidance of entrainment of contaminated aerosols.

In addition to pneumonia, Legionella organisms have been associated with another building-related disease called Pontiac fever, which is self-limited and characterized by fever, chills, headache, and myalgia. It was first described in 1968, in a building-related epidemic of 144 cases in a county health department in Michigan. The attack rate was nearly 100%, with an average incubation period of 36 hours.

Building ventilation characteristics are important factors affecting the spread of contagious infections, such as those due to respiratory viruses. Types of housing with different ventilation characteristics, such as air-conditioned buildings (as compared with tents or naturally ventilated barracks), are associated with increased incidence of respiratory symptoms and signs of communicable disease in military troops. Other airborne infectious diseases, such as tuberculosis, pneumococcal disease, varicella, and measles, may be affected by rates of building ventilation. A major concern in hospitals, prisons, and shelters is control of tuberculosis, for which ventilation and air disinfection techniques are critical. (See also Chapter 13.)

Building-Related Complaints Due to Specific Toxic Agents

Health professionals responding to building-related complaints must also consider specific toxic exposures as a possible explanation. This is particularly important when complaints differ from those of nonspecific building-related illness or occur in epidemic—rather than endemic—fashion. For example, complaints of headache and nausea dictate consideration of carbon monoxide poisoning, which can occur when internal combustion sources are not exhausted to the outdoors or when air intakes entrain fumes from loading docks, parking garages, or boiler-stack emissions. Building-related itching without rash can occur with fibrous glass exposure, which can result when air-duct lining is entrained in the airstream entering the occupied space. Epidemic coughing, dry throat, and eye irritation can result from detergent residues after the misapplication of carpet cleaning products. In instances of building-related complaints associated with specific exposures, a careful evaluation of types of symptoms, their distribution among building occupants by location or job, and their temporal onset may point investigators to the cause and to remediation resources.

INDOOR CARCINOGEN EXPOSURE

Environmental tobacco smoke has been the most common indoor carcinogen, but public tolerance of this exposure is decreasing across the United States, as reflected in state and municipality ordinances prohibiting smoking in workplaces, restaurants, and bars. (See Box 7-1 and Fig. 7-3.) Sadly this is not yet true in many parts of the developing world, where tobacco smoke is still commonly encountered, posing risks for cancer and respiratory disease. Also widely encountered are poorly exhausted combustion products of available and cheap biomass fuels used for heating and cooking. (See Box 7-2.) Even as their precise contribution to cancer risk and respiratory disease burden in children and adults remains under study, there is little controversy that these indoor exposures constitute one of the most prevalent and serious environmental problems in the world.

Sometimes building-related exposures to carcinogens do not cause symptoms in occupants, but still pose a health risk. For example, radon gas emitted from building materials, water, and soil surrounding foundations increases the risk of cancer. Radon exposures can be measured with simple devices. The Environmental Protection Agency (EPA) has issued guidelines for elevated exposures and effective remediation, such as sealing of foundations and subsurface ventilation. Similarly, asbestos in insulation and some building materials in older buildings poses risks of cancer of the lung and other sites (as well as nonmalignant lung disease) if it is disturbed during occupant activities or renovation. Because of latency and dose–response considerations, health professionals are often called to help communicate risks of asbestos exposure to building occupants or the public during removal of asbestos from older buildings. Most states license asbestos-abatement professionals, who are trained to protect remediation workers with respirators and other personal protective equipment, while maintaining negative pressure in asbestos removal areas to prevent asbestos

Box 7-1. Environmental Tobacco Smoke

Kathleen Kreiss

Environmental tobacco smoke (ETS), or secondhand tobacco smoke, contains more than 4,000 compounds, which demonstrate their biologic properties through different mechanisms. Environmental tobacco smoke exposure is associated with increased risks of lung cancer, heart disease, and nonmalignant respiratory disease in people who have never smoked. An estimated 46,000 deaths (range: 22,700 to 69,600) from heart disease and more than 3,400 deaths (range: 3,423 to 8,866) from lung cancer attributable to ETS occur annually in the United States.[1] No risk-free level of exposure has been determined. The only way to fully protect nonsmokers from exposure to ETS is to eliminate smoking in indoor spaces. There are no occupational exposure limits for ETS. Other methods to reduce exposure, such as separating smokers from nonsmokers, cleaning the air, and increasing ventilation in buildings, cannot eliminate ETS exposures of nonsmokers. Homes and personal vehicles remain major venues of exposure to involuntary ETS exposure.

Environmental tobacco smoke exposure, in utero and during the neonatal period and childhood, causes substantial respiratory effects in children. Maternal smoking during pregnancy is associated with lower birthweight, reduced lung growth, and adverse effects on lung function shortly after birth that can persist throughout childhood. Postnatal exposure causes a lower level of lung function during childhood; increased respiratory illnesses in infants and children; middle ear disease (acute and recurrent otitis media and chronic effusion); symptoms of cough, phlegm, wheeze, and breathlessness among school-age children; risk of ever having asthma in schoolchildren; and onset of wheeze-related illnesses in early childhood. ETS exposure is causally related to sudden infant death syndrome. The causal effects of parental smoking are stronger for younger children and for maternal smoking; however, paternal smoking is associated with some of these outcomes even in homes where the mother does not smoke.[2]

Asthmatic adults exposed to ETS have increased acute respiratory symptoms, including cough, chest tightness, and difficulty breathing. Healthy adults also may complain of these acute and chronic symptoms as a result of ETS exposure. There is suggestive evidence that short-term ETS exposure can cause an acute decline in lung function in persons with asthma and that chronic exposure may cause a small decrement in lung function in the general population. Similarly, there is suggestive, but not sufficient, evidence to infer a causal relationship between ETS exposure and adult-onset asthma, worsening of asthma control, risk of chronic obstructive pulmonary disease, and morbidity in persons with chronic obstructive pulmonary disease.

Environmental tobacco smoke also leads to annoyance from odors and sensory irritation of the nose and throat, which accounts for most nonsmokers finding air quality unacceptable in indoor spaces where ETS is present. The odor threshold for respirable suspended particles in environments with cigarette smoking is about 1 $\mu g/m^3$.

In studying exposures to ETS, one often monitors "marker" substances and uses them as indices of exposure. Selection of these compounds is based on their ease of measurement and not their toxicity. They include nicotine, respirable suspended particles, volatile organic compounds, particulate polynuclear aromatic hydrocarbons (PAHs), and aldehydes.

Levels of biomarkers can be measured to determine how much ETS a person has absorbed or metabolized. These biomarkers commonly include carboxyhemoglobin; urinary or serum cotinine, a major metabolite of nicotine; and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol, the major metabolite of the tobacco-specific carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone. Urinary cotinine levels are commonly adjusted for creatinine. The biological half-life of cotinine is approximately 16 to 20 hours; therefore, urinary cotinine is an indicator of ETS exposures from the previous 1 to 2 days. The half-life of the tobacco-specific carcinogen is 40 days, and its urinary metabolite, 45 days.

Restrictions on tobacco smoking indoors are now the norm in the United States in most commercial and government buildings, as well as public transportation facilities and conveyances. The passage of local ordinances has been facilitated by the increasing prevalence of nonsmokers in the population, the quantification of risk from their estimated ETS exposures, and energy savings on ventilation rates in the absence of cigarette smoking.

Some public spaces still permit smoking, especially entertainment and hospitality facilities. Casinos have often remained exempt from laws and regulations mandating smoke-free workplaces. The estimated number of annual deaths from ETS-induced heart disease and lung cancer in Pennsylvania casino workers is five-fold those due to Pennsylvania mining disasters, and the number far exceeds the Occupational Safety and Health Administration (OSHA) level indicating significant risk of health impairment.[3]

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Figure 7-3. Billboard warning the public about the dangers of environmental (secondhand) tobacco smoke. (Photograph by Earl Dotter.)

fibers from entering occupied spaces. For all of these carcinogens, primary prevention is through identification and management.

MULTIPLE CHEMICAL SENSITIVITY

Since the 1980s, a new clinical syndrome—now most widely referred to as multiple chemical sensitivity (MCS)—has been recognized, characterized by symptoms in multiple systems after exposure to low levels of synthetic chemicals.[8] Despite research efforts, its pathophysiologic basis remains uncertain and controversial. Unlike any other building-related illness, it recurs in affected people in a diverse array of environmental situations and cannot be readily reversed by attention to any single exposure situation. The following is a representative example:

A 46-year-old library worker was in good health until the onset of eye, nose, and throat irritation and recurrent headache associated with renovation of the library where she worked. She and many co-workers complained primarily of dust and paint fume exposures, which were poorly controlled initially. After several weeks of effort, the employer succeeded in establishing temporary ventilation for the work area and performing most of the renovation at night. Almost all of the patient's co-workers improved dramatically after these changes were made. She, however, felt no better and began experiencing similar symptoms in her car, at various stores, whenever she was around anything she termed "scented," and especially in the office. She believed she was experiencing effects from the small residual levels of construction-related exposures, but temporary transfer to another part of the library brought no relief. New symptoms, including difficulty breathing, muscle and joint aches, and confusion occurred both at work and at home, triggered by an increasing number of offensive odors, irritants, and products. Efforts to clean her house of such materials, as well as a trial leave of absence from work (without the benefit of workers' compensation), resulted in only minimal improvement.

On clinical evaluation, the patient appeared well and had no abnormal physical findings. Laboratory tests, including a work-up for respiratory and central nervous system abnormalities, were unrevealing. Consultations in pulmonary medicine, rheumatology, and neurology were unhelpful. Attempts at empirical

Box 7-2. Exposure to Biomass Fuel Fumes

John R. Balmes

Half of all people still rely for their daily energy requirements on solid fuels, with even a higher fraction—up to 80%—in rural areas of some countries. These solid fuels are mainly biomass: wood, dung, and crop wastes. Because biomass is typically burned in inefficient, poorly vented stoves indoors—and often in open fires—women and young children are intensely exposed to indoor air pollution (IAP). Levels of exposure to particulate matter (PM) in such homes are often at least an order of magnitude higher than the highest concentrations that occur in the ambient air of developed countries.[1] In addition to PM, carbon monoxide, nitrogen oxides, formaldehyde, and toxic organic compounds, such as benzene; 1,3-butadiene, and benzo(a)pyrene, are present in smoke from biomass combustion, depending on the type of fuel that is burned.[2] In terms of emissions of PM and gases, the combustion of wood and other biomass is qualitatively similar to the burning of tobacco, although without the nicotine.

Cooking with biomass fuels is generally done on unvented stoves, typically consisting of such simple arrangements as three rocks, a U-shaped hole in a block of clay, or a pit in the ground. Combustion under such conditions leads to high emission factors that can lead to extremely high pollutant concentrations near the stove. Pollutant concentrations are further exacerbated by the lack of ventilation that characterizes many kitchens in rural areas of developing countries. Since households in rural areas of these countries often require cooking for many hours daily, exposure to biomass smoke is considerable, especially for women and children. Infants and young children are often carried on their mothers' backs while they cook, so from early infancy children spend hours breathing smoke from cooking or heating fires. In temperate climates and highland areas, people spend more time indoors, and the cold temperatures that characterize these areas require fires that burn over extended periods and tighter house construction for space heating. Therefore, both pollution levels and exposure times increase.

There is great public health impact of the relatively high exposures to PM in homes where cooking is done with inefficient, poorly ventilated stoves using biomass fuels. IAP from solid fuel use is responsible for *(a)* 2.6% of the total global burden of disease in terms of disability-adjusted life years (DALYs), and *(b)* between 1.5 and 2.0 million deaths annually.[3] Indoor air pollution is the second most important environmental risk factor, after poor water, sanitation, and hygiene.

The greatest burden of IAP-related premature deaths is in children with pneumonia. A recent meta-analysis showed a significantly increased overall pooled odds ratio (1.78) for acute lower respiratory tract infection (ALRI) in children exposed to unprocessed solid fuel smoke.[4] This finding is biologically plausible because smoke from biomass impairs respiratory tract defense mechanisms, such as mucociliary clearance and alveolar macrophage function.

While cigarette smoking is the leading preventable cause of chronic obstructive pulmonary disease (COPD) in developed countries, IAP exposure from inefficient burning of solid fuels may be the leading preventable cause of COPD among women in developing countries. Since almost 3 billion people use biomass or coal as their main cooking and heating fuel, the resultant population at risk for COPD (including men) is huge. (In addition, people in developed countries exposed to wood burning in fireplaces and stoves in their homes are at increased risk of COPD.) Cooking with biomass fuel has been associated with both chronic bronchitis and chronic airflow obstruction. A recent intervention trial found a slower rate of decline in lung function in women who used an improved chimney stove.[5]

Given that IAP exposure from solid fuel used for cooking and heating remains common worldwide and that the health impacts of this exposure are substantial, reducing this exposure needs to be a high public health priority.

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therapy with various inhalers, nonsteroidal anti-inflammatory agents, and migraine therapies also failed to relieve her symptoms. Because of the disparity between complaints and findings, she was referred to a psychiatrist who confirmed some depressive features but could not explain her symptoms. She began a trial of selective serotonin reuptake inhibitor (SSRI) antidepressants, but she could not tolerate them and discontinued them after 3 days.

Finally, frustrated by unsympathetic physicians and her employer, she sought evaluation from a nonstandard environmental medicine physician, who advised (a) total avoidance of all chemical exposures, including quitting her job, and (b) nontraditional remedies, based on results of

blood and hair tests in an alternative laboratory, which reported measurable levels of organic chemicals and heavy metals as well as immunologic responses to a range of common chemicals. She continues to have many symptoms.

Although this case occurred in the setting of building-related illness, MCS may develop in occupational and nonoccupational settings, and in people who have experienced one or more episodes of chemically induced illnesses due to solvents, pesticides, or other chemicals. Once MCS begins, however, affected individuals experience symptoms they associate with many types of environmental contaminants in air, food, or water at doses well below those that clinically affect others.[9] Although there is typically no measurable impairment of specific organs, complaints are associated with dysfunction and disability.[10] While MCS as severe as in the aforementioned case is not common, milder variants of chemical intolerance are very prevalent among adults.

There is no general consensus on a definition for MCS, but certain features are sufficiently characteristic to raise suspicion and differentiate it from other occupational and nonoccupational health problems. Its major features are as follows:

- Symptoms usually occur after a single or recurrent occupational or environmental inhalational exposure.
- Symptoms resembling those associated with the preceding exposure begin to occur after exposures to surprisingly lower levels of other common work and household substances, especially irritants and those with a pungent odor.
- Symptoms appear referable to many organ systems, especially the central nervous system.
- Chronic symptoms often make it difficult to discover any relationship between exposures and effects.
- Objective organ impairment is typically absent.
- No other diagnosis easily explains the range of responses or symptoms.

Not every patient meets these criteria precisely. But because the diagnosis of MCS is, in the end, based on subjective information, each point should be carefully considered. Each serves to rule out other clinical disorders that MCS may resemble, such as generalized anxiety disorder, classic sensitization to environmental antigens (such as occupational asthma), late sequelae of organ system damage (such as reactive airways dysfunction syndrome after a toxic inhalation), or systemic disease (such as systemic lupus erythematosus). On the other hand, the diagnosis of MCS does not require the exclusion of all other possibilities, and exhaustive testing is not required in most cases.

The sequence of pathologic events that leads from apparently self-limited episodes of an environmental exposure to the development of MCS in certain people is not known. Most available data are descriptive. And debate over the etiology of MCS has been heavily dominated by dogma.

Detailed information about the epidemiology of MCS is not available. Estimates of prevalence in the U.S. population range as high as 6%; the rate found in military "controls" for veterans of the Persian Gulf War was about 2.5%, while veterans of this war suffered MCS-like symptoms twice as frequently.[11] Although many people find chemicals and other odors objectionable and report life modifications to avoid exposure to them, MCS in the clinically overt form remains uncommon. Other observations include the following:

- Multiple chemical sensitivity syndrome occurs most commonly in midlife.
- Women are more frequently affected than men.
- Some host factor or susceptibility is important because mass outbreaks have been uncommon, and only a small fraction of victims of chemical overexposures acquire MCS or a similar disorder.
- Several classes of chemicals have been commonly implicated in the initial presentation of MCS, specifically organic solvents, pesticides, and respiratory irritants. In a "sick building" situation, some patients evolve from nonspecific building-related illness into MCS.

Natural History

Multiple chemical sensitivity syndrome has not yet been studied enough to delineate its clinical

course completely. But there is little evidence that it is progressive, and it is not lethal. Complete remissions are equally unlikely. There is no established treatment for MCS.

Primary prevention strategies cannot be developed without knowledge of the pathogenesis of the disorder or the host risk factors that predispose some people to become affected.

Secondary prevention would appear to offer some greater control opportunities, although no specific interventions have been studied. Because psychological factors may play a role in victims of occupational overexposures, careful and early management of people seeking care after acute toxic exposures or symptoms related to buildings is advisable even when the prognosis from the exposure itself is good. Patients seen in clinics or emergency departments immediately after acute exposures should be assessed for their reactions to the events and should probably receive very close follow-up when undue concerns of long-term effects or persistent symptoms are noted. Efforts should be made for such patients to prevent recurrences of symptomatic exposures, which may be precipitants of MCS by whatever mechanism is causal. Aggressive interventions or those that lead to unnecessary limitation of activity should be avoided.

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FURTHER READING

Black DW, Doebbling BN, Voelker MD, et al. Multiple chemical sensitivity syndrome: symptom prevalence and risk factors in a military population. Archives of Internal Medicine 2000; 160: 1169–1176.
This study concluded that self-reported symptoms suggestive of multiple chemical sensitivity syndrome are relatively frequent in a military population and are more common among Persian Gulf War veterans than comparable controls.

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Mendell MJ, Cozen M, Lei-Gomez Q, et al. Indicators of moisture and ventilation system contamination in U.S. office buildings as risk factors for respiratory and mucous membrane symptoms: analyses of the EPA BASE data. Journal of Occupational and Environmental Hygiene 2006; 3: 225–233.
Even in non-compliant office buildings, symptoms are associated with remediable building environmental conditions.

Sparks PJ, Daniell W, Black DW, et al. Multiple chemical sensitivity: a clinical perspective. I. Case definition, theories of pathogenesis, and research needs. Journal of Occupational Medicine 1994; 36: 718–730.
This paper and the one that follows comprise a refereed review of multiple chemical sensitivity syndrome. This review article explores four theories of causation of MCS syndrome and suggests areas for further research. Although published in 1994, these two papers still comprise the best and most balanced review in this format of this complex subject.

Sparks PJ, Daniell W, Black DW, et al. Multiple chemical sensitivity: a clinical perspective. II. Evaluation, diagnostic testing, treatment, and social considerations. Journal of Occupational Medicine 1994; 36: 731–737.
This review article proposes strategies for clinical evaluation and management of patients with MCS using a biopsychosocial model of illness.

Saito M, Kumano H, Yoshiuchi K, et al. Symptom profile of multiple chemical sensitivity in actual life. Psychosomatic Medicine 2005; 67: 318–325.
This study, which examined real-time, self-reported experiences of MCS patients coupled with real-time assessment of the environments that triggered symptoms, concluded that patients with multiple chemical sensitivity do not have either somatic or psychological symptoms under chemical-free conditions, and that symptoms may be provoked only when exposed to chemicals.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

8

Water Contamination and Wastewater Treatment

Jeffery A. Foran

We agree to halve, by the year 2015, the proportion of people who are unable to reach or to afford safe drinking water and the proportion of people who do not have access to basic sanitation.

—United Nations Millennium Development Goals, 2000

Water is essential for life, and the earth has over 1 billion cubic kilometers—2.6 x 10^{20} gallons, or 240 million cubic miles—of it. But only a very small percentage is freshwater, and even less is available for human use. Effectively, about 0.01% of all of the water in the world is usable, and this water is not evenly distributed among countries and regions. Indeed, the World Health Organization (WHO) reports that more than 1 billion people in low- and middle-income countries lack access to safe water for drinking, personal hygiene, and domestic use, and nearly 2 billion people lack access to adequate sanitation facilities. Lack of access to clean drinking water leads to nearly 250 million cases of water-related disease and between 5 and 10 million deaths annually.

The average person needs to consume a minimum of 5 liters (1.3 gallons) of water a day to survive in a moderate climate at an average activity level. The minimum amount of water needed for drinking, cooking, bathing, and sanitation is between 50 and 100 liters (13 to 26 gallons) a day. However, rates of water use among people in different countries are markedly different. For example, the average person in Somalia uses 8.9 liters (2.3 gallons) of water a day, while the average person in the United States uses between 250 to 300 liters (65 to 78 gallons) of water a day for drinking, cooking, bathing, and watering domestic property. Similarly, each individual in North America has, on average, access to over 6,000 cubic meters of stored water (in reservoirs), while each individual in the poorest countries in Africa has, on average, access to less than 700 cubic meters and each person in Ethiopia has, on average, access to less than 50 cubic meters of stored water.[1,2]

As of 2000, 3% of the world's population faced "water scarcity" situations (less than 1,000 cubic meters of water available per person per year) and 5% faced "water stress" (1,000 to 2,000 cubic meters of water available per person per year). It is estimated that by 2025, 7% of the world's population will face water scarcity and 31% will face water stress. The total population in countries facing water stress or water scarcity is projected to grow from 480 million in 2000 to nearly 3 billion in 2025.[2]

WHO projects that water scarcity will not affect all countries and regions in the same ways. For example, population increases and growing demands are projected to push many Asian countries into water scarcity. By 2025, nearly 230 million Africans will face water scarcity and 460 million will live in water-stressed countries, with much of the burden falling on North Africa and sub-Saharan Africa.

After water availability, the single greatest hazard associated with drinking water worldwide is microbial contamination. Access to safe (uncontaminated) water—treated or untreated—is unevenly distributed throughout the world. Nine hundred million people suffer from water-related diarrheal illness each year, resulting in 2 million deaths. Many of these people live in low- and middle-income countries. Those at greatest risk are children and older people. Many more suffer from other water-related diseases. The United Nations estimates that, each year, water-related diseases account for more than 5 million deaths, and more than 2 billion people suffer from diseases linked to contaminated water. Sixty percent of infant deaths worldwide are caused, in part, by water-related infectious and parasitic diseases.

Advanced water treatment in many developed countries has reduced pathogen concentrations to levels that pose little threat to public health. However, pathogen contamination of water supplies has not been eliminated in developed countries, including those that employ sophisticated water-treatment technologies. In some countries, including the United States, pathogen contamination is a reemerging threat as a result of *(a)* relatively new and newly discovered pathogens, such as Cryptosporidium, that are resistant to conventional treatment; and *(b)* newly recognized potential sources of water contamination, such as bioterrorism.

Human health has also suffered as a result of chemical contamination of water supplies in both developed and developing countries. Chemical contaminants in surface and ground water may occur naturally or from industry, agriculture, and other human activities (Fig. 8-1). The nature and sources of chemical contamination may be similar or differ greatly among developed and developing countries.

Figure 8-1. Water pollution from point sources, such as this one, has accounted for the killing of many fish. (Photograph by Earl Dotter.)

The global disparities in access to clean freshwater are readily apparent. The adverse effects of these disparities on individual and community health as well as on ecosystems are becoming better understood. This chapter, which addresses water quality and its effects on health, provides overviews of *(a)* pathogen and chemical contamination of surface water and groundwater, *(b)* contaminant sources, *(c)* effects of exposure to contaminants on human health, *(d)* approaches to treatment of sanitary waste and drinking water (Boxes 8-1 and 8-2), and *(e)* regulatory and nonregulatory approaches to address contamination of surface water, groundwater, and drinking water.

CONTAMINANTS OF GROUND AND SURFACE WATER

There are many contaminants of surface water and groundwater from natural and human

Box 8-1. Generalized Steps in the Treatment of Sanitary Waste Prior to Its Discharge to Surface Waters

Screening

Removal of items, such as wood, rocks, and dead animals, prior to wastewater entering the treatment plant. Most of these materials are sent to a landfill.

Pumping

Gravity moves sewage from homes and businesses to the treatment plant. If the plant is built above the ground level, wastewater must be pumped up to the aeration tanks, where gravity moves wastewater through the treatment process.

Aeration

Aeration causes some dissolved gases that cause taste and odor problems, such as hydrogen sulfide, to be released from the water. Wastewater then enters a series of long, parallel concrete tanks. Each tank is divided into two sections. In the first section, air is pumped through the water. As organic matter decays, it uses up oxygen. Aeration replenishes oxygen. Bubbling oxygen through the water also keeps the organic material suspended while it forces grit (coffee grounds, sand, and other small, dense particles) to settle out. Grit is pumped out of the tanks and taken to landfills.

Sludge Removal

Wastewater then enters the second section or sedimentation tanks. The sludge, the organic portion of the sewage, settles out of the wastewater and is pumped out of the tanks. Some of the water is removed in a step called thickening. The sludge is processed in large tanks called digesters.

Scum Removal

As sludge is settling to the bottom of the sedimentation tanks, lighter materials, termed scum, float to the surface. This scum includes grease, oils, plastics, and soap. Slow-moving rakes skim the scum off the surface of the wastewater. Scum is thickened and pumped to the digesters along with the sludge. After solids are removed, the liquid sewage is filtered through a substance, usually sand, by the action of gravity. This method removes almost all bacteria, reduces turbidity and color as well as odors, reduces the amount of iron, and removes most other solid particles from the water. Water is sometimes filtered through carbon particles to remove organic particles.

Disinfection

Finally, the wastewater flows into a tank where chlorine is added to kill bacteria. The chlorine is mostly eliminated as the bacteria are destroyed, but sometimes it is neutralized by adding other chemicals. This protects fish and other aquatic organisms as the treated waste is discharged to surface waters. The treated water, called effluent, is then discharged to a local river, lake, or the ocean.

Residuals

Treating wastewater includes dealing with the solid-waste material. Solids are kept for 20 to 30 days in large, heated enclosed tanks, called digesters, where bacteria break down (digest) the material, reducing its volume and odors, and organisms that can cause disease are removed. The finished product is sent to landfills or is sometimes used as fertilizer.

Source: http://ga.water.usgs.gov/edu/wwvisit.html

Box 8-2. General Steps Used in the Treatment of Drinking Water

Aeration

Water is mixed to liberate dissolved gases and to suspend particles in the water column.

Flocculation

Materials and particles present in drinking water (clay, organic material, metals, and microorganisms) are often quite small and will not settle out from the water column without assistance. To help the settling process, "coagulating" compounds are added to the water. Suspended particles stick to these compounds and create large and heavy clumps of material.

Sedimentation

Water is left undisturbed to allow the heavy clumps of particles and coagulants to settle out.

Filtration

Water is run through a series of filters that trap and remove particles still remaining in the water column. Typically, beds of sand or charcoal are used to accomplish this task.

Disinfection

Water, now largely free of particles and microorganisms, is treated to destroy any remaining disease-causing pathogens, commonly done with chlorination (the same process used to eliminate pathogens in swimming pools), ozone, or ultraviolet radiation. Water is now safe to drink and is sent to pumping stations for distribution.

sources, including the following: discharges of pathogenic and chemical contaminants from industry and wastewater treatment plants; runoff of pesticides, nutrients, and pathogens from agriculture regions; household use of cleaners, pharmaceuticals, and pesticides; leaking septic systems; and storm water runoff. Human exposure to water contaminants occurs from two primary sources: *(a)* direct exposure through ingestion of drinking water, dermal absorption, and inhalation; and *(b)* accumulation of contaminants in aquatic organisms and, in turn, human consumption of these organisms. The sources, exposure routes, and health effects of a representative set of contaminants in surface water and groundwater are discussed next.

Pathogens

Human history has been plagued by disease and suffering associated with pathogen-contaminated drinking water. John Snow identified contaminated drinking water as the source of a cholera epidemic that killed more than 600 people in London in 1854. Subsequently, water treatment, primarily with chlorine, became widespread and reduced pathogen-associated diseases. However, many developing countries continue to have inadequate water treatment capability and significant public health threats as a result, including cholera, typhoid, and other diseases associated with pathogen-contaminated drinking water.

Pathogen contamination of drinking water is not limited to developing countries and regions. Pathogen-associated disease reemerged during the 1990s as an important public health threat in countries and regions with advanced water treatment. In 1993, an outbreak of cryptosporidiosis in Milwaukee, Wisconsin, affected 400,000 people and more than 100 people died as a result. The outbreak was likely due to both *(a)* runoff from agricultural areas polluted with feces from cattle or other animals, and *(b)* water treatment that was inadequate to kill Cryptosporidium oocysts. Parts of Milwaukee have taken steps to improve water treatment to prevent future Cryptosporidium outbreaks, including the use of advanced filtration, ozonation, and ultraviolet (UV) treatment. However, other emerging problems associated with pathogen contamination of water threaten public health in both developing and developed countries.

Bioterrorism

An emerging potential source of pathogen contamination of drinking water supplies is bioterrorism.[3] Pathogens, such as *Clostridium perfringens* and the bacteria that cause anthrax and plague, and biotoxins, such as botulinum, aflatoxin, and ricin, have been weaponized. They are potentially resistant to disinfection by chlorination, and they are stable for relatively long periods in water.[4] While water supply systems provide some dilution, sophisticated technologies, such as use of microcapsules containing pathogens or biotoxins, could be used to disperse human pathogens in drinking water systems. An attack could be enhanced by introduction of an agent into the distribution system.

While the probability of terrorist contamination of drinking water is extremely low, the consequences could be very great. Therefore, preventing human exposure to pathogens is critically important and requires rapid detection in source water and water distribution systems. Rapid detection technologies, such as DNA microchip arrays, immunologic techniques, micro-robots, optical technologies, flow cytometry, and molecular probes, are under development. However, none is available commercially, and none have been tested in large drinking water systems.

Contamination at Swimming Beaches

While exposure to pathogens is typically through consumption of contaminated drinking water, ingestion of pathogen-contaminated water while swimming has also become a public health concern. Swimming beaches in the United States are visited by over one-third of U.S. residents. Epidemiological studies have demonstrated that 1 in 10 people becomes ill after swimming at beaches that were open—and considered safe—for swimming. Contamination of water at swimming beaches causes, or has the potential to cause, several types of diseases, including ear, nose, and throat infections (such as swimmer's ear—otitis externa), respiratory infections, and diarrheal diseases. Some of these disorders may be life-threatening to young children, older

people, and individuals with compromised immune systems.

The Natural Resources Defense Council (NRDC) reports that, during 2007, there were more than 20,000 days of swimming-beach closings and advisories, with over 10,000 due to stormwater pollution. (The Environmental Protection Agency [EPA] estimates that more than 10 trillion gallons of untreated stormwater enters U.S. surface waters annually.) More than 4,000 beach-closing days and advisories were due to sewage spills and overflows. The NRDC also reports that nearly 60% of closings and advisories were caused by unsafe bacterial levels (exceeding water quality standards), while approximately 40% were either precautionary (after a heavy rainstorm) or due to a specific pollution event.

Public health agencies typically take the lead in monitoring water quality at swimming beaches. Monitoring focuses almost exclusively on *Escherichia coli* or fecal coliforms, although monitoring of other parameters, such as pH, nutrients, and temperature, is also conducted. *E. coli* and fecal coliforms occur in the gastrointestinal tracts of higher animals and, as a result, are common in animal feces. Although the strain of *E. coli* commonly found in fecal-contaminated water and fecal coliforms does not cause human disease, coliform contamination is used as an indicator of other human pathogens, such as Giardia and Cryptosporidium.

Until recently, monitoring for pathogens at swimming beaches was sporadic at best. However, the Beaches Environmental Assessment and Coastal Health (BEACH) Act of 2000 provided funding for coastal states, including those in the Great Lakes region, to test water at swimming beaches for fecal contamination. Water quality monitoring now occurs much more frequently as a result. However, there are still deficiencies in water quality monitoring at beaches and in source identification and remediation. Beach Protection Act bills pending in Congress would reauthorize and strengthen the BEACH Act of 2000 by providing funding to identify and clean up contaminant sources. The strengthened bills would also require states to use rapid testing methods for pathogen indicators so that the public could be warned of contamination in a timely manner.

Chemical Contaminants

Lead

Lead occurs naturally in the earth's crust and is used in a variety of industrial applications. Previous use of lead in gasoline in the United States contaminated surface waters, although at very low levels that have not likely resulted in significant human exposure.

Because of its malleability and corrosion resistance, elemental lead has been used in water supply pipes since Roman times. (The word *plumbing* comes from the Latin for "lead.") In older cities, public water supply pipes may still contain lead, although more than 99% of all public drinking water systems have lead concentrations less than 0.005 ppm. However, lead concentrations in the water of homes and other buildings may be significantly higher and may pose a threat to human health. Homes built before 1986 are more likely to have lead pipes, joints, and solder, although new homes are also at risk. Even pipes that are considered "lead-free" may contain up to 8% lead and can leave significant amounts of lead in the water for the first several months after their installation.

The acidity of drinking water plays an important role in the availability of lead, with higher concentrations occurring in waters that are acidic. Acidic water (water with pH below 6.0) corrodes leaded pipes and solder, and results in leaching of lead into the water distribution system. When water is acidic and remains in contact with the pipe for hours, the lead concentration in the first draw may be considerable.

The EPA's maximum contaminant level goal (MCLG) for lead in drinking water is zero. However, the EPA has not developed a reference dose (RfD) or maximum contaminant level (MCL) for lead in water because health effects occur at very low levels and likely lack a threshold. The EPA requires drinking water systems to install or improve corrosion control to minimize lead levels at the tap, install treatment to reduce lead in source water entering the distribution system, and replace lead service lines when more than 10% of targeted tap samples exceed lead concentrations of 15 μg/L. Drinking water systems are also required to conduct public education programs if levels remain above 15 μg/L after reduction actions are taken. Where lead contamination

occurs as a result of plumbing, removal of the existing plumbing and installation of lead-free plumbing and fixtures should prevent further exposure. When this is not practical, running water for 30 seconds before drinking or cooking will reduce the lead concentrations, particularly when water has not been used for a prolonged period. (See also Chapters 11 and 19.)

Arsenic

Arsenic, an element that occurs naturally in soil and rock, is released to the environment via leaching to water and from anthropogenic sources, including ore-smelting operations. The average concentration of arsenic in surface water and groundwater is about 1 ppb, although much higher concentrations can occur locally.

Arsenic in water can be lethal at concentrations of 50 to 60 ppm. At concentrations as low as 300 ppb, arsenic can cause nausea, vomiting, and diarrhea. Chronic exposure to lower levels of arsenic can cause skin changes, including darkening and small corns or warts. The International Agency for Research on Cancer (IARC), the EPA, and the National Toxicology Program (NTP) have classified arsenic as a known human carcinogen. The EPA has set an MCL for arsenic in drinking water of 10 ppb (μg/L).

Arsenic has been found in groundwater throughout the United States (Fig. 8-2), in some cases at concentrations greater than the EPA drinking water standard. However, the public health toll associated with these concentrations pales in comparison to the disaster that has occurred in Bangladesh and in West Bengal, India, where millions of people drink from groundwater heavily contaminated with arsenic.

Bangladesh and West Bengal have some of the highest rates of waterborne infectious disease worldwide, including shigellosis, typhoid, cholera, and hepatitis A. To address this problem, well water was heavily promoted and developed as a safe alternative to untreated surface water, and people were instructed to rely on groundwater as their primary source of drinking water. As a result, the incidence of waterborne infectious disease declined dramatically. However, in the 1980s, evidence of arsenic contamination

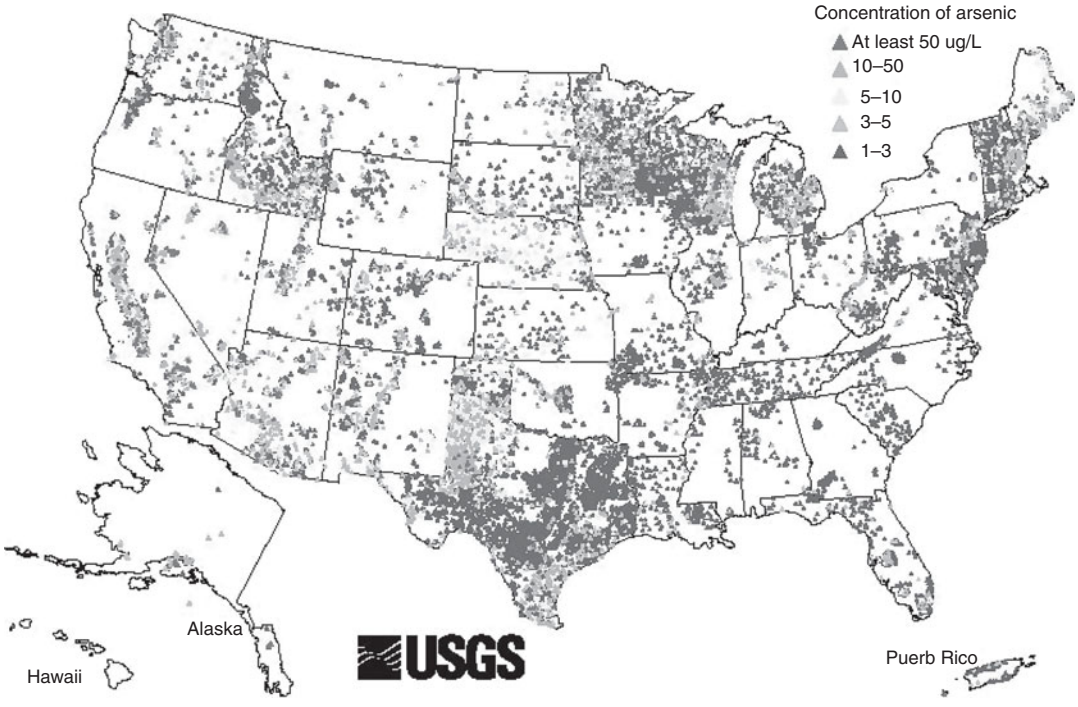

Figure 8-2. Arsenic concentrations in groundwater in the United States. (*Source*: Ryker SJ. Mapping arsenic in groundwater. Geotimes 2001; 46: 34-36.)

was found in groundwater, and, during the mid-1990s, the issue gained broad public attention. WHO estimates that more than 30 million people in Bangladesh may be exposed to arsenic concentrations in drinking water greater than 50 ppb (Fig. 8-3).

WHO also estimates that drinking arsenic-contaminated water in Bangladesh has caused over 100,000 cases of skin disorders. Ultimately, skin and internal cancers caused by arsenic exposure will become major health issues in the region with considerable associated social and economic hardship. Extensive water quality testing and on-site mitigation, with use of deep, arsenic-free wells, rainwater harvesting, installation of treatment plants, and extensive training and education have been implemented. However, the efficacy of these programs in reducing arsenic exposure and the risk of associated disease has not been assessed.[5]

Atrazine

Atrazine, a triazine pesticide, has been used for over 35 years and is applied to more than 65% of U.S. corn acreage, as well as sorghum, sugar cane, macadamia nuts, and conifer trees. In the United States, approximately 10 million pounds of this restricted-use pesticide are applied annually.

The use of atrazine results in runoff, leaching, and volatilization from agricultural soils, and transport to surface water, groundwater, and the atmosphere. The EPA estimates that atrazine

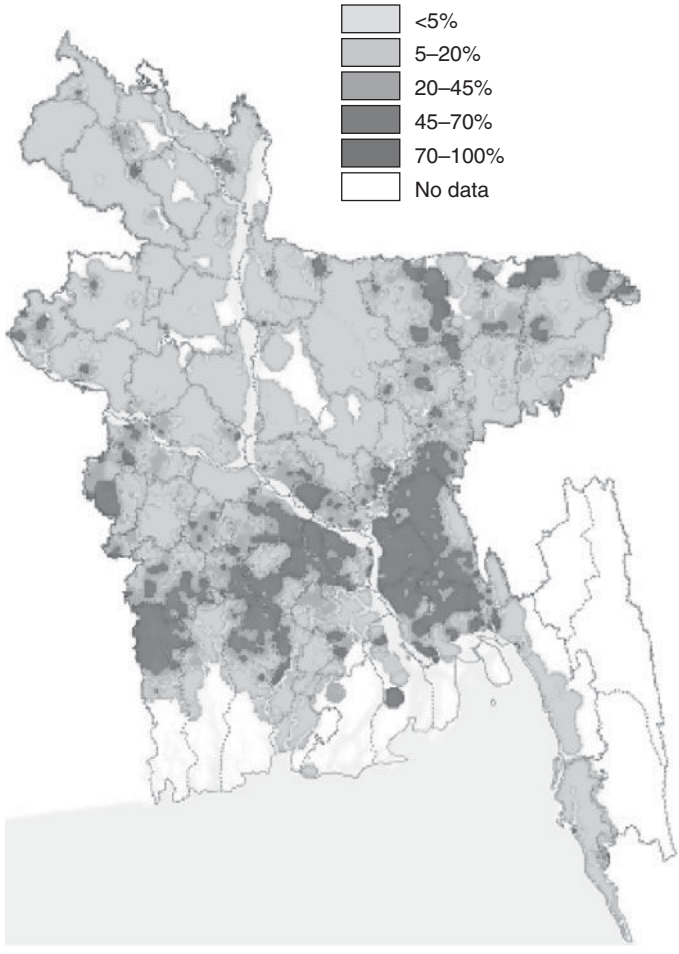

Figure 8-3. Probability of groundwater concentration exceeding 50 ppb (μg/L) in Bangladesh. (*Source*: McArthur JM, Ravenscroft P, Safiullah S, Thirlwall F. Arsenic in groundwater: testing pollution mechanisms for sedimentary aquifers in Bangladesh. Water Resources Research 2001; 37: 109–117.)

is present in over 1,500 community water supplies and over 70,000 rural domestic wells nationwide. Atrazine has been detected in drinking water in over 40% of municipal wells tested in Midwestern states and in over 31% of drinking water wells tested in Maine. It has been found in more than 98% of samples collected from Midwestern streams, rivers, and lakes after it was used on crops. It has also been the most commonly detected pesticide in southern Florida canals.

As would be expected of a broad-spectrum herbicide, atrazine is toxic to primary producers (plants and algae) in surface waters. Chronic, lower-level atrazine exposure causes changes in community structure and function by changing species composition of aquatic plant and algal communities and the productivity of these systems. In turn, the feeding behavior and efficiency of organisms that consume plants and algae are affected, resulting in changes in species assemblages and communities at higher levels in the food chain.

Atrazine has relatively low mammalian acute toxicity (its LD_{50} in rats is greater than 1,000 ppm). However, because of its widespread use and its occurrence in surface and ground water, concern has arisen over adverse effects in humans and on aquatic or semi-aquatic organisms associated with chronic, low-level exposures. Atrazine may cause cancer in humans and affect human reproduction and development via disruption of the endocrine system. Atrazine also causes developmental and reproductive toxicity in laboratory animals and in naturally occurring amphibians.

Because of its widespread use, its occurrence in groundwater and surface water systems, and concern with its potential to cause cancer, endocrine disruption, and reproductive and developmental effects in human and nonhuman organisms, there have been calls to further restrict or ban the use of atrazine in the United States. These calls have been resisted by the pesticide's manufacturers and users, and a highly charged debate about atrazine's regulation has ensued (Box 8-3).

Mercury

Mercury is an important contaminant of many surface water systems; it accumulates in fish and other aquatic organisms that feed high in the food chain. People consuming mercury-contaminated aquatic organisms (primarily fish)—the most important, nonoccupational source of human exposure to mercury—may be exposed to concentrations that pose threats to health.

Mercury is a naturally occurring element that is found in the earth's crust. It occurs in surface waters as a result of direct solubilization and from direct and indirect industrial discharges. Discharges from chloralkali plants, leaking landfills, incineration of mercury-containing products, and combustion of coal are important sources of mercury contamination of surface water (Box 8-4). While some of these discharges are direct and result in local contamination, incineration of mercury-containing products, such as in medical waste, and combustion of coal used for electrical generation discharge mercury to the atmosphere, where it is transported long distances and deposited in lakes. Nearly 80% of anthropogenic emissions of mercury to the air come from fossil fuel combustion, mining and smelting, and incineration of waste.

Inorganic mercury that enters lakes is relatively insoluble; however, it is readily transformed by bacteria to its organic form (methyl mercury), which accumulates in the tissues of aquatic organisms, including fish. Because biomagnification (accumulation and concentration up the food chain) is the predominant mechanism of accumulation in aquatic ecosystems, mercury concentrations are highest in fish that feed at the top of the food chain, such as pike, shark, tuna, and swordfish. Mercury concentrations in these fish may be biomagnified as much as 100,000 times over concentrations in surface water. The concentration of mercury in aquatic food chains is influenced by pH, with more accumulation occurring in water with lower pH. Acid precipitation, which results from the discharge of sulfates and nitrates from combustion of fossil fuels, may therefore play an important role in mercury accumulation in organisms in acidified lakes.

At high concentrations, mercury can damage the brain, kidneys, and other organs. Chronic prenatal exposure to lower concentrations of methylmercury and exposure of infants via breast milk cause developmental disorders, such

Box 8-3. The Debate over Regulation of Atrazine

While several adverse health effects have been associated with low-level, chronic exposure to atrazine, the most controversial are the potential to cause cancer and adverse effects on reproduction and development (in humans and amphibians) via disruption of the endocrine system. In 2003, the Environmental Protection Agency (EPA) stated that atrazine is "not likely to be carcinogenic in humans," despite a different conclusion of its Science Advisory Board (SAB).[1] Studies that the EPA and the SAB evaluated showed significantly elevated rates of prostate cancer among men exposed to atrazine in a pesticide manufacturing plant; however, enhanced detection through aggressive screening impaired definitive determination of atrazine's potential carcinogenicity. Despite confounding effects of screening and limitations of sample size, the SAB stated that there was sufficient evidence to conclude that atrazine played a role in the increased cancer rate at the plant, and that screening was only a partial explanation for the cancer increase.

Studies have also demonstrated an elevated risk of intrauterine growth retardation (IUGR) in the offspring of women who obtained water from atrazine-contaminated wells. However, while atrazine's reproductive and developmental toxicity as well as its endocrine disruption characteristics have been confirmed in studies of laboratory animals, a definite causal relationship between IUGR and human atrazine exposure has not been established because other pesticides in drinking water have also been correlated with IUGR.

Atrazine's endocrine-disrupting potential and its reproductive and developmental toxicity are noteworthy in that they appear to also affect animals in the wild, with attendant concern for ecological effects. Atrazine has been associated with endocrine disruption and developmental abnormalities in amphibians. The EPA conducted an assessment of the literature on these adverse effects and concluded that "there is sufficient evidence to formulate a hypothesis that atrazine exposure may impact gonadal development in amphibians but there are currently insufficient data to confirm or refute the hypothesis."[1] Despite relatively extensive literature that describes these effects, debate among academic, government, and industry scientists continues around atrazine's potential to cause developmental abnormalities in amphibians.

In 2003, the EPA approved a reregistration request for atrazine in the United States in light of scientific uncertainties and despite the conclusions of the SAB regarding human carcinogenicity and reproductive and developmental toxicity of atrazine in humans and amphibians. While allowing continued use of atrazine, the EPA requested additional information and studies of the pesticide's effects in humans and amphibians. In 2007, the EPA Scientific Advisory Panel (SAP) reviewed studies and data collected since 2003 and concluded that data are still not available to reject the EPA hypothesis that atrazine, at environmentally relevant exposure concentrations, adversely affects amphibian gonadal development. In contrast, the European Union and several European countries have banned the use of atrazine because of its widespread occurrence in drinking water and its potential health effects in humans and amphibians—a decision based on the Precautionary Principle for the management of potentially hazardous chemicals.

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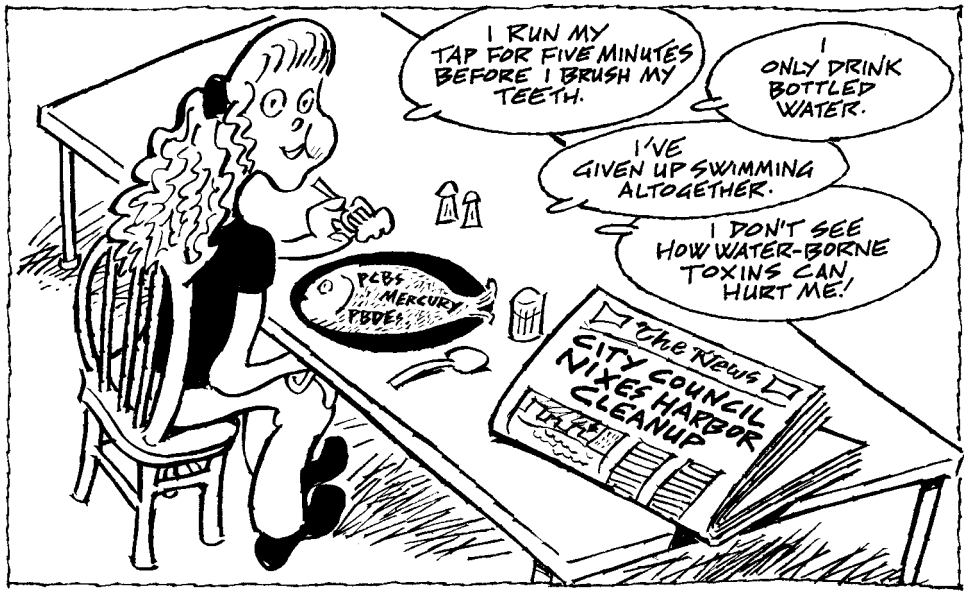

(Drawing by Nick Thorkelson.)

Box 8-4. Coal Waste and Water Quality

The environmental impacts of contaminants, including mercury and greenhouse gases such as carbon dioxide, discharged from coal-fired power plants are well known. Less well known are the impacts of coal combustion wastes (CCWs) on human health and the environment. Coal-fired power plants in the United States produce about 129 million tons of waste per year.[1] These wastes, which are typically stored in surface impoundments and landfills, contain hazardous metals, such as arsenic, lead, and mercury, that pollute air, soil, groundwater, and lakes, streams, and rivers. Humans are exposed to pollutants from CCWs by drinking contaminated groundwater, breathing polluted air, eating fish from contaminated lakes and streams, and consuming vegetables grown in polluted soils. Contaminants in coal waste also harm plants and animals in terrestrial and aquatic ecosystems. According to the Environmental Protection Agency (EPA), pollutants in CCWs have poisoned surface water and groundwater supplies in at least 23 states.[2]

The EPA recently conducted a risk assessment of stored CCWs and determined that they pose very high risks to humans and the environment. For example, the EPA found that the human cancer risk posed by arsenic in CCWs was 900 times greater than safe levels. The EPA also found that boron, lead, arsenic, cobalt, selenium, and barium in CCWs posed risks to birds, frogs, fish, and other organisms that were up to 2,000 times safe levels. Critics of the EPA analysis suggest that risks are overstated and the stored CCWs are unlikely to enter groundwater and surface water. Recent events suggest otherwise.

In late 2008, a containment structure surrounding stored sludge from a coal-burning power plant near Harriman, Tennessee, collapsed. The spill released over 1 billion gallons of coal ash into the Emory River, a tributary of the Clinch River and ultimately the Tennessee River, which serve as drinking water sources. The spilled ash contained heavy metals, including chromium, molybdenum, strontium, antimony, cobalt, thallium, arsenic, barium, beryllium, lead, selenium, boron, vanadium, mercury, and zinc. In the weeks following the spill, sampling revealed that concentrations of several toxic metals violated federal drinking water standards and Tennessee state water quality criteria. The direct impacts of the spill on human health and the environment will not be known for a long time, although a dredging program has begun to remove some of the ash that was deposited in river sediments near the spill site.

While the Tennessee coal ash spill is the largest in U.S. history, it is not an isolated event. Spills in Martin County, Kentucky (300 million gallons), Buffalo Creek, West Virginia (130 million gallons), and elsewhere have caused deaths and devastating effects on the environment. Yet the EPA has not regulated the storage or disposal of CCWs in any substantial way, leaving what little regulation exists to states—resulting in an uneven array of rules inadequate to protect people and the environment. The EPA's own risk assessment of CCW storage confirms that disposal of coal wastes in surface impoundments, such as the site in Tennessee, and in other types of landfills pose considerable risks to people and the environment. Citizen and environmental groups have recently called on the EPA to promulgate minimum federal standards for safe disposal of CCWs, specifically to prevent the open dumping of CCWs and thereby protect human health and the environment from contaminants in them. As of mid-2009, the EPA had not promulgated regulations to manage CCW storage or disposal.

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as delayed onset of walking, and abnormalities of language, attention, and memory.

While a contaminant of many foods, most adult intake of mercury is from seafood. Concentrations of mercury in shark, swordfish, tile fish, mackerel, and albacore tuna occur at levels that have triggered warnings to women of childbearing age, pregnant women, nursing mothers, and young children to avoid consumption of these fish.[6] Similarly, 30 state natural resource and health agencies in the United States have advised these same individuals to reduce or avoid consumption of fish, including perch, northern and walleye pike, musky, and other species caught recreationally.[7]

Polychlorinated Biphenyls

Polychlorinated biphenyls (PCBs) are representative of a class of nonpolar, chlorinated organic compounds that include DDT, chlorinated dioxins, toxaphene, and many others. Polychlorinated biphenyls and other compounds in this class are relatively insoluble in water, persistent in some environmental compartments (such as sediments), and accumulate to a high degree in animals and plants. While they are present in water at extremely low concentrations, they occur at very high concentrations in aquatic organisms, such as fish, posing health risks to people who consume them.

Polychlorinated biphenyls were manufactured and sold in the United States from 1929 to 1977, when the EPA banned their manufacture. During this period, over 1 billion pounds were produced. Despite a ban on their manufacture, PCBs continue to be encountered in various products and applications, including transformers and capacitors, heat-transfer fluids, flame retardants, inks, adhesives, carbonless duplicating paper, paints, pesticide extenders, plasticizers, and wire insulators. They have also been found in over 500 hazardous waste sites in the United States.

Polychlorinated biphenyls concentrate in the fatty tissues of aquatic organisms, including fish. Bioconcentration and biomagnification of PCBs in upper levels of the food chain can lead to concentrations in the tissues of predatory fish that are over 1 million times greater than concentrations in surrounding water. As a result, fish are the most significant source of PCB exposure to humans and other fish-eating animals.

Polychlorinated biphenyls are classified as probable human carcinogens by IARC, the EPA, and the NTP. The hepatotoxic effects of PCBs include induction of microsomal enzymes, liver enlargement, increased serum levels of liver-related enzymes and lipids, altered porphyrin and vitamin A metabolism, and histopathologic alterations that progress to noncancerous lesions and tumors. PCBs also cause adverse dermal effects (chloracne), ocular effects (Meibomian gland hypersecretion and abnormal conjunctival pigmentation), and immunological effects (including increased susceptibility to respiratory tract infections, increased prevalence of ear infections in infants, decreased antibody levels, and changes in T lymphocytes). Exposure to PCBs has also been associated with adverse effects on sperm morphology and production and menstrual disorders. Anthropometric effects, including reductions in head circumference and birthweight, and neurobehavioral abnormalities, including decreased neuromuscular maturity, abnormal reflexes, reduced psychomotor scores, impairment of short-term memory, decreases in visual recognition, and reduced activity levels have been observed in children exposed to PCBs in utero. Some of these effects have persisted into later childhood.

As a result of high PCB concentrations in the tissues of many fish species, warnings have been issued to reduce consumption of the most contaminated species, including salmon, trout, and walleye caught by recreational anglers in the Great Lakes and from other inland water bodies. Concern has also been raised recently around PCB contamination in farm-raised Atlantic salmon.[8]

Polybrominated Diphenyl Ethers

Polybrominated diphenyl ethers (PBDEs) comprise a relatively new class of compounds used as flame retardants in many commercial and household products. The use of PBDEs has increased dramatically in recent years, with annual sales reaching more than 70,000 metric tons.[9] Widespread use has resulted in migration from commercial products to the environment, including surface water and aquatic organisms (primarily fish), with human exposure occurring through consumption of contaminated fish. Polybrominated diphenyl ethers are present in human blood, milk, and fatty tissues. Concentrations in people have increased 100-fold over the past 30 years, with a doubling time of about 5 years; and concentrations in North Americans are significantly higher than concentrations in Europeans.[9]

Polybrominated diphenyl ethers are relatively insoluble in water but highly lipophilic; thus, they bioaccumulate in fish and other aquatic organisms as well as terrestrial species, including humans, that consume aquatic organisms. Concentrations of PBDEs in fish are highly variable, depending on the type of fish and its location. For instance, PBDE concentrations in fish from Europe are about one-tenth those in fish from North America, likely due to the proximity of fish feeding areas to PBDE sources.[9] Regional distribution of PBDEs may also be associated with Europe's more aggressive approach of banning PBDE manufacture and use.

Some PBDE congeners are metabolically active and induce hepatic cytochrome P450 IIB1 and IA1. They also have weak or moderate binding affinity to the Ah receptor. PBDEs disrupt spontaneous behavior, impair learning and memory, and induce other neurotoxic effects in adult mice exposed neonatally. Polybrominated diphenyl

ethers are endocrine disruptors, altering thyroid hormone homeostasis and causing a dose-dependent depletion of thyroxine. They are agonists of estrogen receptors (both ERα and ERβ), an effect that may be enhanced by in vivo metabolism. Polybrominated diphenyl ethers have not been demonstrated to be carcinogenic in rodent bioassays, although some concern for PBDE carcinogenesis continues to be raised. If humans are as sensitive as experimental animals to the adverse effects of PBDEs, current concentrations in humans may leave little or no margin of safety—arguing for close evaluation of the management of PBDEs and potentially aggressive regulation.

Disinfection By-products

Disinfection by-products (DBPs) are created by the interaction of organic matter in source waters with chlorine and other water disinfectants. Disinfection by-products include trihalomethanes, such as chloroform; haloacetic acids, such as trichloroacetic acid; bromate, which is formed when ozone is used for water disinfection; and chlorite, formed when chlorine dioxide is used for disinfection. The health effects of exposure to these substances include carcinogenicity (primarily bladder, colon, and rectal cancer) and reproductive and developmental effects, including spontaneous abortions, stillbirths, neural tube defects, preterm births, intrauterine growth retardation, and low birthweight.

Disinfection by-products are formed by treatment of drinking water. Without treatment, the occurrence of pathogens that cause cholera, typhoid, cryptosporidiosis, and other diseases would increase with commensurate threats to public health. To address the DBP problem, the EPA issued the Stage 1 Disinfectants and Disinfection Byproducts Rule in 1998, which requires drinking-water treatment plants to attain certain levels of disinfection and, at the same time, reduce DBPs to specified levels prior to distributing water. The Rule also sets goals for the complete removal of some DBPs, although deadlines to achieve these goals were not specified. Treatment plants that use surface waters with high concentrations of organic materials were also required to reduce the concentrations of these materials to specified levels prior to treatment to decrease or avoid the formation of DBPs. The EPA issued the Stage 2 Disinfectants and Disinfection Byproducts (DBP) rule in 2006. The Stage 2 rule refines the Stage 1 rule and attempts to strengthen health protection for customers of systems that deliver disinfected water. The rule focuses specifically on two important DBPs: trihalomethanes and haloacetic acids.

The EPA estimates that the nationwide cost of complying with the Stage 1 rule is about $700 million, with an additional cost of about $80 million annually for compliance with the Stage 2 rule. However, the agency also indicates that the benefits of implementation of the rules, which include the prevention of cancer as well as reproductive and developmental disorders, far outweigh the costs. For example, the EPA estimates that nearly 300 cases of bladder cancer will be prevented by implementation of the Stage 2 rule, providing an annual benefit of $1.5 billion. Additional benefits, including prevention of reproductive and developmental effects, will also accrue from implementation of the Stage 2 rule, although the EPA has not estimated their economic benefits.

REGULATING AND MANAGING WATER QUALITY

Water quality and the adverse health effects associated with water pollution can be managed by *(a)* preventing pollution before it occurs, *(b)* treating water after pollution has occurred, and *(c)* implementation of public health practices that reduce or eliminate human exposure when pollution prevention and treatment have not occurred or are ineffective.

Treatment-Based Approaches

The primary statutes to manage water quality in the United States are the Clean Water Act and the Safe Drinking Water Act. The Clean Water Act, adopted in 1972, emphasizes treatment of wastes before they are discharged to surface water. Treatment thresholds are guided by chemical-specific water quality criteria, which are risk-based contaminant concentrations that,

if not exceeded, should prevent adverse impacts of pollutants on human health and the environment. Treatment thresholds are also based on technology guidelines that require industrial sectors to install the best available, economically achievable levels of water treatment to remove pollutants prior to their discharge to surface waters. The Clean Water Act has been remarkably successful in reducing toxic chemicals, such as PCBs, and pathogenic pollutants, such as bacteria and viruses, discharged from point sources—waste pipes of industrial facilities and wastewater treatment plants. As a result, surface waters are cleaner now than prior to the adoption of the Act. However, the focus of the Act on point sources has addressed only a portion of surface water quality problems in the United States.

Nonpoint source pollution, also called polluted runoff, enters lakes and streams from farms and animal feeding operations, leaking hazardous waste and sanitary landfills, septic systems, stormwater runoff that carries pollutants from city streets and sidewalks to surface waters, and the atmosphere. Nonpoint contaminants, such as PCBs (from contaminated sediments), mercury (atmospheric deposition), atrazine (from agricultural runoff), and many pathogens (from animal feeding operations, leaky septic systems, and runoff from urban areas) enter surface waters relatively uncontrolled by the Clean Water Act.

Prior to 1972, discharges of pollutants, such as PCBs from paper mills, occurred with little control and accumulated to very high concentrations in the sediments of lakes and rivers. They have persisted in sediments and are resuspended in surface waters during floods and other disturbances—a particularly challenging nonpoint pollutant source. In the Great Lakes region, dozens of hotspots (areas of concern) have been identified, where sediments are highly contaminated with persistent, bioaccumulative toxicants, such as PCBs. In only a few cases are efforts underway to cap or remove contaminated sediments, often at very significant expense. However, without cleanup, these contaminants will continue to accumulate in fish and other organisms that will be consumed by humans and wildlife.

The Safe Drinking Water Act, passed in 1974, is a treatment-based statute designed to control and reduce toxic and pathogenic compounds in drinking water, which in the United States is provided by over 170,000 treatment plants. The Act gives the EPA authority to set national, health-based standards for both naturally occurring drinking-water contaminants, such as bacteria and viruses, and contaminants of anthropogenic origin, such as lead and atrazine. Implementation of the standards occurs typically at the treatment plant, with enforcement provided either by states or the EPA. While much of the Act focuses on treatment to reduce contaminant concentrations at the tap, revisions in 1996 gave the EPA and the states greater authority to protect ground and surface water that serves as a source of drinking water. The Act also requires water suppliers to notify the public when there is a contamination problem in a drinking water system and treatment plants to provide annual reports to their users on the quality of their tap water.

Sometimes an approach to manage water quality to protect human health leads to unintended consequences. In 2002, elevated lead levels were discovered in water supplied to more than 6,000 homes in Washington, DC. Chlorination of the city's water to kill pathogens, as required by the Safe Drinking Water Act, created carcinogenic disinfection by-products. To reduce disinfection by-products as required by the EPA under the Safe Drinking Water Act, corrosive chloramines were added to the city's water, mobilizing lead in its aging pipes and resulting in lead concentrations in the drinking water of some homes 20 times greater than the EPA recommended level. Ultimately, 23,000 lead-containing water service pipes will need to be replaced. As of July 2007, about 11,000 pipes had been replaced, and, since 2008, homeowners have been encouraged to replace private portions of water pipes containing lead. The city government has informed the population about the lead hazard and provided recommendations to reduce lead exposure. In addition, the city has optimized corrosion control by adding orthophosphate, a corrosion inhibitor, during water treatment. These measures have helped reduce lead levels in tap water; in 2008, the city

declared its water distribution system virtually free of lead.

Exposure Reduction

The Clean Water Act has been relatively effective in regulating the discharge of contaminants from point sources, and the Safe Drinking Water Act has accomplished much of its goal of ensuring that contaminants do not occur in drinking water. However, these statutes have not addressed the vast quantities of in-place pollutants (in sediments) and other nonpoint sources of pollutants, such as leaking hazardous waste landfills or the atmosphere. As a result, PCBs, mercury, and other bioaccumulative contaminants have become concentrated in the tissues of fish and other aquatic organisms, posing threats to human health and the environment.

Regulation of contaminants in fish sold commercially, such as PCBs and mercury, occurs under the Federal Food Drug and Cosmetic Act (FFDCA). The Food and Drug Administration (FDA) sets regulatory thresholds, called tolerance levels, for PCBs, mercury, and other compounds in fish. When the concentration of a contaminant in fish or other foods exceeds a tolerance level, the FDA can remove the food from commercial markets or it can issue a consumption warning, as it did in 2001 for mercury in shark, swordfish, tilefish, and mackerel. However, many fish are caught and consumed by sport or recreational anglers, and contaminants in these fish are not regulated by the FDA.

The EPA has developed methods to manage the health risks of toxicant exposure through consumption of contaminated fish caught by sport or recreational anglers. Its risk-based method is used to develop fish consumption advisories for compounds, such as PCBs and mercury, that are commonly found in sport-caught fish. Consumption advisories are typically issued by states and warn anglers and their families to restrict or eliminate consumption of particular species and size classes, based on tissue concentrations of individual contaminants and combinations of contaminants.

The risk-based approach to consumption advisories developed by the EPA conflicts, in many cases, with the tolerance levels set by the FDA for commercially sold fish. (One important exception is mercury, for which the FDA and EPA have developed a consensus approach for management in fish tissues.) For example, a PCB concentration of 1 mg/kg (ppm) will trigger stringent, "do-not-eat" consumption advice for a recreationally caught salmon in the Great Lakes, while the same salmon may be sold in commercial markets without restriction since this concentration is below the FDA tolerance level for PCBs (2 mg/kg). This issue gained significant attention in 2004 when some types of commercially sold salmon were found to have concentrations of PCBs, toxaphene, dieldrin, and other contaminants at levels that would trigger stringent EPA-based consumption advice, but no action by the FDA.[8] The difference in the two approaches is attributable to the reliance of the EPA on a public health protective, risk-based approach to the development of fish consumption advisories, while the FDA incorporates considerations not based on health, such as economic benefit and analytical detection capabilities in the development of tolerance levels. The FDA has also fallen behind in updating its tolerance levels for most contaminants in fish; for example, the 20-year-old tolerance level for PCBs is one of the most recent levels established by the FDA.

Source Control and Prevention

Water quality managers have recognized the limitations of end-of-pipe, treatment-based controls as a water quality management tool. This approach is not useful for many of the nonpoint sources that plague surface waters. It also becomes cost prohibitive as toxicant concentrations have been reduced to very low, but still harmful, levels. As a result, attention has turned to prevention-based approaches for the management of water quality. In 1992, a cooperative project of more than 80 public, private, and nonprofit groups brought together by the Water Environment Federation produced a consensus report, "A National Water Agenda for the 21st Century." Popularly known as Water Quality 2000, the project produced a vision statement and a goal for the nation's waters of protecting and enhancing water quality that supports

society and natural systems. To achieve this goal, it called for consideration of *(a)* all phases of the water cycle in the structuring of management approaches; *(b)* water as one part of a total environmental management plan to avoid transferring problems from one environmental medium to another; *(c)* the link between land use and water quality; *(d)* the relationship between water quality policy in the United States and global environmental issues; *(e)* promotion of source reduction and waste minimization; and *(f)* water conservation and reuse.

Promotion of source reduction and waste minimization are underway in industry and agriculture and in many communities. Recycling and reuse of industrial waste has reduced point sources of pollutants and, concurrently, saved money by decreasing the need to purchase raw or unused resources. Similarly, measures in agriculture are being implemented to change crop rotation and tillage practices to reduce the need for large quantities of pesticides. Organic farming practices and produce from organic farms are increasing in popularity, with the concurrent benefit of reducing both leaching of pesticides to groundwater and runoff to surface water.

THE FUTURE OF WATER QUALITY MANAGEMENT

Billions of people live without access to safe, clean water. Targeted approaches to point and nonpoint source controls of contaminants in the United States and other higher income countries either are not available or are irrelevant to address the water needs of people in lower- and middle-income countries experiencing water stress. In 2000, the United Nations set millennium development goals (MDGs) to address the needs of the world's poorest people. The goals focus on child and maternal mortality, hunger, malnutrition, education, and, pertinent to this chapter, lack of water and sanitation. For example, it sets a target of halving, by 2015, the proportion of people who are unable to reach or to afford safe drinking water and the proportion of people who do not have access to basic sanitation. All nations combined are on track to meet the MDG drinking water target, although some regions, such as sub-Saharan Africa, are falling considerably behind. Unfortunately, many regions will likely fail to meet sanitation targets, including countries in Southern Asia and sub-Saharan Africa.

Water management is challenged by a convergence of water quality and water quantity issues. Technologies are available that can conserve enough water to reduce stress on threatened natural resources while accommodating agricultural, industrial, and residential uses.[2] By 2020, enough water could be saved by conservation during indoor residential use to meet the needs of over 5 million people, and sustainable irrigation practices could save 450,000 acre-feet of water per year, enough to satisfy the needs of another 3.6 million people. In some cases, water that is conserved is also water that does not have to be treated and discharged, eliminating the costs and adverse effects of treatment and discharge processes. However, water distribution and water quality issues will continue to impose constraints on addressing the world's water needs.

While conservation will play an important role in addressing both water quantity and quality, global management of water will be challenged by existing and new stressors. The world's growing population, which relies on the 0.01% of water worldwide that is usable, poses formidable challenges for water quality managers, health professionals, and government officials. The global effects of climate change also pose daunting challenges (Chapter 5). The United Nations Intergovernmental Panel on Climate Change predicts that increases in global mean temperature due to continued accumulation of greenhouse gases may account for up to 20% of the global increase in water scarcity. Climate change–induced disruption of traditional weather and runoff patterns could increase the frequency and severity of drought and floods, with attendant impacts on the structure and function of riparian ecosystems as well as on human health and humanmade structures. Climate change also will impact stratification and mixing patterns in surface waters, altering nutrient and contaminant cycles with potentially profound effects on aquatic communities and organisms—including humans—that rely on aquatic systems for sustenance. The climate change–induced emergence of new or modified pathogens will pose additional challenges to

water treatment systems designed to protect people and marine and freshwater ecosystems.

There has been much progress worldwide in increasing access to safe drinking water. The number of people with access to safe drinking water increased from 4 billion in 1990 to over 5 billion in 2006. (In addition, the number of people in developing countries with improved sanitation facilities increased by over 1 billion from 1990 to 2006.) Hopefully, this progress will continue so that eventually all people worldwide will have adequate access to safe drinking water.

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9

Food Safety

Craig W. Hedberg

Foodborne illnesses occur as the result of dynamic interactions among agents, hosts, and the environments in which these interactions occur (Fig. 9-1). This is a fundamental concept that can help us understand the nature of threats to food safety and to develop strategies to prevent the transmission of foodborne illness.

For example, *Escherichia coli* O157:H7 are enteric bacteria that are carried by cattle. During slaughter, the surfaces of carcasses may be contaminated. Carcass trimmings may be ground, which spreads contamination throughout large batches of ground beef. Because *E. coli* O157:H7 can cause bloody diarrhea in people of all ages and a life-threatening complication of the kidneys in young children and elderly people, the presence of *E. coli* O157:H7 in ground beef is an important public health problem. However, if a hamburger made from contaminated ground beef is thoroughly cooked, the bacteria will be destroyed, and no transmission will occur.

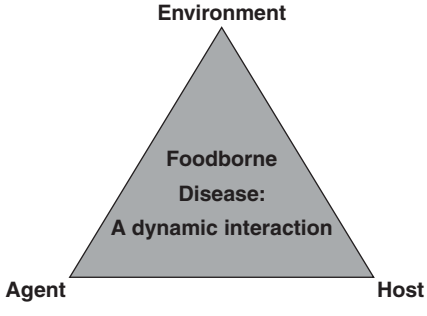

Figure 9-1. This triangle demonstrates the dynamic interactions between agent, host, and environment that result in the occurrence of foodborne disease.

Similarly, a salad maker in a restaurant who is infected with hepatitis A virus (HAV) and has poor personal hygiene and hand-washing practices may contaminate salads served to the restaurant's patrons. However, if the restaurant's patrons have been vaccinated against HAV, they would no longer be susceptible to infection, and no transmission of illness would occur.

These two examples highlight the interactions among agents, hosts, and environments that lead to disease transmission or, alternatively, can be altered to prevent transmission. For each of these diseases, a much broader range of environmental, technical, and policy interventions are used, and could be used, to prevent transmission, as described later in this chapter.

Food safety can be viewed in terms of a specific food product or an entire food system. For a specific food product, this requires a systematic approach that accomplishes the following:

- Characterizes the food item or process
- Identifies hazards that may occur with the food item
- Identifies points at which hazards contaminate the food item, enter the process, or can be eliminated or controlled at a level below which illness would be expected to occur
- Monitors control points
- Initiates corrective action when the process is out of control

All of these functions have corresponding functions that can be described for an entire food system. Food safety represents the combined use of all of these tools to detect, prevent, and abate foodborne illness hazards in the food supply. Because these hazards are the result of dynamic interactions, food safety is a constantly moving target that depends on a strong system of public health surveillance to *(a)* detect new and emerging food safety hazards, and *(b)* provide feedback on the efficacy of control strategies.

PUBLIC HEALTH SURVEILLANCE FOR FOODBORNE DISEASES: THE KEY TO HAZARD IDENTIFICATION

Public health surveillance is the ongoing, systematic collection, analysis, interpretation, and dissemination of data regarding a health event for use in public health action to reduce morbidity and mortality and to improve health.[1] Historically, foodborne disease surveillance has been conducted to accomplish the following:[2]

- Control and prevent outbreaks
- Determine the causes of foodborne disease
- Monitor trends in occurrence of foodborne disease

Accomplishing these tasks requires the coordination of three separate but related activities: *(a)* case surveillance for specific foodborne pathogens, such as Salmonella and *E. coli* O157:H7; *(b)* collection and evaluation of complaints of foodborne illness from consumers; and *(c)* investigation of foodborne illness outbreaks identified from pathogen-specific surveillance or consumer complaints.

The role of surveillance in driving the cycle of public health prevention related to foodborne disease outbreaks has been depicted by the Centers for Disease Control and Prevention (CDC) (Fig. 9-2). Surveillance identifies outbreaks, which are then investigated using a combination of epidemiological, laboratory, and environmental methods. Results of these investigations can *(a)* lead to direct public health interventions, or *(b)* stimulate applied targeted research to improve existing interventions or develop new ones. As new interventions are implemented, surveillance monitors their effectiveness, based on the occurrence of new outbreaks.

Although data on foodborne illness surveillance are frequently compiled at a national level, responsibility for foodborne disease surveillance in the United States resides at the state or local level, as authorized by state laws and regulations governing the reporting of communicable diseases. These laws vary widely, by state; there is therefore no consistent organization of foodborne disease surveillance in the United States.[3] For example, a 2007 survey of state health departments found that 12 states conducted foodborne disease surveillance by a central state office; 11 states, by regional state offices; and

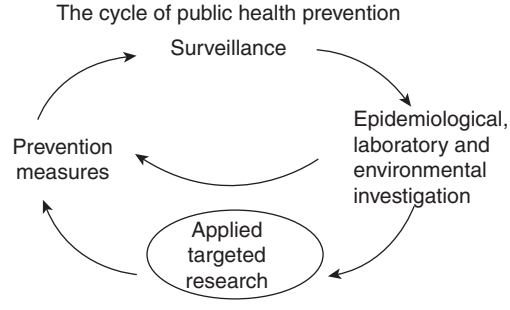

Figure 9-2. This drawing demonstrates how surveillance for foodborne disease outbreaks leads to preventive measures to improve food safety. (Adapted from: Tauxe RV. Molecular subtyping and the transformation of public health. Foodborne Pathogens and Disease 2006; 3: 4-8.)

27 states, primarily by local health agencies.[4] In addition to differing legal structures, resources to conduct surveillance activities vary widely by state and contribute to observed disparities in the distribution of reported foodborne outbreaks.[5]

In the United States, the CDC compiles reports of nationally notifiable diseases.[6] Unfortunately, only a few important foodborne diseases are nationally notifiable, and reported case counts do not provide any information about the source of the illnesses. In order to provide better data to determine trends in the occurrence of foodborne disease and to evaluate the causes of sporadic infections, the CDC established in 1996 the Active Surveillance Network for Foodborne Diseases (FoodNet). FoodNet has conducted, since 1996, active, population-based surveillance for seven major bacterial causes of foodborne disease at five sites, and, since 2007, at 10 sites (with 46 million people, 15% of the U.S. population).[7] FoodNet data provided the framework for the CDC's estimate that the annual burden of foodborne illness in the United States is 76 million cases, 323,000 hospital admissions, and 5,000 deaths.[8]

Trend data for the occurrence of *E. coli* O157:H7 and Salmonella infections have been very useful for evaluating the effectiveness of the regulatory efforts of the Food Safety Inspection Service of the U.S. Department of Agriculture to reduce foodborne illnesses associated with meat and poultry products. Results of sporadic case-control studies conducted by FoodNet have demonstrated a reduced occurrence of *E. coli* O157:H7 infections associated with undercooked hamburgers served in fast-food restaurants.[9] However, they have also highlighted an increased risk of infections of *E. coli* O157:H7, Campylobacter, and Salmonella generally associated with eating foods prepared outside the home.[10] These studies have therefore provided important information about the risks of foodborne disease transmission associated with restaurants that have helped explain and confirm the importance of restaurants as settings for foodborne disease outbreaks.

Although FoodNet has provided valuable information regarding the incidence and cause of sporadic infections, it was not established to detect or investigate outbreaks of foodborne disease. To enhance the usefulness of pathogen-specific surveillance, the CDC established the national molecular subtyping network for foodborne disease surveillance (PulseNet) in 1998. PulseNet is a national network of public health and food regulatory agency laboratories that perform standardized molecular subtyping of *E. coli* O157:H7, Salmonella, and Listeria by pulsed-field gel electrophoresis (PFGE). Patterns from cases are submitted electronically to the CDC, stored in a database, and periodically reviewed to detect unusual, large, or multistate clusters that may represent a foodborne outbreak.[11] PulseNet has been responsible for the identification of many recent multistate outbreaks, and it has greatly enhanced the public health system's ability to identify new and emerging food hazards, such as the role of fresh produce items and processed foods associated with *E. coli* O157:H7 and Salmonella.[2,5]

The CDC collects data on foodborne disease outbreaks (FBDOs) from all states and territories through the Foodborne Disease Outbreak Surveillance System.[2] The overall goal of a foodborne outbreak investigation is to rapidly obtain sufficient information to implement specific interventions to abate the outbreak. Secondary goals of outbreak investigations include identifying the agent, vehicle, source of contamination, and factors contributing to the occurrence of the outbreak. Identifying these factors is critical for identifying new hazards, evaluating the effectiveness of existing control measures, and developing new prevention methods.

Results from 2006 are useful to illustrate the potential benefits and limitations of foodborne outbreak surveillance.[5] A total of 1,270 outbreaks were reported, resulting in 27,634 cases and 11 deaths. An etiology could be confirmed for only 624 (59%) outbreaks. Norovirus accounted for 54% of confirmed outbreaks and 40% of all foodborne outbreaks reported. The next most common cause was Salmonella, which accounted for 18% of outbreaks with a confirmed etiology and 9% of all foodborne outbreaks reported. However, among 11 multistate outbreaks detected, four were attributed to Salmonella and four were attributed to *E. coli* O157:H7. The Salmonella outbreaks were transmitted by tomatoes

(2 outbreaks, 307 cases), peanut butter (715 cases), and fruit salad (41 cases). The *E. coli* O157:H7 outbreaks were transmitted by leafy vegetables (3 outbreaks, 395 cases) and beef (44 cases). An outbreak attributed to *C. botulinum* toxin was transmitted by carrot juice (4 cases).[5] The implications of these outbreaks for abating hazards in the food system are discussed later. Assessment of pathogen-commodity pairs identified the following pathogens as causing the most outbreak-associated illnesses:[5] *Clostridium perfringens* in poultry (902 cases); Salmonella in fruits and nuts (776 cases); norovirus in leafy vegetables (657 cases); Shigatoxin-producing *E. coli* in leafy vegetables (398 cases); Salmonella in vine-stalk vegetables, such as tomatoes (331 cases); and *Vibrio parahaemolyticus* in mollusks (223 cases).

The CDC's electronic Foodborne Outbreak Reporting System collects available information on etiology, number of cases and hospitalizations, and implicated vehicle for each outbreak reported. States also report information on the outbreak setting and factors identified as contributing to the occurrence of the outbreak. Therefore, these data provide a rich framework for analyzing the relative contributions of raw material source contamination, cross-contamination, or ill food workers as the likely causes of foodborne outbreaks in restaurants or other settings. Unfortunately, concerns over the quality and completeness of these data have limited the willingness of the CDC or other investigators to analyze these data and present the findings, contributing to a self-perpetuating problem. Because the data are not being actively used and reported, states see little benefit in making a greater effort to improve the quality or completeness of the data on contributing factors.

Based on the results of surveillance of sporadic infections and outbreaks of foodborne illness, three major foodborne disease problems can be identified:

- Pathogen contamination of raw ingredients
- Scales of production and distribution that turn minor errors into large outbreaks
- Contamination by food handlers of ready-to-eat foods

Abating Hazards in the Food System: Preventing Contamination, Controlling Amplification or Spread, and Reducing or Eliminating the Hazard

Preventing Contamination

As a general principle of environmental microbiology, it is always better to prevent contamination from occurring, rather than to manage the contamination or try to eliminate it during the processing of a food product. However, some contamination of these raw ingredients is inevitable, given the process of growing fresh fruits and vegetables in an open field, or of converting living animals into meat and poultry products in an abattoir. For foods processed with a heat treatment or other control measure capable of killing pathogens, the primary concern is to prevent contamination from environmental sources in the postprocessing environment. In a restaurant setting, persistent concerns are cross-contamination from raw to ready-to-eat foods, or contamination of ready-to-eat foods from infected food workers.

When dealing with issues of chemical contamination, preventing contamination is paramount, since most toxic chemicals are resistant to inactivation by heat or chemical sanitizers. The series of outbreaks and recalls due to contamination of wheat gluten and milk with melamine in China was especially disturbing.[12] These events occurred because of the intentional addition of melamine to these products to increase their apparent protein content. This type of economic fraud has far-reaching implications for food safety and defense that go beyond normal considerations of food safety from a microbiological standpoint. Food processors need to know their suppliers and have some system for ensuring the integrity of their supply chain to prevent future recurrence of this type of problem. Supply chain integrity, supported by a strong regulatory structure that can respond to cross-border contamination issues, is a prerequisite for food safety in a global economy.

Several outbreaks highlight the importance and challenges of preventing microbial contamination. During the summer of 2006, there were 238 cases of *E. coli* O157:H7 infection, including

31 cases of hemolytic uremic syndrome (HUS) and five deaths, caused by spinach that was produced on a single U.S. farm and harvested on a single day.[13] Extensive environmental evaluations were conducted to identify the source of contamination in this outbreak. Multiple strains of *E. coli* O157:H7 were isolated from feces of cattle on a nearby ranch, soil in the cattle pasture, feces of feral swine, surface water, and river sediment samples. The outbreak-associated strain was isolated only from cattle and feral swine feces.[14] Because the environmental evaluations were conducted 2 to 3 months following the contamination event, it is possible that transient contamination of water used in irrigation or processing of the spinach may have caused the outbreak. However, several lines of evidence suggest that the feral swine may have been the critical vector of *E. coli* O157:H7 from the cattle ranch to the spinach field.

First, at the time of the environmental evaluations, the outbreak-associated strains were only isolated from feces of cattle and feral swine. Although the cattle were restricted to their pasture land and had no direct access to the spinach fields, feral swine were observed in both cattle pastures and spinach fields.[14] There was no evidence that cattle manure had been directly applied to the spinach field, or that the spinach field could have been contaminated from flooding events or runoff from the cattle pasture.

In addition, following this outbreak, a company committed $2 million for a novel academic research program to fast-track studies of *E. coli* O157:H7 in leafy greens.[15] Results of these studies established that spinach and leaf lettuce plants do not take up *E. coli* O157:H7 through their roots under field conditions, and that application of bacteria on leaves is unlikely to lead to internalization and distribution of the bacteria throughout the plants' vegetative tissues. In contrast, *E. coli* O157:H7 applied to cut surfaces were readily taken up and transported through vascular tissue. In addition, blades used to harvest and core head lettuce were shown to be capable of contaminating multiple heads of lettuce following the initial contamination of the blade.[16] These findings support the suggestion that feral swine feces in the spinach field may have contaminated harvesting equipment and may have been transferred to the freshly harvested spinach. Uptake of *E. coli* O157:H7 at the cut end of the spinach would have likely led to internalization of the contamination and reduced the effectiveness of subsequent efforts to wash and sanitize spinach leaves.[17]

Table 9-1. The Principles of Hazard Analysis and Critical Control Point (HACCP) Systems

HACCP involves a systematic process of managing food safety threats to a specific food item or process that is based on the following seven tasks:

1. Identify hazards that are reasonably likely to occur in the food item.
2. Determine the critical control points (CCPs) at which the hazard can be prevented, minimized, or eliminated.
3. Establish critical limits to assure the effective control of the hazard at the CCP.
4. Establish monitoring procedures to detect violations of the critical limits.
5. Establish corrective actions to prevent the distribution of potentially contaminated foods and to regain control over the process.
6. Establish verification procedures to ensure that corrective actions are taken when critical limits are exceeded.
7. Establish record-keeping and documentation procedures, to assure that monitoring procedures and corrective actions are being properly performed.

For food items such as fresh produce that will be consumed without an effective treatment measure, preventing contamination at the time of harvest is critical. The investigations of factors associated with outbreaks such as this one have been used to help develop and update good agricultural practices (GAPs) for the production of fresh produce items.[18] While the universal application of GAPs should reduce the frequency and scope of produce-associated outbreaks, GAPs provide only general guidance, are not specific to particular locations or plant varieties, and do not identify critical points at which contamination can be prevented or controlled. While GAPs represent an important safety measure for fruit and vegetable production, they therefore fall short of constituting a hazard analysis and critical control point (HACCP) system (Table 9-1).[19]

Controlling Amplification or Spread

HACCP was originally established as a process control system to protect the safety of foods served to astronauts in space. It has evolved to become a major framework for food safety, although the use of HACCP has been adopted as

a regulatory standard for relatively few food products, as discussed later. One notable success of the application of HACCP to fresh produce has been the implementation of regulations requiring the development of HACCP plans for the production of fruit juices. During the 1990s, a series of *E. coli* O157:H7 and Cryptosporidium outbreaks were associated with consumption of unpasteurized apple juice and cider.[20] Several outbreaks of Salmonella infection were associated with unpasteurized orange juice. In response to these outbreaks, the Food and Drug Administration (FDA) implemented process control measures to regulate the production of fruit juices. These rules were phased in over a 3-year period to accommodate the needs of small producers. In particular, the rules specified the implementation of treatments that could accomplish a 5-log reduction in the presence of *E. coli* O157:H7 as a critical control point in the HACCP plan.[21] Surveillance by the CDC has demonstrated that fewer juice-associated outbreaks have been reported since implementation of these rules. However, outbreaks continue to be associated with processors who are exempt from the rule or who are not in compliance.[20]

Since 1999, the CDC and FDA have recommended that persons at high risk for complications of infection with Salmonella and *E. coli* O157:H7, such as older people, young children, and those with compromised immune systems, not eat raw sprouts. At the same time, the FDA issued *Guidance for Industry for Reducing Microbial Food Safety Hazards for Sprouted Seeds*, which emphasizes the importance of GAPs for the production of seeds and good manufacturing practices for manufacturing, packaging, and holding human food. Two specific interventions are recommended: antimicrobial treatment of seeds to reduce pathogens, and testing of spent irrigation water for the presence of pathogens before products are distributed.[22] This second intervention is recommended because available antimicrobial treatments cannot eliminate all pathogens from seeds and because the conditions required to germinate the seeds and grow the sprouts are also ideal conditions to grow Salmonella and *E. coli* O157:H7. Therefore, any contaminants that survive the treatment would be likely to replicate to dangerous levels prior to distribution.

In contrast to the implementation of the juice HACCP regulation, the FDA's sprout guidance has not been effective at reducing the occurrence of sprout-associated outbreaks. Thirteen Salmonella and three *E. coli* O157:H7 outbreaks that were reported to the CDC from 2000 through 2007 were linked to sprouts.[23] Inadequate disinfection, sampling, and testing procedures, and incorrect interpretation of test results, were identified in some of these investigations.[23] The continuing occurrence of similar outbreaks indicates a need to determine the extent to which the guidance is being implemented. Additional methods to prevent, detect, and eliminate contamination of seeds and sprouts may also be needed.[23]

Another outbreak highlights ongoing problems associated with alfalfa sprouts. On February 24, 2009, the Nebraska Department of Health and Human Services identified six isolates of *Salmonella saintpaul,* with collection dates from February 7 to February 14. Because *Salmonella saintpaul* is not a commonly detected serotype, investigation of this cluster led to the identification of an outbreak involving 228 cases in 13 states. The outbreak investigation ultimately implicated alfalfa sprouts produced at multiple facilities that used seeds from a common grower.

Tracebacks from the initial outbreak investigation identified multiple retail sprout sources that all originated at the same sprouting facility in Omaha, Nebraska (facility A). Facility A produced several types of sprouts that were distributed to locations within a 250-mile radius. The facility reported following FDA guidance for reducing microbial food safety hazards for sprouted seeds.[23] This included treating seeds with 20,000 ppm calcium hypochlorite and culturing seed irrigation water for Salmonella and *Escherichia coli* O157:H7 after 48 hours of germination. The facility reported that it had no positive test results during January and February.[23] However, a sample of facility A alfalfa sprouts, collected from a Nebraska restaurant on February 28, grew *Salmonella typhimurium.* On March 3, the sprouting facility initiated a voluntary recall.[23]

In mid-April, 42 additional cases of *Salmonella saintpaul* with identical PFGE patterns were identified in 10 states. Although cases had

onset of illness after March 15, at least 20 of those affected recalled eating sprouts, which were traced through multiple growing facilities in three states to the same seed source that originally supplied facility A in Nebraska.[23] Results strongly suggested that the seeds were contaminated. The degree to which the various sprout growers involved had appropriately and consistently implemented FDA recommendations is not clear. On May 1, the FDA alerted sprout growers and retailers that a seed supplier was withdrawing voluntarily all lots of alfalfa seeds with a specific three-digit prefix from the market.[23]

Contamination of alfalfa seeds represents a special concern because the raw seed is not a desirable food item, and sprouting the seeds necessarily amplifies the initial contamination. For many raw food items, contamination may be a sporadic occurrence, and the levels of contamination may be low. For example, raw shell eggs emerged as a public health problem during the 1980s due to transovarial transmission of *Salmonella enteritidis* from infected hens. However, only 1 in 1,000 to 10,000 shell eggs was contaminated with *S. enteritidis*, and typically, only a few cells of *S. enteritidis* were deposited in each egg. Egg-associated outbreaks were caused by practices such as pooling multiple eggs together, letting the pooled egg mix sit on a kitchen counter out of refrigeration for several hours, and using the pooled egg mix to make lightly cooked food items. Therefore, the low numbers present in a few eggs were amplified sufficiently to provide an infectious dose to many patrons.[24] The same patterns of amplification have been demonstrated in outbreaks of Salmonella associated with tomatoes and in a series of Shigella outbreaks traced to contaminated parsley imported from Mexico, in which the parsley was chopped, held in a large bowl in the kitchen, and sprinkled as a garnish on dishes ready to be served.[25] In many of these restaurants, food workers and servers became infected as a result of handling and eating contaminated foods. These ill food workers served as additional sources for amplifying transmission during the outbreaks. Therefore, maintaining temperature control and preventing cross-contamination can effectively reduce the risk of an outbreak associated with contaminated food items or ingredients.

The role of infected food workers in transmitting foodborne illness in restaurants is most notable in outbreaks due to norovirus.[5] The CDC's environmental health specialists network (EHS-Net) conducted systematic environmental evaluations in 22 restaurants in which outbreaks had occurred and 347 restaurants in which outbreaks had not occurred during the 2002–2003 period.[26] Norovirus accounted for 42% of the outbreaks studied, and infected food workers and bare-hand contact with food were commonly identified contributing factors. Neither the presence of a certified kitchen manager nor the presence of policies regarding employee health significantly affected the identification of an infected person or carrier as a contributing factor. These findings suggested that a lack of effective monitoring of employee illness or a lack of commitment to enforcing policies regarding ill food workers was an antecedent condition that fostered the transmission of norovirus in restaurants.[26] These same antecedent conditions may also contribute to the role of infected food workers in transmitting Salmonella in restaurants.[27] Whether they are actively contributing to transmission to restaurant patrons or merely serving as indicators of transmission within the restaurant, illnesses among food workers are important health events that should be actively monitored by restaurant managers.

Reducing or Eliminating the Hazard

The final critical control point to abating food safety hazards is to reduce or eliminate the hazard. Cooking is an effective way to destroy foodborne pathogens such as *E. coli* O157:H7 and Salmonella that are associated with raw meat and poultry products. Cooking times and temperatures required to ensure the microbial safety of these foods are well characterized, and have been widely publicized and incorporated into food safety regulations. For example, poultry should be cooked to an internal temperature of 165°F to kill Campylobacter and Salmonella, while a hamburger should be cooked to 160°F to ensure destruction of *E. coli* O157:H7. Unfortunately, most households do not have thermometers that are suitable for measuring the internal temperature of a hamburger.[28]

Pasteurization is another highly successful public health intervention that has virtually

eliminated transmission of foodborne pathogens through milk.[29] The success of pasteurization is demonstrated by the occurrence of outbreaks among persons who seek out raw milk. For example, in 2006, 10 outbreaks reported to the CDC involving 137 cases were attributed to unpasteurized (raw) milk. Pathogens associated with these outbreaks included Campylobacter (six outbreaks), *E. coli* O157:H7 (two outbreaks), Salmonella (one outbreak), and Listeria (one outbreak), resulting in 11 hospitalizations and 1 death.[5] Because these agents can be found on dairy farms and may contaminate raw milk, the absence of outbreaks associated with pasteurized milk confirms the effectiveness of the intervention. The success of milk pasteurization has led to the development of pasteurization conditions that have been validated to achieve a five-log reduction of *Salmonella enteritidis* in shell eggs, without coagulating egg proteins and destroying the physical characteristics or culinary uses of the raw egg.[30]

The success of pasteurization has also provided a model for the use of irradiation as a public health intervention to reduce food safety hazards associated with contamination of raw ingredients.[31] The FDA approved the use of irradiation to control microbes in spices and dried herbs in 1983.[32] Following nationwide concern regarding the emergence of *E. coli* O157:H7 in ground beef during the 1990s, the FDA approved in 1997 the use of irradiation to control pathogenic bacteria in red meat. Two years later, the U.S. Department of Agriculture (USDA) amended its regulations to allow irradiation of refrigerated and frozen uncooked meat, and commercially irradiated ground beef became available in 2000.[32] In 2008, the FDA approved the use of irradiation to control pathogens in fresh spinach and head lettuce.

Although low-dose irradiation has been shown to be effective at reducing levels of pathogen contamination in these products without materially affecting the nutritional content or sensory appeal of these products, it has not yet gained widespread acceptance in the marketplace.[33,34] One issue that has inhibited consumer response is the requirement for these products to carry a logo called the radura in addition to the phrase "Treated with radiation" or "Treated by irradiation." Consumers continue to shun the technology despite consistent reassurances from public health professionals that there is no risk associated with the consumption of food that has received low-dose irradiation, and that these food products are inherently safer microbiologically.[35] The USDA is considering an alternative approach that would allow the use of low-dose irradiation of beef carcasses to kill pathogenic bacteria on the surface.[36] This approach would offer the benefits of eliminating the pathogen in the early stages of processing, thereby reducing the potential for contamination of finished raw meat products, such as ground beef. It would also avoid the labeling problem posed by irradiation of finished products, since it would be considered a "processing aid" rather than a "food additive."[36]

REGULATORY POLICIES AND EDUCATION: PROHIBITING BAD PRACTICES AND INSTITUTIONALIZING GOOD PRACTICES

Regulation of food production historically developed as a tool to prohibit bad practices that jeopardized the safety of food. Both the Food and Drugs Act and the Meat Inspection Act were passed in 1906 in response to unsanitary conditions in meatpacking plants and the use of poisonous preservatives and dyes in foods.[37] These first federal food safety laws—which are still in force in the United States—established the jurisdictions of the USDA for meat and poultry products and the FDA for the rest of the food system. The occurrence of high-profile, multistate foodborne disease outbreaks since 2000 and the lack of progress demonstrated by FoodNet toward meeting *Healthy People 2010* targets for reduction of foodborne pathogens have driven Congress to act to ensure that "Federal food safety standard setting, inspection, enforcement, and research efforts should be based on the best available science and public health considerations, and food safety resources should be systematically deployed in ways that most effectively prevent food-borne illness."[38]

The development of HACCP and its regulatory use in the production of meat, poultry, and juice marked a shift away from a strict regulatory structure based on specific rules and enforcement

to one in which the food industry was charged with identifying problems and developing solutions, under the scrutiny of regulatory oversight. This shift represents an important distinction, which is needed to deal with the growing complexity of food production and distribution systems that are not easily governed by a simple and consistent set of specific rules.

In the restaurant setting, this shift has been accompanied by inspections systems that have moved away from basic observations of the cleanliness of floors, walls, and ceilings to risk-based inspections that focus on managers demonstrating knowledge of food safety.[39] Promoting "active managerial control" in restaurant operations and using inspections as opportunities to consult with managers to help them solve food safety problems have been shown to improve restaurant operations.[40] However, institutionalizing good practices still requires regulatory oversight with strong enforcement capabilities.

Although most food producers strive to produce safe and wholesome food in a profitable manner, there are some who are willing to jeopardize safety in order to make money. The most egregious example of this irresponsibility caused an outbreak involving at least 529 cases of Salmonella in 43 states and Canada with at least 116 hospitalizations and 8 deaths from September 2008 to January 2009.[41] Following the recognition of unusual multistate *Salmonella typhimurium* clusters by PulseNet, investigations by state and local health departments led to the identification of peanut butter and peanut-containing products as the source of the outbreak. The outbreak strains were confirmed in unopened peanut butter containers produced at a single facility in Georgia. During investigations of this facility, it was disclosed that the company had been testing its peanut butter for Salmonella so that it could provide a "Salmonella-free" certificate of analysis to its customers. In fact, several samples from the company during the outbreak period were positive. However, when confronted with a positive sample, the company retested the product. If the follow-up sample was negative, the product was shipped with the "Salmonella-free" certificate of analysis. Neither the company nor the laboratories that performed these tests for the company were required to report the results to regulatory authorities. As a result, an ongoing problem within the plant was ignored until the size of the outbreak finally allowed investigators to identify its source.

Education of managers and food workers is critical to food safety. Education of consumers is also important to help them reduce their risk for foodborne illness by following safe food-handling and food-preparation recommendations and by avoiding consumption of potentially hazardous foods. In particular, the importance of hand washing should be emphasized in general education programs, beginning in preschool. Because the food industry is one of the major sources of employment in the economy, all consumer education activities should help to prepare food industry workers for safe food-handling practices at work. (Detailed information on food safety practices is available at: http://www.foodsafety.gov and http://www.fightbac.org.)

CONCLUSION

Although methods to abate specific hazards, such as *E. coli* O157:H7 contamination of ground beef, can be developed solely based on knowledge of the agent and the characteristics of the food system, public health surveillance is needed to monitor the effectiveness of control measures on a population basis. Monitoring systems at a plant level may not be sufficiently sensitive to prevent the release of large quantities of contaminated products. People represent the ultimate bioassay for this type of contamination, and epidemiology is the tool needed to detect it.

The unique ability of public health surveillance to identify new hazards and provide population-based evaluation of the effectiveness of control measures make it a prerequisite for effective food control programs and a primary tool for food control research. Improving public health surveillance for foodborne diseases will require considerable investments at multiple levels. However, such investments are critical to the improvement of our food safety systems.

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10

Hazardous Waste

Denny Dobbin, Rodney D. Turpin, Ken Silver, and Michelle Watters

People have been generating waste since the beginning of recorded history. Prehistoric waste was largely related to food, including hunting and gathering: piles of discarded shells and animal bones, broken pottery, and primitive tools. Only since the Industrial Revolution in the nineteenth century has waste become a significant societal problem—dumped onto grounds, released into water, and dispersed into the air. Unmanaged garbage has supported proliferation of pathogen-transmitting vectors, such as rodents and insects.

Waste has increasingly become recognized for its impacts on public health, the environment, and land use. In the twentieth century, advances in chemical technology, especially synthesis of organic chemicals, has exponentially increased the volume of toxic material produced and resultant waste generated. And nuclear power generation has led to much radioactive and highly toxic waste that will persist for many years and is difficult to manage.

As we produce, use, and discard products, we may generate waste at each stage—"cradle to grave." We process raw materials, creating waste. We produce and package goods, creating waste. We consume or otherwise use products, creating waste. And we discard products at the end of their useful life, creating waste. All this waste needs to be treated and disposed.

Hazardous waste can adversely affect workers and community residents. Workers are potentially at risk of harm at each of the cradle-to-grave stages. They are engaged in production, where hazardous waste is generated. They are engaged in treatment, storage, and disposal of waste. They are engaged in remediation of uncontrolled hazardous waste sites. Additionally, they respond to emergencies, including spills of hazardous materials. Emergency responders may be at the greatest risk because of the urgent and chaotic nature of their work.

Community exposure to hazardous waste disproportionately affects the poor and minorities. They have historically been less able to control the location of waste sites; they are often exposed to multiple environmental stressors and suffer a variety of adverse impacts, ranging from chronic disease to loss of property value. Hazardous exposures faced by community residents tend to be at lower concentrations, but of larger duration, than those of workers.

DEFINITION OF HAZARDOUS WASTE

Hazardous waste is defined as discarded solid or liquid material that may, directly or indirectly,

cause adverse health effects, unless properly treated, stored, or disposed in a manner that meets specific governmental regulatory definitions. For example, *radioactive waste*, comprised of discarded materials and products that emit harmful radiation, can include spent nuclear fuel rods, high-level radioactive material left over from producing nuclear weapons, mill tailings (radioactive sands left over after uranium metal is separated from its ore), and other low-level radioactive waste Unserviceable material remaining from any process of manufacture or extraction may include unused raw materials, useless by-products, or products so damaged as to be useless or unable to be sold.

Industrial waste includes manufacturing waste, waste from mining and other mineral extraction, coal combustion, and gas and oil production. *Municipal solid waste* includes household garbage, food waste, and trash from public and commercial buildings in communities. *Medical waste* includes waste from clinics, hospitals, and biomedical research laboratories.

The Environmental Protection Agency (EPA) estimates that, by volume, 94% of waste is industrial waste; 5%, hazardous waste; and 1%, municipal solid waste. Radioactive and medical waste represent, by volume, less than 0.1% of total waste. Typical per-capita daily generation of waste in the United States includes about 4 pounds of municipal waste, about 10 pounds of hazardous waste, as much as 300 pounds of industrial waste, and as little as 1 ounce of medical waste.

Each year in the United States, as much as 700 million tons of legally defined waste is produced by as many as 200,000 generators. Chemical manufacturers generate approximately 80% of the total. More than 90% of hazardous waste is discharged as wastewater from industrial production streams. The remaining 10% includes inorganic solids, including contaminated soil and metals; organic solvents in liquid form; and sludge and other residues from water-pollution and air-pollution control systems.

All chemical wastes are toxic, but only those that are specifically designated as toxic by regulation are considered hazardous legally. In regulatory practice, hazardous waste has a precise legal meaning in statutes enacted to control threats to the environment and human health. Defining or listing a material as hazardous waste sets in motion a series of controls and actions to contain the material. However, just because a chemical is *defined* as "not hazardous" does not mean that it *is* not hazardous. Lobbying by special-interest trade associations has led to the exclusion of some chemicals from hazard regulation. Economic hardship and market conditions are often cited as reasons for excluding some chemicals from hazard regulation.

A waste may be classified as "hazardous" if it meets one of the following four characteristics:

- *Ignitability*: The relative likelihood that a chemical will burst into flame (has a flashpoint at or less than 140°F)
- *Corrosivity*: The potential of strong acids (with a pH of 2 or less) and strong bases (with a pH of 12.5 or more) to eat through steel or chemically burn living organisms
- *Reactivity*: The potential for a chemical waste to explode or emit toxic gases
- *Toxicity*: The capability of a chemical to poison living organisms

ADVERSE HEALTH EFFECTS

When evaluating adverse health effects of industrial chemicals discharged or dumped as waste into the environment, extrapolation from occupational data may be helpful. However, standard assumptions and methods that apply to workplaces must be adapted. In the community, as compared with this workplace, people are generally exposed to much lower levels of chemicals and to more complex mixtures of chemicals. Occupational exposure standards for chemicals, designed primarily to protect healthy, working men for 8-hour work shifts, are not sufficient to protect community residents exposed to chemicals—including children, pregnant women (and their fetuses), older people, and people homebound with chronic illnesses or injuries (Fig. 10-1).

In environmental epidemiology, nondifferential misclassification of exposure can obscure exposure–disease relationships. Patterns of exposure to chemicals in the environment can be difficult to characterize in time and space, due to *(a)* variations in daily activities of people,

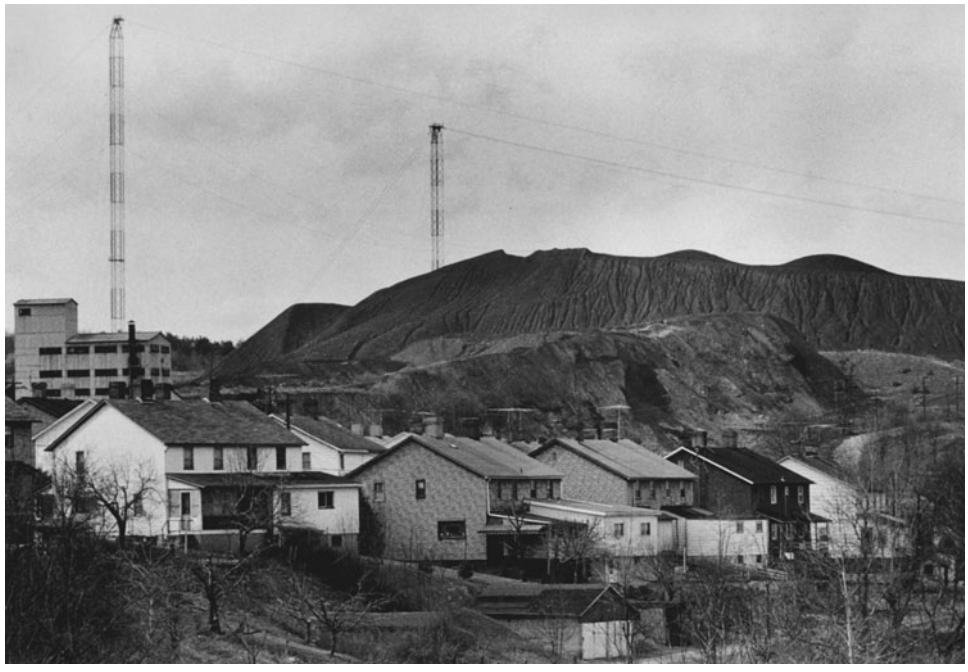

Figure 10-1. "Company town" in Pennsylvania with coal slag heaps. (Photograph by Earl Dotter.)

and *(b)* episodic and seasonal fluctuations in highly complex physical processes, such as flow of groundwater and change in atmospheric conditions.

Compared to industrywide studies that may enroll thousands of workers, epidemiologic studies of people living around hazardous waste sites tend to be based on neighborhood-sized populations, making it difficult to obtain findings that are statistically significant. Government agencies and clean-up consultants often construct computerized risk assessment models that are based on a few exposure measurements. Clean-up priorities are strongly influenced by predictions based on these models, typically in the form of a risk of cancer in the range of 1 in 10,000 (10^{-4}) to 1 in 1,000,000 (10^{-6}). Seldom do epidemiologic studies or personal health data inform clean-up decisions.

Investigations of possible health effects from hazardous waste sites are performed in the most public of settings. Residents of a community often want scientists to confirm that their health effects are associated with exposure to hazardous waste.[1] Ideally, cluster investigations, which may be necessary initially, lead to communication among scientists and community residents on the desirability and feasibility of population-based epidemiologic studies. Early stages of this communication can be marked by conflict between community residents with real-life tragedies and scientists with cool rationality. To respond effectively to communities, public health professionals need to be able to educate the public and health care providers, actively listen, and respectfully support the detective work of community residents (popular, or participatory, epidemiology).[2,3] Health officials accustomed to doing "just science" often quickly discover they must share power with citizens' groups.[4]

The term "hazardous waste" is a social construct: Its meaning changes with societal perceptions and concerns. Originally, it denoted industrial liquid chemicals leaking from corroded barrels at disposal sites that migrated into the drinking water, backyards, basements, and sumps of nearby residences. Love Canal, a community built on a hazardous waste site in upstate New York, was the site of an historic event in the 1970s, when residents noted a wide range of adverse health effects that seemed to be related

to the site. Over time, however, it was recognized that Love Canal was not that unusual; any garbage dump that was active before about 1985 might, in fact, be a hazardous waste site. One-third of the hazardous waste sites on the current federal Superfund list were once "sanitary" landfills.

The term "hazardous waste" also applies to problems as disparate as *(a)* one-time environmental releases of chemicals, such as by spills, leaks, fires, and explosions, and *(b)* long-term problems, such as the presence of radioactive and chemical wastes from the Cold War era at facilities of the U.S. Department of Energy. The wide variety of contaminants, settings, and populations available for study has challenged scientists, required innovation, and generated a social movement that has facilitated cooperative projects among communities and public health scientists.

With the enactment of federal community right-to-know legislation in 1986, which required industrial facilities emitting toxic substances to file public reports, the term "hazardous waste" expanded to include air and water pathways of factory pollution. Increasingly, stringent disposal regulations spurred the expansion of a "treatment, storage, and disposal" industry for chemical wastes. This industry generally took the path of least resistance and expanded into minority communities—in both urban and rural areas. And the environmental justice movement was born.[5]

To overcome the limited statistical power of neighborhood-sized populations, environmental epidemiologists seek ways to increase the size of study groups and/or the numbers of countable events. Registries of adverse birth outcomes have revealed associations between *(a)* proximity to hazardous waste sites, and *(b)* low birthweight and certain congenital anomalies, including neural tube and cardiac defects.[6–10] A crude exposure surrogate of proximity to hazardous waste sites enables epidemiologists to include geographic areas large enough to capture many rare outcomes. Alternatively, many countable events can be identified by studying symptom clusters in neighborhood-sized populations.[11,12] Recall bias and confounding are major challenges in surveys of the prevalence of symptoms among those residing close to these sites. However, some increases in the prevalence of symptoms among exposed residents persist after controlling for recall bias and confounding.[13] Innovations in geographic methods have allowed for exploratory studies of possible associations between environmental exposures and various health outcomes. For example, use of geographic information systems (GISs) has furnished strong evidence of the disproportionate presence of hazardous waste generators in minority communities.[14]

Biological markers show promise as refined measures of exposures to synthetic organic chemicals. For example, in a longitudinal study of a Native American community, the impact of public health advisories recommending pregnant women to reduce their fish intake was measured by a decline in the concentration of PCBs in breast milk.[15] However, use of biological markers as precursors of disease still presents major interpretative challenges. Application of a battery of immune markers may reveal a *statistically* significant association with an exposure, but its *clinical* significance may remain unknown.[16] Among genetic markers, chromosomal abnormalities are generally recognized as steps in—or very near to—causal pathways to cancer and certain congenital anomalies; however, the clinical significance of sister chromatid exchanges and point mutations in specific genes is not well understood. Standardized test batteries for neurobehavioral and reproductive functions, which are used in investigations of hazardous waste sites, rest on strong *clinical* foundations. But when these test batteries are used in *field* investigations, they may be vulnerable to recall bias, confounding, and other problems.[17,18] Psychosocial influences on recall bias of people living near a known hazardous waste site have been poorly quantified.[19]

HAZARDOUS WASTE MANAGEMENT

Hazardous waste management includes treatment, storage, and disposal. It may also include reclamation and incineration—depending on technology, cost, regulation, and physical and chemical properties. Treatment, such as diluting or neutralizing a strong acid or base, can alter the chemical and physical composition of waste,

making it less likely to cause harm. Treatment also includes filtering, solidifying, or evaporating waste. Waste treatment is often done in surface impoundments, such as diked lagoons or ponds, where hazardous waste is temporarily contained. These sites, which are open surface facilities that can hold liquid or partially solidified hazardous waste, vary widely in size. To prevent leakage, barriers of clay or other impermeable material are used to line these holding areas, and groundwater is monitored for possible contamination. Hazardous waste may also be stored before treatment or disposal. For example, a treatment operator may hold a volume of waste targeted for reclamation until markets are favorable to sell the reclaimed product.

Disposal is defined as burial of hazardous waste. Land disposal, which is regulated, includes injection into deep wells, landfilling, and land farming. Injection used for disposing about 10% of hazardous waste requires wells with depths greater than drinking-water aquifers. Abandoned oil and gas wells in Louisiana and Texas are often used for this purpose. Landfills are holes below ground level where hazardous waste may be stored permanently. Landfills must be lined with impermeable material, such as clay. Liquids that leach out must be collected and treated. Groundwater must be periodically tested by monitoring wells surrounding the site. Some hazardous wastes are banned from landfills, and generators must treat certain wastes to lessen their toxicity before they are sent to landfills. In land farming, which relies on bacterial decomposition of hazardous waste, waste from petroleum refineries or elsewhere, is sprayed onto land that is then tilled to mix waste, soil, and oxygen from the air with nutrients and bacteria. The resultant mixture enhances breakdown of waste into safer substances.

Incineration (thermal decomposition of material) is a method used for hazardous waste comprised of organic compounds that can be broken down to simpler chemical components in kilns containing a flame at 1600° F or higher. While waste volume is greatly reduced, resultant emissions contaminate the air, and ashes may contain high concentrations of toxic heavy metals, such as mercury. Since incineration is relatively expensive, less than 1% of hazardous waste in the United States is treated in this manner. Incinerators come in a variety of forms that are appropriate to the waste being treated. For example, rotary kilns are used for solids; injection incinerators, for liquids; and specially designed furnaces, for explosives. Properly operating incineration—with accurately controlled temperature, turbulence, and oxygen concentrations—breaks down organic waste into carbon dioxide, water, and ash. If combustion is incomplete, carbon monoxide may form. Measurement of carbon monoxide in flue gas is used to determine the effectiveness of incinerator performance. Although in reduced volume, inorganic hazardous waste may still be hazardous after incineration.

Hazardous waste may be reclaimed as a commercially useful product. Heavy metals, such as lead, can be reclaimed or recycled from discarded lead-acid batteries; silver can be reclaimed from certain types of photographic processes; and spent degreasing solvents can be cleaned and reclaimed. *Pollution prevention* and *toxic use reduction* rely on rehabilitation of hazardous waste that would otherwise be discarded. Some hazardous wastes can be reclaimed in other than their original forms. One popular means of reclamation is use of combustible hazardous waste as fuel in kilns to produce cement. Further attempts at waste minimization, cradle-to-grave stewardship, recycling, and reclamation can reduce hazardous waste volumes and increase efficiency of production.

The Resource Conservation and Recovery Act (RCRA) of 1976, administered by the EPA, is the primary federal legislation for managing solid waste, including hazardous waste. RCRA is structured around controlling hazardous waste from cradle to grave, including generation, treatment, storage, and disposal. RCRA covers waste from mines, municipalities, and oil and gas production, manufacturing, and coal production facilities. (See Chapter 30.)

REMEDIATING ABANDONED AND ILLEGAL HAZARDOUS WASTE SITES

Public awareness of hazardous waste was heightened in the second half of the twentieth century with reports of abandoned and uncontrolled hazardous waste sites and spilled, or illegally

dumped, hazardous waste that posed a threat to public health or the environment. Too often, extensive sites were abandoned, with no one to hold responsible for cleaning them up. A series of legislative acts to address the problem became known as the "Superfund Law." To support remediation of abandoned sites, the U.S. federal government established "Superfund" from taxes on waste generators, with the principle that the polluter should pay. Superfund began with enactment of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) in 1980. It was updated and improved by the Superfund Amendments and Reauthorization Act (SARA) in 1986. Administered by the EPA, Superfund established *(a)* liability, in the event of releases or spills for generators, transporters, and managers of hazardous waste; and *(b)* a nearly $9 billion fund, which has been used for remediation of hazardous waste sites when those responsible for them cannot be identified or lack resources to conduct clean-up. (Superfund excludes petroleum products and radioactive material waste.) (See Box 10-1 and Chapter 30.)

Superfund clean-up is based on a 10-step approach:

1. *Site discovery*: Sites that require remediation are identified.
2. *Preliminary assessment*: Information on a discovered site is assessed to determine Superfund eligibility.
3. *Site inspection*: Preliminary data and samples are collected to determine the degree of hazard that the site presents to public health and whether emergency action is required.

Box 10-1. Asbestos in Libby, Montana

In the early 1920s, a vermiculite mine was opened in Libby, a small community in northwest Montana. The mine was eventually purchased by W. R. Grace & Company. During its 70 years of operation, the mine produced millions of tons of vermiculite. The vermiculite ore was processed for many commercial and consumer applications, including insulation, garden products, and building material. Dust from the processing facility settled in homes. Children played on waste piles created from processing the ore. And community residents used waste ore and contaminated vermiculite in their gardens, on their driveways, and elsewhere at their homes. Until the mine closed in 1990, Libby vermiculite ore was shipped to over 240 locations throughout the United States for processing and packaging.

While most vermiculite products pose no health problems, the raw vermiculite ore mined at Libby contained up to 26% naturally occurring asbestos. Inhalational exposure to asbestos is associated with several serious health problems, including asbestosis, pleural plaques, lung cancer, and mesothelioma.

In 1999, the Agency for Toxic Substances and Disease Registry (ATSDR) was asked to evaluate the health concerns related to asbestos exposure in Libby. Using death certificate data for the 20-year period from 1979 to 1998, ATSDR investigators found that deaths from asbestosis were 65 to 80 times higher in Libby than for the rest of the United States. Lung cancer was 20 to 30 times higher than expected, and mesothelioma mortality was also elevated.

Over 7,300 people who had lived or worked in Libby for at least 6 months before 1991 were examined. Of those tested, 18% had pleural abnormalities consistent with asbestos exposure. The risk of having a lung abnormality was increased in former mine or mill workers, smokers, and those who had played on the vermiculite piles.

The adverse health effects in workers and community residents in Libby suggested that workers and community residents at other W. R. Grace & Company sites that processed or packaged asbestos-contaminated vermiculite from Libby were also at risk for adverse health effects. In partnership with state health agencies, ATSDR conducted health consultations at 28 selected sites across the United States. It concluded that employees, their families, and people living near one of these 28 sites may have been exposed to amphibole asbestos from vermiculite mined in Libby, Montana, between the 1920s and early 1990s.

There is concern about the use of the vermiculite products, especially attic insulation products, sold under the brand name Zonolite. Many homes in the United States, especially those built before the early 1970s, may contain vermiculite-building products contaminated with asbestos. While the products do not normally present a health hazard, disturbing them may release fibers that may become airborne and subsequently inhaled. Additional information concerning Zonolite/vermiculite insulation can be found at: http://www.epa.gov/asbestos.

In 2009, the Environmental Protection Agency (EPA) determined that a public health emergency existed at the site. By then, hundreds of cases of asbestos-related disease had been documented in this small community. This was the first time the EPA had determined under CERCLA that conditions at a site constituted a public health emergency. This determination recognized the serious health impact from the contamination at Libby and underscored the need for further medical care and public health measures for area residents who had been, or might still be, exposed to asbestos.

4. *Hazard ranking system*: Site data are accumulated and used for ranking of the site to determine its inclusion on the National Priorities List (NPL).
5. *NPL*: Since Superfund resources are to be directed toward remediating the sites with the greatest public health hazard, the EPA maintains a list, ranked by the degree of threat to human health and the environment. Sites on the NPL list are eligible for clean-up funding.
6. *Remedial investigations/feasibility studies*: Following listing of a site on the NPL, in-depth analyses of the site are conducted to *(a)* establish with greater detail and specificity the nature and extent of its health and safety risks, and *(b)* develop recommendations for clean-up.
7. *Remedy selection*: After analysis of recommended alternatives, a preferred remedial action is proposed and made available for public comment in a "record of decision" document. After considering public comment, officials choose a specific remedy.
8. *Remedial design*: After the remedy is chosen, an engineering phase begins to plan and design the remediation.
9. *Remedial action*: The remedy is implemented in the construction phase.
10. *Project close-out*: After the remediation is complete, the project is ended and the site removed from the NPL.

In fiscal year 2008, the EPA reported to have conducted or overseen 691 ongoing clean-up construction projects under the Superfund Remedial and Federal Facility Response Program. As of May 26, 2009, there were 1,264 hazardous waste sites on the National Priority List; 332 sites had been delisted, and new sites have been proposed to be added. An updated list of NPL sites may be found at: http://www.epa.gov/superfund/sites/npl/npl.htm.

In 1998, the EPA developed a concept of "brownfields" clean-up. A "brownfields" site is defined as "real property, the expansion, redevelopment, or reuse of which may be complicated by the presence or potential presence of a hazardous substance, pollutant, or contaminant." Decision makers involved with preparing brownfields sites for productive reuse often require technical and legal assistance to fully understand the complexities of investigating and cleaning up contaminated sites. Additional information about "brownfields" programs may be found at: http://www.brownfieldstsc.org/.

THE ROLE OF THE U.S. PUBLIC HEALTH SERVICE AND OTHER FEDERAL GOVERNMENT ENTITIES

Congress recognized the significance of the public health impact of unregulated hazardous waste and releases as it enacted legislation. Superfund has helped address public health issues by establishing appropriate federal programs to be responsive in areas where there was a need. For example, a basic research program was established at NIEHS to answer questions about preventing health effects related to hazardous waste. Congress recognized the need to protect workers who would be involved in hazardous waste operations and emergency response and directed the Occupational Safety and Health Administration (OSHA) to promulgate protective workplace standards and occupational safety and health training (Fig. 10-2). As part of worker protection, Congress also provided for model training programs to be supported through grants administered by NIEHS.

Congress also recognized the need to address the health concerns of communities and, as a follow-up to CERCLA, established the Agency for Toxic Substances and Disease Registry (ATSDR) in 1980. ATSDR, consolidated administratively with the National Center for Environmental Health (NCEH) at the Centers for Disease Control and Prevention (CDC), has a multidisciplinary staff of epidemiologists, toxicologists, physicians, public health educators, and others. Ten regional offices of the EPA support Superfund and emergency-response activities.

CERCLA was designed to address past hazardous waste disposal activities, even if disposal, or the substance, was not previously considered hazardous. The EPA was mandated to investigate sites, develop clean-up plans, and negotiate payment by responsible parties. ATSDR was designed to work with the EPA by assessing the adverse public health impacts of these sites.

Figure 10-2. Hazardous waste workers in personal protective equipment prepare for training session. (Photograph by Earl Dotter.)

With the Association of Occupational and Environmental Clinics (AOEC) and the EPA, ATSDR developed the Pediatric Environmental Health Specialty Unit (PEHSU) program. Eleven PEHSUs across the United States address pediatric environmental health issues, including evaluation and treatment of children with environmental illnesses, training of pediatricians and other health care providers, and research on children's environmental health problems.

Federal Plans for Emergency Response

Unplanned spills and releases of hazardous waste present special problems for public health protection, depending on the toxicity and physical characteristics of the chemicals involved. Congress anticipated these emergency situations as it enacted hazardous waste–related legislation and provided means to address these situations through governmental agencies.

For multimedia accidental chemical releases, oil spills, and other smaller scale events, fire and police personnel are universally recognized as first responders. Specific large-scale events—such as train derailments, chemical plant explosions, aerospace accidents, and natural disasters—may require other professionals, not generally recognized as first responders, to participate with first responders. (See Chapter 37.)

The National Oil and Hazardous Substance Pollution Contingency Plan (NCP) was promulgated in the 1968 Clean Water Act and has been refined since with the passage of various laws, including Superfund, the Clean Water Act, and the Oil Pollution Act. The original NCP reflected the lessons learned from the 37 million-gallon crude oil spill in Torrey Canyon in 1967. (For comparison, in Alaska in 1989, the Exxon Valdez spilled 11 million gallons of crude oil.) The NCP was the first comprehensive system of accident reporting, spill containment, and clean-up requirements. It established the requirements for response headquarters and national and regional reaction teams, which were the precursors to the current National Response Team (NRT) and Regional Response Teams (RRTs). The NCP identifies the responsibilities of up to 16 participating federal agencies during a

specific emergency. The NRT is responsible for the administration and implementation of the NCP, as well as the planning and coordination of all national response activities.

By 1992, the federal government's emergency response activities were in response to events ranging from natural disasters, such as floods and hurricanes, to accidental releases, such as chemical and oil spills. Several federal agencies developed response plans similar to the NCP, including the Federal Radiological Emergency Plan, the Mass Migration Response Plan, and the U.S. Government Interagency Domestic Terrorism Concept of Operations Plan. State and local governments also developed response teams capable of handling local emergencies. In 1992, the Federal Response Plan (FRP) was published to establish the mechanism and structure by which the federal government could mobilize to address consequences of any major disaster or emergency that overwhelmed the capabilities of state and local governments. The federal departments and agencies agreed to do the following:

- Support the FRP concept of operations and carry out their assigned functional responsibilities
- Cooperate with the federal coordinating officer appointed by the president
- Make maximal use of existing authorities to reduce disaster relief costs
- Form partnerships with counterpart state agencies, voluntary organizations, and the private sector to take advantage of all existing resources
- Develop headquarters and regional planning, exercise, and training activities

Twenty-seven federal departments and agencies have agreed to participate in the FRP.

When releases are serious enough to be considered "nationally significant incidents," the National Response Framework (NRF) is activated and works in conjunction with the National Response System and NCP. The NRF is the federal government's comprehensive, all-hazard approach to crisis management and provides a mechanism for coordinating federal assistance to state governments and localities. The development of the NRF was mandated by the Homeland Security Act of 2002 and Homeland Security Presidential Directive 5. The plan was completed in 2005 and revised after Hurricane Katrina, which struck later that year. The National Response Framework integrates the National Contingency Plan and other national-level contingency plans, and it supercedes the Federal Response Plan, the Domestic Terrorism Concept of Operations Plan, the Federal Radiological Emergency Response Plan, and the original National Response Plan, which was replaced by the National Response Framework in 2008.

CONCLUSION

Uncontrolled hazardous waste can cause acute or chronic disease and threaten the environment. Contaminated land can be a barrier to economic revitalization of a community. Since clean-up of contaminated communities costs more than prevention and control of hazardous waste, more emphasis needs to be placed on prevention and control measures. Through regulation and other systematic approaches, governmental agencies can encourage waste managers to reduce waste at its source, ensure that hazardous and solid wastes are managed safely at industrial facilities, recycle waste to conserve materials or energy, manage waste to prevent spills and releases of toxic materials, and clean up contaminated sites. Federal research and monitoring programs can help identify adverse health effects of hazardous waste, and they can help develop better ways to manage streams of hazardous waste and to clean up uncontrolled sites.

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These are the federal regulations for responding to both oil spills and releases of hazardous substances.

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This was the signed agreement among 27 federal departments and non-governmental agencies that provided the mechanism for coordinating delivery of federal assistance and resources to state and local governments during a major disaster or emergency.

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U.S. Environmental Protection Agency. Current NPL updates: new proposed and new final NPL sites. Available at: http://www.epa.gov/superfund/sites/npl/current.htm

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This Web site includes information on Superfund for fiscal year 2008.

U.S. Environmental Protection Agency. National oil and hazardous substances pollution contingency plan overview. Available at: http://www.epa.gov/oem/content/lawsregs/ncpover.htm
This Web site describes the National Contingency Plan (NCP), the U.S. government blueprint for responding to oil spills and releases of hazardous substance.

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This Web site describes the status and milestones for National Priority Sites.

The Brownfields and Land Revitalization Technology Support Center. Available at: http://www.brownfieldstsc.org/
This Web site describes the Brownfields and Land Revitalization Initiative of the EPA.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the Agency for Toxic Substances and Disease Registry.

11

Chemical Hazards

Michael Gochfeld and Robert Laumbach

Chemicals are most broadly classified as being organic (containing one or more carbon atoms) and inorganic (Table 11-1). Chemicals can also be classified according to their structure, source, use, mechanism of action, environmental properties, and target organ. Sometimes, the only information available for a chemical is its class, such as pesticide or solvent. The classifications and examples presented in this chapter are intended to be illustrative, not comprehensive.

Table 11-1. Chemicals Classified by Structure

Organic Chemicals
Aromatics, such as benzene and benzene derivatives (such as toluene and phenols)
Aliphatics, such as alkanes and alkenes
Chlorinated hydrocarbons, such as chlorinated alkanes and alkenes
Polyaromatic hydrocarbons (PAHs)
Chlorinated polyaromatics, such as dioxins, furans, and polychlorinated biphenyls (PCBs)
Amines
Organic acids, ethers, aldehyes, ketones, and alcohols
Inorganic Chemicals
Heavy metals, such as lead and mercury
Light metals, such as beryllium and lithium
Metalloids, such as arsenic and selenium
Acids and bases
Anions and cations, and salts

CLASSIFICATION BY STRUCTURE

Structure–activity relationships (SARs) are often useful in inferring the toxicity of an unfamiliar chemical from the known activity of familiar, or better-studied, chemicals of similar structure. Since only a few of the thousands of chemicals in commercial use have been adequately tested, reliance on SARs may initially provide the only clues to types of toxicity, if any. For example, chlorinated hydrocarbons with simple chain structure (alkanes) tend to share the common property of central nervous system depression, allowing some to be used as general anesthetics (Fig. 11-1). Although their potency varies by the number of carbon atoms, presence or absence of double bonds, and the presence of chlorine, fluorine, or bromine atoms, their general effects on central nervous system depression are similar. Similarly, many heavy metals are toxic to the proximal kidney tubule, and many hallucinogenic compounds share a common active group. SARs can also be predictive of carcinogenicity identified by long-term animal bioassays (Chapter 17). Yet one must be cautious since chemicals with similar structures do not always have similar properties. In some instances, SARs do not apply; for example, benzene causes leukemia, while closely related compounds, such as phenol (hydroxyl-benzene) and toluene (methyl benzene), apparently do not cause leukemia.

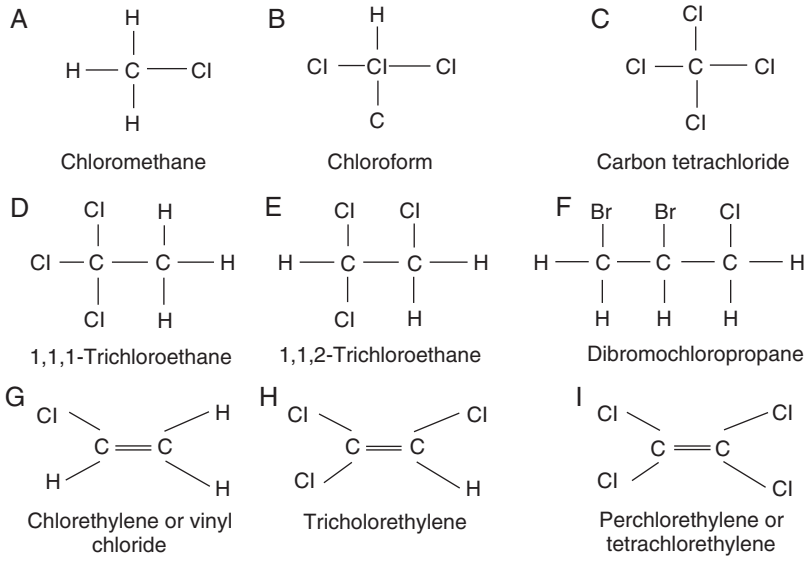


Figure 11-1. Examples of simple chlorinated hydrocarbons. Single carbon compounds are (A) chloromethane; (B) trichloromethane, or chloroform; and (C) tetrachloromethane, or carbon tetrachloride. Two-carbon compounds with a single bond (alkanes) include isomers (D) 1,1,1-trichloroethane and (E) 1,1,2-trichloroethane, as well as (F) dibromochloropropane (DBCP), or 1,2 dibromo,3-chloropropane. The bottom row shows the following chlorinated ethenes, each with a double bond: (G) chloroethene, or vinyl chloride; (H) tricholoroethylene; and (I) tetrachlorethylene, which is better known as perchlorethylene, or "perc."

CLASSIFICATION BY SOURCE

Chemicals can be classified by source (Table 11-2). The word *toxins* refers only to bioactive chemical substances produced naturally by plants and animals. Many plants and animals secrete chemicals designed to protect against being eaten. Monarch caterpillars may incorporate in their tissues alkaloids from the Milkweed leaves they eat, rendering themselves, and subsequently adult butterflies, toxic and unpalatable.

Table 11-2. Chemicals Classified by Source

Natural or Biologic Compounds (Toxins)
Plant
Bacterial
Fungal
Invertebrate
Vertebrate
Synthetic Compounds
Industrial reagent, by-product, or product
Pharmaceutical
Pesticide

Beetles may squirt hot cyanide compounds to deter predators. Plants that have been partially eaten by herbivores may load increased levels of distasteful tannin compounds in newly regenerated leaves. Similarly, many fungi secrete chemicals that inhibit bacterial growth, generating odors that we recognize as "spoilage." Hymenoptera (such as bees and wasps), spiders, and some snakes have developed a variety of neurotoxic and hematotoxic venoms for immobilizing prey. There is great diversity of toxins produced in the terrestrial and marine environment by organisms ranging from fungi and dinoflagellates to fish. The fungal toxin aflatoxin B1, which is produced by fungi that contaminate stored beans, peanuts, and other food items, is reputed to be the most toxic compound, by virtue of its very low LD_{50}. In humans, aflatoxin B1 can cause liver cancer. A wide variety of toxins have been adapted into some familiar pharmaceuticals, such as antibiotics. A compendium provides useful information on plant and animal toxins.[1]

CLASSIFICATION BY USE

Very often, the first aspect of a chemical that a worker reports is its use—as a solvent, as a detergent, or substance used for etching. Therefore, a worker may report having been overcome while "using a solvent." Or homeowners may report "some pesticide spray" having made them ill. Examples of common use classes of chemicals that may have toxic effects are shown in Table 11-3.

Pharmaceutical agents and controlled substances, including alcohol and tobacco, are grouped together because very high concentrations of bioactive agents tend to be often deliberately introduced into the body. Many abused substances that were originally developed as pharmaceuticals, such as amphetamines, barbiturates, and narcotics, have profound toxic effects—quite apart from their addictive properties. By whatever route and whether legal or illicit, these chemicals are used because of their high level of bioactivity. Toxicity may occur from excessive doses. Even when the dosage used is in the therapeutic range, there may be undesired side effects that are manifestations of toxicity. These effects may occur frequently, such as the soporific effects of the antihistamine diphenhydramine, or rarely, such as anaphylaxis due to penicillin.

The most widespread toxic exposures involve the chronic inhalation of tobacco smoke by smokers and bystanders (see Box 7-1 in Chapter 7), and chronic overconsumption of ethanol. Because many pharmaceuticals affect the same enzymes that metabolize occupational chemicals, it is important to evaluate the medication a person may be taking to assess whether there may be dangerous interactions with chemicals at work, in hobbies, or at home.

Table 11-3. Chemicals Classified by Use

Solvents
Fuels
Paints, dyes, and coatings
Glues
Pesticides
Pharmaceutical agents and controlled substances
Detergents and cleansers
Acids and bases

CLASSIFICATION BY MECHANISM OF ACTION

Chemicals can be classified by mechanism of action (Table 11-4). For example, substances that block cellular respiration are categorized as asphyxiants. Methane and other gases are toxicologically inert, but they can function as simple asphyxiants by displacing oxygen from inhaled air, thereby decreasing the oxygen saturation of the blood and reducing its availability to cells. Chemical asphyxiants include carbon monoxide (CO), hydrogen sulfide, and cyanide. CO has a strong affinity for hemoglobin, and the resultant carboxyhemoglobin is unable to transport oxygen from the lungs to cells elsewhere in the body. Much exciting research in modern toxicology focuses on the mechanisms by which a bioactive substance interacts with and alters its target organs to produce its adverse effects. Targets may include organelles, membranes, hormone receptors, cells, and biochemical reactions.

CLASSIFICATION BY ENVIRONMENTAL PROPERTIES

The Environmental Protection Agency (EPA) has identified toxic chemicals that are persistent and bioaccumulative (PBTs) in the environment and the human body (Table 11-5). These are mainly chlorinated hydrocarbon pesticides and related compounds, which break down very

Table 11-4. Chemicals Classified by Mechanism of Action

Metabolic poisons and chemical asphyxiants
Hematotoxins, such as by methemoglobinemia (nitrites) and by hemolysis (arsine)
Binders to macromolecules, such as DNA or protein
Hydrogen ion effects, such as acids and bases
Enzyme inducers
Genotoxins, including mutagens and carcinogens
Cell membrane disrupters
Competitive binders of active sites or receptors
Formers of free radicals and active oxygen
Chemicals causing redox reactions
Chemicals interfering with signal transduction
Chemicals interfering with hormone activity
Sensitizers
Irritants

Table 11-5. Persistent Bioaccumulative and Toxic Chemicals (PBTs)

Aldrin/dieldrin	Mercury
Benzo(a)pyrene	Mirex
Chlordane	Octachlorostyrene
DDT, DDD, and DDE	Polychlorinated biphenyls (PCBs)
Hexachlorobenzene	Dioxins and furans
Alkyl-lead	Toxaphene

Source: http://www.epa.gov/pbt/.

Table 11-6. Chemicals Classified by Target Organ

Neurotoxins
Hematotoxins
Nephrotoxins
Hepatotoxins
Cardiotoxins
Pulmonary toxins
Endocrine disruptors
Dermatotoxins
Reproductive agents and teratogens

slowly under ambient conditions (Fig. 11-2). Some can be degraded by light, others by microorganisms. Most PBTs undergo some bioamplification through the food chain, so that organisms higher on the food chain, such as large predatory fish or birds, have higher tissue concentrations than organisms lower down.

CLASSIFICATION BY TARGET ORGAN

Toxic chemicals can be classified by target organ (Table 11-6). Once absorbed into the bloodstream, chemicals are distributed to most organs, but may have their major effects on a specific organ system. There may be differences between acute and chronic toxicity. For example, benzene is acutely neurotoxic (like many small organic molecules), but it is its chronic toxicity to the bone marrow that results in interference with normal production of red blood cells, white blood cells, and platelets, causing pancytopenia and leukemias. Once absorbed, lead is distributed to many organs, including bone, where it is stored in place of calcium; however, it is the neurotoxic effects of lead on both the developing brain and the mature brain that are the most important toxic effects of lead.

Figure 11-2. Aerial spraying of pesticides in an orchard in California. (Photograph by Earl Dotter.)

PROPERTIES OF CHEMICALS

Chemical Species

A chemical variant of a metal is called a *species*. Different chemical species can have different chemical and toxicological properties. For example, trivalent chromium (Cr-III) and hexavalent chromium (Cr-VI), which differ in oxidation state, also differ in toxicity, carcinogenicity, and ability to pass through cell membranes. They represent different chromium species, yet they are interconvertible, depending on whether they are in an oxidizing or reducing environment. Cr-III is an essential nutrient, while Cr-VI is a lung and gastrointestinal tract carcinogen. Quantifying Cr-III and Cr-VI in environmental media or biological samples is complicated by the potential for redox reactions, which can occur in the environment, during transport to a laboratory, or during analysis. Depending on how a soil sample is collected, transported, and analyzed, Cr-III can be oxidized and appear spuriously as Cr-VI when analyzed; or Cr-VI might be inadvertently reduced to Cr-III and analyzed as such.

The same chemical, such as mercury, may exist in several different chemical species. Slight modifications may have profound effects. Elemental mercury, often referred to as quicksilver, is a dense silvery liquid, with a specific gravity of 13.6. It is one of the few elements that are liquid at ambient temperature and also volatile, giving off an odorless, colorless, but highly toxic vapor that is readily inhaled and absorbed into pulmonary capillaries. Mercury compounds used in industry are often inorganic salts, while many of the biocidal mercurial compounds, such as phenylmercuric acetate, are organic chemicals. The methylmercury produced in aquatic sediments by anaerobic bacteria from elemental or inorganic mercury biomagnifies in the food chain from plankton to small fish to large fish, causing organic mercury poisoning in people who consume large amounts of fish.

In general, organic forms of metals have a different spectrum of toxicity than the inorganic forms of the same metals. Thus, organic mercury and organotin compounds are more highly toxic than the corresponding inorganic forms. Both have been used extensively as biocides, especially in antifouling paints used for ships to inhibit the growth of barnacles. As mercury and tin have leached out of paints into the sea, they have adversely impacted marine organisms and disrupted aquatic ecosystems. However, for some elements, such as arsenic, the inorganic compounds are more highly toxic than the organic ones.

Isomers and Congeners

Two chemical compounds that have the same chemical formula, but differ in structure, are called *isomers*. Figure 11-3 shows two isomers of the common fuel, butane, which as a four-carbon chain can appear as either normal (linear) butane or branched isobutane. *Congeners* have the same basic structure, but different numbers of atoms. For instance, dichlorophenol and trichlorophenol are congeners, while 2,4-dichlorophenol and 2,5-dichlorophenol are isomers. There are 209 different isomers and congeners of polychlorinated biphenyls (PCBs), differing in the number and position of chlorine atoms on the two attached benzene rings. Several of these compounds have four chlorines and therefore are isomers of tetrachlorobiphenyl. The toxicity of isomers and congeners and their behavior in the human body may vary greatly.

Different chlorinated dibenzodioxins vary by orders of magnitude in their toxicity. The most

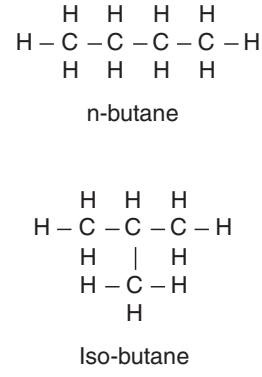


Figure 11-3. n-butane and iso-butane (2-methyl propane) are isomers of butane.

toxic of these is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD, or dioxin), which was a contaminant in the production of the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Each of the dioxin congeners can be assigned a *toxicity potency* (toxic equivalency factor, or TEF) relative to TCDD, which is given a value of 1. Some PCBs are considered dioxin-like in their biological effects and are assigned a TEF, while other PCBs, including the most common isomer (PCB-153), are not.

Polarity and Solubility

Polar compounds are readily soluble in water; *nonpolar compounds* are relatively insoluble in water but dissolve readily in a variety of organic solvents. Some compounds are insoluble in virtually all solvents, while a few dissolve readily in water and organic solvents. The transition from polar to nonpolar is a continuum and is measured by the octanol:water partitioning coefficient. Nonpolar compounds have higher octanol solubility; polar compounds have higher water solubility.

Mixtures

Most toxicologic research is conducted with single chemicals. However, people are generally exposed to multiple chemicals, presenting a challenge for epidemiologists and others attempting to determine the adverse effects of individual chemicals and of the combined exposure to multiple chemicals (Fig. 11-4). Chemicals may interact in the environmental medium, or in the lung, intestinal tract, or in other organs. Interactions may reduce or enhance the toxicity. There are a few examples of true synergy, in which the resulting toxicity is equal to the product of the effects of individual chemicals. The most notable examples of synergy in causing lung cancer are cigarette smoking with radon and cigarette smoking with asbestos.

There is now an abundance of reliable sources of information on toxic chemicals (see Further Reading at end of this chapter). The following section focuses on selected chemicals and groups of chemicals to illustrate some of the main themes in occupational and environmental health and toxicology. (See Chapter 25.)

Figure 11-4. A barefoot worker pours molten iron into molds in a foundry in the Philippines as sunlight penetrates the emission-laden atmosphere containing carbon monoxide, crystalline silica, hydrocarbons, and carbon particles. The foundry was studied as part of an International Labor Organization–sponsored industrial hygiene course for government labor inspectors. (Photograph by Aaron Sussell.)

Inorganic Compounds

CHEMICAL ASPHYXIANTS

Chemical asphyxiants act by blocking the body's ability to use oxygen to support the basic life functions of cells.

Carbon Monoxide

CASE 1

In December, a 50-year-old woman was brought to the emergency department of a small rural hospital after collapsing at work at an onion farm. She reported no previous episodes of syncope or chest pain and had no

significant past medical history other than treatment for mild hypertension. She was doing her ordinary work at the farm's packing shed, preparing onions for shipment, when she suddenly became dizzy and lost consciousness out. Her electrocardiogram (ECG) showed mild ischemic changes, and she was admitted to the intensive care unit for observation.

The next afternoon, two other workers from the same farm were brought to the emergency department complaining of headaches, dizziness, and nausea. Blood samples were drawn for determination of carboxyhemoglobin (COHb) concentration, and both workers had slightly increased levels (about 10%). Interpretation was complicated because more than 30 minutes had elapsed before they reached the hospital from the farm, and it was unclear whether they had been treated with oxygen during that time. The emergency department physician contacted the farm owner, who reported that he had called the gas company to check the propane heaters used in the barn. They had tested the barn with a "gas meter" and found no problem with carbon monoxide (CO) or other gases.

The two workers went back to work the next morning and again became ill. They returned to the emergency department. This time, their COHb levels were elevated (between 14% and 16%). A nurse from a local occupational health program was notified and visited the farm that afternoon. In discussing the situation with the farmer and other workers, she found a number of potential problems. Temperatures in the barn had been very cold. There was little ventilation. Several small propane heaters provided some heat. A propane-powered forklift was used intermittently in the barn. Because of weather conditions, the doors to the barn had been kept closed for the previous several days.

The nurse requested that an industrial hygienist visit the facility to conduct further air sampling. He arrived the next day. Long-term personal samples taken that day showed acceptable CO levels—up to 24 ppm, compared with the Occupational Safety and Health Administration (OSHA) standard of 50 ppm. However, short-term samples showed levels up to 100 ppm at some locations, especially around the forklift. Doors in the facility were kept open during the day that sampling took place. Based on these findings, the farmer obtained a battery-powered forklift and improved ventilation in the facility.

CO is the most common chemical asphyxiant. It is produced by incomplete combustion, such as in automobile exhaust, indoor use of diesel forklifts, charcoal cooking indoors, or malfunctioning heating systems. Fatal CO poisoning has also occurred in outdoor settings where local conditions create the potential for high levels of gas to accumulate, such as near the exhaust of recreational boating craft. CO continues to be a very common cause of death in the workplace and elsewhere. Whereas the oxygen transport of hemoglobin relies on oxygen binding to hemoglobin in the lung and then being released in target tissues, CO binds 240 times more strongly than oxygen to hemoglobin (forming COHb), so that oxygen is not released in tissues. With continued CO exposure, the amount of COHb increases, leaving less and less unreacted hemoglobin to transport oxygen. Symptoms are nonspecific (Table 11-7). Onset is insidious. People become sleepy, without realizing that they are being poisoned. Drivers exposed to CO from faulty exhaust systems may fall asleep while driving. In acute exposure, the first symptom is usually headache, progressing to nausea, weakness, dizziness, and confusion. CO exposure should be considered in patients who collapse at work or report sudden headaches, lightheadedness, dizziness, or nausea. More severe poisoning can lead to unconsciousness and death.

The standard laboratory test for CO exposure is determination of the COHb concentration in the blood; this reveals the proportion of hemoglobin that is bound to CO. Normal levels in nonsmokers range up to 4%, and smokers can

Table 11-7. Symptoms and Signs Associated with Increasing Blood Levels of Carbon Monoxide

Percentage of Carbon Monoxide in Blood	Symptoms and Signs
10–20	Headache and shortness of breath
20–30	Nausea, dizziness, severe headache, and difficulty concentrating
30–40	Lethargy, fainting, visual and auditory impairment, and chest pain
40–50	Fainting, rapid heart rate, and seizures
>50	Coma, convulsions, and death

Source: Sullivan JB Jr, Krieger GR (eds.). Clinical environmental health and toxic exposures (2nd ed.). Philadelphia: Lippincott, Williams & Wilkins, 2001, p. 725.

have levels as high as 8%. Serious medical problems usually do not develop unless levels exceed 20%. However, patients with ischemic heart disease are especially susceptible to the effects of CO. Interpretation of COHb levels is challenging because they return to normal within hours (even faster in patients who have been given oxygen). Therefore, if a patient collapses at work, is given oxygen, and is then brought to the emergency department, the COHb level measured in the emergency department may be well below the peak level the patient reached during the exposure. Hyperbaric oxygen therapy is used for severe CO poisoning, but even with such treatment permanent neurologic damage may occur.

Intermittent or episodic exposures to increased concentrations of CO can increase the risk of cardiovascular disease among groups such as tunnel workers and highway toll collectors. However, such exposures can be difficult to detect. In the case presented here, the original testing by the "gas meter" might have occurred when the ventilation was especially good (such as with a breeze blowing through the barn), or the instruments might have been insensitive to slight CO elevations. Similarly, a worker's exposure from the forklift could vary with time and location in the facility. In this case, sampling with better instrumentation revealed the source of CO.

In chronic CO poisoning, effects may be direct or indirect from relative hypoxia. CO, including from cigarettes, is implicated in accelerating atherogenesis and promoting acute coronary hypoxia. Incomplete recovery from acute CO poisoning, especially after coma, may include residual neurobehavioral and cognitive impairment. Household CO poisoning from faulty heating systems can be averted by alarms. (See Chapter 23.)

Other Chemical Asphyxiants

Asphyxiants usually are grouped into two major categories. Simple or inert asphyxiants, such as propane or hydrogen, act by displacing oxygen in the atmosphere. The most common scenario for this type of asphyxiation is work in a confined space, such as a manhole or a storage tank. OSHA requires special precautions for work in confined spaces, such as warning signs, air testing before entry, and the use of supplied-air respirators.

Chemical or toxic asphyxiants include a number of chemicals that interfere with the transport, delivery, or utilization of oxygen in

(Drawing by Nick Thorkelson.)

the body. In addition to CO, common examples include hydrogen sulfide (H_2S) and hydrogen cyanide (HCN). Although these chemicals are sometimes used in a workplace, more frequently they are produced as a result of some other process, such as combustion or chemical mixing, and the asphyxiation occurs accidentally as a result of that process.

HCN gas, a cellular asphyxiant, is acutely toxic and capable of causing death within seconds by poisoning cellular respiration. Workers with known potential for cyanide exposure should be trained to protect themselves against the known hazard of working with HCN gas under normal and upset operating conditions. However, they may be unaware of the potential of cyanide-containing salts to release HCN on contact with any acid and even with water. Cyanide compounds are used in the plating industry and in the commercial extraction of gold and silver from metal ores. Alkaline cyanide solutions are used to reclaim metals from jewelry. Metals are extracted from the metal cyanide solutions by cathodic plating, the basis for the application of metal cyanide in the plating industry. Cyanide compounds were formerly widely used as rodenticides and in the fumigation of ships. HCN is most commonly produced when acids come into contact with cyanide compounds. The burning of acrylonitrile plastics can also produce significant levels of HCN. Exposure to levels of about 100 ppm for 30 to 60 minutes can be fatal. HCN has a characteristic bitter almond taste, although not all people can identify it. In acute exposures, a person may absorb a lethal dose before the taste can be identified. The primary toxicity of HCN is its poisoning of the cytochrome electron transport system, blocking the oxidative phosphorylation formation of ATP, the energetic basis of cellular function. Initial symptoms include headache and palpitations, progressing to dyspnea and then convulsions. Treatment with sodium nitrite, sodium thiosulfate, and amyl nitrite can be effective, but must be started almost immediately. Blood cyanide levels can be used to monitor the effectiveness of treatment.

Acute H_2S poisoning may occur in a number of workplace settings, including leather tanning, sewage treatment, and oil drilling. H_2S is a common cause of work-related fatalities in oil fields in the southwestern United States, where it occurs naturally as a contaminant of natural gas.

Hydrogen sulfide, a colorless, chemical asphyxiant gas accounts for the rotten-egg odor associated with gas oozing out of marshes. It has been used in chemical analytic laboratories, metallurgy, and chemical synthesis, and as a chemical disinfectant. At intermediate concentrations, H_2S acts as an irritant of the eyes and the respiratory tract. Although its odor is characteristic even at 1 ppm, exposure to a concentration above 150 ppm paralyzes the sense of smell before the odor is identified, so the worker is unaware of the exposure. Acute neurotoxic effects occur at 500 ppm. Delayed pulmonary edema has been reported. Death may occur at 700 ppm. At higher levels, acute respiratory paralysis, or "knockdown," occurs. Serial fatalities may then take place as unprotected co-workers, attempting to rescue the fallen worker, also die.

The outbreak of CO poisoning, which was described in Case 1 earlier, and consideration of other asphyxiants highlight several important principles:

1. Not every toxic exposure is exotic. Such familiar items as a forklift can cause fatal exposures.
2. Occupational medicine principles can be directly applicable to the general environment. For example, many cases of CO poisoning occur in the home and are caused by faulty heaters.
3. Workers, when exposed to an asphyxiant or intoxicant, become less alert and less able to react quickly to hazards. This is a form of synergy, which increases the risk of injuries, further exposures, and other mishaps on and off the job.
4. In an environment with very high gas concentrations, every breath boosts the blood level of the gas, and toxicity can develop remarkably rapidly. Such acute toxicity is common in enclosed spaces, affecting not only the primary victims, but also co-workers who rush to provide assistance.
5. When a worker is found dead or unconscious after an unknown exposure, a blood sample should always be taken. COHb

levels, thiosulfate levels, and other evidence suggesting acute asphyxiation should be obtained routinely.

The most important principle illustrated by asphyxiants is the primacy of prevention. Asphyxiation can almost always be anticipated. The hazards of confined spaces, forklifts, and other sources are well recognized. Once anticipated, exposures can be prevented by some combination of usual measures: *(a)* minimizing the formation of the asphyxiant, *(b)* proper ventilation, *(c)* proper work practices, *(d)* worker training, and *(e)* personal protective equipment.

METALS

Many metals, such as cobalt, copper, chromium, iron, manganese, vanadium, iron, and zinc, have been identified as essential for human growth and physiology. Deficiency of any of these elements can lead to poor growth or abnormal function. Among metalloids (nonmetallic elements that have some of the characteristics of metals), selenium is essential; there is controversy over whether arsenic is essential in some animals, but it is not essential in humans. For some trace elements, such as cobalt, the daily dietary requirement is very low (measured in nanograms); for others (macronutrients), such as iron, milligram amounts are required daily. Essential elements may be toxic in high doses. Metals are often incorporated into biocides, such as lead arsenate insecticides, mancozeb (a manganese and zinc carbamate fungicide), mercurial fungicides, and copper algaecides. Arsenic compounds have been widely used to defoliate cotton prior to harvesting.

Lead

CASE 2

A 29-year-old laborer who worked intermittently for a construction firm that did bridge repair work complained to his family physician of intermittent stomach pains of several weeks' duration. The pain was not associated with meals. Onset had been gradual. He had no associated systemic or gastrointestinal symptoms. He had not experienced any unusual stress at home or at work. He reported drinking one or two cans of beer per day. Physical examination was normal. His physician treated him with antacids and his pain gradually resolved.

Approximately 2 months later, the patient returned complaining of more severe epigastric pain, associated with abdominal cramping, headaches, and fatigue. He recently started working at a new site, where he had used an oxyacetylene torch to remove paint from an old bridge. After consultation with an occupational medicine physician, the family physician obtained a blood lead level (BLL), which was 75 μg/dL. The patient stopped doing paint removal work, and his symptoms gradually improved. Within 4 weeks, his BLL decreased to 35 μg/dL. The contractor provided a ventilation system for use when paint was being removed from bridges. Subsequent quarterly monitoring of the patient's BLL showed a gradual further decline.

Although the use of lead pigment was discontinued in most paints in the United States by the 1970s, older lead-containing paints still cover many interior and exterior surfaces in older buildings and continue to be used on steel structures. This exposure accounts for many cases of childhood lead poisoning. In addition, painters and other workers conducting renovation work on buildings with lead paint can be significantly exposed to lead. Burning or torching of the surface to remove the paint produces a lead fume, which is readily absorbed through the respiratory tract.

Lead is a metal for which there is no known beneficial biological role. Lead poisoning among ship builders was described by Pliny the Elder (79 C.E.). Lead occurs in one oxidation state (+2). It is easy to analyze in biologic and environmental media because few things interfere with its measurement.

Childhood lead poisoning is caused primarily by ingestion and inhalation of paint dust and ingestion of paint chips by infants and toddlers. Lead impairs cognitive development in children even at low doses.[2,3]

Occupational exposures to lead are mainly by inhalation of lead dust and fume, although

ingestion can occur when workers smoke or eat on the job or where hygiene is poor. Lead exposure begins with mining and smelting, then continues into the industries that make lead products, including lead alloys and lead pigments, and those that fabricate products containing lead. A heavy, dense metal, lead is used in X-ray shields and weights, ranging from gram weights on fishing lines to multi-ton weights to stabilize construction cranes. Many lead-using factories have moved from developed to developing countries. Lead acid batteries continue to be manufactured in the United States, but in decreasing numbers.

The combination of improved industrial hygiene and closure of lead factories greatly reduced the number of severe occupational lead poisoning cases in the United States. However, there remains an informal sector of backyard smelters, in which lead plates from vehicle batteries are melted to recover and recycle the lead. This source of urban lead exposure is difficult to detect and regulate. As environmental awareness led to removal of leaded paint from many structures in the 1980s, a new source of lead exposure emerged for workers performing or working near abrasive blasting, burning, welding, and cutting of steel structures, such as bridges that had been coated with anti-rust lead paint. To keep lead from contaminating the environment, lead-removal workers were enclosed in tents, which greatly increased their respiratory exposure to lead fume and dust.

Lead exposure is conveniently measured by analysis of lead in whole blood. During the period of use of leaded gasoline as an anti-knock agent from the 1920s to the 1970s, BLLs in the United States averaged around 15 μg/dL. Since the removal of lead from gasoline, starting in the late 1970s, average BLLs have declined to below 2 μg/dL (Fig. 11-5).

Lead is readily absorbed from the lungs and gastrointestinal tract. In adults, about 10% of ingested lead is absorbed; in children, about 50% is absorbed. Once absorbed, lead, some of it bound to protein, travels through the blood and gains access to all organs, including the brain.

Lead adversely affects many organ systems. Lead inhibits two enzymes: *(a)* delta-aminolevulinic acid dehydratase, which converts delta-aminolevulinic acid to porphobilinogen; and *(b)* ferrochelatase, which inserts iron into the

(Drawing by Nick Thorkelson.)

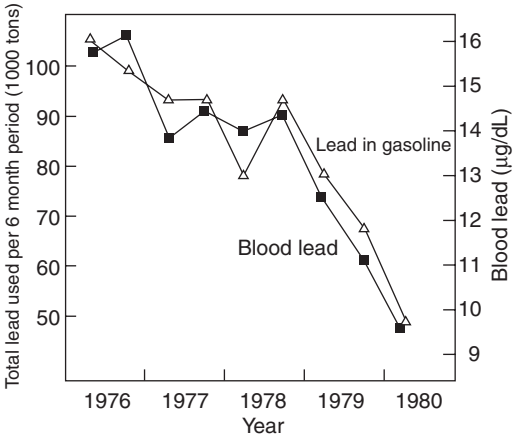


Figure 11-5. This graph tracks the dramatic decline in blood lead levels coincident with the reduction of leaded gasoline production in the United States (1976–1980). The decline reached 2.9 μg/dL by 1990 and has continued so that the mean blood lead level is below 2 μg/dL. (*Source*: U.S. Environmental Protection Agency.)

heme molecule. As a result, lead creates a build-up of protoporphyrin. Clinically apparent anemia may not occur until the BLL is above 50 μg/dL. Elevated blood pressure can occur when the BLL is approximately 20 μg/dL or higher. Peripheral nerve dysfunction starts to occur around 40 μg/dL. Moderate hypertension is associated with elevated BLLs and may be partially related to toxic effects on the proximal renal tubule. Population increases in blood pressure have been associated with increases in lead exposure at very low levels.

The brain is a target organ for lead. Cognitive impairment and sleep disturbances are often reported in adults with BLLs of 50 μg/dL or even lower. Encephalopathy may occur if the BLL reaches 100 μg/dL. Peripheral neuropathy may cause weakness and slowed reflexes. Motor neuron neuropathy predominates in lead poisoning, and "foot drop" is a classical symptom, often first detected by abnormal gait with circumduction of one foot. (See Chapter 19.) Classical lead poisoning, characterized by motor and sensory peripheral neuropathy leading to foot drop, gastrointestinal colic, weakness, and depression, is rarely encountered in developed countries.

BLL is a marker only for recent exposure to lead. Lead accumulates in organs, primarily bone, where K-wave X-ray fluorescence (K-XRF) can measure cumulative dose. Even in the presence of low current BLLs, a high bone lead, as measured by K-XRF, may be associated with chronic disease.[4] In lead-exposed workers, there is a close correlation between bone lead level and BLLs.[5] The EDTA-mobilization test, in which administration of this chelating agent is followed by increased excretion of lead in the urine, can help assess body burden of lead and lead exposure in the distant past.

Lead was used as a pigment in oil-based paint. Approximately two-thirds of homes in the United States constructed before 1940 and one-half built between 1940 and 1960 contain heavily leaded paint. In 1978, the Consumer Product Safety Commission lowered the legal maximum lead content in most types of paint to 0.06%, a trace amount.[6] Residential and commercial painters encounter lead exposure when they prepare surfaces for painting by burning, scraping, sanding, or chiseling old paint. Buildings constructed before 1960 have a high likelihood of having leaded paint in them, which may have been covered by subsequent layers of paint.

The main form of organic lead in commerce was tetraethyl lead (TEL) added to gasoline as an anti-knock additive for automobile engines. The lead industry mounted an extensive campaign in the 1920s to gain acceptance of TEL, and its pervasive use in gasoline since then led to accumulation of lead in the environment and in people worldwide. Acute organic lead poisoning was reported in the TEL workers as early as 1924. By the 1970s, the average BLL in the United States was 15 μg/dL. Cognitive impairment occurs in children even at BLLs below 5 μg/dL. Two generations of children, from the 1930s to the 1980s, suffered at least subclinical impairment by exposure to environmental lead. The decline in the use of leaded gasoline during the 1970s was accompanied by a decline in BLL (Fig. 11-5). Although lead is no longer used as an additive in most gasoline, it is still used in certain fuels, such as aviation fuel. The story of lead in gasoline reflects major failures and successes of public health as well as corruption and integrity in science.[7,8]

In developed countries, concerns about lead focus mainly on *(a)* maternal exposure before

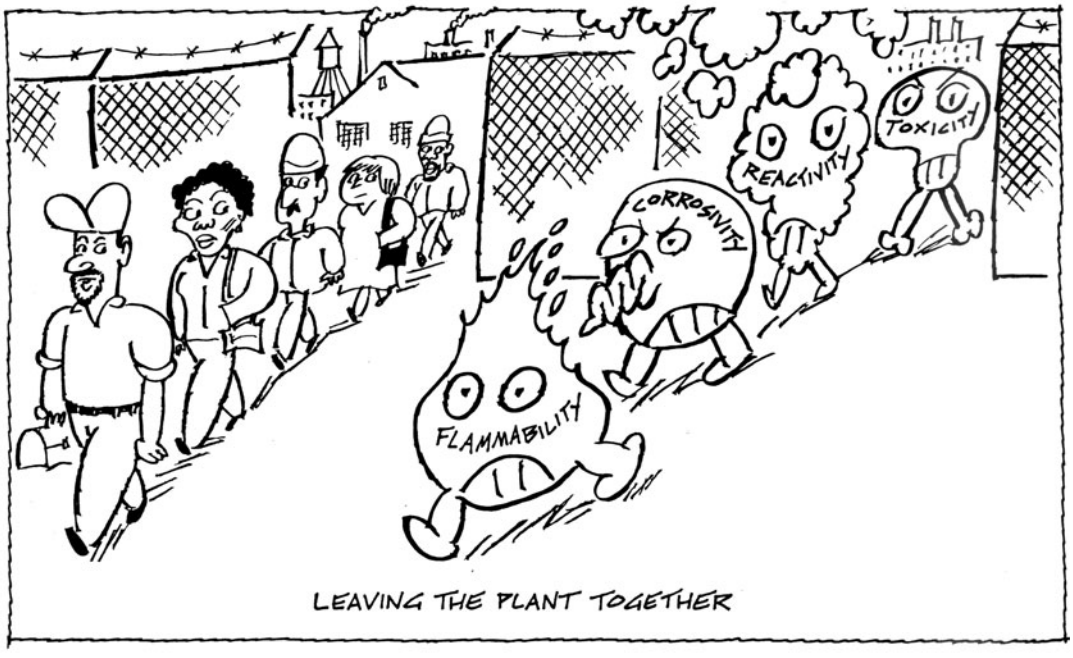
and during pregnancy, and *(b)* lead ingestion during infancy, both of which interfere with the developing nervous system, causing cognitive impairment.[9] As the mean population BLL has fallen below 10 μg/dL, it has been recognized that the cognitive impairment due to lead exposure occurs even below 5 μg/dL, suggesting that there is no threshold for this effect.[3,10]

Lead dust from occupational exposure can be transported home on workers' clothes, skin, and hair, thus exposing children and other household members. Even in the 1990s, children of lead workers had elevated BLLs attributable to this type of paraoccupational exposure.[11]

Most states require clinical laboratories to report some or all BLL test results.[12] When funding allows, states investigate the source of reported elevated lead exposure. Such investigations are sometimes impeded by rules regarding privacy. Elevated BLLs in children are reportable to the Centers for Disease Control and Prevention (CDC) through state and local health departments. (See Chapter 3.)

The OSHA lead standard, promulgated in 1978, covers "general industry" (foundries and factories); a 1993 OSHA standard covers the construction industry (including exposures due to welding and lead abatement, like in Case 2). Both standards require employers to conduct air monitoring to determine whether workers are exposed to lead. Employers are required to keep airborne lead levels at or below the permissible exposure limit (PEL) level of 50 μg/m^3 (8-hour time-weighted average). Both standards require training and education as well as air sampling. If airborne lead exceeds 30 μg/m^3, the action level, medical surveillance is required. If airborne lead exceeds 50 μg/m^3, employers must provide personal protective equipment, sanitation, and separation of work and street clothing.

The most important part of the OSHA lead standard for general industry is its medical removal clause with a rate retention provision. If a worker's BLL exceeds 60 μg/dL at any time or exceeds 50 μg/dL as the average of the last three tests (or of all tests within the past 6 months), the worker must be removed from exposure—with no loss of pay, seniority, or benefits. The worker may be returned to work when the BLL drops below 40 μg/dL. These standards, which have not been changed since first promulgated, are seriously out of date, allowing workers to maintain BLLs around 40 μg/dL, a level that is associated with adverse health effects.[13]



Workers can inadvertently bring toxic chemicals home with them. (Drawing by Nick Thorkelson.)

With comparable exposure, workers who smoke at work and those who do not use gloves or wash hands before eating have more lead exposure than other workers.[14]

Lead-acid batteries are now the main use for lead, and recovery and reuse of lead from spent batteries is important economically and environmentally.[15] An undocumented industry in both developed and developing countries is small-scale, often family-operated, battery-recovery, which frequently results in household and neighborhood lead exposure.

Some drinking water sources naturally contain high levels of lead (and other elements); others have been contaminated by lead from industrial or hazardous waste sites. The use of lead pipes and solder in plumbing fixtures also provides a direct exposure pathway. Some foods are high in lead because of environmental soil contamination, from past use of lead-containing pesticides and other sources (Fig. 11-6). In the past, lead chromate dyes used in food packaging were identified as a potential source of lead exposure to children.[16] The EPA has developed the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children, which is available on its Web site, that allows the user to vary inputs from food, water, soil, and air to obtain a predicted distribution of BLLs for exposed children of varying ages.[17]

There are still other exposures of adults and children to lead. Whereas lead toys were once commonplace, obvious, and identified as such, lead contamination of toys imported from China emerged as a major public health issue in 2008. Scrap lead has also been melted into inexpensive jewelry. Lead has been identified in cosmetics, home remedies, and dietary supplements, especially from Latin American and Asian countries.

Chelation treatment for lead poisoning relies on the use of sulfur-containing drugs that circulate through the body, binding lead and other metal atoms and extracting them from tissues so they can be excreted in the urine. Chelation is a controversial topic because it has been recommended by alternative healers for many conditions for which there is no evidence of benefit. Chelation is sometimes warranted and necessary for symptomatic patients with acute lead toxicity, especially if their clinical status is worsening. Chelation is of questionable benefit for chronic lead poisoning because it may suddenly mobilize lead sequestered in bone and redistribute it to other organs, including the brain.[18] Chelation is potentially harmful if the source of exposure has not been identified and controlled.

Figure 11-6. While a resident looks on, a National Institute for Occupational Safety and Health physician and health scientist collects a residential soil sample as part of a Pan American Health Organization and Centers for Disease Control and Prevention study to assess potential contamination of a community and "take-home" heavy metals related to a large tin smelter in Bolivia. (Photograph by Aaron Sussell.)

Dimercaptosuccinic acid (DMSA) has been approved by the Food and Drug Administration (FDA) for the treatment of childhood lead poisoning, although its efficacy has not been demonstrated for treating children with BLLs below 25 μg/dL.[19] It is unethical to treat lead-exposed workers with DMSA or other chelating drugs for prophylaxis while they are still being exposed to lead and this practice is specifically prohibited in the OSHA lead standard.

Mercury

Mercury, another element with no known essential function in the body, has many toxic effects.[20,21] It exists as elemental mercury (Hg^o), or quicksilver; as inorganic mercury, either as mercurous (Hg+) or mercuric (Hg++) salts; and in organic forms, mainly methylmercury (MeHg), dimethylmercury, ethylmercury, and phenylmercuric acetate. Elemental mercury, which is liquid at ambient temperatures, evaporates so that it can potentially saturate the air in closed rooms. Leaking or broken mercury-containing instruments, such as thermometers or sphygmomanometers, can be a source of mercury poisoning. Fine mercury droplets with greater surface-to-volume ratios release mercury more quickly than larger pooled droplets. Workers can be exposed to mercury during mining and distillation, production and use of mercury-containing products, and as bystanders when mercury-containing devices break.

Mercury is used in the manufacture of monitoring instruments and in certain industrial processes. It is important to distinguish the form of mercury (metallic, inorganic, or organic) when evaluating toxic effects. Metallic and inorganic mercury affect the nervous system and the kidneys. At high doses, exposed persons undergo personality changes, such as irritability, shyness, and paranoia (a syndrome called erethism); tremor; and peripheral neuropathy. Lower doses cause more subtle forms of these problems, such as visuomotor changes on neurobehavioral testing and slowed nerve conduction velocity. Kidney toxicity includes both glomerular and tubular dysfunction, with proteinuria and, in severe cases, impaired creatinine clearance. Exposure to metallic mercury is usually monitored through determinations of urine mercury levels, although blood levels may also be useful.

Organic mercury compounds (usually methylmercury) are sometimes encountered in workplace settings, but they are better known from outbreaks related to environmental contamination (usually human exposure to contaminated fish). These exposures have been associated with severe disease of the central and peripheral nervous systems and birth defects in children of pregnant women exposed to high levels of methylmercury.

In the past, the felt-hat industry used mercuric nitrate to break down hairs to produce felt. Some hatters had very high mercury levels and many showed symptoms of chronic mercurialism, including a tremor known as hatters' shakes and erethism. Community exposure to elemental mercury can also occur. Several volumes describe the history and status of mercury mining, exposure, and toxicity.[20,21] Ramazzini (1713) wrote eloquently about the maladies of mercury exposed gilders, mirror-makers, and even physicians. Of the mirror makers he wrote: "(T)hey learn by experience…how malignant is mercury….Those who make the mirrors become palsied and asthmatic from handling mercury. At Venice, on the island called Murano, where huge mirrors are made, you may see these workmen gazing with reluctance and scowling at the reflection of their own suffering in their mirrors and cursing the trade they have chosen."

All forms of mercury are toxic and potentially lethal to humans and animals. Mercurial compounds have been widely used for their biocidal properties, such as in antiseptics, antibacterials, and fungicides. Phenylmercuric acetate (PMA) has been used as a biocide in antifouling marine paints and indoor latex paint. In 1990, the EPA banned mercury for use in indoor paint. Increases in indoor mold and related symptoms since then may have been partially attributable to the removal of mercurial fungicides from indoor paint.

The dense liquid properties of mercury, which has a specific gravity of 13.6 and a low coefficient of expansion, have made it valuable in instruments for measuring temperature and air pressure. Many thermometers and barometers have contained mercury. Many states, recognizing broken thermometers as a preventable source of mercury exposure, have banned the sale of mercury thermometers. Hospitals have phased out many mercury-containing products; breakage of or a spill from one of these products may result in costly cleanups. Spillage during improper maintenance of gas meters that contain mercury can be a source of household mercury exposure.

The main route of exposure to elemental mercury is by inhalation of vapor. Contrary to popular opinion, elemental mercury is not absorbed through the skin and only negligibly in the gastrointestinal (GI) tract. Mercury has been used

as a flexible weight in long tubes by gastroenterologists to dilate constrictions or obstructions in the GI tract; this use is being phased out. Physicians have been exposed to mercury when they have injected it into these tubes, which can rupture and release mercury into a patient's intestine or peritoneal cavity.[22]

Dental amalgams, popularly known as "silver amalgams" and comprised of about 50% mercury, have been used for a century in dentistry. These amalgams, which are durable, but not impervious to wear, release small amounts of mercury vapor, especially during chewing. Amalgams contribute up to about 1 μg of mercury daily in the urine of people who have them. Mercury has been rapidly replaced in dental practice by composite materials. Controversies arose during the 1990s, when many people were being urged to have their amalgams removed to alleviate nonspecific symptoms, usually not including the classic symptoms of elemental mercury poisoning. The American Dental Association (ADA) stated that it was unethical for dentists to recommend amalgam removal for health reasons, although they could remove them at the request of patients. The ADA contends that, except for uncommon local allergic reactions, there is no evidence of systemic harm from amalgams.[23] In Europe, dental amalgams are being phased out as part of a more aggressive program to reduce all uses of mercury.

During the late 1990s, many dentists began to develop mercury-free practices. Dental insurance companies reimbursed patients only for the cost of amalgams, not for the higher cost of composite fillings, thereby encouraging less affluent patients to settle for amalgam fillings. However, during removal of amalgams, patients have temporary increases in blood mercury levels, followed by decreases—with the biological half-life of mercury being about 2 months. Swallowing bits of amalgam is probably of negligible importance since elemental mercury is not well absorbed from the intestine.

Over many centuries, mercury has been used for many medicinal purposes, including treating syphilis. Mercury compounds, especially thimerosal (ethylmercury), was widely used as an antiseptic (in mercurochrome and merthiolate). These products are still sold in pharmacies, but they now contain other antiseptics. Thimerosal, used extensively as an antimicrobial to stabilize vaccines and other biologics, has been removed from vaccines for children and infants. Mercurial compounds are used by traditional healers in several cultures.[24] Calomel (mercurous chloride) was widely used for several disorders as a cathartic and purgative agent against parasites, and for diphtheria by promoting the sloughing of mucosa in the throat.[25] Calomel, which was incorporated into teething powders used for infants, caused acrodynia (pink disease) with redness and pain in the hands and feet; it took many years before officials were convinced that calomel caused acrodynia and banned its use.

Minamata disease refers to severe methylmercury poisoning. In the mid-1950s, in fishing communities on Minamata Bay in southern Japan, people who regularly consumed fish from the bay developed a disorder that affected the cerebellum and peripheral nerves. Inorganic mercury had been released into Minamata Bay in effluent from a nearby factory and subsequently transformed into organic mercury through the action of aquatic microorganisms, and then bioamplified through the food chain. Mild cases involved numbness and tingling and burning sensations around the mouth and in the fingers. More serious cases progressed to cognitive impairment, slurred speech, unsteady gait, deafness, tunnel vision, blindness, and death. There were hundreds of cases and at least 35 deaths.[26] Infants born to exposed mothers suffered congenital Minamata disease, characterized by motor impairment, mental retardation, and blindness. The syndrome was reproduced in cats that were fed fish from the bay.

Organomercurial fungicides, including those with methylmercury, were widely used as fungicides for protection of grain stored for seeding. In the 1970s, epidemics in Iraq and Guatemala occurred inadvertently when people used mercury-treated grain for baking, rather than planting, and became severely poisoned. Mercury is no longer approved for seed treatment. The use of phenylmercuric acetate as an anti-barnacle/anti-fouling additive to boat paints has also been banned, although its replacement (tributyltin) is equally harmful to aquatic life. Mercury was added to indoor latex paint as a preservative and to prevent mold growth and prolong shelf-life. All of the biocidal properties

of mercury supported manufacturing industries in which workers were exposed to organic mercurial compounds. The formulation and application of mercury-containing fungicides exposed factory and agricultural workers.

Inorganic mercury, from both industrial and natural sources, can be converted to methylmercury in the sediment of bodies of water by anaerobic bacteria. Methylmercury absorbed by microorganisms, bioaccumulates at every stage of the food chain, reaching 0.2 μg/g (0.2 ppm) in herbivorous fish and exceeding 1 ppm in many predatory fish, such as tuna, swordfish, and shark. Although the FDA has set an action level (the level at which commercial fish could be seized) of 1 ppm of methylmercury for commercially sold fish, mercury levels have been found as high as 4 ppm in fish being sold in supermarkets. (By comparison, in Minamata Bay, mercury levels in fish exceeded 10 ppm.) People who consume fish frequently may ingest significant quantities of mercury and, as a result, develop symptoms.

Elemental and inorganic mercury are excreted mainly in urine, but methylmercury is excreted mainly in feces. Therefore, urine is not a sensitive indicator of methylmercury ingestion. Some methylmercury deposits in hair, which grows at a rate of about 1 cm per month, are used to identify chronology of exposure. Most people have hair levels below 1 ppm, but hair levels above 8 ppm have been associated with symptoms of mercury poisoning. Levels above 50 ppm were seen in some people with Minamata disease.

People who frequently eat large predatory fish, such as swordfish and tuna, are vulnerable to developing mercury poisoning. However, fish consumption has also been shown to benefit pregnancy outcome and reduce the risk of cardiovascular disease. These benefits have been attributed, in part, to omega-3 polyunsaturated fatty acids (PUFAs) and selenium present in fish. Benefits can also be achieved by consuming fish-oil supplements. Balancing the benefits of fish intake with contaminants, such as methylmercury and polychlorinated biphenyls (PCBs), has been challenging and controversial. People who daily consume very large amounts of fish, including members of families that do subsistence fishing, often have elevated mercury levels, even if they eat fish that has low levels of mercury. Selenium offers some protection against mercury poisoning, especially if the selenium-to-mercury molar ratio exceeds 7. Most predatory fish with high mercury levels are not high in PUFAs. However, salmon, herring, and some other fish are low in mercury and high in PUFAs.

Although minute amounts of dimethylmercury are present with methylmercury in aquatic environments or in hazardous waste, dimethylmercury is mainly a laboratory reagent used, for example, to calibrate nuclear magnetic resonance equipment. There are very few reports of poisoning due to dimethylmercury. However, a leading chemist died from mercury poisoning 10 months after she spilled a few drops of dimethylmercury on her hand on which she had worn a latex glove. (Dimethylmercury readily passes through latex and is readily absorbed from the skin.) Her blood mercury levels exceeded 1,000 μg/L.

Whereas chelation is appropriate for patients with acute and/or worsening symptoms of mercury poisoning, it is not generally useful for people with chronic exposure, such as from daily consumption of fish. When there has been chronic exposure to mercury, the source should be identified and controlled.

Countries, including the United States and those in the European Union (EU), have endorsed the phase-out of mercury wherever possible.[27] The EU intends to eliminate exports of any mercury or mercury-containing products. Only a few types of batteries now contain mercury. Household fever thermometers containing mercury have been banned in many states. Hospitals have reduced their reliance on mercury-containing devices. Automobile manufacturers have reduced their use of mercury-containing switches.

In developing countries, mercury is widely used in small-scale gold mining, where riverbed gravel is washed with water and mixed with mercury to extract gold. The resulting gold amalgam is then heated to drive off the mercury. This is often done in homes, thereby exposing children and adults. Many governments distribute mercury to miners free of charge to acquire gold for international trade. (See Box 11-1 for a description of an ecohealth approach to mercury contamination in a gold-mining area in Brazil.)

Box 11-1. An Ecohealth Approach to Mercury Contamination

Donna Mergler

During the past 15 years, an interdisciplinary team of Canadian and Brazilian researchers from the natural, health, and social sciences have used an ecosystem approach, with a strong participatory research component, to examine pathways of mercury contamination, to assess human exposure to mercury and health effects, and to evaluate mitigation measures. This work, known as the Caruso Project, has been done in collaboration with communities on the Tapajós River, a major tributary of the Amazon.

Early reports of high levels of mercury in fish and people in the area attributed the source of mercury to gold mining. However, the project's research found that large-scale deforestation was the major culprit, mainly due to "slash-and-burn" agricultural practices, followed by heavy rains on the denuded land washing the mercury-laden soils into the river. The deforested areas are increasing in size due to in-migration of poor people from Northeast Brazil and the need to clear the forest to grow food. Climatic conditions and aquatic vegetation provide favorable conditions for mercury methylation, accelerating the incorporation of mercury into the food chain and contaminating fish, a dietary mainstay of this population. Mercury concentrations in fish in the area vary greatly, depending upon feeding habits, growth rate, age, and location. Mercury concentrations in human hair and blood increase with fish consumption, are higher among people who eat more piscivorous (fish-eating) fish, and vary seasonally. Mercury exposure was found to be associated with visual and motor deficits, changes in cardiovascular function, and biomarkers of oxidative stress.

Since eating fish is highly nutritious, research questions have focused on other dietary factors that could influence mercury absorption, metabolism, and toxicity, such as fruit, selenium, and omega-3 fatty acids. An exploratory study, coordinated by a village midwife, included 26 village women who, over a 12-month period, kept daily food-frequency diaries. Results suggested that, controlling for fish consumption, those who ate more fruit had lower blood and hair mercury concentrations. These results were confirmed by larger studies. Another series of studies examined the role of selenium, which in this population, may be markedly increased in blood due to consumption of Brazil nuts. Plasma selenium was found to be correlated with improved performance on several motor and visual tests, independent of mercury.

At each stage, the integrated results of this research were returned to the communities, and village workshops examined short-, medium-, and long-term solutions concerning dietary, fishing, and farming practices. People were encouraged to "Eat more fish that don't eat other fish," since herbivorous fish contain less mercury. In one village, reassessment of fish consumption, exposures, and neurological outcomes 5 years later showed that the villagers ate the same amount of fish, but they had increased the proportion of fish that did not eat other fish. And mercury levels had decreased by 40%. Motor function of villagers improved, but their visual function continued to decline as a function of prior exposure. An illustrated booklet informed the population of the mercury cycle and its effects, emphasizing preventive actions and dietary changes that would reduce exposure while continuing to eat fish.

Parallel to the work on diet, nutrition, and mercury exposure, the environment team has been working with fishers to identify "hot spots" for methylation, and with farmers to modify agro-forestry practices to reduce soil erosion. A major project is currently underway with experimental reforestation plots. Studies of social communication networks are helping to find ways to implement programs for improving the environment and health. The success of this project is attributed to the collaboration of scientists of various disciplines and community participants, working together in an ecosystem approach to improve human health.

Where mercury is still in use, recycling of mercury, rather than mining of virgin mercury, should provide a sufficient amount. Surplus mercury could be inactivated, possibly by forming insoluble mercury sulfide.

Cadmium

Cadmium, another metal without known essential or beneficial biological functions, is nephrotoxic and carcinogenic. Because some cadmium is present in lead and zinc ores, cadmium pollution occurs near lead and zinc smelters. Cadmium has been used in alloys with other metals, in paint pigments, in nickel-cadmium batteries (now almost phased out), and in electroplating. It has also been used to stabilize plastic.

Cadmium fume can be absorbed through the lungs. Plants, especially rice, can absorb cadmium from soil, thereby making food a significant source of cadmium exposure. Cadmium is present in tobacco and tobacco smoke. Once absorbed, cadmium is stored in soft tissues. Most of the body burden is in the kidney, where cadmium causes tubular damage. Cadmium has a long biological half-life (10 to 30 years). Cadmium exposure can be monitored by determining cadmium concentrations in urine or blood.

Cadmium induces the liver to synthesize metallothionein, a low-molecular-weight, sulfur-rich transport molecule, to which it binds avidly. The metallothionein-cadmium complex is transported in the blood to other organs. Metallothionein normally transports and regulates zinc, a cofactor for many enzymes.

Exposure to cadmium occurs during the smelting or refining of other metals or during torch cutting of steel that has been coated with cadmium. Acute cadmium poisoning is dramatic, but not common. Cadmium has a relatively low boiling point (765°C), so that cadmium fume is generated at temperatures that do not release fumes from other metals. Cadmium fume is toxic to alveoli, resulting in cadmium-fume fever and pulmonary edema about 6 to 12 hours after exposure. It can be fatal.

Chronic lung and kidney disease developed in cadmium-exposed workers in Europe employed from the 1950s to 1970s, when exposures were higher than today. Chronic kidney disease begins with damage to proximal tubular cells, resulting in proteinuria, which can be monitored by increased urine levels of beta-2-microglobulin, retinol binding protein (RBP), or the brush border enzyme n-acetyl glucosaminidase (NAG). With low exposures, these changes are reversible. But with higher exposures, a tipping point is reached beyond which renal damage is progressive, even when exposure is terminated. Tubular proteinuria is an early warning sign of possibly significant kidney damage.[28]

Arsenic

Technically a metalloid rather than a true metal, arsenic is widely known as a classical poison. The organic forms that occur in nature are much less toxic than the inorganic forms. Inorganic arsenic occurs in a trivalent state (arsenites) and a pentavalent state (arsenates). Soluble inorganic arsenicals are readily absorbed from the intestine. Pentavalent compounds are reduced to trivalent compounds, which undergo oxidative methylation to form monomethyl and dimethyl arsenous acids, which are water-soluble and eliminated in the urine. (Methylation capability varies among individuals due to genetic polymorphisms in methyltransferase genes.) The main form of organic arsenic in seafood is arsenobetaine, which undergoes little metabolism and is quickly eliminated in the urine. Therefore, biomonitoring of persons for arsenic exposure must analyze urine for both inorganic and organic arsenic compounds.

Occupational exposures to arsenic occur in the smelting of metal ores (especially lead, zinc, and copper), the production and application of arsenical pesticides, the microelectronics industry (mainly in production of gallium arsenide), and the pharmaceutical industry (which produces some anti-helminthic arsenical medications). However, most arsenic poisoning is due to nonoccupational exposure in drinking water from wells that tap aquifers with naturally occurring inorganic arsenic. This exposure to arsenic is widespread in Bangladesh and elsewhere in South Asia. In the United States, where arsenical insecticides were widely used into the 1960s and arsenical defoliants were widely used more recently, high residues of arsenic in soil, impede the conversion of contaminated land from agricultural to residential use.

Ongoing arsenic exposure can be evaluated with a 24-hour urine sample, which can provide an accurate indication of exposure. Urinary arsenic determinations usually include total arsenic and inorganic arsenic. The blood arsenic level may be informative, but the half-life of inorganic arsenic in blood is short (about 2 hours). If the suspected exposure has ended more than 2 weeks before, hair or nail samples may provide the best indicator of the exposure.

Arsenic is released to the environment in some industrial and chemical processes, including smelting of some metal ores. It is readily absorbed from air, food, or water; distributes widely in the body; and is toxic to all organ systems. Acute occupational poisoning, which is rare, can be manifested with such symptoms as abdominal pain and cramping, difficulty swallowing, vomiting, watery diarrhea, and gastrointestinal bleeding. Shock and death can occur. Survivors may have liver and kidney damage and seizures.

Chronic arsenic poisoning can be manifested with hyperpigmentation of the skin and warty keratoses, especially on the soles of the feet and other parts of the body not exposed to the sun; ischemic heart disease; liver damage; anemia; lung irritation; diabetes; and a symmetrical

distal polyneuropathy. People exposed to arsenic in drinking water are at increased risk of diabetes and hypertension. Arsenic can cause skin, liver, lung, and bladder cancer. Arsenic is unique among known human carcinogens in that there is no reliable animal model. Exposure to arsenic is usually monitored by measuring urinary arsenic levels.

Arsine (arsenic trihydride) is a colorless, odorless gas that is formed when acids leach arsenic ions out of metals. Occupational exposure occurs in metallurgical work, including etching and lead-acid battery manufacture. The use of acid cleaners to dislodge reaction residues from metal reactor vessels may generate arsine gas, and workers inside these vessels have been poisoned, sometimes fatally. Arsine gas is also used, in closed systems, in the doping process of microelectronic chips to form gallium arsenide. Arsine is a potent hemolytic agent. Following a prodrome of malaise, headache, trouble breathing, and vomiting, an exposed worker may experience severe abdominal pain and sudden onset of jaundice. Severe anemia may occur, with free hemoglobin in the serum and blood in the urine. In severe poisoning, kidney failure may occur.

Manganese

Manganese is an essential trace element, but long-term inhalation of manganese dust or fumes can cause a parkinsonian syndrome by its toxic effect on the basal ganglia. Welders who use manganese-coated welding rods, workers in foundries, ceramics workers, workers who burn or grind ferromanganese alloy steel, and agricultural workers who spray manganese-containing fungicides are among those workers who may be exposed to manganese. The proposed addition to gasoline of an organic manganese compound as an anti-knock agent has been highly controversial.

Nickel

Nickel has important industrial uses in stainless steel, electroplating, batteries, coins, jewelry, and pigments. Nickel is absorbed from the lungs (about 20% to 35%) and the gastrointestinal tract (up to 40% from water), and it is excreted mainly in the urine. Nickel is an essential trace element, but it is toxic to the skin, respiratory tract, and the immune system. The most common adverse health effect is skin sensitization (in about 10% to 20% of the population), which results in contact dermatitis. At high occupational levels, nickel is toxic to both the upper and lower respiratory tract. Inhalation of some nickel compounds, especially nickel subsulfide, in the smelting and refining industries can cause lung and nasal cancer, and these compounds are considered human carcinogens by the International Agency for Research on Cancer (IARC) and the EPA. Nickel carbonyl, a highly reactive compound formed when nickel reacts with carbon monoxide, is rapidly absorbed and causes acute toxicity.

Beryllium

Beryllium, a light metal with high tensile strength, is used as a modifier in nuclear reactors to capture neutrons. It is used in electronics and some other industries. Until beryllium was removed from fluorescent light bulbs, bulb manufacturing workers were at increased risk for beryllium poisoning.[8] Machinists working with beryllium have potential exposure. Beryllium can cause acute or chronic poisoning as well as cancer. It is also immunotoxic, as demonstrated by a lymphocyte proliferation test (LPT), which can identify beryllium sensitization. Lymphocytes sensitized by beryllium undergo transformation and proliferate upon subsequent exposure to the antigen.

Chronic beryllium disease, resulting from inhalation of beryllium, is characterized by pulmonary granulomas that can reduce pulmonary function and cause dyspnea on exertion. Chest X-rays show diffuse infiltrates and enlarged lymph nodes. Lymphocyte transformation testing on blood or bronchial alveolar lavage fluid can assist with the early diagnosis of berylliosis. Sarcoidosis, a serious granulomatous disease of unknown etiology, can present with similar symptoms, and some cases initially diagnosed as sarcoidosis have proven to be beryllium disease.

Dr. Harriet Hardy, one of the founders of occupational medicine in the United States, established a registry of cases of beryllium disease that helped to increase recognition and understanding of this disease.

INORGANIC ACIDS AND BASES

A wide variety of corrosive and caustic inorganic acids and bases are present in workplaces and homes. When acids dissolve in water, they release H^+ ions; when bases dissolve in water, they release OH^- ions. At low concentrations, these compounds irritate the skin, eyes, and mucous membranes of the respiratory tract; at high concentrations, they produce chemical burns of these tissues.

The pH of a substance reflects its H+ concentration. The pH of water, which is completely neutral, is 7. Strong acids have a pH below 2; strong bases have a pH above 12. The pH of blood is maintained by homeostatic actions of the lung and kidney at about 7.4. The pH of stomach contents is generally below 2.

Hydrofluoric acid (HF), which is highly reactive and intensely corrosive, is used in chemical syntheses. In liquid and gaseous forms, it is used in manufacturing chemicals, etching glass and metals, pickling stainless steel, and etching computer chips. Even a few drops of HF on the skin can burn deeply—even down to bone. Small HF burns may require hospitalization. Exposure to higher concentrations or large volumes of HF may be fatal. Treatment requires removal from exposure and decontamination of the burned area, and infiltration and intravenous treatment with calcium gluconate (which precipitates calcium fluoride).

Organic Compounds

A large variety of organic compounds are used in commerce, most of which have not been subject to extensive toxicologic testing or epidemiologic study. Some OSHA PELs that were established in 1970 are now outdated. In some instances, industries have established guidelines for occupational exposure limits (OELs) for organic compounds in the absence of appropriate OSHA PELs.

Only a small subset of organic compounds are described in this chapter to represent the main classes of these chemicals to which people are exposed in their workplaces, their homes, and their communities. Many organic compounds are nonpolar and therefore soluble in organic solvents, but not in water. Many are easily absorbed through the skin. Organic compounds of low molecular weight are volatile. Many polyaromatic organic compounds are considered to be semivolatile. (Organometallic compounds have been described earlier.)

Alkanes are compounds that range from methane, which has one carbon atom, to long-chain compounds of high molecular weight, which have many carbon atoms. They can be saturated (have no double bonds) or unsaturated (have double bonds), and they may form straight-chain or branched-chain isomers. At ambient temperature, the alkanes with one to four carbon atoms (methane, ethane, propane, and butane) are gases, while pentane and alkanes with more carbon atoms are liquids. Gasoline is a mixture of medium-chain alkanes (5 to 12 carbon atoms) that is produced from fractional distillation of crude oil.

Like other alkanes of low molecular weight, n-hexane is an acute depressant of the central nervous system (CNS). However, its unique metabolite, 2,5-hexanedione, causes an irreversible "dying-back" axonopathy, manifest by a peripheral neuropathy in a glove-and-stocking distribution. The effect is specific to the 2,5-dione structure, and neither n-pentane nor n-heptane has this metabolite or this toxicity. (See Chapter 19.)

ORGANIC SOLVENTS

CASE 3

During a routine medical examination, a 24-year-old man reported problems with concentration. He frequently lost his train of thought, forgot what he was saying in midsentence, and had been told by friends that he seemed to be forgetful. He also felt excessively tired after waking in the morning and at the end of his workday. He had occasional listlessness and frequent headaches. At work, he often felt drunk or dizzy. Several times, he had misunderstood simple instructions from his supervisor. All of these problems had developed insidiously during the previous 2 years. The patient thought that other employees in his area of the plant had complained of similar symptoms. He noted some

relief during a week-long fishing vacation. He denied appetite or bowel changes, sweating, weight loss, fever, chills, palpitations, syncope, seizures, trembling hands, peripheral tingling sensations, and changes in strength or sensual acuity. He drank alcohol occasionally. He denied recreational drug use and cigarette smoking.

This man had worked for approximately 3 years as a car painter in a repair garage for railroad cars. At his physician's urging, he compiled a list of substances to which he had been exposed (see Table 11-8).

When his plant had been inspected by OSHA 1 year before, only minor safety violations had been found. Physical examination, including a careful neurologic examination, was completely normal. Routine hematologic and biochemical tests, thyroid function studies, and a heterophile antibody assay were all negative. His erythrocyte sedimentation rate was 3 mm/hour (normal). He had slight elevations of serum gamma-glutamyl transpeptidase (SGGT) and alkaline phosphatase.

This case illustrates many problems that confront clinicians. The patient reported vague, nonspecific symptoms that a busy clinician might easily dismiss. However, many toxins produce such nonspecific symptoms. In addition, the patient had multiple chemical exposures, making it difficult to identify one specific cause. This patient was unusual in that he was able to provide a list of his exposures, although this list had limitations. For example, it included two (fictional) trade names of chemicals whose identities were not known and might be difficult to determine. The absence of OSHA citations 1 year earlier may suggest—but not prove—that all exposures were then at permissible levels. However, the OSHA inspection might have been directed only at safety—not health—hazards, the plant may have been cleaned up just before the inspections, workplace conditions could have deteriorated in the year since the inspections, and new production processes could have been initiated or new materials introduced since then. In any event, all the symptoms that this man reported have been associated with exposure to concentrations of organic solvents below permissible exposure levels, so even substances in a well-maintained workplace could be the cause of his symptoms.

Organic solvents are frequently used in many types of work, including oil refining and petrochemical production, plastics manufacturing, painting, and building maintenance. Often several different solvents are present in one product, and multiple products that contain solvents may be used in a workplace. Some products, such as paints, glues, and pesticides, are mixtures containing substantial proportions of solvents. The formulation of products containing solvents has changed over time because of economic factors and concern about the toxicity of specific solvents. These factors may make it difficult to determine a worker's exposure over a period of time.

As illustrated in Case 3, many organic solvents target the nervous system, causing both acute effects (narcosis) and, in some persons, chronic neurobehavioral effects. In addition, several specific solvents, including carbon disulfide, n-hexane, and methyl n-butyl ketone (MBK), cause a peripheral neuropathy characterized by loss of distal sensation, progressing to include motor weakness and even paralysis. The disease may progress for several months after exposure has ceased, and permanent damage may occur. (See also Chapter 19.)

Table 11-8. List of Substances to Which Man in Case 3 Was Exposed

Paint Solvents	Paint Binders	Other Substances
Toluene	Acrylic resin	Organic dyes
Xylene	Urethane resin	Inorganic dyes
Ethanol	Bindex 284	Zinc chromates
Isopropanol	Solution Z-92	Titanium dioxide
Butanol		Catalysts
Ethyl acetate		
Ethyl glycol		
Acetone		
Methyl ethyl ketone (MEK)		

Several organic solvents cause other toxic effects. Benzene was commonly used as an industrial and commercial solvent in the past. High exposures suppress the bone marrow, sometimes leading to anemia or pancytopenia. Benzene is also a potent carcinogen, leading to leukemia and other hematopoietic malignancies. Because of this toxicity, benzene is now used much less frequently, although exposures continue to occur in the petrochemical industry and in some other industries. Some other hydrocarbons, including ethylene oxide, the chloromethyl ethers, and epichlorhydrin, are also known carcinogens. Many other solvents are suspected of being carcinogenic, including several halogenated compounds.

Many organic solvents are hepatotoxic. Carbon tetrachloride, chloroform, and tetrachloroethane can cause hepatic necrosis. Long-term exposure to carbon tetrachloride has been associated with the development of cirrhosis. Dimethyl formamide (DMF) and 2-nitropropane have caused outbreaks of chemically induced liver disease in exposed workers.

Some organic solvents, including the glycol ethers and ethylene oxide, adversely affect the reproductive system (Chapter 20). Solvents frequently cause skin irritation; these chemicals dry the skin by removing natural skin oils (Chapter 22). Many organic solvents are also acute respiratory tract irritants (Chapter 18).

The diagnosis of health problems related to solvent exposure heavily depends on a thorough occupational and environmental history (Chapter 2). Biological monitoring may be helpful for ongoing exposures, but it is not useful for evaluating past exposures, because most solvents are metabolized and cleared from the body relatively quickly.

Control of solvent exposure is based on a careful evaluation of how exposure has occurred. Work procedures and practices are often important determinants of solvent exposure for many people who work with organic solvents, such as painters. Preventive measures include substitution with a less toxic substance, changed work practices, and personal protective equipment. Traditional engineering methods, such as ventilation and containment, are also useful.

Many of the low-molecular-weight aromatic and aliphatic compounds are widely used in a great variety of industrial processes as solvents or raw materials. They are volatile, and many are neurotoxic. These compounds have been used as anesthetics or abused for the "highs" they induce.

Benzene (C_6H_6), a colorless, pleasant-smelling volatile liquid, is the parent aromatic compound. It has been widely used as a solvent and as a raw material for building a wide variety of chemicals. Benzene is produced during the refining of petroleum. As a group, benzene, toluene, ethylbenzene, and xylene are referred to as BTEX compounds. Substitution has greatly reduced exposure to benzene; it is no longer used as a solvent and degreasing agent when other less toxic—but not innocuous compounds—can fulfill these functions. Benzene exposure still occurs in a variety of industries, including perfume manufacture, chemical research and development, and shoe manufacture. Benzene is present, in a concentration up to 10%, in unleaded gasoline, resulting in some benzene exposure to gas station attendants. It is also a constituent of cigarette smoke.

Like all organic solvents, benzene is a defatting agent; therefore, it can cause dermatitis and can be absorbed through skin. However, inhalation of benzene vapor is the main route of entry. About 50% of an inhaled dose is absorbed into the bloodstream. Benzene is very rapidly metabolized, in at least two pathways, to muconaldehyde and muconic acid or to quinones and phenol. Muconaldehyde and quinones are toxic to the bone marrow, causing anemias and leukemias. Measuring benzene in blood is not useful since it is cleared very quickly. Urinary phenol has been used as a nonspecific biomarker of benzene exposure.

Like other organic compounds of low molecular weight, benzene readily crosses the blood–brain barrier. Acute exposure to benzene causes central nervous system (CNS) depression, ranging from lightheadedness and headache to coma, convulsions, and death. However, it is the chronic toxicity of benzene that is of greatest concern. Benzene depresses bone marrow production of red blood cells, white blood cells, and platelets, sometimes leading to pancytopenia and aplastic anemia, which is often irreversible and fatal. Benzene causes a wide variety of lymphohematopoietic diseases. The oldest, strongest epidemiologic evidence links benzene with acute

myelogenous (myeloid) leukemia (AML). However, multiple studies now show excesses of all other types of leukemia as well as multiple myeloma and non-Hodgkin lymphoma in benzene-exposed cohorts.

At its inception, OSHA adopted the benzene TLV of ACGIH (10 ppm) as its PEL. But as epidemiologic evidence regarding leukemia increased, OSHA revised the (8-hour time-weighted-average) standard, in 1978, to 1 ppm. In 1980, in response to an industry challenge, the Supreme Court invalidated the standard because OSHA had failed to provide "significant risk of material health impairment." Part of the controversy hinged on whether aplastic anemia was a necessary precursor of leukemia—if a 10 ppm standard were adequate to prevent aplastic anemia, it should also prevent leukemia. During the 1980s, studies by IARC, EPA, and the National Institute for Occupational Safety and Health (NIOSH) provided convincing evidence of excess leukemia risk at exposures below 10 ppm, and, in 1987, OSHA promulgated and successfully defended a benzene standard of 1 ppm.

Toluene and xylene, both of which are methylated benzene compounds with many industrial applications, share the CNS depression properties of benzene, but they do not depress the bone marrow or cause leukemia.

Halogenated aliphatic solvents include a broad array of generally nonreactive, usually volatile liquids of low molecular weight, all of which depress the CNS and cause dermatitis. Most are hepatotoxic and nephrotoxic. Some are well-documented carcinogens. Long-term exposure can cause irreversible cognitive impairment. The most commonly encountered halogenated aliphatic solvents have one to four carbon atoms and up to one double bond. Among the best-known chemicals in this group are carbon tetrachloride (tetrachloromethane), chloroform (trichloromethane), methylene chloride (dichloromethane), trichloroethylene (trichloroethene), and perchlorethylene (tetrachloroethene). Dibromochloropropane (DBCP), an effective nematocide, was found to be a testicular poison (Chapter 20). (See Fig. 11-1.)

Bioactivity, toxic potency, and hazard of halogenated aliphatic solvents vary by structure and exposure. Many of these chemicals have been used as solvents in degreasing, cleaning, manufacturing, and laboratory work. Many workers have at least some potential for exposure to these chemicals. Dyes, paints, glues, pharmaceuticals, personal-care products, agrochemicals, and many other substances are either produced with these solvents or contain them as "inert ingredients."

In the past 50 years, use of specific chemicals as anesthetics and solvents has dramatically changed, in part because of concerns about their toxicity. Chloroform was replaced by other anesthetics because of toxicity and efficacy. Carbon tetrachloride was replaced as a solvent and spot remover because of its liver toxicity. Trichloroethylene has been largely replaced by the somewhat less toxic trichloroethane and, for dry cleaning, by tetrachlorethylene (perchloroethylene, or "perc"), which is now being phased out because of its toxicity, in favor of "green" cleaning.

Occupations with regular use of solvents include chemical production; laboratory research; production and application of paints, dyes, and coatings; printing; metalworking and machine degreasing; development and production of pharmaceuticals; oil and chemical refining and production; and gas station work. Solvents are found in many household products, including cleaning solutions, paints and stains, paint thinners, cosmetics, nail polish remover, and pesticides.

Solvents are often used as mixtures, such as gasoline, kerosene, and paint thinners, making it hard to evaluate the contribution of single compounds. *White spirit* refers to mixtures of solvents containing both aromatic and aliphatic components. Volatility varies with the composition of low- and high-molecular-weight constituents and workplace temperature. Area air monitoring and personal breathing-zone air monitoring are useful in determining exposure potential. Personal breathing-zone monitoring is more reliable for individual exposure assessment since workers may move into and out of areas where air concentrations of solvents are high. Biological monitoring is useful in those few situations where there is a specific marker of exposure for a specific solvent.

OTHER ORGANIC CHEMICALS

Vinyl chloride monomer (VCM), a gas, is the raw material for polyvinyl chloride (PVC), a

ubiquitous plastic. Although PVC is inert, VCM is highly reactive. In the 1960s, VCM was found to cause acroosteolysis, a disorder characterized by osteoporosis, Raynaud phenomenon affecting the fingers, and skin thickening and nodule formation on the arms. In 1974, there was a report of three workers with angiosarcoma of the liver (an extremely rare disease) who had been exposed to VCM at a plant in Kentucky. Recognition of VCM as a carcinogen led to OSHA lowering the PEL from 500 to 1 ppm. Despite initial protests by industry, controlling occupational exposure to VCM below this PEL was feasible and even profitable. Polyvinyl chloride continues to be widely produced and has many uses, including water pipes and construction material.

Methyl tert butyl ether (MTBE) has been widely used as a gasoline additive (oxygenate) to improve its octane rating, promote more complete combustion, and reduce release of carbon monoxide, oxides of nitrogen, and other products of incomplete combustion. From about 1990 to 2006, MTBE was present in gasoline in concentrations higher than 10% (by volume). Many people who were exposed to MBTE reported symptoms of eye and respiratory tract irritation. In 2004, based partly on irritation and partly on carcinogenesis in several animals, several U.S. states banned MTBE. In 2006, gasoline companies stopped adding MTBE to gasoline, partly because of legal issues resulting from contamination of drinking-water supplies by MTBE spills and leaking underground gasoline storage tanks.

Bis(chloromethyl) ether (BCME) is a highly reactive chemical that is a known human carcinogen, which has been established as a cause of oat cell lung cancer among workers. This type of cancer, which is not associated with smoking, occurred in workers exposed to BCME alone and, to a lesser degree, in workers exposed to chloromethyl methyl ether that had BCME as a low-level contaminant.[29]

Formaldehyde, a highly reactive chemical that is used widely, has been recognized as a carcinogen for more than 30 years. A gas at ambient temperature, it has a characteristic odor. It irritates the eyes and, because it is highly soluble in water, it irritates the upper respiratory tract. It is also a potent sensitizer. Formaldehyde is used in producing plastics, resins, particle board, and paper. After the 1973 energy crisis, urea-formaldehyde foam was widely used as a retrofitting insulating material that was injected into the walls of homes. However, excess application resulted in off-gassing of formaldehyde and serious symptoms in residents of homes where it was used. Formaldehyde in solution (formalin) has been used as a preservative, exposing pathologists, laboratory technicians, and students in anatomy laboratories as well as museum workers and embalmers. Engineering controls can limit formaldehyde exposure. In anatomy and pathology laboratories, workers and students often use respiratory protection. Phenol has been substituted for formaldehyde as an anatomic preservative; however, workers who are sensitive to formaldehyde may eventually have to change occupations.

Formaldehyde is genotoxic, causing chromosomal breaks and forming cross-links in DNA and between DNA and proteins. Studies have found brain cancer, colon cancer, and leukemia among exposed pathologists and morticians and evidence for lung cancer in exposed industrial workers. In 2004, IARC finally determined that formaldehyde was a Group 1 carcinogen.[30]

Isocyanates are highly reactive compounds of low molecular weight that contain an -NCO group. They react with hydroxyl groups to form polyurethane polymers that are used in foams, thermoplastics, spandex fibers, and polyurethane coatings. Exposed workers include plastic manufacturers, foam makers, and producers of a wide range of polyurethane products. Incineration of polyurethane compounds releases isocyanates. Isocyanates are both strong irritants and sensitizers of the skin and respiratory tract. Isocyanates can cause or exacerbate asthma. Some isocyanates are animal carcinogens. Diisocyanates (with two –NCO groups) are the isocyanates that are most widely used, especially methylene diphenyl diisocyanate (MDI) and toluene diisocyanate (TDI).

Endocrine-Disrupting Chemicals

Endocrine disruption refers to any mechanism that increases or decreases the function of

the endocrine system. Chemicals may mimic some of the actions of hormones. For example, xenobiotics that mimic estrogen, known as *environmental estrogens*, may bind to estrogen receptors, either activating them or preventing estrogen from having its normal effects. Chemicals may stimulate the formation or release of estrogen or may induce enzymes that break it down. Endocrine disruptors may act on the hypothalamic-pituitary-adrenal axis, the thyroid, or sex hormone systems. For example, some PCBs and polybrominated biphenyls (PBBs) interfere with thyroid function, perhaps because of their structural resemblance to a thyroid hormone precursor.

DDT, an organochlorine insecticide, was the first endocrine disruptor identified. In the 1960s, researchers found that DDT interfered with reproduction in birds, causing them to lay thin-shelled eggs that broke during incubation. DDT and its metabolites are bioaccumulative compounds that are biomagnified in the food chain. As a result, fish-eating birds, such as bald eagles, brown pelicans, and ospreys, and bird-eating hawks, such as Peregrine falcons, suffered decades of reproductive failure due to DDT. Their populations recovered slowly after organochlorine use was greatly reduced and intensively managed. Among the multiple adverse effects on reproduction was induction of an enzyme that broke down both DDT and endogenous estrogens, thereby causing reproductive impairment.[31]

Endocrine disruptors have serious effects on aquatic wildlife, causing abnormal sexual development, sterility, and death of embryos.[32] In humans, endocrine disruptors adversely affect prenatal and infant development, especially of the urogenital system. Halogenated organic compounds, including polychlorinated dioxins and furans, disrupt male reproductive development, leading to undescended testes, hypospadias, and shortening of the penis. In the 1950s, diethylstilbestrol (DES), which was prescribed to sustain pregnancy in women who showed signs of impending miscarriage, resulted in many cases of vaginal cancer in their daughters. In addition, grandsons of these women have had an increased rate of hypospadias.[33] Precocious puberty in girls, occurring as young as age 7, has been attributed to endocrine disrupting compounds, such as phthalates and PBBs.

Occupational exposure to endocrine disruptors also occurs. Men engaged in the manufacture of birth-control chemicals have experienced feminization, with loss of hair, increased pitch of voice, and gynecomastia. Maternal occupational exposure to phthalates or hair sprays has increased their likelihood of giving birth to boys with hypospadias, although this risk has been modified by folate supplements during pregnancy.[34]

Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) comprise a family of over 100 organic compounds built around two to six aromatic rings. They are formed by the incomplete combustion of petroleum compounds, coal, natural gas, wood, garbage, tobacco, and even food. PAHs are components of coal tar, creosote, and asphalt. They are ubiquitous in urban environments, and detectable even in pristine environments due to atmospheric transport and deposition. PAHs with two to three rings are more volatile than those with four or more rings. They generally occur as mixtures. Benzo[a]pyrene (BaP), a carcinogenic PAH that typically comprises about 10% of PAH mixtures, is thought to be the primary carcinogen in tobacco smoke, charcoal-broiled meat, and chimney soot.

People are exposed to PAHs by inhalation, often from combustion of tobacco, wood, or gasoline, and by ingestion of food containing traces of them. PAHs are released from forest fires and volcanoes, and from automobile exhaust, heating systems, and industrial plants. Soils near roadways have PAHs that may be a source of exposure to toddlers and gardeners. Occupational exposures occur in mines; refineries; metal works; factories producing coal tar, coke, and asphalt; smokehouses; and other smoky environments.

Seventeen PAHs are widespread in the environment.[35] Several are carcinogenic, with BaP being the most widespread and best-studied. PAHs irritate the skin. They vary in solubility in water (although most have low water solubility) and volatility. However, they adsorb readily to

atmospheric particulates, enabling them to be transported long distances.

Coal-gas plants (manufactured gas plants) operated in many U.S. cities and towns from the late 1800s to about 1920. As they became obsolete, surface structures were removed and the land was leveled for residential development, covering over underground structures and pools of tar. PAHs, tar residues, and benzene and other volatile compounds eventually contaminated many homes that were built in those sites. Much remediation was performed in the 1980s and 1990s.

Polychlorinated Polyaromatic Compounds

Many isomers and congeners of polychlorinated biphenyls (PCBs) were produced and formulated into compounds (Arochlors) for commercial and industrial use. Depending on the number and location of chlorine atoms, PCB molecules can be flat (coplanar) or bent. PCBs demonstrate little acute toxicity, but, in animals, they produce a chronic syndrome with weight loss, chloracne, alopecia, skin edema, swelling around the eyes, shrinkage of the thymus and lymph glands, liver enlargement, bone marrow depression, and reproductive abnormalities. The syndrome is triggered through the aryl hydrocarbon (Ah) receptor. PCBs are well-documented animal carcinogens, causing liver cancer in several species, with the potency varying with degree of chlorination. PCBs are not genotoxic, but they can act as promoters of liver or lung cancer in animals. There is also evidence that they can cause cancer in humans.

A hallmark of exposure to PCBs and related compounds is *chloracne*. Chloracne lesions are indistinguishable from adolescent acne, but they are often dense and occur on more parts of the body, including the face, ears, abdomen, and scrotum. Chloracne is a cutaneous manifestation of systemic exposure. PCBs can cause hepatotoxicity, manifest by abnormal liver enzymes, but usually not by jaundice or clinically apparent disease. Women exposed prenatally to PCBs have had infants with lower birthweight, smaller head circumference, and impaired psychomotor and cognitive development.[36]

Although high blood levels of PCBs were widespread in the 1970s, the average concentration in blood has declined slowly since the imposition of restrictions on PCB production and use in the United States and Europe in the 1970s. In Yusho, Japan, in 1968, and Yu-Cheng, Taiwan, in 1979, epidemics of furan poisoning occurred, from contamination of rice oil with dibenzofurans, which were derived from PCBs that had leaked from heating coils into the oil. Adults suffered chloracne and acute liver damage, which was fatal in some cases. Fetal exposure was associated with abnormal urogenital tract development and developmental neurotoxicity, including reduced psychomotor performance and spatial learning and impaired cognitive development.

Dioxins and furans are unwanted by-products of chemical synthesis or breakdown. *Dibenzodioxins* have two benzene rings connected through two oxygen atoms; *dibenzofurans* have two benzene rings connected by one oxygen atom. There are potentially 74 different dibenzodioxins and 134 different dibenzofurans. 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), or *dioxin*, is considered to be the most toxic synthetic chemical. It was present as a contaminant in the manufacture of the commonly used herbicide, 2,4,5-trichlorophenoxy acetic acid (2,4,5-T), which comprised 50% of Agent Orange, which was widely used to defoliate forests in Vietnam.

Animal species vary widely in their susceptibility to TCDD toxicity. It affects many organ systems. Although it is not strongly genotoxic, it is a human carcinogen. Other dioxins and furans as well as some PCBs are given a toxic equivalency (TEQ) rating in comparison with TCDD. The natural combustion of wood yields dioxins and furans. Combustion of synthetic chlorinated compounds and application of chlorine bleaching compounds to wood yield chlorinated dioxins, such as TCDD, and chlorinated furans. The average background concentration of TCDD in body fat is about 4 parts per trillion (ng/g of lipid); in contrast, military personnel exposed to Agent Orange in Vietnam often had levels 10 times higher.[37]

Dioxins and furans can be ingested, inhaled, or absorbed through the skin; ingestion is generally the major route of entry. Dioxins can cause

chloracne, induce liver enzymes, and exert anti-estrogenic activity.

Organic Pesticides

CASE 4

A 38-year-old woman presented to the emergency department of a rural hospital complaining of a severe rash. The rash first appeared on her forearms several weeks before, and during the previous 2 weeks, had become more severe and spread to her face and neck. Itching kept her from sleeping for the previous 3 nights. She had no previous history of skin problems. She suspected that chemical exposures at work caused the rash. She worked at a greenhouse, where she had contact with pesticides, fungicides, fertilizers, and cleaning materials. Physical examination revealed a severe maculopapular rash on her hands, forearms, face, and neck. An emergency department physician treated her with topical steroids and an antihistamine, and gave her free samples of each and prescriptions for more medication when these ran out. He referred her to a community clinic for follow-up, where she was seen 2 weeks later. The rash was then still severe. She had taken the free samples provided at the emergency department, but she could not afford to fill the prescriptions.

The physician at the community clinic asked her about chemicals she used at work, but she could not name any. She did not apply pesticides or fungicides, but she was exposed to them when the greenhouses were sprayed before she arrived at work each day and when she handled flowers. The physician provided her with medication for her rash, advised her to return in 2 weeks, and asked her to try to get the names of the pesticides and fungicides that were used at work.

Soon after, the physician at the community clinic saw two more workers from the greenhouse who presented with recent onset of headache and nausea. He reported the cluster of these patients from the same workplace to the state pesticide enforcement agency. Inspectors toured the greenhouse, where they found problems with labeling of pesticides and disposal practices, but no serious violations of regulations. After the inspection, the owner of the greenhouse changed pesticide application procedures. All three patients recovered. When they returned to work, they were assigned to another area of the greenhouse, which did not have any hazardous exposures.

The worker in this case probably was acutely exposed to an organophosphate by inhalation (from pesticide fogging) and dermal absorption (from contact with pesticide-contaminated plants and surfaces). Once absorbed, organophosphates are metabolized by enzymes in the liver to form molecules that bind with acetylcholinesterase at cholinergic nerve endings, both centrally and peripherally. As a result, organophosphate poisoning causes a predictable constellation of muscarinic, nicotinic, and CNS symptoms. Typical symptoms include miosis, salivation, sweating, and muscle fasciculation. At high exposures, diarrhea, incontinence, wheezing, bradycardia, and convulsions may occur. Severe cases can be fatal.

Cholinesterase inhibition can be measured by determining cholinesterase levels, but these measurements are difficult to interpret because the normal range is wide, accuracy of laboratories varies widely, and cholinesterase levels may quickly return to normal after exposure ceases. Cholinesterase levels are most useful for ongoing monitoring, if baseline levels are known, and in monitoring recovery from acute toxicity. Acute poisoning can be treated with atropine, with or without pralidoxime. A delayed neurotoxicity syndrome, with weakness, paresthesias, and paralysis of the lower legs, may occur in people chronically exposed or severely acutely exposed to organophosphate pesticides.

Pesticides include insecticides, acaricides, molluscicides, nematocides, rodenticides, herbicides, and fungicides. They are designed to repel or kill living organisms ("pests"). Unlike most of the chemicals described above, they are used specifically because of their toxicity. Although most pesticide use occurs in agricultural settings, people may also be occupationally exposed from pesticide use for structural pest control.

Pesticides, as a class of chemicals, affect almost every organ system, but individual pesticides usually have more specific toxicity. Because most pesticides are toxic to people, new generations of pesticides are being designed to reduce

human toxicity. Pesticides kill or repel "pests." Research and development have long aimed at developing pesticides that are target specific, with low toxicity for nontarget organisms, especially humans. Compounds such as sulfur were used as pesticides more than 2,000 years ago. Many other pesticides also have a long history.[38]

Workers are exposed to pesticides during production of active compounds, formulation of commercial products, preparation and application (during mixing, loading, spraying, and cleaning equipment), and contact with treated crops (during inspection, weeding, and picking). Often agricultural field workers without special protection may be inadvertently exposed to nearby spray applications by wind drift. For many pesticides a *reentry time* (the time between application and when workers can, without protective equipment, enter a field to harvest the crops) of 1 to 7 days is specified. For many pesticides, efficacy requires achieving fine aerosols that can be easily inhaled. Exposure can occur by inhalation of vapors, dusts, or mists; dermal absorption; or ingestion—by eating food that has been sprayed or eating in the field without washing or changing clothes.

Fumigants

Fumigants are used to fumigate buildings, warehouses, ship holds, and soil to eliminate rodent and invertebrate pests. They are highly toxic to people. For example, methyl bromide, the use of which has decreased because of high toxicity and its contribution to ozone depletion, can acutely affect the nervous and respiratory systems and the gastrointestinal tract. Metam sodium, a soil fumigant, is less toxic and is replacing methyl bromide. However, a huge spill of metam sodium into the Sacramento River, in 1991, killed aquatic organisms for many miles and caused headache, nausea, and shortness of breath in people living along the river and in spill-response workers. Symptoms of irritant asthma persisted.[39]

Ethylene dibromide (EDB), a strong alkylating agent, was widely used to fumigate stored grain. EDB is readily absorbed through the skin and by inhalation or ingestion. It is highly irritating to mucous membranes and is hepatotoxic. It is an animal carcinogen and is considered a probable human carcinogen (IARC Group 2A). Workers involved in fumigating and those who prematurely entered fumigated structures were exposed. EDB was banned because of evidence of its carcinogenicity and the presence of significant residues in flour, cake mixes, and commercial baked goods.

Ethylene oxide (EtO), a highly reactive chemical, is used in chemical synthesis and in sterilizing reusable equipment and supplies in hospitals. Chemical operators and health care workers are exposed. It is considered a human carcinogen (IARC Group 1) and has been implicated in causing cancer of the blood and lymph systems.[40]

Carbon disulfide (CS_2) has been used to fumigate museum cases and agricultural products. Exposed workers in the rubber and rayon industries have developed acute psychoses at very high levels of exposure and cardiovascular disorders at intermediate levels. It causes a distal axonopathy.

Rodenticides

Rodenticides can have high specificity for rats and mice and can be incorporated into stable baits, some of which kill quickly without exciting suspicion or aversion by the animals. They may also be used against other mammalian pests, including gophers, squirrels, and coyotes. Other rodenticides contain the anticoagulant warfarin, which leads to a deficiency of vitamin K (necessary for blood clotting) and causes poisoned animals to bleed to death, but not before they have carried poisoned food back to their dens. These chemicals have a high potential to cause human poisoning, which may occur during production and formulation and in suicide attempts. Because they are highly toxic to people, rodenticides should only be used by trained personnel.

Insecticides

Insect pests are widespread in homes and on farms. A large variety of insecticides have been produced and formulated into hundreds of products. In *organic farming*, synthetic pesticides are not allowed, but naturally occurring compounds are permitted. Integrated pest management (IPM) uses ecological approaches such as interspersing different crops, introducing biological

controls, and selecting resistant strains, allowing crop production with little or no insecticide use.

Organochlorines (OCs) include *(a)* chlorinated ethanes, such as DDT; *(b)* cyclodiene chemicals, such as chlordane, aldrin, and dieldrin; and *(c)* saturated ring compounds, such as cyclohexanes (which include lindane). From the 1940s to the 1970s, OCs were widely used in agriculture and in programs to control human lice, structural termites, and malarial mosquitoes. As insects developed resistance, the value of the OCs declined. However, it was mainly their environmental and biological persistence that led to their elimination. DDT use in the United States was banned in 1972. Acute toxicity of OCs is lower than for the organophosphates, but higher than some pyrethroids. Some OCs have caused hepatotoxicity in formulation workers. They have been associated with Parkinson disease.[41] Chlordane was widely used as an agricultural insecticide until about 1978, after which its use became more restricted. From 1983 to 1988, it was frequently used around building foundations for termite control. Chlordane was banned in the United States for termite control in 1988 because of its carcinogenicity, environmental persistence, and bioaccumulation. Manufacture for export still continues.

DDT in talc was dusted on soldiers and others to kill lice, and lindane continues to be used for body lice control. DDT is still used in many developing countries for malaria control. DDT has low dermal absorption and low human toxicity when ingested. Acute exposure to high doses, as in suicide attempts, first manifests as a tingling sensation of the mouth and lips and progresses to muscle fasciculations and twitching, "jumpiness," involuntary movements, and tremors, which may progress to convulsions and death from respiratory failure. Chronic toxicity from DDT involves the liver. DDT is a potent inducer of liver enzymes, especially those in the CYP2B and CYP3A groups. DDT metabolites (DDD and DDE) cause liver and lung tumors in rodents, and DDT is considered a possible human carcinogen. DDT was probably the first widely recognized endocrine disrupting chemical. o,p-DDT activates the estrogen receptor and mimics estrogen, while its metabolite, p,p'-DDE, blocks the androgen receptor. However, in the long term, these effects are overridden by enzyme induction, which accelerates breakdown of estrogen.

Two other OCs are mirex and chlordecone. Mirex is an OC that has been used mainly to control fire ants in the southern United States. Chlordecone (Kepone) was used extensively to control cockroaches and other pests in homes, offices, and animal facilities. A chlordecone poisoning outbreak affected 148 workers in a Kepone factory in Virginia in the mid-1970s. Workers experienced a variety of effects, including reduced or absent sperm.

Organophosphates (OPs) include some nerve gases, such as sarin and soman, that have the highest toxicity to humans, potentially causing rapid death after inhalation or dermal exposure to very low concentrations. Introduced into agriculture in the 1930s, OPs became widely used by the 1950s and are now almost universally used in agriculture. They include hundreds of compounds that have been synthesized to improve specificity and overcome resistance. OPs act by binding to the enzyme acetylcholinesterase, thereby inactivating it and preventing it from breaking down the neurotransmitter acetylcholine at every synapse and nerve–muscle junction. Once this enzyme is inactivated, nerve impulses are unable to cross the synapse or nerve–muscle junction, thereby causing paralysis.

Some OPs are directly toxic to mammals, whereas others are oxidized to a toxic metabolite. Metabolism of OP is very complex and involves several enzymes. For example, CYP2C19 activates diazinon, CYP3A4 activates parathion, and CYP2B6 activates chlorpyrifos. Chlorpyrifos and diazinon, OPs with relatively low acute toxicity to humans, have been widely used. Chlorpyrifos largely replaced chlordane in household treatment for termites. Both of these compounds have been restricted because of concerns over chronic toxicity. In addition to removing exposure and decontaminating the exposed person's clothing and body, treatment includes pralidoxime (2-PAM), which removes the OP from acetylcholinesterase, restoring the active site to normal function. Exposure can be measured for some OPs by quantifying specific metabolites in urine, such as diethylphosphate, a metabolite of parathion, diazinon, and chlorpyrifos.

Pyridostigmine, which also protects acetylcholinesterase from OPs, was taken prophylactically by U.S. troops in the Persian Gulf War in 1991.

Children are more susceptible to OP poisoning, probably due to their slower metabolism. Children who accompany parents into farm fields or work alongside them are therefore at especially high risk.

Carbamates, such as carbaryl and aldicarb, are widely used insecticides that have variable human toxicity. Formulations containing solvents facilitate the dermal absorption of these compounds. Metabolism reduces the toxicity of carbaryl and increases the toxicity of aldicarb. Like OPs, these compounds are also anticholinesterases. However, unlike OPs, this effect is spontaneously reversible; recovery can occur within hours after an acute poisoning. Atropine, rather than 2-PAM, is used for treatment. The water-soluble aldicarb has high acute toxicity, but its anticholinergic effects are spontaneously reversible after a few hours. It is a systemic pesticide and has caused outbreaks of poisoning when used on hydroponic crops, such as cucumbers.

Pyrethrins, which are natural products produced by Chrysanthenum-related plants, are used to deter insects. Pyrethroids, such as deltamethrin and resmethrin, are synthetic analogs. Both pyrethrins and pyrethroids are effective, do not promote insect resistance, have low mammalian toxicity, and break down relatively quickly in the environment. Therefore, they are widely used in homes and fields. Pyrethroids have been medically used to treat lice and mites. These chemicals disrupt sodium channels, which are necessary for transmission of nerve impulses along an axon. They are rapidly metabolized in the body. There are very few documented cases of human poisoning or death.

Nicotine, an alkaloid concentrated in tobacco plants, has been used in some pesticidal applications. It activates the nicotinic receptors, a subset of acetylcholine receptors, producing an initial polarization followed by receptor paralysis. Acute poisoning, characterized by nausea and vomiting, weakness, rapid heart rate, and even death, is very rare.

Rotenone, used to kill insects and mites, rarely causes acute poisoning. However, it causes selective nerve damage in experimental animals that is conducive to Parkinson disease. It has been linked to Parkinson disease in humans.[42]

N,N-diethyl-m-toluamide (DEET) is an insect repellent that is widely used for personal application. Its use increased greatly after recognition of widespread West Nile virus transmission in the late 1990s. Formulations vary from weak (5%) to very strong (higher than 90%), with most in the 20% to 40% range. DEET is used to repel insects, fleas, and ticks when applied to skin and clothing. Higher concentrations offer longer protection. DEET is absorbed through the skin. It has low acute toxicity, but it has been implicated as a neurotoxicant in children, leading to the recommendation that children under age 12 should not use repellants with more than 10% DEET.

Herbicides, which are toxic to plants, are not likely to have high human toxicity but can have both local and systemic toxic effects. By absorbing and incorporating chemicals, plants may convey toxic chemicals from water and soil to consumers. Some herbicides inhibit photosynthesis, such as triazines, including atrazine. Some inhibit plant respiration. Some act as growth regulators, such as the phenoxy acids. Some inhibit protein or lipid synthesis or block enzymes, such as glyphosphate (Roundup).

Chlorphenoxy compounds include 2,4-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-T. These chemicals mimic growth hormone, producing lethal overgrowth in plants. They work in dicotyledenous plants, but not in monocots, such as grasses; therefore, these chemicals suppress broad-leaved weeds in lawns, without harming grass. Chlorophenoxy compounds have been associated with soft tissue sarcomas in exposed forestry workers. Agent Orange—which was a mixture of the butyl esters of 2,4-D and 2,4,5-T, contaminated with up to 50 μg/g of TCDD—caused adverse health effects, such as birth defects and cancer, among those exposed.[43]

Bipyridyl herbicides include paraquat, a nonselective, fast-acting contact herbicide used for weed control. It is highly acutely toxic to humans, but not genotoxic or carcinogenic. Inhalation of paraquat, during spraying or by smoking tobacco or marijuana sprayed with paraquat, causes lung toxicity. Paraquat has been implicated in many cases of acute human poisoning. It is irritating to the skin, but not well absorbed through the skin.

It also damages dopaminergic neurons and can cause or accelerate Parkinson disease, especially in combination with exposure to maneb, which contains manganese—another cause of parkinsonism.[44,45]

Triazines include atrazine, a widely used herbicide. Triazines have low dermal and oral toxicity. Atrazine is not genotoxic, but it causes mammary tumors in rats. Because of its adverse effects, including developmental defects in frogs, it has been banned in some European countries.

Glyphosphate kills plants, but it has very low toxicity for animals. This herbicide is widely used by homeowners in their gardens. A surfactant in a common formulation probably accounts for its irritant effects. Genetic engineering of crops to be resistant to glyphosphate allows this broad-spectrum herbicide to be used for weed control on farms.

Fungicides

Plant pathogens, such as viruses and fungi, are extremely difficult to control. Chemical fungicides are most effective when used prophylactically on plants, prior to infection by the fungal spores; however, some achieve cures after infestation, either on contact or systemically. They generally have low mammalian toxicity; however, some fungicides are genotoxic and contribute significantly to estimates of cancer risk from food residues. Fungicides are also used to treat wood and seeds to prevent mold growth. Methylmercury is a very effective fungicide, but it was banned after an outbreak of mercury poisoning in Iraq that was caused by ingestion of treated seed.[46]

Antineoplastic Drugs

Hospital employees involved in preparing and administering chemotherapy can be exposed to highly toxic drugs.[47] Antineoplastic drugs, which are cytotoxic,[48] can cause adverse reproductive outcomes, especially fetal loss during the first trimester of pregnancy, in nurses who mix these drugs. Use of fluorescent tracers has illustrated substantial opportunity for exposure during mixing. As a result, most hospitals require that mixing of these drugs be done in the pharmacy, not on the ward.

THE PRECAUTIONARY PRINCIPLE FOR NEW CHEMICALS

In 1998, a consensus statement described the *precautionary principle* as follows: "When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically."[49] The statement then listed four central components of this principle: *(a)* taking preventive action in the face of uncertainty; *(b)* shifting the burden of proof to the proponents of an activity; *(c)* exploring a wide range of alternatives to possibly harmful actions; and *(d)* increasing public participation in decision making.[49] Regulatory policy is a sluggish and reactive way to reduce worker and public exposure to chemicals and is fraught with delays over uncertainty.[8] Precautionary approaches provide an opportunity to identify potential hazards and limit exposure, before widespread exposure and harm has occurred.[50] The statement "Further research is needed" should never become a euphemism for failure to act.[51]

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12

Physical Hazards

This chapter describes physical exposures that occur over time that can cause human illness. These hazards transfer energy in a variety of forms, such as vibration, heat (into or away from individuals), electromagnetic energy, and increased or decreased atmospheric pressure. Noise, a physical hazard, is addressed together with hearing impairment in Chapter 21. Safety hazards, which result in the acute transmission of uncontrolled energy to a vulnerable individual, are classified separately because they result in instantaneous effects, or injuries, rather than illnesses—although the distinction is one of convenience and definitions may overlap (see Chapter 15). Biomechanical hazards, such as the repetitive lifting, stooping, and reaching that result in musculoskeletal disorders, are also addressed separately (see Chapters 16 and 27), although some people classify them together with physical hazards.

The underlying science that explores measurement and intervention to reduce physical exposures is based in physics, the study of the relationship between energy and matter. The sections included in this chapter address the physics of vibration, thermal stress (both hot and cold), and both ionizing and nonionizing electromagnetic radiation. These exposures are widespread in industry, nature, and various community and medical settings. Additional physical hazards not addressed here include low-pressure environments that may cause high-altitude illness or illness due to loss of pressurization in aircraft. High-pressure environments are encountered by those who work underwater; they are also used therapeutically for some medical conditions.

12A

Vibration

Martin G. Cherniack

Vibration is traditionally divided into whole-body vibration (WBV), having particular pertinence to vehicle seat design, and segmental or hand-arm vibration (HAV), affecting the hand and arm. For segmental vibration, health effects are usually related to energy transfer to the upper extremity from either powered tools or from stationery sources producing oscillatory vibration, such as mounted drills and pedestal grinders. There are parallel physical principles that apply to both sound and vibration, and, in the case of WBV, the psychological and physiological effects of sound and vibration are often intertwined. (For pertinent definitions, see Box 12A-1.)

Vibration is a complex physical exposure, which lends itself to electrophysical measurement. There are also well-accepted methodologies for exposure measurement and for the evaluation of human health effects. Chronic vibration from hand tools is associated with Raynaud phenomenon, a signature disorder of blood vessels, and mechanoreceptor dysfunction of peripheral nerves in the fingertips. There is both overlap with and distinction from other work-related musculoskeletal, neuromuscular, and neurovascular disorders that affect the upper extremity.

Adverse health effects from segmental vibration involving measurable neurologic and arterial dysfunction have led to the development of quantitative approaches to surveillance and industrial control. In 1986, the International Standards Organization (ISO) published methods for measuring segmental vibration and controlling its exposure.[1] The ISO standards were subsequently revised with similar, but even stricter, recommendations in 2001.[2,3] ISO standards 5349.1 and 5349.2 are central to consensus measurement of exposure. ISO standard 5349.1 defines vibration parameters, such as acceleration, a hand-oriented coordinate system, a hand-arm measurement filter, and a dose–response relationship. The American National Standards Institute (ANSI) adopted the current ISO approach in 2006.[4]

Consensus standards emphasize specific quantitative medical tests and the transformation of tool-based measurements into exposure limits and metrics for disease prevention. These accepted international approaches to measurement reflect the technical feasibility of translating the vibration from hand tools into physical principles: the frequency distribution of oscillations; the direction, velocity, and acceleration of those oscillations; and the impulsiveness, or force range, expressed in each impact cycle. Each of these physical characteristics has a bearing on symptoms and tissue injuries, especially in the palms and digits, but also more proximally in the shoulder and neck. Hand-arm vibration and

Box 12A-1. Definitions

Root mean square (RMS): The square root of the arithmetic mean of the squares of a series of numbers. RMS is a traditional way of reporting multifrequency magnitudes, such as tool acceleration.

Weighted curves: The progressive filtering, or downweighting, of accelerations as they exceed 16 Hz, commonly expressed as a_w. Vibration is usually reported as a weighted (filtered) or unweighted acceleration.

Acceleration: Time rate of change in velocity (expressed as m/sec^2 or as gravity); the second derivative of displacement with respect to time. Force from vibratory tools is usually expressed in terms of acceleration.

Frequency: Number of oscillations per unit of time; 1 hertz (Hz) = 1 cycle/second. Oscillatory vibration is defined as ≥125 Hz.

Raynaud phenomenon: A sometimes painful condition, affecting the fingers or toes, that is due to compromised circulation and is provoked by the cold. It causes the digits to turn white because of lack of blood supply. It is commonly divided into an idiopathic, or primary, condition and secondary Raynaud phenomenon. The responses associated with vibratory exposure—occupational Raynaud phenomenon or vibration white finger (VWF) disorder—are examples of secondary Raynaud phenomenon.

Mechanoreceptor: A sensory nerve organelle that records dimensions of touch, including gap, texture, and movement. The three best-known mechanoreceptors are the SAI (Merkel disc), FAI (Meissner corpuscle), and FAII (Pacinian corpuscle).

WBV are both called vector quantities, which means that vibratory motion has a direction and a magnitude or intensity. Magnitude is usually expressed as root-mean-square (rms) acceleration; direction is described by three linear motions (x, y, and z axes) and three rotational motions. For purposes of simplicity, only the three linear directions are made in studies for measuring exposure.

The approach to measurement for WBV is similar in principle, although exposure magnitudes and targeted frequencies are considerably lower. The association with health effects is, however, quite different. There is a general delineation between acute physical and psychological effects that involve loss of proficiency or fatigue and chronic health effects, the most important being low back disorders. Because low back disorders are common and have many causes, their association with WBV is more challenging to assess.

Segmental vibration does not exist as an independent phenomenon. Workers with hand-tool exposure are subject to the same combination of biomechanical and intrinsic risks that accompany other forms of hand-intensive work. As a consequence, the successful reduction of segmental vibration as a physical exposure may not reduce other disorders, which may complicate recognition and attribution.

SEGMENTAL (HAND-ARM) VIBRATION

A Brief History

The deleterious effects on the peripheral nerves and small vessels of the upper extremity from hand-transmitted vibration produced by power tools have been documented for almost a century. The clinical recognition and environmental control of HAV is based on the reduction of the most prominent sign and symptom complex: cold-related finger blanching (Raynaud phenomenon). In 1918, the pioneering occupational medicine physician Alice Hamilton first described this disorder in the United States among Indiana quarry workers using air-powered tools.[5] Subsequent studies have confirmed a strong effect between duration and intensity of vibration exposure and the onset of acquired neurologic and vascular symptoms.[6]

There is a variety of tool types and qualities of exposure that are associated with vibration-related upper-extremity disorders. The most recognized sources are air-powered rotary tools, such as grinders, sanders, and cutting wheels. However, gasoline-powered oscillating tools, such as chainsaws and brush cutters, have a classic association with disease in the forestry industry. The recent transition from air-powered to battery-powered tools, especially in construction, results in exposures having a narrower frequency range, but without necessarily reducing exposure risks.[7] Power tools, such as chipping hammers and pavement breakers, have greater impact (cycles that are defined by transient peak levels of force followed by rapid diminution). In the past three decades, characterization of the exposure–response relationship, disease identification, and anti-vibration tool design have helped to reduce exposure and cumulative

incidence of traumatic vasospasm and vibration white finger (VWF). By the mid-1980s, investigators had recognized that the typical vascular disorder could occur independent of injuries to fingertip mechanoreceptors, experienced as paresthesias and reduced hand function. Thus, the term hand-arm vibration syndrome (HAVS) came into general use, with the accompanying Stockholm Workshop Scale designed to independently assess vascular and neurologic effects.[8,9] The Stockholm Workshop Scale provided a consensus rating system for the two hallmark disorders: *(a)* vasospasm and disturbances of digital circulation, and *(b)* diffuse digital neuropathy. There has been less agreement about associations with other disorders, including *(a)* carpal tunnel syndrome (CTS), which is often confounded by biomechanical factors[10]; *(b)* a pattern of upper-extremity muscle weakness[11]; and *(c)* bone and joint disorders, such as traumatic osteoarthritis and problems with the elbows, shoulders, and neck, which are potential sites of energy absorption.[12] (See Chapter 16.)

Hand-arm vibration syndrome poses more than an incidental problem. In 1990, the National Institute for Occupational Safety and Health (NIOSH) warned that up to 2 million U.S. workers were exposed to vibration at magnitudes and frequencies sufficient to provoke injury with a symptom prevalence approaching 40%.[6] The general decline in metalworking in industrial countries, coupled with anti-vibration tools having damping and balancing features, has significantly reduced symptom rates in specific industries, most notably in forestry. In the longest established longitudinal study of vibratory disease, Finnish forest workers were surveyed cross-sectionally and as an inception cohort 11 times from 1976 to 1995.[13] Vibration exposures were reduced after the introduction of anti-vibration chainsaws. During this same interval, the prevalence of VWF declined from 40% to 4% of the population. From 1990 to 1995, VWF symptoms declined from 17% to 8% in the original population. The prevalence of hand and finger numbness from fell from 78% to 28% in 1990, then increasing to 40% in 1995; these symptoms were more suggestive of CTS. While the physiologic response to vibratory stimuli is only partly understood and the association of symptoms with the use of high-frequency oscillatory devices in medically related fields, such as orthopedics and dentistry, raise new areas of concern,[14–17] HAVS in traditional work settings is largely preventable.

This point is further confirmed by a longitudinal study of Italian and Swedish workers from 2003 to 2006, meant to assess inception of disease in young workers and progression of disease in those more chronically exposed.[18,19] At current reduced levels of exposure, there was limited progression of abnormal vascular findings in Italian forest and stone workers. A sample of young Swedish workers had very subtle changes in quantitative sensory tests, but limited morbidity.

Exposure and Its Measurement

Vibration is a physical factor, expressible in precise units: frequency in Hz, acceleration in sec^2 (g's), and cycles in milliseconds. This offers highly accessible measurement with available instrumentation, principally accelerometry and frequency spectrum analysis. The ISO has taken a consensus regulatory approach to tool design by assuming that a relationship exists between the relative hazards presented by vibration at different frequencies. This is achieved by *(a)* frequency weighting of the acceleration time history, $a_w(t)$, and *(b)* temporal summation, by means of energy averaging over the duration of an 8-hour work shift. Customarily, frequency-specific acceleration is reported at one-third octave bands and downweighted above 16 Hz, so that frequencies above 250 Hz have minimal effect on exposure calculations. (NIOSH had recommended that this downweighting be abandoned.) The implications are significant. For example, a small burring machine that produced a frequency-weighted acceleration of 5.6 m/sec^2 was found to have an unweighted acceleration of 146.8 m/sec^2.[20] Appreciating that these differences are substantial is not a critique of international standardization, but it helps to explain why future scientific controversy is likely.

The exposure–response relationship presumed by the ISO is represented in Figure 12A-1. However, translating exposure from laboratory measurement into the work environment is not straightforward. Vibratory characteristics are

(Drawing by Nick Thorkelson.)

highly tool specific. Chainsaws and drills, for example, are primarily oscillatory and continuous-energy tools. Impact wrenches and rivet guns have large physical displacements and are highly impulsive. Tools such as nut runners have major nonvibratory biomechanical features. Therefore, simple generic measurements may not capture the extent of a potential tool-specific hazard. Tool-specific characteristics also influence the approach to exposure control. While personal protective equipment (PPE) is not a first-line approach to industrial control, some anti-vibration gloves are particularly effective at exposure frequencies >150 Hz, where the greatest risks of tissue injury are likely to occur. The addition of appropriate gloves adds an additional level of protection.[21]

Frequency, direction of vibration, and the position of the arm and hand all have an effect on impedance to and absorption of vibration energy. Push and pull as well as grip force affect transmission.[22] Perhaps the most problematic area involves high-impulse acceleration. The ISO and American National Standards Institute (ANSI) weighted curves treat all vibration as harmonic, ignoring impact forces and instantaneous peak accelerations that may exceed $10^5 m/sec^2$ (10,000 g's). The dramatic reduction in vascular symptoms occurring with the introduction of anti-vibration chainsaws in the 1970s was better explained by the flattening of high transient accelerations than by a reduction in rms.[23] Also vascular symptoms, which were consistently underestimated by ISO 5349.1 for pedestal grinding and stone cutting, were better accounted for when high-peak impulsivity was factored into the exposure model. This is consistent with, but does not fully explain, the high prevalence of VWF in platers and riveters, who use high-impulse tools for only a few minutes a day.[24] A similar problem arises in the setting of tools that oscillate at very high frequencies, such as small precision drills and saws. Most measurement protocols exclude frequencies that exceed 1,500 Hz. There are sound reasons for this: *(a)* energy transfer is directly related to velocity, so that its first derivative, acceleration, is seemingly discounted at high frequencies; and *(b)* there is no physiological evidence that the Pacinian corpuscle, the principal fingertip mechanoreceptor, responds above 1,500 Hz. Nevertheless, neurologic and vascular symptoms have been highly concentrated in select working populations using these types of tools.

Perhaps because of these complexities, the European Union has advanced practical regulations[25] that set a low action level for vibration of $a_w(t)$ at 2.5 m/sec^2 and daily exposure limit of 5.0 m/sec^2. The CEN (European Commission for Standardization/Comité Européen de Normalisation) approach emphasizes tool design and exposure control and is based on expert opinion, and more loosely on epidemiology, to reflect feasibility and a presumed modest risk of disease in less than 10% of those exposed. This simplifies the dimensions of human exposure and sets a threshold, below which disease is expected to be uncommon. A caveat articulated by those familiar with the European system is that single-value exposure limit thresholds cannot be a substitute for a full array of preventive measures, which include warnings, health surveillance, instruction on the proper use of tools, tool maintenance, restricting daily maximum use, selecting appropriate gloves, and addressing the more general work environment.[26] While member states within the European Union have until 2013 to phase in these regulations, they already apply to manufacturers selling new tools within the EU.

Pathology

Sensorineural symptoms in the hands are the most common clinical presentation in industrial workers exposed to vibratory tools. Sensory nerve conduction velocity (SNCV) in the digits of vibration-exposed symptomatic workers is slowed, especially in proximal segments.[27,28] These deficits in the function of distal myelinated nerve fibers are distinct from the characteristic slowing in the wrist-palm segment that occurs in CTS. Injuries to small nerve fibers in the fingertips are distinct from demyelinating nerve compression disorders, such as CTS, that affect larger nerve fibers. When changes in tactile function associated with occupational use of vibratory tools are examined in each of the three mechanoreceptor populations at a fingertip—Meissner corpuscles, Merkel discs, and Pacinian corpuscles—the chronic changes in thresholds, determined by vibrometry, occur differentially among the three populations (Table 12A-1).[29] It appears that at least transient deficits in mechanoreceptor function occur at higher oscillatory frequencies (125 or more Hz) than are emphasized in international standards. Mechanoreceptor dysfunction impairs both sensation and muscular responses, such as grip.

The hallmark symptom associated with segmental vibration, digital vasospasm, predominates in the hand most exposed. However, companion vasoconstriction in the hand not exposed to vibration suggests central sympathetic mechanisms affecting peripheral blood vessels.[30] The characteristic clinical finding is a local blanching of the skin of the fingers that is well demarcated from surrounding skin and occurs in paroxysmal attacks, usually induced by exposure to cold. Dramatic reductions in digital artery diameter provoked by cold, measured either by ultrasound or plethysmography, present similarly, regardless of whether Raynaud phenomenon is work-induced, idiopathic, or secondary to a systemic collagen vascular disease.[31] Sensitive and specific tests include plethysmographic measurement of cold-induced arterial effects through the use of *(a)* strain gauges that measure finger systolic blood pressure (FSBP) and *(b)* locally applied cold provocation.[32] Although intimal injury has been identified in finger biopsy on a few advanced cases,[33] this is exceptional; arteriography and baseline noninvasive studies are usually normal. Thus, pathology is best understood in physiologic terms.[34]

There are two limitations to this type of measurement: *(a)* specialized devices are uncommon, and more conventional ice-water baths are unreliable; and *(b)* plethysmography measures full finger perfusion, which may be intact in nutritive capillaries while cutaneous blood flow is reduced. Thus, laser Doppler, a method which primarily measures cutaneous flow, produces significantly more abnormal tests than plethysmography,[35] and, in subjects with VWF, blood pressure appears to normalize in the setting of cold stress, whereas skin temperature (cutaneous perfusion) and symptoms remain unchanged.[36] Accordingly, *(a)* conventional noninvasive vascular measurements, such as Doppler, pulse volume, and photoplethysmography, are without value in the absence of controlled cold provocation; and *(b)* crude immersion challenge with measurement of skin temperature is not an adequate procedure.

Clinical Presentation and Diagnoses

The hallmark sign of clinical disease related to HAV is a well-delineated patchy blanching of the fingers, occurring at sites of greatest exposure,

Table 12A-1. Mechanoreceptors and Their Function

Type	Property	Mechanoreceptor	Location	Innervation	Sensation	Peak Response (Hz)
A- Beta	Slow adapting SA I	Merkel neurite	Basal layer epidermis	Single nerve	Fixed touch Edge and gap/Braille receptor	4–8
A- Beta	Fast adapting FA I	Meissner corpuscle	Dermal papillary ridge	Multiple nerves	2-point Slippage 5 mm gap	26–32
A- Beta	Fast adapting FA II	Pacinian corpuscle	Deep dermal and subcutaneous	Single nerve (lamellar)	Movement and pressure	125

following exposure of the hands or the whole body to a cold environment. Signs and symptoms of VWF are a subset of those designated by the acronym HAVS. They include the following:

1. White fingers
2. Peripheral neuropathy, with or without increased cold sensitivity
3. Distal compressive and demyelinating neuropathies of digital nerves, the median nerve at the carpal tunnel, and, less plausibly, the ulnar nerve
4. Musculoskeletal disturbances, such as weakness, lancinating forearm pain, and bone and joint degeneration

Finger blanching and small-fiber neuropathy are fairly specific, but nerve compression disorders and musculoskeletal pain and joint degeneration are nonspecific. Diagnosis of a case of HAVS is based on clinical assessment: a history of exposure and complaints, physical examination, and, often, laboratory studies. To some extent, the diagnosis of HAVS is exclusionary, since peripheral nerve dysfunction and Raynaud phenomenon can reflect serious underlying disorders that are unrelated to exposure to vibration.

Even classic VWF can be problematic because pathology may arise in either the arterial or cutaneous circulation. In addition, temperature sensitivity and hand weakness may be representative of injury to small nerve fibers or to compression-related conduction block. In cases where CTS has been ruled out, a finding of diffusely distributed reduction of skin sensitivity and/or paresthesia and an increased sensitivity to cold are more likely due to mechanoreceptor injury. This type of injury, in contrast to typical CTS, is aggravated acutely by exposure to vibratory tools, is less intermittent, and is induced by arm position. The association between segmental vibration and upper-extremity osteoarthritis is more ambiguous; bone and joint changes are very difficult to differentiate from the effects of other ergonomic hazards or aging.

In patients with early symptoms of classical VWF, there is the possibility of other, even multiple, pathologies. These patients often present with symptoms that are more diverse than is anticipated by conventional diagnostic criteria. Their complaints may be due to vibration damage or other pathology but also to variants of normal vascular physiology. In an industrial population already self-selected by hand-intensive work, it is relatively uncommon to uncover underlying systemic diseases, such as thyroid disease, diabetes, early presentations of collagen vascular disease, or vascular symptoms attributable to circulating cold agglutinins or cryoglobulins.

Reduction of exposure magnitude through tool design or transfer of individuals showing symptoms, or even a subclinical stage of the disorder, has produced a shift in the tenor of differential diagnosis. As vibratory exposures are contained and job tasks diversified, pure HAVS cases may be replaced by more obscure presentations. Because there are recoverable components, diminished exposure can produce a pattern where the incidence of new symptoms is balanced by symptom recovery. This changes the pattern from a purely exposure–response disease and toward a cumulative trauma disorder.

In a setting where exposures to HAV are high or where there is a need for legal-administrative criteria, the accent is necessarily on HAVS-specific medical history questions and on tests that are also specific to vibration-related disease, such as small-fiber nerve tests and cold-challenge tests. However, exposure control, longer employment duration before symptom onset, and the conflicting presence of biomechanically induced or naturally occurring injury change the context of diagnostic testing. A lower prevalence of hallmark HAVS sequelae and a higher prevalence of mixed disorders require a broader and nonexclusionary approach to differential diagnosis. These issues are demonstrated in the two cases presented next.

CASE 1: TYPICAL CASE OF HAND-ARM VIBRATION SYNDROME

A man who had worked 27 years as a shipyard welder, having worked 2 to 3 hours a day on a pneumatic grinder and needle gun, had a 10-year history of cold intolerance and a 5-year history of blanching of his fingers when the temperature was less than 10°C. He had nocturnal paresthesias in all of his fingers and

paresthesias at work, resolving 4 to 8 hours after a work shift. He also noted diminished grip strength and clumsiness while attempting fine coordinated movements. On physical examination, he had a positive Tinel sign, cold hands, and diffuse musculotendinous pain. Both his antinuclear antibodies and rheumatoid factor were negative. Nerve conduction velocity revealed a sensory latency of 4.4 m/sec (normal: <4.4 m/sec) in both the right and left distal median nerve. Grip strength was 30 kg for the right hand and 25 kg for the left hand. Two-point discrimination was 5 to 6 mm (normal: 6–7 mm). Monofilaments were perceived at 0.16 gm (normal: ≤0.07 gm).

CASE 2: ATYPICAL CASE OF POSSIBLE HAND-ARM VIBRATION SYNDROME

A man who had worked 15 years as a carpenter, with intensive use of electric sanders, drills, and saws, had a 5-year history of cold intolerance with no clear blanching of his fingers. He tended to avoid cold exposure whenever possible. He had nocturnal paresthesias and moderate paresthesias at work with pinching of his thumb and second and third fingers. All the time at work, however, he had mild paresthesias in all fingers. He also noted hand cramps with painless claw-like rigidity and diffuse joint pain. On physical examination, he had a positive Tinel sign at the wrist and elbow, a positive Adson sign, and decreased range of motion at the wrists. He had arthralgias in his carpometacarpal joints and lateral epicondylar pain. Nerve conduction velocity tests revealed a sensory latency of 4.7 m/sec^2 in the distal right median nerve and 4.2 m/sec^2 in the distal left median nerve, and 4.1 m/sec^2 in the distal right ulnar nerve and 4.2 m/sec^2 in the distal left ulnar nerve. His grip strength was 25 kg in his right hand and 42 kg in his left hand. Two-point discrimination was 5.6 mm, and monofilaments were 2.0 gm.

In Case 1, there was a classic pattern of long exposure to air-powered tools, typical cold-induced finger blanching, a pattern of hand paresthesias, and dysfunction that was not clearly attributable to nerve compression. In Case 2, exposures were more diverse and there was a strongly suggestive pattern of CTS. The raised sensory latency, diminished strength in the dominant hand, and nighttime paresthesias and positional relief were all typical for median nerve compression at the wrist. Also present were the type of chronically acquired musculoskeletal injuries, such as epicondylitis and proximal nerve compression, that occur more commonly in manual work and may have a waxing-and-waning course. In Case 2, HAVS cannot be assumed, which raises an issue of specialized diagnosis.

While clinicians experienced in the treatment of neuromuscular disorders may regret the insufficiencies of diagnostic tests, HAVS is distinguished from other occupationally related upper-extremity disorders because of the availability of signature tests, such as *(a)* cold-challenge plethysmography or other cold-provocation tests for the diagnosis of Raynaud, and *(b)* quantitative sensory tests (QSTs) for detecting small-fiber neuropathies. Nerve conduction studies, used in the diagnosis of work-related entrapment neuropathies, are notably less specific and less physiologic in their application to this type of injury. However, specialized tests are not generally available, and it is an error to proceed to specialized functional tests in the absence of judgment based on clinical anamnesis and a detailed physical exam. An approach to evaluation is summarized in Box 12A-2. The extensiveness of the test battery will depend on several considerations, including the clinical presentation, medico-legal criteria, and the likelihood that a patient will be available for sequential follow-up. Therefore, the list can be regarded as either complete or contingent.

In the classical case of white fingers and neurologic disturbances due to damage of small nerve fibers, there are few therapeutic options. Therapeutic interventions are available, but they should be provided only under experienced clinical supervision.

The goal of rehabilitation should always be to transfer the affected worker to an alternative job, where hands are not exposed to hand-transmitted vibration. In such a situation, disability assistance services would also come into play. Increasingly, the combination of anti-vibration tools with gloves may enable some workers with mild or quiescent symptoms to return to work with reduced exposure. These decisions should

Box 12A-2. Standard Elements in Diagnosing Hand-Arm Vibration Syndrome

- Medical history, including an occupational history
- Physical examination of the upper extremity focused on vascular, neurologic, and musculoskeletal signs
- Provocative clinical tests: Tinel (wrist and elbow), Adson, Roos, and Wright
- Standardized and exclusionary criteria for clinical diagnosis of proximal disorders of the shoulder and neck
- Tests of individual muscle strength and pinch and grip strength
- Electromyographic studies, cold-challenge tests (finger systolic blood pressure), and quantitative sensory tests (vibro-tactile thresholds [VTTs] and temperature thresholds), as indicated
- Radiographic imaging of cervical spine and shoulders, as indicated
- Blood tests (complete blood count, sedimentation rate, blood viscosity, glucose, uric acid, rheumatoid factor, cryoglobulins, serum protein electrophoresis, and immunoglobulins), as clinically indicated.
- Autoimmune serology (such as antinuclear antibodies, anti-DNA antibodies, antinucleolar antibodies, anticentromere antibodies, ENA [extractable nuclear antigen] antibodies, and anticardiolipin antibodies), as clinically indicated.

be made with caution, and on an individual basis.

WHOLE-BODY VIBRATION

Whole-body vibration is transmitted to the anatomic supporting surfaces, especially the legs when standing and the buttocks and back when sitting. A major emphasis for risk evaluation and remediation has been on vehicular seating in forklifts, construction vehicles, and off-road vehicles. While transient alterations in psychomotor, physiological, and psychological function have been attributed to WBV, the greatest concerns and controversies involve chronic low back pain. Common sources of WBV are listed in Table 12A-2.

Table 12A-2. Sources of Whole-Body Vibration

Activity	Source
Warehousing and material handling	Forklifts
Construction	Cranes, power shovels, bulldozers, off-road trucks, and tractors
Farming	Tractors
Transportation	Subways, buses, trains, helicopters, and tractor-trailers
Buildings	Subway and rail vibration, and ventilation systems

Exposure and Its Measurement

The principles involved in measuring WBV are analogous to those employed for segmental vibration. The control of human exposure is dependent on frequency, magnitude (expressed as rms acceleration), and duration of contact. An ISO standard sets reproducible conditions for measurement and prevention.[37] There is a separate ISO standard that addresses sound and vibration coming from rail systems.[38] As is the case for segmental vibration, measurements proceed in three orthogonal directions; however, the planes are adapted to the trunk, rather than the hand and arm. Measurements include one longitudinal direction (buttocks to head, or az), and two transverse directions (chest to back, or ax; and right to left side, or ay). The standard provides numerical limits for exposure to vibrations transmitted from solid surfaces to the human body in the frequency range of 1 to 80 Hz. This is a critical distinction, differentiating WBV from HAVS, for which ISO recommends frequency measurement through 1200 Hz.[1] The European Physical Agents Directive[25] sets, in member states, exposure limits, which were begun to be enforced in 2005. They are as follows:

- A daily exposure action value of 0.5 m/sec^2 (or, at the choice of the EU member state, a vibration dose value of 9.1 m/sec$^{1.75}$)
- A daily exposure limit of 1.15 m/sec^2 (or, at the choice of the EU member state, a vibration dose value of 21 m/sec$^{1.75}$)

Individual member states may have more restrictive limits. There is no standard for HAV or WBV in the United States.

Health Effects of Whole-Body Vibration

The lower frequencies characteristic of WBV involve potential resonant frequencies affecting

the musculoskeletal system. Parts of the human body have their own resonant frequencies and do not vibrate as a single mass. The resulting response to exposure is complex, since amplification or diminution of the vibratory input will be affected differentially by different parts of the body due to intrinsic resonant frequencies. The most effective exciting frequency for vertical vibration delivered to the feet or buttocks occurs at 4 and 8 Hz.[39,40] Vibratory frequencies between 2.5 and 5.0 Hz generate strong resonance in the vertebrae of the neck and lumbar region with amplification up to 240%. Between 4 and 6 Hz, resonance in the trunk may be doubled. Vibrations between 20 and 30 Hz set up the strongest resonance between the head and the shoulders, with amplification up to 350%. In a human body, this may create chronic stresses and sometimes even permanent damage to the affected organs or body parts.[41] Thus, principal health effects are expected and measured at frequencies that are below those of hand-arm exposure.

Not surprisingly, the greatest attention to harmful effects from WBV in the workforce has been directed to low back disease in vehicle drivers. The European Agency for Safety and Health at Work consolidated several national surveys indicating that the range of WBV exposure from commercial vehicles ranged from 5% to 25%, primarily in men.[42] It has concluded that the action level for WBV in the Physical Agents Directive would be exceeded by drivers of most off-road machines and by drivers in the agricultural and forestry tractors. In contrast, it concluded that the limit value would be rarely exceeded except for bulldozers and dumpers as well as forklift operators working on rough surfaces. This conclusion appears to have been confirmed in accompanying epidemiological studies in Italy, Sweden, the Netherlands, and the United Kingdom.[43] Taxi drivers, police, and on-road truck drivers rarely had exposures that exceeded the action level, and limits were exceeded in special cases, such as in surface mining and with loaders. Musculoskeletal symptoms, including low back pain, were associated with duration of driving, but they were not consistently associated with WBV level. Studies of truck drivers alone have also been inconclusive concerning whether there is a causative association between WBV and low back pain.[44]

Beyond the focus on low back disease, the ISO standard on WBV exposure is directed to three different areas of health effects: reduced comfort, fatigue-decreased proficiency, and exposure limits. The reduced comfort boundary is applicable where passenger comfort is of concern, such as on trains, subways, and buses. The fatigue-decreased proficiency boundary is applicable to situations where maintaining operator efficiency of a vehicle is of concern, where, for example, work demands include reading gauges and screens, performing fine manipulation, and maintaining cognitive function. The comfort boundary and the fatigue-decreased proficiency boundary are terms used by ISO to define specific thresholds where rider comfort is no longer preserved and where operators/drivers demonstrate decreased performance. Exposure limit, which is established by the ISO at twice the fatigue-decreased proficiency boundary, applies to situations where worker health and safety may be compromised by chronic injuries to the back, neck, and internal organs.

Fatigue-decreased proficiency and exposure are the outcomes of primary interest in occupational settings. The exposure limit is calculated by multiplying accelerations by 2. For an 8-hour work shift, rms acceleration of 0.315 m/sec^2 is the upper boundary if fatigue-decreased proficiency is the criterion, and 0.63 m/sec^2 if health effects determine an exposure limit. These limits are often exceeded under field conditions when earth-moving equipment is used.

Databases on WBV exposure from industrial vehicles are available online.[45] Profiles based on new equipment are misleading. Vehicle age and maintenance, the terrain, seat and cab design, and the presence of other vibrating equipment on the vehicle also determine the WBV magnitude of exposure in field settings. Exposure from WBV is not the only factor adversely affecting equipment operators.[46,47] Awkward sitting postures often required by drivers can adversely affect back health. Drivers must also resort to side and outside views and drive backwards, forcing them to adopt twisted postures. Depending on shift and overtime schedules, drivers may work continuously for over 12 hours. Awkward postures, repetition, long duration, and/or forceful exertion are considered risk factors for the development of musculoskeletal disorders

(see Chapter 16). In addition, poor ergonomic designs of cabs and seats as well as inaccessible control gear, such as pedals and steering wheels, will adversely affect workers. Cab and seat design modification most commonly address acceleration to the spine in az axis. However, chest-back and side-side exposures are expected to produce more adverse effects at similar levels of exposure.

The damaging effects from WBV on other organ systems are less clear. One study demonstrated reduced gastric motility following exposure on a vibrating platform, but the magnitude of exposure (2 m/sec^2) and the frequencies (10 to 40 Hz) were relatively high.[48] More serious concerns have been raised about fetal injury, based on exposures to pregnant animals.[49] Although there is no commonly accepted evidence of pregnancy risk in humans, several European countries place vibration exposure restrictions on pregnant women.

Vibration in Buildings

One of the most important concerns with WBV includes low-level exposure in buildings. Other human health effects are presumed to be subtle and largely psychological or psychophysical. The extent of the exposed population and the importance of cognitive acuity make this an area for targeted investigation. Vibration in buildings can be constant, intermittent, or impulsive, and it can occur as typical vertical WBV from the floor or, more commonly, as a composition of gross structural vibration and reaction. Resonance occurs at very low frequency in tall buildings (1 Hz), but wood-framed walls may resonate at 10 to 25 Hz, and masonry at 50 Hz. Current ISO and ANSI standards set exposure limits well below the point where WBV is expected to produce fatigue or generate loss of proficiency; but in residential buildings, vibration often proves irritating just above the level of perception.

Prevention and Remediation

The control of WBV rests more on selection of the appropriate measurement standard and prophylactic design rather than on health surveillance or medical evaluation. For example, in transportation the use of absorbent or "air-ride" seats reflects a focused effort to reduce vertical vibration. However, more sophisticated designs that provide front-back attenuation, cab isolation, and vehicle suspension represent a more effective integrated approach. In the industrial environment, anti-shock mounting of machinery, remote manipulation, and vibration-isolated pulpits provide additional measures of protection. Independent of etiology, in construction and earth-moving, low back pain can be alleviated by seat and cab design. Even if the origins and pathology of low back pain remain unidentified, a work environment protected against WBV can reduce symptoms and preserve function.

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12B

Extremes of Temperature

Ann M. Krake

This section of the chapter describes extremes of temperature, their adverse health effects, evaluation and assessment, and strategies for prevention of heat- and cold-related illnesses. Box 12B-1 details physical hazards related to hyperbaric and hypobaric environments and their adverse health effects.

HOT ENVIRONMENTS

Environmental Heat Stress and Heat Strain

CASE 1

An 86-year-old woman was found unresponsive in her bedroom. She had no known medical history, but her grandson reported that she kept her bedroom windows closed for the previous 7 days during a heat wave. She had no fan or air conditioning. A rectal temperature taken at the hospital was 42.2°C (108°F). She had died of heat stroke.

The Centers for Disease Control and Prevention (CDC) has reported that an average of about 700 people die each year in the United States from exposure to excessive heat in work, home, and community settings.[1] About half of such deaths are "due to weather conditions."[2] The CDC's most recent analysis suggests that the number of reported heat-related deaths underestimates the actual number. The analysis counted both hyperthermia (abnormally high body temperature) as an underlying cause of death on the death certificate and also counted hyperthermia as a contributing factor. Including both indicators increased the number of heat-related deaths by 54%.[1] Extreme heat continues to account for 20% of deaths due to natural hazards in the United States, more than any other specific cause combined.[3]

Heat waves are defined as consecutive days of air temperature 32.2°C (90°F) or greater. In the United States, those who live in the South and in the Great Plains/Midwestern states are most at risk during heat waves.[2] During the 1990s, two heat waves struck Chicago, killing over 1,000 people. Hundreds died in Europe during the summer of 2003, with early onset of hot weather, unusually high temperatures, and prolonged heat-stress conditions.

Elderly people are most vulnerable to extreme heat. Of the 522 deaths that occurred in Chicago during the July 12–16, 1995, heat wave, 371 (73%) were age 65 or older. In Rome in 2003, the greatest mortality increases were also seen in people age 65 years or older and living in the most economically disadvantaged areas. Other factors that may have contributed to mortality

Box 12B-1. Physical Hazards Related to Hyperbaric and Hypobaric Environments and Their Adverse Health Effects

John Halpin

The physiology of the human body is well suited and well adapted to functioning at an atmospheric pressure equal to 760 mm Hg. This pressure has been designated as a standard reference point of 1 Atmosphere Absolute (1 ATA). Above or below this pressure (under hyperbaric or hypobaric conditions), the body is subject to various physical hazards due to the change in pressure, or as a direct result of high or low pressure.

Hyperbaric environments are most commonly encountered in a diving setting, but they include any situation in which compressed air is required, including caisson operations, underwater tunneling, and tending patients in a hyperbaric chamber. The most common health problem occurring in hyperbaric environments is known as barotrauma. It involves an imbalance in pressure of air cavities and sinuses within the body as they are subjected to an acute change in pressure. A classic example is middle-ear barotrauma, in which a pressure imbalance develops between the middle ear and the external ear canal, causing tympanic membrane trauma and acute ear pain. A related, but much more serious, form of barotrauma is known as pulmonary overinflation syndrome, in which the lungs become overinflated due to expansion of the air within them during ascent and reach a breaking point at which alveolar capillaries rupture. This can result in pneumothorax, mediastinal emphysema, and arterial gas embolism. Arterial gas embolism, resulting from the introduction of compressed air into the arterial bloodstream, has various neurologic manifestations, including confusion, weakness resembling a stroke, and loss of consciousness.

While functioning in hyperbaric conditions, the lungs absorb copious nitrogen, in the form of dissolved gas in the blood. When ambient pressure is reduced rapidly upon ascent, nitrogen can reform into gas bubbles in the blood and lead to impaired circulation. The resulting array of clinical symptoms is commonly known as decompression sickness (DCS). Pain alone, typically in the joints and muscles, is a less serious form of DCS, which often occurs about 4 to 6 hours after ascent from depth, and is known as Type I DCS. Involvement of the neurologic and cardiopulmonary systems represents a more serious form, Type II DCS. It can manifest as paralysis, severe cough, and severe shortness of breath, and it may be fatal. It requires immediate oxygen therapy and recompression in a hyperbaric chamber.

Two other disorders related to hyperbaric environments occur as a direct result of the increased partial pressure of gas at depth: nitrogen narcosis and oxygen toxicity. At increased partial pressures, nitrogen can exert a narcotic effect—similar to the effects of alcohol intoxication, which increases as depth increases. Even at 1 ATA, but especially under increased partial pressure at depth, oxygen can become neurotoxic, with paresthesias, tinnitus, visual changes, confusion, nausea, vertigo, and seizures. Preventive measures in hyperbaric environments include managing time spent at depth and carefully controlling the rate of descent and ascent. More specific recommendations can be found by referring to dive tables and instructions at the Divers Alert Network (http://www.diversalertnetwork.org) and National Association of Underwater Instructors (NAUI) (http://www.naui.org).

Hypobaric environments are commonly encountered by those in high-altitude mountain settings as well as pilots and passengers in unpressurized aircraft. At altitudes greater than 2,500 meters (about 8,000 feet) the partial pressure of oxygen becomes significantly reduced such that altitude-related illnesses can begin to occur, especially in those who have not acclimatized by ascending too rapidly. The most common form of altitude illness, known as acute mountain sickness (AMS), is characterized by headache, nausea, vomiting, fatigue, and loss of appetite. It is thought to occur due to an imbalance between hypoxic-induced cerebral vasodilation and hypocarbia-induced cerebral vasoconstriction. It is best treated with administration of oxygen while lowering the victim to an altitude where symptoms resolve. It can be prevented by use of acetazolamide and dexamethasone.

A more serious manifestation of altitude illness, high-altitude pulmonary edema (HAPE), affects the lungs. It is believed to occur as a result of hypoxic-induced vasoconstriction and pulmonary capillary leakage. Onset is usually insidious and occurs within 24 to 60 hours of arrival at high altitude, with initial symptoms including shortness of breath, cough, weakness, tachycardia, and headache, which may progress to cough productive of bloody sputum, low-grade fever, and pulmonary congestion. If untreated, coma may ensue as a result of the most serious manifestation of altitude illness, high-altitude cerebral edema (HACE). Prior to coma, severe headache, confusion, ataxia, or hallucinations may indicate that cerebral edema may be occurring. As with AMS, descent with administration of oxygen is the best form of treatment for both HAPE and HACE, although symptoms may also respond to the use of dexamethasone. Preventive measures for hypobaric environments include proper acclimatization, along with an understanding and recognition of symptoms that indicate that descent is warranted. More specific recommendations can be obtained from the International Society for Mountain Medicine (http://www.ismmed.org).

Further Reading

Tetzlaff K, Thorsen E. Breathing at depth: physiologic and clinical aspects of diving while breathing compressed gas. Clinics in Chest Medicine 2005; 26: 355–380.
An excellent recent review of the physics, pathophysiology, and clinical features of breathing in a hyperbaric environment.

Lesham E, Pandey P, Shlim DR, et al. Clinical features of patients with severe altitude illness in Nepal. Journal of Travel Medicine 2008; 15: 315–322.
This article describes the demographic characteristics and clinical course of patients with altitude illness who were evacuated for medical treatment from the mountainous regions of Nepal.

there included lack of quality housing, air conditioning, and access to social services and health care.[4,5] Elderly people have a diminished ability to perspire and therefore have less capacity to release body heat through evaporation—the body's main mechanism for removing heat. People with chronic health problems, especially heart disease, or disabilities are more vulnerable to the adverse effects of heat. Also at much increased risk are people who are not acclimatized to hot weather, those who overexert themselves, obese people, and those who consume alcohol or take medications, including drugs such as antipsychotics, tranquilizers, antidepressants, certain types of sleeping pills, and drugs for Parkinson disease.[6]

CASE 2

A mother in New Jersey left her infant son in a car, with the windows rolled up, for 2 hours. During that time, she checked on him often without realizing that the internal temperature of the car was becoming life-threatening. On her last check, she found her son had passed out. She rushed him to the hospital, but he later died of heat stroke. One hour after his death, the boy's body temperature was 42.2°C (108°F). Outside the vehicle, the temperature was only about 16°C (60°F).[7]

Infants and young children are particularly susceptible to the adverse effects of heat. Children's bodies have a greater ratio of surface area to body mass, so they absorb more heat on a hot day (and lose heat more rapidly on a cold day). In addition, children have a considerably lower sweating capacity than adults, and so they are less able to dissipate body heat by evaporative sweating and cooling.[8] According to a National Highway Traffic Safety Administration report, heat stroke is the leading cause of non-crash vehicle deaths among children.[9] Since 1998, an average of 38 children have died annually in the United States as a result of being left, or becoming trapped, in hot vehicles.[10] The windows of a car act like a greenhouse, trapping sunlight and heat. When the outside temperature is 15°C (60°F), car temperatures can rise well above 43.3°C (110°F). Even with the windows rolled down 2 inches, when the outside temperature is 28.3°C (83°F), the temperature inside the car, in only 15 minutes, can reach 42.8°C (109°F).[11]

Environmental temperatures are steadily rising each year and increasingly severe heat waves are likely. In Metropolitan New York City, by the 2050s, an estimated 47% to 95% more people will die of heat-related deaths than in the 1990s. By 2050, average annual summer temperatures in the New York City area may rise by 1.5°C (2.7°F) to 4°C (7.2°F).[12] In the United States, temperature increases over time are likely to be even more pronounced for southern and southwestern cities.[3] In Memphis, Jackson, and Birmingham, the number of days each year that exceed 38°C (100.4°F) is expected to increase from 3 days currently to 20 days by 2050.[13]

During heat waves, the following measures are recommended:

- Make daily checks on homebound neighbors, friends, and relatives, especially if they are elderly or disabled.
- Never leave children or pets alone in a car, even with the windows open and even on seemingly cool or cloudy days.
- Encourage those without air-conditioned homes to seek out cool places, such as shopping malls and theaters, during the hottest parts of the day.
- Encourage the drinking of nonalcoholic and noncaffeinated beverages.
- Reduce or eliminate strenuous activities.

Occupational Heat Stress and Heat Strain

CASE 3

A male worker, age 56, was harvesting ripe tobacco leaves by hand in North Carolina during his third day on the job. He had come from Mexico on a work visa. Co-workers reported during the mid-afternoon that he seemed confused. When they offered to carry him to the shade and have him drink water, he became combative. About 1½ hours after noticing his confusion, they took him to a

hospital emergency department. His core body temperature was 42.2°C (108°F). Despite treatment, he died. That day, the local high temperature was 34°C (93°F), with 44% relative humidity. Conditions had been similar for the 2 previous days. The man had been given health and safety training on pesticides but not on heat stress.[14]

An estimated 5 to 10 million people in the United States work in industries where heat stress is a potential health and safety hazard.[15] (See Fig. 12B-1.) Between 1992 and 2006, the heat-related average annual death rate in the United States for crop workers—those who work outdoors and are exposed to heat and humidity—was 0.4 per 100,000 workers, almost 20-fold higher than for civilian workers (about 0.02 per 100,000 workers).[14] In all industries from 1992 to 2008, exposure to environmental heat killed at least 488 workers and contact with hot objects or substances killed an additional 189.[16] However, because worksite conditions and job duties are usually not listed on hospital records or death certificates, occupational heat-related illnesses and fatalities are usually underestimated.[17]

Heat-related occupational illness, injury, and strain occur in any situation where total heat load (environmental heat and heat generated by the body's metabolism) exceeds the capacity of the body to maintain normal bodily functions. Situations that have increased potential for causing heat strain include high ambient air temperatures, high humidity, strenuous physical activity, radiant heat sources (such as the sun, ovens, and foundry furnaces), and direct physical contact with hot objects. A hot, humid environment, which impedes evaporative cooling, combined with heavy work activity, poses the highest risk for workers because the metabolic load placed on the body generates even more heat. However, work in cooler, less strenuous environments can also pose a risk, depending upon one's heat tolerance. Potential health risks of working in heat-stress conditions depend—more than any other physical factor—on one's physiology and level of acclimatization. Therefore, professional expertise and judgment in evaluating heat stress and strain in a work environment are vital to preventing heat-related injuries and illnesses in workers.[18]

The National Institute for Occupational Safety and Health (NIOSH) defines total heat *stress* as the sum of the heat generated by the body (metabolic), plus the heat gained from the environment, minus the heat lost from the body to the environment (primarily through evaporation).

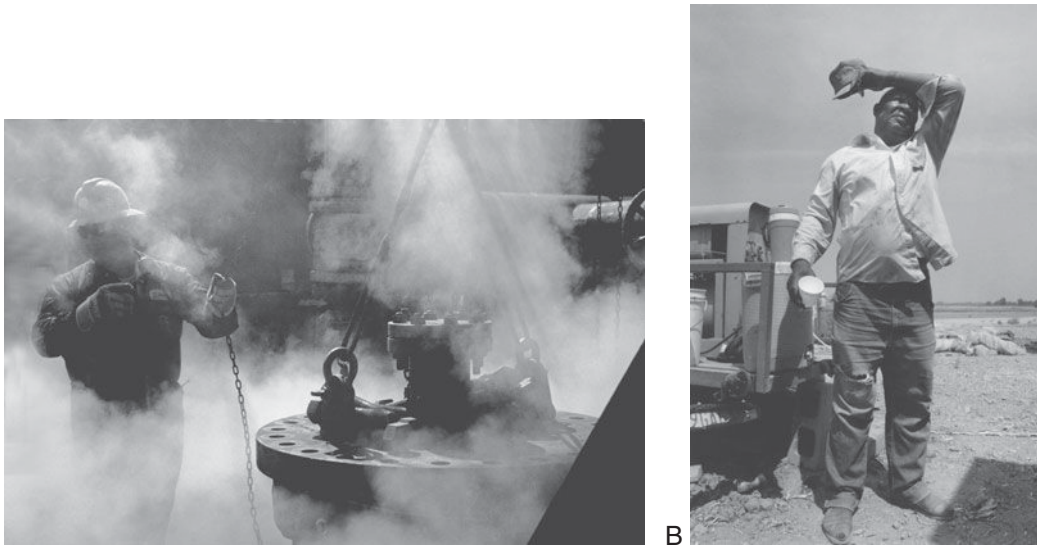

Figure 12B-1. Examples of occupational exposure to extreme heat. (A) Chemical worker exposed to heat while removing vessel lid. (B) Heat-stressed farmworker taking a water break as the ambient air temperature approached 105°F. (Photographs by Earl Dotter.)

Air temperature and velocity, clothing, physical activity, humidity, and radiation are all contributors to the heat stress one experiences.

Heat *strain*, which cannot be reliably predicted from heat stress, is defined as the body's response to the heat stress it experiences. It is unique to each person. Even in the same person given the same exposure, heat strain may be different, depending on current physiological status.[18]

Many bodily responses to heat stress are desirable and beneficial because they help regulate internal temperature and, in situations of appropriate repeated exposure, help the body adapt, or acclimatize, to the work environment. However, at some individually determined stage of heat stress, compensatory measures of the body cannot maintain internal body temperature at a level required for normal functioning. As a result, risks of heat-induced disorders and "accidents" substantially increase.[15]

For normal body function deep-core body temperature must be maintained within the acceptable range of approximately 37°C (98.6°F) ± 1°C (1.8°F), which requires a constant exchange of heat between the body and the environment. The amount of heat to be exchanged is a function of heat produced by the body (metabolic heat) and heat gained from the work environment. The rate of heat exchanged with both hot and cold environments is a function of air temperature, humidity, skin temperature, air velocity, evaporation of sweat, radiant temperature, and type, amount, and characteristics of clothing.[15] The basic heat balance equation, which can also be used to evaluate situations of extreme cold, is as follows:

$$S = (M - W) \pm C \pm R - E$$

where S = change in body heat (either lost or gained by the body); (M – W) = heat produced by metabolism minus heat produced by external work; C = convective and conductive heat exchange; R = radiative heat exchange; and E = body heat lost by evaporation.

Each of the terms in the equation represents a rate of energy transfer—positive values for any of the variables signify that the body is gaining heat in that manner, while negative values indicate a loss of heat. When the body is not thermally challenged, as in homeostasis, there will be no net gain or loss of heat, and S will equal zero. An S greater than zero indicates a heat imbalance, which may lead to heat strain and subsequent heat-related illnesses. The quantity (M – W) describes total body heat produced by combining the metabolic heat gained from work effort minus heat lost due to the external work effort. The metabolic heat value, M, is a combination of the energy expended in doing the work and the energy transformed into heat,[15] which must be removed rapidly from the muscles. When muscle workload is high, so is heat production by the body. A high workload can cause heat gain, even when environmental temperatures feel cool to those performing less strenuous activities. Therefore, the metabolic rate must be considered when evaluating the heat stress of those performing physically demanding work.[18] W represents the amount of energy that is converted from chemical energy to mechanical work, which is usually only about 10% of M. Therefore, because W is small relative to the other routes of heat exchange, this value is usually ignored.

Modes of heat exchange between workers and their environments are convection, radiation, and evaporation. *Convection* refers to the rate of heat exchange between the individual's skin and the air immediately around the skin, if the air is moving. Its value is a function of the difference between the skin and air temperatures and the rate of air movement over the skin. Skin temperature is normally assumed to be 35°C (95°F). Therefore, for a worker wearing a single layer of clothing (long-sleeved work shirt and trousers), an ambient air temperature of greater than 35°C will cause the body to gain heat from the air. In contrast, an ambient air temperature of less than 35°C will cause the body to lose heat into the air by convection.

Conduction, which is the transfer of heat to the skin from direct contact (touch) with hot equipment or floors or from hot liquids, plays a minor role in heat stress other than for brief periods of time when the body may come into contact with such objects. *Radiation*, or radiative heat exchange, also refers to heat that is transferred between the skin and solid surfaces or objects, cold or hot, but without direct skin contact. Working in direct sunlight is an example of radiative heat exposure.

Evaporation of water from the surface of the skin (sweating) is the primary method by which the body regulates internal temperature. Evaporative cooling also occurs from the lungs, but, except for hard work in very dry environments, its contribution to overall heat reduction is minor.[15] The evaporative capacity of the body is a function of ambient air velocity and the water vapor pressure difference between the ambient air and the wetted skin at skin temperature, which is assumed to be 35°C. To solve the equation, metabolic heat produced, air temperature, air water-vapor pressure, wind velocity, and mean radiant temperature all must be measured.[15]

Health Effects of Exposure to Hot Environments

CASE 4

A 43-year-old male, who was a new, seasonal park employee on a day off from work, hiked down a trail in Grand Canyon National Park during the hottest part of the day. Although he was carrying water, he had brought nothing to eat. Park rangers found him that evening wandering around a campground "in a state of shock." His pulse was 100 and his oral temperature was 39.2°C (102.6°F). He was treated for hyponatremia (decreased serum sodium) with intravenous fluids, kept under observation overnight, and medically evacuated the following morning. He later reported that when he was told by hikers coming up the trail that there was no water or shade along the 7-mile route, he decided to continue even more quickly to his destination—the campground at Phantom Ranch at the bottom of the Canyon on the Colorado River. He noticed that although he was drinking and urinating frequently, his urine became clearer the farther he hiked. When asked why he did not try to cool off by getting into the creek running alongside the campground, he told the rangers he did not think to do that and hardly noticed it was there.

The level of heat stress at which excessive heat strain will result depends upon the heat tolerance capabilities of each individual. A person's sensitivity to heat is affected by age, weight, degree of physical fitness, degree of acclimatization, metabolism, level of hydration, use of alcohol or drugs, and medical conditions such as hypertension and diabetes. At greatest risk are the following individuals:

- Unacclimatized workers
- People performing physically strenuous work
- Those with previous heat illness
- Older people
- People with cardiovascular or circulatory disorders
- Those taking medications that impair cooling mechanisms of the body
- People who abuse alcohol or are recovering from recent use
- People in poor physical condition
- Those recovering from illness

Heat disorders and adverse health effects of people exposed to hot work environments include, in increasing order of severity, irritability, lack of judgment and loss of critical thinking skills, skin disorders (such as heat rashes and hives), heat syncope (fainting), heat cramps, heat exhaustion, and heat stroke. Heat syncope happens when blood flow is directed to the skin for cooling, resulting in decreased supply to the brain, and most often strikes workers who stand in place for extended periods in hot environments. Heat cramps, caused by sodium depletion due to sweating, typically occur in the muscles employed in strenuous work. Heat cramps and syncope often accompany *(a)* heat exhaustion, or *(b)* weakness, fatigue, confusion, nausea, and other symptoms that generally prevent a return to work for at least 24 hours to give the individual time to replenish lost electrolytes and body water. A core body temperature (CBT) increase of only 1°C (1.8°F) above normal can adversely affect brain function. As heat-stress levels rise, so do the risks of "accidents" and injuries.[19] The dehydration, sodium loss, and CBT above 38°C (100.4°F) of heat exhaustion are usually due to performing strenuous work in hot conditions with inadequate water and electrolyte intake. Heat exhaustion may lead to heat stroke if the affected person is not quickly cooled and rehydrated.

While *heat exhaustion* victims continue to sweat as their bodies struggle to stay cool, *heat stroke* victims usually stop sweating as their bodies fail to maintain an appropriate core temperature. Heat stroke occurs when hard work, hot environment, and dehydration overload the capacity of the body to cool itself. This thermal regulatory failure is a life-threatening emergency that requires immediate medical attention. Signs and symptoms include irritability, confusion, nausea, convulsions or unconsciousness, hot dry skin, and a CBT above 40.6°C (105°F). Death can result from damage to the brain, heart, liver, or kidneys.[18]

Prolonged increase in CBT and chronic exposures to high levels of heat stress are associated with disorders such as temporary infertility in both men and women, increased heart rate, sleep disorders, fatigue, irritability, kidney stones, and serious gastrointestinal disease. During the first trimester of pregnancy, a sustained CBT greater than 39°C (102.2°F) may endanger the fetus. In addition, one or more occurrences of heat-induced illness predisposes a person to subsequent heat-related injuries and can result in temporary or permanent loss of ability to tolerate heat stress.[15,20]

Acclimatization

Acclimatization is a low-cost, highly effective way to improve the safety, comfort, and productivity of employees in heat-stress situations.[18] It allows workers to withstand heat stress with a reduction in heat strain by a series of physiological adaptations. Acclimatized individuals perspire more abundantly and more uniformly over their body surface and start to sweat earlier than unacclimatized individuals, resulting in lower CBT and lower cardiovascular strain. In addition, acclimatized individuals lose less salt through sweating and can therefore withstand greater water loss.[21]

Working at even a moderate rate in a heat-stress situation brings about physiological changes that substantially improve safety and comfort for those who are healthy. Exposure to heat only, however, will not bring about acclimatization; an elevated metabolic rate, such as occurs during work activities, is also required. The ability of a worker to tolerate heat stress requires integrity of cardiac, pulmonary, and renal functions, the sweating mechanism, the body's fluid and electrolyte balances, and the heat-regulatory mechanism of the central nervous system. Impairment or reduction of any of these functions may interfere with a worker's capacity to acclimatize to heat or, once acclimatized, to perform strenuous work in the heat.[15] Acclimatization at a certain temperature is effective only at that temperature; a person exposed to higher levels of heat stress will not be fully acclimatized at that level—only to the lower level.[18] About 5% of workers may not be able to acclimatize to heat stress adequately.[15]

The effects of acclimatization can begin with as little as 30 minutes of daily physical activity for 1 week. Rapid changes in those first few days give most of the benefits of lower CBT, skin temperature, and heart rate associated with a faster onset of sweating and increased sweat production. Further improvements in water and electrolyte management in the body occur during the second and third weeks. Figure 12B-2 shows a typical acclimatization schedule for workers during a 10-hour shift.

Although heat acclimatization for most individuals begins early during work in the heat, it is also quickly lost if the exposure is discontinued. The loss of acclimatization begins when the activity under heat-stress conditions is discontinued, with a noticeable loss occurring after only 4 days. This loss is usually rapidly made up, so that by Tuesday, workers who were not working on the previous weekend are as well acclimatized as they were on the preceding Friday. However, if there is no exposure to heat stress for 1 to 2 weeks, full acclimatization can require up to an additional 3 weeks of continued physical activity under heat-stress conditions similar to those anticipated for the work.[18] A worker's capacity to acclimatize may be reduced by chronic illness, the use or misuse of pharmacologic agents, a sleep deficit, a suboptimal nutritional state, or a disturbed water and electrolyte balance. In addition, an acute episode of mild illness, especially if it includes fever, vomiting, respiratory impairment, or diarrhea, may cause abrupt, transient loss of acclimatization.[15]

Evaluating and Assessing Heat Stress

In an attempt to understand a worker's level of heat-stress exposure, the *combination* of

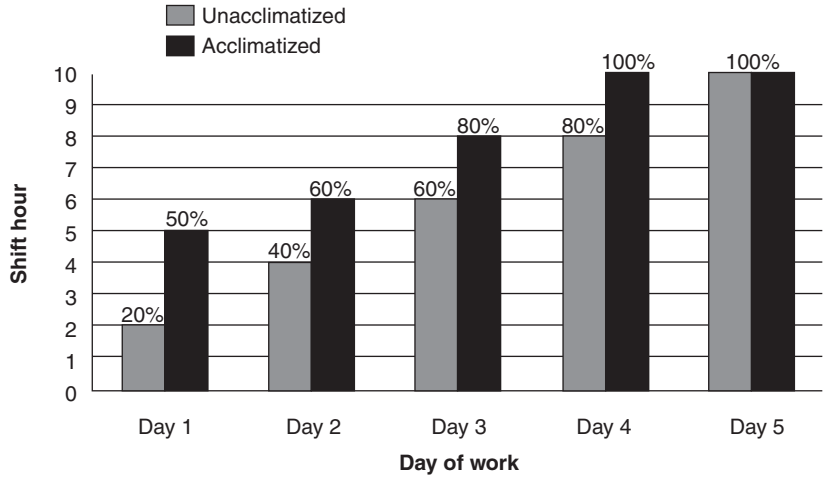

Figure 12B-2. Work schedule for heat acclimatized and unacclimatized employees. (Based on a 10-hour shift.) (*Source*: Adapted from: National Institute for Occupational Safety and Health. Criteria for a recommended standard: occupational exposure to hot environments, rev. DHHS Publication No. (NIOSH) 86-113. Cincinnati, OH: U.S. Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1986, p. 69.)

environmental conditions, work demands, and clothing requirements affect an individual's ability to maintain thermal equilibrium must be evaluated.[18] Assessing heat stress in workers involves measuring the environmental contributions to heat stress at the work location, assessing metabolic work rates for each task, and adjusting for clothing factors.

The wet-bulb globe temperature (WBGT) index, most commonly used to assess the environmental contribution to heat stress, is a simple approximation of the combined effects of air movement, temperature, humidity, and radiative heat.[15] It gives an indication of how a worker feels or perceives the work environment. The WBGT is a function of dry bulb (ambient air) temperature, natural wet bulb temperature (which simulates the effect of evaporative cooling), and black globe temperature (which estimates radiant, or infrared, heat load). Individual and task metabolic rates can be estimated using the table of "Estimated metabolic heat production rates by task analysis" in the NIOSH criteria document *Occupational Exposure to Hot Environments*[15] or by using the work rate categories table by the American Conference of Governmental Industrial Hygienists (ACGIH).[22] In addition, the ACGIH publication *Heat Stress and Strain: Documentation of the TLV (2009 Supplement)* is an excellent resource on the proper use, limitations, and accuracy of the WBGT monitor.[18]

Many heat-stress guidelines have been developed to protect people from heat-related illnesses. The objective of any heat-stress index is to prevent the CBT from rising excessively. Core body temperature should not exceed 38°C (100.4°F) and oral temperature should not exceed 37.5°C (99.5°F) in prolonged daily exposure to heavy work and/or heat.[19] A core body temperature of 39°C (102.2°F) should be considered reason to terminate exposure, even when CBT is being monitored. This does not mean that a worker with a CBT exceeding these levels will necessarily experience adverse health effects; however, the rate of unsafe acts committed by workers increases as does the risk of illness from heat stress.

The Occupational Safety and Health Administration (OSHA) does not have a specific heat-stress standard; however, exposure to heat stress is enforced by the Secretary of Labor under the General Duty Clause of the OSH Act. The OSHA technical manual[20] provides investigation guidelines that approximate those found in the annual ACGIH publication *Threshold Limit Values (TLVs) for Chemical Substances and Physical Agents and Biological Exposure Indices.*[22]

NIOSH recommends that total heat exposure be controlled so that unprotected healthy workers who are medically and physically fit for their required level of activity and are wearing, at most, long-sleeved work shirts and trousers or equivalent, are not exposed to conditions

exceeding the applicable NIOSH criteria: Almost all healthy employees, working in hot environments and exposed to combinations of environmental and metabolic heat less than the NIOSH recommended action limits (RALs) for unacclimatized workers or the NIOSH recommended exposure limits (RELs) for acclimatized workers, should be able to tolerate total heat stress without substantially increasing their risk of incurring acute adverse health effects. No employee should be exposed to metabolic and environmental heat combinations exceeding the applicable ceiling limits (C) of the RALs and RELs, without being provided with, and properly using, appropriate and adequate heat-protective clothing and equipment.[15]

ACGIH guidelines require the use of a decision-making process that provides step-by-step situation-dependent instructions that factor in clothing insulation values and physiological evaluation of heat strain (Fig. 12B-3). ACGIH WBGT screening criteria (Table 12B-1) factor in the ability of the body to cool itself (considering clothing insulation value, humidity, and air movement), and like the NIOSH criteria, can be used to develop work/rest regimens for acclimatized and unacclimatized employees. The ACGIH WBGT-based heat exposure assessment was developed for a traditional work uniform of long-sleeved shirt and pants. It represents conditions under which nearly all adequately hydrated, unmedicated, healthy workers can be repeatedly exposed without adverse health effects. Clothing insulation values and the appropriate WBGT adjustments, as well as descriptors of the other decision-making process components, can be found in *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices* by ACGIH.[22] The ACGIH TLV for heat stress attempts to provide a framework for the control only of heat-related disorders. Although the rates of "accidents" and injuries can increase with increasing levels of heat stress, the TLVs are not directed toward controlling them.

NIOSH and ACGIH criteria can only be used when WBGT data for the immediate work area are available and they must not be used when encapsulating suits or garments that are impermeable or highly resistant to water vapor or air movement are worn. Further assumptions regarding work demands include an 8-hour work day, 5-day work week, two 15-minute breaks, and a 30-minute lunch break, with rest area temperatures the same as, or less than, those in work areas, and "at least some air movement." NIOSH and ACGIH guidelines do not establish a fine line between safe and dangerous levels but, to ensure protection in each situation, require professional judgment and a heat-stress management program.

Evaluating and Assessing Heat Strain

It is impossible to predict in any given environment who will suffer heat strain from excessive heat stress. However, knowing what to look for and how to intercede can prevent injuries and save lives. Supervisors and workers must be educated on *(a)* risk factors for, and early warning signs and symptoms of, heat strain; and *(b)* how to monitor themselves and co-workers. Knowing the signs and symptoms of heat strain is the best way to prevent heat-related illnesses. Observing one symptom deserves attention; observing two or more symptoms requires action.

Physiological monitoring for heat strain becomes necessary when impermeable clothing is worn, when heat-stress screening criteria are exceeded, or when data from a detailed analysis, such as the International Standards Organization (ISO) required sweat rate (SRreq), shows excess heat stress.[22]

One indicator of physiological strain, sustained peak heart rate, is considered by ACGIH to be the best sign of acute, high-level exposure to heat stress. Sustained peak heart rate, defined by ACGIH as 180 beats per minute (bpm) minus an individual's age, is a leading indicator that thermal regulatory control may not be adequate and that an increase in core temperature has, or will soon, occur. Sustained peak heart rate represents an equivalent cardiovascular demand of about 75% of maximum aerobic capacity. During an 8-hour work shift, even if sustained peak demands do not occur, there may still be excessive demand placed on the cardiovascular system. These "chronic" demands can be measured by calculating the average heart rate over the shift. Decreases in physical job performance have been observed when the average heart rate exceeds 115 bpm over the entire shift. This level

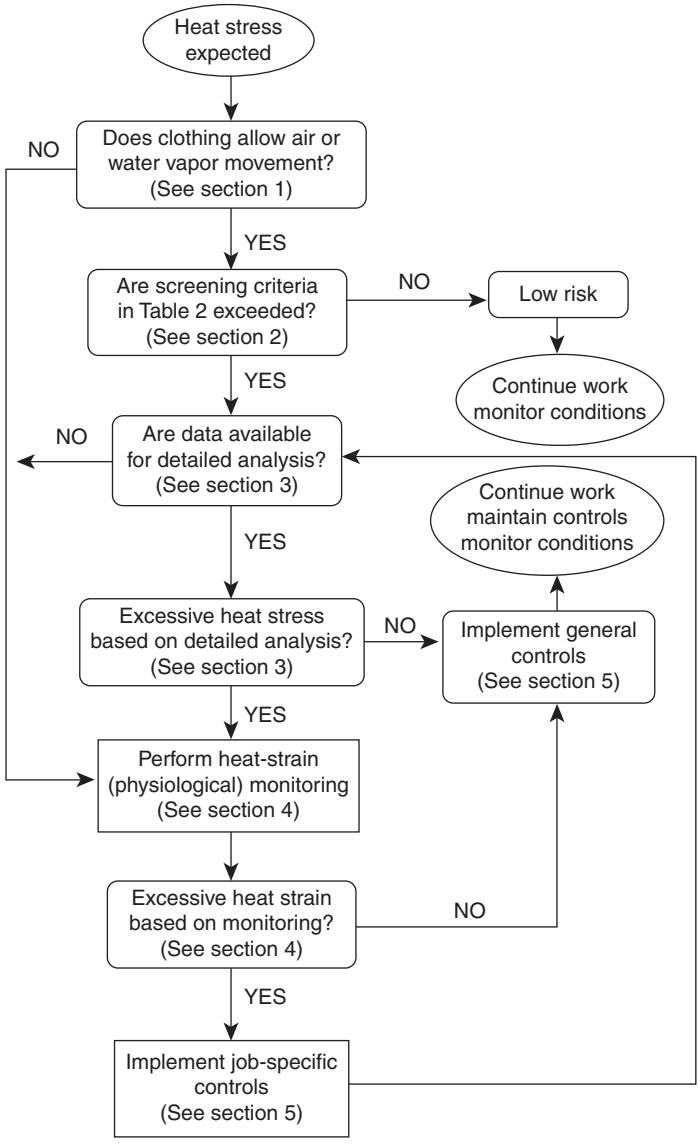

Figure 12B-3. Algorithm for evaluating heat stress and strain. (*Source*: American Conference of Governmental and Industrial Hygienists. 2010 TLVs® and BEIs® Book. Cincinnati, OH: ACGIH®, 2010, p. 210. Copyright 2010. Reprinted with permission.)

is equivalent to working at approximately 35% of maximum aerobic capacity, a level sustainable for 8 hours.

An individual's heat-stress exposure should be discontinued when any of the following excessive heat strain indicators occur: *(a)* for those with normal cardiac performance, heart rate is sustained for several minutes over 180 bpm minus the individual's age in years; *(b)* core body temperature is greater than 38.0°C (100.4°F) for unselected, unacclimatized workers and greater than 38.5°C (101.3°F) for medically fit, heat-acclimatized workers; *(c)* recovery heart rate 1 minute after a peak work effort exceeds 110 bpm; or *(d)* there are symptoms of sudden and severe fatigue, nausea, dizziness, lightheadedness, or the sensation of a pounding pulse within the body. An individual may be at greater risk of heat strain if: *(a)* profuse sweating is sustained over several hours; *(b)* weight loss over a shift is greater than 1.5% of body weight; or *(c)* 24-hour urinary sodium excretion is less than 55 millimoles.

There is a variety of equipment available for monitoring heat strain in an individual. One of

Table 12B-1. Screening Criteria for TLV® and Action Limit for Heat-Stress Exposure

Allocation of Work in a Cycle of Work and Recovery	TLV® (WBGT Values in °C)				Action Limit (WBGT Values in °C)			
	Light	Moderate	Heavy	Very Heavy	Light	Moderate	Heavy	Very Heavy
75 to 100%	31.0	28.0	-	-	28.0	25.0	-	-
50 to 75%	31.0	29.0	27.5	-	28.5	26.0	24.0	-
25 to 50%	32.0	30.0	29.0	28.0	29.5	27.0	25.5	24.5
0 to 25%	32.5	31.5	30.5	30.0	30.0	29.0	28.0	27.0

Wet-bulb globe temperature (WBGT) values are expressed in °C and represent thresholds near the upper limit of the metabolic rate category.

If work and rest environments are different, hourly time-weighted averages (TWAs) should be calculated and used. TWAs for work rates should also be used when the work demands vary within the hour.

Values in this table are applied by reference to the "Work-Rest Regimen" section of the *Documentation* and assume 8-hour work days in a 5-day work week with conventional breaks, as discussed in the *Documentation* (see reference 18). When work-days are extended, consult the "Application of the TLV" section of the *Documentation*.

Because of the physiological strain associated with heavy and very heavy work among less fit workers regardless of WBGT, criteria values are not provided for continuous work and for up to 25% rest in an hour for very heavy. The screening criteria are not recommended, and a detailed analysis and/or physiological monitoring should be used.

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the simplest devices is a standard body-weight scale. Employees should weigh themselves fully clothed, without equipment belts, just before starting a shift and again, wearing the same clothing, just before ending a shift. Weight loss over the shift (hydration status) can then be calculated by subtracting postshift weight from preshift weight and dividing that total by preshift weight. Multiplying by 100 gives percent body weight lost or gained. Weight loss should not exceed 1.5% of total body weight in a work day. If it does, fluid and food intake should be increased until a return to baseline is achieved.

A simple and inexpensive method for monitoring heat strain is to use a heart rate monitor. Currently, three types are available:

1. A wireless strap worn around the chest that sends a signal to a readout display, usually worn on the wrist
2. A monitor worn on the wrist that requires the user to place two fingers on its sensors, after which heart rate is displayed in a few seconds
3. A monitor worn as a ring, which is less accurate[23]

Measurements should be taken at appropriate intervals (that is, 15–30 minutes) covering at least a full 2-hour period during the hottest parts of the day and again at the end of the day to ensure a return to baseline.[15]

There are also currently two methods of monitoring internal (core) body temperature. Both involve swallowing disposable sensors that send a signal to a direct-readout data logger that is worn by the worker. One sensor incorporates a crystal that vibrates in direct proportion to the worker's CBT, while the other is a thermistor-based system. Both offer radiofrequency capabilities and can monitor CBT and heart rate in multiple workers within direct line of sight. Both systems are fairly costly and users should be familiar with the limitations of the equipment prior to use with employees. Also when considering use of this equipment, it is important to remember that most heat-related illnesses may result with little or no increase in CBT and that problems can arise sooner than an increase in CBT is noted. Most heat strain occurs because, at some point during heat exposure, the body is limited in its ability to distribute and store heat. Heat strain can occur without the body experiencing hyperthermia (the increasing inability to maintain thermal equilibrium).

Preventing Heat Stress and Heat Strain

Preventing heat stress and heat strain is the best method of keeping workers safe. Keeping workers fully hydrated is critically important. The thirst mechanism is usually not strong enough

to drive an individual to drink enough to replace the water lost in sweat; therefore, the palatability of fluid replacement is important to ensure adequate rehydration. Adding sweeteners to drinks increases consumption, facilitates sodium and water absorption, and provides energy for muscles engaged in vigorous activities. Ideally, about 5 ounces of fluid at 10°C–15.6°C (50°F–60°F) should be consumed every 15 to 20 minutes.

Prevention always requires professional judgment that considers both environmental conditions and personal risk factors. Administratively, employers can *(a)* evaluate ways to reduce the physical demands of the work, and *(b)* limit work for hot jobs to morning and evening hours and/or institute work/rest regimens. Employers should require workers to use a buddy system, ensure that they are trained in recognizing symptoms of heat strain, and institute preplacement and periodic medical surveillance exams that include individual testing of aerobic capacity and assess mental and medical qualifications. Those with low heat tolerance or poor physical fitness should be excluded from hot jobs. Continuing education programs can provide workers with information on the hazards of heat stress, awareness of its symptoms, and the dangers of using caffeine, drugs, and alcohol while working in hot and physically demanding environments. Workers should be allowed to acclimatize to a hot work environment, which may require additional staffing for the same job until all workers are acclimatized. Employers and supervisors should allow employees to take breaks when they feel they need to, thereby self-limiting their exposure.

Employees can help themselves by ensuring that they are well hydrated, well nourished, and not sleep deprived. Those working in hot, dry environments will also benefit from soaking their clothing with water before and during their shift to aid in evaporative cooling. Equipment such as an ice vest or a capillary cooling system, which can be worn either under air- and vapor-impermeable protective suits or with regular work clothes, will help keep workers cooler during specific short-term, high-intensity tasks. A comprehensive heat-stress management program that includes an emergency action plan for severe heat illness can prevent heat strain in all work environments.

COLD ENVIRONMENTS

CASE 1

One January day, the frozen body of a 93-year-old man was found by neighbors who became concerned when they saw the windows of his house icing over. Four days before, the local utility company had installed on his electric meter a "limiter," which restricts the amount of electricity a house can use, because he had not paid his electric bills. After the limiter was installed, a cold front settled over the area, dropping temperatures to −24°C (−10°F).

CASE 2

An 81-year-old man walked away from his son's home either late at night or early in the morning and was found frozen to death about 200 yards away. The temperature that night was −5°C (5°F). The man had been reportedly diagnosed with dementia and had only recently moved in with his son.

Hypothermia (abnormally low body temperature) causes approximately 700 deaths a year in the United States, half of which occur in people age 65 and older.[24] Like hyperthermia, hypothermia is preventable. Yet dozens of people freeze to death every winter, especially during exceptionally cold periods. Those most at risk include elderly people who live in inadequately heated structures and homeless people. In the United States in 2003, about 70% of those who died of hypothermia were men, with a median age of 61. About 82% of the victims were single, divorced, or widowed. A disproportionate number were members of ethnic minority groups.[25]

Occupational Cold Stress

CASE 3

During construction of the Alaskan pipeline, a man was killed because of the cold-weather clothing he was wearing. The worker had

tunnel vision and impaired hearing because of his wool cap and fur-lined hood. As a result, he failed to see a pickup truck that backed over him. Had he not been wearing a bulky parka, he may have escaped with only minor injuries, but his clothing got entangled in the truck's drive shaft, and he was quickly crushed. One of the lessons of this incident is that failing to manage and properly use personal protective equipment can do more harm than good; ideally every hazard is considered and mitigated on the job.[26]

Occupational illness, injury, and reduced productivity related to cold stress result from a net body heat loss (decrease in CBT) or heat loss from limbs, feet, hands, or head. Workers in agriculture, transportation, oil and gas extraction, construction, warehousing, food production, utilities, and especially those who work outdoors, as open-water divers, and in cold storage, are at increased risk of injuries due to cold stress.

Workers' compensation claims for cold injury reflect the expected association between environmental factors and cold injuries. The highest rates of cold injury occur in the oil and gas industry; trucking and warehousing; protective services; interurban and local transportation; electric, gas, and sanitation; auto sales, service, and repair; food and related products; and heavy construction.[27] Most workers with cold injury are men under 35 years old who do "routine outdoor work."[28] Cold injuries correlate with periods of extreme cold, and, as wind speed increases, the rate of cold injury increases.

Heat loss occurs in the following ways: *(a)* radiation (up to 65%); *(b)* conduction (up to 15%, but greater in cold water where body heat is lost up to 25 times faster)[29]; *(c)* convection (as the wind increases); *(d)* respiration; and *(e)* evaporation. Rates of heat loss by respiration and evaporation depend on the ambient temperature and relative humidity.[30] Working conditions of low temperatures, high winds, wet clothes, or wet body pose the highest risk of developing cold-stress injuries and illnesses. However, hypothermia can result even when air temperatures are above freezing or whenever water temperature is below normal body temperature: 37°C (98.6°F).

The CBT of a worker must remain within 1% to 4% of normal body temperature—no lower than 36°C (96.8°F)—the temperature at which the metabolic rate increases in an attempt to compensate for heat loss and a temperature slightly above the point at which maximal shivering begins. For single occupational exposures, a drop in CBT to no lower than 35°C (95°F) is permissible under ACGIH guidelines.[30] However, CBT is maintained at the expense of other parts of the body, as peripheral blood flow decreases to reduce heat lost from the skin's surface. Therefore, extra care must be taken to protect arms and legs from discomfort and damage.

Health Effects of Exposure to Cold Environments

As with hyperthermia, workers exposed to cold environments often are slow to recognize they are in danger. Hypothermia can lead to carelessness and disorientation especially in those who are not regularly exposed to cold weather and are distracted by other job hazards.[26] Workers beginning to experience hypothermia may shiver and stomp their feet. They may lose coordination, have slurred speech, and fumble hand-held items. As body temperature continues to fall, these symptoms will worsen and shivering will stop. Workers may become unable to walk or stand.

Exposure to a cold environment causes intense stimulation of the sympathetic nervous system, which results in reduced heat loss through the skin (vasoconstriction) and often pain and numbness of fingers and toes. The most common nonfreezing cold injuries are chilblains (pernio) and immersion (trench) foot. With chilblains, repetitive exposure to dry air and temperatures just above freezing to as high as 16°C (60°F) causes damage to the capillary beds in the skin. This damage is permanent and the redness and itching, which typically occur on cheeks, ears, fingers and toes, will return with additional exposure. Trench foot, which can progress to gangrene, is caused from repetitive exposure to a cold, wet environment above the freezing point, conditions common to commercial fishing, for example. Symptoms include tingling or itching, burning, swelling, and in more extreme

cases, blisters.[31] Frostbite, which occurs when the skin tissue falls below 0°C (32°F), can occur when workers touch cold metal or chemicals or wear constrictive clothing or shoes. Frostbite, which can be superficial or deep, is classified in degrees, ranging from redness and numbness (first degree) to frostbite affecting bone and muscle (fourth degree).[24] Freezing in deeper layers of tissue causes the affected area to look waxy and pale and feel hard to the touch.

If exposure to cold continues and CBT falls to around 36°C (96.8°F), metabolic rate, respiration, pulse, and blood pressure increase in an attempt to maintain homeostasis. At a CBT of 35°C (95°F), maximal shivering occurs and physical work and mental processes are impaired. Workers should be removed from the environment when shivering, the most inefficient way of producing heat, becomes evident.[30] Severe hypothermia results if CBT falls below 33°C (91.4°F). Consciousness becomes clouded and progressively lost, respiratory rate and pulse decrease, and blood pressure falls and becomes difficult to measure. The skin becomes cold and may turn bluish. When CBT reaches 28°C (82.4°F), loss of consciousness, little or no breathing, and ventricular fibrillation may occur. Worker fatalities from exposure to cold have almost always resulted from failure to escape from environments with low temperature of air or water.[30]

Workers at greatest risk of cold stress are older workers, those with cardiovascular disorders, and those taking medications that interfere with body temperature regulation or reduce tolerance to working in the cold. The danger of hypothermia is also increased in people who use alcohol and other depressants of the central nervous system. Workers routinely exposed to temperatures below −25°C (−13°F) at wind speeds greater than 2 m/sec (5 mph), or air temperatures below −18°C (0°F) at wind speeds less than 2 m/sec (5 mph) should be medically qualified for work in such environments. Employees suffering from diseases or taking medication that interferes with body temperature regulation or reduces the tolerance for cold work should be excluded from working at or below -1°C (30.2°F).[30]

Workers suffering from cold stress should be moved to a warm, dry area and any wet or tight clothing should be removed. The affected body parts should be protected from further trauma, including *not* rubbing the affected body part because rubbing can cause tissue damage. Affected areas should be soaked in a warm water bath for approximately 25 to 40 minutes, and then dried and wrapped. The skin may blister and appear puffy. Medical attention should always be sought as soon as possible even though normal movement, skin color, and feeling may have returned. One should give workers with hypothermia, if they are alert, warm, sweet, nonalcoholic, noncaffeinated drinks and high-calorie food. If possible, affected workers should move to warm up muscles. If they cannot move, one should place warm water bottles or packs in the axilla, groin, and on the neck and head. One should keep the victim awake and obtain medical attention as soon as possible.

Assessing the Cold Work Environment

Whenever environmental temperatures are expected to go below 16°C (60.8°F), the air temperature should be monitored, and workers performing barehanded tasks for more than 20 minutes should be provided with ways to warm their hands. These may include radiant heaters or warm air jets. When they fall below −1°C (30.2°F), air temperatures should be monitored at least every 4 hours and metal tool handles and control bars should be wrapped in insulating material. Wind speed should also be monitored when it exceeds 2 m/sec (5 mph) or whenever air temperatures drop below −1°C.[29] Workers should be provided with anti-contact gloves, such as those made of silk, to prevent contact frostbite from surfaces that are less than −7°C (19.4°F).

Workers in an environment that is continually at or below −7°C (19.4°F) should be provided a work-warming regimen. Table 12B-2 is an example of a work/warm-up schedule for a 4-hour work shift. Heated shelters stocked with warm, noncaffeinated drinks and food should be readily available. Workers should remove their outer layers of clothing while in the shelter. (The work rate should not be so high that heavy sweating results; however, dry clothing should be available if necessary to prevent a return to the work environment in wet clothing.)

Table 12B-2. Threshold Limit Values (TLVs) Work/Warm-up Schedule for a 4-Hour Shift

Air Temperature,	Sunny Sky	No Noticeable Wind		5 mph Wind		10 mph Wind		15 mph Wind		20 mph Wind	
°C (approx.)	°F (approx.)	Max. Work Period	No. of Breaks	Max. Work Period	No. of Breaks	Max. Work Period	No. of Breaks	Max. Work Period	No. of Breaks	Max. Work Period	No. of Breaks
–26° to –28°	–15° to –19°	(Norm. breaks)	1	(Norm. breaks)	1	75 min	2	55 min	3	40 min	4
–29° to –31°	–20° to –24°	(Norm. breaks)	1	75 min	2	55 min	3	40 min	4	30 min	5
–32° to –34°	–25° to –29°	75 min	2	55 min	3	40 min	4	30 min	5	Nonemergency work should cease	
–35° to –37°	–30° to –34°	55 min	3	40 min	4	30 min	5	Nonemergency work should cease			
–38° to –39°	–35° to –39°	40 min	4	30 min	5	Nonemergency work should cease					
–40° to –42°	–40° to –44°	30 min	5	Nonemergency work should cease							
–43° and below	–45° and below	Nonemergency work should cease									

Notes:

- Schedule applies to any 4-hour work period with moderate to heavy work activity, with warm-up breaks of ten (10) minutes in a warm location. For light-to-moderate work (limited physical movement), apply the schedule one step lower. For example, at −35°C (−30°F) with no noticeable wind (Step 4), a worker at a job with little physical movement should have a maximum work period of 40 minutes with 4 breaks in a 4-hour period (Step 5).
- The following is suggested as a guide for estimating wind velocity if accurate information is not available: 5 mph—light flag moves; 10 mph—light flag fully extended; 15 mph—raises newspaper sheet; 20 mph—blowing and drifting snow.
- If only the wind-chill cooling rate is available, a general rule of thumb for applying it rather than the temperature and wind velocity factors given above would be: *(a)* special warm-up breaks should be initiated at a wind-chill cooling rate of about 1,750 W/m^2; and *(b)* all non-emergency work should have ceased at or before a wind chill of 2,250 W/m^2. In general, the warm-up schedule provided above slightly undercompensates for the wind at the warmer temperatures, assuming acclimatization and clothing appropriate for winter work. On the other hand, the chart slightly overcompensates for the actual temperatures in the colder ranges, because windy conditions rarely prevail at extremely low temperatures.
- Threshold limit values apply only for workers in dry clothing.

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Preventing Cold-Related Injuries

Cold-related injuries can be prevented by developing and using a cold-weather management plan. Plans may include scheduling maintenance and repair jobs during warmer weather or warmer parts of the day, reducing physical demands, using relief workers or assigning extra workers for long, demanding jobs, monitoring workers who are at risk of cold stress, and providing cold-weather training that includes information about risks, prevention, symptoms, monitoring, and personal protective equipment.[32]

Protective clothing is the most important way to avoid cold stress. The following are recommendations for working in cold environments:

Wear at least three layers of clothing:

1. An outer layer, like Gortex or nylon, to break the wind and allow some ventilation
2. A middle layer of down or wool to absorb sweat and provide insulation even when wet
3. An inner layer of cotton or synthetic weave to allow ventilation

Wear a hat. Up to 40% of body heat can be lost when the head is left exposed.

Wear insulated boots or other footwear.

Keep a change of dry clothing available in case work clothes become wet.

Do not wear tight clothing. Loose clothing allows better ventilation.

The thirst mechanism is usually not strong enough to drive an individual to drink enough to replace the water lost in sweat, especially when it is cold, so workers should take care not to become dehydrated. As in hot weather, workers should drink plenty of liquids and avoid caffeine and alcohol. Workers should also take frequent breaks out of the cold, avoid fatigue, and consume warm, high-calorie food to maintain energy reserves. As with heat stress, supervisors should enforce appropriate work schedules and allow workers to interrupt their work if they express extreme discomfort.

When temperatures in the work environment are at or below −12°C (10.4°F), workers should pair up or be under constant protective observation. Workers new to the environment should be allowed to acclimatize. They should not be required to work full-time in the cold during the first few days of work while they adjust to the conditions and protective clothing.[30]

Workers should also be educated about symptoms of cold-related illnesses and should be encouraged to seek shelter and medical attention if they or fellow workers experience pain, numbness or tingling, severe shivering, or drowsiness. In general, workers who are provided with insulated protective clothing, assigned to work in areas where drafts and wet conditions are minimized, and given adequate breaks with access to a warm shelter and food are far less likely to suffer adverse health effects from exposure to cold.

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These two publications are the best technical guides for keeping workers safe from the effects of heat stress and strain. The NIOSH document also covers international and foreign standards and recommendations.

American Conference of Governmental Industrial Hygienists. Cold stress: documentation of the TLVs and BEIs with other worldwide occupational exposure values (CD-ROM-2009). Cincinnati, OH: ACGIH (2001 suppl.), 2009.

This is a useful summary of the issues relevant to protecting workers from the effects of cold on the body.

Crippen E, Davis K, (eds.). Staying alive in the arctic: a cold weather survival manual. American Petroleum Institute, Washington, DC, 1978.

While most workers do not ordinarily face the harsh conditions that builders of the Alaska pipeline did, lessons learned there are still helping safety professionals protect workers from the cold. This publication should be required reading for many cold-weather workers.

Danzl DF. Hypothermia and frostbite. In Fauci AS, Braunwald E, Kasper DL, et al. (eds.). Harrison's principles of internal medicine (17th ed.). New York: McGraw-Hill Medical, 2008, pp. 135–138.

This reference provides a good overview of hypothermia and, more importantly, the exact steps to take when you are faced with a victim of cold stress.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the U.S. Public Health Service, the Bureau of Land Management, or the National Institute for Occupational Safety and Health.

12C

Ionizing and Nonionizing Radiation

John Cardarelli

The term *radiation* initiates certain feelings and responses in people based on their past experiences and knowledge of the subject. Although humans evolved in an environment consisting of ionizing and nonionizing background radiation for millions of years, it was not until 1895 that the discovery of X-rays (ionizing radiation) was made—by Wilhelm Conrad Roentgen. The following year, Antoine Henri Becquerel discovered that uranium emits another form of invisible energy, later termed *radioactivity* by Marie Curie, another pioneer in ionizing radiation.

Since these discoveries, ionizing and nonionizing radiation has successfully been used for beneficial purposes in nearly all industries, employing millions of workers (Table 12C-1). Despite its enormous benefits, ionizing radiation suffers from a negative societal perception, which has been influenced by the nuclear weapons industry and the continuing adverse health effects of workers in this industry. In addition, society often judges ionizing radiation on the basis of highly publicized negative events: use of atomic bombs in Hiroshima and Nagasaki (1945); nuclear power plant accidents at Three Mile Island (1979) and Chernobyl (1986); criticality at a nuclear fuels fabrication facility in Tokaimura, Japan (1999); the dispersion of cesium-137 source from a teletherapy machine in Goiania, Brazil (1987), where four people died and more than 100,000 requested screening for potential contamination; and the concern over the potential use of radioactive "dirty bombs." In contrast, nonionizing radiation has a positive image in society because of its tangible benefits, such as wireless communications, and lack of negative events or strong evidence of adverse health effects.

IONIZING RADIATION

Basic Terminology

Ionizing radiation is caused when an electron is ejected from its atomic structure. *Nonionizing radiation* does not eject electrons, but causes molecules to vibrate. *Exposure* represents the amount of ionizing-radiation energy that is absorbed in air. *Dose* refers to the amount of energy absorbed in a specified material other than air, usually tissue. *Half-life* is the amount of time that it takes for half of an amount of radioactive material to decay. *Activity* represents the decay rate or how quickly that radioactivity material decays. *Risk* is the increment of some adverse health affect associated with a known amount of cumulative radiation dose.

The multiple units and scientific terms used to define and describe ionizing radiation do little to bring understanding to those outside the field. These problems have existed for decades and will continue to exist until scientific organizations,

Table 12C-1. Types of Workers, by Sector, Who May Be Exposed Occupationally to Ionizing and Nonionizing Radiation

- Accelerator personnel
- Department of Defense (DOD)
- Department of Energy (DOE)
  - Contractor employees
  - Visitors
  - Reactor facility employees
  - Weapons fabrication personnel
  - Office workers
  - Uranium fuel cycle
    - Miners
    - Millers
    - Fuel fabricators
    - Fuel processors
    - Uranium enrichment workers
- Educational institutions
- Industry
  - Radiographic workers (in nondestructive testing)
  - Manufacturing workers
  - Distribution workers
  - Well logging workers
- Medical and dental
  - Dental workers
  - Medical clinic workers
  - Nuclear medicine (fluoroscopy) workers
  - Radiologists and radiology technicians
  - Veterinarians
  - Chiropractors
  - Podiatrists
- Nuclear power plant workers
  - Commercial plants
  - Naval fleet and shipyard workers
- Transportation
  - Airline crews
  - Screening personnel
  - Trucking and other shipping workers
- Regulatory
  - Inspectors
- Research
- Other

Source: Adapted from National Council on Radiation Protection and Measurements. NCRP Report No. 101: Exposure of the U.S. population from occupational radiation. Washington, DC: NCRP, 1989; and National Council on Radiation Protection and Measurements. NCRP Report No. 160: Ionizing radiation exposure of the population of the United States. Washington, DC: NCRP, 2009.

industries, and national governments achieve harmonization. Scientific organizations are providing leadership by publishing internationally recognized standards through the International Commission for Radiation Protection (ICRP) and the International Atomic Energy Agency (IAEA). In contrast, industry continues to produce instrumentation that provides results in conventional units (Table 12C-2), and governments, with their complex legislative processes, struggle to keep abreast of changing developments. With consistent use of terms and units, those outside the field will begin to better understand ionizing radiation.

The Basics of Ionizing Radiation

The various types of radiation and how they interact with matter can be described by our understanding of the atom. *Alpha radiation* (α), which consists of alpha particles, each of which is a helium nucleus (two protons and two neutrons), is typically associated with heavy elements, such as radon, radium, uranium, and plutonium. It is a large, positively charged particle and easily interacts with other atoms to quickly deposit its energy. Depending on its energy, which is measured in million electronvolts (MeV), an alpha particle can travel up to 10 cm in air. However, most only penetrate 1 to 3 cm (less than 5 MeV) before dissipating all their energy. Alpha particles with at least 7.5 MeV can penetrate the nominal protective layer of the skin (0.07 mm), but only 14% of all alpha

Table 12C-2. Radiation Units

Parameter	Conventional Units	SI Units
Exposure	**Roentgen (R)** 87.6 ergs per gram (air) 2.58×10^{-4} Coulomb/kg (air)	**Coulombs/kg**
Dose	**rad** 100 ergs per gram (tissue) 1 rad = 0.01 Gy	**Gray (Gy)** 1 Gy = 100 rad
Dose equivalent	**rem** 1 rad x w_r* 1 rem = 0.01 Sv	**Sievert (Sv)** 1 Sv = 100 rem
Activity	**Curie (Ci)** 3.7×10^{10} decays per second	**Becquerel (Bq)** 1 decay per second

*w_r = Radiation weighting factor, which is a dimensionless number that depends on the way in which the energy of the radiation is distributed along its path through the tissue. In general, it is 20 for exposures to alpha particles, 1 for exposures to beta particles and gamma and X-rays, and 5 to 20 for exposures to neutrons.

emissions occur above this level of energy, and most of these 14% are manmade with very short half-lives. Therefore, alpha radiation does not pose an external hazard to people, who are easily shielded (such as by air, skin, or paper), but it can be hazardous if the emitting radionuclide is inhaled, ingested, or injected into the body.

Beta radiation (β) consists of electrons emitted by atoms. The mass of an electron is 1/1,836 that of a proton, which permits it to penetrate further into materials or tissue.[1] Due to their small size and charge, beta particles can travel about 12 feet per MeV in air and need only 0.07 MeV to penetrate the skin. Most beta particles do not normally penetrate beyond the top layer of skin, but exposure to higher-energy beta particles (>0.07 MeV) can cause skin burns. Beta radiation is easily shielded with plastic, glass, or metals, but layers of plastic or glass are preferred in the workplace because X-rays can be produced from beta interactions with metal shielding. These characteristics make beta radiation both an external and internal hazard to people.

Photon (gamma or X-ray) radiation is a form of electromagnetic radiation, such as light, with energies high enough to cause ionization. There are several differences between gamma rays and X-rays, most importantly their points of origin. Gamma rays originate from within nuclei of atoms, whereas X-rays originate from surrounding orbital electrons. Gamma-ray emissions are very specific and are often used to identify radionuclides with special instruments. X-ray emissions are generally not specific because they are produced artificially by the rapid slowing down of an electron beam (*bremstrahlung radiation*). Since the rate of slowing is not specific, various X-ray energies exist within a continuum of energies that peak at the maximum energy of the incident electron beam or beta particle on the target surface. *Characteristic X-rays* are one exception where X-rays with specific energies are emitted due to the specific energy levels between electron shells. An electron shifting from a higher energy shell to a lower energy shell will emit an X-ray of fixed energy that is equal to the energy difference between shells. Gamma rays commonly encountered in medical and industrial workplaces are generally higher in energy (0.2 MeV to 1.5 MeV) than X-rays (typically less than 0.5 MeV). Finally, photon-emitting radionuclides found in natural background generally have higher energies (such as potassium-40, with 1.46 MeV) than artificial radionuclides (such as cobalt-60 with 1.125 MeV and 1.17 MeV, and cesium-137 with 0.662 MeV).

Neutron radiation is essentially zero for background radiation levels at ground level. It is an occupational concern only at commercial nuclear power plants and research facilities, and for airplanes and spacecraft. Neutrons have no charge. Therefore, they are not influenced by other charged particles and can easily penetrate materials. Water and concrete are effective shielding materials because they contain many similar-sized atoms close to that of a neutron (hydrogenous materials). As the neutrons penetrate these materials, they interact with the atomic nuclei of the material—like billiard balls. Neutron radiation is also capable of creating radioactive materials through a process called *activation*. When a neutron is absorbed by an atomic nucleus, the atom becomes "excited" and often releases the excess energy in the form of other types of radiation, especially protons. Protons originate from the nucleus and hold an electric charge of +1 elementary charge. Since the number of protons in the nucleus identifies an element, any change in this number will change the element and its chemical properties. The most common activation product encountered in various industries is cobalt-60.

Measuring External and Internal Doses

Alpha, beta, gamma, and X-radiation do not cause the body to become radioactive. However, most materials in their natural state, including body tissues, contain measurable amounts of radioactivity, especially potassium-40, and uranium and thorium decay products. *External doses* occur when the body is irradiated by a radioactive source outside the body. This dose can be measured by using a dosimeter. A qualified individual should select the most appropriate dosimeter for a given application, based on its advantages and limitations (Table 12C-3). All these technologies can be built to detect the types of radiation (beta, gamma, or neutron) and their respective energies, but they cannot identify the specific radionuclide emitting the energy.

Table 12C-3. Advantages and Limitations of Dosimeter Types

Dosimeter Type	Advantage	Limitations
Pocket ion-chambers	Immediate readout	False-positives (impact sensitive)
	Reusable	Requires minor maintenance
	Cost-efficient	
	Some can differentiate between gamma rays and neutrons	
Film	Provides a permanent record of dose (re-readable)	Sensitive to light
	Can differentiate between beta particle, gamma ray, and neutron exposures	Higher limit of detection
		Measurements must be processed
	Provides an integrated dose	Variation from batch emulsions
	Can estimate energy level of radiation	Chemical processing variables
	Simple design	
Thermoluminescent (TLD)	Can differentiate between beta particle, gamma ray, and neutron exposures	Measurement must be processed
		Not a permanent record
	Provides integrated dose	Some TLD materials subject to fading (resulting in underreporting of dose)
	Can estimate energy level of radiation	
	Lower limit of detection	
	Simple design	Potential for false-positives
Optical stimulated luminescent (OSL)	Similar to TLDs	Measurements must be processed
	Distinguishes static from dynamic exposure conditions	Sensitive to light
	Provides a permanent record (reanalysis; dose verification)	
	Quicker readout (within seconds)	
	Reduced potential for false-positives	
Electronic	Immediate readout	Expensive
	Can differentiate between beta particle, gamma ray, and neutron exposures	Requires calibration and maintenance
		Availability in large numbers may be limited during periods of high demand
	Provides integrated dose	
	Can estimate energy level of radiation	
	Lower limit of detection	Sophisticated design
	Data-logging capabilities	
	Some provide visual and audible warnings	
	Telemetry	

Other types of detectors and electronics are needed to identify radionuclides. Additional considerations when selecting a dosimeter include the type of radiation encountered, the monitoring frequency (immediate, hourly, weekly, monthly, or quarterly), the required sensitivity, processing time, and cost.

Internal doses occur when a radioactive material enters the body via inhalation, ingestion, injection, or absorption through the skin and deposits it energy in the tissues. Typical external and internal radioactive sources and their industrial uses are listed in Table 12C-4. Doses from internal exposures are more difficult to assess than external exposures because individual characteristics, such as diet, health status, and age, vary greatly within a population. In an attempt to standardize the dosimetry methodology, the International Commission on Radiological Protection (ICRP) has developed sophisticated human reference models to estimate internal doses.[2,3] These models often govern the airborne radiation concentration limits (derived air concentrations, or DACs) for workplaces. Internal dose estimates are determined by direct measurements and/or biological sample analyses. Direct measurements, such as of thyroid, bone, or the entire body, employ very sensitive instruments that measure photon radiation (gamma or characteristic X-rays) emitted from within the body. Specific gamma energies identify radionuclides, while measurements estimate the amounts internally deposited. These data are then used, together with knowledge of the initial time of exposure and the ICRP standardized models, to estimate dose. Biological samples, such as urine, feces, exhaled breath, sweat, and hair, are used when the type of exposure,

chemical properties (such as solubility), and radionuclide are known. The amount of radioactive material measured in these samples can estimate internal dose by using the ICRP models. In the workplace, both methods are used to refine internal dose estimates, as more information is obtained on the individual's elimination rate of the radioactive materials.

Background Radiation and the Environment

The National Commission on Radiological Protection (NCRP) published a complete review of all radiation exposures to U.S. residents for 2006, such as terrestrial, cosmic, medical, consumer-product, security, research, and occupational exposures.[4] The average annual dose was 6.2 mSv (620 mrem), which represented a 170% increase over the average annual dose to U.S. residents from the late 1980s (3.6 mSv or 360 mrem) (Fig. 12C-1).[5]

Radon and thoron exposure, which is responsible for about 36% of background dose (about 2.28 mSv), is highest where naturally occurring radioactive material (NORM) (uranium and thorium) is found. Cosmic radiation, which accounts for about 5% (0.33 mSv) of background levels, increases at higher altitudes and at latitude positions closer to the poles. Terrestial radiation (rocks and soil) accounts for about 3% (0.21 mSv). Internal exposures (radioactive substances inside the body, especially potassium-40) account for about 5% (0.29 mSv). Occupational and manmade radiation sources (radiation from consumer products, industrial uses, and security uses, and education and research) account for about 2% (0.14 mSv). Medical exposures, which account for 49% (3 mSv) of the total annual dose, increased 700% since the 1980s—due mainly to increases in medical imaging and the size of the U.S. population.

Adverse Health Effects

Health effects from radiation exposures vary with the type, amount, and duration of exposure. When radiation exposes a cell, it may *(a)* pass through without doing any damage; *(b)* interact and damage the cell, but with later repair by the cell; *(c)* interact and damage the cell in such a way that it continues to reproduce itself in a damaged state; or *(d)* kill the cell. The death of a single cell may not be harmful; however, if many cells are killed within an organ, then that organ may not function properly. The likelihood of damage is also related to the mitotic cycle of the cell. In 1906, the Law of Bergonie and Tribondeau concluded that the most radiosensitive cells have a high division rate and a long dividing future, and are not specialized.

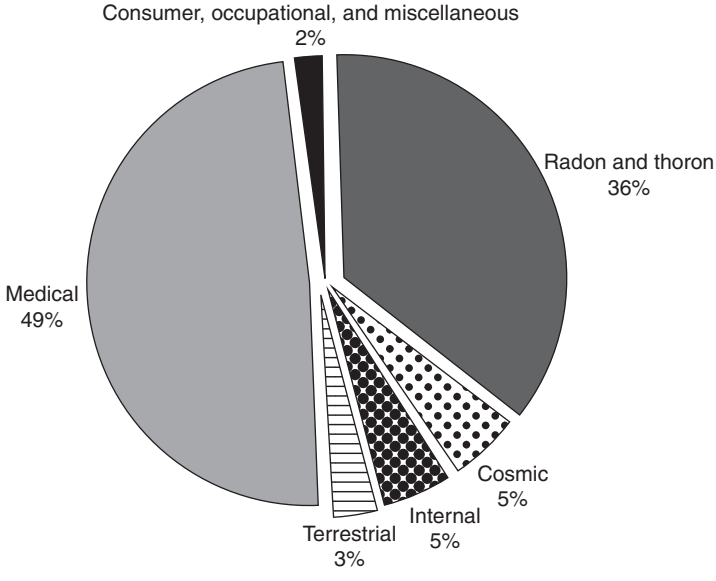

Figure 12C-1. Sources of average annual dose of ionizing radiation to the U.S. population. (*Source*: Reference #4.)

In general, tissues that are young and rapidly growing are most likely to be radiosensitive. Therefore, mature lymphocytes are more radiosensitive than (in order) intestinal crypt cells, mature spermatocytes, erythorcytes, and nerve cells.

The acute health effects of ionizing radiation are nonstochastic, in that the severity of the effect varies with the dose. They occur within a range of minutes to days after exposure. If the dose is kept below a given threshold, usually about 0.25 Gy (25 rad), no effect will be observed. Above this value, especially above 1 Gy (100 rad), acute radiation sickness develops, which includes the hematopoietic syndrome, gastrointestinal syndrome, central nervous system syndrome, and cutaneous radiation syndrome, the last of which often complicates the recovery process of the affected individual due to an increase potential for infection.

The hematopoietic syndrome, which results from penetrating gamma or X-ray doses ranging between 2 to 10 Gy (200 to 1000 rads) is characterized by deficiencies of white blood cells overall, lymphocytes, and platelets. It consists of four phases: prodromal (with nausea, vomiting, and anorexia lasting up to 48 hours), latent (without symptoms, but with changes in blood elements lasting up to 3 weeks), bone marrow depression, and recovery (Fig. 12C-2).

The gastrointestinal syndrome, which results from penetrating gamma or X-ray doses greater than 10 Gy (1000 rads), is characterized by an immediate onset of nausea, vomiting, and diarrhea, followed by a short latent period. Severe dehydration is caused by the massive denuding of the gastrointestinal tract. Most patients do not survive.

The central nervous syndrome, which results from penetrating gamma or X-ray doses above 100 Gy (10,000 rads), is characterized by vomiting and diarrhea (within minutes of exposure), confusion, disorientation, hypotension, and fever, resulting in death within a short time.

The severity of the cutaneous syndrome is determined by the dose of *beta radiation*, energy of the radiation, and type of exposure (skin contamination, contact with contaminated clothing, or distant exposure). Effects depend on uniformity of exposures and location of

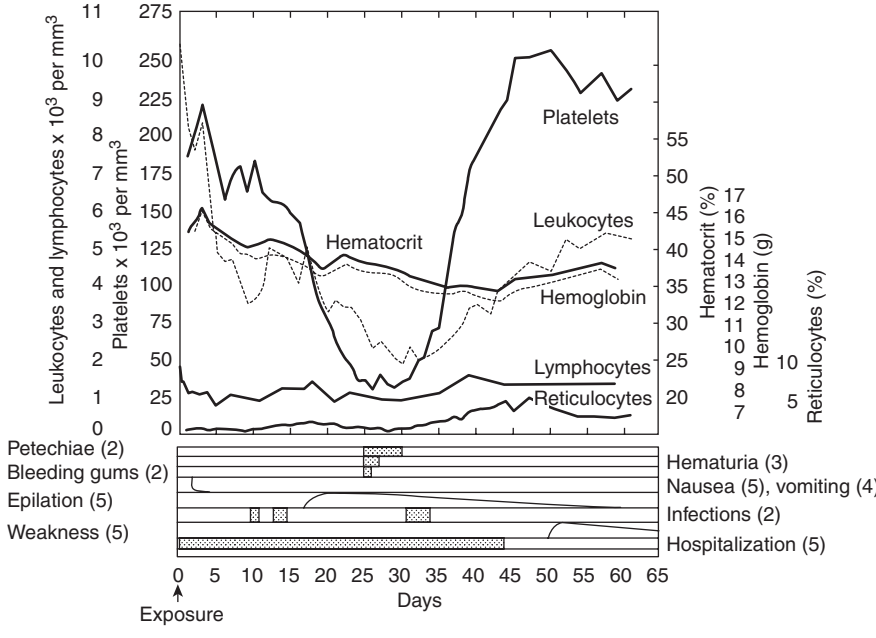

Figure 12C-2. Hematologic values, symptoms, and clinical signs in five men exposed to whole-body irradiation in a criticality accident. The blood counts are the average value for the five men; the figures in parentheses denote the numbers showing the symptoms and signs indicated. (*Source*: From Andrews GA, Sitterson EW, Kretchmar AL, et al. Criticality accidents at the Y-12 plant. In: Diagnosis and treatment of acute radiation injury. Geneva: World Health Organization, 1961: 27–48.)

contamination on the body. Most radiosensitive are moist areas (axilla, groin, and skin folds), followed by the inner aspect of the neck, the antecubital and popliteal spaces, the flexor surfaces of the extremities, and the chest, abdomen, face, and back. Least sensitive are the nape of the neck, scalp, palms, and soles. The larger the area irradiated, the lower the dose needed for adverse reactions. Conversely, the smaller the area irradiated, the higher the dose needed for a similar reaction. A temporal scheme classifies the effects as acute effects (within the first 6 months), subacute effects (within the second 6 months), in the chronic clinical period (2 to 5 years), and in the late clinical period (after 5 years). The skin response depends on the dose. Skin responses include erythema (3–10 Gy; 14–21 days), hair loss (>3 Gy; 14–18 days), dry desquamation (8–12 Gy; 25–30 days), moist desquamation (15–20 Gy; 20–28 days), blister formation (15–25 Gy; 15–25 days), ulceration (>20 Gy, 14–21 days), and necrosis (>25 Gy; >21 days). Workers in the commercial nuclear power industry can face a unique skin hazard of highly localized, radioactive material (usually cobalt-60 or cesium-137), called *hot particles*, *fleas*, or *specks*. These particles range from 1 to 100 μm in diameter, deliver very high doses to a local area, and are difficult to remove. In the event of a terrorist attack involving nuclear material (involving fission) or radioactive (nonfissile) material, these particles may become a primary radiological concern, but they are not likely to result in whole-body doses resulting in death.

Chronic effects (stochastic effects) are those in which the probability of the effect increases with increasing dose, without a threshold. Any dose has a probability of causing the effect; however, the severity of the effect remains unchanged. Cancer and hereditary effects are examples of stochastic effects. The international scientific community has adopted a linear, no-threshold, dose–response model to set occupational dose limits, based primarily on atomic bomb survivors and people exposed medically. There is little controversy about the linear response between high cumulative doses (>1 Gy; 100 rads) and adverse health affects. However, controversy continues as to whether the linear no-threshold model is appropriate for lower cumulative doses (measured in Sv or rem) and dose-rate (measured in Sv/hour or mrem/hour) as found in the workplace.[6] Over the past several decades, several response models have been studied and proposed in the scientific literature, including the linear quadratic model (cancer risk increases exponentially with dose), the threshold model (cancer risk does not exist until dose reaches a particular level), the supra-linear model (cancer risk is substantially increased at lower dose and dose-rate levels), and the hormesis model (a health benefit is recognized at low levels and cancer risk only becomes a concern at a particular dose level) (Fig. 12C-3).[7]

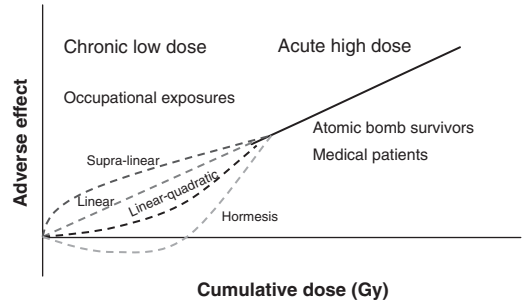

Figure 12C-3. Health effects associated with dose of ionizing radiation.

Pregnancy Issues

Thousands of pregnant workers are exposed to ionizing radiation each year. Inadequate knowledge about ionizing radiation contributes to much anxiety and unnecessary consideration of pregnancy termination. Fears and concerns can often be alleviated by an understanding that the radiation risks during pregnancy are related to radiation dose and stage of pregnancy. Preconception irradiation of either parent's gonads does not result in increased risk of future cancer or congenital malformations in the child. Radiation risks are most significant during organogenesis in the early fetal period and lower in the second and third trimesters. Congenital malformations, which have a threshold ranging between 0.1 to 0.2 Gy (10 to 20 rads), typically involve the central nervous system. Fetal doses of 0.1 Gy are not reached even with three pelvic computed tomography (CT) scans or 20 conventional diagnostic X-ray examinations. Ionizing radiation increases the risk of leukemia and

other malignancies in adults and children under 18 years of age. The embryo/fetus is assumed to be at about the same risk for carcinogenic effects as children. After exposure in utero to 0.01 Gy, the absolute risk of a fatal cancer from birth to age 15 is about 1 in 1,700. This suggests that the probability of bearing a healthy child is very high, even if the pregnant worker receives a radiation dose that exceeds the occupational dose limit for nonpregnant workers. These risks must be considered in the context of the occurrence of abnormal pregnancy outcomes of pregnant women who are not exposed to radiation: spontaneous abortion, more than 15%; genetic abnormalities, 4% to 10%; intrauterine growth retardation, 4%; and major malformations, 2% to 4%. (See Chapter 20.)

Exposures of more than 0.1 Gy (10 rads) are extremely rare in the workplace, especially if a woman informs her employer of her pregnancy. The dose to a declared pregnant worker is limited in the United States to 0.005 Gy (0.5 rad) per pregnancy (one-tenth of the occupational dose limit for nonpregnant workers). The ICRP states that pregnant workers may work where there is a potential for exposure to ionizing radiation as long as there is reasonable assurance that the fetal dose can be kept below 0.001 Gy (0.1 rad) above background throughout the pregnancy. This dose is about the same as that which all people receive annually from penetrating natural background radiation, excluding radon, and one-fiftieth of the nonpregnant occupational dose limit of 0.05 Gy (5 rads).

Termination of pregnancy is rarely contemplated due to of an occupational exposure, but it may become a dominant concern after an attack with a nuclear weapon or "dirty bomb." High fetal doses (0.1 to 1.0 Gy; 10 to 100 rads) during late pregnancy are *not* likely to result in congenital malformations since all the organs have been formed by then. There is less than a 1% chance that childhood cancer or leukemia will result from a fetal dose of about 0.1 Gy (10 rads). Therefore, termination of pregnancy at fetal doses less than 0.1 Gy (10 rads) is not justified on the basis of radiation risk. As the fetal dose increases to above 0.5 Gy (50 rads), there can be significant fetal damage, depending on the stage of the pregnancy. At fetal doses between 0.1 and 0.5 Gy (10 and 50 rads), decisions should be based upon individual circumstances.[8]

Radiation Protection

Radiation protection standards have evolved since the discovery of X-rays in 1895, and they continue to undergo changes, additions, and revisions. International and national organizations recommend scientifically based protection standards, and national governments promulgate regulations with occupational dose limits (Table 12C-4). The latest recommendations differ from regulatory standards since these are

Table 12C-4. Occupational Dose Limits or Recommendations (Annual Unless Otherwise Specified)

Dose Limits	DOE	NRC	OSHA	NCRP (1993)	ICRP* (1991)
Occupational	50 mSv (external plus internal doses)	50 mSv (external plus internal doses)	12.5 mSv per quarter for the whole body	50 mSv	20 mSv averaged over 5 years (100 mSv in 5 years), with a further provision that the effective dose should not exceed 50 mSv in any single year
Lens of eye	150 mSv	150 mSv	12.5 mSv per quarter	150 mSv	150 mSv
Hand and forearms, feet and ankles	500 mSv	500 mSv	187.5 mSv per quarter	500 mSv	500 mSv
Skin	500 mSv	500 mSv	75 mSv per quarter	500 mSv	500 mSv
Cumulative	None	None	50(N-18) mSv; N =age (years)	10 mSv × age (years)	100 mSv in 5 years

*The 2005 ICRP recommendations continue to endorse these limits.

DOE, Department of Energy; ICRP, International Commission on Radiological Protection; NCRP, National Commission on Radiological Protection; NRC, Nuclear Regulatory Commission; OSHA, Occupational Safety and Health Administration.

based on recent findings from the Radiation Effects Research Foundation and the United Nations Scientific Committee on the Effects of Atomic Radiation. These findings suggest that radiation risk has substantially increased for workers by a factor of about four. The most recent dose-limit recommendations were reduced in order to be commensurate with the basic philosophy that radiation workers ought to have at least the same level of protection as those in safe industries (about 1 death per 10,000 workers per year).[9]

Radon

Occupational exposure limits for radon and radon progeny (radon daughters) were derived to protect the health of underground miners over a working lifetime of 30 years.[10–11] When radon gas and radon progeny are inhaled, the radiation dose is primarily caused by the (short-lived) radon progeny. Because it was not feasible to routinely measure individual radon progeny, the concept of the working level (WL) was introduced and defined as 1.3×10^5 MeV of alpha radiation emitted from the short-lived radon progeny in 1 liter of air. An exposure of 1 WL for a working period of 1 month (170 hours) results in a cumulative exposure of 1 working level month (WLM). A WLM, which is the common unit of measurement for human exposure to radon progeny, is the basis for the occupational exposure limits (Table 12C-5).

Radiation Protection Programs

Radiation protection programs, which reflect application of management's responsibilities for radiation protection and safety, implement policies, procedures, and organizational structures commensurate with the nature and extent of radiation risks (Fig. 12C-4). Three principles of radiation protection and safety include justifying, limiting, and optimizing exposures. Radiation exposures may be justified if an activity produces sufficient benefit—considering social, economic, and other relevant factors—to offset the harm it might cause exposed workers.

Table 12C-5. Radon Gas and Radon Progeny Occupational Exposure Limits

Time Period	Description	Units	Limit and Application	
IAEA annual average over 5 years	Potential alpha energy intake	J	0.017	Progeny
		MeV	1.1×10^{11}	Progeny
	Potential alpha energy exposures	J·h/m^3	0.014	Progeny
		Bq·h/m^3	2.5×10^6	Gas
		WLM	4.0	Progeny
		pCi·h/L	$6.76 \times 10^{4*}$	Gas
IAEA maximum in a single year	Potential alpha energy intake	J	0.042	Progeny
		MeV	2.6×10^{11}	Progeny
	Potential alpha energy exposures	J·h/m^3	0.035	Gas
		Bq·h/m^3	$6.3 \times 10^{6*}$	Gas
		WLM	10	Progeny
		pCi·h/L	$1.7 \times 10^{5*}$	Gas
NIOSH recommended exposure limits	Potential alpha energy exposures	J·h/m^3	0.0035	Gas
		Bq·h/m^3	$6.3 \times 10^{5*}$	Gas
		WLM	1.0	Progeny
		pCi·h/L	$1.7 \times 10^{4*}$	Gas
	Average work shift concentration	WL	0.083	Progeny
		pCi/L	8.3*	Gas
OSHA permissible exposure limits	Average concentration for workers under age 18	pCi/L	3	Gas
	Average concentration for adult workers (40-hour week)	pCi/L	100	Gas
	Must post airborne radioactivity for weekly average concentrations	pCi/L	25	Gas

*These time-integrated activity concentrations relate to the equilibrium equivalent concentration of radon. The associated time integrated concentration of radon gas is obtained by dividing the appropriate equilibrium factor (usually recognized as 0.5).

IAEA, International Atomic Energy Agency; NIOSH, National Institute for Occupational Safety and Health; OSHA, Occupational Safety and Health Administration; J, joules; MeV, million electron-volts; h, hours; m, meters; Bq, becquerels; WLM, working level months; Ci, curies; L, liters; WL, working levels.

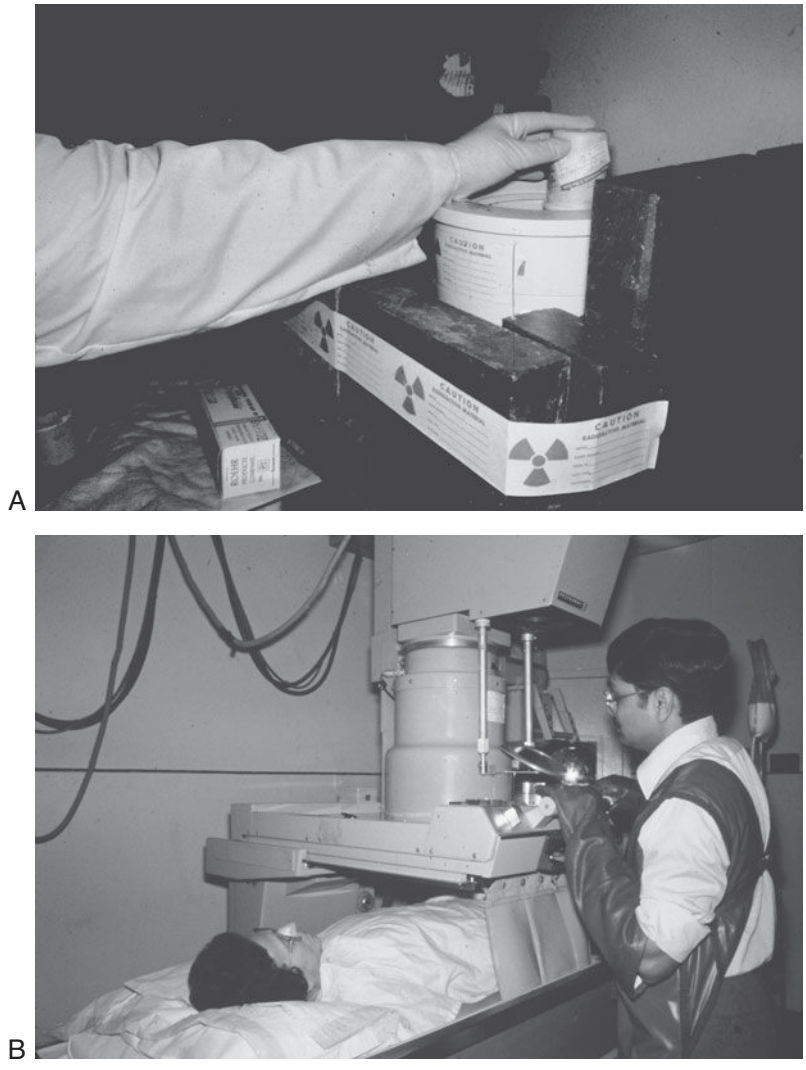

Figure 12C-4. Examples of protection of health care workers from ionizing radiation. (A) Worker wearing leather glove in nuclear medicine supply storage area. (B) X-ray technician wearing protective apron. (Photographs by Earl Dotter.)

Dose limitation is necessary to limit the risk of stochastic effects from exposures considered to be unacceptable. Protection and safety should be optimized to ensure that the magnitude of worker doses, the number of workers exposed, and the likelihood of incurring exposure are all kept as low as reasonably achievable, after accounting for social and economic factors (cost and cost-effectiveness of engineering controls, emergency response activities, and the potential impact to the public). A "safety culture," which contributes to a successful radiation protection program, depends on management's commitment to encourage a questioning and learning attitude toward protection and safety and to discourage complacency. A neutral or negative attitude by management toward radiological protection can cause unnecessary or excessive radiation exposure in the workplace. So can inaccurate or incomplete radiation surveys, inadequately prepared radiological work permits, failure of radiological technicians to react to changing or unusual conditions, failure of workers to follow procedures, and inadequate involvement of supervisors.

The basic structure of a radiation protection program should include the following:

1. Assignment of responsibilities to various levels of management
2. Designation of controlled or supervised areas
3. Local rules for workers to follow and the supervision of work (site-specific considerations and accountability procedures)
4. Arrangement for monitoring workers and the workplace with appropriate dosimeters and instrumentation
5. A system to record and report all relevant information to appropriate decision makers
6. Education and training programs on the nature of the hazards, protection, and safety
7. Methods to periodically review and audit performance of the program
8. Emergency response plans
9. A health surveillance program
10. A quality assurance and quality control program

Emergency Response and Recovery

Terrorist attacks have focused attention on preparedness to address large-scale radiological and nuclear threats as well as threats of small-scale industrial radiation releases. Since 9/11, response capabilities of federal and state governments in the United States have been improved by creating the Department of Homeland Security, consolidating many federal emergency-response plans into the National Response Framework, and providing funding to state and local governments. Emergency-response workers may be highly exposed to radiation at levels requiring additional precautions and medical intervention. (See Chapter 37.) Most important for health professionals responding to emergencies is to always treat life-threatening injuries first before addressing radioactive contamination or radiation exposure. Even if people have been heavily irradiated or contaminated with radioactive material, they should be first evaluated for other forms of injury, such as mechanical trauma, burns, and smoke inhalation. One should be especially cautious of wounds containing metallic objects because these can be a major source of radiation. Decisions during an initial response to a large-scale radiological incident are based on protecting life and critical infrastructure. Decisions during the recovery phase that follow include consideration of law enforcement, mass casualties, damage to infrastructure, psychosocial impacts, and environmental concerns.[12] The Department of Homeland Security provides guidance to state and local officials for determining appropriate clean-up levels under various circumstances.[13] *Protective action guides* (PAGs) support actions to protect the public and emergency workers responding to or recovering from a radiological or nuclear incident.

Helpful Government Web Sites

Centers for Disease Control and Prevention (CDC). Available at: http://www.cdc.gov/nceh/radiation/default.htm

Environmental Protection Agency Radiation Protection Programs. Available at: http://www.epa.gov/radiation/

Food and Drug Administration Center for Devices and Radiological Health. Available at: http://www.fda.gov/RadiationEmittingProducts/default.htm

Federal Emergency Management Agency (FEMA). Available at: http://www.fema.gov/

International Atomic Energy Agency (IAEA). Available at: http://www.iaea.org/

National Library of Medicine. Available at: http://www.remm.nlm.gov/

Occupational Safety and Health Administration (OSHA). Available at: http://www.osha.gov/SLTC/radiation/index.html

Helpful Web Sites of Scientific Organizations

American Association of Physicists in Medicine. Available at: http://www.aapm.org/

American Association of Radon Scientists and Technologists. Available at: http://www.aarst.org/

Conference on Radiation Control Program Directors. Available at: http://www.crcpd.org/

Health Physics Society. http://www.hps.org/

International Commission on Radiological Protection. Available at: http://www.icrp.org/
International Radiation Protection Association. Available at: http://www.irpa.net/

NONIONIZING RADIATION

Everyone is exposed daily to nonionizing radiation, which is both naturally occurring and manmade. It can be beneficial or detrimental to those exposed. Like ionizing radiation, one cannot see it—except for visible light (wavelength = 400 to 760 nm), taste it, or smell it. But unlike ionizing radiation, one may be able to feel it by sensing heat or through electrostimulation. The phenomenon of hearing certain radio frequencies is also a well-established biological effect with no known adverse health consequences. A quiet environment is needed for these radio frequency (RF)-induced sounds (similar to other common sounds) to be heard. Nonionizing radiation is the energy absorbed by any material without causing ionization (ejection of electrons surrounding the atoms within the material). It takes many forms, including television and radio signals, radar, pager and cordless as well as cellular phone signals, microwaves, visible light, infrared and ultraviolet light, and lasers. The presence of nonionizing radiation is growing, fueling anxiety and speculation about its possible adverse health effects. Levels of exposure will continue to grow as technology advances and as society increasingly demands the conveniences it brings.

The electromagnetic spectrum includes ionizing and nonionizing radiation (Fig. 12C-5). All nonionizing radiation presents in *electromagnetic fields* (EMFs), which can be described by frequency or corresponding wavelength by the equation:

$$\lambda = c / f$$

where λ = wavelength in meters (m), c = velocity of light (about 300,000,000 meters per second), and f = frequency in cycles per second (Hertz, or Hz). Most of the nonionizing radiation spectrum is partitioned into specified radiofrequency bands. Hazards potentially associated with exposure to EMFs in various bands may result in *(a)* currents produced in the body by contact with energized sources or without such contact (electrostimulation), *(b)* increased core-body temperature, or *(c)* increased body surface temperature (Table 12C-6). How efficiently these fields interact with the body depends on several factors. For example, materials with high water content (muscles) absorb EMF energy at higher rates than dry materials. The absorption rate is higher when *(a)* the incident electric field is parallel to the body, and *(b)* the incident magnetic field is perpendicular to a larger cross-sectional area. Sharp corners, edges, and points concentrate electric fields. Depth of penetration of EMF energy decreases as conductivity or frequency increases and as wavelengths decrease.

Electric fields (E) exist when electric charges exert forces on one another, regardless of whether they are in motion. Electric field strength describes the strength of forces on charges (in volts per meter, V/m). Electric fields can be visualized as lines of force that emanate from a positively charged object to a negatively charged object.

Magnetic field strength (H), measured in amperes per meter (A/m), is associated with the strength of these additional forces on moving charges. An ampere is the SI unit for electric current. Magnetic fields exist in a direction perpendicular to the direction of the electrical current, and their intensity is proportional to amount of current present. Magnetic fields are related to another quantity called the magnetic flux density (B) by $B = \mu H$, where μ is the permeability of the medium. B is the sum of the components of magnetic fields passing through a given area and is the quantity used for hazard evaluation. Its SI unit is the telsa (T) and the conventional unit is the gauss (G) (1 T = 10,000 G). A useful factor to convert B and H is 1 G = 80 A/m.

The relationship between the E- and H-fields is described by the *power density*, which is the power incident on a surface per unit surface area. Abbreviated as S, it can be calculated from E- or H-field measurements by the following equation:

$$S = E^2 / 377 \text{ or } 377H^2$$

where S = power density in watts per square meter (W/m^2 or VA/m^2), E = electric field

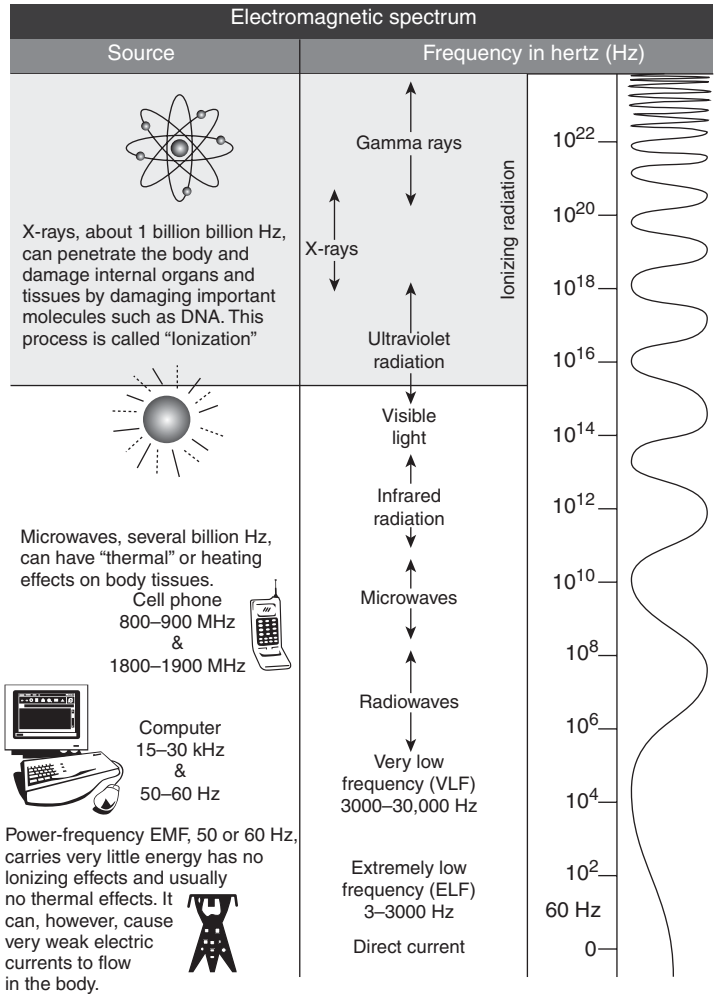

Figure 12C-5. Electromagnetic spectrum. (From EMF in the workplace. Washington, DC: Department of Energy, National Institute for Occupational Safety and Health, and National Institute of Environmental Health Sciences, 1996.) Note: Extremely low frequency (ELF) is defined as 30 to 300 Hz by the National Council on Radiation Protection and Measurements Report 119.

strength measurement (V/m), H = magnetic field strength measurement (A/m), and 377 = the constant = the impedance of free space (in ohms, [Ω], or V/A). Impedance describes the resistance experienced by electromagnetic radiation traveling through space. The quantitative nature of the electromagnetic fields changes with increasing distance from the source. These distances are classified as near-field, far-field, and intermediate-field. In the near field, a distance from the source to about one-sixth of the associated wavelength, E- and H-fields are not perpendicular because the radiator is not an ideal source. These differences prevent the use of the power density equation, which was cited earlier, and require the measurement of individual components of the E- and H-field strengths. At distances greater than about one-half of the wavelength from the source, called the far field, the E- and H-fields are perpendicular, allowing the use of the power density equation. In this region, the E- and H-field strengths decrease linearly with distance from the source, and the power density decreases as the square of the distance from the source. The distance between the near and far fields, the intermediate field, is a transitional region where the power density equation still does not apply. The E- and H-field strengths decrease linearly with distance following a 1/r relationship. (The r is the radius from the source.)

Exposure Limits

The transfer of energy from electric and magnetic fields in any material is described by the

Table 12C-6. Frequency Bands and Their Associated Biological Impacts

Band	Frequency Range (Hz)	Wavelength Range (m)	Biological Impact
SELF Sub extremely low frequency	0–30	0–10^7	0–10^5 Hz, 30×10^6–3,000 m:
ELF Extremely low frequency	30–300	10^7–10^6	Electrostimulation (primary dosimetric parameter is internal current density)
VF Voice frequency	300–3,000	10^6–10^5	
VLF Very low frequency	3,000–3×10^4	10^5–10^4	
LF Low frequency	3×10^4–3×10^5	10^4–10^3	10^5–6×10^9 Hz, 3,000–0.05 m:
MF Medium frequency	3×10^5–3×10^6	10^3–10^2	Specific absorption rates (heating effects)
HF High frequency	3×10^6–3×10^7	10^2–10	
VHF Very high frequency	3×10^7–3×10^8	10–1	
UHF Ultrahigh frequency	3×10^8–3×10^9	1–0.1	
SHF Super-high frequency	3×10^9–3×10^{10}	0.1–10^{-2}	Above 6×10^9 Hz, below 0.05 m:
EHF Extremely high frequency	3×10^{10}–3×10^{11}	10^{-2}–10^{-3}	Surface heating (radiant)
SEHF Supra extremely high frequency	3×10^{11}–3×10^{12}	10^{-3}–10^{-4}	
Infrared radiation	**IR-C**	0.3 μm–1 mm	Corneal burns, thermal skin burns
	IR-B	1.4 μm–0.3 μm	
	IR-A	0.7 μm–1.4 μm	Retinal burns, cataracts of lens, thermal skin burns
Visible light		400–760 nm	Retinal burns, thermal skin burns
Ultraviolet radiation	**UV-A**	400–320 nm	Cataract of lens, thermal skin burns
	UV-B	320–280 nm	Corneal injuries Cataracts of lens,
	UV-C	280–200 nm	photokeratitis, photoconjunctivitis, erythema

specific absorption rate (SAR). "Specific" refers to the normalization to mass of the material exposed, "absorption" refers to the absorption of the energy in a specific medium (tissue), and "rate" refers to the time rate of change of the energy absorption. The SAR is the most reliable indicator or predictor of the potential for biological effects in test animals and a measure of what is happening inside the human body. It is expressed in units of watts per kilogram (W/kg) or milliwatts per gram (mW/g). Since SAR is difficult to evaluate or measure outside the laboratory, the measurable quantities of magnetic or electric field strengths and power density as well as induced and contact currents are used to define the RF environment (Fig. 12C-6). They have been correlated with SAR to determine the maximum permissible exposure (MPE) levels (Table 12C-7). In the far field (greater than about one-half wavelength from RF source), measuring field strengths or power density provides reliable exposure assessments. In the near field or in contact with RF sources and/or other metallic objects (where many occupational exposures occur), induced and contact current measurements provide the most reliable exposure evaluations. Measuring field strengths or power density is unreliable near or in contact with RF sources or other metallic objects. The MPE values provided are those from the Institute of Electrical and Electronics Engineers, Inc., standard, which incorporate the latest scientific findings and recommendations for occupational exposures.[14] Guidelines for limiting RF exposure

Figure 12C-6. Researchers conducting radiofrequency (RF) measurements near television broadcast antenna atop a high-rise building.

Table 12C-7. Maximum Permissible Exposure for Occupational Environments[a]

Frequency Range (MHz)	RMS E-Field[b] Strength (V/m)	RMS H-Field[b] Strength (A/m)	Power Density (S) E-field, H-field (W/m^2)	Averaging Time $\|E\|^2$, $\|H\|^2$ or S (min)
0.1 - 1.0	1,842	$16.3/f_M$	$(9{,}000/f_M^2, 100{,}000/f_M^2)$[c]	6
1.0 - 30	$1{,}842/f_M$	$16.3/f_M$	$(9{,}000/f_M^2, 100{,}000/f_M^2)$	6
30 - 100	61.4	$16.3/f_M$	$(10{,}100{,}000/f_M^2)$	6
100 - 300	61.4	0.163	10	6
300 - 3,000			$f_M/30$	6
3000 - 30,000			100	$19.63/f_G^{1.079}$
30,000 - 300,000			100	$2.524/f_G^{0.476}$

Induced and Contact Currents: Occupational Environment

	Maximum Current (mA)[d]			
			Contact	
	Through Both Feet	**Through Each Foot**	**Grasp**	**Touch**
0.003 - 0.1	$2.0f_K$	$1.0f_K$	$1.0f_K$	$0.5f_K$
0.1 - 110	200	100	100	50

F_K = frequency in kHz; f_M = frequency in MHz; f_G = frequency in GHz.

[a] An occupational environment is also called a controlled environment—an area where the occupancy and activity of those within it are subject to control and accountability, as established by a radio frequency (RF) safety program for the purpose of protection from RF exposure hazards.

[b] For exposures that are uniform over the dimension of the body, such as certain far-field exposures, the exposure field strengths and power densities are compared with the MPEs in this table. For nonuniform exposures, the mean values of the exposure fields, as obtained by spatially averaging the squares of the field strengths or averaging the power densities over an area equivalent to the vertical cross-section of the human body, or a smaller area depending on the frequency, are compared with the MPEs in this table.

[c] These plane-wave equivalent power density values are commonly used as a convenient comparison with MPEs at higher frequencies and are displayed on some instruments in use.

[d] The averaging time for determination of contact current limits is 6 minutes.

Source: Data from the Institute for Electrical and Electronics Engineers (2005), Reference #14.

have also been developed by several other scientific organizations and government agencies, but the differences are minor and work is underway to harmonize the various exposure limits.[15–16] In the case of exposure of the whole body, a human adult (height = 175 cm) absorbs RF energy most efficiently when the wavelength is 40% of the long axis of the body and parallel to the incident E-field vector. This occurs at a frequency of about 70 megahertz (MHz). The RF exposure limits, which are called basic restrictions, reflect this dependency on frequency and were derived from a SAR of 4 W/kg for those frequencies associated with heating affects (100 kiloHertz to 3 gigaHertz). In terms of human metabolic heat production, 4 W/kg represents a moderate activity level, such as with housecleaning. A safety factor of 10 was applied resulting in an RF exposure limit of 0.4 W/kg, virtually an indistinguishable heating effect from normal temperature variation, exercise, or exposure to the sun. For localized exposures in an occupational environment where the field strength is more than 20 times the spatial average, the SAR should not exceed 10 W/kg. For the extremities and the pinna (the cartilaginous projection portion of the outer ear consisting of the helix, lobule, and anti-helix), the SAR should not exceed 20 W/kg. Radio frequency exposures below this level are intended to prevent adverse health effects. Exposures in excess of the limits are not necessarily harmful. However, without intended lifesaving or medical benefits, these situations are not recommended.

Interpreting Radio Frequency Measurement Data

Occupational limits (sometimes referred to as a controlled environment) apply to persons exposed at work, provided they are fully aware of the potential for their exposure and can exercise control over it. One should understand there are three fundamental concepts when interpreting measurement data: *(a)* the difference between exposure and emission limits, *(b)* spatial averaging, and *(c)* time averaging.

Emission limits are the maximum power output authorized by government authorities for companies or individuals. However, these transmitting signals are often not emitted at the maximum power output. This is especially true for cell-phone base stations or towers, since the amount of power used is proportional to the number of calls handled. For this reason, it is important to note that the emission limit (maximum power output) may not be directly related to exposure potentials. Unlike emission limits, exposure guidelines apply to *exposure limits*, and they are relevant only to locations that are accessible by workers.

Spatial Averaging

The exposure limits are based on the concept that the exposures are applied to a whole-body averaged SAR. This means that spot measurements exceeding the stated exposure limits do not imply noncompliance or harmful exposure scenarios if the spatial average of RF fields over the body does not exceed limits. A spatial average measurement may consist of three or more measurements averaged together that span a length of an adult.

Time Averaging

Another feature of the exposure guidelines is that EMF exposures should be averaged over a 6-minute period for workplaces (controlled environments). To apply field measurements to exposure limits properly, one must consider the length of time individuals are exposed. For example, during any given 6-minute period, workers could be exposed to twice the applicable limit for 3 minutes as long as they are not exposed for the preceding or following 3 minutes. Similarly, a worker could be exposed at three times the limit for 2 minutes as long as no exposure occurs during the preceding or subsequent 4 minutes.

Protective Measures

Engineering Controls

Protection of workers from unnecessary or excessive exposure to RF radiation is accomplished through engineering and administrative controls. Engineering controls are preferred since they eliminate or reduce the potential exposures at the source, but they require a sophisticated level of knowledge to install. Improperly installed controls may enhance worker exposures. Interlocks, shielding, bonding,

grounding, and filtering are some of the more common controls employed. The Occupational Safety and Health Administration (OSHA) requires a lock-out/tag-out program for working with sources of hazardous energies, which may include installing many of the RF controls described earlier.

The effectiveness of shielding materials varies with the material, geometry, frequency, and where the field reduction is measured. Some are more effective for reducing electric fields, while others are more suitable for reducing magnetic fields. One of the most recognizable types of shielding is that used on microwave ovens. The perforated screen is designed to allow penetration of visible light (wavelength about 0.7×10^{-6} to 0.4×10^{-6} meters, or 430 million to 750 million Hz), but it prevents leakage of microwave radiation (wavelength about 12 cm, or 2,450 MHz). Perforated or continuous shielding materials reduce exposures by reflection, absorption (attenuation), and internal reflection. The proper selection of material is complex and should be done by qualified individuals.

Techniques that may supplement the use of engineering controls include prudent placement of RF sources, resonant frequency shift, and personal protective equipment (shoes, clothing, and special suits). Consideration should be given to building-construction materials and layout when installing RF equipment to reduce or prevent unnecessary enhancement of reflected energy at the worker's location. If the operating frequencies are around 10 to 40 MHz, the whole-body SAR may be reduced by *resonant frequency shift*, separating the body from the ground plane by a small distance with electrically insulating materials. This measure reduces the worker's absorption characteristics by reducing the flow of current from the body to a grounded surface. Resonant frequency shift may be especially useful for dielectric-heater (plastic-sealer) operators by having them stand on nonconductive platforms made of wood or rubber. For factory worksites, metal-reinforced concrete floors act as ground planes. Footwear that reduces the grounding effect also achieves the same effect as a resonant frequency shift. The level of RF exposure reduction is dependent on the RF frequency and the types of shoes and socks worn by the worker. Wool socks and rubber-soled shoes provide the greatest reduction for frequencies below 100 MHz (wavelengths above 3 meters). Protective suits may be helpful when work must be done in "hot" areas, such as continuous-radar, onboard naval vessels, and some communication and broadcast environments. Suit material is typically wool, polyester, or nylon impregnated with a highly conductive threaded metal. Some are more effective than others depending on frequency, orientation of the worker in the environment relative to the incident electric fields, and construction of openings for feet, hands, and head. Washing these suits may reduce their protective capabilities. Some experts recommend against use of RF-protective suits because they may be hazardous to individuals nearby the wearer, and they may increase the hazard to the wearer by allowing closer proximity to open circuits that may act as secondary sources.

Administrative Controls

Administrative controls include increasing the distance between the source and workers (often used and easy to bypass), controlling the duration of exposure, restricting access, placing warning signs, providing training commensurate with the level of potential hazard, and real-time monitoring via dosimetry. Horizontal and vertical distance should be considered when determining the appropriate distance, which is often the distance that results in a radiation level equal to the limit (the *hazard distance*). There is no simple way to calculate the reduction of field strength with distance since the calculation depends on so many factors; however, some researchers measured magnetic field strengths that showed a reduction by $1/r^5$ for induction heaters.[17] Controlling the duration of exposure is achieved by applying the time-averaging technique discussed earlier. Finally, real-time monitoring devices (dosimeters) are especially useful in identifying potentially harmful exposures, allowing the recipient to take protective actions and reduce risk of injury. Dosimeters provide an audible and visual alarm when exposures exceed a predetermined level (usually 50% of the maximum permissible exposure), and they allow the wearer to quickly identify if changes occur during work activities.

Health Effects Associated with Electromagnetic Frequencies below 100 kHz

Exposures to electric and magnetic fields emanating from the generation, transmission, and use of electricity have been studied extensively. Recommendations of various scientific organizations and regulatory agencies acknowledge controversy regarding the potential health effects of chronic low-level EMF exposures. However, there is no convincing evidence of a health risk.[18,19] One of the most comprehensive reviews of health effects associated with extremely low frequency (ELF) exposures was published by the International Agency for Research on Cancer (IARC),[20] which found the following:

1. Limited evidence in humans for the carcinogenicity of ELF magnetic fields in relation to childhood leukemia
2. Inadequate evidence in humans for the carcinogenicity of ELF magnetic fields in relation to all other cancers
3. Inadequate evidence in humans for the carcinogenicity of static electric or magnetic fields and ELF electric fields
4. Inadequate evidence in experimental animals for the carcinogenicity of ELF magnetic fields
5. No available data for the carcinogenicity of static electric or magnetic fields and ELF electric fields in experimental animals

IARC concluded that ELF magnetic fields are possibly carcinogenic to humans, and that static electric and magnetic fields and ELF electric fields are not classifiable as to their carcinogenicity to humans.

Health Effects Associated with Electromagnetic Fields above 100 kHz

More than 100 million Americans use wireless communication devices, with 50,000 new users daily.[21] If the use of wireless communication devices is ever associated with even the slightest increase in risk of adverse health effects, it could become a significant public health problem. At frequencies above 100 kHz, studies support the basic restrictions and MPE recommendation described earlier. These recommendations were made on the basis of a comprehensive review of the scientific data to protect against established adverse health effects from RF exposures. An adverse health effect is defined as a harmful change in health that is supported by the consistent findings in the peer-reviewed literature, demonstrated by independent laboratories, with consensus in the scientific community. The established adverse health effects associated with RF exposure above the basic restrictions and MPEs are as follows: *(a)* aversive or painful electrostimulation due to excessive RF internal electric fields, *(b)* RF shock or burns due to contact with excessively high RF voltages, *(c)* heating pain or tissue burns due to excessive localized RF exposures, and *(d)* behavioral disruption, heat exhaustion, or heat stroke due to excessive whole-body RF exposures.[14] Adverse effects do not include effects such as biological effects (sensations) without a harmful health effect, indirect effects caused by electromagnetic interference with electronic devices, or changes in subjective feelings of well-being that are a result of anxiety about RF effects or impact.

Debate continues on the level of protection necessary to prevent long-term health effects from RF exposures. The World Health Organization (WHO) and many European countries promote a precautionary approach by discouraging the widespread use of mobile phones by children for nonessential calls. Children may be more likely to develop adverse effects because their nervous systems are still developing, and they will face a lifetime of various hazardous exposures.[22] The Russian National Committee on Non-Ionizing Radiation Protection extends the WHO recommendations for children to pregnant women and to those suffering from specific diseases, and it recommends that duration of cellular phone calls be limited to 3 minutes each with at least 15 minutes between calls. The United States does not necessarily endorse the precautionary approach because without clear, convincing epidemiologic evidence that a health hazard exists from RF exposures, this approach could negatively impact growth and development of the telecommunications industry.

Cancer-related studies on animals provide no evidence of physiological, pathological, or

disease-specific effects of long-term RF exposures. Likewise, epidemiological studies show no clear or consistent evidence to indicate a causal role of RF exposures in human cancer or other disease endpoints at exposures below the basic restrictions and MPEs. However, it is scientifically impossible to prove absolute safety (the null hypothesis) of any physical agent. Many of the original studies lacked adequate exposure-assessment information and biological measures, and included confounding factors, such as multiple sources. More recent studies have benefited from improved dosimetry and modeling techniques, as well as better clinical testing protocols.

Infrared and Ultraviolet Radiation

Infrared radiation (IR) lies at frequencies higher than those of radar waves and microwaves (Table 12C-6). Nearly half of the sun's radiant energy is emitted as IR. Infrared radiation is highly absorbed by water and the Earth's atmosphere and invisible to the eye. However, its warmth can be detected by the skin. All objects with temperatures above absolute zero emit IR. In industry, significant levels of IR are produced directly by lamps and indirectly by heat sources, such as heating and drying devices. The primary biological effect of IR is thermal due to absorption in the water within body tissues. For this reason, IR cannot penetrate the skin, but leaves a sensation of heat, which often serves as an adequate warning sign to take protective action or risk skin burns. The lens of the eye is particularly vulnerable to IR because the lens has no heat sensors and a poor heat-dissipating mechanism. Cataracts may be produced by chronic IR exposure at levels far below those that cause skin burns. Occupations typically at risk of IR exposure include glass blowers, furnace workers, foundry workers, blacksmiths, solderers, oven operators, those who work near baking and drying heat lamps, and movie projectionists. Like RF radiation, IR exposure limits are frequency based; however, they represent conditions under which it is believed that nearly all healthy workers may be repeatedly exposed without acute adverse effects. The limits for IR most recognized in the scientific community are published by the American Conference of Governmental Industrial Hygienists (ACGIH).[23] Control of an IR hazard requires *(a)* shielding of the IR source and eye protection with appropriate IR filters, *(b)* maximizing the distance between workers and the IR source, and *(c)* reducing the time spent in areas with high levels of IR exposure.

Ultraviolet radiation (UVR) is produced by the sun and artificially by incandescent, fluorescent, and discharge types of light sources. It is characterized by three distinct energy bands known as UV-A (400 to 320 nm), UV-B (320 to 280 nm), and UV-C (280 to 200 nm). The first two bands are principal UV components in sunlight. Nearly all UV-A reaches the Earth's surface, but most UV-B is absorbed by the stratospheric ozone layer. UV-C is completely absorbed by the ozone layer and oxygen in the air, but it can be artificially produced. Industrial sources of UVR include welding arcs, plasma torches, electric arc furnaces (full spectrum of UVR), germicidal and black-light lamps (mostly UV-C), and certain type of lasers (full spectrum of UVR). Because wavelengths of UVR are so short, UVR presents a surface heating hazard.

The most common health effect from overexposure to UVR is sunburn (erythema). Chronic low-level UVR exposure from the sun is also associated with various skin effects, including skin cancer (basal cell carcinoma, squamous cell carcinoma, and malignant melanoma), premature aging of the skin, solar elastosis (wrinkling), and solar keratoses (premalignant lesions). Basal cell carcinoma and malignant melanoma are more strongly associated with a history of multiple episodes of sunburn, whereas squamous cell carcinoma is associated with total exposure. UVR exposure has also been associated with suppressing the immune system and developing cortical cataracts (UV-B exposure). Photosensitizing agents, such as coal tar, plants (including figs, lemon and lime rinds, celery, and parsnips), and pharmaceutical drugs (including chlorpromazine, chlorpropamide, and tolbutamide) can increase susceptibility to UVR. All these effects vary with individual susceptibilities. Lighter skin is more susceptible than darker skin, and people on medicine for diabetes are more susceptible. And they vary with geographic location (UVR levels are highest near the equator, at higher altitudes, when the sun is directly overhead,

when there is no cloud cover or ozone coverage, during the summer, and in highly reflective environments. Acute high-level UVR exposures, especially from UV-B, result in eye injuries, which are often only recognized several hours after the exposure. Photokeratitis (inflammation of the cornea) and photoconjunctivitis (inflammation of the thin transparent mucous membrane lining the inner surface of the eyelids) are usually reversible within several days. Intense UVR exposure also has an indirect impact on health through its ability to cause photochemical reactions. Small amounts of oxygen and nitrogen can be converted into ozone and oxides of nitrogen, which are respiratory irritants. Halogenated hydrocarbon solvent vapors can decompose into toxic gases, such as perchloroethylene decomposing to hydrogen chloride and trichloroethylene decomposing to phosgene. Chronic low-level UVR exposures can be controlled by use of protective clothing, eyewear, and sunscreen lotions, and by reduction of duration of exposure. Controlling UVR from acute high-level photochemical exposures may require local exhaust ventilation and isolation of UVR sources from industrial processes that involve solvents. Only qualified personnel should determine the effectiveness of any particular form of personal protection. (See Chapter 22.)

Laser Radiation

Laser is an acronym for *l*ight *a*mplification by the *s*timulated *e*mission of *r*adiation. Uses in industry include heat treatment, glazing, alloying, cladding, cleaning, brazing, soldering, conduction welding, penetration welding, cutting, hole drilling, marking, trimming, and photolithography (Fig. 12C-7).[24] Health and safety decisions are based on the class of laser and the wavelength of the laser source. The hazard classification system places lasers into four categories depending on their potential to cause harm from direct beam exposures (Table 12C-8). These exposures may result in at least four types of injury to the eyes and skin, each requiring a special consideration for selecting the appropriate personal protective equipment (Table 12C-9). However, nonbeam laser hazards constitute the greatest source of noncompliance with federal safety codes. Sources of nonbeam hazards include *(a)* improper electrical design or improper use of grounding, components, or shielding; *(b)* lack of knowledge for production of laser-generated air contaminants (LGACs); *(c)* unwanted plasma radiation; *(d)* excessive noise levels; *(e)* inadequate ventilation controls; *(f)* fire hazards; *(g)* explosive hazards from high-pressure tubes; *(h)* exposure to toxic chemicals

Figure 12C-7. Carpenter using laser machinery. (Photograph by Earl Dotter.)

Table 12C-8. Laser Classification

Class of Laser*	Hazard Potential
1	Pose no potential for injury. No safety measures required to either the eye or skin.
2, 2a	Visible beam posing no significant potential for injury. Blinking response limits exposure.
3, 3a, 3b	Modest potential for injury. Normal aversion response is not sufficient to limit eye exposure to a safe level. Skin hazards normally do not exist. May require safety precautions and personal protective equipment. Class 3b lasers require more safety precautions than class 3a.
4	Serious potential for injury of the eye and skin. Requires safety precautions and personal protective equipment. Diffuse reflection viewing hazard. Potential fire hazard. Most laser systems for cutting, heat treating, and welding are Class 4.

*When Class 3 and 4 lasers are fully enclosed to prevent potentially hazardous laser radiation exposures, the system may be classified as a Class 1 system.

and laser dyes; and *(i)* fire hazards. Most of these hazards are associated with Class 3b and 4 lasers. In practice, it is always desirable to totally enclose the laser and beam path to prevent both direct-beam and nonbeam exposures.

Unlike most other workplace hazards, there is generally no need to perform workplace measurements for lasers because of highly confined beam dimensions, minimal likelihood of changing beam paths, and the difficulty and expense of using laser radiometers. However, measurements must be performed by manufacturers to ensure proper laser classification. Laser safety standards are published by government agencies, and by independent and industrial standards organizations. In the United States, the American National Standards Institute (ANSI) has developed the *Standard for the Safe Use of Lasers* (Z136.1) and publishes general safety requirements for users. Although this standard is not a law, it forms the basis for OSHA and many states' regulations. There are other laser safety standards and state-specific regulations, but they apply primarily to Class 3b and 4 installations and maintenance activities.

The International Organization for Standardization (ISO) and International Electrotechnical Commission (IEC) have published standards similar to those in the United States. Two requirements in the ISO documents that affect manufacturers are that *(a)* all systems must be Class 1 during operation, and *(b)* manufacturers must specify which materials that equipment is designed to process. A Class 1 laser rating can be achieved by installing appropriate engineering controls.

Controlling all aspects of potential laser exposures is complex and requires a qualified individual to assess direct and nonbeam hazards. Control measures include process isolation, local-exhaust and building ventilation, training and education, restricted access, proper housekeeping, preventive maintenance, and use of appropriate personal protective equipment.

Table 12C-9. Laser Injuries

Type of Hazard	Laser Wavelength (nm)	Target Tissue	Comment
Ultraviolet photochemical injury	180 to 400	Skin	Eye protection is required whenever a bluish-white light is seen at the laser focal point.
	180 to 400	Cornea	
	295 to 380	Lens	
Blue-light photochemical injury	400 to 550	Retina	Retinal burn (has been termed "eclipse blindness")
Thermal injury	400 to 1,400	Retina	Nd: YAG lasers pose the greatest risk because beam image can be intensified about 100,000 times.
	1,400 nm to 1 mm	Skin	Most common injury from laser radiation exposure
		Cornea	Biggest concern with CO_2 lasers
		Conjunctiva	
Near-infrared thermal injury	800 to 3,000	Lens	Results from molten metal or large, heated surface during treatment. This hazard is only of concern for repeated, chronic exposures.

Nd: YAG, neodymium-doped yttrium aluminum garnet.

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The findings and conclusions in this chapter are those of the author and do not necessarily represent the views of the Environmental Protection Agency.

13

Biological Hazards

Mark Russi

Infectious hazards exert a massive toll on humanity. They will continue to do so despite our best efforts, aided inexorably by natural selection, population pressure, poverty, and an increasingly contiguous world. To distinguish occupational infections among the vast array of infections caused by human pathogens requires an assessment of situations likely to *(a)* enhance contact between workers and microbes or *(b)* alter the usual microbial environment.

Examples include workplaces in which the sick are cared for (Figs. 13-1 and 13-2); contact occurs with animals and the zoonotic illnesses they may harbor; enhanced contact is likely with arthropod disease vectors or environmental fungi; or exposure to an altered range of diseases in the general environment exists, such as in tropical countries and settings where many people live or train in close proximity.

In developed countries, the most important setting in which increased contact exists with a broad range of human diseases is health care, where workers are exposed to pathogens spread by direct, droplet, airborne, fecal-oral, or bloodborne transmission. Infectious diseases spread by airborne or droplet transmission include tuberculosis (TB), influenza, pertussis, varicella, parvovirus B19, measles, and rubella. Principal bloodborne pathogens of concern are the human immunodeficiency virus (HIV), hepatitis B virus (HBV), and hepatitis C virus (HCV). Fecal-oral transmission of Salmonella and Shigella bacteria, enteroviruses, and hepatitis A virus may occur in hospitals and other work settings. In recent years, health care institutions have had to prepare for *(a)* newly emergent organisms, such as those that cause severe acute respiratory syndrome (SARS) and H1N1 influenza, and *(b)* the threat of bioterrorism.

Beyond health care, other infectious diseases pose increased risks to a wide spectrum of workers. Zoonoses may occur by direct contact with animals or their respiratory secretions or excreta. Veterinarians, farmers, cat and dog breeders, and animal handlers are among the occupations at heightened risk. Outdoor work settings increase the risks of arthropod-borne diseases and fungal infections for forestry, farm, construction, landscape, and other workers because of increased risk of contact with mosquitoes and ticks and increased exposure to *Coccidioides immitis*, *Histoplasma capsulatum*, or other pathogens in soil and dust. Workers assigned to or native in developing countries are exposed to

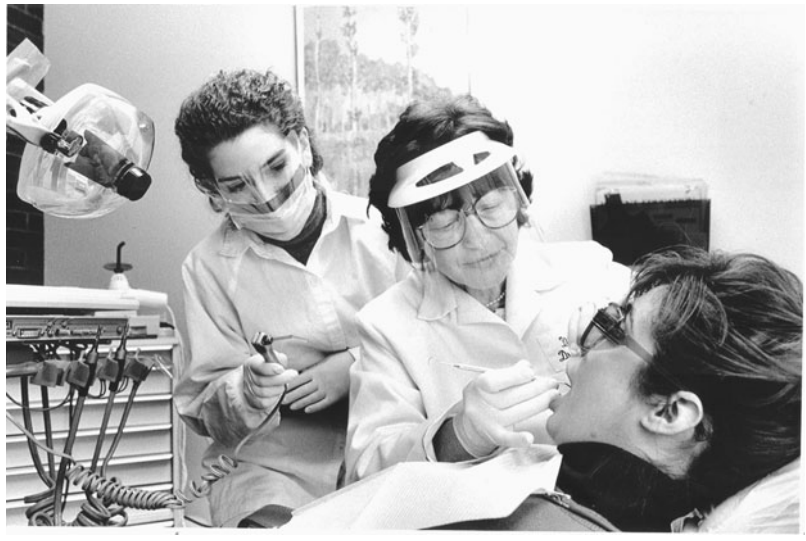

Figure 13-1. Dentists and dental technicians are at increased risk of exposure to HIV, hepatitis B and C viruses, and other pathogens. They require protection from splashes or sprays of infectious materials. The worker closest to the patient is wearing a face shield, while the other worker wears a combination surgical mask and eye protector. (Photograph by Marvin Lewiton.)

Figure 13-2. Workers in an HIV/AIDS laboratory are at risk of acquiring HIV infection. This photograph shows HIV/AIDS laboratory workers using personnel protective equipment and exhaust ventilation under a hood. (Photograph by Earl Dotter.)

agents that cause endemic infectious diseases, such as malaria and other parasitic diseases, nematode infestations, and viral and bacterial illnesses.

BIOLOGICAL HAZARDS IN HEALTH CARE AND LABORATORY SETTINGS

Bloodborne Pathogens

Prevention

More than 500,000 needlesticks occur annually in the United States, of which at least 5,000 involve HIV-contaminated blood. Unfortunately, underreporting of blood and body fluid exposures is very common. Studies of percutaneous exposures with hollow-bore needles have demonstrated significant differences between the number of needlesticks reported and the number estimated retrospectively by questionnaires. In the operating room, where injuries may occur during as many as 15% of all procedures and blood contact may occur in as many as 50%,[1,2] underreporting is substantial—one study found that only approximately 2% to 11% of blood exposures were reported.[3] Because early prophylactic therapy is indicated for certain exposures, underreporting places health care workers at unnecessary risk of infection.

Guidelines and regulations have been designed to reduce bloodborne exposures among health care workers. Universal Precautions, developed by the Centers for Disease Control and Prevention (CDC) in 1987, were incorporated into the Occupational Safety and Health Administration (OSHA) Bloodborne Pathogen Standard of 1991 along with a requirement for annual training, exposure reduction planning, implementing engineering controls, and providing HBV vaccine to potentially exposed health care workers. In 1995, Standard Precautions were introduced, combining Universal Precautions with isolation of body substances, to establish a single set of procedures for patient care and handling of blood and other potentially infectious body fluids.

Needlestick injuries can be reduced by educational programs and replacement of standard instruments with safer devices (Fig. 13-3). Use of phlebotomy devices with engineered safety features and needleless intravenous delivery systems have reduced needlestick injuries. Use of blunt needles for certain procedures has reduced percutaneous injuries among operating room personnel. The Needlestick Safety and Prevention Act of 2000, which recognized the potential for safer devices to reduce bloodborne pathogen exposures among health care workers, mandated OSHA to amend the Bloodborne Pathogen Standard so that employers would be required to document consideration and use of effective safer medical devices to eliminate or minimize occupational exposure to blood.[4]

Although a broad range of infections can be transmitted percutaneously or mucocutaneously, the bloodborne pathogens of greatest significance for health care workers are HIV, HBV, and HCV.

Human Immunodeficiency Virus

As of mid-2009, the CDC had documented 57 health care workers in the United States to have become HIV-positive following occupational

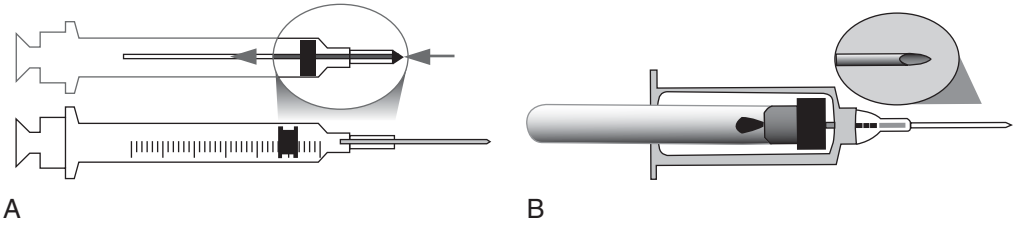

Figure 13-3. Shown here are two devices that help prevent accidental needlesticks (sharp sticks). (A) Syringe with retractable needle: After the needle is used an extra push on the plunger retracts the needle into the syringe, thus removing the hazard of needle exposure. (B) Blunt-tipped blood-drawing needle: After blood is drawn, a push on the collection tube moves the blunt-tip needle forward through the needle and past the sharp needle point. The blunt point tip of this needle can be activated before it is removed from the vein or artery.

exposure: 24 nurses, 19 laboratory workers, 6 physicians, 2 surgical technicians, 2 housekeepers or maintenance workers, 1 dialysis technician, 1 respiratory therapist, 1 health aide, and 1 morgue technician. Of these 57 health care workers, 48 had percutaneous exposure to HIV, 5 mucocutaneous exposure, 2 both percutaneous and mucocutaneous exposure, and 2 exposure from an unknown route.

There appears to be a 0.3% risk of HIV seroconversion following needlestick exposure. A higher risk of seroconversion is associated with deep injury, visible contamination of a device with blood, needle placement directly into an artery or vein, and exposure to a person with a high titer of HIV. Risk of seroconversion following mucous membrane exposure has been estimated to be 0.09%.[5] The risk of seroconversion following isolated skin exposure has not been quantified, but is likely to be extremely low.

The U.S. Public Health Service recommends antiretroviral medications for prophylactic treatment of people exposed to HIV-contaminated blood or body fluids. Several lines of evidence support use of prophylaxis, including a case-control study of health care workers who became HIV positive following bloodborne occupational exposure to HIV,[6] and a study of HIV-positive pregnant women administered zidovudine during pregnancy.[7] Drug efficacy is decreased if not begun soon after exposure or prematurely discontinued.

Drug resistance is a major challenge to the efficacy of antiretroviral therapy. Seroconversions have occasionally occurred after bloodborne HIV exposure, despite prophylaxis with one or more antiretrovirals, possibly due to viral resistance, late initiation of therapy, inadequate duration of therapy, or an overwhelming inoculum of virus. In prescribing combination antiretroviral therapy to exposed health care workers, probable patterns of viral resistance should be considered, based on the medication history of the source patient. Drug toxicities also should be monitored closely in health care workers receiving prophylaxis. A broad range of mild and serious side effects has been reported, including fulminant hepatic failure requiring liver transplantation.[8]

Many people who are potentially exposed to HIV at work are concerned that they may place sexual partners or other family members at risk. Many worry about future pregnancies and career options. Clinicians treating HIV-exposed workers should counsel them on barrier protection to prevent pregnancy and disease transmission, and clinicians may also counsel sexual partners and family members, if necessary.

Health care workers exposed to HIV-infected blood or body fluids should receive prophylaxis as soon as possible following exposure. Health care workers working in HIV-endemic countries where antiretrovirals may not be readily available should be provided access to them for prophylaxis.[9]

Hepatitis B Virus

Due to the implementation of Standard Precautions in medical centers and widespread hepatitis B vaccination, the estimated incidence of HBV infections among health care workers is approximately one-fifth that of the general population. Among unvaccinated health care workers, percutaneous exposure to HBV-infected blood confers a seroconversion risk of 1% to 6% if the source patient is e-antigen negative, and 22% to 31% if the source patient is e-antigen positive.[10] Viral titers may be as high as 1 billion virions per millilter of blood or serous fluid; however, they are usually several orders of magnitude lower in saliva, semen, and vaginal secretions. Hepatitis B virus is resistant to drying, ambient temperatures, simple detergents, and alcohol. It may survive on environmental surfaces for up to 1 week.[11] An HBV-contaminated sharp object may pose a threat to health care workers for several days after last contact with a source patient.

Less than half of people who become infected with HBV manifest acute symptoms. Acute illness generally consists of several weeks of malaise, jaundice, and anorexia. Fulminant hepatitis may develop in approximately 1% of patients. Chronic HBV infection develops in approximately 5% of those infected and is usually accompanied by persistent presence of hepatitis B surface antigen (HBsAg) in the blood for more than 6 months. In those whose infections do not become chronic, hepatitis B surface antibody (anti-HBs) develops as HBsAg levels fall. IgM antibodies to hepatitis B core antigen (HBcAg) indicate current infection, while IgG

core antibodies indicate past infection. The e antigen, which is separated from HBcAg during intracellular processing, is a marker of HBcAg production and viral replication. Cirrhosis develops in approximately 20% to 35% of people with chronic HBV infection, 20% of whom will develop hepatocellular carcinoma.

Administration of hepatitis B vaccine generates immunity in more than 90% of people who receive three vaccine doses. Once established, immunity appears to persist even as anti-HBs titers fall or become undetectable, although the number of years during which immunity is sustained is not known. Periodic booster doses are not recommended. Individuals who do not produce anti-HBs following vaccination, however, should again receive three doses of vaccine. Those who do not mount an anti-HBs response to the vaccine following three additional doses should be counseled regarding their susceptibility to HBV and receive hepatitis B immune globulin (HBIG) and possibly additional vaccine if exposed percutaneously or mucocutaneously to HBV-contaminated blood or body fluids. HBIG, which should be administered as soon as possible following exposure, is approximately 75% effective in preventing HBV infection in those without vaccine-induced protection.

The single most effective step to prevent HBV infection among health care workers is vaccination. Despite an OSHA requirement that employers provide vaccine free of charge to health care workers, a surprising number of workers remain at risk. A survey of transplant surgeons revealed that approximately 20% were not fully vaccinated.[12]

Hepatitis C Virus

Among health care workers, the prevalence of HCV infection is about the same as that of the general population: 1.5%. Following percutaneous exposure of health care workers to infected blood, the risk of hepatitis C seroconversion ranges from 0% to 10%, with an average of 1.8%.[13] Infection following mucocutaneous exposure appears to be much less common. The incubation period for hepatitis C varies from 2 to 24 weeks, with an average of 6 to 7 weeks. Antibodies to HCV (anti-HCV) may be detected within 5 to 6 weeks of infection, and they may persist regardless of whether virus is actively replicating. Most people who become infected with HCV have no acute symptoms. Chronic hepatitis develops in approximately 85% of those infected.

No hepatitis C vaccine is available. Administration of immune serum globulin is ineffective. Interferon alpha-2b is effective in treating chronic HCV infection. Treatment during acute HIV infection or early in the course of chronic HCV infection may be associated with higher cure rates.[14,15] Symptomatic patients with acute hepatitis C are more likely to spontaneously clear the virus than are patients with asymptomatic infection.[16]

For those who are acutely infected and symptomatic, delaying therapy with interferon, or interferon and ribavirin, until approximately 12 weeks after onset of symptoms can reduce unnecessary therapy in those destined to clear HCV spontaneously. Given the lower apparent likelihood of spontaneous clearance among those with asymptomatic acute infections, initiation of therapy after infection is documented by seroconversion, and polymerase chain reaction (PCR) assay may be prudent. Given the high cure rates associated with acute therapy and the toxicities of interferon and ribavirin, there is no role for prophylaxis in individuals exposed percutaneously or mucocutaneously to HCV-infected blood or other body fluids. Exposed individuals should be monitored at 6 weeks, 3 months, and 6 months for seroconversion. Testing with PCR may be used to detect early infection or to confirm presence of virus.

Other Infections

Tuberculosis

Following a resurgence of TB in the United States during the 1980s and early 1990s, disease incidence has fallen in recent years, although TB remains the single most important infectious cause of death worldwide. It is important to distinguish between *infection* with the organism (*Mycobacterium tuberculosis*) that causes TB and *active disease*. Approximately 95% of people who become infected will contain the organism with a healthy immune response and never develop active disease. Such people have latent infections, which are not contagious. Risk for developing active disease is highest within the first 2 years

of infection. It is increased when the infected person's immune response is compromised, which may occur with HIV infection, malnutrition, cancer chemotherapy, diabetes mellitus, or other diseases. In 2008 in the United States, 12,898 cases of active TB were reported. The incidence rate of 4.2 per 100,000 population represented a decrease of about 50% since 1992, when cases most recently peaked. Almost 60% of active TB cases in the United States occur among those born in other countries; their rate is 10 times that of those born in the United States. In 2008, Mexico, the Philippines, Vietnam, and India were the native countries of approximately half of these patients. Among cases where susceptibility testing was performed, the proportion of active TB patients in 2007 in the United States with multidrug resistant tuberculosis (MDR TB) was 1%. Eighty percent of these patients had been born in other countries.

Without careful adherence to engineering, administrative, and personal protective controls, health care workers remain at increased risk for active TB. In response to increasing tuberculosis rates in the late 1980s and early 1990s and occupational transmission in several medical centers, the CDC issued guidelines recommending that health care facilities at high risk for TB transmission develop and implement programs to prevent occupational exposure.[17] CDC guidelines, most recently updated in 2005, address early identification of potentially contagious patients, engineering controls to minimize spread within a medical center, use of personal protective equipment, and medical surveillance among health care workers. For the potentially contagious patient placed in negative-pressure isolation, work-practice controls include respiratory isolation signage, use of N95 respirators by all persons entering the isolation room, and restriction of diagnostic and therapeutic procedures to negative-pressure isolation settings.

Tuberculin skin testing (TST) for tuberculosis, which is based on a healthy immune response to the presence of *Mycobacterium tuberculosis*, may be positive in persons with latent infection and those with active disease. A decreasing incidence of TST conversion among health care workers during the past 15 years is testament to the success of administrative, engineering, and personal protective controls in health care facilities.[18] Administrative controls (early isolation of suspected tuberculosis patients) and engineering controls (adequate ventilation rates) have been strongly associated with reduced rates of TST conversion among health care workers.[19,20] In the past, outbreaks in health care facilities caused substantial morbidity among health care workers. In 11 outbreaks from 1928 to 1991, TST conversion rates ranged from 15% to 100%, and active TB occurred in 11% to 61% of skin-test converters. However, health care workers who were already TST positive at time of exposure did not develop elevated risk of active TB.[21]

Tuberculin skin testing is the most widely used method for TB surveillance among health care workers. Health care workers with previously negative tuberculin tests must be tested at time of hire. Because skin-test positivity can wane over time and can be "boosted" by repeated skin testing, those in whom testing has not been performed within the preceding year should receive a two-step test (TST test repeated several weeks following an initial test) to ensure adequate TST sensitivity. In addition to prior infection, vaccination with BCG (Bacillus Calmette-Guerin)—a live, attenuated form of *Mycobacterium bovis* used in many developing countries to reduce TB infections among children—may produce a positive skin test either initially or on two-step testing, especially if BCG vaccination has been recent. A new infection requires an intensive public health search for a source patient and carries with it specific recommendations for chemoprophylaxis. Therefore, one should establish accurate baseline skin-testing results to avoid mistakenly identifying a "boosted" response as a new infection.

The recommended frequency of ongoing testing is based on community TB prevalence and frequency of inpatient TB admissions. People with documented positive TSTs should not be retested. They should be monitored for symptoms suggestive of active TB. After TST conversion and a negative chest X-ray, additional screening chest X-rays should not be done. Tuberculin skin testing reactions may be suppressed by illnesses or medications that alter the normal immune response, and they may be difficult to interpret in areas where non-TB mycobacterial infections are common.

An alternative or supplemental screening method involves assays that measure the release of interferon gamma (IFN-g) in whole blood incubated with tuberculosis-like synthetic peptides, such as QuantiFERON-TB Gold (QFT-g), QuantiFERON-*TB* Gold In-Tube, and T-SPOT.TB. There is much variability in sensitivity for both TST and IFN-g release assays, and no clear advantage of either test. In non-BCG vaccinated populations, specificity for both types of tests is 96% or higher.[22] The principal advantage of QFT-g is its greater specificity among people who have been vaccinated with BCG, probably because peptides used in QFT-g assays are not found in BCG vaccine. Therefore, for people who have received BCG vaccine, QFT-g assays are often used after a positive TST. A negative QFT-g result in people who have received BCG vaccine indicates a lower likelihood of latent TB; a positive result is presumptive evidence of latent or active disease.

OSHA requires employers to meet the general duty clause of the OSH Act—to provide a workplace free of recognized hazards. This requirement includes identifying potential respiratory hazards and providing a respiratory protection program specific to the hazard.

Newly Emergent Acute Respiratory Diseases: Severe Acute Respiratory Syndrome, Novel H1N1 Influenza

From November 2002 through July 2003, over 8,000 people worldwide—including more than 1,700 health care workers—contracted severe acute respiratory syndrome (SARS), a new human respiratory disease caused by a novel coronavirus. The disease appeared to be transmitted primarily by droplets and direct contact. In some hospitals, attack rates among health care workers were nearly 60%, primarily because of delayed recognition of SARS. Worldwide, SARS caused 774 deaths in 2002 and 2003, with a case fatality rate of 9.6%.

After an incubation period of 2 to 10 days, symptoms included fever, chills, rigors, headache, malaise, and diarrhea. Effects on the lower respiratory tract followed—the usual cause of death. The case-fatality rate varied; in one series, it was 3% in patients under 60, and 54% in those 60 or older.[23] Even when health care workers used personal protective equipment, some patient-care activities, such as intubation, were associated with increased risk of SARS transmission.[24]

While most patients with SARS did not transmit it to others, some "superspreaders" accounted for widespread transmission under certain circumstances, such as in hotels, apartment buildings, and hospitals, and on airplanes. A SARS infection in a physician, who had treated SARS patients in China and was later admitted to a Hong Kong hospital, eventually led to SARS in about 100 hospital workers and epidemics in several other countries. Another superspreading event, which occurred at an apartment complex in Hong Kong, produced more than 320 cases. Since the epidemic in 2003, there have been a few SARS cases, associated primarily with laboratory exposures; secondary spread has been limited.

In the spring of 2009, a novel H1N1 influenza virus, derived from swine, avian, and human strains, emerged from rural Mexico. Novel H1N1 spread rapidly, primarily via droplet spread, and the World Health Organization (WHO) declared a phase 6 pandemic within months. The case-fatality rate of H1N1 influenza has been estimated at 0.4%. In contrast to seasonal influenza, novel H1N1 has disproportionately affected children as well as adults below the age of 60. In New York, for example, an early focus of the epidemic, only 5% of those hospitalized were older than 60. More severe disease and higher case fatality have occurred among pregnant women and people with chronic medical problems, including asthma, other chronic lung disease, diabetes, immunosuppression, and obesity.

The main hazard to health care workers occurs when patients acutely ill with H1N1 influenza are not promptly diagnosed, when they are not properly isolated, and when health care workers do not use recommended personal protective equipment.[25] Universal Respiratory Etiquette, which requires that symptomatic patients don surgical masks when entering a health care facility, would decrease the risk of transmission to health care workers. Compliance with this guidance, however, is limited. To better protect health care workers during respiratory disease epidemics, hospitals may consider having emergency-department patients don surgical masks at time

of triage, at least until a thorough clinical assessment is completed. Oseltamivir prophylaxis has been recommended for health care workers who have had unprotected exposure to novel H1N1 patients.

BIOLOGIC HAZARDS IN SCHOOLS, HEALTH CARE FACILITIES, AND OTHER WORKPLACES

Measles

Despite control of measles in the general population, health care facilities should continue to maintain measles vaccination programs for their personnel because several past measles epidemics have been linked to health care facilities. Since measles may be spread by large droplets and airborne transmission, precautions must be used when caring for patients with confirmed or suspected measles. Hospitals, schools, and day care centers should be vigilant for imported measles cases, especially from Europe and Asia.

Rubella

The principal hazard of rubella is its potential to adversely affect fetal development. In 1980 in an outbreak, 47 (13%) of health care workers at a Boston hospital developed rubella; one health care worker terminated her pregnancy. In another outbreak, 56 hospital employees developed rubella; three women terminated their pregnancies. More recently, outbreaks have tended to occur in nonhospital workplaces that employ a large proportion of foreign-born workers.[26] Rubella, which is spread by droplet transmission, is most contagious at the time the rash is erupting, although virus may be shed from 1 week before to 5 to 7 days after the onset of rash. Infants with congenital rubella may excrete virus for years. Droplet precautions must be used when caring for patients with rubella, and health care workers should be vaccinated if they do not have evidence of rubella immunity.

Mumps

Mumps is an acute viral syndrome, which may cause parotitis and less frequently deafness, orchitis, oophoritis, and mastitis. Transmission occurs from direct contact or via respiratory droplets. Incubation is usually 16 to 18 days. Infected people may be contagious prior to manifesting symptoms. One dose of measles-mumps-rubella (MMR) vaccine protects 80% of vaccinees from mumps; two doses protect 90%.

An outbreak, which occurred in Iowa and surrounding states in 2005 and 2006, primarily involved people age 18 to 25, most of whom had been vaccinated. The viral genotype was identical to one associated with a large mumps outbreak in the United Kingdom, which also occurred principally among unvaccinated individuals. Due to their increased risk of acquiring and transmitting mumps, health care workers should receive two doses of MMR vaccine. Persons suspected of having mumps should remain isolated for 9 days following onset of symptoms.

Varicella (Chicken Pox)

Varicella may be spread by contact with infected lesions or by airborne transmission. The incubation period ranges from 10 to 21 days. People at risk for severe disease include immunocompromised individuals, pregnant women, and premature infants. Adults generally have more severe disease than children. From 1990 to 1994, fewer than 5% of varicella cases in the United States occurred among adults older than 20, but they accounted for 55% of varicella-related deaths. Outbreaks may occur in hospitals when personnel without immunity care for patients with unrecognized disease. A varicella vaccine was licensed in 1994 and is recommended for nonimmune health care workers, teachers of young children, day care workers, military personnel, those who work in institutions and prisons, and international travelers. It is contraindicated for pregnant women. Because the vaccine provides only partial protection in some people, many medical centers only require health care workers who have immunity from natural disease to care for infected patients—a practice that will need to be reevaluated as the epidemiology of varicella changes due to widespread vaccination of children. Exposed hospital personnel who do not have varicella immunity

should be furloughed from patient contact from days 10 to 21 after contact with an infected patient.

Parvovirus B19

Episodes of parvovirus transmission to health care workers occur infrequently. Risk of infection among school and day care teachers generally exceeds that of health care workers. Parvovirus is spread via large droplets, direct contact, or fomites. Patients with erythema infectiosum rash (fifth disease) are contagious before the appearance of the rash. Infected adults generally suffer a self-limited viral arthropathy. Those with parvovirus-associated aplastic crisis are contagious for up to 1 week following onset of illness. Infected immunocompromised persons may be contagious for years. Patients hospitalized during a phase of disease when transmission may occur should be treated using droplet precautions. When women become infected during the first half of pregnancy, there is a small risk of fetal death due to hydrops or spontaneous abortion.

Pertussis

Pertussis, which is easily spread by droplets or direct contact, has an attack rate of 80% in unvaccinated individuals. Estimates of annual incidence in the United States range from 800,000 to 3.3 million cases.[27] Several pertussis outbreaks have occurred in hospitals and involved health care workers.[28] Infants are at highest risk of death.

An acellular pertussis vaccine (Tdap) is recommended to the general population—with an accelerated schedule of vaccination to health care workers. Vaccine efficacy is approximately 92%. Antibiotic prophylaxis with trimethoprim-sulfamethoxazole, clarithromycin, or azithromycin continues to be recommended following acute unprotected exposure to a pertussis patient, regardless of whether the exposed individual has received Tdap.

Seasonal Influenza

More than 110,000 U.S. residents are hospitalized annually due to influenza or its complications, and approximately 36,000 die each year. Hospitals have a high risk of influenza transmission, which generally occurs by large droplets. Patients hospitalized with influenza should be treated using droplet precautions. In adults, virus may be shed from 1 day prior to illness to 7 days after onset. Children may excrete virus for longer periods. The most effective means of prevention is annual vaccination, which is specifically recommended for health care workers. Vaccine consists of killed virus from three strains (H3N2, H1N1, and B) designed to closely match circulating strains. Vaccine generally prevents disease in 70% to 90% of healthy immunized adults when the match of circulating and vaccine strains is close. Antivirals may be administered during outbreaks to prevent influenza in nonimmunized adults. However, oseltamivir resistance in seasonal H1N1 strains and adamantane (rimantadine and amantadine) resistance in H3N2 strains have become widespread. Adamantanes are also ineffective against influenza B.

In 1997, the first instance of transmission of avian influenza (H5N1) to a human was documented. Since then, more than 400 cases have been documented, occurring principally in children and in adults under the age of 40. The case-fatality rate has been approximately 60%. Human-to-human transmission is very rare. The acquisition by the virus of the ability to transmit readily from human to human would represent a global public health emergency.

Hepatitis A

Although outbreaks of hepatitis A have occurred in health care facilities, its prevalence among health care workers is similar to that in the general population. The CDC does not recommend hepatitis A vaccination for health care workers. Transmission has occurred in hospitals *(a)* during care of patients with diarrhea who were later discovered to be acutely infected with HAV, and *(b)* through contamination of food due to improper hand washing after patient care. Outbreaks may occur in day care centers, especially if there is community transmission, but day care center workers do not have increased prevalence of infection.[29]

Agents of Bioterrorism

Following the 9/11 attack on the World Trade Center and the dissemination of anthrax spores through the U.S. mail during 2001, there has been increased attention on preparedness for terrorist attacks. Bioterrorism agents are viewed as credible threats due to their capacity for widespread dissemination and potential to affect or kill many people. The CDC classifies such agents into three categories. Category A agents can be easily disseminated or transmitted from person to person, can result in high case-fatality rates, can have major public health impact, might cause public panic and social disruption, and require special action for public health preparedness. Category B agents are moderately easy to disseminate, result in moderate morbidity rates and low case-fatality rates, and require specific enhancements of diagnostic capacity and disease surveillance. Category C agents could be engineered for mass dissemination in the future due to their availability, ease of production and dissemination, and potential for high morbidity and case-fatality rates.

Category A agents (and the diseases they cause) are *Bacillus anthracis* (anthrax), Clostridium botulinum toxin, *Yersinia pestis* (plague), *Variola major* (smallpox), *Francisella tularensis* (tularemia), and Ebola, Marburg, Lassa, and Machupo viruses (viral hemorrhagic fevers). Category B agents (and the diseases they cause) are Brucella species (brucellosis), Epsilon toxin of *Clostridium perfringens*, food safety threats (Salmonella species, *Escherichia coli* 0157:H7, and Shigella) (see Chapter 9), *Burkholderia mallei* (glanders), *Burkholderia pseudomallei* (melioidosis), *Chlamydia psittaci* (psittacosis), *Coxiella burnetii* (Q fever), ricin toxin, staphylococcal enterotoxin B, *Rickettsia prowazekii* (typhus fever), viral encephalitis (alphaviruses [such as Venezuelan equine encephalitits, eastern equine encephalitis, and western equine encephalitis]), and water safety threats (such as *Vibrio cholerae* and *Cryptosporidium parvum*). Category C agents include agents that cause "emerging infections," such as Nipah virus and hantavirus.

Agents of bioterrorism vary widely in their propensity for transmission from person to person. Standard precautions are all that is required to prevent transmission to health care workers caring for those affected by anthrax, tularemia, Q fever, and biological toxins. However, patients who have not been adequately decontaminated may harbor disease agents on their skin or clothing that could cause disease in health care providers. For some agents, such as smallpox virus and Lassa fever virus, the primary means of transmission is close contact, but isolated examples of airborne spread dictate use of respiratory protection when providing patient care.[30,31] Health care workers should use droplet precautions when caring for patients with pneumonic plague and viral encephalitis viruses, and contact precautions when caring for patients with brucellosis.

As a result of the 2001 dissemination of anthrax spores, 23 people contracted inhalational or cutaneous disease, 17 of whom survived. They had symptoms that included fever, flu-like symptoms, cough, dyspnea, pleuritic chest pain, nausea, vomiting, headache, and chest discomfort. Presence of shortness of breath, nausea, and vomiting and lack of rhinorrhea helped to distinguish the initial clinical presentation of anthrax from influenza or influenza-like illness. For affected postal workers, the most important factor in survival was physicians' clinical suspicion of anthrax, based on occupational history, which led them to initially obtain blood cultures.

Considerable attention has been directed to smallpox virus as a potential biological weapon, although the only known remaining stocks of the virus are safeguarded in the United States and Russia. A major campaign was undertaken by the U.S. government to vaccinate military personnel stationed in areas considered to be at risk and health care workers who might need to care for smallpox victims in a bioterrorist attack. Many military personnel were vaccinated. Due to widespread concern about adverse effects of smallpox vaccine, a smaller than anticipated proportion of health care workers chose to be vaccinated. Common adverse effects of vaccination include fever, lymph node swelling, and injection site pain. Less common, potentially serious adverse effects include erythema multiforme, generalized vaccinia, myocarditis, transmission to contacts, and inadvertent inoculation (such as people inoculating their eyes with virus shed from the vaccine site). Because of marginal acceptance by health care workers of this smallpox

vaccination campaign, policy has been changed to rapidly vaccinate health care workers if smallpox cases occur.

Measures to enhance preparedness for bioterrorist attacks have included the following:

- Upgrading of epidemiological detection systems to recognize unusual disease clusters that may indicate exposure to bioterrorist agents
- Building capacity of laboratories to detect bioterrorism-related agents
- Improving communication systems among first responders, law enforcement personnel, and staff medical centers
- Increasing awareness of bioterrorism-related disease among physicians
- Stockpiling vaccines and drugs
- Improving supply lines for rapid delivery of vaccines and drugs
- Performing research on relevant diagnostic tests, vaccines, and therapies

BIOLOGIC HAZARDS ASSOCIATED WITH ANIMAL CONTACT

Many bacterial, fungal, parasitic, viral, and rickettsial diseases can be transmitted from animals to humans (Table 13-1). Workers who have frequent contact with wild animals, farm animals, or domestic pets are at increased risk.

Workers including park rangers, hunters, ranchers, forestry workers, trappers, fur traders, geologists, other scientific field workers, butchers, rendering workers, expedition leaders, and zoo workers have contact with wild animals. These include rats, mice, bats, rabbits, raccoons, skunks, deer, and bison. For some diseases, relatively close animal contact is required for transmission; for others, illness may occur after ingesting small amounts of water or food contaminated by animal waste, such as giardiasis, or by breathing dusts contaminated with animal excrement, such as histoplasmosis. Workers are at increased risk for brucellosis (if they have contact with, for example, bison or deer), raccoon roundworm (raccoons), giardiasis (water contaminated by animal excrement), hantavirus infection (wild mice), histoplasmosis (bat guano), lymphocytic choriomeningitis (rodents or house mice), tuberculosis (deer, elk, or bison), plague (wild rodents), rabies (raccoons, skunks, or bats), and tularemia (rodents, rabbits, or hares). Chronic wasting disease of deer and elk, which is endemic in Colorado, Wyoming, and Nebraska, may be caused by a prion; it is not clear if this represents a threat to humans.

Contact with Macaque monkeys, which may occur in an animal laboratory setting, a monkey cell culture facility, or among veterinarians, is associated with transmission of herpes B simiae, which can cause fatal encephalomyelitis in humans. Infection is caused by animal bites, scratches, or exposure to the tissues or secretions of Macaques. Immediate and thorough wound cleansing is indicated following a Macaque bite. Prophylactic treatment with acyclovir or valacyclovir is indicated for percutaneous or mucocutaneous exposures to potentially infected animals.

Farm workers and those who process farm products, such as meatpackers, butchers, and slaughterhouse workers, may be exposed to cattle, sheep, pigs, goats, domestic fowl, horses, and other animals. Farm workers have much contact with livestock and livestock waste. Agents that may be transmitted in the farm environment include Brucella, Campylobacter, Cryptosporidium, *Escherichia coli* 0157:H7, *Coxiella burnetti*, rabies virus, ringworm, Salmonella, and *Yersinia enterocolitica*. Bovine spongiform encephalopathy (BSE, or "mad cow disease"), a neurological degenerative disease of cattle, is likely caused by a prion; consumption of contaminated meat has been strongly associated with variant Creuzfeldt-Jakob disease in humans; farm workers, however, have not been a high-risk group for this disease.

Enhanced contact with pet animals may occur among breeders, delivery personnel, veterinarians, pet shop workers, and others. Illnesses associated with dogs include brucellosis (rare), Campylobacter infection, cryptosporidiosis, giardiasis, leptospirosis, Lyme disease, Q fever, rabies, Rocky Mountain spotted fever, salmonellosis, and infestations with tapeworm, hookworm, ringworm, and roundworm. Many of these same illnesses are associated with cats. Cat scratch disease, caused by *Bartonella henselae*, and plague (rarely) can also be transmitted from cats. Bird-associated illnesses may occur among

Table 13-1. Zoonoses and Transmitting Animals

Zoonoses	Transmitting Animals
Bacterial Diseases	
Brucellosis (Brucella spp.)	Farm animals and dogs
Campylobacteriosis (Campylobacter spp.)	Cats, dogs, farm animals, and improper food preparation
Cat scratch disease or cat scratch fever (*Bartonella henselae*)	Cat scratches and bites
Escherichia coli O157:H7 infection	Cattle and improper food preparation
Fish tuberculosis (Mycobacterium spp.)	Fish and aquarium water
Leptospirosis (Leptospira spp.)	Livestock, dogs, rodents, wildlife, and contaminated water
Lyme disease (*Borrelia burgdorferi* infection)	Dogs and ticks
Plague (*Yersinia pestis*)	Wild rodents, cats, and fleas
Psittacosis (*Chlamydia psittaci*)	Pet birds, including parrots and parakeets
Q fever (*Coxiella burnetti*)	Cattle, sheep, goats, dogs, and cats
Salmonellosis (Salmonella spp.)	Reptiles, birds, dogs, cats, horses, farm animals, and improper food preparation
Tuberculosis, or TB (*Mycobacterium tuberculosis*)	Deer, elk, bison, and cattle
Tularemia (*Francisella tularensis*)	Sheep and wildlife, especially rodents and rabbits
Yersiniosis (*Yersinia enterocolitica*)	Dogs, cats, and farm animals. Also associated with improper preparation of chitterlings
Fungal Diseases	
Cryptococcosis (Cryptococcus spp.)	Wild birds, especially pigeon droppings
Histoplasmosis (Histoplasma spp.)	Bat guano (stool)
Ringworm (Microsporum spp. and Trichophyton spp.)	Mammals, including dogs, cats, horses, and farm animals
Parasitic Diseases	
Cryptosporidiosis (Cryptosporidium spp.)	Cats, dogs, and farm animals
Giardiasis (*Giardia lamblia*)	Various animals and water
Hookworm (*Ancylostoma caninum*, *Ancylostoma braziliense*, *Uncinaria stenocephals*)	Dogs and their environment
Leishmaniasis (Leishmania spp.)	Dogs and sand flies
Raccoon roundworm infection (*Baylisascaris procyonis*)	Raccoons
Roundworm (*Toxocara canis*, *T. cati*, and *Toxocaris leonina*)	Cats, dogs, and their environment
Tapeworm infection (*Dipylidium caninum*)	Flea infections in cats and dogs
Toxoplasmosis (*Toxoplasma gondii*)	Cats and their environment
Viral Diseases	
Hantavirus (hantavirus pulmonary syndrome)	Wild mice
Herpes B (Herpesvirus 1)	Macaque monkeys
Lymphocytic choriomeningitis	Rodents such as rats, guinea pigs, and house mice
Monkeypox	Recently suspected to be associated with prairie dogs, Gambian rats, and rabbits
Rabies	Mammals, including dogs, cats, horses, and wildlife
West Nile virus	Spread by mosquitoes; can affect birds, horses, and other mammals
Rickettsial Diseases	
Rocky Mountain spotted fever (*Rickettsia rickettsii*)	Dogs and ticks
Other (Prion?)	
Bovine spongiform encephalopathy (mad cow disease)	Associated with cattle

veterinarians, pet shop workers, poultry workers, and bird breeders, including psittacosis (parrots and parakeets), Q fever (ducks and geese), cryptococcosis (wild bird and pigeon droppings), and salmonellosis (chickens, baby chicks, and ducklings). Human cases of monkeypox have been reported in association with pet prairie dogs. Smallpox virus is closely related to monkeypox virus. Smallpox vaccine, which may be 85% protective against monkeypox, is recommended for workers investigating monkeypox outbreaks or involved in caring for infected people or animals.

BIOLOGIC HAZARDS ASSOCIATED WITH ARTHROPOD VECTORS

Contact with arthropod vectors, especially mosquitoes and ticks, may occur frequently among

park rangers, landscapers, nursery workers, farmers, ranchers, trappers, construction workers, soldiers, and others who work outside.

Several types of encephalitis are transmitted in the United States by mosquito vectors. Eastern equine encephalititis, transmitted primarily from birds to humans by mosquitoes, accounts for an average of five reported cases a year in the United States, but it has a 35% case-fatality rate. Coastal areas and freshwater swamps have the highest transmission risk. Western equine encephalitis, also rare, has a lower case-fatality rate. LaCrosse encephalitis, which is reported in the United States approximately 70 times a year, is typically transmitted from chipmunks or squirrels to humans by the treehole mosquito (*Aedes triseriatus*). Workers in woodland areas are at increased risk. St. Louis encephalitis, 4,658 cases of which were reported in the United States between 1964 and 2006, is transmitted from birds to humans primarily by Culex mosquitoes. In temperate areas of the United States, St. Louis encephalitis occurs primarily during the late summer and early fall; in southern states, it may occur year-round.

West Nile virus, a flavivirus common in Africa, West Asia, and the Middle East, is closely related to St. Louis encephalitis virus. It appears to have been introduced to the eastern U.S. during 1999 or earlier. It is transmitted primarily from birds to humans by mosquitoes, with outbreaks in temperate regions predominating in late summer and early fall. Year-round transmission takes place in milder climates. During 2008, there were more than 1,300 reported cases and 44 deaths in the United States.

Tick bites represent another occupational hazard for outdoor workers. The most important illnesses associated with tick vectors are Lyme disease, Rocky Mountain spotted fever, babesiosis, and ehrlichiosis. Lyme disease is caused by *Borrelia burgdorferi* and transmitted to humans by black-legged ticks (*Ixodes scapularis* in north central and northeastern United States, and *Ixodes pacificus* on the Pacific coast). Infection is most likely to be transmitted if the tick has fed for at least 2 days. Workers in woodland areas of the northeastern and north central United States, and a limited region of the northwestern Pacific coast, are at highest risk. If recognized early, Lyme disease can be effectively treated with oral antibiotics. Rocky Mountain spotted fever is caused by *Rickettsia rickettsii* and spread either by *(a)* the American dog tick, which predominates in central and eastern areas of the United States and the California coast; or *(b)* the Rocky Mountain wood tick, which predominates in the Rocky Mountains. Most infections are transmitted from April through September.

Babesiosis is caused by Babesia protozoan parasites (primarily *Babesia divergens* and *Babesia microti*). Disease, which is spread from mice to humans primarily by the *Ixodes scapularis* tick, is characterized by fevers, chills, myalgias, hepatosplenomegaly, and hemolytic anemia. Ehrlichiosis is caused primarily by three distinct bacterial species of the genus Ehrlichia. In the United States, ehrlichiosis due to *Ehrlichia chaffeensis* occurs primarily in the southeastern and south central states and is transmitted by the lone star tick, *Amblyomma americanum.* Ehrlichia ewingii has caused a few human cases of ehrlichiosis in Missouri, Oklahoma, and Tennessee. Human granulocytic ehrlichiosis is caused by a third Ehrlichia species, and it is transmitted by black-legged ticks (*Ixodes scapularis* and *Ixodes pacificus*).

Preventive measures that should be implemented by outdoor workers to prevent transmission of mosquito- or tickborne illnesses include wearing lightly colored, long-sleeved shirts tucked into pants and lightly colored, long pants tucked into socks, using DEET-containing insect repellants, using mosquito netting if sleeping outdoors, avoiding outdoor work at dawn and dusk, and checking of skin and hair for ticks daily. Permethrin-containing repellants may be used on clothing, shoes, bed nets, and camping gear.

TRAVELERS' HEALTH

Detailed discussion of diseases typically encountered in tropical and developing countries, as well as their prevention and treatment can be found at http://www.cdc.gov/travel. The most common cause of illness in travelers is contamination of food or water. Travelers' diarrhea can be due to bacteria, including *E. coli*, Salmonella, and *Vibrio cholerae*; viruses; or parasites. Many illnesses are transmitted to travelers via arthropod vectors,

including malaria, yellow fever, dengue, filariasis, leishmaniasis, trypanosomiasis, and onchocerciasis. Schistosomiasis can be transmitted through the skin during swimming in freshwater. Vaccination and prophylaxis vary by destination country. General recommendations for travelers include frequent hand washing, drinking only bottled or boiled water or canned drinks, eating only thoroughly cooked food or self-peeled fruits and vegetables, complying with any recommended malaria prophylaxis, and protecting oneself from mosquitoes. One should not eat food purchased from street vendors, drink beverages with ice, eat unpasteurized dairy products, handle animals, or swim in freshwater. Prior to departure, workers assigned to tropical or developing countries, should consult with a travel medicine specialist.

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14

Occupational Stress

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Occupational stress can be defined as the harmful physical and emotional responses that occur when the requirements of the job do not match the capabilities, resources, or needs of the worker. Despite recognition by law, medicine, and the insurance industry of the nature of occupational stress, it remains a nebulous construct for many people.

During the past two decades, research consistently documented the same phenomenon in developed countries: As the pace of competition increased and a truly global marketplace developed, occupational stress and its consequences greatly increased. Increased work hours, increased pressure, increased insecurity, and many other organizational stressors were shown to have immediate and long-term deleterious consequences for both individuals and organizations.

The current worldwide economic recession, which began in late 2007 and has been the longest since World War II, has had enormous consequences for working conditions and the nature and intensity of job stress—consequences that will likely affect the lives of workers for many years.

Downsizings, layoffs, mergers, restructuring, and bankruptcies have led to many workers losing their jobs. For example, by spring of 2009, the unemployment rate in the United States reached 9.5% and initial claims for unemployment insurance reached a 26-year high. Job loss can have devastating consequences, putting unemployed workers at risk for physical illness, marital strain, anxiety, depression, and even suicide. In addition, high unemployment rates can profoundly affect the lives of young people trying to enter the workforce and older workers seeking to reenter it or to remain there.

Macroeconomic changes, such as recessions, can also increase job stress for workers in nearly all industry sectors and organizational strata, because they lead to changes in job structure. For example, millions of workers who have been shifted to unfamiliar tasks with new supervisors and fewer health and retirement benefits generally feel they must work longer and harder to maintain their standard of living.

A BRIEF HISTORY OF JOB STRESS RESEARCH

Occupational stress, as a field of inquiry examining job conditions and their health and performance consequences, is a relatively new area of research that crystallized in the early 1970s. Its conceptual roots can be traced to Hans Selye's animal research and Walter Cannon's work on

the physiological concomitants of emotion.[1] In the early 1930s, Hans Selye discovered that a wide variety of noxious stimuli (which he later referred to as stressors), such as exposure to temperature extremes, physical injury, and injection of toxic substances, evoked identical patterns of physiological changes in laboratory animals. In each case, the cortex of the adrenal gland became enlarged, the thymus and other lymphatic structures became involuted, and ulcers developed in the stomach and intestines. These effects occurred regardless of the stressor and were superimposed on any specific effects associated with the individual agents. Some years later, Selye described this somatic response as the general adaptation syndrome (GAS) and defined stress as the nonspecific response of the body to any demand. His mention of "nervous stimuli" among the "stressor" agents capable of eliciting the GAS energized researchers in psychosomatic medicine. Cannon had earlier laid the groundwork, by describing the "fight-or-flight" response, for an understanding of how emotions affect physiological functions and disease states. This response, elicited by potentially dangerous situations, included increased heart rate and blood pressure, redistribution of blood flow to the brain and major muscle groups, and a decrease in vegetative functions (digestive activity). Cannon also advanced the concept of "physiologic homeostasis" and developed an "engineering" concept of stress and strain—with stress as the input and strain as the response. Cannon proposed that critical stress levels were capable of producing strain in homeostatic mechanisms. Although he used the term somewhat casually, Cannon, like Selye, conceived of "stress" as involving physical as well as emotional stimuli.

In the 1960s and 1970s, Richard Lazarus and his colleagues added immensely to the study of stress by describing in specific terms how an organism's perceptions or appraisals of objective events determine their health relevance. He described cognitive appraisal as an intrapsychic process that translates objective events into stressful experiences. This formulation recognized that subjective factors can play a much larger role in the experience of stress than objective events, since a given objective event can be perceived positively by one person and negatively by another.

The study of occupational stress was given impetus in the early 1970s by the establishment of the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA). The importance of behavioral and motivational factors was acknowledged in provisions of the Occupational Safety and Health Act (OSH Act), which directed NIOSH to include psychological, behavioral, and motivational factors in researching problems of worker safety and health and in developing remedial approaches for these problems. Job-related hazards were interpreted broadly to include conditions of a psychological nature—undue task demands, work conditions, or work regimens that may adversely affect workers' physical or mental health.

NIOSH has sponsored and conducted many research studies that have helped shape the course of job stress research in the United States. For example, in 1988, NIOSH proposed a national strategy for prevention of work-related psychological disorders. Key elements in this prevention strategy include abatement of known job risk factors, research to improve understanding of these risk factors, surveillance to detect and track risk factors, education and training to facilitate recognition of risk factors and their control, and improved mental health services.[2]

In 1996, NIOSH identified "organization of work" as one of the 21 priority research topics for the next decade and developed a comprehensive research agenda for investigating and reducing occupational safety and health risks associated with the rapidly changing nature of work.[3] This document described how macrolevel forces impact occupational stress. For example, national and international economic, legal, political, technological, and demographic forces influence production methods, human resource policies, management structures, and supervisory practices. These factors, in turn, directly impact the work context, influencing the nature of jobs and the tasks that comprise them. Fueled by global competition, organizational downsizing and restructuring has influenced not only the way work is performed but also—as many laid-off workers can attest—whether work is available to perform.

A MODEL OF JOB STRESS AND HEALTH

Working conditions play a primary role in causing job stress. However, the role of individual factors cannot be ignored. Exposure to stressful working conditions (job stressors) can have a direct influence on worker safety and health.[4] However, individual and situational factors can intervene to strengthen or weaken this influence (Fig. 14-1). Individual and situational factors can modulate the effects of job stressors on the risk of illness and injury in different ways: They can decrease them, leave them unchanged, or potentiate them.

Based upon this view of job stress, a paradigm of stress was developed by researchers at NIOSH to guide efforts at examining the relationship between working conditions and health consequences (Fig. 14-2). In this paradigm, job stress is viewed as a situation in which job stressors—alone or in combination with other stressors—interact with individual worker characteristics and result in an acute disruption of psychological or physiological homeostasis. This disruption (often called job strain) can be psychological (disruption in affect or cognition); physiological; or behavioral. Job strain, if prolonged, can lead to cardiovascular disease, psychological disorders, musculoskeletal disorders, and other diseases. In addition, job stressors are probably linked to risk of workplace injury and violence. Job stressors can also cause "organizational strain" in the form of increased absenteeism, decreased performance, increased injury rate, and increased loss of employees to other jobs.

Job stressors generally fall into three very broad categories: job/task demands, organizational factors, and physical conditions. Examples of common stressors in each category are briefly described in the following sections.

Job and Task Demands

Workload is related to stress (Figs. 14-3, 14-4, and 14-5). Overwork causes negative psychological, physiological, and behavioral strains. Working excessive hours or performing more than one job has been associated with adverse health consequences, including poor perceived health, increased injury rates, and increased cardiovascular disease morbidity and mortality.[5] (See Box 14-1.) Workload and work pace are especially important when work hours are increasing.

Shift work, a work-related stressor, is another job demand associated with health and safety consequences. (See Box 14-2.) Working rotating

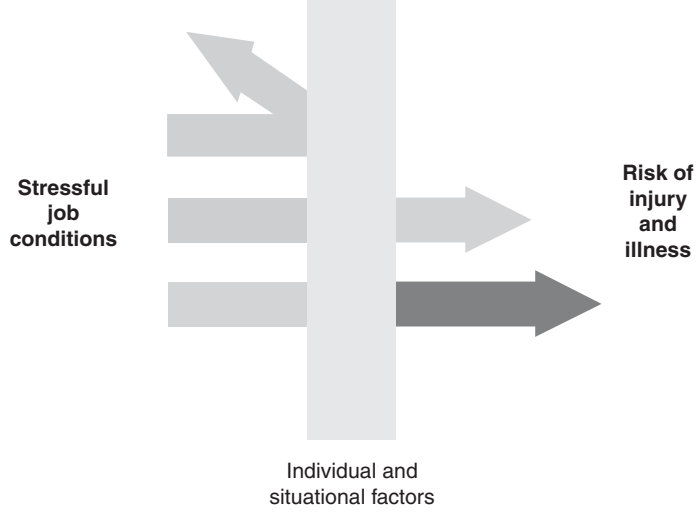

Figure 14-1. This National Institute for Occupational Safety and Health model of job stress illustrates the different roles that individual and situational factors can have in reducing the impact of job stress (top arrow), having no effect on job stress (middle arrow), or exacerbating job stress (bottom arrow). (*Source*: National Institute for Occupational Safety and Health. Stress at work. DHHS NIOSH Publication No. 99-101. Washington, DC: NIOSH, 1999.)

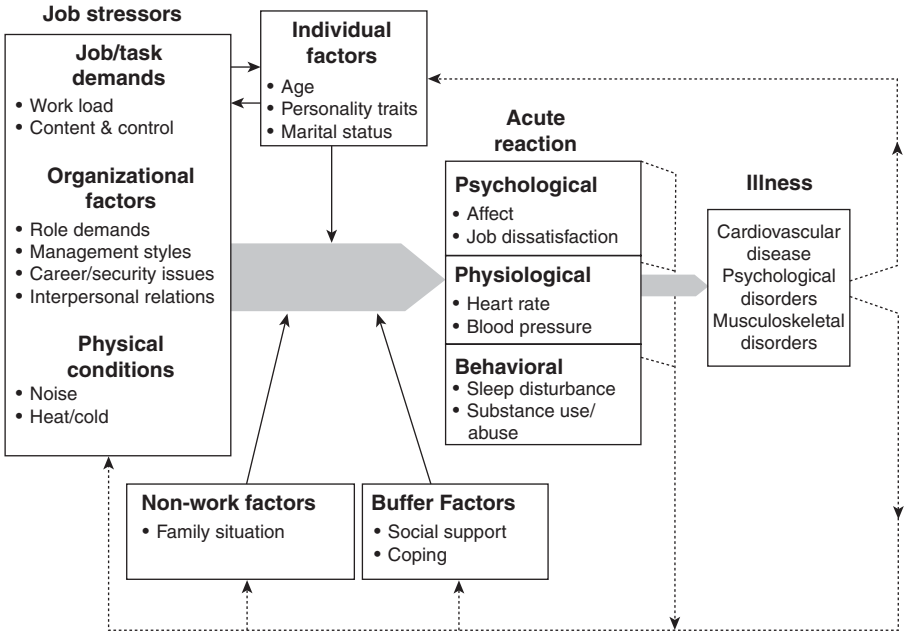

Figure 14-2. Job stressors and their consequences.

Figure 14-3. Garment workers, who often work on a piecework basis, are often under much stress at work. (Photograph by Earl Dotter.)

shifts or permanent night work results in disruption of social activities and physiological circadian rhythms, impairing alertness and the sleep cycle.[6] Employees report that working nights or overtime affects their mental and physical health. Their decreased alertness makes them more prone to errors and increases their risk for injuries. Most workers work shifts because it is required or because no other work is available. Workers experience friction between shift work and their family and social life. Rotating shift work is associated with cardiovascular

Figure 14-4. Automobile assembly line workers face the stresses of machine-paced work. (Photograph by Earl Dotter.)

Figure 14-5. Secretaries are among the most highly stressed workers. (Photograph by Earl Dotter.)

and gastrointestinal disease. All of these effects are sufficiently well established to provide the basis of labor law in the European Union, which regulates the scheduling of shifts and rest days.

Narrow, fragmented, invariant, and short-cycle tasks that provide little stimulation, allow little use of skills, or expression of creativity are job characteristics that are considered stressors in the NIOSH model.[2] Robert Karasek's *demand-control-social support model* is perhaps the best known of all models relating such job characteristics to well-being.[7] This model proposes first that high job demands, lack of job control (referred to as low *decision latitude*), and lack of social support predict strain outcomes. In addition, it suggests that demands, control, and social support interact to predict strain, such that high control and high social support buffer the effects of job demands on strain outcomes.

The ever-growing number of studies using the model suggests support for the first hypothesis—the main effects of demand, control, and social support—and limited support for the hypothesized interaction among these factors. The combination of low decision latitude and high

Box 14-1. Time, Work, Stress, and Well-Being in Society

Sherry L. Baron and SangWoo Tak

One of the most pronounced changes affecting working people and their families is how they experience and perceive time as a result of changes in demographics, society, technology, and work organization. The average work week in the United States has not changed significantly over the past several decades—with, on average, men working about 41 hours and women about 39 hours weekly.[1] But workers' experience of time has changed dramatically. Failure to balance the competing time demands from one's work and family can have adverse effects on the health and well-being of workers, their partners, and their children—an important focus for research. Time demands can increase work stress, which is associated with both adverse mental and physical health outcomes (Chapter 19).

While average work time has not increased, several factors have transformed the way that workers and their families experience time. The most dramatic factor has been the rapid increase in women's participation in the workforce. In 1970, in 42% of married couples, both spouses worked; in 2009, in 74% of married couples, both spouses worked. Over the same period, the number and percentage of single-parent families increased from about 10% to about 20%. As a result, spouses have less time with each other, and parents have less time with their children.

The schedule challenges of dual-earning and single-parent families are made worse by the increasing proportion of workers employed in jobs requiring work hours outside of the standard 9-to-5 work day and the standard Monday-to-Friday work week. In one of three dual-earner families and one in five-single parent families with children under 14, a parent works either a rotating or a nonstandard work shift.[2] These proportions are even greater in low-income families that are most likely to face financial stress and complex work schedules. Nonstandard work shifts can have adverse effects on family activities, especially activities that require involvement of parents at their children's schools or other activities associated with standard schedules. Seventy percent of parents in the United States complain that they have insufficient time with their children, and 38% of U.S. residents say they always "feel rushed." Difficulty of parents in meeting parental responsibilities, such as caring for children, meeting with their teachers, and chaperoning school trips, also has consequences.

Workers on shift work and long work hours often have difficulty maintaining their own hobbies and leisure activities. They experience lack of time to spend with friends who do not work shifts and find it difficult to participate in sports or other nonwork activities because of varying shift schedules. They may feel that they do not have the energy or free time to participate in hobbies. Tensions and problems among spouses can arise from not having enough time to spend with each other. Those who work long hours and perform shift work have higher divorce rates. Nonstandard work schedules may become more common, especially in service jobs and other low-paying occupations.

The experience and perception of time at work has also changed. As productivity at work continues to increase, introduction of new technology and the intensification of job tasks may mean that workers experience greater job demands. Downsizing and outsourcing often require professionals and managers to work longer hours, take work home with them, and intensify workloads on other workers. Increased demand for after-hour and weekend services and demands for increased productivity can result in more nonstandard work weeks and work shifts for hourly workers, including rotating shifts, night work, and split shifts. While the status of salaried professionals allows them more flexibility to leave work early in the event of a family responsibility, hourly workers usually are not allowed such flexibility.[3]

Fatigue on the night shift is reflected in performance. Studies have demonstrated that errors by meter readers peaked on the night shift, that telephone operators connected calls considerably more slowly at night, and that train engineers failed to operate their alerting safety device more often at night. Concerns about resident physician performance after prolonged shifts have led to changes in residency on-call rules. Motor vehicle crashes increase after prolonged shifts. Perhaps the best approach to limiting work hours is to place limits on the acceptable level of fatigue or risk, rather than on any specific feature or features of the work schedule. The Fatigue Index of the Health and Safety Executive in the United Kingdom can be used as a tool to enable organizations to assess whether work schedules are likely to be associated with undue levels of fatigue.

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Further Reading

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Jacobs JA, Gerson K. The time divide: work, family and gender inequality. Cambridge, MA: Harvard University Press, 2004.
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Provides more information on the health effects of shift work and long work hours.

(*Continued*)

Box 14-1. Time, Work, Stress, and Well-Being in Society (Continued)

Presser HB. Working in a 24/7 economy: challenges for American families. New York: Russell Sage Press, 2003.
A comprehensive analysis of the impact of longer workdays and extended workweeks on the health and well-being of workers and their families.

Work, Family and Health: A federally funded research initiative. Available at: http://www.kpchr.org/workfamilyhealthnetwork/public/default.aspx
This federally funded initiative is conducting research regarding the health effects of and potential interventions for work-family stress, including time constraints.

Box 14-2. Shift Work

David H. Wegman and SangWoo Tak

Shift work is an imprecise concept, although it usually refers to a work activity scheduled outside standard daytime hours (standard being a period of 8 hours between 7 a.m. and 7 p.m.), where there may be a handover of duty from one individual or work group to another on the same job within a 24-hour period. Examples of shift work are as follows: *(a)* work during the afternoon, night, or weekend outside standard daytime work hours; *(b)* extended work periods of 12 hours or more, often associated with compressing the working week; *(c)* rotating hours of work; *(d)* "split shifts," where work periods are divided into two distinct parts with several hours break in between periods; *(e)* overtime work; and *(f)* standby (on-call) work.

It is difficult to classify the many types of shift work. The following factors contribute to types of shift work: shift pattern (permanent or rotating, forward or backward rotation, and fast or slow rotation); shift timing (night, early morning, afternoon, and daytime); shift duration (8 hours, 12 hours, more than 12 hours, shifts of varying length, and split shifts), and the scheduling of rest breaks within or between shifts.

In general, there are four major types of work schedules: day work, permanently displaced work hours, rotating shift work, and roster work. Day work involves work periods between approximately 7 a.m. and 7 p.m. Permanently displaced work hours require the person to work either a morning shift (approximately 6 a.m. to 2 p.m.), an afternoon shift (approximately 2 p.m. to 10 p.m.), or a night shift (approximately 10 p.m. to 6 a.m.). Rotating shift work involves alternation between two or three shifts. Two-shift work usually involves morning and afternoon shifts, whereas three-shift work also includes the night shift. Three-shift work is often subdivided according to the number of teams used to cover the 24 hours of the work cycle—usually three to six teams, depending on the speed of rotation (number of consecutive shifts of the same type). Roster work is similar to rotating shift work, but it may be less regular, more flexible, and less geared to specific teams. It is used in service-oriented occupations, such as transport, health care, and law enforcement. In most industrialized countries, approximately one-third of workers have some form of "non-day work" (shift work); approximately 5% to 10% have shift work that includes night work.

Effects on Health and Well-Being

A general review of research on shift work is available in a special issue of *Applied Ergonomics*.[1] A summary of the health effects of shift work has been provided in a review article.[2]

Sleep

The dominant health problem reported by shift workers is disturbed sleep and wakefulness, affecting at least three-fourths of shift workers. Sleep loss is primarily taken out of stage 2 sleep and rapid eye movement (REM) sleep. In addition, the time taken to fall asleep (sleep latency) is usually shorter. The level of sleep disturbances in shift workers is comparable to that seen in insomniacs.

Alertness, Performance, and Safety

Lack of quality sleep, disruption of the internal body clock, or prolonged exertion can result in the decline in mental and/or physical performance. Shift workers complain about fatigue and sleepiness, which is especially increased on the night shift, negligible on the afternoon shift, and intermediate on the morning shift. The maximum is reached toward the early morning (5 to 7 a.m.). Frequent incidents of falling asleep occur during the night shift, which has been documented in process operators, truck drivers, train drivers, and pilots. Even though one-fourth of night-shift workers exhibit sleep incidents, most workers seem unaware of them, suggesting an inability to judge one's true level of sleepiness.

Sleepiness on the night shift is reflected in performance. A classic study showed that errors in meter readings over a period of 20 years in a gas works had a pronounced peak on the night shift. Other studies demonstrated that telephone operators connected calls considerably more slowly at night, and that train engineers failed to operate their alerting safety device more often at night. Performance may be reduced to levels comparable with those present after considerable alcohol consumption. There is evidence that the Challenger Space Shuttle disaster and the nuclear power plant incidents at Chernobyl, Three Mile Island, the David Beese reactor in Ohio, and the Rancho Seco reactor in California were due to fatigue-related errors during night work. The risk of errors, accidents, and injuries is higher on the night shift and is likely to rise with increasing shift length over 8 hours. These risks are also increased by successive shifts—especially night shifts—and having an insufficient number of rest breaks.

(*Continued*)

Box 14-2. Shift Work (Continued)

Other Effects on Health and Well-Being

The rate of gastrointestinal complaints is greater among night-shift workers than among day workers. The incidence rate of coronary artery disease is increased in male shift workers, as compared with men who work days. (A high prevalence of smoking among shift workers might contribute to these two increased rates.) A few studies of pregnant shift workers suggest an increased risk of miscarriage and lower birthweight of infants of mothers who work irregular hours, but the study did not suggest a risk of birth defects. The International Agency for Research on Cancer (IARC) has classified "shift work that involves circadian or chronodisruption" as a probable carcinogen to humans (Group 2A).[3] People likely exposed to circadian disruption have high risks of breast cancer and prostate cancer. Night shift workers have high risks of endometrial cancer.

Health problems in shift workers usually increase with age and with increasing exposure to shift work. Being a "morning-type" person, as opposed to an "evening-type" person, is associated with poorer adjustment to shift work. Gender is not related to shift-work tolerance, although the extra burden of housework may put women at a disadvantage. Good physical condition of workers may facilitate shift work.

One of the major effects of shift work is the interference of work hours with various social activities. Therefore, direct time conflict reduces the amount of time available to spend with family and friends or in recreation or volunteer activities.

Factors Affecting Adjustment

Shift System Characteristics

An important shift system characteristic is the number of consecutive night shifts. The circadian system and sleep do not adjust (improve) much across a series of night shifts even in permanent night workers. Therefore, a series of more than four night shifts might be expected to be especially taxing. If performance capacity needs to remain high during night work, a schedule of permanent night shifts for some workers may be preferable, with other workers working a two-shift schedule (only morning and afternoon shifts).

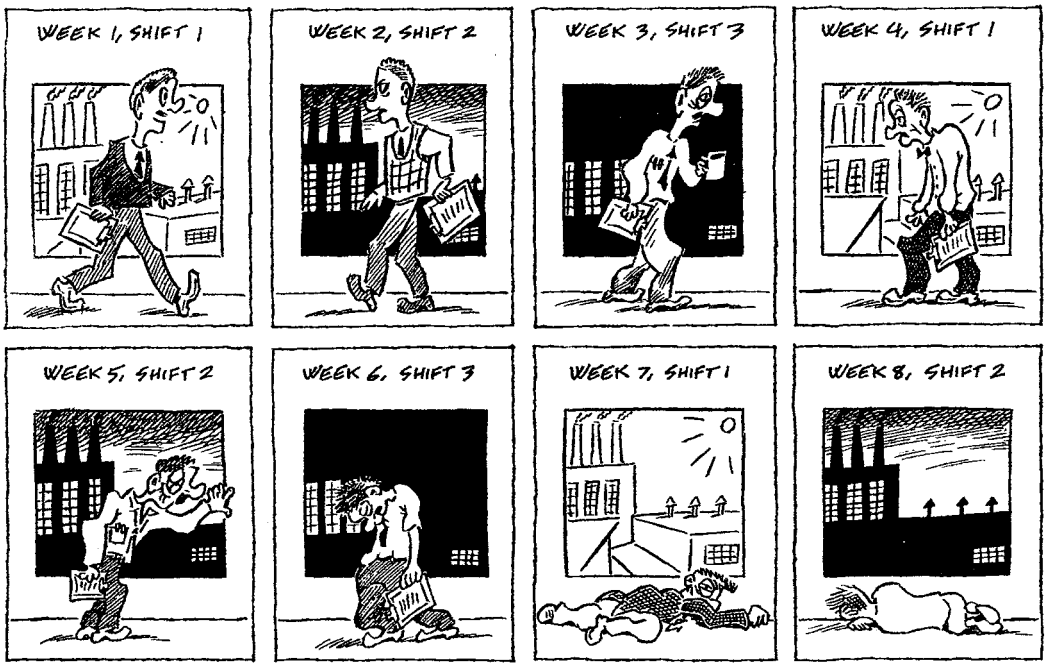
More workers are working longer shifts (up to 10 to 12 hours) on fewer work days, a schedule that permits long sequences of free time and reduced commuting to and from work. Having a second job may exacerbate the effects of long shifts or lack of recovery days.

For rotating shift workers, schedules that delay (rotate clockwise: morning-afternoon-night) are considered preferable to schedules that advance (rotate counterclockwise). There have been, however, few practical tests of this theory, especially in relation to sleepiness.

Preventive Measures

The United Kingdom Health and Safety Executive has published *Managing shiftwork: Health and safety guidance* (2006), which is a good general reference.[4] The following preventive measures should be considered:

- Avoid quick changes of work schedule.
- Maintain daily rest of at least at 11 hours.
- Avoid double shifts or other greatly extended work shifts.



(Drawing by Nick Thorkelson.)

(*Continued*)

Box 14-2. Shift Work (Continued)

- Avoid morning shifts starting before 6 a.m.
- Intersperse rest days during the shift cycle.
- Schedule naps during the night shift.
- Provide long sequences of days off and few weekends with work.
- Avoid having a morning shift immediately after a night or evening shift.
- Avoid long (more than three shifts) sequences of night or morning shifts (rotate rapidly).
- Offer workers a choice between permanent and rotating shift schedules.
- Plan night shifts at the end of the shift cycle.
- Give shift workers older than 45 years of age the right to transfer to day work.
- Rotate shifts forward (clockwise).
- Build regular weekend breaks into the shift schedule.

The most important individual preventive measure is good sleep hygiene, including sleeping in a dark, cool, sound-insulated bedroom; using ear plugs; and informing family and friends about one's sleep schedule. Another important preventive measure is strategic sleeping. For night-shift work, the sleep period should be between 2 and 9 p.m. If this is not socially feasible, the next best alternative is to have a moderate morning sleep and then to add a 2-hour nap in the evening. Workers should avoid intake of major meals during the night shift.

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psychological demands is a risk factor for cardiovascular mortality.[8] Indeed, it is widely accepted that worker control or discretion over working conditions is integral to worker health. The theoretic basis and specific mechanisms of the effects of control on health, however, are not clear.

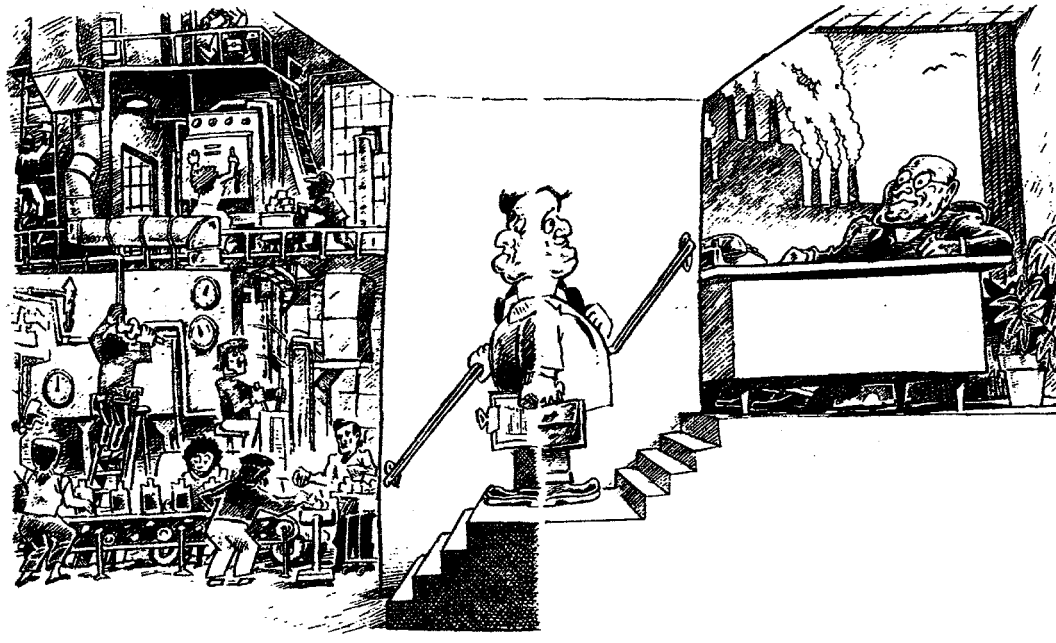
CASE 1

A 42-year-old woman left her production job 3 years ago to become a customer representative at a large telecommunications company. In her new job, a computer routes the calls and, to her, the calls never seem to stop. She even has to schedule her bathroom breaks. All day she hears complaints from unhappy customers. She cannot promise anything to them without getting the approval of her boss. She is caught between company policy and what the customer wants. The other customer representatives are so busy and uptight about rumors of possible outsourcing that none of them talk to each other. They all remain in their small work cubicles until the end of their work shifts. She has migraine headaches and hypertension. To make matters worse, her mother's health is deteriorating, but she has not accumulated much sick time and fears losing what little she has to take care of her mother.

Organizational Factors

Many studies have examined the psychological and physical effects of various role-related demands in organizations. Role conflict exists whenever individuals face incompatible demands from two or more sources. Role ambiguity reflects the uncertainty employees experience about what is expected of them in their jobs. Interrole conflict exists when employees face incompatible demands from two or more roles. The most common form of interrole conflict is work-family conflict, in which the demands of work conflict with the roles of parent and spouse. Each of these role-related stressors has been linked to strain and, in some cases, illness outcomes. Given the revolutionary changes in the way that work has been structured and performed in recent years, these stressors are also believed to be highly prevalent and problematic.[3]

Various management styles, including total or partial intolerance of worker participation in decision making, lack of effective consultation, and excessive restrictions on worker behavior, are also stressful. Of these style characteristics, exclusion from decision making has been shown to be related to a variety of strain outcomes, including lowered self-esteem, low job satisfaction, and overall poor physical and mental health. By contrast, studies have demonstrated



A supervisor's job may be highly stressful due to its high degree of role conflict. (Drawing by Nick Thorkelson.)

that greater participation in decision making has led to greater job satisfaction, lower employee turnover, better supervisor–subordinate relationships, and greater productivity. Increasing worker participation seems to result in less work-related psychological strain.

Stressors include career-related concerns, such as job insecurity, fear of job obsolescence, under- and overpromotion, and more generally concerns about career development.

The effort–reward imbalance model of job stress has focused attention on the role of organizational rewards as a job stressor.[9] This model proposes that strain and ill health result when financial, esteem, and career rewards are not consistent with efforts—the strivings of individuals to meet the demands and obligations of the job. Workers attempt to maintain a state of equilibrium between efforts and rewards. Those who cannot maintain this balance over time will eventually become ill. In addition, the theory proposes that a high level of overcommitment (a personality factor) may increase the risk of ill health and that workers reporting high levels of imbalance in combination with high levels of overcommittment may be at even higher risk of ill health. While a variety of studies have generally found support for the imbalance portion of the theory,[10] the full model has not yet been adequately tested.

Very recently, the Job Demands-Resources (JD-R) model of job stress was introduced as an alternative to the demand-control and effort-rewards models.[11] In this more comprehensive model, various job "resources," such as social support, career opportunities, performance feedback, and supervisory coaching, are viewed as having the potential to buffer the effects of various physical psychological and organizational demands. However, evidence for buffering has come largely from studies examining psychological effects, such as job burnout.

CASE 2

A 35-year-old man, who is a single parent of three young children, has worked for 12 years in a medium-sized electronics assembly plant. Six months ago, because of lagging product

sales, the parent company implemented a major reorganization that resulted in downsizing and extensive layoffs. Because of his relative seniority, this man escaped the initial round of job cuts, but he remained worried about the future. In the meantime, his job changed dramatically. Another worker and he started doing the work that three had done before. He started working 12-hour shifts, 6 days a week, which created many problems for him at home. In addition, he was assigned job tasks that he has never been trained to perform, and there has been much confusion at the plant concerning job responsibilities and how tasks are to be performed. Worker morale has worsened. And company officials have been considering bringing in consultants to figure out a better way to get the work done.

Interpersonal Relations

Poor interpersonal relations in the workplace, resulting from poor leadership or aggression and even violence, are stressors that result in strain consequences. Although incidents of physical violence are relatively rare, they have a dramatic effect on individual and organizational well-being. Aggression in the workplace, much more prevalent than violence, is associated with impaired physical and psychological health. Poor-quality leadership has been associated with increased levels of employee strain. Employees who perceive their supervisors as abusive experience low levels of job and life satisfaction, lower levels of commitment to work, increased work–family conflict, more psychosomatic symptoms, and psychological distress.

Physical Conditions

Adverse environmental conditions exacerbate overall job demands placed on employees, thus lowering worker tolerance to other stressors and decreasing worker motivation. Environmental conditions, including excessive noise, temperature extremes, poor ventilation, inadequate lighting, and poor ergonomic design have been linked to employee physical and psychological health complaints as well as attitude and behavior problems. For example, outbreaks of mass psychogenic illness (often called collective stress response) can occur in workplaces that employees regard as physically uncomfortable. Psychological job stressors appear to produce increments in muscle tension that may exacerbate muscle loads and symptoms resulting from physical task demands.[12]

MODERATING FACTORS

Several personal and situational characteristics can modify the way individual workers exposed to a work environment perceive or react to it. These characteristics, known as "moderators," are depicted in Figure 14-2, in the blocks labeled individual factors, nonwork factors, and buffer factors.[1]

Individual Factors

The most widely discussed personal characteristic related to stress at work has been the Type A behavior pattern, characterized by intense striving for achievement, competitiveness, time urgency, excessive drive, and overcommitment to vocation or profession. While investigators in the past have reported the Type A pattern to be independently associated with coronary artery disease, more recent studies have suggested that the variables of hostility, cynicism, anger, irritability, and suspicion may be the primary pathogenic component of the Type A pattern found to be significant in earlier studies. Similarly, while earlier studies suggested an interaction between certain job stressors and Type A characteristics that may lead to heart disease, overall the evidence that Type A persons are more adversely affected by various job stressors is limited.

The hardy personality style and an internal locus of control are also thought to mediate the stressor–illness relationship. Hardy persons are believed to possess various beliefs and tendencies that are useful in coping with stressors, such as optimistic appraisals of events and decisive actions in coping. Hardy persons report less illness in the presence of stressors. Persons with an internal locus of control—a general belief that events in life are controlled by their actions—also have shown a consistent tendency to report

better health than those who believe that life events are beyond their control.

Stage of career development, although little studied, may also moderate the stressor–illness relationship. For example, work experience (job tenure) seems to moderate worker responses to negative events at work. For workers in mid-career, job stressors may lose potency while stressful events outside the job domain become increasingly deleterious.[13]

Factors Outside of Work

Workers do not leave their family and personal problems behind when they go to work, and they do not forget job-related problems on returning home. Difficult transportation between home and work, child care needs, and availability of community resources may also moderate home and work stress. Nearly all models of job stress acknowledge extraoccupational factors and their potential interaction with work in affecting health outcomes. Few studies, however, have attempted to examine the respective health effects of job and extraorganizational stressors. While some investigators have incorporated generic stressful life events scales into job stress surveys, these scales provide only crude indications of social, familial, and financial stressors. In future studies, more attention needs to be paid to nonwork factors. Interpersonal, marital, financial, and child-rearing stressors can exacerbate existing job stressors to promote acute stress reactions. Alternatively, the absence of extraorganizational problems may make stressful job situations more tolerable (less stressful) and may impede the development of stress reactions. Environmental factors are recognized modifiers within the job stress model. For example, a worker living in a noisy, high-crime neighborhood will be exposed to added stress and may be unable to recover from stress endured at work. Or a worker may be subjected to significant stress because of a long and difficult commute between home and work. In contrast, the environment a worker lives in can offer good opportunities to reduce stress, such as by biking, running, and walking, or to enhance social interaction among neighbors.

Buffer Factors

Social Support

Stress researchers have long sought to identify buffer factors that may reduce or eliminate the effects of job stressors, such as the degree of social support an individual worker receives from work and nonwork sources. However, evidence for a buffering effect of social support has been mixed, perhaps due to differences among researchers in conceptualizing and measuring support.

Coping

Another potential buffering factor is coping. Coping is a transactional process that is modified continuously by experience within and between stressful episodes. A specific coping strategy that alleviates stress in one situation may not alleviate stress, or may actually increase it, in other situations. Coping responses that people use depend on their social and psychological resources. Social supports and psychological resources, like mastery and self-esteem, are what people draw upon in developing coping strategies. These resources vary by socioeconomic status, with people who are better educated and more affluent possessing more resources and a wider range of coping alternatives. No single coping response is uniformly protective across work and nonwork situations. However, having a large and varied coping repertoire can be helpful in reducing stressor–strain relationships. While various coping responses have been found to be effective in the areas of marriage, child rearing, and household finances, coping is often ineffective for work-related problems. This may be due to the impersonal nature of work and the lack of worker control over job stressors.

Lifestyle Factors

Lifestyle factors, such as physical fitness and exercise, smoking cessation, sound nutrition, and stress management, have the potential to buffer the health effects of job stressors, but clear evidence for such a buffering effect is lacking. However, such evidence could result from a current NIOSH initiative (NIOSH WorkLife), which is facilitating collaboration among occupational

safety and health professionals and health promotion specialists to develop and implement workplace programs that prevent occupational illness and injury, promote health, and optimize the health of workers. (See Chapter 38.)

PATHOPHYSIOLOGICAL CORRELATES OF JOB STRESS

Little is known about the pathophysiological mechanisms that underlie the relationships between stress and disease. Both direct and indirect pathways have been described or postulated. The direct pathways that are thought to play a role are disregulations of the neurohormonal system (pituitary-adrenocortical axis), the autonomic nervous system, and the immune system. A combination of these pathways, influenced by genetic factors, probably links exposure to job stressors and adverse health effects. An indirect pathway links job and non-job stressors to high-risk behaviors and, in turn, to adverse health effects. For example, strain effects from rotating shift work directly influence circadian rhythm, with resultant changes in the autonomic nervous system and the immune system.

To further complicate these relationships, job stressors can be seen as influencing risk factors for coronary heart disease or triggers for acute myocardial infarction. Acutely, stress increases catecholamine levels, leading to increased heart rate and blood pressure, decreased plasma volume, coronary constriction, and increased lipid levels, platelet activity, coagulation, and inflammation. Chronically, stress causes autonomic imbalance, neurohormonal changes (such as elevated cortisol and norepinephrine levels), a pro-coagulant state (characterized by blood hypercoaguability), and increased lipid levels. Immune system responses may mediate some of these relationships. Animal studies have demonstrated that experimentally induced stress increases susceptibility to a variety of infectious agents and the incidence and rate of growth of lymphomas and ovarian and pulmonary tumors. Some human studies have shown that psychosocial factors, including stressful life events, are related to immunological disorders, such as asthma, allergies, and autoimmune diseases. And stress has been linked to changes in levels of circulating antibodies, lymphocyte cytotoxicity, and lymphocyte proliferation.

PREVENTION AND INTERVENTION

The gap between etiologic and intervention-related knowledge in the realm of occupational stress is great. Despite the ever-burgeoning literature on the nature, causes, and physical and psychological consequences of occupational stress, surprisingly little is known about intervention for occupational stress. Views differ regarding the importance of worker characteristics versus working conditions as "the" major cause of organizational stress; these views have, in part, led to the development and use of primary, secondary, and tertiary prevention (intervention) approaches for occupational stress. The aim of primary prevention intervention is to reduce risk factors or job stressors. The aim of secondary prevention intervention for occupational stress is to alter the ways that individuals respond to risks or job stressors. And the aim of tertiary prevention intervention is to heal those who have been traumatized. Research on primary and tertiary prevention intervention has been reviewed by this author,[14,15] while secondary prevention intervention has been the subject of reviews by Murphy[16] and van der Klink and colleagues.[17] The following provides a brief overview of research findings for all three types of intervention.

Primary Prevention Interventions

Primary prevention interventions can be characterized as either psychosocial or sociotechnical. Psychosocial interventions focus primarily on human processes and psychosocial aspects of the work setting and aim to reduce stress by changing workers' perceptions of the work environment; they may also include modifications of objective working conditions. In contrast, sociotechnical interventions focus primarily on changes to objective working conditions and are considered to have implications for work-related stress. Some interventions involve elements of both approaches.

Psychosocial Interventions

Most primary prevention interventions appear to be psychosocial. Many are based upon the principles of participatory action research (PAR), in which researchers and workers collaborate in a process of data-guided problem solving to improve the organization's ability to provide workers with desired outcomes and to contribute to general operational knowledge. Participatory action research involves workers and experts from outside the workplace, in an empowering process of defining problems (identifying stressors), developing intervention strategies, introducing changes that benefit employees, and measuring outcomes. Some PAR interventions have specifically focused on efforts to redesign work or work processes. In general, there is very limited evidence for the efficacy of PAR and other participatory-type interventions; studies evaluating its efficacy tend to be methodologically weak, difficult to interpret, and causally ambiguous. When found, the effects of the interventions have often been on job satisfaction and perceptions of the working environment; few effects on health-related outcomes have been reported. It is unclear whether the general lack of health benefits are due to ineffective interventions, the insufficient duration of the studies, or the nature of the health-outcome variables studied. Moreover, which effects are attributable to the act of participating in the intervention and which are attributable to changes in working conditions or processes resulting from the intervention are unclear.

There is, however, some evidence for the efficacy of psychosocial interventions focused upon supervisors and managers, rather than workers. While few in number, these interventions resulted in positive organizationally relevant outcomes and found modest positive effects on individual well-being. An intriguing aspect is that the effects on well-being may extend beyond the supervisors and managers themselves, possibly representing a potentially effective and seemingly cost-efficient approach to primary prevention. No firm conclusions, however, can be drawn.

Sociotechnical Interventions

In contrast, sociotechnical interventions are generally not a result of employee–employer or employee–employer–researcher collaboration. Sociotechnical interventions have generally involved changing only a very limited variety of objective working conditions, such as workloads, work schedules, and work processes. However, as a whole, sociotechnical intervention studies provide more consistent and robust evidence for the efficacy of the intervention than psychosocial intervention studies. In addition to incorporating self-report measures of affect, such as job satisfaction, anxiety, and depression, most of these studies have incorporated objective outcome measures, such as blood pressure, job performance, and sickness absence, in the study design. In general, these studies have also tended to utilize more rigorous experimental and quasi-experimental designs.

Secondary Prevention Interventions

Secondary prevention interventions, often termed "stress management" or stress management training (SMT), involve techniques and procedures designed to help workers modify their appraisal of stressful situations and/or to deal with the symptoms of stress. Typically, such interventions are prescriptive, person-oriented, relaxation-based techniques, such as biofeedback, progressive muscle relaxation meditation, and cognitive-behavioral skills training. They differ from other health-promotion programs in the variety of training techniques and wide range of health-outcome measures used to assess program effectiveness. In contrast to primary prevention interventions, they do not seek to alter the sources of stress at work (job stressors) through organizational change strategies or job redesign.

Cognitive-behavioral skills training, frequently used in stress management, involves techniques designed to modify the appraisal processes that determine perceived stressfulness of situations and to develop behavioral skills for managing stressors. It helps people to restructure their thinking patterns through cognitive restructuring. In general, it can reduce psychological strain, especially anxiety, and improve organizationally relevant outcomes, such as job satisfaction. However, it has not shown consistent improvement of physiological strains.

In contrast, muscle relaxation techniques can benefit some physiological strains, such as blood pressure, but not others. Such techniques involve focusing one's attention on muscle activity, learning to identify even small amounts of tension in a muscle group, and practicing releasing of tension from muscles.

Meditation methods used in worksite stress-management studies, often secular versions of transcendental meditation, involve sitting upright in a comfortable position, in a quiet place, with eyes closed, and mentally repeating a mantra while maintaining a passive attitude. The few studies that have examined the efficacy of such worksite-based meditation provide consistent evidence that they reduce psychological, physiological, and behavioral strain.

Combinations of two or more stress-management approaches into a single intervention are frequently used. The most common combination and most effective seems to be muscle relaxation coupled with cognitive-behavioral skills training.

Tertiary Prevention Interventions

Tertiary interventions involve treatment of the physical, psychological, or behavioral consequences of exposures to job stressors. The following is an overview of these interventions that have often been implemented in organizations.

Medical Care

Many large companies have occupational medicine departments that offer services that include urgent medical care, employee examinations, disability reviews, health promotion activities, and referrals for medical treatment. (See Chapter 28.) In general, these departments are not structured to provide extensive or long-term care for stress-related illness or injury and must rely on making referrals to appropriate health care providers. Mental health problems related to job stress can present special challenges to occupational medicine departments that may not be well equipped either to deal with them or to make referrals.

Counseling and psychotherapy are commonly used methods to treat individuals suffering from work-related mental health problems. Common techniques of psychotherapy and counseling include behavioral and cognitive therapy, supportive counseling, and insight-oriented psychotherapy. Counseling and psychotherapy can have marked benefits on symptom reduction, but they may not have a beneficial impact on work performance (as measured by reduced absenteeism).

Many companies offer limited counseling at the workplace through employee assistance programs (EAPs) that often provide a variety of mental health–related services. Employees can refer themselves to EAPs or be referred by management. The goals of an EAP are to restore employees to full productivity by *(1)* identifying those with drug abuse, and those with emotional or behavioral problems that result in deficient work performance; *(2)* motivating these employees to seek help; *(3)* providing short-term professional counseling assistance and referral; *(4)* directing employees toward the best assistance available; and *(5)* providing continuing support and guidance throughout the problem-solving period. Very few studies have addressed the cost-effectiveness of EAPs. There is little agreement on evaluation methodology. And some have questioned whether there should be any economic evaluation of EAP. However, reduced health claims, financial savings, lower absenteeism rates, and increased return on investment have been reported. (See Chapter 19.)

For many employees, a stigma continues to be associated with psychological treatment of any kind. This fear, along with concerns regarding confidentiality, may limit the use of workplace-based mental health resources. Employees may also feel that the company has a vested interest in their productivity that is of greater importance than their health. This concern may be exacerbated by EAPs being gatekeepers with financial incentives to not refer employees for more sophisticated and long-term care and instead refer them to mental health care providers with limited training who may charge the employer less money. Psychologists, psychiatrists, and social workers seem to achieve equally positive outcomes, whereas other counseling professionals who generally charge less money do not appear to achieve outcomes as positive. There are paradoxes embedded in the very nature of EAPs, which lead to conflicting demands and occupational stress for EAP professionals, such

as pressures to provide short-term individual solutions to what may be long-term structural problems.[18]

Implications for Practice and Policy

A tremendous gulf exists between our knowledge regarding job stress and the most efficacious and economical means of preventing it and treating its consequences in the workplace. There is only limited evidence that certain primary prevention interventions have worked. Those that focus on a few stressors and those that do not introduce too many changes too quickly appear to be the most successful. Before primary prevention interventions are designed and implemented, the most prevalent and problematic stressors must be identified and prioritized according to their potency and amenability to meaningful change.[19] Practitioners and researchers should target appropriate objective and subjective outcomes for assessing the efficacy of interventions as well as valid and reliable measures of these outcomes. Objective measures that are organizationally relevant need to be included, without which other organizations will be reluctant to implement these interventions.

Job stress interventions are all too often implemented in relative isolation from one another within an organization. For example, management, human resources, the medical department, and/or the EAP may be given the responsibility for an intervention and there may be little involvement or cooperation of other organizational structures. Any and all interventions for job stress should be integrated within the organization as a whole.

Given that each intervention may not be possible, or effective, for all stressors, a comprehensive approach to the issue of job stress should include all three forms of intervention. Pragmatically, the only way that organizations can reduce risks of occupational stress and resulting organizational costs is to implement effective programs aimed at primary intervention, buffering or remediation (secondary intervention), and treatment (tertiary prevention).

Finally, there is a need to explore the effectiveness of "countervailing interventions" that focus on increasing the positive experience of work rather than decreasing the negative aspects.[14] This approach may be important and viable to improving well-being at work for several reasons. First, enhancing the positive experience of work is consistent with an emergent body of literature showing that characteristics such as hope, self-efficacy, and optimism can be influenced by the workplace. Interventions that enhance these aspects of well-being may a powerful countervailing force that counteracts the effects of job stressors. Second, some research has suggested that mental health is a function of the ratio of positive to negative experiences and that, by extension, interventions that enhance positive work experiences could change the ratio in favor of enhanced mental health. Third, such an approach is consistent with empirical observations that positive experiences in the workplace, such as trust in management and being exposed to positive leadership styles, predict well-being. The range of what might be considered a countervailing intervention may be quite broad.

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Provides in-depth coverage of occupational stress and intervention for occupational stress in today's workplace.

Naswell K, Hellgren J, Sverke M (eds.). The individual in the changing work life. Cambridge, England: Cambridge University Press, 2008.
Provides a good international perspective on occupational stress.

WEB SITES

http://www/cdc.gov/niosh/topics/stress/
This site, sponsored by NIOSH, provides information about job stress and health and links to other sources of information on job stress.

http://www/cdc.gov/niosh/worklife/
This site describes the NIOSH WorkLife Initiative, which seeks to improve worker health through better work-based programs, policies, practices, and benefits.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

SECTION III

ADVERSE HEALTH EFFECTS

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15

Injuries and Occupational Safety

Dawn N. Castillo, Timothy J. Pizatella, and Nancy A. Stout

Occupational injuries are caused by acute exposure in the workplace to physical agents, such as mechanical energy, electricity, chemicals, and ionizing radiation, or from the sudden lack of essential agents, such as oxygen or heat. Examples of events that can lead to worker injury include motor vehicle crashes, assaults, falls, being caught in parts of machinery, being struck by tools or objects, and electrocutions. Resultant injuries include fractures, lacerations, abrasions, burns, amputations, poisonings, and damage to internal organs.

Occupational and nonoccupational injuries represent a serious public health problem (Box 15-1). More than 5,000 workers died from occupational injuries in the United States in 2008.[1] Another 3.5 million workers sustained nonfatal injuries in 2008;[2] this estimate is conservative because it relies on employer reporting, excludes important groups of workers (such as workers who are self-employed and workers on small farms), and may miss counting many cases.[3] An estimated 3.4 million workers were treated in emergency departments for work-related injuries and illnesses in 2004, with approximately 2% of them being hospitalized immediately or transferred to another hospital, such as a trauma or burn center. Although these data include illnesses, more than 90% are injuries.[4] The direct cost of serious occupational injuries in the United States in 2007 was estimated at $53 billion,[5] an amount that includes only wages and medical payments to workers whose injuries resulted in more than 6 days away from work.

CAUSES OF INJURY

Although the immediate cause of injury is exposure to energy or deprivation from essential agents, injury events arise from a complex interaction of factors associated with materials and equipment used in work processes, the work environment, and the worker. These factors include the following: physical hazards in workplaces or work settings, hazards and safety features of machinery and tools, the development and implementation of safe work practices, the organization of work, the design of workplaces, the safety culture of the employer, availability and use of personal protective equipment (PPE), demographic characteristics of workers, experience and knowledge of workers, and economic and social factors.

Box 15-1. Injuries are a Major Public Health Problem

In addition to the workplace, injuries occur at home and school, while traveling, and during recreation. In the United States, injuries are the leading cause of death for persons aged 1 to 44 years, surpassing deaths from cancer, heart disease, and infectious diseases. In 2007 in the United States, 182,479 injury deaths occurred (age-adjusted rate of 59 per 100,000 persons). Injuries contributed to more than 3.8 million years of potential life lost before age 65. In 2008, an estimated 30 million nonfatal injuries required treatment in an emergency department (age-adjusted rate of 9,909 per 100,000 persons).[1]

Many injury causes are common in multiple environments, such as the workplace and home; others are more common in the workplace. Transportation events, violence, falls, and being struck by objects are examples of injury causes that are common in multiple settings; machinery, electrocutions, explosions, and overexertion injuries are more common in the workplace. Strategies for reducing and preventing injuries in multiple settings include changes to the environment (such as changes in roadway design), regulatory policy (such as specifying product safety parameters), and educational approaches. Broad injury prevention measures, such as those focused on improving roadway safety, improve workplace safety. And injury prevention measures in the workplace complement those occurring in other settings.

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CASE 1

An 18-year-old construction worker was using a roller/compactor to compact soil that would be the foundation for a future townhouse. The foundation plot was next to uncompacted soil with a downward slope of approximately 45 degrees. Although the incident was not witnessed, machine tracks suggest that the construction worker maneuvered the roller/compactor partly onto the uncompacted soil next to the foundation plot, which caused it to overturn down the slope. The construction worker was thrown from the roller/compactor and crushed by its roll bar that landed on his back. An unknown amount of time after the rollover, a co-worker noticed the overturned machine. Emergency medical services were called and co-workers used an excavator to lift the roller/compactor off of the worker who was pronounced dead at the scene.[6]

This case illustrates how the occurrence of occupational injury events can be influenced by a variety of factors and circumstances. Some of the contributory factors are clear, whereas others are surmised:

- The residential construction worksite included sloped land that posed risks for operation of equipment such as roller/compactors. The roller/compactor manufacturer recommends that the machine not be used on slopes exceeding a 17-degree incline. The machine was being operated near a 45-degree incline.
- The roller/compactor was equipped with an interlocked seat belt that prevented machine operation unless the seat belt was buckled; however, it appears that the construction worker was sitting on the buckled seat belt, thus overriding this safety feature. Seat belts on machines such as roller/compactors serve to keep workers in a protective envelope or zone in the event of a rollover.
- The small company that employed the construction worker had some elements of a safety program—such as hazard training, monthly employer/management meetings to discuss safety practices, daily worksite inspections, and a progressive disciplinary system for unsafe practices, but the circumstances of this incident illustrate lapses and inadequacies in the program. The foreman and project managers reportedly assumed that each other had trained the construction worker, and they did not realize that the construction worker's training had been limited to observing a co-worker operate the roller/compactor, then demonstrating his ability to operate the machine. If a daily worksite inspection took place, it apparently did not result in any worker guidance

or change in procedures to address the uncompacted sloped soil next to the townhouse foundation. The nonuse of the roller/compactor seat belt by the construction worker was apparently not observed or went uncorrected by management.
- The organization of work, in which workers were expected to work independently and frequently alone, contributed to a delay in lifesaving efforts and may have contributed to the failure to address the hazards of the sloped uncompacted soil and the construction worker not wearing the roller/compactor seat belt.
- The young construction worker apparently was confident in his skills, despite what appeared to be very limited experience and training. This could have contributed to his not recognizing the dangers of the sloped and uncompacted soil and working without the seat belt.
- The construction worker was a recent immigrant from Mexico and did not speak English. To communicate with the worker, the foreman and project managers used another worker as a translator. This could have contributed to the inadequate training and supervision provided to the worker, and the lack of appreciation for the worker's inexperience and limited skills.
- The social and economic realities of construction work can result in high worker turnover, pressures to complete jobs despite unanticipated setbacks, and unanticipated hiring needs, all factors that might have contributed to the young construction worker being allowed to work with minimal training and supervision. The young construction worker had been hired and worked the previous workday to help complete the townhouse project. The task of compacting the townhouse foundation soil was necessary because the ground had previously failed a soil inspection. Pressures to complete the job may have contributed to the lapses in training and safety oversight.

This case illustrates how injury events can arise from a complex array of factors, not all of which contribute equally to an injury event. In addition, the responsibilities for a safe work environment and safe work practices are not borne equally by all involved parties. Employers bear the greatest responsibilities, as they are responsible for providing a safe work environment, including the identification of potential safety hazards and the implementation of hazard controls and safe work practices and procedures. However, workers are also responsible for following established procedures and for reporting safety hazards to employers.

THE EPIDEMIOLOGY OF INJURIES

Occupational injuries are not random events. They cluster or are associated with specific types of workplaces and jobs, workplace exposures, and worker characteristics. Because occupational injuries are not random, they can be anticipated and steps can be taken to prevent them.

Epidemiologic data allow those involved in injury prevention efforts to target groups and settings with high numbers or rates of occupational injuries, and to anticipate and take steps to prevent injuries in specific workplaces or work settings. Epidemiologic data on fatal and nonfatal occupational injuries differ and thus are addressed separately. Both categories of injuries require attention—fatal injuries, because they represent the most severe consequence of occupational injury and are devastating to families, communities, and workplaces; and nonfatal injuries, because of the sheer volume and aggregate costs to workers, families, employers, and society as a whole.

Fatal Injuries

In the United States, data on occupational injury deaths are considered to be very complete. Beginning in 1992, the U.S. Bureau of Labor Statistics (BLS) began collecting data through the Census of Fatal Occupational Injuries (CFOI), which uses multiple sources of data and involves verification of the work-relatedness of deaths.[1] A less complete system based only on death certificates, the National Traumatic Occupational Fatalities (NTOF) system, provides additional data since 1980.[7] Data, such as medical-examiner records, also exist at the state level.

In 2008, there were 5,214 occupational injury deaths in the United States—3.7 occupational injury deaths for every 100,000 U.S. full-time equivalent workers in 2008.[8] The distribution and risks for fatal occupational injury differ by demographic characteristics of workers. Men account for more than 90% of occupational fatalities and have occupational fatality rates approximately 10 times higher than those for women.[7–9] In 2008, of all occupational fatal injuries, 70% were among white non-Hispanic workers, 15% among Hispanic workers, 10% among black non-Hispanic workers, 3% among Asian workers, and 1% among American Indians or Alaska Natives.[1] Hispanic workers have fatality rates consistently higher than the average for all workers,[8] and they are a priority population for fatal occupational injury prevention (Box 15-2). Of all fatal occupational injuries in 2008, 63% occurred to workers between 25 and 54 years of age, 9% to workers younger than 25, and 28% to workers 55 and older.[1] Rates of fatal occupational injury generally increase with age, with the highest rates among workers 65 and older.[7–9] The youngest and oldest workers present both challenges and opportunities for occupational injury prevention (Box 15-3).

In 2008, of all occupational injury deaths, 80% were among wage and salary workers; the remainder were among self-employed workers, whose fatality rate is more than three times greater than that of wage and salary employees.[8] The types of jobs held by self-employed workers explain some of this difference.[10] For example, high proportions of the self-employed work in agriculture and construction, two industries with the highest rates of fatal injury.[8,9]

Transportation-related events accounted for 41% of the 5,214 occupational injury deaths in the United States in 2008 (Fig. 15-1). These events involved motor vehicles and mobile equipment, such as tractors and forklifts; occurred on and off the highway; and included pedestrians and bystanders as well as operators and drivers.[9] Work-related road crashes provide unique challenges and opportunities for prevention (Box 15-4). Contact with objects or equipment accounted for 18% of the fatalities, including being struck by falling objects, being caught in running equipment or machinery, and being caught in or crushed by collapsing materials, such as in trench cave-ins or collapsing buildings. Assaults and violent acts accounted for 16% of fatalities in 2008, with most of them involving homicides and some involving suicides. Violence-related injuries occur in a variety of work situations, and consequently prevention strategies vary (Box 15-5). Falls, mostly to a lower level, accounted for another 13% of the fatalities. Exposure to harmful substances or environments, such as electric current, temperature extremes, hazardous substances, and oxygen deficiency, accounted for 8% of fatalities. Fires and explosions accounted for 3% of the fatalities.[9] Demographic characteristics vary; for example, homicide accounts for a higher proportion of deaths among women than in men.[7,9]

Box 15-2. Hispanics Are a Priority Population for Occupational Injury Prevention

Concomitant with increases of Hispanics in the U.S. population, the proportion of Hispanics in the workforce has increased and is expected to continue to increase. The number of Hispanics in the U.S. workforce increased 54% between 1998 and 2008, and it is expected to increase another 33% by 2018, to more than 29 million Hispanic workers.

Hispanics frequently work in the most hazardous jobs, which helps explain their higher rates of fatal injuries. Fatality rates are highest for foreign-born Hispanic workers. Most of the fatally injured foreign-born Hispanic workers are from Mexico. It is not known to what extent language, literacy, culture, and vulnerable employment situations (such as work as a day laborer and illegal immigration status) contribute to the high injury death rate among foreign-born Hispanics. The National Institute for Occupational Safety and Health (NIOSH) and the National Institute of Environmental Health Sciences (NIEHS) have funded research projects to identify unique risks for Hispanic and immigrant workers and develop and evaluate unique prevention approaches, such as utilizing community-based organizations to communicate safety and health information to Spanish-speaking and immigrant workers.

Many groups are responding to the need for communication of occupational safety and health information to Spanish-speaking and foreign-born workers, addressing issues of language, literacy, and culture.

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Box 15-3. The Youngest and Oldest Workers Present Challenges and Opportunities for Prevention

The U.S. workforce is characterized by involvement of workers from early adolescence to beyond traditional retirement ages. The United States is somewhat unique among industrialized nations in the high participation of youth less than 18 years of age in the workforce. Youth employment can begin as early as the middle school years (11 through 13 years of age), and by the time that young people graduate from high school (17 and 18 years of age), nearly 80% have reported working. As the U.S. population has aged and people have lived longer than in the past, the number of older workers has increased—and this number is expected to continue to grow. The number of workers 55 years and older increased 63% between 1998 and 2008, and it is expected to increase an additional 43% by 2018 to nearly 40 million. In 2009, there were an estimated 1.2 million workers 75 years and older in the United States.[1]

Because of their biologic, social, and economic characteristics, the youngest and oldest workers have unique patterns and risks for work-related injuries. While younger workers have lower rates than older workers for fatal injuries, their rates for nonfatal occupational injury are higher. The higher rates of nonfatal injury are frequently attributed to less experience and training on safety hazards in the workplace. In contrast, the oldest workers have the highest rates of fatal occupational injury, lower rates of nonfatal injury, and longer recovery times once injured. Decreased physical ability to tolerate and recover from injuries may account for the longer recovery times and increased fatality rates. While normal decrements in health associated with aging, such as reductions in visual acuity and slower reaction times, would theoretically lead to increased injuries among older workers, it would appear that work and life experiences contribute to the lower rates of nonfatal occupational injury among older workers. Furthermore, older workers may be assigned to less physically demanding tasks.

It is important to ensure that employers provide new workers with training on the specific safety hazards in their work environment and guidance on how to safely perform their jobs. Additionally, there is potential value in providing youth with basic training on occupational safety before they enter the workforce, as a means of helping to keep them safe in their first jobs, and potentially contributing to a more safety conscious generation of new workers. Along these lines, NIOSH and its partners have designed curricula that can be integrated into high-school programming or be used in other group settings, such as in apprentice training. Several government and private sector entities have also developed educational materials to increase the safety of young workers up to 24 years of age.

At the other end of the age spectrum, older workers bring a wealth of experience and perspective to the workplace. As the workforce continues to age, it is important to understand workplace programs and policies that reduce the risk for injury among an older population facing the realities of the aging process, and to make reasonable accommodations to increase the safety of older workers. Modifying work tasks to account for age-related decrements in functioning may have the added benefit of increasing safety for workers of all ages.

Reference

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The incidence of occupational injury deaths varies by industry sector (Table 15-1), with the most deaths in 2008 occurring in the construction sector, and the highest fatality rates in the agriculture, forestry, fishing and hunting, and mining sectors.[8,9] Numerous specific industries and occupations have injury rates far in excess of the average for all industries and occupations.[7,8] For example, occupations with fatality rates (deaths per 100,000 full-time equivalent workers) more than 10 times higher than the national average in 2008 include the following: fishers and related fishing workers (128), logging workers (120), aircraft pilots and flight engineers (73), structural iron and steel workers (47), and farmers and ranchers (40).[8,9]

Nonfatal Injuries

There is no single data system in the United States that collects data on all nonfatal occupational injuries. The two primary national sources of data on nonfatal work-related injuries are data from the BLS annual survey of employers[2]

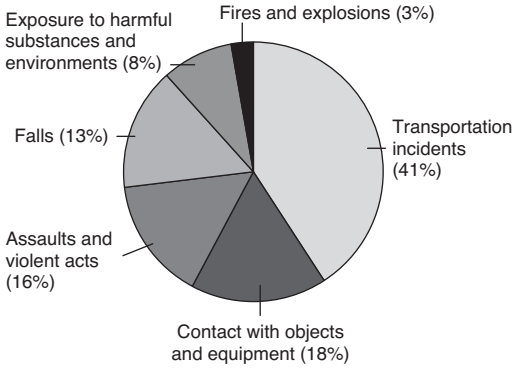

Figure 15-1. Events or exposures leading to occupational injury deaths, United States, 2008. (*Source*: Bureau of Labor Statistics. Census of fatal occupational injuries charts, 1992-2008 (revised data). Washington, DC: U.S. Department of Labor, Bureau of Labor Statistics, 2010. Available at: http://www.bls.gov/iif/oshwc/cfoi/cfch0007.pdf.)

and from emergency departments.[4] Neither system is designed to capture all work-related injuries and both have limitations. The BLS survey is based on employer reports of injuries documented in records required by the Occupational Safety and Health Administration (OSHA). Based on the BLS survey, there were an estimated 3.5 million occupational injuries in 2008.[2] The BLS survey excludes the self-employed, farms with fewer than 11 employees, and federal government employees, and it may miss many cases that should be counted.[3] Data on worker demographics and the circumstances of injuries are available only for lost workday cases in the BLS survey.[11] The emergency department system collects data on injuries treated in a nationally representative sample of emergency departments, with an estimate of 3.4 million occupational injuries and illnesses in 2004.[4] The identification of these cases requires documentation in the emergency department record that the injury was work-related. Research on the completeness of the emergency department data has not been conducted, and information on industry and occupation are not currently available in the emergency department data. An estimated one-third of occupational injuries are treated in emergency departments.[4] Data collected in both systems overlap and are not mutually exclusive. Illnesses, such as dermatitis, are included in both the emergency department data and lost workday data from the BLS employer survey, but they represent less than 10% of cases in both systems.[2,4] Although the data from the BLS survey and emergency departments have limitations and undoubtedly underrepresent the true burden of occupational injuries, they are likely to represent the majority of the more serious injuries, and they provide useful information on epidemiologic patterns of injury. Limited data are also available from the population-based National Health Interview Study (NHIS), which estimated 4 million medically consulted injuries and poisonings that occurred in paid jobs in 2008.[12]

Although not as dramatic as for fatal injuries, differences are seen across demographic categories for nonfatal injuries. Men account for approximately 60% to 70% of nonfatal work-related injuries treated in emergency departments and reported in the NHIS,[4,12,13] but men account for approximately 85% of nonfatal work-related injuries requiring hospitalization.[4] Men have rates that exceed those of women by 58% to 100%.[4,11,13] In 2008, most nonfatal occupational injuries (67%) were among white, non-Hispanic workers, with fewer among Hispanic workers (22%) and black, non-Hispanic workers (7%).[12] About 70% of nonfatal injuries occur among workers 25 to 54 years of age.[4,11,13] Those younger than 25 account for about 20% of injuries treated in emergency departments and reported in the NHIS,[4,13] and 13% of injuries reported by employers as requiring at least 1 day away from work.[11] Workers older than 54 account for 8% to 9% of injuries treated in emergency departments and reported in the NHIS,[4,13] and 16% of injuries reported by employers as requiring at least 1 day away from work.[11] The highest rates of nonfatal occupational injury are among workers about 18 to 24 years of age, with lower rates among workers less than 18 and among older age groups.[4,13] The median number of days away from work, based on employer-reported data, was 8 in 2008, with the median days increasing steadily from a low of 4 days for workers 14 to 15 years of age to a high of 15 days for workers 65 and older.[11]

In 2008, of employer-reported cases, 11% occurred among employees who had worked for less than 3 months for the employer, 20% among employees with 3 to 11 months of service, 36%

Box 15-4. Unique Challenges for Prevention of Roadway Occupational Deaths and Injuries

Roadway crashes are the leading cause of occupational injury deaths in the United States. Between 1992 and 2008, more than 23,000 workers died in highway incidents,[9] averaging more than three deaths daily. Truck drivers account for more roadway fatalities than any other occupational group, and they have the highest rates for roadway worker deaths. However, work-related roadway crashes are not limited to the transportation industry, and many workers in occupations that are not related to transportation are killed each year. Some workers are killed while using vehicles provided by their employers, while others are killed driving their own vehicles to perform their jobs.

Preventing work-related roadway crashes is especially challenging. Unlike other workplaces, the roadway is not a closed environment. Although employers cannot control roadway conditions, they can take a number of steps to help keep their workers safe when driving, such as:

- Implementing and enforcing policies for mandatory use of seat belts
- Ensuring that no workers are assigned to drive on the job if they do not have valid driver's licenses appropriate for the types of vehicles they drive
- Providing fleet vehicles that offer the highest possible levels of occupant protection in the event of a crash
- Maintaining complete and accurate records of workers' driving performance, in addition to driver's license checks for prospective employees and periodic rechecks after hiring, are critical.
- Incorporating fatigue management into safety programs
- Ensuring that workers receive the training necessary to operate specialized motor vehicles or equipment
- Offering periodic screening of vision and general physical health for all workers for whom driving is a primary job duty
- Avoiding requiring workers to drive irregular hours or to extend their work day far beyond their normal working hours as a result of driving responsibilities
- Establishing schedules that allow drivers to obey speed limits and follow applicable hours-of-service regulations
- Setting safety policy in accordance with state graduated driver licensing laws so that company operations do not place younger workers in violation of these laws
- Assigning driving-related tasks to young drivers in an incremental fashion, beginning with limited driving responsibilities and ending with unrestricted assignments

Many of these recommended employer measures are included in the standard of the American National Standards Institute (ANSI) Safe Practices for Motor Vehicle Operations (Z15.1). This voluntary consensus standard, issued in 2006, provides minimum guidelines for employers to develop a motor vehicle safety program. These guidelines are meant for use by employers with vehicle fleets ranging from one vehicle to hundreds of vehicles.

Employees can also take steps to increase their safety while driving in the performance of their work, including the following:

- Using safety belts
- Avoiding placing or taking cell phone calls while operating a motor vehicle
- Avoiding other activities while driving, such as eating, drinking, or adjusting noncritical vehicle controls
- Never attempting to read or send text messages while driving

Source: Excerpted and updated from: Pratt SG. National Institute for Occupational Safety and Health Hazard Review: Work-related roadway crashes: challenges and opportunities for prevention. DHHS [NIOSH] publication no. 2003-119. Cincinnati, OH: NIOSH, 2003.

Further Reading

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with 1 to 5 years of service, and 31% with more than 5 years of service.[11] Most employer-reported injuries requiring time away from work in 2008 occurred Monday through Friday (86%), and between the hours of 8:00 a.m. and 4:00 p.m. (51%). Fifty percent of the employer-reported injuries occurred between 2 and 8 hours into the work shift, with the largest proportion (20%) occurring 2 to 4 hours into the shift.[11]

The types of events leading to nonfatal occupational injuries follow a different pattern than fatal injuries. The most common events resulting in nonfatal occupational injuries include contact with objects and equipment, bodily reaction and exertion, and falls.[4,11] Figure 15-2 shows the distribution of nonfatal occupational injuries treated and released from emergency departments in 2004. Demographic characteristics vary; for example, bodily reaction and exertion, and falls, account for a higher proportion of injuries among women than in men.[4]

The number and rate of nonfatal injuries by industry division vary greatly from the number and rate for injury deaths (Table 15-2).

Box 15-5. Workplace Violence: A Complex Workplace Injury Phenomenon

Homicide is a leading cause of occupational injury death, and workplace violence accounts for many nonfatal injuries each year. Because of news coverage of sensational and more "newsworthy" events, many assume that disgruntled co-workers and former employees account for the bulk of these injury statistics. In reality, violence caused by co-workers or former employees is a relatively small part of the workplace violence problem in the United States. Most work-related violence in the United States is associated with crime, such as robbery, and violence from clients, customers, or patients.

Violence in the workplace has been categorized into four different types of events:

- *Type I: Criminal Intent*: These situations are typically associated with crimes such as robbery, shoplifting, and loitering. A preexisting relationship does not exist between the employee and the perpetrator, and the perpetrator does not have a legitimate reason for being in the workplace.
- *Type II: Customer or Client*: These situations involve customers or clients who have a legitimate reason for being in the workplace. The violence is associated with a business transaction or service. Perpetrators include customers, clients, patients, and inmates.
- *Type III: Worker-on-Worker:* These situations involve violence between co-workers or violence perpetrated against an employee by a former employee.
- *Type IV: Personal Relationship:* In these situations, the perpetrator has a preexisting relationship with the employee and the violence is associated with the relationship rather than the business. These situations include acts of domestic violence against employees while they are at work.

Workplace violence occurs in a variety of workplaces and occupations, although there are some worker groups at increased risk for the more common Type I and II events, including police and corrections officers, taxi drivers, health care providers, and employees in retail settings.

While workplace violence is a complex phenomenon, there are a variety of strategies that employers and workers can use to reduce the risks for violence—some specific to work settings and tasks, and others more general. Workplace violence prevention strategies include the following: modifying the work setting and tasks to reduce the risks for robbery and/or assault (such as by posting signs in retail settings that minimal cash is kept on hand, providing physical barriers between employees and potential criminals or violent clients, ensuring good lighting, and using surveillance cameras and/or security guards); establishing workplace policies for "zero violence tolerance" and procedures for reporting and following up on all threats or violent acts; and training employees on how to handle criminals or violent customers or clients.

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National Institute for Occupational Safety and Health. Current intelligence bulletin 57: violence in the workplace: risk factors and prevention strategies. DHHS [NIOSH] Publication No. 96-100. Cincinnati, OH: NIOSH, 1996.

Peek-Asa C, Howard J, Vargas L, Kraus J. Incidence of non-fatal workplace assault injuries determined from employer's reports in California. Journal of Occupational and Environmental Medicine 1997; 39: 44–50.

Most injuries in 2008 occurred in the manufacturing sector, and the highest injury rates were in the transportation and warehousing sector.[2] The occupational injury rate in 2008, averaged across all industries and state and local governments, was 4.0 per 100 full-time equivalent workers. Because the BLS annual survey of employers excludes farms with fewer than 11 employees, the numbers of nonfatal occupational injuries reported for the agriculture, forestry, fishing, and hunting sector should be considered as conservative estimates. In a separate survey of U.S farm operators, the number of injuries was much higher than reported in the BLS survey of employers (74,800 occupational injuries; 13.1 injuries per 1,000 workers.)[15]

Clinical Presentation and Course of Injuries

Of all workers with occupational injuries, an estimated 34% are treated in emergency departments in the United States;[4] the remainder are treated at workplaces, and at physician's offices, clinics, and other medical treatment facilities. In 2004, the most common diagnoses of workers treated for occupational injuries in emergency departments were as follows: sprains and strains (28%); lacerations, punctures, amputations, and avulsions (25%); contusions, abrasions, and hematomas (17%); dislocations and fractures (7%); and, burns (3%).[4] Most sprains and strains (55%) were to the trunk area (shoulder, back, chest, or abdomen), followed by the lower extremities (legs, feet, and toes) (25%).

Table 15-1. Number and Rate of Fatal Occupational Injuries, by Industry Sector, United States, 2008

Industry Sector	Number of Fatalities	Fatality Rate*
Construction	975	9.7
Transportation and warehousing	796	14.9
Agriculture, forestry, fishing and hunting	672	30.4
Government	544	2.4
Manufacturing	411	2.5
Professional, scientific, management, administrative	403	2.8
Retail trade	301	2.0
Arts, entertainment, recreation, accommodation, and food services	238	2.2
Wholesale trade	180	4.4
Other services, except public administration	178	2.6
Mining	176	18.1
Educational, health and social services	141	0.7
Finance, insurance, real estate, and rental and leasing	106	1.1
Information	47	1.5
Utilities	37	3.9
Total	**5,214**	**3.7**

Note: For 9 fatalities, industry sector not reported.
*Rate per 100,000 full-time equivalent workers
Source: Bureau of Labor Statistics. Fatal occupational injuries, total hours worked, and rates of fatal occupational injuries by selected worker characteristics, occupations, and industries, civilian workers, 2008. Washington, DC: U.S. Department of Labor, Bureau of Labor Statistics, 2010. Available at: http://www.bls.gov/iif/oshwc/cfoi/cfoi_rates_2008hb.pdf. Accessed on June 18, 2010.

About 75% of the lacerations, punctures, amputations, and avulsions were to the upper extremities (arms, hands, or fingers). Almost 2% of occupational injuries resulted in hospital admission.[4] Dislocations and fractures, caused mostly by falls, accounted for 40% of the hospitalizations among males and 33% among females.

Of the estimated 1.1 million injuries and illnesses with lost work days in 2008, the median time away from work was 8 days. Median time away from work was highest for fractures (28 days), carpal tunnel syndrome (28 days), and amputations (26 days).[11]

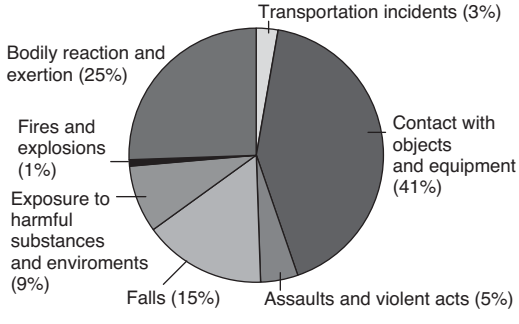

Figure 15-2. Events or exposures leading to occupational injuries treated and released from emergency departments, United States, 2004. (*Source*: Derk SJ, Marsh SM, Jackson LL. Nonfatal occupational injuries and illnesses–United States, 2004. Morbidity and Mortality Weekly Report 2007; 56: 393–397.)

PREVENTION OF INJURIES

The Hierarchical Approach to Occupational Injury Control

Over the years, a number of models for occupational injury control have evolved. Many of these models categorize worker protection strategies based on a hierarchical approach,[16] such as the five-tier model (Table 15-3). The hierarchical approach focuses on *(a)* eliminating hazards through design; *(b)* using safeguards which eliminate or minimize worker exposure to hazards; *(c)* providing warning signs or devices to identify and alert workers to hazards; *(d)* training workers in safe work practices and procedures; and *(e)* using PPE to prevent or minimize worker exposure to hazards or to reduce the severity of an injury if one occurs.

William Haddon, Jr., proposed 10 basic strategies for injury prevention that have several

Table 15-2. Number and Rate of Nonfatal Occupational Injuries, by Industry Sector, United States, 2008

Industry Sector	Number of Injuries	Injury Rate*
State and local government	867,600	5.9
Education and health services	653,600	4.7
Manufacturing	630,600	4.6
Retail trade	520,600	4.3
Leisure and hospitality	368,400	4.1
Construction	314,200	4.6
Professional and business services	249,100	1.8
Transportation and warehousing	233,600	5.5
Wholesale trade	211,300	3.6
Financial activities	101,600	1.4
Other services, except public administration	91,900	3.0
Information	48,700	1.9
Agriculture, forestry, fishing and hunting	43,700	4.9
Mining	23,700	2.9
Utilities	17,600	3.2
Total	**4,376,300**	**4.0**

*Rate per 100 full-time equivalent workers.
Source: Bureau of Labor Statistics. Workplace injuries and illnesses—2008. News Release USDL 09-1302. Washington, DC: U.S. Department of Labor, Bureau of Labor Statistics, 2009.

similarities to the hierarchical approach, such as hazard elimination, hazard reduction, and use of barriers for protection.[17] He also introduced the concept that injury causation was a chain of multifactorial events, each of which provided opportunities for intervention. Herb Linn and Alfred Amendola suggested an approach that, for injury control, combines the public health model with safety engineering analysis.[18] Epidemiology, safety engineering, biomechanics, ergonomics, psychology, safety management, and other types of expertise comprise a multidisciplinary approach that is useful for identifying injury risk factors and developing control strategies.

Three main categories of control strategies correlate with the hierarchical approach: engineering controls, administrative controls, and the use of PPE.

Table 15-3. Safety Hierarchy

Priority Rank	Safety Action
1	Eliminate hazard and/or risk
2	Apply safeguarding technology
3	Use warning signs
4	Train and instruct
5	Use personal protective equipment

Source: Barnett RL, Brickman DB. Safety hierarchy. Journal of Safety Research 1986; 17: 49–55.

Engineering Controls

Engineering controls, also known as passive controls, eliminate hazards through equipment or systems design or prevent worker exposure to hazards through the application of safeguards. Effective hazard elimination and safeguards are designed or retrofitted into equipment, work stations, and work systems to provide protection without direct worker involvement—thus, the term "passive controls." To be most effective, engineering controls must be designed so that they do not adversely interfere with the work process or introduce additional hazards.

The optimal injury control strategy is to eliminate a hazard completely. Frequently, hazard elimination or the reduction of hazard severity can be accomplished through equipment or systems design.

CASE 2

A 36-year-old male Hispanic laborer died after becoming engulfed in sawdust inside a sawmill storage silo. The flat-bottomed silo used a three-armed rotating sweep auger mechanism to funnel stored sawdust through an opening in the silo floor to a transfer auger, which

transported the sawdust to another part of the sawmill for use in generating electricity for the mill. Due to the flat-bottom design of the silo, the sweep auger was prone to frequent clogs, requiring workers to manually unclog the system with rakes and poles. On the day of the incident, the victim entered the silo to manually clear a clog, and, after a short time that he was inside, sawdust that had accumulated on the sides of the silo collapsed, completely engulfing him.

Although several factors contributed to this worker's death, the National Institute for Occupational Safety and Health (NIOSH) recommended retrofitting the silo with a mechanical leveling/raking device that improves the flow of loose materials, such as sawdust, to minimize or eliminate the need for worker entry into this confined space.[19]

Because hazard elimination is not always possible, other control strategies in the hierarchy must be implemented to achieve worker protection. If a hazard cannot be eliminated completely, then the next control level should be to prevent worker exposure through protective safeguarding approaches. These types of safeguards prevent worker exposure to the hazard, as long as the control is in place and functions properly.

For example, many types of industrial equipment require power transmission units that include belts, pulleys, gears, shafts, and other mechanisms necessary for the equipment to function. Workers can be exposed to serious, or even fatal, injury hazards if they contact these rotating or moving components. A fixed barrier guard that completely encloses the power transmission unit is an engineering control that protects workers from being caught in or struck by hazards by preventing worker contact with any moving parts. As long as the guard remains in place, the worker is protected from injury. Another engineering control is an optical sensor, also called a light curtain, used to protect the worker from injury when operating a mechanical power press (Fig. 15-3). The optical sensor is integrated into the press control mechanism so that if any part of the worker's body breaks the plane of light in front of the hazardous point of operation, the downward motion of the press ram cannot be initiated or, if motion has begun, the press ram is automatically disengaged.

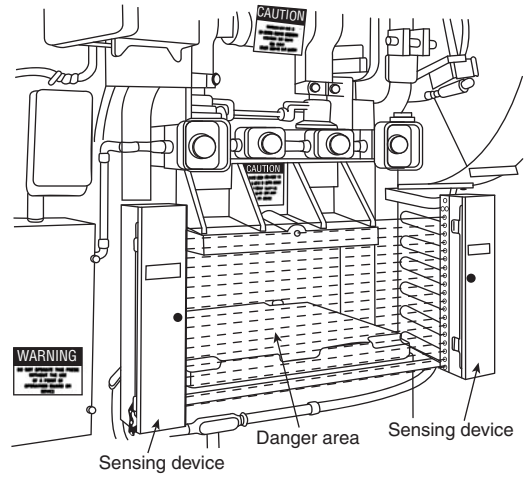

Figure 15-3. Photoelectric (optical) sensor installed on a mechanical power press to protect the point of operation. (*Source*: Occupational Safety and Health Administration. Concepts and techniques of machine safeguarding. Washington, DC: OSHA, 1980.)

Many engineering controls are interlocked to ensure that they cannot be removed without disabling the machine or equipment. An interlock is a device that is integrated into the control mechanism of a machine or work process to prevent the work cycle from being initiated until the interlock is closed, signaling the equipment that the work cycle can be initiated. One example is a skid-steer loader with interlocked driver controls that require the operator be properly positioned inside the equipment, with the seat belt fastened, before the equipment can be started and the bucket raised. Interlocks, which are usually electrical or mechanical controls, need to be designed so that they are not easily bypassed or disabled.

Although engineering controls should be viewed as primary tiers of prevention, it is not always possible to develop such controls for all potentially hazardous work situations. Administrative controls are the next tier for reducing or minimizing worker exposure to injury hazards.

Administrative Controls

Administrative controls are management-directed work practices or procedures which,

when implemented consistently, will reduce the exposure to hazards and the risk of injury. They are sometimes referred to as active controls because they require worker involvement to be effective. The use of warning signs and devices, and worker training on safe work practices and procedures, are considered administrative controls since workers must be actively involved for these to be effective. Workers must adhere to warning signs that identify potential injury hazards and apply the training they have received properly. Other examples of administrative controls include housekeeping procedures requiring that spills or debris be cleaned up quickly to reduce the potential for a slip, trip, or fall injury (Fig. 15-4), and implementation of a hazardous energy control policy for workers performing maintenance activities on a machine. Lockout/tagout procedures are important components of a hazardous energy control policy (Fig. 15-5).

Figure 15-4. Example of poor housekeeping on a construction site. Numerous cords and debris create a potential tripping hazard for workers. (Photograph by Earl Dotter.)

However, to be effective, the procedures must be written and consistently implemented, and workers must be trained in their use.[20]

Personal Protective Equipment

Personal protective equipment consists of devices worn by workers to protect them, by reducing *(a)* the risk that exposure to a hazard will injure the worker or *(b)* the severity of an injury if one does occur. Although the hazard still exists, the potential for worker injury is mitigated by use of PPE. The use of PPE in many work environments and situations is essential

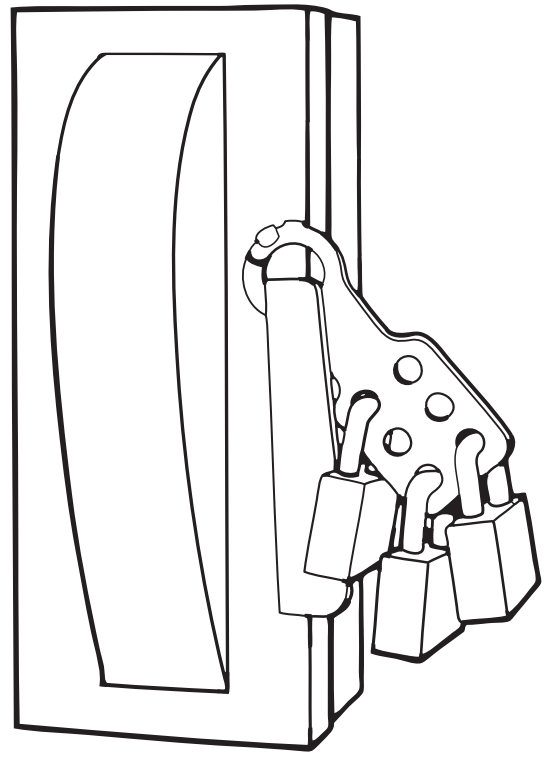

Figure 15-5. Lockout hasp on an electrical control panel, which provides a method for applying a lock (lockout) to the panel during maintenance or repair to ensure that the equipment is not energized until the work has been completed. The control panel should also be tagged (tagout) with a label indicating that work is being performed. Workers should be provided with individually keyed locks, and only the worker who applied the lock should remove it. (*Source*: Occupational Safety and Health Administration. Concepts and techniques of machine safeguarding, Washington, DC: OSHA, 1980.)

for worker protection. However, PPE is usually viewed as the lowest tier in the hierarchy of controls. If hazardous exposures cannot be eliminated through engineering controls or the application of administrative controls, then PPE provides another opportunity for worker protection. Examples of PPE designed to reduce worker injuries include protective hard hats, eyewear and face shields, steel-toed safety shoes, fall restraint devices, and personal flotation devices (Fig. 15-6). When worn properly and consistently, these devices can prevent, or at least reduce the severity of, traumatic injuries. Fall restraint devices, such as lanyards and body harnesses, do not prevent workers from falling, but they protect them from suffering more serious injuries or fatalities due to falls from elevations (Fig. 15-7).

Combined Application of Controls

A comprehensive approach to worker injury prevention efforts inevitably includes all tiers of the control hierarchy to achieve maximum worker protection. In most work environments, a combination of engineering controls, administrative controls, and PPE will be required to have a complete and effective injury prevention program. The following examples illustrate how the combined application of controls can be used to achieve an enhanced level of worker protection.

Tractors equipped with a rollover protective structure, an engineering control, significantly reduce the risk that the operator will be injured in a rollover event (Fig. 15-8). However, more effective protection can be achieved if a seat belt, an administrative control, is worn to keep the operator within the protective envelope of the rollover protective structure. A similar example is the increased protection afforded by the combined use of seat belts, mandated in company safety policies and programs, in motor vehicles that are also equipped with air bags.

Figure 15-6. Example of worker using multiple forms of personal protective equipment, including hard hat, face shield, hearing protection, work gloves, knee pads, and work boots. (Photo courtesy of Mine Safety Appliance Company.)

Training

Training refers to methods to assist individuals in acquiring knowledge (safety information on potential workplace hazards), changing attitudes (perceptions and beliefs regarding safety), and practicing safe work behaviors (organizational, management, or worker performance). Despite inadequate data on the direct relationship between training and injury, evidence suggests a positive impact of training on establishing safe working conditions.[21] Training is one of the key factors accounting for differences between companies with low and high injury rates. It is often critically important for developing and implementing effective hazard control measures.[21,22] Training increases hazard awareness and knowledge, facilitates adoption of safe work practices, and leads to other workplace safety improvements. Training is an administrative control, as workers must properly use training they have received on a consistent basis for it to be effective in preventing injuries.

The elements of effective training programs are *(a)* assessing training needs specific to the work task; *(b)* developing the training program to address these needs specifically; *(c)* setting clear training goals; and *(d)* evaluating the post-training knowledge and skills and providing feedback to the workers.[21] Other important

Figure 15-7. Worker wearing a full-body harness with attached lanyard properly tied off to a life line. (Photo courtesy of the Mine Safety Appliance Company.)

characteristics of a successful program are management commitment to safety and training that is initiated as soon as a worker is hired and then is followed up with periodic retraining and reinforcement.[21,22]

Unique characteristics of the specific workforce must be considered when developing or implementing safety training programs. Language, literacy, cognition, and cultural issues may diminish the effectiveness of training when programs are not tailored to account for unique or diverse characteristics of the workforce. Workplace safety training appears to be most effective when it includes active learning experiences that stress worksite application, and when it is developed and implemented in the context of a broader workplace-based prevention approach.[21]

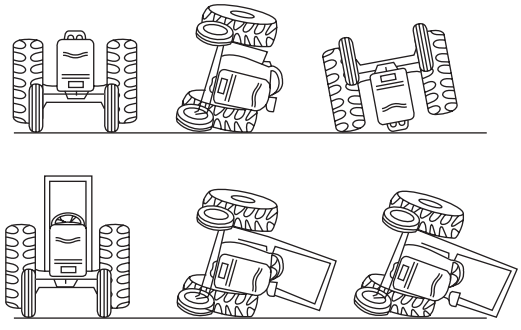

Figure 15-8. Tractor with a two-post roll-over protective structure (ROPS) frame installed. A ROPS is designed to reduce the risk of injury or death by preventing the tractor from rolling onto and crushing the operator. A properly fastened seat belt greatly improves the chances that the operator will stay within the protective envelope provided by the ROPS (the seat). (*Source*: National Institute for Occupational Safety and Health. Safe grain and silage handling. DHHS (NIOSH) Publication No. 95-109. Washington, DC: Author, 1995.)

Standards

Many standards aim at protecting workers from traumatic injury. These standards cover a multitude of hazards and address the work environment, work practices, equipment, PPE, and worker training. The two primary types of worker protection standards consist of *(a)* mandatory standards, such as those promulgated by OSHA or other regulatory agencies, and *(b)* voluntary standards, such as those developed through independent organizations, like the American National Standards Institute (ANSI), through a consensus process involving various stakeholders in an industry—typically including representatives from labor, management, and government. Numerous specifications, codes, and guidelines for machinery, equipment, tools,

and other materials can also assist engineers and designers in developing safer products and systems, many of which have application in the workplace. Examples include the National Electric Code (NEC) published by the National Fire Protection Association (NFPA) and numerous consensus standards from the American Society of Mechanical Engineers (ASME) and the American Society for Testing and Materials (ASTM).

Injury Control: Roles and Responsibilities

Occupational injury prevention is not the sole responsibility of a single person or group. Employers, workers, public health and safety practitioners, researchers, regulators, and policy makers each share in the responsibility for prevention. A multidisciplinary approach involving interaction among diverse groups is crucial to developing and implementing effective occupational injury prevention strategies.

Within an organization, active participation by both management and workers is essential to an effective safety program. Safety and prevention should be integrated throughout the organization with everyone sharing responsibility. Employers are responsible for establishing written safety policy, developing a comprehensive safety program, and effectively implementing that program at the workplace. A competent person or committee should be designated with responsibility for overall planning and implementation of company safety policy. This person or committee should have sufficient knowledge concerning safety policy, standards, regulations, and hazard abatement, and should actively participate with managers and workers in coordinating and overseeing the safety program.

An effective safety program will strive to identify hazards through job safety analysis or other methods of systems safety analysis and will eliminate or control identified hazards through the various approaches previously described. Workers, managers, and safety specialists should work together to analyze the job and potential hazards and to recommend changes or controls to abate them to avoid an injury event. Table 15-4 includes injury hazards with examples from each of the three main categories of hazard control strategies: engineering, administrative, or PPE. The most comprehensive safety programs will typically require strategies from all three categories. In industries or jobs where the work environment is not constant, site hazard assessments should be performed prior to beginning work in any new or changing environment. Occupations such as farming, logging, construction, oil and gas extraction, and mining are characterized by frequently changing work sites and require a site hazard assessment prior to commencing work in any new or changed environment. This requirement is particularly important in industries such as construction and utility maintenance, where worksites change not only from job to job but also from day to day—even hour to hour, with constant potential for new hazards.

Employers are also responsible for ensuring proper maintenance of vehicles, equipment, and machinery and their safety features, such as machine guarding, interlocks, warning systems, and barriers. Where job hazards cannot be eliminated or controlled, employers are responsible for providing appropriate PPE, such as fall arrest systems, respirators, hearing protectors, hard hats, or eye protectors.

Employers must also ensure that workers receive appropriate training in minimizing their risk—including training on safety policy and practice, hazard recognition and control technologies, and the appropriate use of PPE. Enforcement of safety policy is also a critical employer responsibility. The demonstrated commitment of management to safety is a major factor in successful workplace safety programs.[23–25] Employers are more likely to have successful safety programs when they demonstrate concern by having top managers personally involved in safety activities and routinely involve workers in decision making about safety matters. As part of a comprehensive safety program, employers should require systematic reporting and tracking of occupational injuries and assessment of this information for corrective action to prevent similar occurrences.

Workers also play a vital role in workplace safety. Their participation is essential. Workers share in the responsibility for complying with safe work practices and policies, maintaining a safe work area, and using appropriate PPE when required by their employer. Workers should also

Table 15-4. Injury Hazards and Example Control Strategies by Category

Injury Hazard	Engineering Controls	Administrative Controls	Personal Protective Equipment
Motor vehicle crashes	Ensure all vehicles are equipped with air bags	Implement a mandatory seat belt policy	Provide helmets and eye protection for workers whose job requires operating motorcycles or bicycles
Assaults	Install bullet-resistant barriers or enclosures in retail settings	Train workers in nonviolent response when confronted with volatile situations	Provide body armor for public safety workers
Falls from elevation	Install grids or screens over skylight fixtures that meet OSHA standards for protection from falls through skylights	Train workers to set up extension ladders at the proper inclination angle of 75 degrees	Provide personal fall arrest systems during work at elevations
Falls to same level	Redirect downspouts away from walkways with high pedestrian traffic	Implement a policy encouraging workers to clean up or report floor spills promptly	Provide or require workers to wear shoes with slip-resistant soles
Caught in	Ensure that controls on skid-steer loaders are interlocked and require operators to be properly positioned with seat belts fastened before the vehicle can be started and the bucket raised	Develop standard procedures for safely clearing material jams on machinery and equipment	Ensure long hair is tied back or covered when working around machinery with rotating or moving components
Struck by	Install fencing or other physical barriers around robots or other moving equipment, with access through interlocked gates	Minimize forklift traffic during shift changes to reduce exposure to moving forklifts during times when large numbers of workers pass through an area during a short time period	Provide protective hard hats, eyewear, and shoes
Contact with electrical energy	Install ground fault circuit interrupters (GFCIs) in damp or wet locations	Develop and implement a hazardous energy control policy for all maintenance and repair activities	Provide electricians with properly rated di-electric gloves when procedures require work on energized components, such as troubleshooting an electrical panel
Overexertion	Use mechanical lifting devices, such as ceiling mounted cranes, to lift heavy and bulky items	Use job rotation schedules with different physical demands to reduce the frequency of lifting and repetitive motion tasks	Provide workers with nonslip safety gloves during materials handling tasks
Confined spaces	Where possible, locate serviceable components, such as pumps, agitators, and gauges outside of confined spaces so that entry is not required for maintenance, repair, or monitoring	Ensure workers test any confined space for flammable, toxic, or oxygen-deficient atmospheres prior to entry; identify and post warning signs outside of all confined spaces	Provide self-contained breathing apparatus (SCBA) or other appropriate air-supplied respirators if entry is required into spaces with flammable, toxic or oxygen-deficient atmospheres

participate in company-sponsored training. They should report injuries and unsafe conditions for corrective action. As the experts in their jobs, workers should be involved in systems safety analysis and development of safe solutions. Workers' input into recommended design or modification of safety controls, processes, or technology and into the development of safe work practices increases the acceptance of positive changes and, thus, the success of safety programs.

An effective workplace safety program that minimizes injuries results from a multidisciplinary activity that actively involves every level

of the workforce, from the employer and upper-level managers to employee representatives and hourly workers. Each must assume some responsibility for safety and must work together interactively to achieve the common goal of preventing injuries.

Researchers provide science-based approaches to workplace injury prevention. The development of injury prevention strategies and technologies, through laboratory studies and field evaluations, yields evidence-based strategies and solutions to existing and emerging hazards. It is important for researchers and industry to work together in partnership throughout the research process to ensure that prevention strategies are relevant and applicable to the workplace, to demonstrate and evaluate prevention effectiveness in actual work settings, and to facilitate the transfer of research results to implementation and practice in the workplace. Injury prevention

Box 15-6. Unique Role for Public Health Agencies in Occupational Safety

In 2008, the National Institute for Occupational Safety and Health (NIOSH), in conjunction with the Council of State and Territorial Epidemiologists (CSTE), updated the publication *Guidelines for Minimum and Comprehensive State-Based Public Health Activities in Occupational Safety and Health*. This publication highlights the important role of state public health agencies in fostering occupational safety and health, based on the three core functions of public health identified by the Institute of Medicine in 1988: assessment, policy development, and assurance.

Assessment: Assessment involves the regular and systematic collection, analysis, and communication of the public's health, including statistics on health status. There are numerous state-level data sources for assessing occupational injuries that include injuries not captured in the national occupational injury systems overseen by the Bureau of Labor Statistics (BLS). These unique state-level data include hospital discharge data, emergency department data, workers' compensation records, burn center data, and poison control centers' data. CSTE has identified key occupational injury indicators that use existing state-level data to assess and track trends in occupational injuries at the state level, and these have been reported by 15 states to date. In-depth analyses of state-based occupational injury surveillance data have been conducted in several states, leading to state-specific injury prevention efforts, including prevention of burns and injuries among teen workers.

Policy development: Policy development involves the responsibility to develop public health policies based on scientific knowledge. Examples of how state health departments can contribute to sound policy development to improve worker safety include the following: collaborating with stakeholders in establishing statewide occupational safety objectives, such as the Healthy People 2020 objectives for the nation to reduce occupational injuries; collaborating with public health partners to encompass the prevention of occupational injuries in broad state-wide injury prevention programs and plans (such as those focused on reducing transportation injuries and injuries to adolescents); developing programs and working relationships with partners such as state labor departments and OSHA to collectively work toward preventing occupational injuries; and developing program capacity to identify and respond to emerging occupational safety hazards or unique prevention opportunities.

Assurance: Assurance involves making sure that services are available at the state-level to achieve agreed upon goals, such as injury prevention generally, or occupational injury-specific goals. State health departments should have sufficient occupational safety expertise and resources to meet their populations' information needs and to be able to provide appropriate referrals for technical assistance.

Public health agencies have statutory, regulatory, and philosophical commitments to protect the public's health, including vulnerable groups who may fall outside the jurisdiction of federal or state regulatory agencies. The NIOSH/CSTE publication noted above provides guidelines on developing state-based public health programs in occupational safety and health, ranging from minimum activities that can be performed with existing state health department staff and data, to more comprehensive approaches that require additional resources. It is intended that these guidelines will be used by state health agencies to develop the capacity for minimum activities in every state and to enhance existing programs. Numerous examples of state-based public health activities in occupational safety and health suggest that state public health agencies have a critical and complementary role to state labor agencies in preventing occupational injuries.

Further Reading

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research results will only be effective in reducing injuries if they are directly communicated and transferred to employers, trainers, safety practitioners, regulators, and policy makers who can implement research results for prevention action. This research-to-practice process, developing and applying science-based prevention strategies in the workplace, is also a shared responsibility of the multiple entities with vested interest in workplace injury prevention.

Government agencies also play a role in preventing occupational injuries. Federal and state labor agencies are involved in data collection on occupational deaths and injuries through the BLS, and they serve a regulatory function by establishing standards for safe work practices and enforcing those regulations. Federal OSHA, and 27 states and territories authorized by OSHA, promulgate and enforce mandatory minimum standards for occupational safety and health. Federal and state labor agencies also provide consultative services to employers and education to raise awareness about their standards and injury prevention practices. State health departments are involved in occupational safety at varying levels, including the following: the collection, analysis, and interpretation of unique data not collected by BLS; disseminating occupational injury prevention recommendations using state networks; and ensuring that occupational injury prevention is encompassed within state injury prevention plans. Increasing state health department involvement in occupational safety holds considerable potential for improving worker safety (Box 15-6).

Occupational injuries continue to exert too large a toll on the workforce. While the rate of fatal injuries in the United States has decreased markedly over time, the rate of nonfatal injuries has not been reduced as much.[4] The prevention of workplace injuries requires concerted and consistent efforts from multiple parties using multiple strategies. In addition to the primary stakeholders in the workplace, additional groups can help reduce occupational injuries. These groups include researchers who provide the evidence base for effective prevention strategies and technologies, manufacturers and distributors of industrial equipment and tools that design and promote safety features of equipment, insurers who provide monetary incentives for good safety records and practices, and health care providers and public health practitioners who provide their patients and constituents with information on preventing workplace injuries.

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American National Standards Institute Technical Report. Risk assessment and risk reduction—A guide to estimate, evaluate and reduce risks associated with machine tools. B11.TR3: 2000. McLean, VA: The Association For Manufacturing Technology, 2003.
A technical report that is part of the ANSI B11 series pertaining to the design, construction, care, and use of machine tools. This report provides a method for both machine suppliers and users to conduct a risk assessment (analyze hazards) for industrial machinery and related components, and it includes guidance for selecting appropriate safeguarding to reduce the risk of worker injury.

Christoffel T, Gallagher SS. Injury prevention and public health: practical knowledge, skills, and strategies. Gaithersburg, MD: Aspen Publishers, Inc, 1999.
A reference document that includes information on injury epidemiology and prevention strategies. The document includes chapters on conducting injury

surveillance, developing an injury prevention program, and evaluating injury prevention measures.

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A good overall reference on occupational safety and health issues. Provides an overview of standards and codes for workplace safety; identifying and controlling hazards; analyzing safety hazards and conducting incident investigations; and developing and implementing workplace safety programs. Addresses both the engineering and management aspects of occupational injury and disease prevention.

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A reference document that includes occupational injury data from multiple sources, including the BLS Census of Fatal Occupational Injuries and Annual Survey of Occupational Injuries and Illnesses. Data are presented in figures and tables; also included are charts on special topics, such as construction and agricultural injuries; young and older worker injuries; Hispanic worker injuries; and fractures, burns, and amputations.

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This comprehensive manual provides guidance for health and safety education to workers, including guidance specific to health care providers, as well as information for occupational safety and health training resources.

16

Musculoskeletal Disorders

Barbara Silverstein and Bradley Evanoff

Work-related musculoskeletal disorders (WMSDs) are a common result of excessive work-related physical and psychosocial demands. We will first describe WMSDs in terms of magnitude and cost and then proceed to describe recognition, risk factors, and treatment strategies for disorders of the neck and arm (shoulder to the hand), the back, and leg (hip to foot). We will then describe the advantages of an ergonomics program in preventing WMSDs and facilitating return to work for those who have experienced a WMSD.

Work-related musculoskeletal disorders are soft-tissue disorders of nontraumatic origin that are caused or exacerbated by interaction with the work environment. Recognition of the work-relatedness of musculoskeletal disorders (MSDs) goes back at least to the early 1700s, when Bernardino Ramazzini noted the harmful effects of unnatural postures and movements, such as the numbness in the upper extremity in scribes due to "incessant movement of the hand and always in the same direction," or sciatica in potters due to continual turning of the potter's wheel. The general public has used terms such as repetitive strain injury, washerwoman's sprain, telegrapher's cramp, and carpet layer's knee, and, more recently, "mouse hand" or "mouse shoulder" and "cell-phone thumb" to describe relationships between work and MSDs.

The most commonly reported body areas affected by WMSDs are the neck, the upper extremities (arms) and the low back. There is increasing evidence of work-relatedness for some common hip and knee disorders. Tendonitis and tenosynovitis, the most common WMSDs, are inflammatory disorders of the tendon and tendon sheath. Specific examples of these disorders include rotator cuff tendonitis, epicondylitis, extensor and flexor tendonitis of the wrist, and peripatellar tendonitis of the knee. WMSDs can cause pain, burning, and/or numbness and tingling, resulting in losses of work time and productivity. Symptoms can initially be intermittent and mild, but, in the absence of treatment, may progress to become more frequent and severe. Figure 16-1 presents a conceptual model of the contributors to musculoskeletal disorders, which include workplace factors, individual factors, and their interaction. Attribution of musculoskeletal disorders to work activities can be difficult and controversial, as illustrated in Box 16-1.

MAGNITUDE AND COST

For 2007, the Bureau of Labor Statistics (BLS) reported 333,760 WMSDs in private industry in the United States—an annual incidence rate (IR) of 35 per 10,000 workers. Work-related musculoskeletal disorders accounted for 29% of

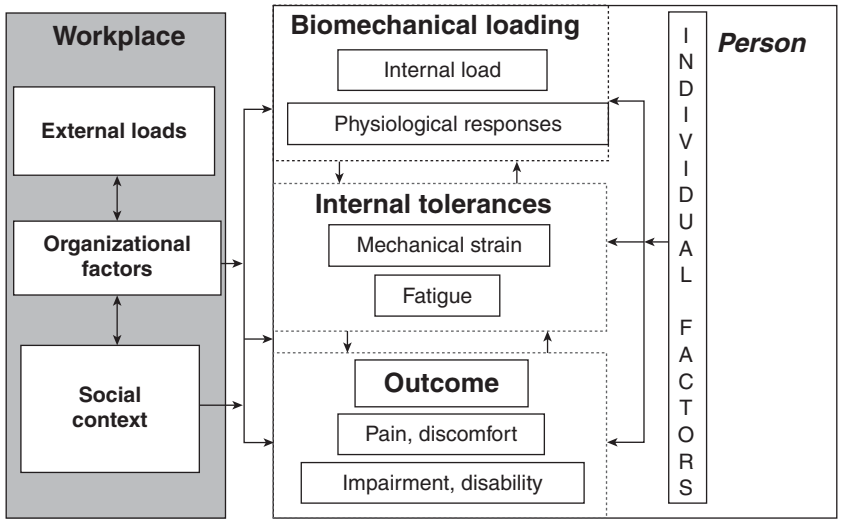

Figure 16-1. Conceptual model of contributors to musculoskeletal disorders. (Adapted from Institute of Medicine. Musculoskeletal disorders and workplace: low back and upper extremities. Washington, DC: National Academies Press, 2001.)

Box 16-1. Plumber's Knee

A plumber was forced to retire at age 50. He was a plumber for 32 years. He spent 65% of his work time kneeling and squatting. This was frequently combined with heavy lifting. This led to numerous knee surgeries.

- First sought treatment for pain and swelling in 1980
- Arthroscopic surgery to repair torn meniscus in the knees in 1985
- Filed initial workers' compensation claim in 1983–1985
- Filed another claim in 1998 because first surgery not fully successful
- In 2003, the Vermont Supreme Court ruled that knee deterioration after 1995 was wholly attributable to the earlier injuries.

Comment: There are at least three features of work-related musculoskeletal disorders that contribute to controversy over attribution: *(a)* gradual onset (days to years), *(b)* none are uniquely caused by work, and *(c)* ubiquity of risk factors. Figure 16-1 is adapted from the 2001 National Research Council Institute of Medicine report on MSDs and the workplace. The basic mechanism for these disorders appears to be overloading tissue tolerance with insufficient recovery time. A variety of individual (gender, age) and lifestyle (obesity, smoking, exercise), biomechanical, organizational, and social factors may contribute to the tension between overload and recovery.

Source: Workplace Ergonomics News 2003; 5: 6.

all injuries and illnesses. On average, they resulted in a median of 9 days away from work. The service and manufacturing sectors accounted for about one-half of all WMSD cases. Nursing aides and orderlies had the highest IR (252 cases per 10,000 workers), followed by laborers and freight handlers (IR = 149) and light-truck and delivery-truck drivers (IR = 117). Changes in BLS case definitions have affected national data collection on WMSDs, likely accounting for more underestimation of true occurrence of these disorders.

The current BLS definition of MSDs (last modified in November 2008) includes cases where the nature of the injury or illness is a sprain, strain, or tear; back pain, hurt back; soreness, pain, or hurt, except the back; carpal tunnel syndrome; a hernia; or a musculoskeletal-system or connective-tissue disease or disorder, when the event or exposure leading to it was bodily reaction/bending, climbing, crawling, reaching, or twisting; overexertion; or repetition. Cases of Raynaud phenomenon, tarsal tunnel syndrome, and herniated spinal discs are not included in this definition; although these disorders may be considered MSDs by others, the BLS survey classifies them in categories that also include non-MSD cases, such as "injuries."

There has been an interesting progression of recording procedures for WMSDs on the Occupational and Safety Administration (OSHA) 200–300 logs. Originally, there was a column (7f) to record "disorders associated with repeated trauma." Then, in 2002, a MSD column was included, only to be removed in 2003. Currently the log has six categories: injuries, skin disorders, respiratory disorders, poisonings, hearing loss, and "all other illnesses." Lack of specific reporting for WMSDs, the most common category of occupational disorders, makes it very difficult to evaluate trends in WMSDs at the national level.

Estimated annual workers' compensation costs for WMSDs in the United States vary between $13 and $20 billion in direct costs.[1] Estimated annual costs of "overexertion injuries" at work in the United States are now $9.8 billion, having decreased about 5% between 1998 and 2007. Estimated annual costs of repetitive motion injuries are now $2.1 billion, having decreased about 35% during the same time period.

Incidence and direct costs for workers' compensation cases of WMSDs by body area and specific conditions have been reported by Washington State (Table 16-1). Indirect costs range from two to five times direct costs. In addition to underreporting of cases in the BLS and workers' compensation data,[2,3] lost work time and reduced productivity probably continue much longer than reported in official statistics.[4] For example, when compared to workers' compensation cases for upper-extremity fracture, workers with carpal tunnel syndrome had not recovered to preclaim annual earnings even 7 years after filing a claim.[5]

Although WMSDs include a diverse group of disorders, the central concern in managing all of these disorders is early recognition and appropriate treatment. Good management of MSDs requires early access to appropriate medical treatment, evaluation of patients' job exposures, and the provision of limited or modified work duties when necessary. Comprehensive programs that integrate ergonomic improvements and medical treatment are effective in reducing the incidence and severity of WMSDs.[6]

Early recognition and treatment of MSDs are essential because they allow earlier treatment of affected workers at a time when treatment can prevent progression to a more severe condition. Workers who are treated in the early stages of disorders have better prognoses and are less likely to have prolonged disability than workers treated only after prolonged duration of symptoms. Conservative management is most effective when begun in the early stages of these

Table 16-1. Work-Related Musculoskeletal Disorders (WMSDs) of the Neck, Back, and Upper Extremity, Washington State Fund Workers' Compensation Claims, 1997–2005

Type	Incidence per 10,000 FTEs	Median Lost Workdays	Median Cost
All	258.0	42	$939
Neck	31.5	53	$950
Back	133.9	24	$834
Sciatica	5.7	260	$22,768
Upper extremity	97.9	74	$948
Shoulder	35.5	83	$1,111
Rotator cuff syndrome	17.3	142	$7,589
Elbow/forearm	16.8	64	$672
Epicondylitis	11.1	92	$1,238
Hand/wrist	49.4	69	$555
Carpal tunnel syndrome	20.4	93	$7,225
Tendonitis	15.3	80	$1,483
Knee	9.8	38	$1,569
Tendonitis/Bursitis	0.5	42	$713

Note: Lost time and costs are for compensable claims (4 or more lost work days). Costs adjusted to 2005 dollars.

FTEs, full-time equivalent employees.

Source: Silverstein B, Adams D. Work-related musculoskeletal disorders of the neck, back, and upper extremity in Washington State, state fund and self insured workers' compensation claims 1997-2005. Technical Report 40-11-2007 SHARP Program. Tumwater, WA: Washington State Department of Labor and Industries, 2007.

disorders.[7] With some disorders, such as carpal tunnel syndrome (CTS), individuals can often be treated conservatively in the early stages of disease, while surgery is often necessary when individuals present with advanced disease. However, when cases of CTS are identified early and have electrodiagnostic confirmation, surgery may result in better return-to-work outcomes.[8] Early detection is necessary to ensure that signs and symptoms of all WMSDs are recognized and treated appropriately through medical management, administrative controls, and job evaluation/and modification.

Both healthy and injured workers can potentially benefit from evaluation of their workplaces for identification of physical stressors that can be reduced or eliminated. Simple modifications can often be made to a workplace that enables the work to be done with less effort. Such modifications can prevent injury and can enable injured workers to safely return to their usual jobs more quickly. Ergonomic evaluation and intervention support successful treatment of workers for WMSDs.[9] When clinicians have more information about patients' job demands and exposures and when worksite modifications reduce physical exposures, early safe return to work is facilitated.[10] This is often the case for non-work-related musculoskeletal disorders as well as those primarily caused or exacerbated by work activities.

Many corporations and medical professionals endorse comprehensive ergonomic programs that incorporate primary prevention of MSDs through ergonomic changes in jobs, early detection of MSDs through surveillance, and early treatment of MSDs with an emphasis on early return to modified work. The American College of Occupational and Environmental Medicine (ACOEM) has released *Occupational Medicine Practice Guidelines*, which describe its recommendations for best medical practice in the diagnosis and treatment of work-related disorders.[9] These recommendations include application of ergonomic principles to job design in order to prevent MSDs, and adjustment of workstations and tools to avoid aggravation of existing disorders. Return of workers to modified work, with specific reduced physical exposures, is strongly recommended as part of treatment. Return to work is most successful when workers return to their original jobs with modifications to reduce physical exposures.

While the main focus of prevention efforts should be on primary prevention—the reduction or elimination of workplace risk factors, workers must have access to appropriate and timely medical care if they are injured. The goals of a medical management program should be to achieve the following:

- Reduce or eliminate symptoms
- Prevent progression of MSDs
- Reduce the duration and severity of functional impairment
- Prevent or reduce the severity of disability

Important elements of such a program include the following:

- Surveillance using workplace medical reports, OSHA-300 logs, annual symptom surveys, and dissemination of findings to the workplace in a timely manner
- Timely access to appropriate health care providers (Box 16-2)
- Ergonomic evaluation of the jobs of injured workers
- Availability of appropriate job modification
- Follow-up of treated workers and coordination with primary prevention efforts

Box 16-2. The Choice of a Health Care Provider for Injured Workers Is Important

Ideally, health care providers should have training or experience in ergonomics and the role of work modifications in the treatment of work-related musculoskeletal disorders. Effective diagnosis and treatment require knowledge of specific job duties. The best way for a health care provider to get knowledge of job duties is through a worksite visit. Since this is impractical in some clinical settings, information about exposures and job duties can also be obtained through a written work description, or a videotape of the job task. Employers should have a contact person with knowledge of job activities and the ability to coordinate appropriate job placement during a recovery period. Working knowledge of the industry and the specific workplace is also needed to make appropriate recommendations regarding temporary or permanent job modifications. Many employers will provide detailed information about job duties and physical exposures to the treating physician. It is difficult to provide optimal care for employees when this information is not available.

The vast majority of injured or symptomatic employees are able to return to productive work quickly, as long as their work is modified to reduce physical exposures to affected body parts. Such job modifications are frequently inexpensive and simple, and they can help employees safely return to work sooner and reduce risk of future injury. Examples of job modifications include the following:

- Training or retraining in work procedures that reduce physical exposure
- Simple job changes to prevent awkward postures, such as a step stool or tilted work surface
- Changes in tool design to reduce awkward postures and high hand forces
- Preventive maintenance to reduce force in tool/equipment use
- Changes in procedures, such as job rotation
- Use of conveyors, hoists, slides, and carts to reduce heavy lifting, pushing, pulling, and carrying

When there is no simple fix to reduce or eliminate physical exposures that are causing or exacerbating WMSDs, temporary job transfer or restrictions are important to allow workers' injuries to heal. Examples of temporary restrictions include the following:

- Reduction in pace or quantity of work
- Restriction of certain tasks
- Limitation of work hours

If an employee is to be transferred to a different job, the employer and the health care provider should assess the new job to ensure that the employee will not be exposed to physical risk factors similar to those on the job that first caused or aggravated the condition. When this cannot be accomplished, temporary removal from work will allow time for healing. In most cases, keeping an injured or symptomatic employee at work in an appropriate modified-duty position is preferable to lost work time.

Successful programs have decreased the length or severity of disability through improved early recognition and management of these disorders and integrating ergonomic interventions with medical treatment of injured workers.[10] For example, an integrated program designed for sheet-metal workers at an aircraft manufacturer combined preplacement evaluations of workers with ongoing surveillance for symptoms and signs of upper-extremity MSDs. Job modification was implemented for those with signs of early disorders, through restriction of work hours and restriction of use of vibrating hand tools. After implementation of this program for screening, surveillance, early medical evaluation, and job modification, workers' compensation costs, time lost from work, and severity of injuries all decreased.[11]

There are many other examples of reduced costs and injury rates after introduction of ergonomic or medical management interventions. Most major corporations have ergonomics programs, recognizing that such programs effectively reduce injuries. Successful approaches have most often used a combination of ergonomic principles for prevention and improved recognition and management of disorders. (See Chapter 27 for a more complete discussion of ergonomics.)

NECK AND UPPER-EXTREMITY DISORDERS

Clinical, laboratory, and epidemiological studies have contributed to the current understanding of the pathophysiology of WMSDs of the upper extremity and neck. Five workplace physical factors are important in the etiology of these disorders:

- Forceful motions
- Repetitive or prolonged duration of motion
- Static or awkward postures
- Hand-arm vibration
- Mechanical stresses

Combinations of risk factors in the same tasks increase the risk.[12–13] The effects of these physical load factors can be exacerbated by workplace psychosocial factors, such as the perception of intense workload, monotonous work, and low levels of social support at work.[12] The way in which work is organized largely determines the physical and psychosocial dimensions of the work. In assessing the role of workplace factors,

duration, frequency, and intensity of the individual and combined factors should be considered.

Physical Load Factors

Repetition and Force

Repetitive motions of the hands, wrists, shoulders, and neck commonly occur in the workplace. A data-entry operator may perform 20,000 keystrokes per hour with forearms pronated and wrists in ulnar deviation. A worker in a meat-processing plant may perform 12,000 knife cuts per day. And a worker on an assembly line may elevate her right shoulder above the level of the acromion 7,500 times per day. Such repetitive motions may eventually exceed the ability of individual muscles, tendons, and nerves to recover from the stress, especially if motions involve forceful or static contractions of muscles.

Failure to recover usually implies some type of tissue damage or dysfunction, which may represent acute inflammation that is totally reversible. In WMSDs, the sites of likely tissue damage are most commonly tendons, tendon sheaths, and tendon attachments to bones, bursae, and joints. Over time, these tissue changes may lead to nerve compression, chronic fibrous reaction in the tendon, tendon rupture, calcium deposits, or formation of fibrous nodules in a tendon.

Abrupt increases in the number of repetitive motions performed by a worker each day can cause tendonitis. New workers performing unaccustomed, forceful, or repetitive work are often at increased risk of developing MSDs.[1] Too many forceful contractions of muscles can cause corresponding tendons to stretch, compressing the microstructures of the tendons and leading to ischemia, microscopic tears in tendons, progressive lengthening, and sliding of tendon fibers through the ground substance matrix. All of these events can cause acute inflammation of tendons. High levels of exposure to the combination of repetitive and forceful movements, especially those of long duration or combined with awkward postures, are strongly associated with several MSDs of the upper extremity.[12–15] There are several aspects of repetitiveness that should be considered, including the velocity and acceleration of movement and amount of recovery time within repetitive cycles or tasks. Force also has several components, including peak force, average force, duration of exertion, and recovery time between exertions. The interaction between hand force and repetition is considered in the threshold limit value for hand activity level (HAL).[16]

Posture, Mechanical Stress, and Vibration

In addition to repetitive and forceful motions, three other exposure variables influencing the development of WMSDs are external mechanical stress, work performed in awkward or static postures, and segmental (localized) vibration.

Mechanical stress in tendons, which results from muscle contractions, is related to the force of the muscle contractions. Posture is also relevant because *(a)* muscles are more susceptible to injury at longer muscle lengths, and *(b)* in some postures, muscles and tendons must undergo more mechanical stress to exert a given amount of force on an object. For example, pinching while the wrist is flexed causes more stress on muscles and tendons than pinching while the wrist is in a neutral posture. When combined with high forces, the amount of damage is even greater.[12]

Another source of mechanical stress results from a work surface or a hand-held tool with hard, sharp edges or the ends of a short handle that press on soft tissues. The tool exerts just as much force on the hand as the hand does on the tool. These stresses can lead to *(a)* neuritis due to forceful contact between one's thumb or fingers and the edge of scissors handles; or *(b)* cubital tunnel syndrome in workers such as microscopists who must position their elbows on a hard surface for long periods. Short-handled tools, such as needle-nosed pliers, can dig into the base of the palm and compress superficial branches of the median nerve.

Work with an arm elevated more than 60 degrees from the trunk is more stressful for rotator cuff tendons than work performed with the arm at one's side. Rotator cuff tendinitis has been associated with a combination of increasing duration of shoulder extension/flexion and high hand forces, such as with pinching.[12,17] Work performed in static postures that require prolonged, low-level muscle contractions of the upper limb or trapezius muscle may also trigger chronic localized pain.

Segmental vibration is transmitted to the upper extremity from impact tools, power tools, and bench-mounted buffers and grinders. Raynaud phenomenon has been associated with several types of power tools, including chain saws, rock drillers, chipping hammers, and grinding tools. (See Chapter 12A.)

Chronic or intermittent pain originating in muscles may be a factor in the development of tension neck syndrome (costoscapular syndrome) and overuse injuries in musicians. Two types of muscle activity may contribute to the development of WMSDs: *(a)* low force with prolonged muscle contractions, such as moderate neck flexion while working at a computer for several hours without rest breaks (note the weight of the head in flexion is equivalent to a bowling ball); and *(b)* infrequent or frequent high-force muscle contractions, such as intermittent use of heavy tools in overhead work. Sustained static contractions can lead to increases in intramuscular pressure, which, in turn, may impair blood flow to muscle cells.

If damage occurs daily from work activity, muscle tissue might not be able to repair the damage as fast as it occurs, leading to chronic muscle damage or dysfunction. A causal factor in some WMSDs may be work activities that lead to sustained, relatively low-level muscle activity or higher-level muscular contractions.

Nonoccupational Factors

In addition to occupational risk factors or exposures, such as repetitive forceful work, personal risk factors may influence the risk of developing WMSDs. For example, forceful repetitive activities combined with, for example, wrist extension, can occur in some recreational activities and contribute to the development of WMSDs. Age and gender may possibly be associated with some WMSDs. For virtually all upper-extremity disorders, obesity is a significant factor.[12,18] Obesity may reduce carpal tunnel space or place heavier loads on shoulder and elbow tendons when in awkward postures. Nonoccupational factors for CTS include coexisting medical conditions, such as obesity, rheumatoid arthritis, diabetes mellitus, pregnancy, and acute trauma. Few personal factors are strong predictors of susceptibility to upper-extremity WMSDs after work organization and psychosocial and physical load factors have been considered.

Psychosocial Factors

Psychosocial factors may be important in both the initial development of WMSDs and the subsequent long-term disability that sometimes occurs. (See Chapter 14.) Few studies have rigorously investigated either psychosocial factors or the combined effects of psychosocial and physical factors.[19] The effects of psychosocial factors may operate indirectly by altering muscle tension or other physiologic processes and decreasing micropauses in muscle activity and, in turn, influencing pain perception. Psychological factors may be particularly important in determining whether specific MSDs evolve into chronic pain syndromes due to responses of the central nervous system to high job stress. Psychosocial factors appear to be somewhat more important in disorders of the neck and shoulder muscles than in tendon-related disorders of the forearm and the wrist.

Psychosocial factors are more predictive of some MSD outcomes, such as disability, than of others, such as incidence of symptoms. The risk of upper-limb disorders is increased by high structural constraints and perception of low decision latitude, and by high strain and low levels of social support at work.[12,20] Several measures have been used to define intense or stressful workloads, such as lack of control over how work is done, perceived time pressure, deadlines, work pressure, or lack of workload variability.[19]

Studies that have addressed psychosocial factors have often used the demand-control-support model originally introduced by Robert Karasek and Töres Theorell.[21] In this model, high levels of psychological job demands may contribute to the development of WMSDs when they occur in an occupational setting in which workers have *(a)* little ability to decide what to do or how to do a particular job task, and *(b)* little opportunity to use or develop job skills. Further, these adverse effects are hypothesized to occur more frequently in a work environment in which there is little social support from co-workers or supervisors. Low job satisfaction has not been consistently identified as an important risk factor for WMSDs.

Diagnosis

This broad group of work-related disorders of the neck and upper extremity has a diverse set of symptoms and physical findings. The evaluation of a patient for a suspected work-related disorder should have three major components: *(a)* a history of present illness, *(b)* a physical examination of the upper extremity and the neck, and *(c)* assessment of the work setting and tasks.[22]

The history of present illness should fully characterize the symptoms by determining the location, radiation, duration, evolution, time patterns, and exacerbating factors. The worker's description of work activities is useful. The worker should be asked to describe the nature of specific work tasks by risk factors (forceful exertions, repetitive activities, and other adverse exposures). For example, a worker who for 8 hours a day uses a vibrating jackhammer to perform a task that is repeated every 30 seconds may be at high risk for CTS. Similarly, a repetitive job that requires the arms to be held overhead during most of the work shift may increase the risk of a rotator cuff tendonitis. Because specific job tasks can vary within even a high-risk occupation, a careful history of specific job tasks should be obtained.

When a worker who has been performing the same job for a considerable period develops an MSD, the history should be directed not only at the chronic stable exposures but also at acute factors, such as changes in work tasks, tools, materials, or work pace or duration (more overtime, longer workdays, or fewer days off—with less time for recovery from fatigue and occult injury).

Determining whether the individual has a predisposing medical condition, such as previous injury to the symptomatic area, is also important. Nonoccupational exposure to risk factors can be a potential confounding influence and should be elicited when the worker is interviewed. However, the cause of MSDs is frequently multifactorial, and the presence of nonoccupational risk factors does not negate the importance of coexisting occupational exposures.

Surveillance and epidemiological studies have identified a number of industries and occupations associated with risks of CTS or other upper-extremity disorders. Awareness of these findings can alert physicians to the industries and occupations in which adverse exposures are more common. Table 16-2 provides examples from Washington State workers' compensation data of common occupations and industries with more than 2.5 times the expected rate for WMSDs based on all industries. It is likely that there are also "high-risk" jobs in "low-risk" industries.

The physical examination is an important part of evaluation of patients with WMSDs. An examination of the upper extremity typically involves inspection, assessment of the range of motion, strength, palpation, and evaluation of peripheral nerve function.

One of the main objectives of the physical examination is to precisely determine the structures in the upper extremities that are the anatomic source of symptoms. Numbness and paresthesias often result from peripheral nerve compression, but there are many other reasons why there might be numbness and tingling in the fingers. Increased pain on resisted maneuvers often results from lesions in a tendon or at its insertion. In some cases, it is not possible to determine the precise source of pain in the upper extremity; in others, it is possible to determine the specific disorder that is present. The severity of these disorders ranges from very mild, with no significant impairment of the ability to work, to very severe. Guidelines have been published to establish standardized methods for diagnosis, especially for epidemiological studies but also for clinicians.[22–25]

In addition to the disorders with specific findings on physical examination, workers in certain occupations, such as keyboard operators, musicians, and newspaper reporters, often have an increased rate of complaints of pain in the upper extremity or neck. These symptoms are similar to those of low back pain because a specific anatomic source of the pain often cannot readily be identified on clinical evaluation. As with low back pain, these types of pain are common, often intermittent, and sometimes lead to substantial disability and impairment.

Diagnosis of a WMSD is based on a three-step process:

1. *Determination of whether the individual has a specific disorder*, such as flexor tendonitis

Table 16-2. Most Frequent Occupations in High-Risk Industries for Compensable Work-Related Musculoskeletal Disorder (WMSD) Claims in Washington State

Industries	Occupations
Forest nurseries and forest product gathering	Nursery workers
	Laborers/farmworkers
	Production inspecting/packing
	Floral design
Masonry, stonework, tile, plastering	Drywall installers
	Insulation installers
	Brick masons
Roofing	Roofers
	Carpenters
	Laborers
Meat products	Butchers and meatcutters
	Laborers and freight stocking/handling
	Hand packers
Dairy products	Laborers and freight handlers/stockers
	Truck drivers
	Hand packers
Sawmills	Lumber handlers
Millwork	Laborers
	Woodworking machine operators
	Assemblers
	Cabinetmakers
Iron and steel foundries	Mold and core workers
	Furnace/oven workers
	Grind/polish machine operators
	Laborers
	Machine operators
Heating, ventilation, and air conditioning	Welders/cutters
	Assemblers/fabricators
	Laborers
	Grinding/polishing machine operators
Nursing and personal care facilities	Nursing aides and orderlies
	Health aides
	Licensed practical nurses and registered nurses
	Maids/housekeeping workers
Local and suburban passenger transport	Emergency medical technicians
	Bus drivers
	Physician assistants/registered nurses
	Mechanics
	Taxi/drivers
Trucking and courier services	Truck drivers
	Freight handlers/stockers
	Refuse and recyclable collectors
	Grader/sorters
Air transportation, scheduled and air courier services	Freight/stock handlers
	Flight attendants
	Couriers/messengers
	Transport/ticket/reservations workers
	Mechanics
Examples of high-risk occupations that cross over most industries	Housekeeping/janitorial workers
	Data-entry operators
	Stockers/receivers
	Assembly and packaging workers
Dairy product manufacturing	Laborers and freight stockers
	Production workers
	Truck drivers (heavy and light)
	Packagers and package handlers

(*Continued*)

Table 16-2. Most Frequent Occupations in High-Risk Industries for Compensable Work-Related Musculoskeletal Disorder (WMSD) Claims in Washington State (Continued)

Industries	Occupations
Waste collection	Refuse and recycled materials collectors
	Truck drivers (heavy and light)
	Laborers
	Bus and truck mechanics
	Welders and cutters
Nursing care facilities	Nursing aides and orderlies

Source: Silverstein B, Kalat J, Fan ZJ. Work-related musculoskeletal disorders of the neck, back, and upper extremity in Washington State, state fund and self insured workers' compensation claims, 1993–2001. Tumwater, WA: Washington State Department of Labor and Industries, 2003.

of the forearm. This is usually based on the history and physical examination.

2. *Obtaining evidence from a detailed occupational history*, or—better yet—from *(a)* direct observation of the workplace or representative videotapes of substantial exposure to specific occupational risk factors, and *(b)* review of detailed job descriptions and job safety analyses. Although direct observation of the workplace is often required to determine more precisely the level of exposure to risk factors in specific job tasks, descriptions by workers may identify high-risk exposures with sufficient accuracy to make a correct diagnosis. Analysis of health surveillance data, such as OSHA logs or workers' compensation records for the specific workplace, may be particularly helpful in confirming that a particular job is associated with an increased risk of a WMSD. To facilitate return-to-work evaluations, some employers now provide health care providers with a videotape or DVD of the job that the worker normally performs. This may be useful in determining the approximate level of exposures. Table 16-3 provides an illustrative list of exposures of concern ("caution zone jobs") identified by Washington State as a guideline for implementing ergonomics activities, including raising employee awareness.[26] This is not an exhaustive list of exposures of concern, but it does provide a practical guide for frequently observed exposures in many workplaces.
3. *Consideration of nonoccupational causes as possible primary causal factors or extenuating factors*, based on the history and physical examination. Review and analysis of surveillance and epidemiologic data of similar work may provide information on the relative contributions of occupational and nonoccupational factors in causing a specific WMSD in the worker's occupation and industry. With the exception of tests for abnormalities in nerve conduction, elaborate diagnostic or laboratory studies are often not necessary, unless the worker *(a)* has a history of trauma, *(b)* has symptoms suggestive of underlying systemic disease, or *(c)* fails to improve with conservative treatment.

The most difficult part of the diagnosis of WMSDs is determination of the relative contribution of occupational factors in causation. The critical question is: Was the exposure of sufficient intensity, frequency, and duration to have caused or aggravated the condition? Because intense periods of high exposure as short as a few days can cause lateral epicondylitis or other WMSDs, one should estimate the intensity and frequency of exposure. It is not uncommon for there to be simultaneous exposure to multiple risk factors, such as repetitive and forceful exertions of the hands, shoulder abduction, and vibration from hand tools. There are no simple rules for assessing whether exposure has been of sufficient intensity, frequency, and duration to cause a specific disorder in a specific person.

Table 16-3. Caution Zone Risk Factors, Washington State Ergonomics Rule/Guideline, 2000

Movements or postures that are a regular and foreseeable part of the job, occurring more than 1 day per week and more frequently than 1 week per year	
Awkward postures	Working with the hand(s) above the head, or the elbow(s) above the shoulders, more than 2 hours total per day
	Working with the neck or back bent more than 30 degrees (without support and without the ability to vary posture) more than 2 hours total per day
	Squatting more than 2 hours total per day
	Kneeling more than 2 hours total per day
High hand forces	Pinching an unsupported object(s) weighing 2 or more pounds per hand, or pinching with a force of 4 or more pounds per hand, more than 2 hours per day (comparable to pinching half a ream of paper)
	Gripping an unsupported objects(s) weighing 10 or more pounds per hand, or gripping with a force of 10 or more pounds per hand, more than 2 hours total per day (comparable to clamping light-duty automotive jumper cables onto a battery)
Highly repetitive motions	Repeating the same motion with the neck, shoulders, elbows, wrists, or hands (excluding keying activities) with little or no variation every few seconds, more than 2 hours total per day
	Performing intensive keying more than 4 hours total per day
Repeated impacts	Using the hand (heel/base of palm) or knee as a hammer more than 10 times per hour, more than 2 hours total per day
Frequent, awkward, or heavy lifting	Lifting objects weighing more than 75 pounds once per day or more than 55 pounds more than 10 times per day
	Lifting objects weighing more than 10 pounds if done more than twice per minute, more than 2 hours total per day
	Lifting objects weighing more than 25 pounds above the shoulders, below the knees, or at arms length more than 25 times per day
Moderate to high hand-arm vibration	Using impact wrenches, carpet strippers, chain saws, percussive tools (jack hammers, scalers, riveting or chipping hammers) or other tools that typically have high vibration levels, more than 30 minutes total per day
	Using grinders, sanders, jigsaws, or other hand tools that typically have moderate vibration levels more than 2 hours total per day

Neck Disorders

Nonradiating neck pain is often called "tension neck syndrome," suggesting muscular origin. Nonradicular radiating neck pain is often reported by patients with neck-shoulder pain. It is important to distinguish this pain from cervical osteoarthritis or cervical nerve root compression. Pain in the upper extremity on active or passive cervical rotation is often observed in nonradicular radiating pain.[23]

Neck disorders of nontraumatic origin are frequent and involve primarily muscles in the neck-shoulder region. According to workers' compensation claims data, annual incidence of neck disorders is 31.5 per 10,000 full-time equivalent employees (FTEs) (Table 16-1). Most of these involve nonspecific neck pain. Many studies of neck pain also include the neck/shoulder region, primarily due to upper trapezius pain. (In some languages, neck and shoulder are not differentiated.) The annual incidence rate of neck pain lasting more than 1 week in office environments is about 34%, and of radiating neck pain about 14%. Table 16-4 summarizes risk factors for neck and neck/shoulder disorders. Among office workers, women report neck pain about six times as frequently as men. The combination of high mental stress and limited physical exercise increases risk about six-fold. Several work factors have been associated with lost workdays (sick leave) due to neck pain, including jobs that involve prolonged flexion and rotation of the neck and jobs that involve a limited role in decision making.[27]

Among nurses, increased risk of neck/shoulder pain occurs with patient-handling tasks involving pushing/pulling and reaching. When neck/shoulder complaints are combined with pressure tenderness, prevalence is about 7% and the annual incidence rate about 2%. Workers who perform highly repetitive shoulder work (16 to 40 movements per minute) and/or forceful work

Table 16-4. Risk Factors for Nontraumatic Neck and Neck/Shoulder Disorders

Individual factors	Age
	Female gender (may be a function of gender segregation)
	Little physical exercise
Physical work factors	Prolonged seated work
	Neck flexion, rotation
	Prolonged shoulder shrugging
	Repetitive shoulder or hand work
	Inappropriate keyboard location
Psychosocial factors	Low decision latitude
	High demands
	High mental stress
Jobs with high-risk activities	Dental workers
	Microscopists
	Video display terminal workers
	Surgeons
	Nurses/assistants
	Electronics assemblers

have two to four times the risk.[17,20] Prolonged neck flexion and lack of recovery time from highly repetitive work also increase risk. Perceived job demands almost double the risk. Those experiencing a recent increase in exposure (prolonged work using monitors, keyboards, and mice, or work above the shoulder) are more likely to seek health care than those who have been exposed long-term, suggesting a short induction time.

Shoulder Disorders

Rotator cuff tendonitis is one of the most frequent and costly upper-extremity disorders associated with work activities. In Washington State during the 1998–2007 period, the average cost of a workers' compensation compensable claim for rotator cuff tendonitis was $35,000, largely due to extensive lost work time and frequent surgery. The rotator cuff is made up of four interrelated muscles arising from the scapula and attaching to the tuberosities that allow the humeral head to rotate: The supraspinatus stabilizes and abducts the arm, and the infraspinatus, teres minor, and subscapularis stabilize and externally rotate the head of the humerus. The long head of the biceps muscle stabilizes and flexes the humeral head and the elbow. The supraspinatus is most active in the initial phase of abduction, whereas the deltoid is more active higher in the arc, but both are required for full power. Above 90 degrees, the rotator cuff force decreases, making the joint more susceptible to injury. Usually rotator cuff disease initially occurs after intensive activity of the shoulder, followed by remission with rest or treatment. Symptoms can become constant, especially with activities that are overhead and require arm strength. Slow onset of localized pain that increases with activity suggests rotator cuff tendonitis, especially when pain is above or lateral to the shoulder. In contrast, sudden onset of pain suggests traumatic fracture, dislocation, or rotator cuff tear.

Table 16-5 summarizes risk factors for shoulder disorders. Rotator cuff disease is more common after age 40 (with onset generally around age 55) and in men. Repetitive overhead activities and sports predispose to rotator cuff tendonitis. In working populations, repetitive, prolonged, and forceful shoulder work increases the risk of shoulder tendonitis three-fold.[27–29]

Table 16-5. Risk Factors for Nontraumatic Shoulder Disorders

Individual factors	Age
	Obesity
	Male gender
	Lack of physical exercise
Physical work factors	Repetitive shoulder work
	Repetitive hand work with tools
	High hand force
	Working above shoulder height
	Working in a bent posture
	Physically strenuous work
	Shoulder angle greater than 45 degrees static or repetitively
Psychosocial factors	Low decision latitude
	Monotonous work
	Mental stress
	High job demands
	Depression
Jobs with high-risk activities	Truck drivers
	Carpenters
	Welders
	Drywall installers
	Meat packers
	Assembly workers
	Masons
	Nursing assistants
	Freight handlers
	Garbage collectors

Half of individuals with shoulder tendonitis due to repetitive work recover within 10 months, but recovery is slowed with increasing age. Newly employed workers are at increased risk of shoulder pain if they are lifting heavy weights, lifting with one hand, lifting above shoulder height, or pushing or pulling heavy loads. There is some indication that monotonous work and depression are independent risk factors, but not as important risk factors as repetitive use of tools or low decision latitude.[30] The 1-year incidence rate of rotator cuff in symptomatic computer users has been reported as about 2%.

Bicipital tendonitis presents with pain in the anterior shoulder, occasionally radiating down to the elbow. It is aggravated by activities requiring shoulder flexion, forearm supination, or elbow flexion with forceful exertions. In the early stages, pain is worst at onset and completion of the activity, gradually becoming constant. On physical examination, pain in the bicipital groove is exacerbated with resisted arm flexion with a supinated forearm and full elbow extension, or on resisted supination. It is less frequently reported than rotator cuff tendonitis.

Elbow and Forearm Disorders

Epicondylitis is characterized by pain at muscle–tendon junctions or insertion points of forearm flexor (medial) or extensor (lateral) tendons. Pain is usually localized around the epicondyle, but it may radiate distally to the forearm. Lateral epicondylitis (tennis elbow) is more frequently reported than medial epicondylitis (golfer's elbow)—five times more frequently in the Washington State workers' compensation data. Lateral epicondylitis is a result of inflammation at the muscular origin of the forearm extensors, primarily the extensor carpi radialis brevis, leading to micro tears with subsequent fibrosis. Medial epicondylitis involves primarily the flexor/pronator muscles at their origin on the anterior medial epicondyle; less often it affects other flexor tendons. Concurrent compression of the ulnar nerve in or around the medial epicondyle groove has been estimated to occur in half of the cases. Epicondylitis can occur in data-entry operators and industrial workers, primarily in those using forceful twisting motions, such as in using screwdrivers. Force increases risk. However, the longer the duration of keyboarding combined with unsupported awkward postures, the greater the risk. Frequent "microbreaks" combined with varying tasks that use different muscle groups, can usually reduce the severity and incidence of epicondylitis.

Repetitive forceful stress at the musculotendinous junction and its origin at the epicondyle cause an acute tendonitis, and tendinosis in its more chronic form, due to failure of the tendon to heal. Peak incidence occurs in people 20 to 49 years old; males account for two-thirds of the cases. Onset can accompany an acute injury, but more commonly it is associated with repetitive use of the extensor/supinator or flexor/pronator muscles. Work activities, such as using a screwdriver or hammer, increase risk. The frequency of forceful exertions as well as the combination of supination and lifting increase risk above either alone, when controlling for personal factors; social support reduces risk.[31] In repetitive work environments, incidence of lateral epicondylitis is approximately 12%. It increases with age, number of other upper limb diagnoses, and "turn-and-screw" motions.[31–32] In a working population, prevalence of medial epicondylitis is about 5% and its annual incidence rate is about 1.5%. Forceful work increases risk. Medial epicondylitis is often found with other upper-limb disorders in working populations. Approximately 80% of patients recover within 3 years. Table 16-6 summarizes risk factors for elbow and forearm disorders.

Diagnostic criteria include intermittent to continuous pain in the epicondylar area, pain on resisted wrist extension (lateral), or resisted pronation (medial). Symptoms often last up to 1 year, irrespective of treatment. They are exacerbated by forceful gripping activities. Poor prognoses are associated with intensive manual work and high baseline pain. Several studies have reported elbow/forearm pain in occupational computer users.

Hand and Wrist Disorders

The most frequent hand and wrist diagnoses are tendonitis and CTS (Fig. 16-2). The incidence of workers' compensation claims for all nontraumatic hand and wrist disorders in the United States is 57 per 10,000 FTEs, with an average cost

Table 16-6. Risk Factors for Nontraumatic Elbow/Forearm Disorders

Individual factors	Age
	Other work-related musculoskeletal disorders
Physical work factors	Driving screws
	Tightening with force
Psychosocial factors	Low discretion
	High demands
	High mental stress
Jobs with high-risk activities	Carpenters
	Machinists
	Laborers
	Plumbers
	Assembly work with hand tools
	Hairdressers
	Drywall installers
	Hand packers
	Electricians
	Bus drivers
	Welders
	Grinders/polishers
	Butchers/meatcutters
	Kitchen/food preparation

Figure 16-2. Woman in Nicaragua cutting meat. A high degree of hand force and frequent repetition combine to make this a high-risk job for development of carpal tunnel syndrome and tendonitis. In addition, this woman faces the potential hazards of cuts and neck strain. (Photograph by Barbara Silverstein.)

of almost $12,000. The claims incidence rate for tendonitis is 16 per 10,000 FTEs, with an average cost of about $15,600 and an average of 274 (median 81) lost days from work . (See Table 16-1 for data from Washington State.) Table 16-7 summarizes the risk factors for CTS and tendonitis.

Carpal Tunnel Syndrome

Carpal tunnel syndrome, which is characterized by pain, paresthesias, and/or weakness in the median nerve distribution of the hand, is due to entrapment of the median nerve in the carpal tunnel at the wrist. It is diagnosed by history of numbness and tingling in the median nerve distribution of the hand along with electrodiagnostic testing. Criteria for diagnosis include symptoms in the median nerve distribution of the hand and a positive electrodiagnostic test. Further information can be found at: http://www.lni.wa.gov/ClaimsIns/Providers/ProviderIndex/default.asp.

Table 16-7. Risk Factors for Nontraumatic Carpal Tunnel Syndrome, Tendonitis

Individual factors	Age
	Obesity
	Female gender
	Pregnancy
	Rheumatoid arthritis, diabetes, hypothyroidism, hypertension
Physical work factors	High-force, highly repetitive work, hand-arm vibration
	Repetitive pinching, tightening, or holding with force
	Repetitive hitting
Psychosocial factors	Low discretion
	Low job satisfaction
	High demands
	Poor social support
	High mental stress
Jobs with high-risk activities	Meat cutters
	Lumber turners
	Food processors
	Carpenters
	Assembly work with hand tools
	Foundry workers
	Hairdressers
	Kitchen workers
	Laborers
	Machine operators
	Sewing operators
	Hand packers
	Typists
	Stock handlers/baggers
	Roofers

Personal risk factors include diabetes mellitus, hypothyroidism, obesity, rheumatoid arthritis, older age, and female gender. In work-related CTS, the gender difference may be due to a greater proportion of women in more repetitive work and a greater proportion of men in more forceful work—and a greater willingness of women to report symptoms.[34] Work-related physical factors include highly repetitive or forceful hand work (especially pinching), sustained awkward wrist postures, and hand-arm vibration. The more these factors occur simultaneously, the greater the risk.[18,34] Although frequently reported in the news media, CTS is not primarily associated with computer work. Among computer users, the annual CTS incidence rate is approximately 0.9%, compared to an annual incidence rate of 14.7% for extensor tendonitis in the first dorsal compartment.[35] Meatpackers, assembly-line workers, and other workers with high-force and high-repetition tasks appear to be at much higher risk for CTS than computer users. Workers who perform repetitive forceful work have a high percentage of CTS. For example, the prevalence of CTS in the clothing, food, and assembly sectors in France ranges from 11% to 17%. Carpal tunnel syndrome has been associated with hand-arm vibration, but it is difficult to separate vibration from high hand force. When workers are exposed to high force and high repetition simultaneously, risk of CTS increases dramatically.

Carpal tunnel syndrome is characterized by pain, numbness, and tingling in the median nerve distribution. Electrodiagnostic (nerve conduction) tests are typically used to confirm the diagnosis; however, some definitions of CTS cases include only symptoms and physical examination maneuvers. The case in Box 16-3 illustrates the intermittent and progressive nature of most work-related disorders of the upper extremity, especially CTS—the best known of these disorders.

Ulnar nerve entrapment at the wrist (Guyon canal), which usually presents as a motor lesion, is much less frequently reported than median nerve entrapment in the carpal tunnel. Cubital tunnel syndrome (frequently called student's elbow or "Saturday night palsy") results from compression of the ulnar nerve due to prolonged weight bearing on the elbow. Radial nerve entrapment, which is less common than ulnar nerve entrapment, may be related to repetitive upper-arm activities requiring gripping and squeezing.

Hand and Wrist Tendonitis/Tenosynovitis

Tendonitis causes pain over the tendon close to where it is inserted in the muscle, and it can cause mild swelling over the tendon. Tendonitis worsens with repetitive forceful motion. The highest risk of hand and wrist tendonitis is associated with a combination of high hand force and high hand repetition. There are many types of tendonitis associated with the numerous different tendons in the hand and wrist. DeQuervain's tendonitis, the most common type, presents with a history of repetitive pinching and pain along the radial aspect of the wrist below the base of the thumb (elicited with the Finkelstein test—passive ulnar deviation with the thumb inside a closed fist). DeQuervain's tendonitis has an annual incidence of 15% among intensive computer users. DeQuervain's tendonitis, as is true for most other forms of tendonitis, worsens with activity and improves with rest. "Trigger finger" (volar flexor tenosynovitis) presents with tenderness at the proximal end of the tendon sheath, in the distal palm, and with a catching of the tendon when the finger is flexed. There is frequently palpable tendon thickening and nodularity.

Treatment and Prognosis

The goals of treatment are elimination or reduction in symptoms and impairment, and return to work under conditions that will protect health. These goals can be most easily achieved by early and conservative treatment. Early treatment of WMSDs is less difficult and less costly, often reduces the need for surgical procedures, decreases absence from work, shortens stressful exposures, and increases effectiveness of treatment. Early interaction among the health care provider, the worker, and the employer facilitates safe and successful return to work. This ergonomics approach has been very successful for workers' compensation claimants for back and upper-limb problems.[6–8]

The initial goals of treatment are to limit tissue damage, dysfunction, and inflammation and to assist the repair of tissue damage. Symptoms can

Box 16-3. Carpal Tunnel Syndrome Case

A 31-year-old, right-handed man had been employed in a variety of automobile manufacturing jobs for 13 years. Two years ago, he switched to a new plant and was assigned to a job that required him to manipulate a spot-welding machine beneath cars moving overhead. He completed four welds/minute on each car. The metal handles of the spot welder required substantial force for appropriate positioning, and they were manually repositioned four times per car. The worker's wrists were in extreme extension for a substantial portion of the job cycle.

When the worker started on this job, the work shift was 9 hours for 6 days per week. After 3 weeks on the job, he noted that he had pain in both wrists, numbness and tingling in the first four fingers of his left hand, at first only at night, a few nights each week, after he had fallen asleep. When he awoke at night with the numbness, it was alleviated by shaking his hands. Gradually, over the next several months, the numbness and pain worsened in both frequency and intensity. His left hand would feel numb by the end of the work shift, and any time he was driving his hands would become numb. Because he liked his job and did not want to be placed on restriction, which would mean he could not work overtime, he decided to visit his private physician rather than the company physician. He also was not sure that the company physician would be very sympathetic to his complaints.

The physician found on physical examination that the worker had decreased sensitivity to light touch in the left index and middle fingers and a positive wrist flexion-nerve compression test of the left hand. She suspected carpal tunnel syndrome (CTS) and believed that the disorder might be work-related because the patient was young, male, and had no other risk factors, such as diabetes, past history of wrist fracture, or recent trauma to the wrist. The physician discussed job changes with the patient. She also prescribed wrist splints to be used at night.

The splints relieved some of the nighttime numbness for a period. However, over the next 6 months, the symptoms became present most of the time, and he thought that his left hand was becoming weaker. Similar symptoms also developed in his right hand.

The patient felt he could no longer do his job and returned to his physician, who ordered nerve conduction tests that showed slowing of median sensory nerve impulse conduction in the carpal tunnel, more so on the right than the left. She referred him to a hand surgeon.

One year after the problem was first noted, the worker had surgery, first on the left hand and then on the right. After surgery, the company placed him in a transitional work center for a 3-month period, where he worked at his own pace and had no symptoms. He then returned to the assembly line, with the restriction that he not use welding guns or air-powered hand tools. When he worked on the line, he occasionally had symptoms, but they were substantially less intense and less frequent than before.

He later transferred to a warehouse, because he felt that he would have a better chance of avoiding long layoffs there. His job required use of a stapling gun to seal packages. Three weeks after beginning this job, his symptoms began to return with their former intensity. Through ordinary channels, he immediately sought and was given a transfer to a position driving a forklift truck. This change reduced, but did not eliminate, his symptoms. Currently, he has numbness, tingling, and pain in the fingers of both hands about twice a month. Playing volleyball usually triggers a severe attack. With the use of nighttime splints, he can sleep through most nights without awakening. Although he believes that his hands are weaker than before the symptoms developed, he is still able to perform his job. He has decided that he will continue working as long as the symptoms remain at no more than the present level.

be relieved with anti-inflammatory medications, rest (sometimes facilitated by night splints), and application of heat or cold. Physical therapy techniques, such as stretching exercises, are used to help relieve symptoms, ensure normal joint motion, and recondition muscles after periods of rest or reduced use. If these more conservative measures fail to reduce symptoms and impairment, steroid injections or surgical treatments may be helpful. Surgery, even in CTS, may be ineffective if the worker is returned to the former job without eliminating or reducing the WMSD hazards that were present. Because few scientifically valid studies have evaluated the long-term effectiveness of the treatment of WMSDs of the limb and neck, an empiric approach is indicated. One year after carpal tunnel release surgery, distal sensory latencies remain abnormal in about 80% of patients.

Resting of the symptomatic part of the upper extremity is the most important part of treatment. In addition to engineering changes, restricted duty, job rotation, or temporary transfer may be effective. For job rotation or temporary transfer to be effective, the new job duties must result in a net reduction in level of exposure. It is often necessary to conduct an evaluation of the new duties to determine whether a reduction in exposure will occur. The magnitude of reduction required to facilitate recovery often is not known. In general, the more severe the disorder, the greater the reduction in

magnitude and duration of exposure that will be required. Workers should be removed from the workplace only in severe cases or after less drastic measures have failed to be effective.

Splints and other immobilization devices may provide rest to the symptomatic region of the body. However, they may increase the level of exposure if workers must resist devices to perform regular job tasks. Workers may also adapt to wearing a splint by altering their work activities in a way that leads to substantial stress on another part of the upper extremity, such as the elbow or shoulder. Immobilization or prolonged rest may have direct adverse effects if either leads to muscle atrophy. As a result, careful monitoring is indicated for workers on restricted duty, temporarily transferred to another job, or wearing immobilization devices. In addition, because it is difficult to predict the clinical course of these conditions and because the empiric basis of many treatments is poorly understood, frequent follow-up is desirable. Failure of the treatment to produce improvement over several weeks should lead to thorough reevaluation of the treatment plan and its underlying assumptions.

Many of these conditions resolve within a few weeks with early treatment. The prognosis is generally good with early treatment and reduction in exposure. Results of randomized clinical trials suggest that some people with moderate CTS do better with surgery than splinting (within at least the first 12 months after surgery).[36]

Sometimes CTS and upper-extremity WMSDs follow a course similar to that of chronic severe low back pain. With conservative treatment and appropriate adjustments in the work setting, most individuals improve enough so they can successfully return to work, but a few develop chronic symptoms that are very difficult to treat. In these cases, the physical capabilities of the worker, the work demands, and the psychosocial factors related both to the worker and the employer are important in determining whether the worker successfully returns to work.[7] The ways in which these factors interact are complex. The recognition of psychosocial factors—such as job satisfaction, supervisory and peer support, and negative self-fulfilling beliefs of the worker, the employer, or the health care provider—is important and should not lead to ignoring the role of occupational physical exposures or to "blaming the victim."[35] When the latter occurs, delayed recovery is often attributed to personal weakness, low job satisfaction, or desire for secondary gain. Critical to prevention of these persistent cases is early intervention—an important reason to promote early reporting of symptoms.

Comprehensive programs to address physical reconditioning of workers, psychosocial factors, and workplace factors, such as ongoing exposure, have rapidly developed.[37] A contract between the worker and the health care provider should be established early, explicitly aiming to return the worker safely to work. Diagnosis and treatment of severe or chronic WMSDs can be challenging. Determining level of exposure by history is difficult; usually direct observation of work is preferred or necessary. There is much uncertainty about how to best measure exposure in some workplaces, especially offices. Determining work-relatedness is challenging, with danger of overdiagnosis or underdiagnosis. It is also challenging to recognize when a case is becoming chronic and severe, and when a multidisciplinary approach needs to be considered.

In diagnosing and treating these work-related conditions, carefully obtaining histories and performing physical examinations are important. Extensive objective assessment of the workplace may be necessary. In most cases, a reasonable and often effective approach involves conservative treatment that *(a)* preserves normal physical conditioning, *(b)* relies on reducing exposure while the worker remains at work, and *(c)* incorporates careful monitoring of the worker. Prevention of these disorders requires identification and remediation of adverse exposures.

LOW BACK PAIN

Low back pain is among the most common health complaints among working-age populations worldwide, ranking second only to respiratory illnesses as a symptom-related reason for physician visits. In the United States and other developed countries, about 70% to 80% of adults will experience a significant episode of low back pain at least once in their lives. More than 22 million cases of back pain that last 1 week or

more occur annually in the United States, resulting in almost 150 million lost workdays.[38]

Low back pain is a major cause of disability, limitation of activity, and economic loss in developed countries. Disability due to low back pain is a complicated phenomenon influenced by the physical condition of the affected person, and other personal and societal factors, including medical care, the work environment, and the workers' compensation system. In the United States between 1997 and 2005, there was a 40% decrease in the number of low back pain claims for workers' compensation and an even sharper decrease in workers' compensation payments for low back pain. There has also been a decline over the past decade in reported back and spinal injuries associated with lost workdays.

Nevertheless, low back pain still accounts for a substantial burden of cost and disability. At any given time in the United States, back pain accounts for about 1% of the working-age population being permanently disabled and up to 1% being temporarily disabled. Back disorders remain the most frequent category of chronic conditions causing activity limitation in people under age 45 in the United States. Back pain is the most common reason for filing workers' compensation claims, accounting for 14% to 25% of all claims and 23% to 33% of all workers' compensation claim costs—estimated at more than $9 billion per year. The estimated total economic impact of low back pain in the United States, including lost earnings and other uncompensated losses is $75 to $100 billion.[1] Estimates of the average workers' compensation costs of low back disorders and sciatica are shown in Table 16-1.

While there is widespread agreement about the severity and widespread nature of low back pain, there is much less agreement concerning its etiology or even its definition. There are many clinical definitions of back pain and different ways in which patients can be identified, such as by symptoms, medical treatment, or disability. Most people with low back pain do not see a health care provider. Most cases of low back pain that are seen by a health care provider do not result in a change in work status. Most alterations of work status due to low back pain do not lead to long-term disability. Very different pictures of low back pain may thus emerge from differing case definitions. In a given individual, onset, severity, reporting, and prognosis of low back pain may be influenced by many work and nonwork factors. The presence of personal risk factors in a patient does not rule out work-relatedness, just as work may not be the only cause of an individual's symptoms.

Etiology

Low back pain is associated with work-related lifting and forceful movements, whole-body vibration, heavy physical work, and work in awkward postures (bending and twisting).[37,39,40] Examples of jobs with these risk factors are listed in Table 16-8. Psychosocial factors, such as job satisfaction, personality traits, perception of intensified workload, and job control, are associated with low back pain.

Workplace factors include frequent bending and twisting, heavy physical labor, and prolonged sedentary work. Jobs requiring frequent lifting of objects weighing 25 pounds or more seem to be associated with an increase in risk, as are sudden, unexpected maximal lifting efforts. The effect of lifting may be modified by individual fitness and strength capability and by the rate, position, distance, and height of the lifting task. Other than the weight of the object lifted, the single greatest factor affecting the mechanical strain of lifting is the distance of the object to the spine. The exposure to vibration that accompanies motor vehicle operation (4 to 6 Hz) is also a risk factor for low back pain. Truck drivers, manual material handlers, and nursing personnel have high rates of compensable back pain episodes.

The frequency and severity of low back pain are also associated with many personal and lifestyle factors, including age, gender, physical

Table 16-8. Jobs with High-Risk Activities for Sciatica, Washington State Fund Workers' Compensation Claims, 1993–2001

Nursing aides/orderlies	Nurses
Truck drivers	Construction laborers
Carpenters and apprentices	Garbage collectors
Maids and housekeeping cleaners	Glaziers
Drywall installers	Freight/stock handlers
Carpet installers	Brick masons

fitness, lumbar mobility and strength, tobacco use, nonwork physical activities, past history of low back disorders, and congenital structural abnormalities, such as spondylolisthesis.[41,42]

Diagnosis and Evaluation

Low back pain may arise from *(a)* any of the structures comprising the lumbosacral spine and its associated soft tissues, or *(b)* abdominal, retroperitoneal, or pelvic structures. It may result from local or systemic processes. Even with clinical tests and imaging procedures, however, the causes of most episodes of low back pain remain unclear and perhaps as many as 85% of patients cannot be given a precise pathoanatomical diagnosis. Pain in these cases is typically assumed to be related to soft tissue injury or to degenerative changes. Nonspecific terms, such as sprain or strain, are commonly used to describe the etiology of low back pain. Given the idiopathic nature of most episodes of low back pain, primary goals of evaluation are to identify any of the following:

- Systemic or visceral cause of pain
- Neurologic compromise requiring urgent surgery
- Other findings that influence the choice of therapy or prognosis, including workplace exposures that may incite or exacerbate symptoms

A limited diagnostic evaluation, combined with strong reassurance regarding prognosis and careful attention to the patient's concerns, best serves the needs of most patients. In cases of work-related back pain, one must identify work exposures that may need modification to improve functional recovery or prevent recurrence.

Evaluation should focus on these questions:

1. Is the pain caused by a systemic disease?[43]
2. Is there neurologic compromise that may require evaluation by a surgeon?[43]
3. Is there social or psychological distress that may amplify or prolong the pain?[43]
4. Is it related to work?
5. What is the patient's fitness or capacity for work?

The most important immediate goal of obtaining a history is to determine whether the patient has pain related to a serious local condition, such as a fracture; a systemic disorder, such as a malignancy or infection; or a neurologic disorder requiring evaluation by a surgeon, such as cauda equina syndrome. The history should focus on "red flags" that increase the likelihood of a more serious disorder than nonspecific low back pain:

- History of trauma
- Age over 50 or under 20
- History of malignancy or immune compromise
- Pain that worsens in the supine position
- Recent onset bowel or bladder dysfunction or saddle anesthesia
- Severe or progressive neurologic deficit of the lower extremities[41]

Most cases present with nonspecific low back pain or with symptoms of sciatica. The history should include questions on the following:

1. Previous low back disorders
2. Onset and time course of symptoms
3. Any functional limitations
4. Location of symptoms (including radiation of pain to or paresthesias in the distal lower extremity)
5. Temporal relation to work or other daily activities
6. Other precipitating factors
7. Alcohol or drug abuse
8. Depressive symptoms

These questions may identify factors that amplify or prolong pain and are amenable to specific intervention, and they may help plan the person's return to work.

The history should also include a description of the patient's work activities, including awkward postures, lifting requirements, other forceful movements, whole-body vibration, and need for back bending and twisting. It should also include information on monotonous work, job control, job satisfaction, and social support.

The most important immediate goal of the physical examination is to seek physical signs that may indicate a serious medical condition.

Examination of the lumbosacral spine includes musculoskeletal and neurologic components, and it should proceed according to an organized routine. Unfortunately, most of the items commonly assessed on physical examination have limited prognostic significance and limited reproducibility among different examiners.[44] A baseline physical examination allows clinical progression to be assessed. Beginning with the patient disrobed and standing, the alignment, curvature, and symmetry of the spine, pelvis, and lower extremities are evaluated. Range of motion of the lumbosacral spine is assessed in flexion and extension. Visual estimation of range of motion is adequate for general clinical purposes, although goniometers (instruments that precisely measure angles) can also be used for more accurate measurement. Measurement of minimal distance from fingertips to floor is useful to assess the effect of treatment on combined lumbar and hip mobility. A lateral bending maneuver is performed to each side to assess symmetry and any resultant effect on symptoms. Toe raises, heel walking, and standing on one leg (test for Trendelenburg's sign) assist the evaluation of lower-extremity muscle weakness. A thorough neurologic examination is also essential in patients with sciatica or neurologic complaints in the lower extremity.

Diagnostic tests play a very limited role in initial management of acute low back pain. In the absence of "red flags" in the history, X-rays of the lumbosacral spine are unlikely to change diagnosis or therapy. They are often overused. While these X-rays are appropriate with chronic or recurrent low back pain, they should be performed with acute back pain only to rule out a fracture or a systemic disorder if that is suggested by the history. For patients age 20 to 50 with nonradicular back pain and no suggestive history of potentially serious underlying condition, it is best to wait 4 weeks before performing X-rays. If symptoms have not improved in 4 weeks, plain X-rays of the lumbar spine should be performed—along with a complete blood count and erythrocyte sedimentation rate to help rule out an occult neoplasm or osteomyelitis.[44–45] If a neoplasm or osteomyelitis is suspected, but not detected on the plain X-rays, a bone scan or magnetic resonance imaging (MRI) of the spine should be done.

Patients with radicular back pain will generally derive little benefit from early diagnostic imaging, since many of them will have spontaneous resolution of symptoms; and early surgical management is indicated only with severe or progressive neurologic deficits. Patients with persistent or progressive neurologic deficits and a physical examination consistent with nerve-root impingement should be referred for an MRI to evaluate the anatomic basis of nerve-root symptoms. Patients with more ambiguous nerve-root involvement may benefit from electromyography (EMG) to determine whether nerve root impingement is present. Counseling and education of patients are important, in part to dissuade patients from inappropriate imaging studies. Interpretation of an MRI can be problematic because a substantial proportion of people *without* back pain have disc abnormalities that are revealed by MRI; among asymptomatic adults, 22% to 40% have MRI evidence of disc herniation and 24% to 79% have evidence of a bulging disc. Therefore, anatomic abnormalities seen on MRI must be evaluated critically for their clinical importance in each patient.

Older adults with symptoms suggestive of spinal stenosis (pain or paresthesias in the legs relieved by spinal flexion, or pseudoclaudication) should be evaluated with a computed tomography (CT) scan or an MRI; EMG may be useful to determine the extent of neurologic impairment.

Treatment and Prognosis

Nonspecific Low Back Pain

Evidence-based guidelines for the treatment of low back pain have been provided by several expert panels and are useful in managing most cases.[9] For acute cases, the health care provider should offer a confident and positive approach, which is justified by the generally good prognosis of acute low back pain. Reassurance regarding prognosis should be provided, as many workers with low back pain are apprehensive about potential disability. Early return-to-work activities, with work modifications as necessary, and reestablishment of normal or near-normal activities of daily living are important aspects of care. Although unlikely to be of short-term benefit, measures should be implemented to alter

lifestyle factors associated with low back pain, such as smoking, sedentary lifestyle, and obesity.

Nonsteroidal anti-inflammatory drugs (NSAIDs) provide adequate symptomatic relief for most patients. Opioid analgesics may be considered in those few patients who do not attain adequate symptom relief from NSAIDs; opioid drugs should be used with caution and only for a limited time. Muscle relaxants may also relieve symptoms in some patients. Sedation is a common side effect, although in patients who are having trouble sleeping due to back pain this side effect can be used to therapeutic advantage by taking muscle relaxants in the evening. Physical therapy and spinal manipulation are also effective in providing temporary symptom relief in patients with acute or subacute low back pain. Spinal manipulation or physical therapy can be delayed for 2 or 3 weeks after onset of symptoms because many patients will improve spontaneously within this period of time. Back exercises do not seem to be useful in the acute phase; however, exercise is helpful with chronic back pain and in preventing recurrence. Massage therapy may possibly achieve temporary analgesia. A wide array of alternative therapies that are advocated by practitioners are not consistently effective, including laser stimulation of trigger points, various injection therapies, acupuncture, reflexology, traction, and corsets.

In cases of chronic low back pain, an active exercise program is recommended. Treatment of chronic cases emphasizes strengthening and range-of-motion exercises as well as aerobic conditioning, in the context of recurrent evaluation of physical capacities. Maintaining adherence to an intensive exercise regimen may be difficult. Some patients may benefit from referral to a multidisciplinary pain center, where medical therapies are used simultaneously with supervised graded exercise, cognitive and behavioral therapy, and/or patient education. Antidepressants are useful in the one-third of patients with chronic low back pain who have depression, although it is not clear that they are effective in patients without clinical depression.

In workers who have been temporarily disabled from work due to low back pain, decisions about return to work cannot be made in isolation from knowledge about their work and their workplace. Modification of physical job demands to facilitate early return to work is critical in preventing longer term disability.[42] A combination of rehabilitation and ergonomics interventions is most likely to be successful in returning injured workers to work, with the ergonomics intervention contributing most to this success.[10] Intensive clinical and rehabilitation intervention, without ergonomics intervention, have not significantly reduced the time away from regular work.

Herniated Intervertebral Disc

In the absence of cauda equina syndrome or progressive neurologic deficit, conservative (nonsurgical) management should be pursued for at least 1 month in most cases. After 6 weeks of treatment, only about 10% of patients still have sufficient symptoms for consideration of surgical management. Early treatment parallels the treatment of nonspecific low back pain, with the caveat that the safety and effectiveness of spinal manipulation is not clear. Epidural corticosteroid injections offer temporary symptomatic relief in some patients, and their use may reduce rates of surgery in patients who otherwise would be candidates for surgical decompression.

In patients who still have significant pain or neurologic deficits after 4 weeks, discectomy should be considered to provide quicker symptom relief and return to function. Patients with herniated discs who undergo surgery do not return to work more quickly than those treated with nonsurgical therapies, although surgery appears to lead to improved functional and symptomatic outcomes at 1 year. Long-term outcomes are similar among patients treated with or without surgery. The result of surgical treatment of these patients is strongly related to the findings at surgery. The better defined the clinical syndrome is, the better the surgical outcome will be—with at least partial relief of sciatica in up to 90% of carefully selected patients. Approximately 70% of patients experience relief of back pain. Surgical outcomes also can be adversely affected by unrealistic patient expectations, depression, and substance abuse.

LOWER-EXTREMITY DISORDERS

In comparison to low back pain and upper-extremity disorders, little attention has been

paid to WMSDs of the lower extremities. Except for osteoarthritis, studies of work-related lower-extremity disorders have mostly emphasized traumatic injuries. Although disorders such as Achilles tendonitis, plantar fasciitis, and tarsal tunnel syndrome have been recognized as the result of chronic overuse in athletes, they have not been well characterized among workers. Knee bursitis seems to be associated with kneeling work; for example, laying of floors has been recognized as an occupation with high rates of knee bursitis and other disorders of the lower extremities. There is no clear evidence demonstrating that occupational exposures cause other foot and ankle disorders—but there have been few studies reported on this subject.

Workers' compensation data from Washington State (Table 16-1) indicate that nontraumatic knee disorders are more infrequently accepted for compensation than other nontraumatic MSDs. These data indicate that the incidence rate for nontraumatic knee disorders was 9.8 per 10,000 FTEs and 0.5 per 10,000 FTEs for tendonitis/bursitis. The median costs were $1,569 for nontraumatic knee disorders and $713 for tendonitis/bursitis. The median cost of nontraumatic knee disorders was similar to the median costs of epicondylitis and hand/wrist tendonitis. The industries with the highest claims rate for nontraumatic knee disorders were carpentry and floor work, plumbing, residential construction, and roofing, and for knee tendonitis/bursitis were carpentry and floor work, plumbing, electrical work, masonry/stonework/tile setting, and roofing. The most frequently identified occupations of these claimants were carpenters, plumbers, electricians, carpet layers, and roofers.

The best-studied WMSD of the lower extremities is osteoarthritis of the hip and knee. Osteoarthritis is the most prevalent joint disease, the most common disabling medical condition among older adults, and a leading cause of disability among people during their working years.[46] It can affect one or several joints, commonly the hips, knees, shoulders, and fingers. Among persons age 55 or older, 5% to 15% have evidence of hip osteoarthritis, while knee osteoarthritis is even more common. Osteoarthritis has a wide range of severity, from an asymptomatic state evident only on X-rays to symptomatic states that severely limit working abilities and daily activities. Joint replacement may be performed in severe cases. Osteoarthritis is the leading indication for hip and knee replacement; between 120,000 and 200,000 persons undergo total hip replacement annually in North America. In addition to the personal and social aspects of these diseases, the cost to society is enormous.

Most cases of osteoarthritis are idiopathic, and the biologic or biomechanical processes underlying the disease are largely unknown. One hypothesis is that osteoarthritis occurs when repeated stresses at a joint exceed the ability of joint tissues to withstand those stresses, leading to "microtrauma" and cumulative damage. Heavy physical loading from work or sports may thus play a causal role in osteoarthritis when they create imbalances between mechanical stresses and the ability of joint tissues to withstand those stresses.

Heavy physical work is a risk factor for developing osteoarthritis of the hip.[46,47] Repeated heavy lifting and frequent climbing of stairs are associated with an increased risk of osteoarthritis requiring hip replacement. Osteoarthritis of the knee is associated with occupations requiring frequent knee bending, squatting, heavy lifting, and frequent climbing of stairs.[48] However, some research questions the links between work activities and knee and hip osteoarthritis.

PREVENTION

Preventive strategies based on ergonomic interventions are largely experience-based and have not been comprehensively evaluated by scientific studies. Several systematic reviews of ergonomics programs have been performed and summarized.[49] The best practices are based on integrated approaches to hazard control rather than specific "ergonomic tools." Successful ergonomic programs must be as follows:

- Supported by organizational policy
- Implemented with broad-based ergonomics training, rather than a narrow focus on a few tools or tasks
- Committed to making available to workers appropriate technology for performing their jobs safely

Figure 16-3. In jobs like this, reducing the load on the wrist, elbow, and shoulder can be accomplished by one or more of the following three methods: changing the tool, reorienting it (from vertical to horizontal, or vice versa), and changing the height of the work station—either elevating the worker or lowering the piece being worked on. (Courtesy of Washington Industrial Safety and Health Act Services Demonstration Project.)

Reduction in exposures is the most important approach to prevention. This approach often requires changes in the work station, work process, or use of tools. Appropriate interventions must be specific to the biomechanical risk factors encountered in a particular workplace (Fig. 16-3).

It is useful to consider the relationships among the various factors in the work environment that may contribute to either increased risk or risk reduction. The model developed by Michael J. Smith and Pascale C. Sainfort (now Pascale C. Carayon)[50] is an example that analyzes the relationship among the environment, organization, technology, tasks, and the worker (Fig. 16-4).

For example, hot environments may increase metabolic load, which in turn may make it more difficult for a worker to successfully complete a physically demanding task. The organization of work may involve severe structural constraints where workers have no opportunity to change postures or movements. A task may involve heavy lifting from the floor. A tool may have too large a handle, requiring a more forceful grip. Attention to how these components of work interact can identify risk factors for MSDs or approaches for reducing risks.

Ergonomic principles must be adapted to fit the specific characteristics of each work environment. They should be viewed as a guide, rather than a blueprint. Chapter 27 describes the range of ergonomics measures that can be taken to reduce WMSDs and acute traumatic injuries,

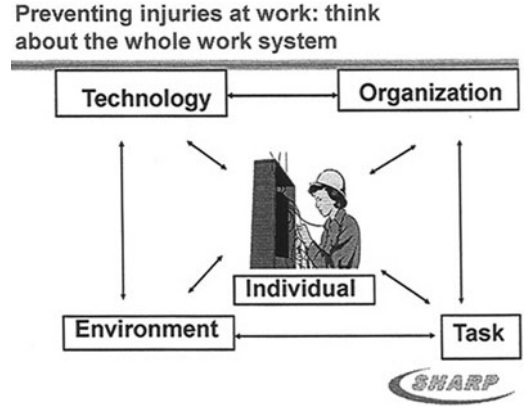

Figure 16-4. Using a work balance model to integrate all of the components of work enhances both health and productivity. (*Source*: Adapted from Smith MJ, Sainfort PC. A balance theory of job design for stress reduction. International Journal of Industrial Ergonomics 1989; 4: 67–79.)

while improving both productivity and the quality of processes and products.

In addition to these engineering controls, there is evidence to support the effectiveness of administrative controls (changing workplace culture), modification of individual risk factors through exercise programs, and the use of programs utilizing a combined approach. Multidisciplinary, participatory approaches that involve employers and employees appear to be successful and foster compliance and acceptance of changes.[51] Sometimes administrative changes, such as work restrictions, or job rotation are useful alternatives, either as preventive or as therapeutic interventions. Use of some types of personal protective equipment, such as palm pads and knee pads are effective. However, one very popular device, lumbar corsets or back belts, do not seem to be effective in reducing the occurrence of low back pain.

In order for work restrictions to be effective in the treatment of injured workers, the health care provider must be specific about the type of work activity that should be avoided or reduced. For example, it is better to limit repetitive hand activities to "fewer than 10 movements per minute for more than 2 hours per day" than to prescribe "no repetitive hand movements during the work shift." Developing specific recommendations for work restrictions is facilitated by viewing videotapes of the usual job of the worker or by obtaining detailed job descriptions from the employer. Job rotation of workers among jobs that require different types of motions or forces may simply expose an even greater number of workers to a considerable degree of risk.

To reduce exposure, the first step required for instituting changes in workstations or work processes is to analyze the specific characteristics of suspected high-risk jobs. Although an industrial engineer or occupational health professional with ergonomics training can conduct the job review, the involvement of those persons who are most knowledgeable about the job is important. Experience has shown that operators and supervisors with limited technical training can successfully identify many of the hazardous aspects of a specific job, and that specific solutions may not be effective or accepted without the involvement of such persons in the job review and development of solutions.

The Hand Activity Level (HAL) Threshold Limit Value[16] is useful for assessing risk in monotask jobs looking at force and repetition. The Strain Index[52] for the distal upper extremity and Rapid Upper Limb Assessment (RULA)[53] tools are useful in performing quick risk assessments. The NIOSH Lifting Equation[54] and the ACGIH TLV for Lifting[55] provide guidance on acceptable lifting, depending on weight, location of the load, and frequency of handling.

After a job analysis has identified the potentially hazardous exposures associated with a particular job, specific solutions should be solicited from those who are knowledgeable about the job. With limited training in the control principles (discussed in the next section), engineers, production employees, and front-line supervisors often propose the most useful methods for eliminating hazardous risk factors. If several factors are present, it can be difficult to determine which is the most detrimental. Where possible, integrated solutions should be developed that reduce multiple risk factors at the same time.

Control of repetitiveness, forcefulness, awkward posture, vibration, mechanical contact stress, and cold are often possible, as illustrated in the following examples.

Control of Repetitiveness

1. Use mechanical assists and other types of automation. For example, in packing operations, use a device, rather than the hands, to transfer parts.
2. Rotate workers among jobs that require different types of motions. Rotation must be viewed as a temporary administrative control, one used only until a more permanent solution can be found.
3. Implement horizontal work enlargement by adding different elements or steps to a job, particularly steps that do not require the same motions as the current work cycle.
4. Increase work allowances or decrease production standards. Management rarely looks on this control strategy favorably.
5. Design a tool for use in either hand and also so that fingers are not used for triggering motions.

Control of Forcefulness

1. Decrease the weight held in the hand by providing adjustable fixtures to hold parts being worked on. Many conventional balancers are available to neutralize tool weight. Articulating arms are used in many plants to hold and manipulate heavy tools into awkward positions.
2. Control torque reaction force in power hand tools by using torque reaction bars, torque-absorbing overhead balancers, and mounted nut-holding devices. Control the time that a worker is exposed to torque reaction by using shut-off rather than stall power tools. Avoid jerky motions by handheld tools.
3. Design jobs so that a power grip rather than a pinch can be used whenever possible. (Maximum voluntary contraction in a power grip is approximately three times greater than in a pinch.)
4. Increase the coefficient of friction on hand tools to reduce slipperiness, such as by use of plastic sleeves that can be slipped over metal handles of tools.
5. Design jobs so that slides or hoists are used to move parts or people, to reduce the amount of lifting, handling, or carrying of parts by the worker (Fig. 16-5).

Control of Awkward Posture

The primary method for reducing awkward postures is to design adjustability of position into the job (Fig. 16-6). Wrist, elbow, and shoulder and back postures required on a job often are determined by the height of the work surface with respect to the location of the worker. A tall worker may use less wrist flexion or ulnar deviation than a shorter worker. Additionally, awkward postures can be reduced by the following procedures:

1. Alter the location or method of the work. For example, in automotive assembly operations, changing the line location at which a particular part is installed may result in easier access.
2. Redesign tools or change the type of tool used. For example, when wrist flexion occurs with a piston-shaped tool that is used on a horizontal surface, correction may involve use of an in-line type tool or lowering of the workstation.
3. Alter the orientation of the work.
4. Avoid job tasks that require shoulder abduction or forward flexion greater than 45 degrees, elbow flexion greater than 110 degrees, wrist flexion more than 20 degrees, wrist extension greater than 30 degrees, or frequent neck rotation, flexion, or extension.
5. Provide support for the forearm when precise finger motions are required, to reduce static muscle loading in the arm and shoulder girdle.

Control of Vibration

1. Do not use impact wrenches or piercing hammers.
2. Use balancers, isolators, and damping materials.
3. Use handle coatings that attenuate vibrations and increase the coefficient of friction to reduce strength requirements.
4. Reduce exposure below ISO standard[56] or Washington State ergonomics appendix B[57] by using alternative tools.

Control of Mechanical Contact Stress

1. Round or flare the edges of sharp objects, such as guards and container edges.
2. Use different types of palm button guards, which allow room for the operator to use the button without contact with the guard.
3. Use palm pads, which may provide some protection until tools can be developed to eliminate hand hammering.
4. Use compliant cushioning material on handles or increase the length of the handles to cause the force to dissipate over a greater surface of the hand.
5. Use different-sized tools for different-sized hands.
6. Avoid narrow tool handles that concentrate large forces onto small areas of the hand.

A

B

Figure 16-5. Risk of low-back injury can be reduced by using an electrical lifting device to reduce load and awkward postures. The photographs demonstrate lifting a patient without such a device (A) and with one (B). (Photographs by Barbara Silverstein.)

Figure 16-6. (A) Traditional method of applying glue to floor posts. (B) New method using commercially available extended gun ($50 retail). A handle was added to the gun to reduce hand/wrist fatigue (parts less than $10). Job times were the same for each of the two method. (Photographs by Barbara Silverstein.)

Control of Cold and Use of Gloves

1. Properly maintain power tool air hoses to eliminate cold-exhaust air leaks onto the workers' hands or arms.
2. Provide a variety of styles and sizes of gloves to ensure proper fit of gloves. Although gloves may protect the hands from cold exposures and cuts, they often decrease grip strength (requiring more forceful exertion), decrease tactile sensitivity, decrease manipulative ability, increase space requirements, and increase the risk of becoming caught in moving parts.
3. Cover only that part of the hand that is necessary for protection. Examples include use of safety tape for the fingertips with fingerless gloves and use of palm pads for the palm.

Other Preventive Strategies

A conditioning process that provides a period of time during which workers can gradually adapt their muscles and tendons to new demands, can be a useful approach for workers in forceful or repetitive jobs. There is some evidence that exercise programs that combine aerobic conditioning with specific strengthening of the back and legs can reduce the frequency of recurrence of low back pain.

Training of new workers in the most efficient and least stressful ways of performing their jobs may also be useful. Similarly, workers with symptoms may, with training, be able to adapt an equally efficient, but less stressful, work method. However, lifting education programs have generally been ineffective at reducing the frequency of occurrence of low back pain. Many other training activities have not been evaluated specifically. Several employers, perceiving long-term benefits from a "phasing-in period," have established transitional or training areas where employees may work at a reduced pace for a limited time. In a survey of 5,000 employers in Washington State, among those who used preventive strategies, a larger percentage reported decreased number and severity of MSDs with engineering and administrative measures (such as task variety, reduced overtime) than with strictly personal controls (such as exercise programs, personal protective equipment).

Development of a replacement process to identify those persons who are at unusually high risk for development of an MSD is the least desirable prevention strategy because there are no scientifically valid screening procedures to identify which persons are at high risk. This shifts the cost of reducing the incidence of symptoms onto the workers (who are denied employment or placement) and increases the costs of the hiring and replacement processes. A recent study evaluating the practice of postoffer preplacement screening for CTS in new workers showed that this practice was not cost-beneficial to the employer.[58] Similarly, the use of preplacement screening with low back X-rays should not be employed, as plain X-rays are not a useful predictor of future low back disorders.

CONCLUSION

Work-related neck and low back pain and disorders of the upper and lower extremity are together among the most common occupational health problems. Although scientific knowledge often limits our ability to determine precisely the role of occupational and nonoccupational factors in the diagnosis of these conditions, substantial progress can be made in reducing their severity by applying existing knowledge about the role of physical factors in these disorders, including forceful repetitive hand work and frequent lifting of heavy objects. Work should be designed to reduce exposure to the known physical risk factors. Encouraging employers to involve employees in decisions that affect the way they perform the job (decision latitude) and also reduce the psychological demands and increase social support will also improve employee health. Encouragement of prompt and appropriately conservative medical evaluation of workers with such disorders can contribute to secondary prevention. Early and safe return to work made possible through ergonomic improvements and modified work regimes have had considerable success. Finally, for the minority of workers with disorders that do not respond to conservative treatment, including reduction in the level of exposure, treatment programs that address all psychosocial and physical aspects of the problem probably have the greatest chance

of preventing permanent disability from these disorders.

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17

Cancer

Elizabeth Ward

Cancer encompasses a broad spectrum of diseases that arise in various organs and tissues throughout the body and have in common the uncontrolled growth of abnormal and potentially lethal cells that lose their differentiation and survive for abnormally long periods. Cancer originates with changes in DNA, or gene expression, that may be triggered by endogenous products of metabolism or exogenous chemicals; physical agents, such as ionizing radiation; or biologic agents, such as viruses, other microorganisms, or their products, such as aflatoxin. Inherited genetic factors play a role in susceptibility to cancer, often by influencing how the body responds to an environmental carcinogen (gene–environment interaction). The human health effects of many recognized environmental carcinogens were first documented through studies of occupational groups with heavy, prolonged exposure.

Cancer is a major public health problem in the United States and throughout the world. Each year, approximately 1.5 million U.S. residents are diagnosed with invasive cancer, and approximately 560,000 die of various cancers. Cancer accounts for almost one-third of deaths in the United States, second only to heart disease. Among men, prostate cancer has the highest incidence, followed by lung cancer and colorectal cancer; among women, breast cancer has the highest incidence, followed by lung cancer and colorectal cancer. In both sexes, the three most common cancer sites account for over half of new cases.[1] Since survival is worse for lung cancer than for other common cancers, lung cancer is the most common cause of cancer death among both men and women.

In the United States and other developed countries, cancer incidence and mortality patterns shifted dramatically during the twentieth century (Fig. 17-1). Most notably, lung cancer in men increased sharply after World War II, peaked in the early 1990s, and declined steadily thereafter; lung cancer in women rose later and only recently began to plateau.[2] These trends largely reflect *(a)* the introduction of manufactured tobacco products early in the twentieth century, and *(b)* differences in men and women in the increase and decline of tobacco smoking. Stomach cancer, one of the major cancers early in the twentieth century, declined steadily during the century, probably due to advances in food preservation, increased availability of fresh fruits and vegetables, and decreases in the prevalence of *Helicobacter pylori* infection. Cervical and colorectal cancer incidence and mortality rates have declined in the United States because of screening and removal of premalignant lesions, early detection, and treatment.

The global burden of cancer is significant. In 2008, an estimated 12.4 million people were newly diagnosed with cancer and 7.6 million

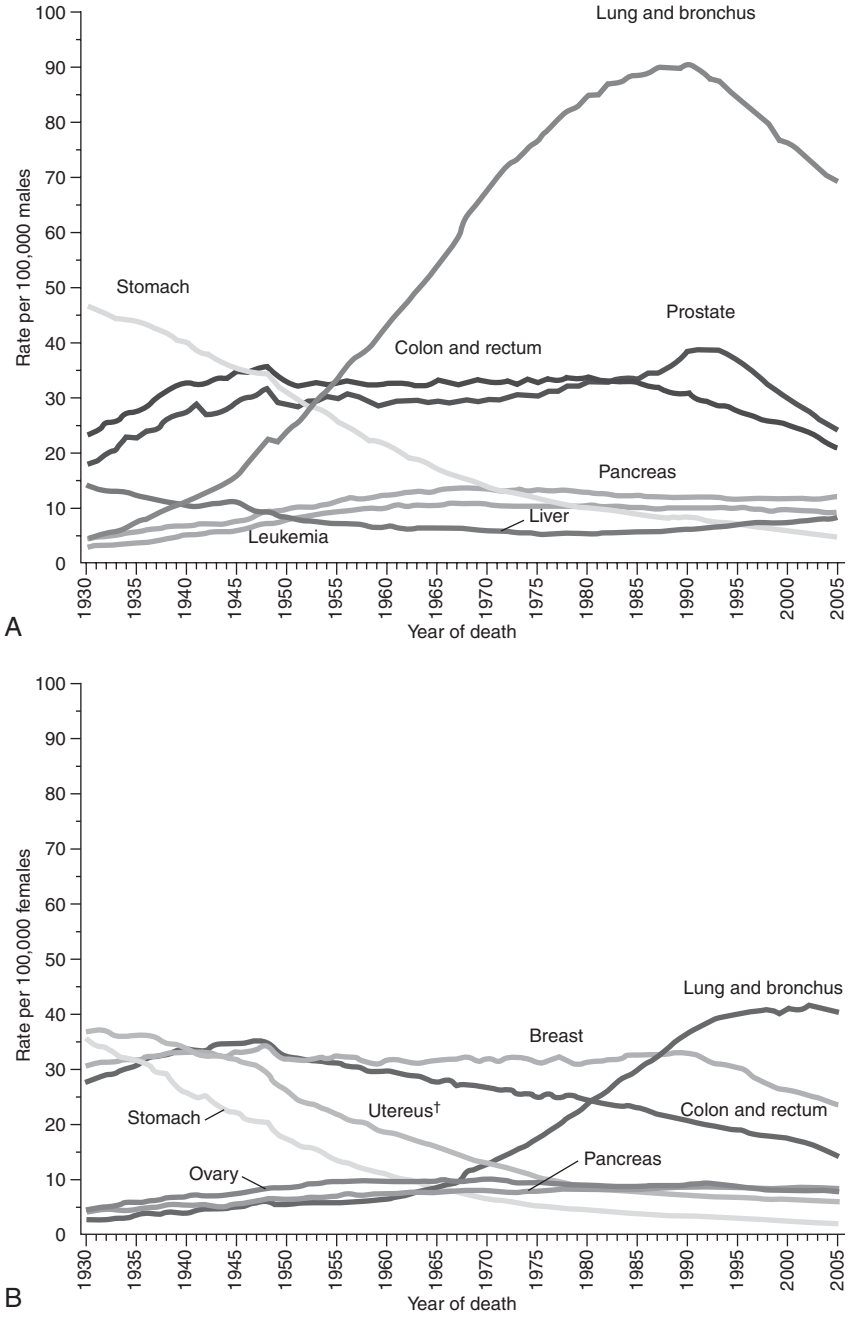


Figure 17-1. Age-adjusted cancer death rates, by site, United States, 1930–2005: (A) Males, (B) Females. (*Source*: American Cancer Society. Cancer facts & figures 2009. Atlanta, GA: American Cancer Society, 2009, p. 2; and National Center for Health Statistics. U.S. mortality data, 1962 to 2005, and U.S. Mortality Volumes, 1930 to 1959. Atlanta, GA: Centers for Disease Control and Prevention, 2008.)

people died from cancer worldwide.[3] In developed countries, the most common types of cancer are lung and bronchus, colon and rectum, breast, and prostate; in developing countries, the most common types of cancer are lung and bronchus, stomach, liver, and breast (Table 17-1). The most common preventable causes of cancer in the United States and other developed countries are cigarette smoking and obesity resulting from dietary patterns and physical inactivity. Other important causes of cancer are occupational exposures, viruses and other biologic agents, reproductive factors, consumption of alcohol, environmental pollution, and ionizing and ultraviolet radiation. In developing countries, infectious agents play a greater role in causation of cancer overall. Among the frequent cancers in men and women in developing countries are stomach cancer associated with *H. pylori* infection, liver cancer associated with hepatitis B virus and hepatitis C virus infection, and cervical cancer caused by human papilloma virus (HPV) infection. It is predicted that cancer will become an even more important cause of premature mortality in developing countries due to increases in cigarette smoking, changing dietary and physical activity patterns, and growth of hazardous industries.

People have been exposed to carcinogenic agents in their environment and cancer has been observed throughout human history. However, industrialization and growth of the

Table 17-1. Ten Leading Sites of New Cancer Cases and Deaths, Developed and Developing Countries, 2007 (in Thousands)

Developed Countries			
Estimated New Cases		Estimated Deaths	
Males	Females	Males	Females
Prostate: 567	Breast: 680	Lung and bronchus: 466	Breast: 204
Lung and bronchus: 529	Colon and rectum: 336	Colon and rectum: 176	Lung and bronchus: 174
Colon and rectum: 388	Lung and bronchus: 210	Prostate: 144	Colon and rectum: 165
Stomach: 215	Corpus uteri: 147	Stomach: 141	Stomach: 90
Urinary bladder: 192	Stomach: 124	Liver: 78	Pancreas: 73
Kidney: 94	Ovary: 103	Pancreas: 78	Ovary: 67
Non-Hodgkin lymphoma: 89	Cervix uteri: 87	Urinary bladder: 57	Cervix uteri: 42
Liver: 81	Non-Hodgkin lymphoma: 72	Esophagus: 55	Liver: 41
Pancreas: 77	Melanoma of the skin: 69	Leukemia: 50	Leukemia: 41
Leukemia: 75	Pancreas: 69	Kidney: 44	Non-Hodgkin lymphoma: 37
All sites*: 2,948	All sites*: 2,478	All sites*: 1,648	All sites*: 1,272
Developing Countries			
Estimated New Cases		Estimated Deaths	
Males	Females	Males	Females
Lung and bronchus: 564	Breast: 593	Lung and bronchus: 496	Cervix uteri: 272
Stomach: 475	Cervix uteri: 473	Liver: 399	Breast: 256
Liver: 424	Stomach: 251	Stomach: 370	Stomach: 199
Esophagus: 301	Lung and bronchus: 225	Esophagus: 247	Lung and bronchus: 198
Colon and rectum: 228	Colon and rectum: 187	Colon and rectum: 138	Liver: 167
Prostate: 195	Liver: 172	Prostate: 107	Esophagus: 129
Oral cavity: 129	Esophagus: 153	Leukemia: 87	Colon and rectum: 112
Urinary bladder: 116	Ovary: 124	Oral cavity: 68	Ovary: 72
Leukemia: 111	Oral cavity: 84	Non-Hodgkin lymphoma: 67	Leukemia: 66
Non-Hodgkin lymphoma: 103	Leukemia: 83	Urinary bladder: 66	Pancreas: 47
All sites*: 3,588	All sites*: 3,168	All sites*: 2,658	All sites*: 2,022

*Excludes nonmelanoma skin cancer.

Source: Garcia, M, Jemal A, Ward E, et al. Global cancer facts and figures 2007. Atlanta, GA: American Cancer Society, 2007.

chemical industry in the early twentieth century created opportunities for concentrated, high-level exposures among working populations. Exposures included *(a)* naturally occurring substances that for the first time were mined and milled for industrial uses, such as asbestos and uranium; *(b)* substances extracted from natural sources, such as benzene from petroleum; and *(c)* newly synthesized substances, such as vinyl chloride. Due to large increases in cancer risk associated with high-level industrial exposures from the middle to the end of the twentieth century, case reports and epidemiologic studies documented high risks of *(a)* bladder cancer among dye workers exposed to the aromatic amines β-naphthylamine and benzidine, *(b)* lung cancer among uranium miners exposed to radon, *(c)* lung and skin cancer in workers exposed to arsenic, and *(d)* lung cancer and pleural and peritoneal mesothelioma among workers exposed to asbestos.

Development of experimental models for carcinogenesis led to formal bioassay programs at the National Cancer Institute (NCI) and the National Toxicology Program (NTP). These testing programs confirmed both the high correlation between carcinogenicity in experimental models and human carcinogenicity, and the scientific basis for prevention of widespread human exposure to carcinogens through toxicologic testing and regulation. Occupational epidemiology studies, many of which were initiated in the 1970s and 1980s, documented the carcinogenicity of asbestos, benzene, beryllium, bis-chloromethyl ether (BCME), coke oven emissions, vinyl chloride, and some other widely used substances.

The proportion of new cancers and deaths in the United States and worldwide that are related to occupational and environmental carcinogens is not precisely known. In 1981, it was estimated that 4% of all cancer deaths in the United States were due to occupational exposures; a more recent estimate is 2.4% to 4.8% (Table 17-2).[4,5] In Great Britain, it was estimated that, in 2004, 8% of cancer deaths among men and 1.5% among women were attributable to occupational carcinogens.[6] Globally, an estimated 10% of lung cancer deaths, 2% of leukemia deaths, and nearly 100% of mesothelioma deaths are attributable to occupation—with occupational exposures resulting, in the year 2000, in 102,000 deaths from lung cancer, 7,000 from leukemia, and 43,000 from mesothelioma.[7]

OCCUPATIONAL AND ENVIRONMENTAL CARCINOGENS

Although there has been much progress in the recognition and control of carcinogenic hazards in the United States and many other developed countries, some of the earliest recognized occupational carcinogens continue to be widely used and inadequately controlled in much of the world today. Lung cancer accounts for more than half of occupational cancer cases worldwide, and asbestos is, by far, the most important occupational exposure accounting for lung cancer.[8] In 2005, the World Health Organization (WHO) estimated that 125 million people worldwide were exposed to asbestos at work,[9] despite the recognition of asbestos-related cancer and lung disease for more than 60 years. In addition, excess risks of lung cancer and mesothelioma, an extremely rare cancer of the pleura and peritoneum strongly associated with asbestos, continue for decades after asbestos exposure has been reduced or eliminated. In the United States, where use of asbestos peaked in the 1970s (Fig. 17-2), an estimated 27.5 million workers were exposed to asbestos from 1940 to 1979, including 18.8 million exposed to more asbestos than the equivalent exposure from 2 months work in primary manufacturing or insulation.[10] It was projected that annual mortality from asbestos-related diseases in the United States would peak in the year 2000 at about 9,700 deaths (including approximately 3,000 from mesothelioma and 4,700 from lung cancer), then decline, but remain substantial, for another three decades. The projections were remarkably accurate for mesothelioma deaths, which rose from 2,482 (14.1 per million) in 1999 to 2,704 (14.0 per million) in 2005.[11] Higher rates of lung cancer mortality are found in counties with shipyards.[12] There has been increasing evidence for associations between asbestos exposure and cancer of other sites, including laryngeal, ovarian, esophageal, stomach, and colorectal cancers.[13,14]

Consumption of asbestos worldwide was estimated at about 2 million metric tons in 2006,

Table 17-2. Estimated Number of Occupationally Related Cancer Deaths for Selected Cancers and Illustrative Exposures, United States, 1997

Cause of Death and Exposure	Number of Deaths*	Estimated Number of Exposed Workers	Estimated Percent of U.S. Workforce Exposed	Relative Risk	Estimated Proportion Due to Occupational Exposures (Percent) (AF)	Estimated Number of Occupationally Related Cancer Deaths
Selected cancers						
Lung cancer	91,289 (M); 61,877 (F)	NA		NA	6.3–13.0 (combined)	9,677–19,901
Chemical exposures					6.1–17.3 (M); 2 (F)	6,807–17,031
Environmental tobacco smoke (ETS) ("never-smokers" only, 10% of all lung cancer deaths)					0.6 (M+F)	870
Indoor radon at work					1.3 (M+F)	2,000
Bladder cancer	7,638 (M); 3,897 (F)	NA		NA	7–19 (M); 3–19 (F)	651–2,191
Mesothelioma	2,081 (M); 548 (F)	NA		NA	85–90 (M); 23–90 (F)	1,895–2,366
Leukemia	19,038 (M+F)				0.8–2.8 (combined)	152–533
Benzene		1,000,000	0.72	2–4	0.8–2.0	
Ethylene oxide		1,000,000	0.72	1.1–3.5	0–1.6	
Ionizing radiation (100+ mSv)		61,700	0.04	1.3–2.1	<0.05	
Ionizing radiation (50–100 mSv)		70,900	0.05	1.1–1.4	<0.05	
Laryngeal cancer	3,016 (M)				1.0–20.0 (combined)	30–603
Sulfuric acid		3,000,000	4.4	1.1–5.0	0.4–15.0	
Mineral oils		4,400,000	6.4	1.1–2.0	0.6–6.0	
Skin cancer	1,407 (M)				1.5–6.0 (combined)	21–84
Polycyclic aromatic hydrocarbons		8,222,800	12.0	1.1–1.5	1.2–5.7	
Arsenic		240,000	0.36	2	0.1	
Sinonasal (SN) and nasopharynx (NP) cancer	303 (SN) (M) and 436 (NP) (M)				33.0–46.0 (SN) and 30.0–42.0 (NP)	231–322 (SN and NP)
Wood dust		4,515,200	6.8	3.1 (SN); 2.4 (NP)	12.5 (SN); 8.7 (NP)	
Nickel compounds		4,000,000	6.0	2.2	6.7 (SN and NP)	
Hexavalent chromium		3,400,000	5.2	5.2–10.8	18.0–33.8 (SN and NP)	
Kidney cancer	7.131					
Coke production		520,000	0.76	2	0.0–2.3	0–164
Liver cancer	7,283					
Vinyl chloride		320,000	0.48	2.5	0.4–1.1	29–80
Total occupationally related cancer deaths						12,686–26,244

F, female; M, male; NA, not available; AF, attributable fraction.
*Attributable fractions taken from the literature; see text.

Source: Steenland K, Burnett C, Lalich N, et al. Dying for work: the magnitude of US mortality from selected causes of death associated with occupation. American Journal of Industrial Medicine 2003; 43: 461–482.

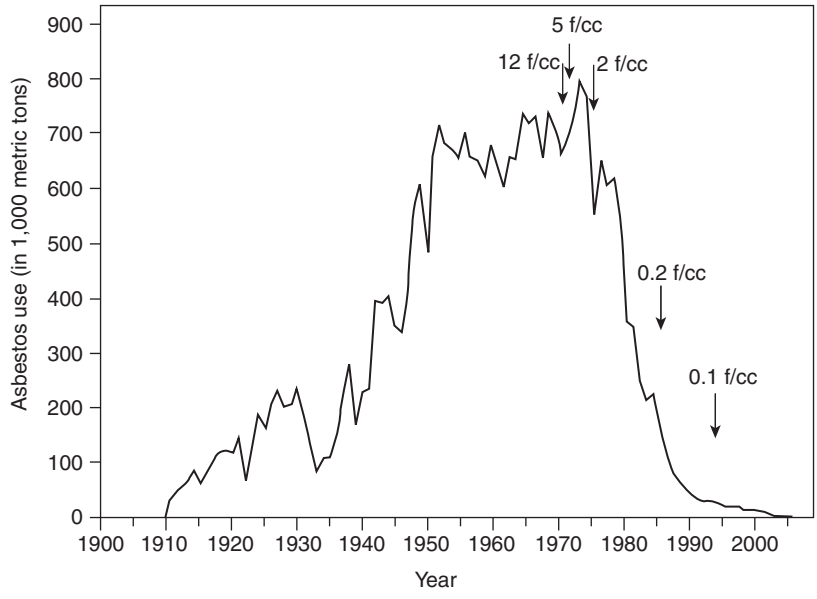


Figure 17-2. Asbestos use and permissible exposure limits (PELs), United States, 1900–2007. Arrows indicate year when Occupational Safety and Health Administration PELs were put in place (12 fibers per cubic centimeter [f/cc] in 1971, 5 f/cc in 1972, 2 f/cc in 1976, 0.2 f/cc in 1986, and 0.1 f/cc in 1994). (*Source*: Centers for Disease Control and Prevention. Malignant mesothelioma mortality–United States, 1999-2005. Morbidity and Mortality Weekly Report 2009; 58: 393-396.)

down from a peak of over 4.5 million tons in 1975. In 2003, eight countries (China, Russia, India, Kazakhstan, Ukraine, Thailand, Brazil, and Iran) accounted for 82% of asbestos consumption.[15] Occupational and environmental exposures in many of these countries have not been well documented, but they may equal or exceed exposures in developed countries when they began to industrialize. In South Africa, which mined asbestos minerals throughout most of the twentieth century, high prevalence of asbestos-related diseases and significant environmental contamination resulted from early use of open-pit mining, widespread use of a manual process (cobbing) to separate asbestos from rock, frequent employment of women and children in asbestos industries, inadequate dust control in mines and mills, and widespread dumping of tailings near population settlements.[16,17] Hazardous working conditions in the asbestos industry there persisted because of the poverty and isolation of the mining regions; the apartheid system, which allowed few employment opportunities for black workers and forced many into migrant labor; and the weakness of government regulation. Since transition to majority rule in 1994, there have been serious efforts by the South African government to assess exposure, involve affected communities in decision making, and raise public awareness about asbestos-induced diseases. Still, the resources that will be required to adequately address the asbestos problem there are staggering, especially in the context of poverty and limited medical resources in many affected communities.[16,17]

In contrast, India is a major importer but only a minor producer of asbestos. Expansion of the asbestos industry in India in the 1990s was associated with drastic reductions in import tariffs on raw asbestos and asbestos-containing products. Exposures there occur in mining, milling, manufacturing of asbestos cement and asbestos textiles, and shipbreaking.[16] Half of the nearly 700 ships that are scrapped worldwide each year are broken down in India. Each ship contains about 5.5 tons of asbestos, which is then resold to manufacture insulation and roofing materials. Dusty conditions, inadequate control technology, and lack of personal protective equipment are common.[18,19] In 2006, WHO stated that the most efficient way to eliminate

asbestos-related diseases is to ban the use of all forms of asbestos; 46 countries, including South Africa, have done so.[20]

Cancers related to asbestos are not confined to workers with high exposure. Mesotheliomas have been observed among household members of asbestos workers and in residents of asbestos-contaminated communities. For example, vermiculite ore from a mine that operated near Libby, Montana, from the early 1920s until 1990 was contaminated with asbestos, during which time it was given free of charge to the community and used extensively in gardens, driveways, public areas, and homes. Fifteen deaths from mesothelioma were identified among workers who mined, milled, and processed vermiculite in Libby and 11 individuals with mesothelioma were identified who did not have occupational exposure. (Two had household exposure to workers and nine had environmental exposure.)[21,22] An investigation of 70 communities across the United States that received asbestos-contaminated vermiculite ore from this mine found 11 communities with excess rates of mesothelioma.[23]

Many mesothelioma cases have also been observed among family members of workers at, and community residents near, an asbestos mine in western Australia. Among the 3,000 women and girls who lived in the mining and milling town of Wittenoom between 1943 and 1992, 40 deaths from mesothelioma had occurred by 2004, representing 8% of total deaths.[24] Mesotheliomas have also been observed in areas of Turkey, Greece, Corsica, New Caledonia, and Cyprus where asbestos and asbestos-like materials present in soil are used to whitewash interior walls of houses. Residents of these areas also have a high prevalence of pleural plaques, a nonspecific effect often due to asbestos exposure.[25,26]

Although some aspects of the history of occupational and environmental cancer related to asbestos are unique, this history has much in common with other occupational and environmental carcinogens. Most cancers caused by occupational or environmental exposures are pathologically and clinically indistinguishable from cancers not caused by these exposures. Therefore, unless elevated risks are exceptionally high, a large and carefully designed epidemiologic study is necessary to determine whether cancer rates are elevated and associated with specific exposures. Mesotheliomas caused by asbestos and angiosarcomas of the liver caused by vinyl chloride and Thorotrast are rare. However, they are notable exceptions in which almost all cases of a very rare histologic type of cancer are associated with a specific exposure.

For mesothelioma, the latency period between onset of exposure to occupational or environmental carcinogens and diagnosis of cancer is often long, with excess disease rates peaking 30 to 40 years after onset of population exposure. Therefore, epidemiologic studies require accurate and complete records of people who were exposed decades ago.

Many occupational carcinogens have other toxic effects often associated with high-level exposure, including pleural plaques and fibrotic lung disease in asbestos workers, myelosuppression in workers exposed to benzene, and liver fibrosis and acroosteolysis (clubbing of the fingers) in workers exposed to vinyl chloride. Although exposure to recognized occupational carcinogens has been curtailed in the United States and Western Europe, production and use has shifted to developing countries, where there is limited documentation of both asbestos exposure and occurrence of cancer and other occupationally related diseases. Although it has been difficult to detect cancer caused by very low exposures to environmental carcinogens in epidemiologic studies, exposures to known carcinogens are widespread and may contribute to cancer incidence and mortality. For example, many people are exposed to low levels of benzene, which is a constituent of cigarette smoke, gasoline vapor, and gasoline engine exhaust (Fig. 17-3).

THE MOLECULAR BASIS OF CANCER

Even among people exposed to high levels of carcinogens for long periods, not all of them develop cancer. Despite their greatly increased relative risk of lung cancer, not all lifetime heavy smokers develop lung cancer, and some nonsmokers with no known risk factors develop lung cancer. Both inherited genes and environmental factors play roles in cancer development. Multiple events occurring in a probabilistic

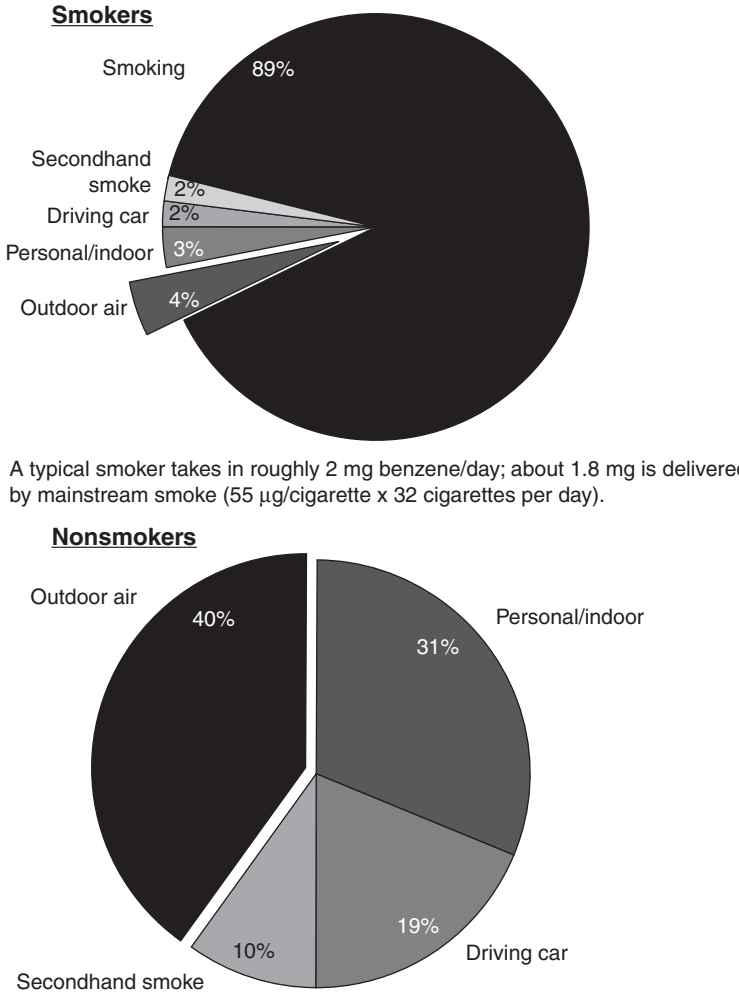


Figure 17-3. Sources of benzene exposure in the general environment. (This figure excludes occupational sources of benzene exposure.) (*Source*: American Cancer Society. Global cancer facts & figures 2006. Atlanta, GA: American Cancer Society, 2006, p. 27; and U.S. Environmental Protection Agency TEAM Studies.)

fashion may influence whether a person develops cancer. A study of twins concluded that environmental factors are the principal cause of most cancers, with a significant role of heritable factors for prostate cancer (42% of the risk possibly explained by inherited genes), colorectal cancer (35%), and breast cancer (27%).[27]

Carcinogenesis is characterized by four stages: initiation, promotion, malignant transformation, and tumor progression. Initiation occurs when a carcinogen interacts with DNA, most often by forming an adduct between the chemical carcinogen or one of its functional groups and a nucleotide in DNA, or by producing a

strand break. If the cell divides before the damage is repaired, an alteration can become permanently fixed as a heritable error that will be passed on to daughter cells. Such heritable changes in DNA structure are called mutations. Many mutations have no apparent effect on gene function. However, when mutations occur in critical areas of genes that regulate cell growth, cell death, or DNA repair, they may predispose clonal expansion and accumulation of further genetic damage. Promoters are substances or processes that contribute to clonal expansion by stimulating initiated cells to replicate, forming benign tumors or hyperplastic lesions. Promotion is thought to be completely reversible. The process of promotion does not cause heritable alterations or mutations. It stimulates cell turnover, so that mutated cells can exploit their selective growth advantage and proliferate, increasing the probability that a cell will acquire additional mutations and become malignant. Unlike promotion, the end result of malignant transformation is irreversible. Tumor progression involves the further steps of local invasion and/or metastasis.

Many carcinogens are able to form DNA adducts, either because they are intrinsically reactive or are activated, through metabolism, to a DNA-reactive form. Classes of organic compounds associated with cancer include alkylating agents, arylalkylating agents, and arylhydroxylamines. Alkylating agents are chemicals that attach alkyl groups, such as methyl or ethyl groups, to nucleotides to form DNA adducts. Examples of carcinogens in this group include nitrosamines and aflatoxin B1, a potent liver carcinogen that can contaminate food products. Arylalkylating agents can transfer aromatic or multiringed compounds to a nucleotide to form an adduct. Examples of such compounds include polycyclic aromatic hydrocarbons (PAHs), such as benzo(a)pyrene (BAP). Arylhydroxylamines are chemicals that transfer aromatic amines to nucleotides to form adducts. Examples of such compounds include the aromatic amines β-naphthylamine and benzidine, which have been responsible for very high rates of bladder cancer among exposed workers. Certain inorganic metals and minerals show carcinogenic activity in people and/or animals, including arsenic, nickel, (hexavalent) chromium, and asbestos.

The mechanisms for carcinogenicity of particles and fibers include both primary genotoxicity through generation of reactive oxygen species and secondary genotoxicity through particle-induced inflammation. Particles may also carry mutagens to the surface and/or inside of cells. Ionizing radiation is a classic cancer initiator.

The mechanism of carcinogenesis from ionizing radiation is believed to involve formation of mutagenic oxygen free radicals in the shell of hydration surrounding DNA. Once formed, the reactive oxygen species, such as hydroxyl radicals and hydrogen peroxide, can induce strand breaks and more than 30 different DNA adducts as well as DNA-protein cross-links.[28] Unrepaired or misrepaired DNA double-strand breaks are thought to be the principal lesions responsible for induction of genetic damage by ionizing radiation in mammalian cells, while base damage is generally the predominant mechanism for the production of such damage by chemical carcinogens.[29]

Metabolic activation is necessary to convert some chemicals to forms that can bond with DNA. For some well-studied chemical carcinogens, the metabolic pathways leading to activation or deactivation influence both target organ specificity and individual susceptibility. Genetic polymorphisms in metabolic enzymes are likely to affect susceptibility to occupational and environmental carcinogens. Studies have examined variation in *(a)* genes, such as CYP1A1, that code for cytochrome P-450s; *(b)* intracellular proteins involved in the metabolism of carcinogenic PAHs to epoxides; *(c)* GSTM1 that codes for a cytosolic enzyme glutathione-S-transferase M1, which can conjugate epoxides of PAHs and aflatoxin; and *(d)* NAT2, which codes for the N-acetylation phenotype associated with metabolism of some carcinogenic aromatic amines. After a carcinogen has reached and interacted with cellular DNA, the carcinogenic process may be arrested by DNA repair, or promoted by factors that increase cell replication or interfere with the programmed death of damaged cells (apoptosis). Thus, the outcome of a carcinogen–DNA interaction may be influenced by factors such as cell division, clonal expression, loss of tumor suppressor function, and other genetic and epigenetic factors.

Although many mutations probably have no effect on cells, mutations occurring in genes that

regulate cell growth are the first step in the evolution of a cancer cell. More than 100 genes can convert normal rodent cells in tissue culture to a transformed phenotype with abnormal growth characteristics in cell culture and the ability to form tumors when explanted into immunocompromised rodents. These dominant transforming genes, called oncogenes, encode proteins involved in signal transduction or cell-cycle regulation. Mutations in these genes may trigger production of oncogenic proteins that increase the proliferation of cells that express them. A set of recessive tumor suppressor genes has been identified. Deletion, point mutation, or inactivation of both gene copies allows cells to proliferate unregulated or with reduced restraints.

An oncogene is an altered form of a normal cellular gene called a proto-oncogene. Proto-oncogenes encode proteins that participate in the regulation of growth and/or differentiation of normal cells, and are involved at various levels in signaling from the extracellular compartment to the nucleus. One of the best-studied examples is the ras oncogene, which was first identified in rat sarcomas. The ras oncogene can be activated by PAHs, N-nitroso compounds, and ionizing radiation, and has been found in a wide variety of human cancers, including bladder cancer, lung cancer, and other cancers of occupational and environmental importance.

Tumor suppressor genes, or anti-oncogenes, are also important. Ordinarily these function to regulate cell growth and stimulate terminal differentiation or trigger apoptosis of damaged cells. When inactivated, they fail to perform these functions, allowing neoplastic transformation to proceed. A prominent example is the p53 gene, located on chromosome 17. Mutations in the p53 gene have been identified in many cancers, including those of the colon, lung, liver, esophagus, and breast; lymphohematopoietic malignancies; and the Li-Fraumeni syndrome of familial multiple cancer susceptibility. Carcinogenic exposures such as aflatoxin and HBV have been associated with specific mutations on the p53 gene, suggesting that some carcinogens may leave a unique genomic "signature."

Epigenetic mechanisms for deactivation of tumor suppressor genes include methylation of DNA in the gene promoter region, a characteristic that has been observed in many cancers. Abnormal promoter hypermethylation can have the same effect as a coding region mutation in inactivating a tumor suppressor gene.[30]

Once a cell is initiated, clonal expansion may occur through a variety of mechanisms. Initiated cells may be more responsive to growth stimulation, may be unable to terminally differentiate, or may become resistant to apoptosis. Clonal expansion increases the probability that cells with critical mutations will acquire additional genetic damage needed for malignant transformation.

The events involved in progression are less well understood than those involved in initiation or promotion. During progression, populations of tumor cells undergo further selection, and the genome becomes unstable, causing chromosomal alterations with increasing frequency. As the progression phase ends, tumor cells have converted to the neoplastic phenotype, characterized by autonomous growth and ability to erode normal tissue barriers.

Endogenous factors, such as hormones, inflammation, and the by-products of metabolism, are major sources of initiating and promoting events.[31] Oxygen reactive species, including superoxide, hydrogen peroxide, and hydroxyl radicals, and singlet oxygen, generated by normal cellular processes, including respiration, inflammation, and phagocytosis, have the ability to induce mutations. Endogenous DNA lesions are genotoxic and induce mutations that are commonly observed in mutated oncogenes and tumor suppressor genes.[32] The levels of oxidative DNA damage reported in many tissues or in animal models of carcinogenesis exceed the levels of lesions induced by exposure to exogenous carcinogenic compounds. Oxidative DNA damage is probably important in the etiology of many human cancers, but we do not know the precise role that it plays in carcinogenesis and how it synergizes with other forms of genetic and epigenetic events to accelerate cell transformation and malignant transformation. The association of cancer with chronic inflammatory diseases, such as gastritis, chronic hepatitis, ulcerative colitis, and pancreatitis, may result from generation of reactive oxygen species.[31]

Endogenous and exogenous hormones also play a role in the development of cancer, and there is increasing use of hormonal medications

for chemoprevention and treatment of cancer. Although metabolites of estradiol and estrone are genotoxic, a major action of these hormones is to accelerate the accumulation of somatic genetic errors. Endogenous and exogenous hormones play an especially important role in development of cancers of the breast, endometrium, ovary, and prostate. Higher cumulative exposure to endogenous estrogen and many ovulating cycles (associated with earlier menarche, later menopause, and lower parity) increases breast cancer risk among women, as does use of combined estrogen and progesterone hormonal therapy for menopausal symptoms. Hormonal medications may have opposing effects in different organs. Oral contraceptives cause a small increase in breast cancer, but a large decrease in ovarian cancer. Tamoxifen, a weak estrogen agonist, is effective in treatment of breast cancer and in the prevention of breast cancer for high-risk women, but it increases the risk of endometrial cancer.

In summary, the development of cancer is a multistage process, involving the activation of oncogenes, deactivation of tumor suppressor genes, disruption of DNA processes, and progressive genetic deterioration. In most instances, the process of carcinogenesis involves a series of probabilistic events, in which both exogenous exposures and endogenous factors influence the outcome.

IDENTIFICATION OF POTENTIAL CARCINOGENS

Substances that have been formally classified as carcinogens are those in which adequate toxicologic and epidemiologic studies have documented a carcinogenic hazard. Although high-quality epidemiologic data provide a strong basis for hazard identification and risk assessment, it is often not possible to conduct definitive studies in humans. There are many animal carcinogens for which definitive epidemiologic studies cannot be performed due to multiple and/or poorly characterized exposures, use in small workplaces, and/or other limitations. Thus, the prevention of occupational and environmental cancer must often rely on extrapolation of findings in toxicological studies to predict effects in humans and establish limits for human exposure. Although the 2-year rodent bioassay is currently the "gold standard" for toxicological testing of carcinogenicity for hazard identification and risk assessment, the time and expense of these assays limit the number of agents that can be tested. Thus, high priority is given to developing both *(a)* alternative testing methods to identify which substances should be tested, and *(b)* testing methods that are highly predictive of cancer in humans.

Predictions Based on Chemical Structure

Knowledge about the relationship of chemical structure and carcinogenic activity can be used to identify potential chemical carcinogens. Computerized databases of carcinogenic and noncarcinogenic chemicals have been developed to relate structure to carcinogenic activity. Using results of rodent bioassays of more than 300 chemicals, a list of chemical structures that correlate with tumorigenicity in rodent tests has been developed. These characteristics, or "structural alerts," indicate chemicals that should be tested extensively and monitored for evidence of carcinogenicity.[33] However, studies comparing the results of widely used computer programs with in-vitro studies have found limited concordance.[34,35]

Toxicologic Testing

Short-Term Tests

Various short-term tests have been developed to determine whether chemicals are mutagenic. Among these, the Ames assay has been the best studied and most extensively used. This test uses special strains of *Salmonella typhimurium* bacteria that are deficient in DNA repair and cannot grow in the absence of histidine. Cultures of *S. typhimurium* are treated with several dose levels of the chemical being tested. Growth of a cell culture in the absence of histidine indicates a mutation causing reversion to the histidine-positive phenotype. Homogenates of mammalian liver, which promote metabolic activation, may be added to the incubation mixture to allow detection of carcinogens that require metabolic activation. In-vitro mammalian cell mutation

assays also exist, including the mouse lymphoma L5178Y (MOLY) assay and the Chinese hamster ovary (CHO) assay. Other short-term tests involve both in-vitro and in-vivo (in animals) induction of chromosome aberrations, sister chromatid exchanges (SCEs), and micronuclei.

The concordance between short-term tests of mutagenicity and the results of chronic bioassays (animal tests) depends on the databases selected for comparison. In general, the concordance has declined over time as an increasing proportion of nongenotoxic carcinogens have been tested in rodent bioassays. An analysis of 59 chemicals classified by the International Agency for Research on Cancer (IARC) as human carcinogens (Group 1) or probable human carcinogens (Group 2A) that had been tested for mutagenicity by multiple methods found positive results on the Salmonella assay for 38 (67%) of the 57 chemicals tested. A total of 93% were mutagenic in in-vivo or in-vitro mammalian tests.[36] Based on the accumulated information, it has been recommended that screening protocols for genetic effects in vitro include tests for both *(a)* gene mutation in Salmonella and/or mammalian cells; and *(b)* chromosome aberrations and numerical chromosome changes (aneuploidy) in mammalian cells.[37,38] Some carcinogens are not detected by mutagenicity assays, including hormonal carcinogens, some metals, agents that have a mode of action involving multiple target organs, and agents with a nongenotoxic mode of action.

Chronic Two-Year Bioassay

The "gold standard" for determining the potential carcinogenic activity of a chemical is the 2-year bioassay in rodents. This assay involves test groups of 50 rats and 50 mice of both sexes and at two or three doses of the test agent. In the United States, the B6C3F1 mouse and the F344 rat are commonly used. At about 8 weeks of age, test animals are placed on the test agent (or placebo) for the remaining 96 weeks of their life span. The test agent may be administered in feed, by gavage (forced feeding), or by inhalation. The maximum dose level used in a 2-year bioassay is determined by the estimated maximally tolerated dose (MTD), usually derived from a 90-day study. The MTD is defined by the Environmental Protection Agency (EPA) as "the highest dose that causes no more than a 10% weight decrement, as compared to appropriate control groups; and does not produce mortality, clinical signs of toxicity, or pathological lesions (other than those that may be related to a neoplastic response) that would be predicted to shorten the animal's natural lifespan." Controversy exists around the use of the MTD, with some scientists claiming that it is not high enough to elicit the anticipated effects, while others voicing concern that the MTD is too high, it may overwhelm mechanisms that defend the host against low exposure levels, and may induce cancer because of toxicity and abnormal cell proliferation. In any case, high exposure levels are necessary to provide meaningful results without requiring studies that are prohibitively large and costly.

Even with the use of high exposure levels, the high cost of 2-year bioassays, estimated to be over $1 million per chemical, limits the number of tests that can be conducted. Genetically altered mice are being evaluated as possible replacements for, or adjuncts to, conventional rodents for bioassays of chemical carcinogenesis. Some researchers speculate that transgenic mice will be well suited to identifying human carcinogens because they already possess altered genes known to be involved in human cancer.[13] However, the results of transgenic carcinogenesis testing must be validated against known and probable human carcinogens before they can be used for risk assessment. Currently, the 2-year bioassay remains the only widely accepted indicator of carcinogenic potential to humans by international and national health and regulatory agencies.

Many substances that are carcinogenic in rodent bioassays have not been adequately studied in humans, usually because an adequate study population has not been identified. Among the substances that have proven to be carcinogenic in animals, all have shown positive results in well-conducted 2-year bioassays. Moreover, between 25% and 30% of established human carcinogens were first identified through animal bioassays. Since animal tests necessarily use high-dose exposures, in most cases human risk assessment requires extrapolating the exposure–response relationship observed in rodent bioassays at higher doses to predict effects in humans at lower doses. Typically, regulatory agencies in the United States have adopted the default

assumption that no threshold level of exposure exists for carcinogenesis. For some chemicals, mechanistic hypotheses have been advanced to suggest that there may be a threshold, but for most carcinogens, it is considered infeasible to generate empirical data on the exposure–response curves at low levels to confirm or refute these hypotheses.

Although the presence or absence of carcinogenicity is similar across many species, the target organ affected by cancer may vary, largely because of differing metabolic pathways, in different species. Benzidine, for example, causes bladder cancer in humans, hepatomas in mice, and intestinal tumors in rats. One database in which the results of animal bioassays are tabulated is the Carcinogenic Potency Database, maintained by the Lawrence Berkeley Laboratory and the University of California, Berkeley. Results are available at the National Library of Medicine Toxicology Data Network (TOXNET) (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?CPDB.htm).

Toxicogenomics

The potential to use toxicogenomic technologies to identify potential human carcinogens and to prioritize substances for 2-year cancer bioassays has been of great interest in the past decade. Incorporation of toxicogenomics into screening tests involves measuring gene, protein, or metabolite changes in response to specific doses of an administered test compound at a specific time point, with or without the parallel development of more traditional markers of toxicity. Toxicogenomic technologies include technologies that analyze DNA sequences (genomics), messenger RNA expression (transcriptomics), proteins in living systems (proteomics), and small-molecule components of living systems (metabolomics). They are used to yield data that can integrate toxicant-specific alterations in gene, protein, and metabolite expression with phenotypic responses of cells, tissues, and organisms.[39,40] These approaches may be especially useful to identify nongenotoxic carcinogens, define groups of substances that may have similar toxicity because they elicit similar cellular responses, and reflect the contemporary understanding that carcinogens can act through multiple modes of action and mechanisms. However, considerable developmental research is needed before these methods are routinely applied in hazard identification, risk assessment, and regulation.

CLASSIFICATION OF CARCINOGENS

In 1969, the IARC initiated its monograph program to evaluate the carcinogenic risk of chemicals to humans and to produce monographs on individual chemicals. (Information on the program can be obtained at http://monographs.iarc.fr/.) The program assembles international groups of experts to critically review and evaluate evidence on the carcinogenicity of a wide range of human exposures. Published data regarding an agent, mixture, or exposure circumstance are reviewed to determine the level of evidence for carcinogenicity in humans and experimental animals.

The criteria for sufficient evidence of carcinogenicity are quite stringent. For humans, sufficient evidence for carcinogenicity requires that "…a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence." For animals, sufficient evidence for carcinogenicity requires "…an increased incidence of malignant neoplasms in *(a)* two or more species of animals or *(b)* in two or more independent studies in one species carried out at different times or in different laboratories or under different protocols. An increased incidence of tumours in both sexes of a single species in a well-conducted study, or a single study in one species or sex, when malignant neoplasms occur to an unusual degree….can also provide sufficient evidence for carcinogenicity in animals."[41] Based on separate evaluations of carcinogenicity in humans and experimental animals, the agent, mixture, or exposure circumstance is classified into one of five groups:

Group 1—Carcinogenic to humans: This category is used when there is sufficient evidence of carcinogenicity in humans. In exceptional circumstances, an agent may be placed in this category when evidence of carcinogenicity in humans is less than sufficient, but there is sufficient evidence of carcinogenicity in animals and strong

evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

Group 2A—Probably carcinogenic to humans: This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence for carcinogenicity in animals. In some cases, an agent may be placed in this category when there is inadequate evidence of carcinogenicity in humans and strong evidence of carcinogenicity in animals and strong evidence that carcinogenesis is mediated by a mechanism that also operates in humans.

Group 2B—Possibly carcinogenic to humans: This category is used for agents for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in animals, or inadequate evidence for carcinogenicity in humans, but sufficient evidence of carcinogenicity in animals.

Group 3—Not classifiable as to its carcinogenicity in humans.

Group 4—Probably not carcinogenic to humans.

IARC treats Group 2A and 2B chemicals as if they present a carcinogenic risk to humans.

Using this classification, IARC has evaluated over 938 chemicals, industrial processes, and personal habits. It has classified 108 agents, mixtures, and exposure circumstances in Group 1, 66 in Group 2A, 248 in Group 2B, 515 in Group 3, and 1 in Group 4. Tables 17-3 and 17-4 list agents, mixtures, and exposure circumstances (such as industrial processes) in Group 1 and Group 2A for which exposures are predominantly occupational or environmental. In 2008, the IARC Monograph Program began updating reviews of all IARC Group 1 carcinogens, which will be published in Monograph 100. (The specific results of the Monograph 100 evaluations are being published in *Lancet Oncology*; the volume on sites and mechanisms will likely not be published until 2011 or 2012.) As part of this process, working groups are asked to identify *(a)* cancer sites for which there is sufficient or limited evidence in animals and humans, and *(b)* likely mechanisms of carcinogenesis for each agent. This information will be compiled in a publicly available database.[42]

The NTP also has a systematic process for evaluating human carcinogens, which classifies agents, mixtures of substances, and exposure circumstances as "known to be human carcinogens" or "reasonably anticipated to be human carcinogens." The *11th Report on Carcinogens*, issued by NTP in 2002, listed 58 substances as "known to be human carcinogens," and 188 as "reasonably anticipated to be human carcinogens."[43]

CARCINOGENS AND PUBLIC HEALTH

Cancer Risk Assessment

In contrast to the IARC and NTP processes which focus on *hazard identification*, risk assessment is a procedure for *characterizing and quantifying the amount of harm expected to result from an exposure*. This process was developed in the 1970s as regulatory agencies attempted to *(a)* set permissible levels of exposure, based on acceptable levels of risk; and *(b)* quantify the amount of benefit that would be expected from regulation at a specific level. While risk assessment is a generic process that can be applied to any risk, including nonmalignant diseases, it is discussed in this chapter because it arose in the context of cancer risk.

The four basic components of risk assessment, which were described by the National Research Council in 1983, can be applied specifically to cancer:

- *Hazard identification* involves a review of the relevant biologic and chemical information bearing on whether an agent may cause cancer.
- *Dose–response assessment* involves quantifying a dosage and evaluating its relationship to the incidence of a specific cancer.
- *Exposure assessment* involves making qualitative or quantitative estimates of the magnitude, duration, and route of exposure.
- *Risk characterization* integrates and summarizes the three preceding elements. Presented with assumptions and uncertainties, it provides an estimate of the risk to public health and a framework to define the significance of the risk.

Table 17-3. Definite Human Carcinogens with Potential for Occupational or Environmental Exposure (IARC Group 1)*

Exposures	Examples of Occurrence	Tumor Sites or Types for Which There Is Sufficient Evidence in Humans	Other Sites or Types with Limited Evidence in Humans
Aflatoxins (naturally occurring mixtures of)	Grains, peanuts (farmworkers)	Liver	
4-Aminobiphenyl	Dye and rubber industry	Bladder	
Arsenic and arsenic compounds	Insecticides, nonferrous metal smelting, mining and milling of ores containing arsenic	Lung, skin, urinary bladder.	Kidney, liver, prostate
Asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthrophyllite)	Mining and milling, insulation, shipyard workers, sheet metal workers, asbestos cement industry	Lung, mesothelioma, larynx, ovary	Colorectum, pharynx, stomach
Benzene	Chemical industry	ANLL	ALL, CLL, MM, NHL
Benzidine	Rubber and dye industries	Bladder	
Benzidine-based dyes	Coloring paper, textiles, and leather		
Benzo[a]pyrene			
Beryllium and beryllium compounds	Beryllium extraction and processing, aircraft and aerospace industries, electronics and nuclear industries	Lung	
1,3-butadiene	Chemical and rubber industries	Hematolymphatic organs	
Bis(chloromethyl) ether (BCME) and chloromethyl methyl ether (CMME)	Chemical industry	Lung	
Cadmium and cadmium compounds	Metalworking industry, batteries, soldering, coatings	Lung	Prostate, kidney
Chromium (VI) compounds	Chromate production plants, dyes and pigments, plating and engraving, chromium ferro-alloy production, stainless steel welding	Lung	Nasal cavity and paranasal sinuses
Coal tar pitches	Coal distillation	Skin, scrotum, lung, bladder	
Coal tars	Coal distillation	Skin, lung	
Dioxin (2,3,7,8-tetrachlorodibenzo-*p*-dioxin)	Herbicide production and application	All sites combined, lung	
Dyes metabolized to benzidine			
Erionite	Environmental (Turkey)	Mesothelioma	
Ethylene oxide	Sterilant in health care settings; chemical component		Lymphoid tumors (NHL, MM, CLL), breast
Formaldehyde	Production, pathologists, medical laboratory technicians, plastics, textile industry	Nasopharyngeal, leukemia	Sinonasal
Gallium arsenite	Used in high-speed semiconductors, high-power microwaves, fiberoptics		
Hepatitis B and C viruses	Health care settings	Liver	
HIV	Health care settings	Sarcoma	
Leather dust		Nasal cavity and paranasal sinuses	
4,4-methylenebis(chloroaniline)	Curing agent in polyurethane industry		
Mineral oils	Machining, jute processing	Skin	
Mustard gas (Sulfur mustard)	Production, war gas (now banned)	Lung	Larynx
2-Naphthylamine	Rubber and dye industries	Bladder	
Neutrons	Radiation workers	Unknown	

(*Continued*)

Table 17-3. Definite Human Carcinogens with Potential for Occupational or Environmental Exposure (IARC Group 1)* (Continued)

Exposures	Examples of Occurrence	Tumor Sites or Types for Which There Is Sufficient Evidence in Humans	Other Sites or Types with Limited Evidence in Humans
Nickel compounds	Nickel refining and smelting	Lung, nasal cavity, and paranasal sinuses	
Phosphorus-32, as phosphate	Phosphate mining and processing	Lung	
Plutonium-239 and decay products, as aerosols	Plutonium production workers	Lung, liver, bone	
Radon and its decay products	Indoor environments, mining	Lung	
Radionuclides, alpha-emitting, internally deposited	Fallout from nuclear explosions and reactor accidents	Lung, bone, leukemia	
Radionuclides, beta-emitting, internally deposited	Fallout from nuclear explosions and reactor accidents	Thyroid, bone	
Radium-222, 224, 226, and 228 and decay products	Uranium mining and milling	Lung	
Schistosoma hematobium infection	Farming and other outdoor work in endemic areas	Bladder	
Shale oils	Energy production	Skin	
Silica dust, crystalline, in the form of quartz or crystobalite	Hard rock mining, sandblasting, glass and porcelain manufacturing	Lung	
Solar radiation	Outdoor work	Skin	
Soots	Chimneys, furnaces	Skin, lung	
Sulfuric acid-containing strong inorganic acid mists	Metal, fertilizer, battery, and petrochemical industries	Larynx, lung, possibly nasal sinus	
Talc (with asbestiform fibers)	Talc mining, pottery manufacturing	See asbestos.	
Ortho-toluidine	Production of dyes, pigments, and rubber chemicals	Bladder	
Vinyl chloride	Plastics industry	Angiosarcoma of the liver, hepatocellular carcinoma	
X- and gamma radiation	Medical, nuclear fuel cycle	Leukemia, thyroid, breast	
Wood dust	Wood and furniture industries	Nasal cavity and paranasal sinuses, nasopharynx	
Exposure Circumstances (Industrial Processes)			
Aluminum production			
Auramine manufacturing			
Boot and shoe manufacturing and repair			
Chimney sweeping			
Coal gasification			
Coke production			
Furniture and cabinet making			
Hematite mining (with radon exposure)			
Inorganic acid mists, strong, containing sulfuric acid			
Involuntary smoking (exposure to secondhand or environmental tobacco smoke)			
Iron and steel founding			
Isopropyl alcohol manufacturing (strong acid process)			
Magenta manufacturing			
Paving and roofing with coal tar pitch			
Painter (occupational exposure as)			
Rubber industry			
Strong inorganic-acid mists containing sulfuric acid (occupational exposure to)			

*Other carcinogens, including medications (especially cancer chemotherapeutic agents, a risk for health care workers), foods, tobacco, and viruses, are classified as IARC Group 1 carcinogens, but are not listed here.

Current as of July 20, 2009. Up-to-date IARC evaluation data can be found at the IARC Web site, http://www.iarc.fr, or more specifically at the Monographs Database Web page, http://193.51.164.11/.

ALL, acute lymphocytic leukemia; ANLL, acute non-lymphocytic leukemia; CLL, chronic lymphocytic leukemia; MM, multiple myeloma; NHL, non-Hodgkin lymphoma; STS, soft tissue sarcoma.

Table 17-4. Probable Human Occupational Carcinogens (IARC Group 2A)*

Exposures	Illustrative Examples of Occurrence
Acrylamide	Polyacrylamide manufacturing
Benz[a]anthracene	Coal distillation
Captafol	Fungicide
Alpha-chlorinated toluenes	Plastics industry
4-Chloro-ortho-toluidine	Dye and chlordimeform manufacture
Creosotes	Wood preservatives
Cyclopenta[*cd*]pyrene	
Dibenz[*a,h*]anthracene	Coal distillation
Dibenzo[*a,l*]pyrene	
Diesel engine exhaust	Motor vehicles
Diethyl sulfate	Petrochemical industry
Dimethylcarbamoyl chloride	Chemical manufacturing
Dimethylhydrazine, 1,2-	Rocket propellants and fuels, boiler water treatments, chemical reactants, medicines, cancer research
Dimethyl sulfate	Former war gas, now used in chemical industry
Epichlorhydrin	Resin manufacturing, solvent
Ethyl carbamate (urethane)	
Ethylene dibromide	Fumigant, gasoline additive
Glycidol	Chemical intermediate, sterilant
Indium phosphide	
High-temperature frying, emissions from	
Lead compounds, inorganic	Lead-acid batteries, metallic alloys, radiation shielding
N-nitrosodiethylamine	Solvent
N-nitrosodimethylamine	Solvent
Nonarsenical insecticides (occupational exposures in spraying and application of)	Agriculture
Polychlorinated biphenyls	Electrical equipment
Styrene-7,8-oxide	Chemical industry
Tetrachlorethylene	Dry cleaning
Trichloroethylene	Metal degreasing
1,2,3-Trichloropropane	Pesticide; rubber manufacturing; solvent
Tris(2,3-dibromopropyl)phosphate	Flame retardant, polystyrene foam manufacturing
Ultraviolet radiation A, B, and C	Outdoor work
Vinyl bromide	Plastic industry
Vinyl fluoride	Chemical industry
Exposure Circumstances	
Art glass, glass containers, and pressed ware (manufacture of)	
Carbon electrode manufacture	
Cobalt metal with tungsten carbide	
Hairdresser or barber (occupational exposure as a)	
Petroleum refining (occupational exposures in)	
Shift work that involves circadian disruption	

*Other probable carcinogens, including medications (especially cancer chemotherapeutic agents, a risk for health care workers), infectious agents, and foods, are classified in Group 2A, but are not listed here.

Current as of July 20, 2009. Up-to-date IARC evaluation data can be found at the IARC Web site, http://www.iarc.fr, or more specifically at the Monographs Database Web page, http://193.51.164.11/.

In risk characterization, the exposure level that will lead to a particular magnitude of risk is estimated, using mathematical models. Different models, based on different biological assumptions, yield different results. Since risk assessment always involves some estimates, uncertainty factors are often used to introduce margins of safety. Physiologically based pharmacokinetic (PBPK) models have been used to *(a)* refine predictions made when extrapolating animal data to humans, and *(b)* assess the human relevance of certain animal tumors.

Risk assessment offers a quantitative approach to assessing the risk of exposures. If the aim of

public policy is to control rather than eliminate carcinogenic exposures, then risk assessment provides a framework for deciding how much exposure to permit. Risk assessment is transparent: The assumptions used are generally made explicit. However, critics point out that many of these assumptions, such as cross-species extrapolation and linear extrapolation to low doses, do not eliminate important scientific uncertainties. In addition, risk assessment is typically performed on one substance at a time, while actual exposures do not occur in isolation. Finally, risk assessment raises ethical concerns, because those who bear the risk are generally not those who benefit from production of a substance being evaluated and they are usually not well represented in quantifying and allocating risk.

Cancer Clusters

A cluster refers to an unusual aggregation of diseases, injuries, or deaths that are grouped together in time and space. Although clusters may be identified by surveillance performed by health professionals, more often suspected clusters are reported to public health agencies by concerned citizens. Responses to inquiries about perceived environmental clusters may consume substantial resources of public health agencies, yet they rarely lead to the identification of etiologic agents. Those clusters that have identified previously unrecognized carcinogens have been clusters of extremely rare diseases and/or clusters of disease in well-defined populations. Historically, the investigation of occupational cancer clusters has led to the identification of several human carcinogens. For example, the associations between vinyl chloride monomer and angiosarcoma of the liver and between bischloromethyl ether (BCME) and oat cell carcinoma of the lung were first suspected by recognizing a cluster of cases at a single company.

Occupational Cancer Clusters

Although investigations of occupational cancer clusters have sometimes led to identification of new hazards, more often concerns about clusters arise from misperceptions of normal patterns of cancer incidence or mortality in working populations. Overall, 44% of U.S. men and 37% of U.S. women will be diagnosed with cancer at some time in their lifetime;[1] thus, occurrence of multiple cancers in a specific group of workers is not uncommon. To respond quickly to concerns about cancer clusters and to monitor and detect any unusual mortality patterns, some large corporations have developed databases for surveillance of deaths among workers. Optimally, a file can be developed of all workers employed by a company in a given period of time, who can then be followed to determine vital status (if they are alive or dead) and causes of death. Surveillance systems based on death certificates alone (proportionate mortality ratio, or PMR, analyses) have also been used. Ideally, work history and exposure information should be included in surveillance databases, so that the rate and causes of death of workers with and without exposures of concern can be analyzed. However, if this is not feasible, nested case-control studies can be performed to evaluate job and exposure histories for cancers or other diseases that appear to be in excess.

When concern about a cancer cluster arises in a company with an ongoing surveillance program, it may be possible to determine fairly quickly whether the number of cases or deaths exceeds the number that would be expected. (See Chapter 24.)

In the absence of a surveillance system, investigation of a cancer cluster in the workplace involves a number of steps:

1. Obtain a list of cancer cases on which the suspicion is based, with as much work history and clinical information as possible, including the date of onset of each cancer and date of hire at the plant for each cancer patient. (Cancers diagnosed before or shortly after hire should be excluded from consideration.)
2. Determine whether cancers are primary or secondary, especially for sites such as liver and brain, where metastatic lesions are common. A suspected cluster based on a variety of common cancer types arising at expected ages is less likely to be occupationally related than a cluster of one type of cancer, especially if the latter is *(a)* at an uncommon anatomical site, *(b)* of an uncommon histological type, or *(c)* is occurring at younger ages than expected.

Similarly, a suspected cluster arising from individuals with diverse jobs and exposures is less likely to be occupationally associated than one arising among workers employed in the same department or with similar exposures. Often, occupational cancer clusters represent cases from current, former, and retired workers.

3. Estimate the number of expected cases in the population at risk, which might include all workers employed at the facility from the time it opened. The number of expected deaths from any specific cancer in a population depends on the total number of workers, when they were hired, their age at hire, and gender and race distribution. It is difficult to estimate this number accurately without conducting a full cohort mortality study.
4. Develop information about the workplace exposures at present and in the past. In those workplaces with exposures to confirmed or suspected carcinogens, there should be heightened concern.
5. Obtain advice from experts early in the process to ensure that planned investigations are designed well and will be likely to yield useful information. This process should be open, with involvement of management and nonmanagement personnel and experts who are considered to be objective and credible.

Community Cancer Clusters

Each year, state and local health departments in the United States respond to more than 1,000 inquiries about suspected cancer clusters. Most states have developed a stepwise approach to triage requests from the public, using established criteria to determine their response. Most of the inquiries about cancer clusters to state health departments are situations that are clearly not clusters and can be resolved by telephone. For the remainder, follow-up is needed, first to confirm the number of persons affected, their age, type of cancer, dates of diagnosis, and other factors, and then to compare cancer incidence in the affected population with background rates in state tumor registries.

Not all suspected cancer clusters can or should be investigated extensively. Increasingly, epidemiologic studies of the community are only conducted when the following conditions are met:

1. The observed number of cases of a specific type of cancer statistically significantly exceeds the number expected.
2. Either the type of cancer or age at onset is highly unusual.
3. The population at risk can be defined.
4. Prolonged exposures to known or suspected carcinogens at levels that exceed environmental limits can be documented.

Rigorous documentation and investigation of a cancer cluster is generally an expensive, multiyear process, complicated by anxiety and pressure to generate information quickly. For example, in 1999, what appeared to be an excessive number of children diagnosed with leukemia while living in Churchill County, Nevada, was brought to the attention of the Nevada State Health Department. After an extensive case-finding effort, it was confirmed that, between 1997 and 2002, 16 children who lived in the county at the time of, or prior to, diagnosis developed leukemia. Among the 16 children, 11 lived in the county at the time of diagnosis, whereas only 1 case in a child residing in the county would have been expected. Statistical testing indicated that the likelihood that this cluster was a random event was very small. Although the number of childhood leukemia cases was unusual, the distribution of leukemia cell types was not. The Nevada State Health Department, in collaboration with the Centers for Disease Control and Prevention (CDC) and other agencies, collected air, water, soil and dust samples from almost 80 homes and tested samples for heavy metals, pesticides, polychlorinated biphenyls (PCBs), and volatile organic compounds (VOCs). Environmental samples were also tested for radon and other radioactive elements. Levels of most contaminants measured were not elevated, compared with national referent data or existing environmental standards. None of the measured contaminants were associated with the occurrence of childhood leukemia. There also were investigations of records of historical exposures in the community and possible exposures from a nearby naval air station.

Although elevated levels of arsenic and tungsten were found in the municipal water supply, an expert panel concluded that this was not a likely explanation for the childhood leukemia cluster, because arsenic does not seem to be related to childhood leukemia and elevated levels of tungsten are found in many parts of Nevada. Continued investigation has not identified any exposures likely to be associated with increased risk of childhood leukemia.[44]

The CDC provides recommendations for local and state health departments on the management and investigation of cancer and other disease clusters reported by the public.[45] Enhanced tools for tracking data on environmental hazards are available through the National Environmental Health Tracking Program (http://www.cdc.gov/nceh/tracking/).[46] Perhaps the most important challenge for public health agencies is to communicate effectively with the public. Informed clinicians can plan an important role by helping to educate patients and their families about cancer and by contributing to public debate and decision making.

Controlling Occupational Cancer

Eliminating or reducing exposure to known or potential carcinogens is central to the prevention of occupational cancer. As described in Chapters 2 and 26, the best approaches to controlling exposure to hazardous substances in the workplace are *(a)* to eliminate exposure altogether by substitution or *(b)* to minimize exposure by engineering controls, such as process enclosure and ventilation. For example, benzene, which causes leukemia and other diseases, has been substituted by toluene and other organic solvents for many of its former uses. Exposure to vinyl chloride monomer, which causes angiosarcoma of the liver and other diseases, was greatly reduced by enclosing the processes where it had been present. Less desirable, but necessary in some settings such as hazardous work clean-up activities, is the use of personal protective equipment (PPE). However, establishment of an adequate program using PPE requires considerable expertise, training, and management commitment. Technical issues include the proper type of dermal and respiratory protection, requirements for fit-testing of respirators, training, maintenance, and monitoring of compliance. Personal protective equipment is uncomfortable to wear, may allow an exposure to hazardous substances if it malfunctions, and may even present its own hazards, such as hyperthermia from working in whole-body protective clothing. Therefore, process changes and environmental controls are almost always preferable to controlling exposure with PPE.

Other aspects of primary prevention include worker training and product labeling to increase awareness of workplace hazards, and training to minimize exposures. Well-designed environmental monitoring programs should be established in workplaces where potential carcinogens are present to ensure effective exposure control. In settings where exposure to a potential carcinogen or other hazardous substance may occur through multiple routes, such as the respiratory system and the skin, biological monitoring of exposure should be considered to ensure adequate control.

Secondary prevention by cancer screening may be warranted for populations of workers with known or suspected increased cancer risks. Screening tests for early detection of bladder cancer have been employed for workers exposed to carcinogenic aromatic amines, such as β-naphthylamine, benzidine, and 4-aminobiphenyl. The two methods most commonly used are urinalysis for microscopic hematuria and urine cytology. Hematuria is relatively sensitive in detecting both superficial and invasive bladder cancer, but its low specificity results in a high false-positive rate, requiring many invasive studies on healthy individuals. Urine cytology has good sensitivity and specificity for high-grade bladder cancer, but poor sensitivity for low-grade, papillary lesions. Early detection may not produce a survival advantage for patients whose disease is detected through such screening. However, highly effective treatment exists for both low-grade and high-grade lesions detected at an early stage. More advanced screening techniques using molecular biomarkers have been successful in predicting risk of bladder cancer in a cohort of benzidine-exposed workers.[47]

Historically, the only methods available for early detection of lung cancer were periodic chest X-rays and/or sputum cytology. Evaluation of

these approaches in the 1970s found that the combination of chest X-rays and sputum cytology tests three times a year yielded a significant increase in lung cancer detection in the study, compared to advising testing once a year. However, no significant decrease in lung cancer mortality occurred with this screening. These and other data supported for many years the recommendation that no routine surveillance for lung cancer be offered, even to high-risk populations.

Recently, studies have established that low-dose spiral computerized tomography (spiral CT) can detect smaller pulmonary abnormalities that may be malignant tumors better than conventional chest X-rays. However, whether such screening improves survival in high-risk populations is not yet known. One should recognize that many of the small nodules detected by spiral CT are not malignant and that chest surgery entails substantial risks. A population-based trial is now underway to compare spiral CT and standard chest X-rays in detecting lung cancer among 50,000 current and former smokers. Trials of spiral CT screening among asbestos-exposed individuals are also underway; however, screening in this population is complicated by a high proportion of false-positive lesions due to interstitial fibrosis and pleural thickening.[48,49]

OCCUPATIONAL AND ENVIRONMENTAL CANCER: SELECTED TOPICS

Environmental causes of cancer are highly diverse, including biological, physical (radiological), and chemical hazards. The topics covered in detail in the next sections were selected based on the magnitude of impact on human cancer to illustrate a diversity of exposures and exposure circumstances and to highlight emerging or newly recognized hazards.

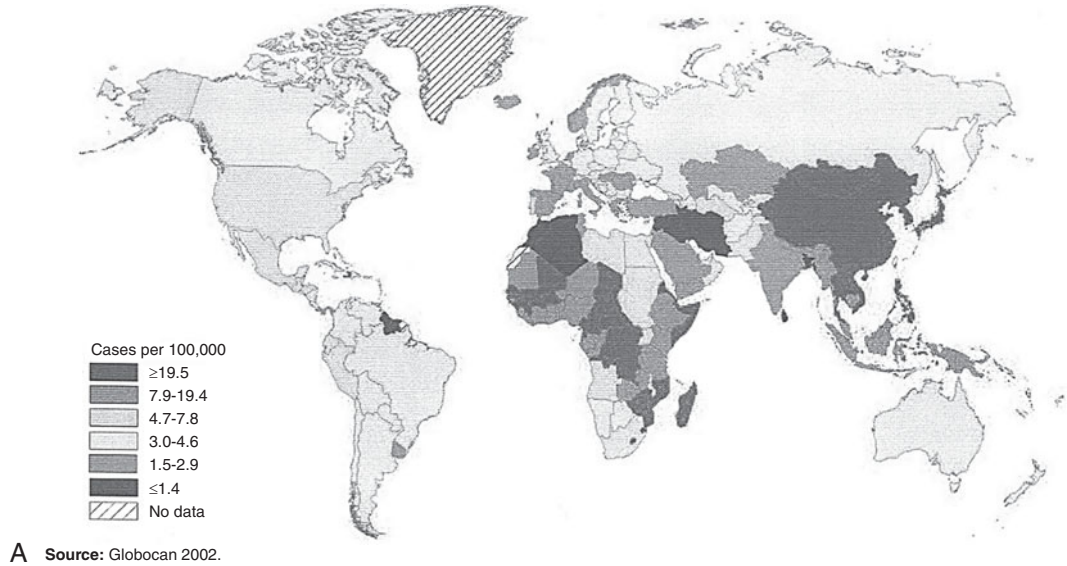
Hepatitis Infection, Aflatoxin Exposure, and Hepatocellular Carcinoma

Hepatocellular carcinoma (HCC) is the sixth most common cancer in the world. Eighty percent of cases occur in developing countries, with incidence rates in males exceeding 40 per 100,000 in some parts of eastern Asia and sub-Saharan Africa (Fig. 17-4). In the United States, the incidence rate in 2002–2006 for males was 10.2 per 100,000 and for females, 3.6 per 100,000. Incidence of HCC and mortality have been increasing in the United States since 1973. Major risk factors for HCC worldwide are chronic infection with hepatitis B virus (HBV) or hepatitis C virus (HCV) and exposure to aflatoxins as food contaminants. Hepatitis B virus accounts for 70% of HCC cases in developing countries.[50]

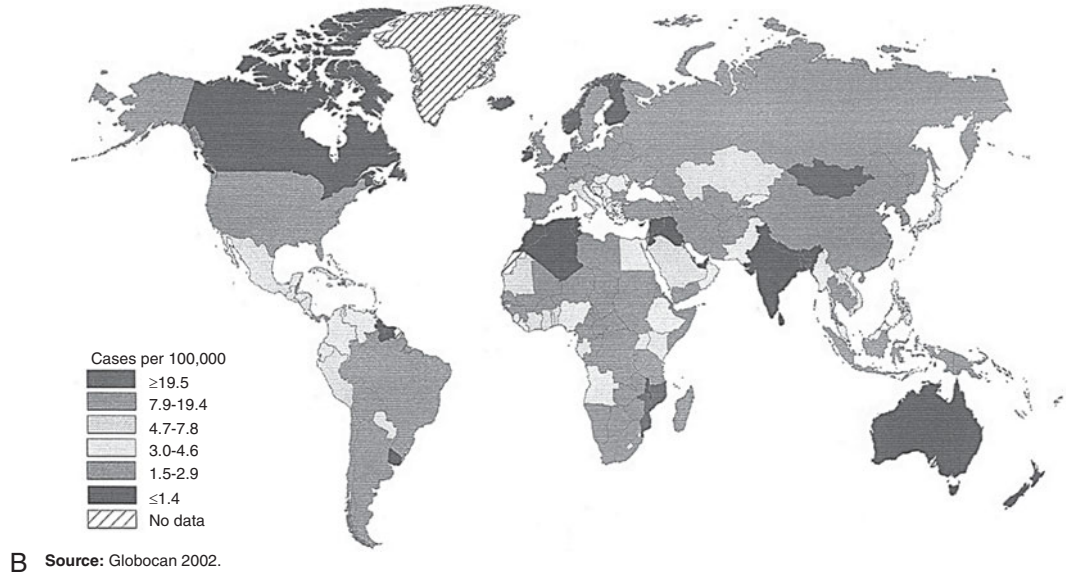
Carriage of HBV is defined as the presence of the hepatitis B surface antigen (HBsAg) in the blood for 6 months or longer. HBsAg is produced by replication of HBV virus in hepatocytes. Carriage of HBV has been associated with a 50-fold increased risk of HCC.[50] The highest HBV carrier rates occur in Africa, China, and Oceania (excluding Australia and New Zealand). The main determinant of HBV carriage is age at infection, with infection in utero or during the first 5 years of life associated with much greater rates of carriage than infection later in life. Approximately 90% of infants infected in utero or around the time of birth and 25% of those infected during the first 5 years of life become carriers, while fewer than 5% of those infected past the age of 10 years become carriers.

In contrast, about 80% of all HCV infections result in carriage. Direct transmission by blood contamination, usually though a needle, is the most important mode of transmission, resulting in a high prevalence among intravenous drug users and onset of infection mainly among teenagers and young adults in most of the world. Prevalence of HCV in Egypt has been detected in 20% of those tested, probably due to transmission of HCV during mass injection of the public for treating schistosomiasis years ago.

The risk of HBV carriage is greatly decreased by HBV vaccination within 48 hours after birth. Risk is further diminished by administration of hepatitis B immune globulin (HBIG) to infants with carrier mothers. However, since HBIG is expensive to manufacture, public health programs in Asia and Africa only use hepatitis B vaccine. Most countries in Africa include HBV vaccine in national immunization programs and about half have achieved over 80% coverage.[20]



A **Source:** Globocan 2002.



B **Source:** Globocan 2002.

Figure 17-4. International variation in age-standardized liver cancer incidence rates: (A) Males, (B) Females. (*Source*: American Cancer Society. Global cancer facts & figures 2007. Atlanta, GA: American Cancer Society, 2007, p. 21.) A color version of these maps is available at: http://ww2.cancer.org/downloads/STT/Global_Facts_and_Figures_2007_rev2.pdf

It is unlikely that a vaccine against HCV will be developed in the near future since HCV is an RNA virus that shows marked genetic heterogeneity. Preventive measures include screening for HCV in donated blood and emphasis on clean, safe needle use.

Occupational exposures to percutaneous injuries are a potential source of infections with bloodborne pathogens among health care workers (see Chapters 13 and 36). Although strategies are available to prevent infections due to sharps injuries, they have not been widely implemented in developing countries. It is estimated that in the year 2000 worldwide, 16,000 new HCV infections and 66,000 new HBV infections may have occurred due to percutaneous injuries.[51]

There are currently 360 million HBV carriers worldwide. One way to decrease their risk of HCC is by reducing contamination of food supplies with aflatoxins, which are mycotoxins

(toxic fungal metabolites) produced by *Aspergillis flavus* and *A. parasiticus*. Aflatoxin contamination of foods occurs mainly in developing countries with hot, humid climates. It is found on a variety of oilseeds and cereal crops. Often the regions with high exposure are the same as those with high HBV infection rates. Aflatoxins are potent hepatocarcinogens. Hepatitis B virus and aflatoxin have a synergistic effect on HCC risk.[50]

A variety of measures can reduce aflatoxin contamination, including pre- and postharvest crop management and dietary change. These measures are especially important in parts of the world where there is a high prevalence of HBV and HCV carriers. Treatment with interferon in patients with HCV-related cirrhosis or HBV-related chronic hepatitis may reduce the risk of HCC.

Indoor Air Pollution from Burning of Solid Fuels in Developing Countries

In developed countries, tobacco smoking is the most important risk factor for lung cancer, with the vast majority of lung cancer cases occurring among smokers. In developing countries, however, nonsmokers, frequently women, form a much larger proportion of patients with lung cancer. For example, two-thirds of lung cancer cases among women in China, India, and Mexico occur among nonsmokers. Household use of solid fuels for cooking and heating is thought to be an important cause of lung cancer among nonsmoking women in developing countries. (See Box 7-2 in Chapter 7.) Globally, almost 3 billion people rely for their primary source of domestic energy on solid fuels, including coal and biomass (fuel from wood, charcoal, crop residues, and dung). Most households in developing countries burn biomass fuels in open fireplaces or in poorly functioning earth or metal stoves. Combustion is incomplete and results in substantial emissions that contain particles, carbon monoxide, nitrous oxides, sulfur oxides (primarily from coal), formaldehyde, and polycyclic organic matter, including carcinogens such as benzo(a)pyrene. Indoor emissions from household combustion of coal increase lung cancer risk—an exposure classified as a Group 1 carcinogen by IARC. The best-documented health effects of emissions from biomass fuels are childhood acute upper respiratory infections and chronic obstructive pulmonary disease. However, studies have found increased lung cancer among people who burned wood for cooking or heating. Based on these studies and toxicologic data, IARC has classified household combustion of biomass fuel (mainly wood) as probably carcinogenic to humans (Group 2A).[52] Indoor air pollution is probably responsible annually for over 2 million excess deaths worldwide, most of which are from acute and chronic respiratory disease. Indoor air pollution is a major public health hazard for many of the world's poorest, most vulnerable people. The development and evaluation of interventions must consider many aspects of household energy supply and utilization, including affordability and sustainability.

Radon Exposure

Environmental (indoor) radon exposure is second only to cigarette smoking as a leading cause of lung cancer.[53] Radon (radon-222) is a naturally occurring decay product of radium-226, the fifth daughter of uranium-238. Two of the decay products of radon-222 emit alpha particles, which are highly effective at damaging tissues. Both uranium-238 and radium-226 are present in most soils and rocks, although concentrations vary widely. Radon exposure in homes and workplaces is largely a result of radon-contaminated gas arising from soil. Although radon is ubiquitous in indoor and outdoor air and also in the air of underground passages and mines, its concentration is increased by the presence of a rich source and by low ventilation of air in contact with the source.

Radon, unlike exposure to cigarette smoke, is naturally occurring and cannot be completely eliminated from homes and workplaces. Within buildings, radon levels are usually highest in the basement due to its proximity to the ground, from which radon-containing soil gas diffuses. Thus, people who spend much of their time in basements, at home or at work, face a greater potential for exposure. The EPA sets action levels for concentration of radon in homes, and provides information about how it is measured and how levels can be reduced, such as by installing ventilation.

Exposure to radon among miners is associated with an increase in lung cancer risk.[54] Studies have consistently demonstrated a linear increase in lung cancer risk with increasing cumulative exposure to radon. Although the increase in relative risk per unit exposure is higher for people who have never smoked than for smokers, the increase in absolute risk is much higher for smokers.

Since average radon exposures among miners have been about 10-fold greater than average indoor exposures, extrapolation of risk assessments from studies in miners to the lung cancer risk for people exposed to radon in their homes may be unreliable. To study the relationship between residential radon exposure and lung cancer risk, several case-control studies in the general population were initiated in the 1980s. Meta-analyses and collaborative analyses of these case-control studies found statistically significant exposure–response trends that are similar to *(a)* exposure–response trends extrapolated from higher dose exposures among miners, and *(b)* relative risks computed directly from studies of miners with low cumulative exposures.[55–57] An estimated 10% to 14% of lung cancer deaths in the United States have been attributed to radon (among "ever-smokers" and "never-smokers" combined).[58] Most radon-related lung cancers occur among "ever-smokers." However, an estimated 2,100 to 2,900 of the 11,000 deaths from lung cancer among nonsmokers in the United States each year are thought to be radon-related. The geologic potential for radon exposure differs throughout the United States (Fig. 17-5). Approximately one-third of radon-induced lung cancer could be prevented if homes with radon concentrations exceeding the EPA action level of 4 picocuries/L in air could reduce radon concentrations below that level.

Environmental Tobacco Smoke (Passive or Involuntary Smoking)

Involuntary smoking consists of exposure to a complex mixture of chemicals generated during

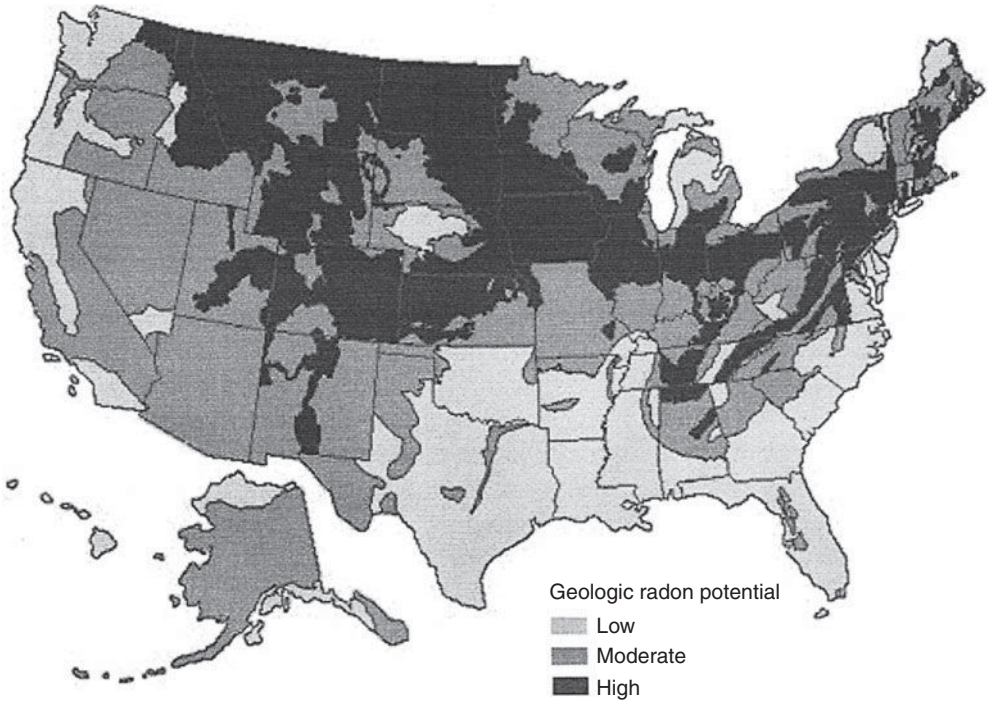


Figure 17-5. Generalized geologic radon potential of the United States. (*Source*: American Cancer Society. Global cancer facts & figures 2006. Atlanta, GA: American Cancer Society, 2006, p. 24; and U.S. Geologic Survey.)

the burning of tobacco products (see Box 7-1 in Chapter 7). It contains sidestream smoke—the material emitted from smoldering tobacco products between puffs—as well as exhaled mainstream smoke. Compounds identified in tobacco smoke include recognized carcinogens, such as 4-aminobiphenyl, arsenic, and benzo(a)pyrene. Numerous studies and meta-analyses have documented increased risk from lung cancer among nonsmokers exposed to environmental tobacco smoke (ETS) in the workplace and home. A large prospective study found that 16% of lung cancer in "never-smokers" and 24% in ex-smokers are attributable to ETS mainly due to work-related exposure.[59] The same study also found an association between exposure to ETS in childhood and increased risk of lung cancer in adulthood.

In response to the health effects associated with cigarette smoking and exposure to ETS, many countries and U.S. states have enacted smoke-free policies, including legislative and other measures to protect against harmful exposure to secondhand smoke. An IARC working group concluded that *(a)* these policies substantially decrease secondhand smoke exposure; *(b)* smoke-free workplaces decrease the prevalence of adult smoking and decrease cigarette consumption in continuing smokers; and *(c)* voluntary smoke-free home policies decrease children's secondhand smoke exposure, decrease adult smoking, and decrease youth smoking.

Shift Work and Circadian Rhythm Disruption

Long-term occupational exposure to light at night and circadian rhythm disruption appears to increase breast cancer risk. Disruption of other hormonal pathways may also lead to increased risk of cancer. An IARC working group concluded that "shift work that involves circadian disruption is probably carcinogenic to humans"[60]—a finding based on limited evidence from epidemiologic studies and sufficient evidence from animal studies. A meta-analysis found an aggregated standardized incidence ratio (SIR) for breast cancer of 1.48 (95% CI 1.31–1.46) among female night workers.[61] More than 20 animal studies have investigated the effect of constant light, dim light at night, simulated chronic jet lag, or circadian timing of carcinogens; most have shown a major increase in tumor incidence. Other studies found a tumorigenic effect of reduced nocturnal melatonin secretions or removal of the pineal gland (where melatonin is produced). Disruption of the circadian system by exposure to light at night results in alteration of sleep-activity patterns, suppression of melatonin production, and deregulation of circadian genes involved in cancer-related pathways. Inactivation of the circadian period gene, Per2, promotes tumor development in mice; in human breast and endometrial tumors, expression of PERIOD genes is inhibited. Suppressed melatonin may also result in increased levels of estrogen, thereby inducing tumors in hormone-sensitive organs, such as the breast.

Although investigation of the role of environmental lighting and circadian rhythm disruption is a relatively new area of research, it is of direct importance in the workplace since 15% to 20% of workers in the United States and Europe are engaged in shift work that involves night work. In addition, one of the defining features of the human environment for the past 120 years has been artificial electric lighting. Changes in the light-dark cycle related to artificial lighting in developed countries in the twentieth century may have contributed to increasing incidence of breast, colon, prostate, and other cancers.[62] (See Box 14-2 in Chapter 14.)

Nanoparticles

In the past decade, increasing numbers of workers have been involved with the production, use and disposal of nanoparticles, which are defined as being under 100 nm at their largest dimension. (See Box 26-2 in Chapter 26.) There are a wide variety of such particles, for which varying physicochemical features (size, shape, composition, charge, crystallinity, solubility, added functional groups, and impurities) can lead to different toxicologic potential. Because development of nanomaterials is new, most exposures occur in pilot or start-up facilities in a wide range of manufacturing and materials sectors.[63,64] Although toxicologic data involving nanoparticles are limited, studies suggest that some materials that are relatively nontoxic in larger particles are highly toxic or carcinogenic in nanoparticles.

Uses of carbon nanotubes have been rapidly increasing in electronics, optics, drug-delivery devices, protective clothing, strengthening of sports equipment, and research. Concerns about the carcinogenicity of carbon nanotubes have been raised because the shape and dimensions of the carbon fibers are similar to those of asbestos. One study has shown that carbon nanotubes cause in the abdominal mesothelium of mice pathological changes similar to those caused by asbestos. More research is needed to determine what types of carbon fibers cause the greatest risk, the extent of exposure needed to produce a carcinogenic effect, and whether exposed humans develop lung disease or mesothelioma. A precautionary approach has been advocated to decrease the possibility of health risks to exposed workers until more definitive research is completed.[65]

CASE STUDIES IN OCCUPATIONAL AND ENVIRONMENTAL CANCER

CASE 1

A computer company has a site with 4,200 employees engaged in research and development, manufacturing, sales and service, and repairs. The human resources director is interested in cancer prevention. What are the most important measures an employer can implement to prevent cancer?

Comment: The first priority of the employer is to ensure that any exposure to potential carcinogens in the workplace is minimized or eliminated. In addition to compliance with regulatory requirements, companies should ensure that appropriate experts review toxicological data on all chemicals used, select the least toxic products, and implement proper exposure controls and monitoring for all potentially toxic substances. Several substances used in the microelectronics industry have recently been evaluated by IARC. It classified gallium arsenide as a Group 1 carcinogen and indium phosphide as a Group 2A carcinogen. Substances with evidence of carcinogenicity in animals should be treated as potentially carcinogenic to humans and appropriate exposure monitoring and control should be implemented, even if not required by regulations. Engineering controls are generally preferred over PPE for exposure control. Depending on the nature and extent of chemical exposures, consideration should be given to change rooms, onsite laundering of work clothing, and other precautions to minimize potential transport of hazardous substances outside the workplace. Health and safety committees, with representation of management and nonmanagement personnel, should review workplace health and safety procedures, prioritize issues, and coordinate measures to inform and educate workers.

Employers can reduce the risk of cancer among all employees by offering health insurance benefits that include coverage for smoking cessation and all recommended types of cancer screening. If employees are or may be at increased risk of cancer due to work exposure, the possibility of offering additional cancer screening should be considered. Regardless of occupational factors, opportunities exist for cancer control and health promotion through workplace programs.

Cigarette smoking and obesity are the two most important causes of cancer in the general population in the United States. Smoke-free workplaces reduce exposure of nonsmokers to ETS, encourage smokers to decrease smoking or quit, and reduce the potential for exposure to workplace chemicals through hand-to-mouth contact while smoking. Transition to a smoke-free workplace should be accompanied by support to help smokers quit, including counseling services and pharmacotherapy. The workplace may also be used to promote healthy diets and physical activity. Provision of healthy food choices in workplace cafeterias may encourage healthy eating habits. On-site exercise facilities or subsidies for health-club memberships can encourage physical activity.

CASE 2

A 44-year-old flight attendant is concerned about breast cancer due to her occupation. Is her concern justified?

Comment: Almost all epidemiologic studies performed on flight attendants have shown a significant excess risk of breast cancer. Occupational exposures that might explain this increased risk include cosmic radiation and circadian rhythm disruption. Although radiation exposures among flight crew members are not routinely monitored, those who regularly fly long distance at high altitudes or on flight routes near the North Pole or South Pole may have higher exposure to ionizing radiation than the average U.S. radiation worker. (See Chapter 12C.) Flight attendants who work during pregnancy may exceed the International Commission on Radiation Protection (ICRP) recommended limit of 1 mSv to the conceptus during pregnancy. Many flight attendants also experience circadian rhythm disruption on flights that cross multiple time zones or occur overnight. The possibility that the increased risk of breast cancer among flight attendants is related to circadian rhythm disruption is supported by a consistent finding of increased breast cancer risk among other groups of female night workers, and studies in experimental animals documenting increased tumor incidence associated with circadian rhythm disruption. Despite increasing evidence for increased risk of breast cancer among flight attendants and the possible role of circadian rhythm disruption, it is still not certain if this association is causal, in part because of the complexity of separating the influence of occupational and nonoccupational risk factors for breast cancer.

Important breast cancer risk factors in the general population include a family history of breast cancer in a parent or sibling, nulliparity, older age at first birth, earlier age at menarche, later age at menopause, use of hormone replacement therapy, and obesity. Although many of these risk factors are not readily modifiable, women interested in reducing their breast cancer risk should be encouraged to maintain a healthy body weight, engage in regular physical activity, and minimize alcohol consumption. The American Cancer Society recommends that women of average risk begin annual clinical breast exam and mammography at age 40, and inform their physician promptly about any changes in their breasts between exams. A woman's personal risk profile for breast cancer should be determined based on family history and other factors. Additional options, including enhanced screening and chemoprophylaxis, should be offered to high-risk women who meet established criteria.

CASE 3

A 67-year-old machinist is diagnosed with rectal cancer. He asks his surgeon if his work exposure to metalworking fluids could have caused it. He wonders whether his son, who now works as a machinist, is at increased risk.

Comment: Millions of gallons of metalworking fluids are used each day in the United States for cutting, milling, drilling, stamping, and grinding. More than 1 million workers are engaged in these activities and potentially exposed to metalworking fluids by inhalation and dermal contact. Metalworking fluids are complex mixtures of chemicals that are classified into three major types: straight oils, soluble oils, and semisynthetic metalworking fluids. The identity and proportion of chemical species in these mixtures are dependent on several factors, including the manufacturer and the cooling and lubrication requirements of the machining process. In addition, several additives are used in metalworking fluids to extend their operational life span, and other substances contaminate the complex mixture during manufacturing. The three major types of metalworking fluids are further described next:

1. *Straight oils* are composed primarily of solvent-refined petroleum oils. Before the 1940s, metalworking fluids were predominantly straight oils. In 1984, untreated and mildly treated mineral oils containing polycyclic aromatic hydrocarbons (PAHs) were classified by IARC as human carcinogens. Highly refined petroleum oils (lubricant base oils) continue to be used in lower production operations and those requiring lubrication. Straight oils may contain elemental sulfur, sulfur compounds, and chlorinated compounds, such as chlorinated paraffins, some of which are carcinogenic.

2. *Soluble oils* are combinations of highly refined mineral oils (30%–85%) and emulsifiers, and they are typically diluted with water at ratios of 1 part concentrate to 5 to 40 parts water. Semi-synthetic metalworking fluids contain a lower proportion of lubricant base oils (5%–30%), a higher proportion of emulsifiers, and 30%–50% water.
3. *Synthetic oils* contain no petroleum oils and may be water-soluble or water-dispersible. They are composed of water with additives, including buffers, such as ethanolamines.

Exposures to metalworking fluids have been associated with increased risks of cancers of several sites, including stomach, esophagus, lung, prostate, brain, colon, rectum, and the hematopoietic system. The specific metalworking fluid constituents or contaminants responsible for increased risks for specific cancers have not been determined.[66]

Several studies have reported an association between exposure to metalworking fluids and rectal cancer. A comprehensive study of the health effects of exposure to metalworking fluids found a significant association between exposure to straight oils and rectal cancer, with a two-fold increased risk among workers with a cumulative exposure of over 3 mg/m^3-years. Risk was greatest for those hired before 1970, perhaps reflecting either less carcinogenicity of more modern metalworking fluids or the relatively short follow-up of workers hired after 1970.

While it is plausible that exposure to metalworking fluids may have contributed to the development of rectal cancer in this machinist, it is unclear whether his son will be at risk as a result of present-day metalworking fluid exposures. Over the past several decades, substantial changes have been made in metalworking, including changing the types of metalworking fluids used; reducing potentially carcinogenic contaminants, such as nitrosamine precursors; and reducing exposure concentrations through process changes and engineering controls—all of which may decrease the risk of rectal cancer among more recent workers. Given that the specific constituent of metalworking fluids responsible for the increased risk of rectal cancer is unknown, increased risk of rectal cancer for workers who began exposure recently cannot be ruled out. Metalworking fluids have been nominated for toxicologic testing by the National Toxicology Program, with the goal of better understanding the carcinogenicity of formulations currently used. The son's individual risk of developing colorectal cancer also depends on whether there have been other family members diagnosed with colorectal cancer at a young age and whether he has certain specific diseases that increase colorectal cancer risk. Colorectal cancer screening can both detect cancer at an early stage and prevent it by removing adenatomatous polyps. Screening for colorectal cancer is recommended for the general population starting at age 50, with earlier and enhanced screening recommended for those at high risk.

CASE 4

A study found that 10 current and former workers at a chemical manufacturing plant with 5,000 workers have developed brain cancer over the past 10 years. The workers' union requests an investigation and asks whether an exposure at our plant is causing brain cancer and what should be done.

Additional investigation identifies two additional cases, for a total of 12. No medical records are available for three of the 12. Five appear to have died of brain cancer, and four appear to have been diagnosed with benign brain tumors. While case confirmation continues, another worker is diagnosed with brain cancer. The local newspaper interviews this worker's wife and writes a story suggesting a company conspiracy to cover up a brain cancer epidemic. Concern begins to center around a plant department with historical exposure to nitrosamines and many other chemicals.

Union and management agree to bring in outside consultants to review the case and exposure information. The consultants state that it is unclear whether the observed cases represent an excess. The number of brain cancer deaths does not greatly exceed the number that might be expected from studies of other cohorts of workers. However, since

ascertainment of cases was not done systematically, it is not known if all brain cancer cases have been identified. The consultants agree that, while brain cancer clusters have been reported in the chemical industry, limited evidence exists for an association between specific chemical exposures and brain cancer. They further agree that, given the wide variety of chemical processes present at the plant, retrospective exposure assessment would be very time-consuming and expensive.

Comment: The best way to approach a possible brain cancer excess at the workplace is to conduct a cohort mortality study. In this situation, such a study could be done quickly if it was confined to employees working there on or after January 1, 1978. Since that date, employees' work histories and demographic information have been computerized and they can be linked with the National Death Index, which can provide accurate information on deaths throughout the United States since 1978. The consultants request a comprehensive industrial hygiene survey, which will be performed by an independent contractor to ensure that current exposure protection is adequate.

The consultants agree to reconvene after the study is completed to review its results. If the study confirms excess mortality from brain cancer, further studies may be indicated to identify high-risk departments and processes. If not, the consultants would recommend that the company continue to conduct mortality surveillance for this cohort of workers.

RESEARCH AND POLICY PRORITIES

A major challenge is to develop better data to evaluate the hazards of chemicals, mixtures, and exposure circumstances in IARC Groups 2A and 2B, and others with some evidence of carcinogenicity in animal or human studies. Historically, many of the recognized human carcinogens were identified in occupational cohorts where very high exposures resulted in very high relative risks. The changing nature of the workplace and increasing complexity of exposures have made such occupational epidemiology studies more difficult. As a result of regulatory and voluntary controls, exposure levels and attendant risks are much lower than in the past. Many exposures are mixtures, and many occupations involve exposure to an ever-changing and diverse array of substances. These changes create the need for more sensitive measures to detect cancer risks in occupational populations. For example, studies may need to incorporate quantitative estimates of risk for multiple exposure agents, examine possible interaction between occupational and nonoccupational exposures, and consider the use of biomarkers to better define intermediate markers related to exposure and biologic effects.

The potential for biomarkers to play a role in improving understanding of human cancer risks has been recognized, but not fully exploited. Biomarkers can play an important role in understanding a number of stages in the process through which exogenous exposures result in cancer, including internal dose, biologically effective dose, early biologic effect, altered structure and function, premalignant changes, and clinical disease. Biomarker data have been used by IARC to support classification of ethylene oxide and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (dioxin) as Group 1 carcinogens, in the absence of definitive evidence of increases in cancer incidence or mortality from epidemiologic studies. Incorporation of biomarkers of genetic susceptibility may play a role in studies of potential occupational and environmental carcinogens. However, current scientific knowledge about genetic susceptibility to environmental and occupational cancer is too limited for clinical application.

There is much scientific controversy and debate about the extrapolation of effects in animals to humans, especially concerning *(a)* low levels of human exposure, and *(b)* the strength of evidence that certain mechanisms of action in rodents are not applicable in humans. This controversy and debate, while highly technical, have practical consequences for the classification of, and exposure control for, chemicals of public health importance. Research conducted to clarify these issues must be objective and supported, at least in part, by public and private institutions with no financial interest in the outcome. In addition, scientific and government agencies considering these issues should ensure representation

from all perspectives, including labor, management, and affected communities.

The Precautionary Principle is aimed at avoiding possible harm associated with suspected, but not definite, environmental risks. In the face of scientific uncertainty about potential carcinogens, it has been advanced to provide a framework within which to consider public health actions. It provides justification for acting in the face of uncertain knowledge to address potential risks from specific environmental exposures. The Precautionary Principle states that appropriate public health action should be taken in response to limited evidence of likely and substantial harm, when that evidence is plausible and credible. The burden of proof is shifted from demonstrating the presence of risk to demonstrating its absence.

Clinicians' roles in confronting occupational cancer vary. They should maintain a high index of suspicion of occupational causes of cancer when treating patients. Clinicians should work to identify past exposures, using the patient's knowledge, toxicological resources, consultants, and other sources of information. In case of ongoing exposures, clinicians should help to reduce or eliminate hazardous exposure. Finally, clinicians should educate patients, employee and employer groups, and communities about the hazards of carcinogenic exposures and ways to prevent them.

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18

Respiratory Disorders

Amy M. Ahasic and David C. Christiani

CASE 1

A 60-year-old man, who had been a sandblaster for 23 years, was hospitalized for the third time in 4 months for shortness of breath. Three years before, he began having shortness of breath and a fast heart rate with moderate exertion. These symptoms increased over the next several months. He was evaluated by the company physician, who told him that he had "bad lungs" but gave him no treatment.

Two years before, his shortness of breath on exertion worsened, and he was hospitalized. Arterial blood gases were PaO_2 87 mm Hg and $PaCO_2$ 31 mm Hg on room air. A chest X-ray showed multiple interstitial nodules. Forced vital capacity (FVC) was 73% of predicted. Diffusing capacity was normal. Tuberculosis smear, culture, and cytology of bronchial washings were all negative. He was sent home without therapy and was told not to return to work. He has not worked since.

Seven months ago, he developed a cough, occasionally productive of thin, clear-to-grayish sputum, and then was admitted to the hospital three more times for increasing shortness of breath. Since the last hospitalization 1 month ago, he has been continuously dependent on oxygen and has remained in bed most of the time.

The patient had smoked one pack of cigarettes per day for 5 years, until quitting 20 years ago. He had no history of asthma, pneumonia, surgery, or allergies.

For 23 years, he operated a sandblasting machine in a basement room (20 by 40 feet). Dust escaped continuously through crevices of the sandblasting unit, and much dust escaped every time he opened the door of the unit to install and remove a piece to be blasted. The windows were closed. An exhaust fan in a wall did not seem to remove any dust. A room fan, installed to circulate the air in the room, often did not work. The patient wore a helmet with a cloth apron that covered his shoulders. When the air in the room was very dusty, he breathed from a tank of compressed air that he wore.

Physical examination revealed a thin man in moderate respiratory distress, sitting hunched over, gasping for breath, with grunting expirations. Pulse was 110, respiratory rate 40, blood pressure 110/80, and temperature 98°F. Lung and heart examinations were normal, except for a systolic ejection murmur and an increased second heart sound over the pulmonic valve area. He had clubbed fingernails and cyanosis. Arterial blood gases on room air revealed significant hypoxemia (PaO_2 39 mm Hg, $PaCO_2$ 38 mm Hg). A chest X-ray showed diffuse, interstitial, small, rounded densities throughout both lung fields and hilar fullness. These densities were judged to be "q"-sized with a 2/2 profusion in all lung fields, using the International Labor Organization (ILO) nomenclature for chest radiographs.[1] The diagnosis of silicosis was made. He remained completely disabled and died 3 months later.*

* Case courtesy of Stephen Hessl, MD, Daniel Hryhorczuk, MD, and Peter Orris, MD, Section of Occupational Medicine, Cook County Hospital, Chicago, Illinois.

This case is a severe case of pneumoconiosis, an occupational respiratory disease. Workplace exposure responsible for such chronic disabling lung disease occurs gradually over long periods. At first, exposures do not result in acute symptoms, but, once symptoms do appear, often little can be done beyond palliative treatment. Unless discovered very early in their course, most work-related respiratory diseases are not curable. Disease prevention is therefore critically important.

Occupational lung disease was observed in ancient times—in the writings of Hippocrates and in pictographs from Egypt. Some of these chronic diseases are still present.

Estimates of the prevalence and incidence of occupational respiratory disease suggest that only a small fraction of chronic occupational respiratory disease is correctly identified as associated with work.

Pneumoconioses and occupational asthma are work-related respiratory diseases that often are not correctly diagnosed. For example, approximately 5% of U.S. residents have physician-diagnosed asthma, but a much larger proportion of people *report* either asthma or wheezing, cough, or other symptoms of bronchial hyperresponsiveness. Physicians who see workers who report wheezing should diagnose the problem and determine whether it is related to work.

AMBIENT AIR POLLUTION

Ambient air pollution has contributed to pulmonary disease since the first use of coal in the fourteenth century. Ambient air pollution significantly increased during the Industrial Revolution in the 1800s in developed countries. Developing countries are now experiencing the adverse health effects of ambient air pollution due largely to increasing dependence on automobiles. Since the early 1990s, many prospective studies have confirmed an association between ambient air pollution and mortality.[2,3] A recent study, for example, found associations between chronic exposure to both ozone and particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and mortality, with ozone contributing specifically to mortality from respiratory disease.[4] (See Chapter 6.)

Ambient air pollution causes a wide range of adverse health outcomes, as demonstrated by acute and chronic morbidity and mortality during and after the serious pollution episodes in Donora, Pennsylvania, in 1948, in London in 1952; and after the World Trade Center attack in New York in 2001.

The public health impact of air pollution on children has been substantial.[5] Both ambient and indoor air pollution increases *(a)* the incidence of acute respiratory hospital admissions in children, *(b)* school absences, and *(c)* medication use in children with asthma (Box 18-1), and decreases peak flow rates in otherwise normal children.

EVALUATION OF INDIVIDUALS

Evaluations of pulmonary response to occupational and environmental exposures should include the following: *(a)* a complete history, including occupational and environmental exposures, tobacco use, and respiratory symptoms (Chapter 2); *(b)* a physical examination, with special attention to breath sounds; *(c)* a chest X-ray, with attention to parenchymal and pleural opacities; and *(d)* pulmonary function tests.

History

Review of symptoms should include questions on chronic cough, chronic sputum production, shortness of breath (dyspnea), wheezing unrelated to respiratory infections, chest tightness, and chest pain. Shortness of breath and cough often occur at night. In occupational asthma and pulmonary edema, symptoms may peak 8 to 16 hours after exposure. Understanding symptom periodicity and timing is important. Respiratory symptoms during the work week that improve on weekends or holidays strongly suggest an occupational disease. Recognizing the temporal relationships of symptoms with nonoccupational exposures may be more difficult, since these exposures may be occurring daily in the home environment. Use of the American Thoracic Society (ATS) Respiratory Symptom Questionnaire[6] has been helpful in systematically obtaining information on respiratory symptoms.

Box 18-1. Childhood Asthma

Asthma, a leading cause of chronic childhood illness, is a major cause of childhood disability and lost days from school. In the United States, childhood asthma prevalence more than doubled between 1980 and the mid-1990s.[1] Although its prevalence appears to have plateaued since then, health care utilization for asthma, especially office visits to physicians, has continued to increase. Data show that:

- About 9% of children (6.5 million) have asthma.
- About 13% of children (9 million) have been diagnosed with asthma at some time.
- About 3% of ambulatory visits and emergency-department visits by children are for asthma.

Black, Native American, and Hispanic children have higher rates of asthma than white children. Prevalence among Puerto Rican children is more than double that among non-Hispanic white children.[1] Hospitalizations, emergency-department visits, and deaths due to asthma are much higher among black children than among any other group of children. Black children have a six-fold higher death rate from asthma than white children, although the rate of ambulatory-care visits by black children is lower than that for children of other racial groups.[1] Overall childhood asthma mortality has declined since 1999, but the asthma mortality among black children has not declined, thus showing an increasing racial disparity.[1]

Some of these disparities arise from differences in environmental risk factors. Early exposure to some allergens may increase risk of asthma. However, early exposure to animal dander, such as among children living on farms, may decrease risk of asthma. Exposure of inner-city children to dust mites and cockroach antigen appears to increase risk.[2] History of severe viral respiratory illness, especially with respiratory syncytial virus (RSV), is another known risk factor, possibly due to damage caused by the virus. However, asthmatic children seem to be predisposed to severe viral respiratory illnesses, so RSV may be unmasking preexisting disease.

Environmental tobacco smoke (see Box 7-1 in Chapter 7) and air pollution exacerbate, and may also possibly cause, childhood asthma. Several criteria pollutants have been associated with increasing respiratory symptoms and decreasing pulmonary function in children, and prenatal exposure to some criteria pollutants negatively affects postnatal lung function. Specifically, higher prenatal exposures to nitrous oxide, carbon monoxide, and PM_{10} have been associated with decreased spirometric values postnatally; higher childhood exposures to nitrous oxide, carbon monoxide, ozone, and PM_{10} have had similar negative effects.[3] Concentrations of sulfur dioxide have also been positively correlated to infant death rates.[4]

Genetic or other mechanisms may also contribute to asthma risk. Atopy is a risk factor for asthma; some children have eczema or allergic rhinitis before they develop asthma. Atopy tends to be inherited; family history of atopy and/or asthma has also been used in predicting a child's risk of asthma.

The natural history of childhood asthma is variable. Some children have resolution of lung function abnormalities without recurrence in adulthood. Still others have persistent asthma without remission. There is some evidence that atopy and a low FEV_1/FVC ratio at age 18 are independent predictors of relapse.[5] Severe or inadequately treated asthma in childhood can result in lifelong impairment in lung function, primarily due to airway remodeling, leading to fixed obstruction. Therefore, asthma exacerbated by work exposures may sometimes be partially attributed to asthma during childhood.

In caring for adults and children with asthma, one should take a thorough occupational and environmental history in order to identify and then reduce exposures that trigger or exacerbate symptoms. Questions should focus on type of housing, presence of smokers in the household, infestations with insects or rodents, mold or musty odors, visible water damage, pets, and other family members affected with asthma. Addressing these triggers is critical to maintaining the respiratory health of asthmatic children. Educating parents on how to reduce their children's exposures to allergens can help improve prognosis. The National Environmental Education Foundation and other organizations and government agencies offer free educational materials for caregivers and others.[6]

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Physical Examination

The most remarkable finding in most people with occupational and environmental respiratory disease is the relative absence of physical signs. However, auscultation can reveal important diagnostic clues. Fine rales may be heard at the lung bases, often at end-inspiration; they are more common in asbestosis than in other interstitial lung diseases. Hearing wheezes and understanding their temporal relationship to exposure may help in evaluating suspected cases of occupational and environmental asthma. A pleural

rub may be heard as a result of a pleural reaction caused by exposure to asbestos. Clubbing of the digits, a nonspecific sign, may be seen rarely in advanced lung diseases, including asbestosis, bronchial carcinoma, and idiopathic pulmonary fibrosis. Signs of right ventricular heart failure, such as pedal edema, may indicate severe lung disease.

Chest X-ray

A chest X-ray should be taken when malignancy, pneumoconiosis, hypersensitivity pneumonitis, or other lung disorders are suspected. For cases of occupational airways disease, such as work-related asthma, chest X-rays are rarely helpful. For suspected cases of pneumoconiosis, a trained "B reader" should interpret the X-ray, using the International Labor Organization (ILO) system for pneumoconiosis (Fig. 18-1).[1] The ILO system permits semiquantitative interpretation of chest X-rays to identify early evidence and progression of parenchymal and pleural disease. It focuses on size, shape, concentration (profusion), and distribution of small parenchymal opacities as well as distribution and extent of pleural thickening or calcification. Rounded opacities in the upper lung fields are usually associated with silicosis, whereas linear (irregular) opacities in the lower lung fields are usually associated with asbestosis (Fig. 18-2). Deviations from these patterns are common; for example, silicosis and coal workers' pneumoconiosis (CWP) can be associated with irregular opacities. Moreover, workers exposed to mixed dusts, such as silica and asbestos, can present with mixed rounded and irregular opacities in any or all lung fields. The ILO system has the advantage of using a standardized set of comparison radiographic films, which can be used to classify X-rays at one point in time or to follow an individual or a population for change over time.

Although chest X-rays present evidence of abnormality, they do not provide information on disability or impairment and do not necessarily correlate well with pulmonary function test findings. A person with severe obstructive disease may show little evidence of it on a chest X-ray. In contrast, a person exposed chronically to iron oxide or tin oxide may show a dramatically abnormal chest X-ray, but little if any pulmonary inflammatory reaction or lung function abnormality (Fig. 18-3). Additionally, conventional chest X-rays may prove to be insensitive to subtle lung abnormalities. Although the ILO system was developed for epidemiologic studies and not for clinical evaluation of individuals, it may be an important facet to the posteroanterior (PA) chest X-ray interpretation by describing types of opacities and their distribution, both of which may be characteristic of a specific pneumoconiosis.

The advent of high-resolution computed tomography (HRCT) scanning has improved dramatically physicians' ability to detect and classify subtle lung diseases that are not or only barely visible on conventional chest radiographs (Fig. 18-4). While there is currently no standardized schema for the use of computed tomography in classification of pneumonconioses, the National Institute for Occupational Safety and Health (NIOSH) has published proceedings of a workshop on the use of digital chest X-rays, since digitized images are now the standard in most hospitals and clinics.[7]

Pulmonary Function Tests

Pulmonary function tests, required for medical surveillance by some Occupational Safety and Health Administration (OSHA) standards, are used commonly and are reliable, reproducible, and easy to perform. In a well-equipped pulmonary function laboratory, spirometry, lung volume determinations, gas exchange analyses, and exercise testing can be performed with relative ease. In a physician's office, only spirometry is readily and inexpensively performed; it does, however, provide much useful information. Most cases of respiratory disease yield abnormal test results or accelerated decline in pulmonary function within the "normal" ranges before onset of clinical symptoms, especially if patients are followed at regular 1- to 3-year intervals. Although pulmonary function tests may demonstrate several patterns of abnormalities, one cannot rely on them alone to determine etiology. Hospital-based tests, such as lung volume determinations, gas exchange analyses, exercise tests, and bronchial challenge tests, can help in refining a diagnosis.

Basic tests of pulmonary function can be obtained with a simple portable spirometer.

I. Size and shape of small opacities

ROUNDED OPACITIES		IRREGULAR OPACITIES	
p	≤ 1.5 mm diameter	s	Fine linear opacities > 1.5 mm width
q	1.6–3.0 mm diameter	t	Medium opacities 1.6–3.0 mm width
r	3.1–10.0 mm diameter	u	Coarse, blotchy opacities 3.1–10.0 mm width

* Size recorded by two letters to distinguish single type from mixed type. For example, q/q if only q opacities are present, but q/t if q opacities predominate but t are also present.

II. Concentration (profusion) and distribution

SMALL OPACITIES

Major categories		Minor divisions			Distribution*	
0	Small opacities absent or less than category 1 normal lung markings visible	0/-	0/0	0/1	RU	LU
1	Small opacities present but few normal lung markings usually visible	1/0	1/1	1/2	RM	LM
2	Small opacities numerous normal lung markings partially obscured	2/1	2/2	2/3	RL	LL
3	Small opacities very numerous normal lung markings totally obscured	3/2	3/3	3/+		

LARGE OPACITIES

A	One or more opacities with greatest summed diameter 1–5 cm
B	One or more opacities larger or more than category A. Total area < Equivalent of right upper zone
C	One or more opacities. total area exceeds equivalent of right upper zone

* Recorded by dividing lungs into 3 regions per side and checking all regions containing the designated small opacities

III. Pleural thickening

WIDTH*	EXTENT*	CALCIFICATION*
a Maximum Up to 5 mm	1 Up to 1/4 lateral wall	1 One or several regions summed diameter ≤ 2 cm
b Maximum 5–10 mm	2 1/4–1/2 lateral wall	2 One or several regions summed diameter 2–10 cm
c Maximum > 10 mm	3 Exceeds 1/2 lateral wall	3 One or several regions summed diameter > 10 cm

* Width estimated only if seen in profile. Extent estimated as maximum length of thickening (profile or face on). Calcification site (diaphragm, wall, other) and extent are noted separately for two sides.

Figure 18-1. Schematic of International Labor Organization classification system for chest X-rays. In addition to these scores, the reader is guided in scoring technical quality of the X-ray (good, acceptable, poor, unacceptable) and in identifying other relevant features (e.g., bullae, cancer, abnormal cardiac size, emphysema, fractured rib, pneumothorax, tuberculosis).

Test results are derived from the volume-time and flow-volume curves (Fig. 18-5). Although several different measurements can be derived from these curves, the simplest and the most generally useful ones for evaluating occupational or environmental respiratory disease are *(a)* forced vital capacity (FVC), *(b)* forced expiratory volume in the first second of forced expiration (FEV_1), and *(c)* the ratio of FEV_1 to FVC. The peak expiratory flow rate (PEF or PEFR) is a measurement that is most useful when tested serially over time, such as throughout the work day or work week. While PEF can be determined by spirometry, workers can also measure and record their own PEFs using a simple handheld peak flow meter (see section on "Work-Related Asthma" later in this chapter). A simple scheme for the interpretation of spirometric measurements is shown in Table 18-1. Results are compared with predicted values based on gender,

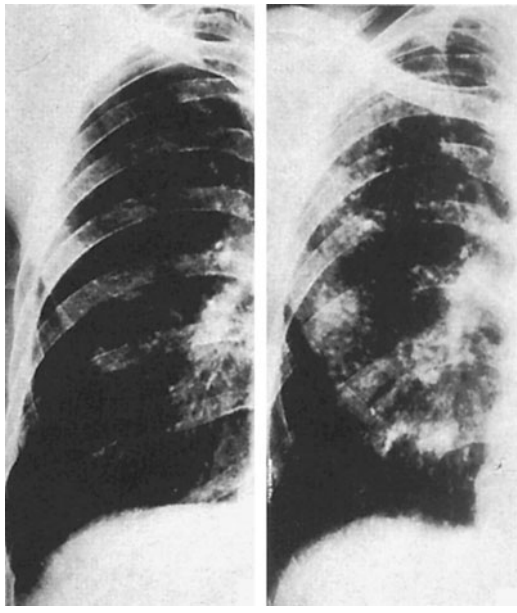

Figure 18-2. Progression of discrete nodules of silicosis over 10 years in a slate quarry worker. (*Source*: Parkes WR. Occupational lung disorders (3rd ed.). London: Butterworths, 1994.)

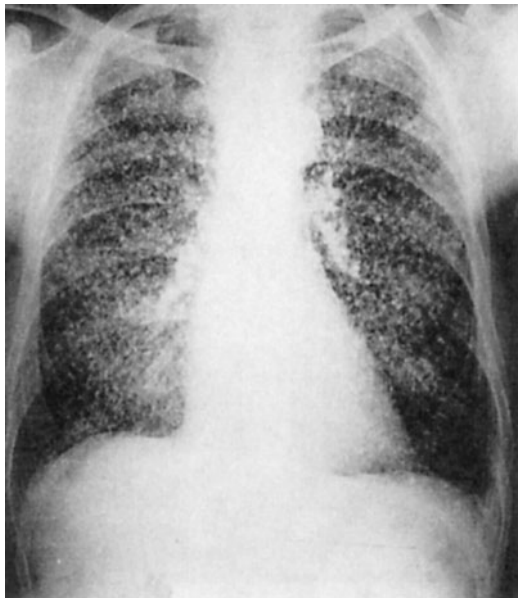

Figure 18-3. Chest X-ray demonstrating stannosis, the benign pneumoconiosis due to the inhalation of tin oxide, in a man who worked as a furnace charger in a smelting works for 42 years. (*Source*: Parkes WR. Occupational lung disorders (3rd ed.). London: Butterworths, 1994.)

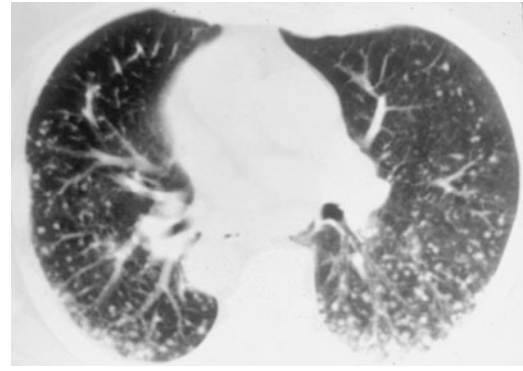

Figure 18-4. High-resolution computed tomogram (HRCT) of a 55-year-old construction worker diagnosed with silicosis. (*Source*: Massachusetts Medical Society. Weekly clinicopathological exercises. New England Journal of Medicine 1995; 333: 20.)

age, and height, and derived from a normal population of nonsmoking adults. Results are then expressed as a percent predicted of the expected value.

Criteria for the proper performance and evaluation of spirometry are based on ATS recommendations.[8-11] Many types of equipment are marketed to provide these tests, yet several have been inadequately standardized. The ATS has provided guidelines on the standardization of spirometry, including information on instrument reliability and test performance.[8,9]

The pneumoconioses silicosis and asbestosis are considered *restrictive* diseases because they result in reduction in total lung capacity. In the absence of significant airways disease, flow rates are maintained and may even be above normal because of decreased lung compliance with increased elastic recoil. Coal workers' pneumoconiosis (CWP), in contrast, is more often an *obstructive* disease, with decreased airflow and normal or increased lung volumes. Occupational asthma is also considered an *obstructive* disease, causing obstruction of airflow without reduction in lung volume. With multiple environmental exposures (including tobacco smoke), a *mixed restrictive-obstructive* disease is frequently present. In addition, some mineral dusts, such as asbestos and coal dust, have been shown to cause abnormalities in both the airways and the interstitium.

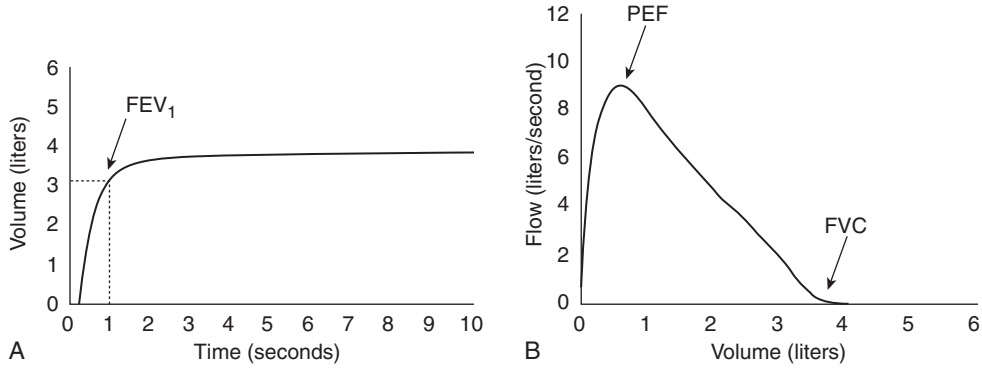

Figure 18-5. Normal spirogram. (A) Volume-time curve and (B) Flow-volume curve. FEV1, forced expiratory volume in the first second; FVC, forced vital capacity; PEF, peak expiratory flow. (*Source*: Adapted from Townsend MC. ACOEM position statement: Spirometry in the occupational setting. American College of Occupational and Environmental Medicine. Journal of Occupational and Environmental Medicine 2000; 42: 228-245.)

Table 18-1. Spirometry Interpretation

	Percentage Predicted*			
Type of Response	FEV_1	FVC	FEV_1/FVC %	Response to Inhaled Bronchodilators
Normal	≥80%	≥80%	≥75%	—
Obstructive	<80%	≥80%†	<75%	±
Restrictive	≥80%	<80%	≥75%	—
Mixed	<80%	<80%	<75%	±

*Predicted FEV_1 and FVC based on Knudson RJ, Lebowitz MD, Holberg CJ, et al. Changes in the normal maximal expiratory flow volume curve with growth and aging. American Review of Respiratory Disease 1983; 127: 725.

†Severe obstruction can result in reduction of FVC also.

FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity.

EVALUATION OF GROUPS

It may not be until a group of individuals is evaluated that a respiratory disease can be associated with work or the ambient environment. For instance, an occupational physician in Missouri recognized a group of eight workers within the community who had developed fixed obstructive lung disease after working at a microwave popcorn plant.[12] He reported this finding to the local health department, which led to an intensive investigation and recognition of a new occupational respiratory disease—now named popcorn worker's lung disease, a major current focus in occupational medicine and a NIOSH initiative. (See section on "Occupational Airways Disease.")

Comparisons with baselines should be performed whenever possible to permit evaluation of change over time in individuals or a group compared to a measured, rather than a predicted, value. Decrements in lung function, development of respiratory symptoms, and recognition of chest X-ray abnormalities are often far more significant when internally compared to previous examinations, rather than externally compared to other individuals or groups. Any worker who may be exposed in the future to respiratory hazards at work should have a baseline pulmonary function test before potential exposure.

The major types of respiratory response to external agents are summarized in Tables 18-2 and 18-3. Occupational lung cancer is discussed in Chapter 17, and work-related infectious diseases of the respiratory tract are discussed in Chapter 13.

Table 18-2. Major Types of Occupational Pulmonary Disease

Pathologic Function Process	Example of Disease or Exposure	Clinical History	Physical Examination	Chest X-ray	Pulmonary Function Pattern
Fibrosis	Silicosis	Dyspnea on exertion, cyanosis, shortness of breath	Clubbing	Nodules	Restrictive or mixed obstructive and restrictive
	Asbestosis	Dyspnea on exertion, cyanosis, shortness of breath	Clubbing, rales	Linear opacities, pleural plaques, and calcifications	DLCO normal or decreased
Reversible airway obstruction (mucus plugging, asthma)	Byssinosis, isocyanate asthma, irritant asthma (RADS)	Cough, chest tightness, shortness of breath, asthma attacks	↑Respiratory rate, wheeze	Usually normal	Normal or obstructive with bronchodilator improvement Normal or high DLCO
Emphysema	Cadmium poisoning (chronic)	Cough, sputum, dyspnea	↑Respiratory rate,↑ expiratory phase	Hyperaeration, bullae	Obstructive low DLCO
Granulomatous lung disease	Beryllium disease	Cough, weight loss, shortness of breath	↑Respiratory rate	Small nodules	Usually restrictive with low DLCO
Bronchiolitis obliterans	Flavoring or flavoring ingredients	Cough, chest tightness, shortness of breath, fixed airway obstruction	↑Respiratory rate, ↑ expiratory phase	Usually normal	Obstructive without bronchodilator improvement DLCO normal
Pulmonary edema	Smoke inhalation (firefighter or house fire victim)	Frothy, bloody sputum production	Coarse, bubbly rales	Diffuse air space opacity	Usually restrictive with decreased DLCO Hypoxemia at rest

DLCO, Diffusing capacity of the lung for carbon monoxide; RADS, Reactive airways dysfunction syndrome.

ACUTE IRRITANT RESPONSES

Irritation in the upper respiratory tract is frequently associated with symptoms due to regional inflammation. Nasal and paranasal sinus irritation can cause congestion that may result in severe frontal headaches, nasal obstruction, runny nose, sneezing, and nosebleeds. Throat inflammation is commonly reported as a dry cough. Laryngeal inflammation can cause hoarseness and, if severe, may result in laryngeal spasms associated with glottal edema, severe anxiety, shortness of breath, and cyanosis.

In the lower airways, in contrast, acute symptoms are often due to bronchospasm. In asthma, there is also thickening of the basement membrane, mucosal edema and infiltration with inflammatory cells, mucus hypersecretion and plugging from proliferation of goblet cells, and increased presence of smooth muscle at preterminal bronchioles.

Occupational asthma is being recognized more frequently. Precipitating agents number over 300 and include industrial cleaning products, such as glutaraldehyde and bleach; soldering fluxes; and epoxy resins (Table 18-4). In addition, many irritant substances not usually associated with asthma can produce bronchial hyperreactivity when high levels of exposure have occurred. Single high-dose exposure to irritants such as ammonia or chlorine can result in nonspecific bronchial hyperreactivity, referred to as reactive airways dysfunction syndrome (RADS) or irritant-induced asthma, which may persist for months to years or may never fully resolve. It is not clear if chronic low-dose irritant exposures can also *cause* RADS or work-related

Table 18-3. Common Environmental Pollutants with Respiratory Effects

Pollutant	Common Sources	Illustrative Health Effects
Sulfur oxides	Coal and oil power plants	Throat irritation
	Oil refineries, smelters	Exacerbation of asthma, chronic bronchitis, and other respiratory illnesses with significant airflow obstruction
	Stoves burning wood, coal, kerosene	
	Industrial chemical manufacture	
Particulates	Motor vehicle exhaust	Increased susceptibility to lung infections
	Fossil-fuel power plants	Exacerbation of asthma, chronic bronchitis, and other respiratory illnesses with significant airflow obstruction
	Heavy construction	
	Natural sources, such as volcanoes, bushfires, windblown dust, and oceans	
Oxides of nitrogen (NO_x)	Motor vehicle exhaust	Throat irritation
	Fossil-fuel power plants	Lung injury
	Oil refineries	Exacerbation of asthma and chronic obstructive pulmonary disease
		Increased susceptibility to lung infections
Ozone (O_3)	Motor vehicle exhaust	Same as NO_x
	Ozone generators	
	Aircraft cabins	
	Power plants	
Carbon monoxide (CO)	Motor vehicle exhaust	Hypoxia leading to heart and nervous system damage, death
	Fossil-fuel burning	
	Kerosene space heaters	
	Incinerators	
	Industrial equipment	
Polycyclic aromatic hydrocarbons (PAHs)	Surface runoff from roads and land surfaces	Lung cancer
	Sewage effluents	
	Diesel exhaust	
	Cigarette smoke	
	Stove smoke	
Radon	Soil, rock, and groundwater	Lung cancer
Asbestos	Asbestos mines and mills	Mesothelioma
	Insulation	Lung cancer
	Building materials	Asbestosis
Arsenic	Copper smelters	Lung cancer
	Cigarette smoke	
	Pressure-treated wood	
	Pesticides	

asthma, although irritants can *exacerbate* preexisting RADS or asthma.

Pulmonary edema and pneumonitis can occur following acute irritation of the deep respiratory tract. Pulmonary edema occurs following extravasation of fluid and cells from the pulmonary capillary bed into the alveoli. Primary pulmonary edema is due to direct toxic action on the capillary walls. For example, exposure to ozone or oxides of nitrogen, common in industrial settings, can cause pulmonary edema. This response can be immediate, such as when a trapped worker cannot escape from a place of high exposure, or delayed, when exposure is lower. In contrast to pulmonary edema, pneumonitis is an inflammation of the lung parenchyma in which cellular infiltration rather than fluid extravasation predominates. It can be caused by exposure to beryllium or cadmium.

Factors Involved in Toxicity

The most widespread causes of acute responses are irritant gases. Water is a major constituent of the respiratory tract lining, and solubility of these gases in water is the most significant factor influencing their site of action. Gases with high solubility act on the upper respiratory tract

Table 18-4. Selected Causes of Occupational Asthma[a]

Agents	Occupations
High–Molecular-Weight Compounds	
Animal products: dander, excreta, serum, secretions, fish glue	Animal handlers in laboratories, research scientists, farmers, grain-store workers, and entomologists
Plants: grain, dust, flour, tobacco, tea, hops, latex, cotton, coffee beans	Grain handlers, tea workers, textile workers, bakers, and workers in natural oil manufacturing, tobacco, food processing, and health care
Enzymes: *B. subtilis*, pancreatic extracts, papain	Bakers and workers in the detergent and pharmaceutical industries
Dyes: anthraquinone, carmine, paraphenyl diamine, henna extract	Fabric and fur dyers, beauticians
Other: crab, prawn	Crab and prawn processors
Low–Molecular-Weight Compounds	
Diisocyanates: toluene diisocyanate, methylene-diphenyldiisocyanate	Polyurethane industry workers, roofers, insulators, painters, plastics workers, workers using varnish, and foundry workers
Anhydrides: phthallic and trimellitic anhydrides	Epoxy resin and plastics workers
Wood dust: oak, mahogany, California redwood, western red cedar	Carpenters, sawmill workers, and furniture makers
Metals: platinum, nickel, chromium, cobalt, vanadium, tungsten carbide	Platinum- and nickel-refining workers and hard-metal workers, platers, and welders
Soldering fluxes	Solderers
Drugs: penicillin, methyldopa, tetracyclines, cephalosporins, psyllium, organophosphates	Pharmaceutical and health care industry workers and farmworkers
Other organic chemicals: urea formaldehyde, dyes, formalin, azodicarbonamide, hexachlorophene, ethylene diamine, dimethyl ethanolamine, polyvinyl chloride, pyrolysates	Workers in chemical, plastics, and rubber industries; hospitals; laboratories; foam insulation manufacture; food wrapping; and spray painting

[a]Mechanism believed to be IgE-mediated for high-molecular-weight compounds and for some low-molecular-weight compounds. The immunologic mechanism for asthma from many low-molecular-weight substances remains undefined.

Source: Venables KM, Chan-Yeung M. Occupational asthma. Lancet 1997; 349: 1465–1469.

within *seconds*. For example, fatal epiglottic edema has been associated with irritants of high solubility, such as ammonia, hydrochloric acid, and hydrofluoric acid. In contrast, moderately soluble gases act on both the upper and lower respiratory tract within *minutes*. Chlorine gas, fluorine gas, and sulfur dioxide are irritants of this type, producing both upper respiratory irritation and bronchoconstriction. The low-solubility irritants, such as ozone, oxides of nitrogen, and phosgene, are most insidious. With few warning signs, they penetrate to the deep portions of the respiratory tract and act predominantly on the alveoli *6 to 24 hours* after exposure. Because of this considerable delay in onset of symptoms, individuals can be exposed to large amounts of these irritants without any warning symptoms.

Other factors influencing the site of action of an irritant gas are intensity and duration of exposure. The amount of exposure depends not only on air concentrations but also on work effort. A worker with a sedentary job exposed to a given concentration of a respiratory irritant receives a much lower dose than a worker with an active job requiring rapid breathing and a high minute ventilation (the product of tidal volume and respiratory rate).

A final element that influences the site of action is interaction, such as synergism or antagonism. Sulfur dioxide and water droplets are synergistic, combining to deliver a sulfuric acid–like vapor to the respiratory tract. Ammonia and sulfur dioxide, however, are antagonistic and together produce less response than either can individually. The presence of a carrier, such as an aerosol, may increase the effect of an irritant gas. For example, sulfur dioxide may cause a moderate effect and a sodium chloride aerosol no effect on the respiratory tract; however, concurrent respiratory exposure to both of these substances may result in a marked effect because the aerosol delivers the sulfur dioxide more deeply into the lung.

Highly Soluble Irritants

Primary examples of highly soluble irritants are *(a)* ammonia, used as a soil fertilizer and in the manufacture of dyes, chemicals, plastics, and explosives, in tanning leather, and as a household cleaner; *(b)* hydrochloric acid (hydrogen chloride), used in chemical manufacturing, electroplating, and metal pickling; and *(c)* hydrofluoric acid (hydrogen fluoride), used predominantly for etching and polishing of glass, as a chemical catalyst in the manufacture of plastics, as an insecticide, and for removal of sand from metal castings in foundry operations.

The primary physical effects of highly water-soluble irritants are first perception of their odors and then irritation of the eyes, the nose, and sometimes the throat. In high doses, the respiratory rate can increase and bronchospasm can occur. Lower respiratory tract effects, however, do not occur unless the person is severely overexposed or trapped in an environment. The irritant effects are powerful and usually provide adequate warning to prevent overexposure of people free to escape from exposure. The history and physical examination are the most important parts of evaluating people exposed to irritants. Pulmonary function tests may show significant airflow limitation, reflecting bronchospasm shortly after exposure. Chest X-rays are not helpful unless there is pulmonary edema.

Management of reactions to these irritants is immediate removal of the worker and, if breathing is labored or hypoxemia is present, administration of oxygen. If severe exposure or loss of consciousness occurs, observation in a hospital for development of pulmonary edema is advisable.

Prevention of exposures relies on proper industrial hygiene practices with local exhaust ventilation as an essential component. Respirators should be used only as a temporary control measure in an emergency. If respirators are required to prevent overexposure, workers must be trained in their proper use and maintenance.

CASE 2

A 25-year-old man came to the emergency room with acid burns. Before taking a job as an electroplater 5 weeks before, he was in perfect health. On the first day at this job, he developed itching. He then developed sores, which healed with scars, at sites of splashes of workplace chemicals. After 4 days on this job, he developed a runny nose, throat irritation, and a productive cough. He also noted some shortness of breath at work.

His work involved dipping metal parts into tanks containing chrome solutions and acid. He wore a paper mask (disposable respirator), rubber gloves, and an apron, but no eye protection. Although heavy fumes were present in the $60 \times 20 \times 14$-foot room, there was no ventilation. None of the other eight workers in the room seemed to have similar medical problems.

Past history revealed three prior hospitalizations for pneumonia, but not asthma or allergies. He smoked about four cigarettes a day.

From age 16 to 18, he worked as a sheet metal punch-press operator for a tool and die company. At age 18, he worked as a drip-pan cleaner for a soup company. From age 19 to 21, he was a student in an auto mechanic school. From age 21 to 24, he occasionally worked as a gas station attendant.

Physical examination was normal, except for multiple areas of round, irregularly shaped, depigmented, 1 mm atrophic scars on both forearms and exposed areas of the anterior thorax and face; a 4 mm, rounded, punched-out ulcer, with a thickened, indurated, undermined border and an erythematous base on his left cheek; an erythematous pharynx; and bilateral conjunctivitis. His nasal septum was not perforated. Patch tests with dichromate, nickel, and cobalt were all negative. A chest X-ray was normal.

He was diagnosed with irritation and inflammation of the upper respiratory tract and an irritant contact dermatitis, both due to chromic acid mist. His symptoms resolved with removal from exposure. Periodic medical monitoring was advised to provide early diagnosis of a possible cancer of the nasal sinuses, for which he may be at increased risk because of his chromium exposure. Finally, a follow-up industrial hygiene survey of the workplace was initiated to control exposures for the other exposed workers.†

† Case courtesy of Stephen Hessl, MD, Daniel Hryhorczuk, MD, and Peter Orris, MD, Section of Occupational Medicine, Cook County Hospital, Chicago, Illinois.

Many small electroplating firms have no local ventilation over open vats of chromic and other acids. Frequently, a high level of chrome or other metals in the fumes is liberated when metal parts are immersed for plating. Chrome and chromic acid mist are local irritants. Primarily in hexavalent forms, chromium is considered to be a carcinogen; epidemiologic studies have shown an elevated lung cancer risk among exposed workers.[13] (See Chapter 17.)

Moderately Soluble Irritants

The moderately soluble irritants include chlorine, fluorine, and sulfur dioxide. Chlorine is widely used in the chemical industry to synthesize various chlorinated hydrocarbons, whereas outside the chemical industry its major use is in water purification and as a bleach in the paper industry. Fluorine is used in the conversion of uranium tetrafluoride to uranium hexafluoride, in the development of fluorocarbons, and as an oxidizing agent. Fluoride is used in the electrolytic manufacture of aluminum, as a flux in smelting operations, in coatings of welding rods, and as an additive to drinking water. Sulfur dioxide is commonly used as a disinfectant, a fumigant, and as a bleach for wood pulp, and it is formed as a by-product of coal burning, smelter processes, and the paper industry. Sulfur dioxide is a colorless, highly water-soluble gas, and hence it affects mostly the upper respiratory tract and has limited deposition in the lower airways. However, during exercise, the resultant increased minute ventilation may result in greater lower airway deposition than what would be usually expected. When sulfur dioxide is released into the atmosphere, it combines with water, metals, and other pollutants to form aerosols, most importantly sulfuric acid, metallic acids, and ammonium sulfates. These aerosols induce asthmatic responses.

Particulate air pollution, consisting of particles suspended in the air after various forms of combustion or other industrial activity, increases morbidity and mortality.[14] In contrast to particles greater than 10 μm in diameter (PM_{10}), particles less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) are carried deep into the lungs and are more likely to be deposited in the lower airways.

These irritants, like the highly soluble ones, initially cause irritation of mucous membranes, often manifested by persistent cough. Acute symptoms are usually of short duration. Low levels of continuous exposures, which are better tolerated than exposures to highly soluble irritants, may cause obstructive respiratory disease.

In addition, these irritants can lead to other health problems. Chlorine gas can corrode teeth, and fluorine can cause chemical skin burns. Chronic exposure to fluoride is associated with increased bone density, cartilage calcification, discoloration of teeth in children, and possibly rheumatologic syndromes, such as seronegative arthritis. Sulfur dioxide exposure can cause bronchospasm, especially in people with asthma, and may eventually contribute to chronic obstructive pulmonary disease. Management and prevention are similar as for highly soluble irritants. Pulmonary function tests, especially spirometry, are recommended in surveillance programs for individuals with chronic exposure.

Low-Solubility Irritants

Usually, the effects of irritants with low solubility are mild throat irritation and occasional headache. Much more significant is pulmonary edema, which manifests 6 to 24 hours after exposure, preceded by symptoms of bronchospasm, such as chest tightness and wheezing (Fig. 18-6). Although management and prevention are similar to those for highly soluble irritants, overnight observation of patients is frequently necessary, when excess exposure has occurred, because of the insidious onset of pulmonary edema.

Two of the most commonly produced industrial and urban pollutants are ozone and oxides of nitrogen, which are usually produced by the action of sunlight on the waste products of the internal combustion engine. Both are present in welding fumes, and therefore are found in many work environments. Ozone is used as a disinfectant; as a bleach in the food, textile, and pulp and paper industries; and as an oxidizing agent. Oxides of nitrogen are used in chemical and fertilizer manufacture and in metal processing and cleaning operations.

Of great concern are unburned hydrocarbons and nitrogen dioxide, which are the main constituents of smog, commonly seen in Los Angeles,

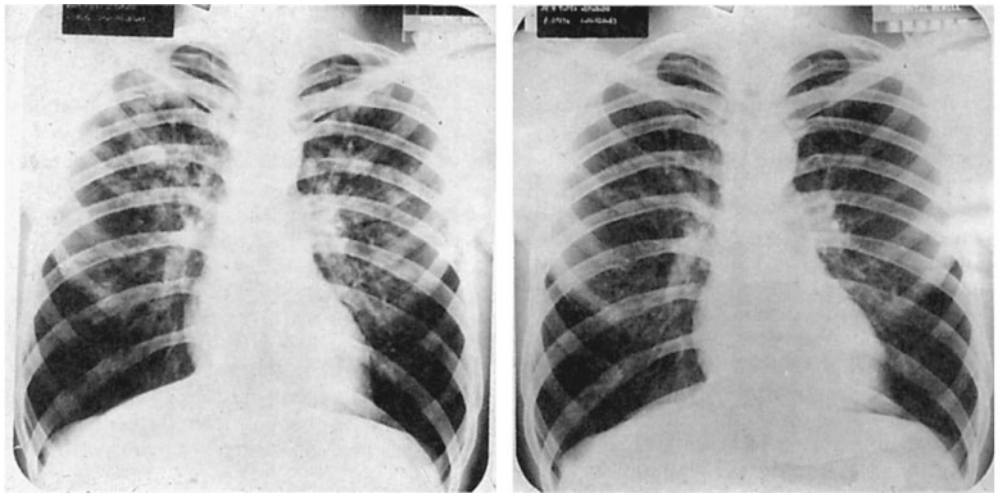

Figure 18-6. Chest X-rays in a copper miner. (A) Twenty-four hours after overexposure to oxides of nitrogen. Pulmonary edema is evident. (B) One week after exposure, there is resolution of pulmonary edema. (Courtesy of the late Benjamin G. Ferris, MD, Harvard School of Public Health, Boston, Massachusetts.)

Mexico City, Bangkok, and other large cities. Levels are usually lowest in the morning, highest at midday, and taper off after sunset. Higher levels are found in the summer, when sunlight is more intense and temperatures are higher.

Ozone and oxides of nitrogen are relatively insoluble in water, and therefore reach deeper into the respiratory tract as compared with sulfur dioxide and other gases. Chronic exposure to oxides of nitrogen may result in bronchiolitis obliterans, an obstructive deficit on spirometry, and a chest X-ray with evidence of pulmonary edema and bilateral patchy airspace opacities.

Silo filler's disease results from exposures to oxides of nitrogen in the upper chambers of grain silos due to anaerobic fermentation of green silage. Its brownish color is an important warning sign for farmers. Acute overexposure and death have resulted from inadequately ventilated silos.

Exposure to ozone levels even below the U.S. National Ambient Air Quality Standard of 80 ppb for 8 hours has resulted in symptoms and decreases in peak expiratory flow. These exposure levels are lower than in Los Angeles and the northeastern United States.

The Environmental Protection Agency (EPA) has developed an Air Quality Index (AQI) that gives a numeric score (0 to 500) for air quality on a given day. The AQI considers five major pollutants: ozone, sulfur dioxide, nitrogen dioxide, carbon monoxide, and particulate pollution. An AQI is calculated for each; the reported AQI is the highest of these five scores. Values between 101 and 150 have been designated "unhealthy for sensitive groups." The EPA defines "sensitive groups" as persons with heart or lung disease, older adults, and children. People who are active outdoors may also be sensitive to ozone. On days with high AQIs, those who are in "sensitive groups" are recommended to stay indoors, close car windows, and use air conditioning to protect against exposure and adverse effects of ozone and other pollutants. AQIs above 150 are considered "unhealthy" for the general public, and those above 300 are considered "hazardous." More information on ambient ozone and on air quality standards can be found at: http://www.AIRNow.gov.

NONIRRITANT EXPOSURES

Carbon Monoxide

Carbon monoxide is emitted mainly from internal combustion engines used in motor vehicles. Other causes of exposure include incomplete combustion of coal, paper, wood, oil, gas, or any other carbonaceous material. Carbon monoxide adversely affects blood oxygenation. The affinity of carbon monoxide for hemoglobin is 240 times

more than the affinity of oxygen for hemoglobin, thus severely reducing the ability of the blood to transport oxygen. Carbon monoxide can cause severe hypoxia, resulting in fatal neurological and cardiac consequences. (See Chapters 11 and 23.)

Indoor Air Pollution

Indoor air pollution, in homes and nonfactory public buildings such as office buildings, schools, and hospitals, can cause mucous membrane irritation, discomfort, illness, and even death. Indoor air pollution is a major problem in developing countries, due to the burning of biomass in homes.[15] The World Health Organization estimates that 2 to 3 million deaths occur annually in developing countries due to severe indoor pollution, with women and children affected more frequently than men. High levels of indoor air pollution are strongly associated with respiratory illnesses in children under age 5. Residents of developed countries are also exposed to high levels of indoor pollution, due to tight building construction and use of building materials that have high levels of volatile organic compounds.

In developing nations, the exposure of high levels of indoor pollution occurs during the burning of biomass, such as wood, crop residues, and animal dung. Homes that use biomass for cooking expose residents to mean 24-hour carbon monoxide levels estimated from 2 to 50 ppm, rising to 10 to 500 ppm during cooking. (The EPA 8-hour standard for carbon monoxide is 9 ppm.)[16] (See Chapter 7.)

OCCUPATIONAL AIRWAYS DISEASE

CASE 3

An 18-year-old woman, with an 8-week history of wheezing, a productive cough, and shortness of breath and a 1-day history of cyanosis of her fingertips presented to an emergency department. She had begun working at a tool supply and manufacturing company 9 weeks before—1 week before her symptoms began. Her usual job was grinding carbide-steel drill bits. Part of her work involved sharpening drill bits on a machine that generated copious metal dust, often covering the machine and her face, hands, and clothes. There was no exhaust ventilation, and no respiratory protection was provided.

After her respiratory symptoms began 8 weeks before, she was temporarily assigned to cleaning drill bits in a solvent bath. On this job, she felt lightheaded, but had no difficulty breathing. After a long holiday weekend, she was again assigned to drill-bit grinding and, after several hours, developed a cough. The next day, the cough worsened and she experienced shortness of breath, prompting a second visit to her physician. When she improved from that episode, she returned to work again and experienced exacerbation of coughing and shortness of breath, prompting her emergency department visit.

Past medical history revealed childhood seasonal rhinitis, but no asthma, eczema, or other allergies. There was no family history of allergies or asthma.

Physical examination revealed a pulse rate of 128 and a respiratory rate of 40. She had cyanosis of the lips and fingertips. Chest examination revealed diffuse bilateral wheezes and use of accessory muscles for breathing.

Arterial blood gases on room air at rest revealed a markedly low PaO_2 (39 mm Hg). Spirometry showed a normal FVC, but a markedly abnormal FEV_1 (53% of predicted). A chest X-ray was normal. White blood cell count was elevated at 11,200 cells/mm^3, with increased eosinophils (10%).

She was treated with oxygen, bronchodilators, and steroids. She improved clinically and by the second day her FEV_1 had improved to 82% of predicted.

Her physician learned from the state occupational safety and health agency that carbide-steel bit alloys contain nickel, cobalt, chromium, vanadium, molybdenum, and tungsten and that grinding these bits can produce cobalt and tungsten carbide dusts, which can cause pulmonary sensitization.

The diagnosis in this case was occupational asthma. No specific agent was confirmed to be responsible, but the presence of tungsten carbide and cobalt dusts indicated that they probably caused her asthma. Since changing jobs, she has felt well and has not had further bronchospasm.‡

‡ Case courtesy of the late James Keogh, MD, University of Maryland School of Medicine, Balitmore, Maryland (unpublished curriculum materials).

Work-Related Asthma

According to the Asthma in America survey, approximately 7% of U.S. residents currently have physician-diagnosed asthma.[17] Worldwide, as many as 300 million people may be affected. An estimated 9% to 15% of new-onset adult asthma in industrialized countries is attributable to occupational factors.[18,19] Work-related asthma (WRA) refers to asthma that is induced by inhalation exposures in the workplace. Work-related asthma encompasses asthma developing de novo in the workplace—occupational asthma (OA)—as well as preexisting asthma that is triggered or exacerbated by workplace exposures—work-exacerbated asthma (WEA). Work-exacerbated asthma may occur in 25% or more or patients with preexisting asthma.[19] A wide variety of materials and circumstances can cause occupational asthma (Table 18-4). In addition to industrial exposures, asthma can be triggered in the workplace by exertion, cold air, tobacco smoke, and other factors. All people with asthma are at risk for WEA. Inciting conditions may be chemical, biologic, or physical. Prevalence of WRA may be underestimated by population surveys because many workers with WRA may have left their jobs to avoid symptoms.

Immunologic WRA is the most common form of WRA, accounting for more than 90% of cases. Primary etiologic agents are high-molecular-weight (HMW) compounds, usually proteins of animal or vegetable origin. Most HMW compounds result in an immunoglobulin E (IgE)-mediated reaction, the typical allergic or atopic immune response. Common examples of HMW triggers include latex and animal dander. Immunologic WRA can also be triggered by low-molecular weight (LMW) compounds. Some LMW compounds (such as diisocyanates and platinum) act through an IgE-mediated mechanism, but most LMW compounds (such as glutaraldehyde) act through non-IgE-mediated mechanisms. There is always a latency period between exposure and development of immunologic WRA as the immune system becomes primed. Latency periods among compounds widely vary, from hours to months or longer. Once a worker is sensitized, the exposure needed to produce symptoms typically becomes increasingly smaller. Atopy is a risk factor for immunologic WRA caused by HMW compounds. However, atopy is not a good predictor of sensitization.[18]

Nonimmunologic WRA accounts for less than 10% of WRA cases. It differs from immunologic WRA in that no immune sensitization occurs, and therefore no latency period is necessary. Nonimmunologic WRA may occur after a single high-level exposure to a workplace irritant, such as hydrochloric acid or other acids, and chlorine bleach or other bases. Some compounds that can cause sensitization are also irritants at certain concentrations. For example, glutaraldehyde may cause immunologic WRA in some workers and a nonimmunologic irritant response in others. Many irritants also cause skin reactions, which, in turn, can increase the risk for immunologic WRA. Many workers are exposed to both irritants and sensitizers; if an irritant causes skin breakdown, a worker may have increased exposure to a sensitizer through areas of open skin.

A diagnosis of WRA should be considered in all workers with new-onset asthma and those with previously quiescent or well-controlled asthma who experience increased symptoms of asthma. Diagnosis of WRA depends greatly on the occupational and environmental history and the symptom history, which is more sensitive than specific. Workers report wheezing, chest tightness, shortness of breath, or cough as typical symptoms, although nasal and sinus symptoms may also occur. Symptoms may develop during the work day and improve in the evening away from work. Symptoms may also worsen throughout the work week and improve over weekends or vacations. Some responses are delayed, however, and workers may report symptoms primarily at night or on weekends, with fewer symptoms at work. If exposure has been prolonged, symptoms may be persistent, losing any temporal pattern with work. In addition, removal from exposure does not always lead to complete recovery. One meta-analysis estimated that only 32% of workers have complete symptomatic recovery after cessation of exposure to the causative agent.[20] This meta-analysis also found that older age and a longer period of symptomatic exposure were negative prognostic indicators for recovery.

A high suspicion for WRA should prompt a thorough exposure history, including exposures

at previous jobs. Making a diagnosis of WRA can be challenging. The American College of Chest Physicians (ACCP) has published recent guidelines on diagnosis and management of work-related asthma, including a useful flowsheet for clinical evaluation.[19] Diagnostic workup begins with confirming asthma, using pre- and postbronchodilator spirometry testing, if spirometry is normal, or by methacholine challenge testing. Once asthma is confirmed, a relationship to work, and ideally to a specific workplace exposure, should be determined. Establishing a relationship of asthma to work can include serial measurements of peak expiratory flow rate (PEFR) throughout the day, using a hand-held flow meter. For example, a worker can measure PEFR four times daily (on both work days and non-work days) for 2 weeks, which may help establish a clear pattern. Alternatively, workers can have office-based spirometry performed on them before and after work shifts in a company-based or other occupational medicine clinic. A decrease of at least 300 mL, or 10%, of the FEV_1 (measured as the mean of the two best of three acceptable results each time) between the beginning and end of the first shift of the work week suggests a work-related effect.

To establish a relationship with a specific exposure, blood tests (such as IgE specific to the suspected exposure) or skin-prick testing using the suspected substance can be helpful. Unfortunately, such testing is not available for most exposures, and positive serum or skin testing does not diagnose WRA due to that exposure—it only establishes sensitization to the exposure. Many more workers are sensitized than have symptoms or disease. However, the ACCP recommends that such additional testing be done whenever available because the more positive findings that can be established, including work-related symptoms or positive methacholine challenge, the more certain the relationship to work becomes.[19]

Although it is done elsewhere in the world, specific inhalation challenge testing is not typically available in the United States because of risk of anaphylaxis or other serious reactions. In a specific challenge test, the suspected exposure (instead of methacholine) is inhaled to provoke bronchospasm.

Acute care for those with attacks of WRA is the same as for any other cases of asthma. Long-term management, however, almost always requires removal from exposure, which is especially critical in immunologic WRA since even very low levels of exposure can trigger symptoms in a sensitized worker. Pharmacologic management of chronic WRA is also similar to that of non-work-related asthma. Close monitoring of symptoms and lung function should be maintained for a person who must continue exposure to a suspected offending agent.

Finally, when a diagnosis of WRA cannot be established, other related conditions need to be considered, including vocal cord dysfunction, eosinophilic bronchitis, upper respiratory tract irritation, hypersensitivity pneumonitis, and psychogenic factors. However, it is important to remember that asthma (including WRA) can coexist with these other conditions.

Occupational Chronic Obstructive Pulmonary Disease

Occupational exposures may be a much more important contributor to the development of chronic obstructive pulmonary disease (COPD) than previously recognized. As many as 20% of cases of COPD can be attributed to occupational exposures to various vapors, gases, dusts, and fumes.[21] Some workplace factors may even double the risk of COPD.

Chronic bronchitis is probably the most frequent chronic respiratory response to external agents. A clinical diagnosis must satisfy the following ATS criteria: recurrent productive cough, occurring four to six times a day, at least 4 days of the week, for at least 3 months during the year, for at least 2 consecutive years. The definition of simple bronchitis—the production of phlegm on most days for as much as 3 months of the year—can be used to identify individuals with the most important symptoms. The excess mucus production associated with bronchitis often causes airflow obstruction. Chronic bronchitis is frequently superimposed on other respiratory diseases due to cigarette smoke and/or occupational hazards, including mineral dusts and fumes (such as from coal, fibrous glass, asbestos, metals, and oils); organic dusts (such as from cotton and grains); irritants (such as

ozone and oxides of nitrogen); plastic compounds (such as phenolics and isocyanates); acids; and smoke (such as from fires).

Flavorings-Related Lung Disease

In 2000, an occupational physician reported eight cases of bronchiolitis obliterans in former workers of a Missouri microwave popcorn plant.[12] Four of these individuals were awaiting lung transplantation. NIOSH conducted medical and environmental examinations that identified diacetyl (2,3-butanedione), a chemical used in butter flavoring, as the responsible agent.[22] Additional affected workers were identified at the plant and in California,[23] the United Kingdom, and elsewhere. Since some affected workers had been employed by plants that made artificial flavoring, NIOSH named this clinical entity "flavorings-related lung disease." It is also known as "flavorings-related bronchiolitis obliterans" and "popcorn worker's lung disease."

Symptoms include nonproductive cough, wheezing, dyspnea on exertion, and occasionally fever, night sweats, and weight loss. Symptoms generally develop gradually and worsen. Periods away from work generally do not improve symptoms.

Diagnostic evaluation includes an occupational history, with a focus on diacetyl exposure if flavorings-related lung disease is suspected. Spirometry usually reveals fixed obstruction (a reduction in FEV_1 and FEV_1/FVC that does not reverse with bronchodilator administration). Lung volume testing may show hyperinflation, since air-trapping is a key feature of bronchiolitis obliterans. Unlike emphysema or various causes of interstitial lung disease, the diffusing capacity is usually normal. Imaging tends to be normal, or it shows only hyperinflation or air-trapping. High-resolution computed tomography sometimes reveals thickened airway walls. Most cases recognized thus far have not responded to medical treatment. More information on this disease can be found on the NIOSH Web site.[24]

HYPERSENSITIVITY PNEUMONITIS

Hypersensitivity pneumonitis (HP), previously known as extrinsic allergic alveolitis, refers to reactions associated with the most illustrative of all occupational disease names (Table 18-5). Hypersensitivity pneumonitis results from an immunologic response to inhaled antigens from

Table 18-5. Examples of Hypersensitivity Pneumonitis

Disease	Antigenic Material	Antigen
Farmer's lung	Moldy hay or grain	Thermophilic actinomycetes
Bagassosis	Moldy sugar cane	
Mushroom worker's lung	Mushroom compost	
Humidifier fever	Dust from contaminated air conditioners or furnaces	
Maple bark disease	Moldy maple bark	*Cryptostroma* species
Sequoiosis	Redwood dust	*Graphium* species, *Pullularia* species
Bird fancier's lung	Avian droppings or feathers	Avian proteins
Pituitary snuff taker's lung	Pituitary powder	Bovine or porcine proteins
Suberosis	Moldy cork dust	*Penicillium* species
Paprika splitter's lung	Paprika dust	*Mucor stolonifer*
Malt worker's lung	Malt dust	*Aspergillus clavatus* or *Aspergillus fumigatus*
Fishmeal worker's lung	Fishmeal	Fishmeal dust
Miller's lung	Infested wheat flour	*Sitophilus granarius* (wheat weevil)
Stipatosis	Esparto fibers	*Aspergillus fumigatus*
Metalworking fluid – associated HP	Metalworking fluids (coolants)	*Mycobacterium chelonae* *Pseudomonas nitroreducens*
Furrier's lung	Animal pelts	Animal fur dust
Coffee worker's lung	Coffee beans	Coffee bean dust
Chemical worker's lung	Urethane foam and finish	Isocyanates (such as toluene diisocyanate), anhydrides

organic or LMW compounds, commonly fungi or thermophilic bacteria, that are present in a surprising variety of settings. Generally, these antigens are less than 3 μm in diameter, enabling them to deposit deep in the terminal bronchioles and alveoli, where the reaction occurs. These antigens are then cleared via lymphatic channels, draining into the hilar lymph nodes, inducing the immunologic response,[25] which is mediated by immunoglobulin G (IgG). The immune response is also characterized by complement activation and stimulation of alveolar macrophages. Repeated exposure to the antigen can lead to fibrosis. Once hypersensitivity is established, small doses of antigen may trigger episodes of alveolitis.

A worker with hypersensitivity pneumonitis typically has shortness of breath and a nonproductive cough, but little or no wheezing. In acute episodes, sudden onset of respiratory symptoms, fever, and chills is dramatic. Physical examination may reveal rapid breathing, fine basilar rales, and hypoxemia. Pulmonary function tests may show marked reduction in lung volumes consistent with restrictive disease. Arterial blood gas measurements generally show an increased alveolar–arterial oxygen difference and a reduced diffusing capacity. Other diagnostic testing for HP may include measuring various antibodies (precipitins) in serum, which are highly sensitive but not specific, and demonstrating a serum antibody response, which alone is not itself diagnostic. A conventional chest X-ray and HRCT can be helpful in acute episodes by revealing patchy infiltrates or a diffuse, fine micronodular shadowing. Radiographic findings are often evanescent, however, and are therefore easily missed in acute episodes. Bronchoalveolar lavage fluid may show lymphocytosis with a predominance of CD8 T cells. Biopsies generally reveal poorly formed granulomata and nonspecific findings, such as bronchiolitis, lymphocyte infiltration, foamy macrophages, and areas of fibrosis.

If the worker is removed from exposure, symptoms and signs usually disappear in 1 to 2 weeks. In severe cases, corticosteroid treatment is required. If repeated exposures occur, especially at relatively low levels that cause only mild symptoms, a more chronic disease may develop. The worker may be unaware of the association of symptoms with work exposure because symptoms can be similar to a persistent or intermittent case of the "flu." Over a period of months, however, there is usually gradual onset of dyspnea, which can be accompanied by weight loss and lethargy. Physical examination findings are similar to those in the acute reaction, although the patient may appear less acutely ill and may demonstrate finger clubbing. The chest X-ray, however, is more suggestive of chronic interstitial fibrosis and pulmonary function tests show a restrictive defect. The disease may progress to severe dyspnea, and the end result can resemble idiopathic pulmonary fibrosis (IPF), both clinically and histologically. The risk of fibrosis may depend in part, on host factors, including genetic markers. For example, there are familial forms of HP. And a phenotype similar to IPF, with clubbing and poor prognosis, has been described in Mexican pigeon fanciers, who have a high prevalence of HLA-DR7.[25]

Prevention rests on removal from exposure. This can be more readily accomplished than with asthma because environmental controls can focus on the elimination of conditions that foster bacterial or fungal growth. Process changes may also be necessary to prevent antigen production, and local exhaust ventilation—rather than, or in conjunction with, personal protective equipment (masks)—should be used.

Hypersensitivity Pneumonitis Associated with Metalworking Fluids

Outbreaks of hypersensitivity pneumonitis have been described in numerous workers as shown in Table 18-5. Recently, it has been seen in workers exposed to metalworking fluids (coolants) contaminated with microbial flora. Exposed workers have also developed other occupational lung diseases, such as work-related asthma. The metalworking fluids implicated in these outbreaks are water-based, and microbial flora contaminants have appeared to be the causative agents.[26] Sputum cultures of those affected in these outbreaks have revealed gram-positive bacteria and strains of *Pseudomonas*, and, most consistently, "rapid-growing" nontuberculous mycobacterial (NTMB) species, especially *Mycobacterium chelonae* or *Mycobacterium immunogenum*. Outbreaks of skin disease have

also been reported, consistent with the spectrum of NTMB-caused disease.

BYSSINOSIS AND OTHER DISEASES CAUSED BY ORGANIC DUSTS

Some types of airway constriction are believed to be due to direct toxic effects on the airways. Byssinosis (meaning "white thread" in Greek) is associated with exposure to cotton, hemp, and flax processing. It has been called "brown lung" (a misnomer because the lungs are not brown), analogous to the term "black lung" used to describe CWP.

Byssinosis has been shown to develop in response to dust exposure in cotton processing, but prevalence can range from 2% to 50%. It is especially prevalent among cotton workers in the initial, very dusty operations where bales are broken open, blown (to separate impurities from fibers), and carded (to arrange the fibers into parallel threads). A lower prevalence of disease occurs in workers in the spinning, winding, and twisting areas, where dust levels are lower. The lowest prevalence of byssinosis has been found among weavers, who experience the lowest dust exposure. Processing of cloth is practically free of cotton dust, as in the manufacture of denim, which is washed during dyeing before thread is spun. Byssinosis has also been described in nontextile sectors where cotton is processed, such as cottonseed oil, cotton-waste utilization, and garneting (bedding and batting) industries. The same syndrome has been shown to occur in workers exposed in processing soft hemp, flax, and sisal.

Byssinosis is characterized by shortness of breath and chest tightness. These symptoms are most prominent on the first day of the work week or after being away from the workplace over an extended period of time (which explains why it is sometimes called "Monday morning tightness"). No previous exposure is necessary for symptoms to develop. Symptoms are often associated with changes in pulmonary function. Characteristically, there is a decrease in FEV_1 during the Monday work shift or during the first day back at work after at least 2 consecutive days off. Because workers do not normally lose lung function during a work day, an acute loss of at least 10% or 300 mL (whichever is greater) in an individual, or 3% or 75 mL (whichever is greater) in a group of 20 or more workers, can be considered significant enough to require further investigation. Over time, cotton dust workers have an accelerated decrement in FEV_1, consistent with fixed airflow obstruction and chronic obstructive lung disease. Diagnosis is based mainly on symptoms; no characteristic examination or chest X-ray findings are associated with byssinosis. Therefore, the patient should be questioned systematically about symptoms.

It is assumed that byssinosis progresses if duration of exposure to sufficiently high dust levels is prolonged. Mild or early byssinosis is probably reversible if exposure ceases, but longstanding disease is irreversible. People with severe byssinosis are rarely seen in an industrial survey because they are too disabled to work. The end stage of the disease is fixed airway obstruction with hyperinflation and air-trapping. Cigarette smokers are at increased risk of irreversible byssinosis.

Much research has been done on possible etiologic mechanisms and effects. Extracts of cotton bract, a leaf-like part of the cotton flower, have been shown to release pharmacologic mediators, such as histamine, and prostaglandins. It seems likely that the mechanism of byssinosis involves stimulation of the same inflammatory receptors by endotoxin and by cotton dust. Gram-negative bacterial endotoxin contaminates cotton fiber, and aqueous extracts of endotoxin have produced acute symptoms and lung function declines.

Two other respiratory conditions are associated with work in the cotton industry:

1. *Mill fever*: This self-limited condition usually begins on first exposure to a cotton-dust environment. It lasts for 2 or 3 days and has no known sequelae. It is a "flu"-like illness characterized by headache, malaise, and fever—symptoms similar to metal fume fever and polymer fume fever. Mill fever is probably related to gram-negative bacterial material in mill dust; it usually affects workers only once, but after prolonged absence from a mill, reexposure may trigger another attack. This syndrome is also referred to as organic dust toxic syndrome, a form of inhalation fever.

2. *Weaver's cough*: Weavers have experienced outbreaks of acute respiratory illness characterized by a dry cough, although their dust exposure is comparatively low. It may result from sizing material or from mildewed yarn that is sometimes found in high-humidity weaving rooms.

Other organic/vegetable materials are associated with obstructive respiratory diseases, including flax (baker's asthma), swine confinement buildings (acute airflow obstruction), and wood dust (asthma and chronic airflow obstruction). It appears that chronic exposure to organic dusts can result in both acute and chronic lung disease.[27]

PNEUMOCONIOSES AND OTHER CHRONIC RESPIRATORY TRACT RESPONSES

Pulmonary fibrosis is a well-documented environmental and work-related chronic pulmonary reaction. This condition, which varies according to inciting agent, intensity, and duration of exposure, is generally referred to as a pneumoconiosis. It is usually due to an inorganic dust or coal that must be of respirable size (less than 5 μm) to reach terminal bronchioles and alveoli. Dust of this size is not visible, and so its presence may not be recognized by workers. There are two typical patterns of fibrosis: *(a)* localized and nodular, usually peribronchial, fibrosis; and *(b)* diffuse interstitial fibrosis. Both lead to a restrictive lung disease pattern on spirometry. The clinical features of all pneumoconioses are similar: initial nonproductive cough, shortness of breath of increasing severity, and, in the later stages, productive cough, distant breath sounds, and signs of right heart failure. The pneumoconioses are often associated with obstructive airways disease caused by the same agents or concomitant cigarette smoking during a working lifetime.

Silicate-Related Disease: Silicosis

Crystalline silica (SiO_2) is a major component of the earth's crust. Therefore, exposure occurs in a wide variety of settings, such as mining, quarrying, and stone cutting; foundry operations; ceramics and vitreous enameling; and in use of fillers for paints and rubber.

The World Health Organization estimates that at least 1.7 million workers in the United States are exposed to silica, and 10% are at risk for developing silicosis. Worldwide, there are estimated to be tens of millions of workers exposed to silica. China recorded more than 500,000 cases of silicosis between 1991 and 1995. In the United Kingdom, there were 1,164 reported new cases in 2002. In the United States, reports suggest that there are still around 5,000 new cases of silicosis and 300 deaths annually. No distinct clinical features can be cited beyond the ones already listed, but there is a distinct pathologic process. Silicosis occurs more frequently in the upper lobes of the lungs, with nodules varying in size from microscopic to 6 mm in diameter. In severe cases, nodules coalesce and become fibrotic masses several centimeters in diameter. Nodules are firm and intact with a whorled pattern, and rarely cavitate (Fig. 18-7). Microscopically, the nodules are hyalinized, with a well-organized circular pattern of fibers in a cellular capsule. The amount of fibrosis appears proportional to the free silica content and to the duration of exposure. Fibrosis progresses even

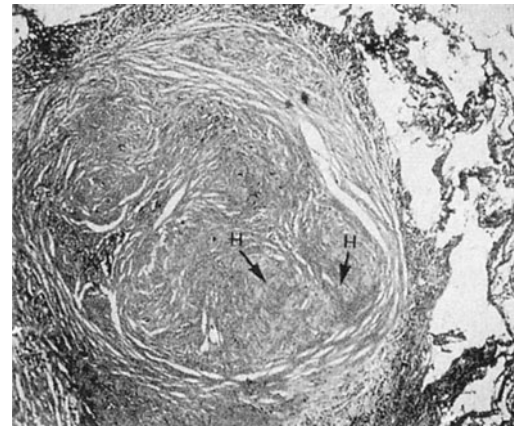

Figure 18-7. Microscopic section of a typical silicotic nodule showing the concentric (onion skin) arrangement of collagen fibers, some of which are hyalinized (H); lack of dust pigmentation; and peripheral cellularity. The lesion is clearly demarcated from adjacent lung tissue, which is substantially normal. (*Source*: Parkes WR. Occupational lung disorders (3rd ed.). London: Butterworths, 1994.)

after removal from exposure. Except in acute silicosis, symptoms usually do not occur until 10 to 20 years after initiation of exposure. Evidence of pathologic response to silica exposure exists well before symptoms occur.

Evaluation of workers exposed to silica includes lung function tests (that may show reduced FVC or total lung capacity or mixed obstructive and restrictive patterns, and reduced diffusing capacity), a chest X-ray (that may appear more abnormal than the lung function tests), and determination of (a reduced) hemoglobin oxygen saturation on exercise. As the disease progresses, there can be decreased oxygen saturation at rest and reduced total lung capacity. The chest X-ray usually shows rounded opacities, localized initially to the upper lung fields (see Fig. 18-2). The size and distribution of these opacities increase over time, and "eggshell" calcification of hilar lymph nodes occurs in some cases.

Chronic silicosis is classified either as simple or complicated, although there is a continuum between these two forms of the disease. The simple form is noted on the chest film by the presence of multiple, small, round opacities, usually in the upper zones. The concentrations of these opacities are used in classifying simple silicosis (ILO profusion categories 1 to 3).[1] Although simple silicosis alone is not a common cause of disability, it can contribute to disability as well as progress to complicated silicosis. In progressive massive fibrosis (PMF), several of the simple nodules appear to aggregate and produce larger conglomerate lesions, which enlarge and encroach upon the vascular bed and airways (ILO parenchymal categories A, B, and C). The extent of lung function impairment appears directly related to the radiographic size of the lesions and is most severe in categories B and C.

An important complication of silicosis is tuberculosis (TB). There is an increased incidence of TB among workers in the mining, quarrying, and tunneling industries, and steel and iron foundries. Workers exposed to silica may be at increased risk of TB, even in the absence of radiographic evidence for silicosis. Infections with atypical mycobacteria, such as *Mycobacterium kansasii* and *Mycobacterium avium-intracellulare*, can also occur and are related to the geographic distribution of these organisms. Treatment for such cases may require more vigorous drug treatment than for TB cases without silicosis. No relationship has yet been shown between silicosis and cigarette smoking. Another potential complication of silica exposure is lung cancer. Epidemiologic studies have demonstrated a link between silica exposure and lung cancer, and the International Agency for Research on Cancer (IARC) has classified silica as a Group 1 carcinogen.

Prevention of silicosis focuses on reduction of exposure through wet processes, isolation of dusty work, and local exhaust ventilation. Annual TB screening by purified protein derivative (PPD) skin testing or, if the PPD is positive, chest radiography is essential in silica-exposed workers. There is an ongoing U.S. national effort to eliminate silicosis in all sandblasting operations. Elimination of silicosis from individual work practices could substantially reduce the at-risk population.

Acute silicosis, a distinct entity, is a devastating disease. It is due to extraordinarily high exposures to small silica particles (1 to 2 μm). These exposures occur in abrasive sandblasting and in the production and use of ground silica. In one of the most tragic industrial disasters in U.S. history, approximately 3,000 miners employed to build the Hawk's Nest Tunnel through Gauley Mountain in West Virginia, starting in 1927, were exposed to massive amounts of silica due, in part, to the mineral content of the rock they were cutting. No personal protective equipment was provided or used. According to a monument erected by the West Virginia Department of Culture and History, there were 109 workers, mainly black migrant workers, who died. However, Congressional hearings later revealed that 476 workers had died, and the number may have actually been higher.

Symptoms of acute silicosis include dyspnea progressing rapidly over a few weeks, weight loss, productive cough, and sometimes pleuritic pain. Diminished resonance on percussion of the chest and rales on auscultation can be found. Lung function tests show a marked restrictive defect, with a substantial decrement in total lung capacity. The chest X-ray has a diffuse "ground-glass" or miliary TB–like appearance, rather than the appearance in classic nodular silicosis. The pathologic process in this disease is characterized

by widespread fibrosis, with a diffuse interstitial, macroscopic appearance, and a microscopic appearance and chemical constituency resembling pulmonary alveolar proteinosis, but with doubly refractile particles of silica lying free within the alveolar exudate. Disease onset usually occurs 6 months to 2 years after initial exposure. Acute silicosis is often fatal, usually within 1 year of diagnosis.

Diatomaceous earth is an amorphous silica material mined predominantly in the western United States. It is used as a filler in paints and plastics, as a heat and acoustic insulator, as a filter for water and wine, and as an abrasive. In contrast to the various forms of crystalline silica, amorphous silica has relatively low pathogenicity. However, some processes using diatomaceous earth include heating (calcinating) it to remove organic material. This heating process can produce up to 60% crystalline silica as cristobalite, which is highly fibrogenic. Exposure to this form of diatomaceous earth, therefore, must be treated the same as exposure to crystalline silica.

Silica appears in a wide variety of minerals in different combined forms known as silicates. Many of these silicates, such as asbestos, kaolin, and talc, also cause pneumoconiosis, but the types of pneumoconiosis that they produce have features distinct from those of silicosis. Asbestos is the most widespread and best known of the silicates. It causes asbestosis and several types of cancers. (See Chapter 17.)

Silicate-Related Disease: Asbestosis

Asbestos is a fibro-silicate that appears in nature in four major types (chrysotile, crocidolite, amosite, and anthophyllite) that produce similar chronic respiratory reactions. All four types are comprised of fibers that are indestructible at temperatures as high as 800°C. Use and production of these materials greatly increased in the twentieth century. According to the EPA, U.S. consumption peaked in 1973 at 885,000 tons.[28] And while asbestos is no longer produced in the United States, the U.S. Geologic Survey (USGS) estimated that 1,730 metric tons of asbestos were imported in 2007 for use in manufacturing roofing, coatings, friction products, and other materials. As of 2008, OSHA estimated that 1.3 million U.S. workers in construction and general industry are still at risk of asbestos exposure; for 2002, the Mine Safety and Health Administration (MSHA) estimated that an additional 44,000 miners and mine workers might also be at risk.[29] Asbestos has been used in a variety of applications: asbestos cement products (tiles, roofing, and drain pipes), floor tile, insulation and fireproofing (in construction and shipbuilding), textiles (for heat resistance), asbestos paper (in insulating and gaskets), and friction materials (brake linings and clutch pads). Probably the most hazardous current exposures occur in repair and demolition of buildings and ships, and in a variety of maintenance jobs where exposures may be unsuspected by the workers (Fig. 18-8). In the United States and many other countries, the construction industry is the major source of asbestos exposure to workers, mainly from disrupting previously installed asbestos products. The effect of asbestos exposure in the community can be significant, as seen in Libby, Montana. Strip mining, transportation, and processing of vermiculite ore containing asbestiform minerals was conducted in and around Libby from 1923 until 1990. As a result, asbestos-related lung diseases have been observed in Libby residents and workers at the mine. Numerous potential exposure sources existed, including direct exposure at work or in the community, use of vermiculite ore waste at home, and inadvertent exposure of family members via dust brought home on workers' clothes, skin, and hair. Among former workers at the Libby mine, 29% had pleural changes consistent with asbestos exposure in 2004–2005, compared to 2% in 1980. These pleural changes are directly related to cumulative fiber exposure.[30]

The main symptoms of asbestosis are cough and shortness of breath, which may be more severe than the appearance of the chest X-ray might indicate. In 20% of those affected, basilar rales are present, heard best at end inspiration or early expiration. Pleural rubs and pleural effusions can occur. Benign asbestos effusions can occur less than 10 years after initial exposure, and they are generally small and unilateral, and may resolve spontaneously. They can be associated with pain and fever, however. Although not common, pleuritic pain or chest tightness is

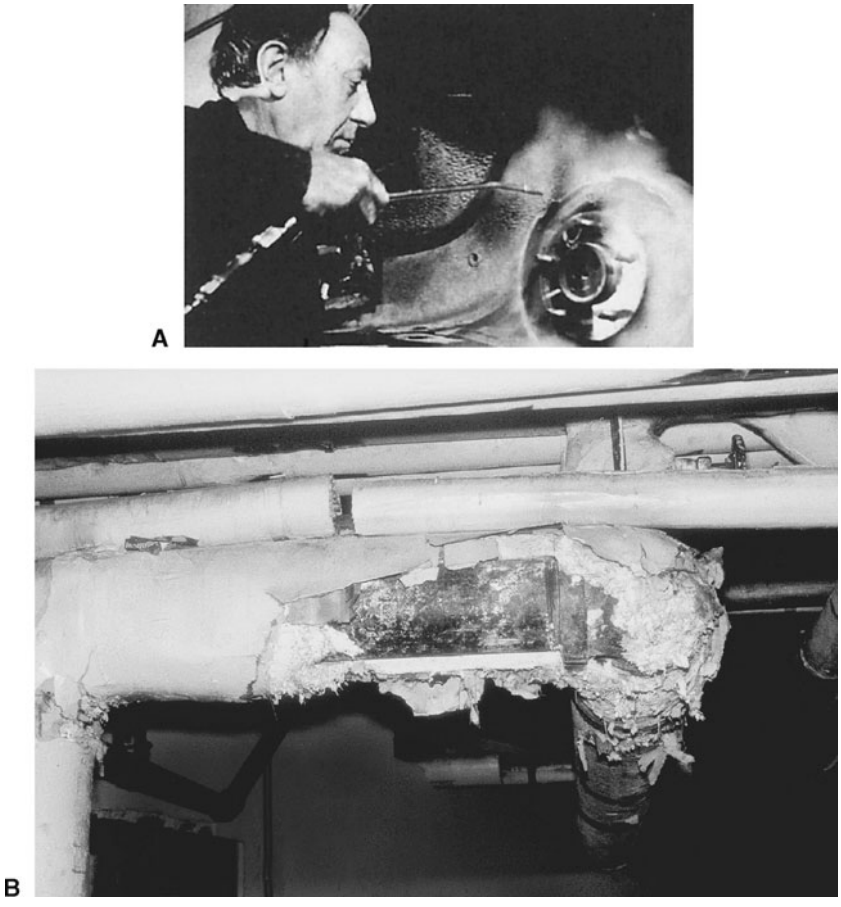

Figure 18-8. Examples of occupational exposure to asbestos: (A) Brake mechanic exposed to asbestos fibers while using compressed air to clean brake drum. (Photograph by Nick Kaufman.) (B) Exposed asbestos pipe insulation. (Photograph by Earl Dotter.)

more frequent in asbestos-related disease than in other pneumoconioses. Pleural effusion or pleuritic pain in a person with a history of asbestos exposure should always prompt an evaluation for mesothelioma.

Pathologically, the lung appears macroscopically as a small, pale, firm, and rubbery organ with fibrotic adherent pleura. The cut surface shows patchy to widespread fibrosis, and the lower lobes are more frequently affected than the upper. The microscopic appearance is characterized by interstitial fibrosis. Chest X-rays show widespread irregular (linear) opacities, more common in the lower lung fields.

Asbestos (or ferruginous) bodies can be seen microscopically in sputum and lung tissue. These are dumbbell-shaped bodies, 20 to 150 μm in width, that appear to be fibers covered by a mucopolysaccharide layer. Iron pigment (from hemoglobin breakdown) makes them appear golden-brown. They are not diagnostic of asbestos-related disease, but, when present even in small numbers in sputum or tissue sections, they indicate substantial occupational exposure to airborne asbestos fibers. Most urban dwellers in industrialized countries have a measurable asbestos burden, but the concentrations of asbestos bodies in nonoccupationally (or paraoccupationally) exposed populations are orders of magnitude lower than in those with occupational exposures. In the "background" population of urban dwellers, 50 to 100 microscopic sections of lung would have to be searched to find a single asbestos body, whereas people with

very early asbestosis have asbestos bodies in nearly every section. Those with more severe asbestosis usually have many asbestos bodies per section. Asbestos bodies may also be found in other parts of the body besides the lungs; they form round fibers that are transported by lung lymphatics into the circulation.

A particular feature of asbestos exposure, unlike other pneumoconioses, is the frequent presence of asbestos-induced circumscribed pleural fibrosis, known as pleural plaques, which are sometimes the only evidence of exposure. These plaques can calcify, may be bilateral, and are located more commonly in the parietal pleura. Sometimes, these plaques may provide evidence for prior asbestos exposure or help explain why pulmonary function tests are abnormal (Fig. 18-9).

Pleural plaques are one manifestation of the rather marked pleural reaction to asbestos fibers. Other such evidence seen on chest X-rays is a "shaggy"-appearing cardiac or diaphragmatic border. An early, nonspecific sign is a blunted costophrenic angle. Diffuse pleural thickening also occurs, probably less commonly than the more specific pleural plaques. Asbestos-induced diffuse visceral pleural fibrosis may also occur and may impair lung function. Advanced pleural fibrosis may act like a cuirass, severely constricting breathing and leading to respiratory failure.

The evaluation of an individual suspected of having asbestosis includes determining whether there has been a history of exposure; a physical examination to ascertain whether rales are present; a chest X-ray, which may show irregular linear opacities and a variety of pleural reactions; and pulmonary function tests, which may show evidence of restrictive disease and a decreased diffusing capacity. In addition, the peribronchiolar fibrosis may have an obstructive component. Hence, in both nonsmokers and smokers with asbestosis (as with all pneumoconioses), a mixed restrictive–obstructive pattern may be seen.

Asbestosis, like silicosis, may progress after removal from exposure. Asbestos exposure, even without asbestosis, carries with it the added risk

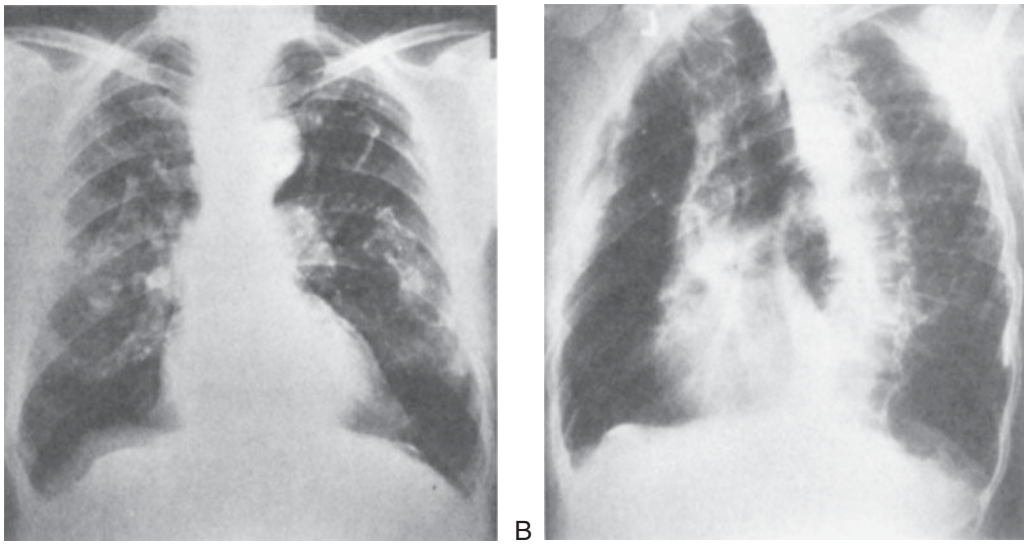

Figure 18-9. Bilateral calcified pleural plaques on chest walls and diaphragm, on two posteroanterior chest films. (A) Note irregular outline and variable density of the large lesion seen en face, in the mid-right lung field, and the rim of calcification along the left cardiac border. The small, rounded lesions also represent calcification in plaques and are not intrapulmonary. (B) The large plaque in the right lung field on the posteroanterior film is seen end-on against the chest wall (left field). There is no evidence of diffuse interstitial pulmonary fibrosis in either film. The patient, 65, had been an insulation worker from 1925 to 1932. There were no crackles in the lungs. Lung function testing showed severe airflow obstruction and hyperinflation. (*Source*: Parkes WR. Occupational lung disorders (3rd ed.). London: Butterworths, 1994.)

of mesotheliomas of the pleura and peritoneum, lung cancer, and cancers of the gastrointestinal tract and other organs. (See Chapter 17.) Prevention focuses on substitution with materials such as fibrous glass, use of wet processes to reduce dust generation, local exhaust ventilation to capture the dust that is generated, and respiratory protection. Exposed patients who smoke should be advised to stop smoking, in part because the risk factors of cigarette smoking and asbestos for (nonmesothelioma) lung cancer are synergistic—that is, the risk from each exposure is multiplied together to cause a much higher risk in people who have both exposures.

In 2004, the ATS published an official statement on the diagnosis and initial management of nonmalignant disease related to asbestos.[31] Nonmalignant asbestos-related disease includes asbestosis; pleural plaques, thickening, or fibrosis; benign asbestos pleural effusion; and airflow obstruction. This statement outlines diagnostic criteria, recommended clinical evaluation, expected disease outcomes, and implications of diagnosis for patient management. The statement is available online at the ATS Web site (http://www.thoracic.org).

Pneumoconioses Due to Talc and Kaolin

Talc is a hydrated magnesium silicate that occurs in a variety of natural forms. The two major types are nonfibrous and fibrous. The nonfibrous forms, such as those found in Vermont, are free of both crystalline silica and fibrous asbestos tremolite; the fibrous forms, such as those found in New York State, can contain up to 70% fibrous material, including amphibole forms of asbestos. Talc exposures occur mainly during its use as an additive to paints and as a lubricant in the rubber industry, especially in innertubes. Evidence suggests that high doses of nonfibrous talc or moderate doses of fibrous talc accumulated over a long time can result in chronic respiratory disease known as talcosis, with the same symptoms as other pneumoconioses.

Pathologically, the macroscopic appearance of the lung is characterized by poorly structured nodules, unlike the firm nodules of silicosis or the diffuse fibrosis of asbestosis. The microscopic appearance consists of ill-defined nodules with some diffuse interstitial fibrosis. Evaluation of people exposed to talc includes pulmonary function tests and chest X-rays. Chest X-rays may show both nodular and linear opacities and also pleural plaques. Studies addressing the possibility of a cancer risk associated with fibrous talc exposure have found a four-fold increase in risk of lung cancer in New York State talc miners.

Kaolin (China clay) is a hydrated aluminum silicate found in the United States (in a band from Georgia to Missouri), India, and China. It is used in ceramics; as a filler in paper, rubber, paint, and plastic products; and as a mild soap abrasive. Kaolin is not particularly hazardous in the mining processes because it is usually a wet ore and mined by jet-water mining techniques.

The pneumoconiosis resulting from chronic exposures to kaolin dust (kaolinosis) produces no unique clinical features. Pathologically, the macroscopic appearance is one of immature silicotic nodules, although conglomerate nodules may appear. Pleural involvement occurs only if the lung is massively involved. The microscopic appearance consists of nodules with randomly distributed collagen.

Coal Workers' Pneumoconiosis

In the United States until the 1960s, coal workers' respiratory disease was considered a variant of silicosis and was often known as an anthracosilicosis. It is now clear that coal workers' pneuomoconiosis (CWP) is an etiologically distinct entity that can be induced by both coal dust and pure carbon. Coal workers' pneuomoconiosis exists both in uncomplicated and complicated forms; the latter, known as progressive massive fibrosis (PMF), is the most severe and disabling form of the disease. Although exposure to coal dust occurs most commonly in underground mines, there is also some exposure in handling and transport of coal. Significant exposure also occurs in the trimming or leveling of coal in ships when preparing material for transport.

Uncomplicated CWP increases the likelihood for future development of the complicated form. Diagnoses of CWP have relied primarily on chest X-rays, which show nodular opacities of less than 1 cm (mostly less than 3 mm) in diameter. Progressive massive fibrosis, in contrast, is seen on chest X-rays as the development of

conglomerations of these small opacities to sizes greater than 1 cm in diameter.

In the early stages, CWP is asymptomatic. Initial symptoms are dyspnea (breathlessness) on exertion with progressive reduction in exercise tolerance. As nodular conglomeration begins and PMF is diagnosed, symptoms become more severe, with marked exertional dyspnea, severe disability, or total incapacity. There is general agreement that PMF leads to premature disability and death. No such agreement, however, exists for the impact of simple CWP.

Coal dust also contributes independently to the disability observed in coal workers through the production of chronic bronchitis, airways obstruction, and emphysema. The occurrence of bronchitis and loss of pulmonary function are both dose-dependent on coal dust exposure, in smokers and nonsmokers. The greater the intensity and duration of exposure (cumulative exposure), the more likely that a miner will get one of these diseases. Concomitant silicosis can occur as well if the quartz content of the coal is high. These two diseases may present in any combination.

Pathologically, CWP appears macroscopically as soft, black, indurated nodules. Microscopic observation shows dust in and around macrophages near respiratory bronchioles. Nodules show random collagen distribution and the lung shows centrilobular emphysema. Chest X-rays show widely distributed, small, round opacities.

In PMF, the large conglomerate masses have variable shapes and do not respect the architecture of the lung. The surfaces are hard, rubbery, and black, and cavitation often occurs (Fig. 18-10). Copious, black sputum is often produced.

Microscopically, the appearance is not distinct from the simple nodules. Chest X-rays show large conglomerate opacities (Fig. 18-11). Although evaluation for CWP is the same as for the other pneumoconioses, a particular feature affecting evaluation is the federal Mine Safety and Health Act of 1977, which prescribes what types of abnormalities make a person eligible for disability benefits. Because these are subject to continuous revision, consultation with the Mine Safety and Health Administration in the U.S. Department of Labor is advisable. Miners have special rights to a low-dust environment with increased medical monitoring if they are found to have CWP, and they have the right to permanent removal from the high-dust environment with wage retention. These rights are unique among workers in the United States, although arguably such an approach should be applied in the prevention of all pneumoconioses.

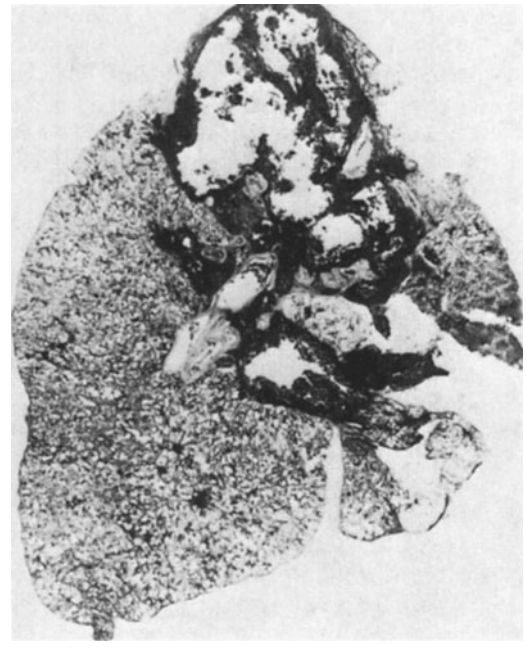

Figure 18-10. Gough section of lung of coal worker with 18 years of mining experience, which had been completed 20 years prior to death. It shows cavitation as well as centrilobular emphysema, which was present in both lungs. (Courtesy of J. C. Wagner, MRC Pneumoconiosis Unit, Llandough Hospital, Penarth, Wales, United Kingdom.)

Rheumatoid pneumonconiosis (Caplan syndrome) occurs when pneumoconiosis (silicosis, asbestosis, or CWP) is accompanied by rheumatoid arthritis. First described in 1953 by Dr. Anthony Caplan in a group of 51 Welsh coal miners, Caplan syndrome is defined by an increased number of nodules relative to other patients with pneumoconiosis, but less respiratory impairment in many cases. Radiographically, there are multiple well-defined bilateral nodules or masses, primarily peripheral in distribution. The pathologic appearance is that of alternate black and gray-white bands of material in conglomerate masses that frequently cavitate or calcify. Patients also have rheumatoid arthritis clinically, and they usually have positive serologies for rheumatoid disease as well.

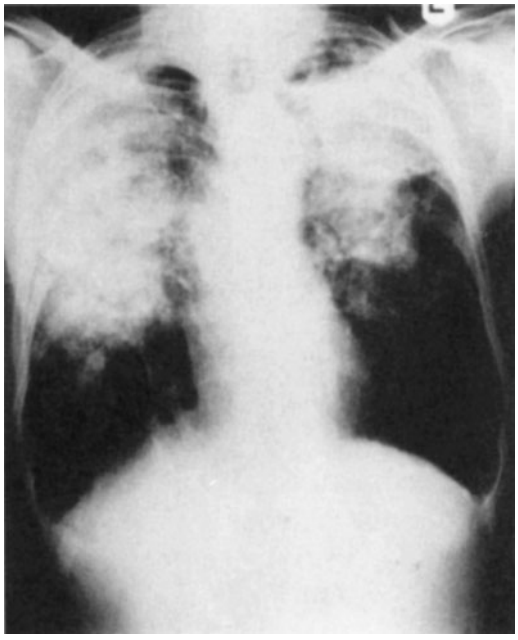

Figure 18-11. Chest X-ray of coal worker whose lung section appears in Figure 18-10, taken 2 weeks before death. The appearance is classic for progressive massive fibrosis, with large conglomerate masses in both lung fields. (Courtesy of J. C. Wagner, MRC Pneumoconiosis Unit, Llandough Hospital, Penarth, Wales, United Kingdom.)

Flock Worker's Lung

An emerging pneumoconiosis is chronic interstitial lung disease in workers employed in the nylon flocking industry. Index cases were first described in plants in Rhode Island and Canada in the early 1990s.[32] Since then, more cases have been reported. Processes in the flocking industry include cutting nylon tow (cables of synthetic monofilaments), flocking the cut fibers (applying them to an adhesive-coated substrate), dying the nylon flock, and other finishing processes.[33] Environmental studies of flocking plants have identified multiple airborne exposures, including bioaerosols, nylon fibers, and tannic acid. Prior to cutting and flocking, the nylon polyamide fibers were believed by manufacturers to be of nonrespirable size, but air sampling conducted by NIOSH has revealed long, thin, respirable-size nylon particles. While there are still additional candidate exposures, the respirable fibers are currently believed to be the causative agent in flock worker's lung.[34]

In the initial case series, median latency was 6 years from date of hire to onset of symptoms.[33] Most common clinical symptoms include dry cough and dyspnea. Chest X-ray and HRCT findings have included patchy infiltrates with a "ground-glass" appearance, and occasional areas of fibrosis. Bronchoalveolar lavage fluid often has a high proportion of eosinophils. The most consistent histopathologic findings on lung biopsy specimens have been a pattern of nonspecific interstitial pneumonia (NSIP), lymphoid nodules, and lymphocytic bronchiolitis without evidence of granulomata. However, other patterns, including bronchiolitis obliterans organizing pneumonia (BOOP), can occur. The long-term natural history of this disease is not yet known, but most workers have had symptomatic and X-ray improvement after cessation of exposure.

Miscellaneous Inorganic Dust Disorders

Fibrous glass and related products, referred to as synthetic vitreous fibers (SVFs), manmade vitreous fibers, manmade mineral fibers (MMMFs), or very fine vitreous fibers, have been used for insulation purposes for over 60 years. More recently, they have played an important role as an asbestos substitute. Synthetic vitreous fibers are amorphous silicates with a length-to-diameter ratio of greater than 3:1. They are made mainly from rock, slag, glass, or kaolin clay, and they can be divided into three main groups: mineral wool, fibrous glass, and ceramic fiber.

Synthetic vitreous fibers can induce skin, eye, and upper respiratory tract irritant responses. There have been few case reports of pulmonary disease due to SVF exposure. In general, respiratory symptoms are absent or mild, and chest X-rays and pulmonary function tests are normal. Limited studies of workers exposed to fine-diameter fibers have revealed evidence of irregular opacities consistent with pneumoconiosis. An excess of pleural changes (especially pleural plaques) without abnormalities in lung function has been demonstrated in workers involved in the production of refractory ceramic fibers. There is growing concern about the possible carcinogenicity of these very fine fibers. IARC has classified special-purpose glass fibers, such as E-glass and "475" glass fibers, and also ceramic

fibers as possibly carcinogenic to humans, and it has determined that insulation glass wool, continuous glass filament, rock (stone) wool, and slag wool are not classifiable as to their carcinogenicity to humans. Studies of workers exposed to glass wool, continuous glass filament, rock (stone), and slag wool have not provided consistent evidence of an association between exposure to fibers and risk for lung cancer or mesothelioma. There is limited epidemiological data to permit an adequate evaluation of the cancer risk associated with exposure to refractory ceramic fibers. In chronic inhalation studies, ceramic fibers have produced an increase in the incidence of mesothelioma in hamsters and an increased incidence of lung tumors in rats. Because of persistent uncertainties, occupational exposures to SVFs should be lowered as much as possible with engineering controls, proper worker training, and safe work practices.

Individual exposures to iron dusts, particularly those resulting from steel-grinding operations, welding, or foundry work, are common. The only clinical effect of pure iron oxide exposure is a reddish-brown coloring of the sputum. Lung function tests show no clinical abnormality, whereas chest X-rays show many small (0.5 to 2.0 mm) opacities without confluence (as with stannosis; see Fig. 18-3). Lung sections show macrophages laden with iron dust but without fibrosis or cellular reaction. With removal from further iron oxide dust exposure, the radiographic abnormalities slowly resolve. Similar results can be seen in exposures to tin, barium, and antimony.

Chronic Beryllium Disease

Chronic beryllium disease (CBD) is the most well-known and best-characterized occupational granulomatous lung disease. It is a systemic disease that affects the lungs disproportionately to other organs. It develops in workers who make metal alloys containing beryllium, a strong, lightweight, nonmagnetic metal used in industries such as aerospace and defense, high-end audio and electronics equipment, and nuclear weapons design. Workers are exposed by inhaling beryllium-containing dust, which can result in immune sensitization in susceptible workers. Sensitization can then progress to CBD, but not all sensitized workers develop symptomatic disease.

A granuloma is a cellular immune response to an inciting agent in which immune cells organize into microscopic clusters or nodules. Macrophages or multinucleated giant cells (fused macrophages) are at the core of the granuloma, and other immune cells, such as lymphocytes and neutrophils, as well as fibroblasts and collagen may also be present. Granulomata can be the result of infectious agents, usually *M. tuberculosis* or NMTB, or noninfectious agents.

Chronic beryllium disease may be clinically indistinguishable from sarcoidosis, an idiopathic systemic granulomatous disease that also affects the lung disproportionately. Serum angiotensin-converting enzyme levels may be elevated, as in sarcoidosis, although this is not a highly sensitive test in either disease. A more specific test to determine cell-mediated immunity to beryllium is the beryllium lymphocyte proliferation test (BeLPT) on peripheral lymphocytes or lymphocytes from bronchoalveolar lavage samples. The BeLPT is a highly specific and sensitive test for sensitization to beryllium, but alone it is not diagnostic of CBD.

As in sarcoidosis, the pattern of pulmonary function testing can be extremely variable. Most patients with CBD develop restrictive disease, often with impaired gas exchange marked by a decrease in diffusing capacity. However, obstructive disease, isolated gas exchange abnormalities, and normal pulmonary function tests can all be seen. Chest X-rays or computed tomography scans frequently show hilar and/or mediastinal lymphadenopathy, as in sarcoidosis, and a variety of parenchymal findings, such as nodules and opacities with a "ground-glass" appearance.

Transbronchial or surgical lung biopsies or samples of affected lymph nodes will show non-caseating (non-necrotic) granulomata, which, together with evidence of sensitization to beryllium (such as a positive BeLPT), are generally diagnostic of CBD.

Genetic polymorphisms in the major histocompatibility (MHC) complex have been identified as a major susceptibility factor for sensitization to beryllium. In a small series of CBD cases and controls, polymorphisms in the human leukocyte antigen gene HLA-DPB1 have been identified as conferring susceptibility.[35]

MHC (HLA) Class II antigens are involved in the T cell response to external antigens. T cells are also part of the immune response that results in granuloma formation. Although BeLPT has been used to monitor workers potentially exposed, genetic testing of prospective workers remains a controversial ethical question, and thus far, this discovery has not been used routinely to identify workers at risk for beryllium sensitization, and thus CBD.

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19

Neurologic and Psychiatric Disorders

Edward L. Baker, Jr., and Nancy L. Fiedler

A 29-year-old man was seen after 8 years of employment in a chloralkali plant, where he was primarily employed in maintenance and operation of the electrolytic cells. Four years after beginning work in the plant, he began to notice increased nervousness and irritability. His nervousness continued for 2 years; he then began to experience episodes of severe depression. At that time, he also experienced a tremor of the hands, bleeding gums, easy fatigability, increased salivation, and loss of appetite. He sustained an injury to his left Achilles tendon and was away from work for 7 months, during which time most of his symptoms improved, but tremulousness, nervousness, and depression remained.

This man and his wife reported that before his employment at the plant he was outgoing, calm, and patient. He had been a military policeman in the U.S. Marines and did not experience emotional upsets during this tour of duty despite significant stress.

Urine mercury monitoring, which had been performed by his employer during the entire period of employment, had demonstrated numerous values over 500 µg/L, the highest of which was 736 µg/L in his fifth year of employment (normal range in the general population = 5 to 30 µg/L).

Physical examination performed at the end of his 7-month absence from work showed no evidence of tremor, a mild loss of pinprick sensation on the dorsal aspect of his arms, and an otherwise normal neurologic examination. Lines of increased pigmentation were observed at the gingival margins of several teeth.

Neuropsychological testing showed mild defects in his ability to perform mental calculations and in his immediate verbal and visual memory. Written spelling was particularly impaired, with an inability to copy simple sentences. He could not concentrate on various tasks and, as a result, his performance was erratic, with incorrect answers to simple questions and correct answers to more difficult ones. He was emotionally labile in the test situation, appearing anxious and depressed. He displayed average performance on tests of manual dexterity.

This patient's illness was manifested primarily by emotional disturbances and deficits on standardized tasks of psychological performance. He showed no particular deficits in memory, psychomotor performance, learning ability, or recall of current events. His most striking deficit was one of impaired concentration, which resulted in erratic performance on various tests. These effects were still detected months after he was removed from mercury exposure.

The occurrence of neurobehavioral and psychiatric disorders among workers in various occupations and among people exposed to neurotoxins in their communities is of increasing concern.

The neurotoxic effects of chemicals such as lead or mercury are well known, but as new substances are introduced into industry and commerce, neurologic and psychiatric disorders associated with them are being recognized. For example, in the 1970s, an industrial catalyst, dimethylaminopropionitrile (DMAPN), was found to cause an autonomic neuropathy affecting the bladder in workers producing polyurethane foam.[1] Another such discovery occurred when peripheral neuropathy was diagnosed in employees of a coated-fabrics plant and traced to the solvent, methyl n-butyl ketone (MBK). In some instances, as in the aforementioned case study, specific chemical substances are responsible for characteristic pathologic processes within the nervous system. However, in many other situations, workers have symptoms characteristic of both neurologic and psychiatric disease due to exposure to mixtures of chemicals and adverse psychosocial factors.

NEUROLOGIC DISORDERS

For more than 100 years, exposure to toxins has been known to affect behavior. During the past 50 years, quantitative methods applied to the study of behavioral abnormalities during and after toxin exposure have demonstrated a wide range of clinical and subclinical effects for numerous substances. Neuroimaging techniques, such as positron emission tomography (PET), single photon emission computed tomography (SPECT), and functional magnetic resonance imaging (fMRI), are helping to improve our understanding of the impact of neurotoxic agents on the central nervous system, and nerve conduction studies are helping to quantify dysfunction of the peripheral nervous system. Many neurotoxic agents produce a dose-related spectrum of impairment, ranging from mild slowing of nerve conduction velocity or prolongation in reaction time to more serious neuropathy and encephalopathy.

Pathophysiology

Peripheral Nervous System (PNS) Effects

Two basic forms of damage to peripheral nerves occur as a result of exposure to neurotoxins: segmental demyelination and axonal degeneration. Segmental demyelination results from primary destruction of the neuronal myelin sheath, with relative sparing of the axons. This process begins at the nodes of Ranvier and results in slowing of nerve conduction. Characteristically, there is no evidence of muscle denervation, although disuse atrophy may occur if paralysis is prolonged. As remyelination begins, recovery is rapid and usually complete.

Axonal degeneration is associated with metabolic derangement of the entire neuron and is manifested by degeneration of the distal portion of the nerve fiber. Myelin sheath degeneration may occur secondarily. Nerve conduction rates are usually normal until the condition is relatively far advanced. Distal muscles show changes of denervation. Recovery may occur by axonal regeneration, but it is very slow and often incomplete.

In some instances, axonal degeneration and segmental demyelination coexist, presumably as a result of secondary effects derived from damage to each system. Therefore, although the classic descriptions of these syndromes hold in experimental models, the clinical manifestations of neuropathy in exposed individuals may represent a combination of both pathologic processes. In addition, it is not clearly understood at what dose or after how long a duration of exposure these syndromes occur.

Central Nervous System (CNS) Effects

Lead, chlordecone (Kepone), carbon monoxide, organic solvents, and other chemicals can significantly disrupt the metabolism of neurotransmitters, affecting dopamine, norepinephrine, gamma-aminobutyric acid (GABA), and serotonin. This disruption of neurotransmitters correlates with behavioral aberrations in experimental animals. In addition, many industrial solvents cause acute depression of synaptic transmission in the central nervous system, resulting in drowsiness and weakness. Such mechanisms may be responsible for the manifestations of CNS toxicity induced by neurotoxic substances.

Combined Peripheral Nervous System and Central Nervous System Effects

Certain neurotoxins cause distal degeneration of axons in both the CNS and PNS. This form of axonal degeneration was originally described as

"dying back" neuropathy. In view of the association of CNS and PNS degeneration, it has been suggested that this process be referred to as "central-peripheral distal axonopathy." Substances associated with this effect include acrylamide, n-hexane, MBK, carbon disulfide, and organophosphorus compounds, most notably triorthocresyl phosphate (TOCP).

Characteristically, distal degeneration occurs within the long nerve fiber tracts of both the CNS and PNS. Once degeneration begins peripherally, it becomes more severe in the initially affected nerve segments while progressing centrally to involve more proximal segments of nerve fibers. Within the spinal cord, the long ascending and descending tracts (the spinocerebellar and corticospinal tracts) appear to be the most severely affected. Involved fiber tracts demonstrate axonal swellings, which are often focal and are associated with neurofilament accumulation within the axon. Although the length of the axon is a key determinant of fiber susceptibility, fiber diameter may also be important: Large-diameter, myelinated fibers are more frequently affected.

The precise locus of the metabolic derangement that is responsible for these manifestations of axonal damage is unknown. Chemical substances may bind to the inactive intraaxonal enzyme systems required for maintenance of normal axonal transport mechanisms. Magnetic resonance imaging may be useful in the clinical evaluation of individuals with combined central and peripheral effects of toxin exposure.

Manifestations

Peripheral Nervous System

Virtually all of the toxins that affect the peripheral nervous system cause a mixed sensorimotor peripheral neuropathy. The initial manifestations of this disorder consist of intermittent numbness and tingling in the hands and feet; motor weakness in the feet or hands may develop somewhat later and progress to the development of an ataxic gait or an inability to grasp heavy objects. Although the distal portion of the extremities is involved initially and to a greater degree, severely affected patients may also have proximal muscle weakness and muscle atrophy. Nerve biopsies in affected persons have shown axonal swellings and paranodal myelin retraction. Extensor muscle groups usually manifest weakness before flexors do.

Although the manifestations are somewhat similar from one toxin to another, certain specific characteristics are unique to individual agents (Table 19-1). Painful limbs and increased sensitivity of the feet to touch are particularly characteristic of arsenical neuropathy. Sensory involvement predominates in the relatively rare neuropathy seen with alkyl mercury poisoning. Both motor and sensory disorders are observed in the neuropathies associated with exposure to n-hexane, MBK, and acrylamide.

The peripheral neuropathy associated with lead exposure is unusual because only the motor system is involved. The most characteristic early manifestation of peripheral neuropathy due to lead is wrist extensor weakness. Reports of involvement of the lower extremities, resulting in ankle drop, were made during the 1930s, when cabaret dancers consumed lead-contaminated illicit whiskey and developed lead neuropathy in the muscles that they used most actively. Overt wrist drop, which was a characteristic manifestation of lead neuropathy in reports of many years ago, is rare today. Significant lead exposure more frequently results in associated changes in electromyograms (EMGs).

The development of these syndromes is usually insidious. Very slow development of numbness and tingling of the fingers and toes occurs over several weeks and may then be followed by motor weakness. With several toxins, including acrylamide, n-hexane, and MBK, the neuropathy may progress even after the worker is removed from exposure. This deterioration may continue for 3 to 4 weeks; at that point, recovery may begin. The duration of the recovery process is proportional to the degree of severity of neuropathy: Less severely affected patients may experience total resolution in 3 to 6 months, whereas those with advanced disease may continue to have signs and symptoms 1 to 2 years later.

Physical examination of affected workers shows a characteristic distribution of sensory loss, particularly to pain and temperature discrimination (Fig. 19-1). Frequently, vibration sensation is impaired, and touch perception,

Table 19-1. Peripheral Nervous System Effects of Occupational Toxins*

Effect	Toxin	Comments
Motor neuropathy	Lead	Primarily wrist extensors Wrist drop and ankle drop rare
Mixed sensorimotor neuropathy	Acrylamide	Ataxia common Desquamation of hands and soles Sweating of palms
	Arsenic	Distal paresthesias earliest symptom Painful limbs (especially in calves) Hyperpathia of feet Weakness prominent in legs
	Carbon disulfide	Peripheral neuropathy (mild) CNS effects more important
	Carbon monoxide	Only seen after severe intoxication
	DDT	Only seen with ingestion
	n-hexane	Distal paresthesias and motor weakness Weight loss, fatigue, and muscle cramps common
	Methyl n-butyl ketone	Distal paresthesias and motor weakness Weight loss, fatigue, and muscle cramps common
	Mercury	Predominantly distal sensory involvement More common with alkyl mercury exposure
	Organophosphate insecticides (selected agents)	Delayed onset following single exposure (usually nonoccupational)

*Includes most, but not all, neurotoxic substances associated with listed conditions.
CNS, central nervous system.

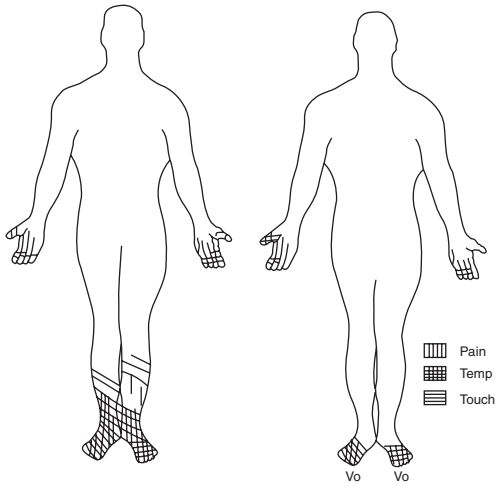


Figure 19-1. Two cases of methyl n-butyl ketone (MBK) neuropathy, representing range of losses in cutaneous and vibration sensation. (*Source*: From Allen N, Mendell JR, Billmaier DJ, et al. Toxic polyneuropathy due to methyl n-butyl ketone. Archives of Neurology 1975; 32: 209–218.)

particularly with acrylamide poisoning, is lost. Tremor of the hands is particularly common in several types of chemical intoxication, usually a resting tremor that is not increased with movement. The tremor seen with chlordecone poisoning is a common manifestation of the disease and has characteristic features: It is irregular, nonpurposive, and most severe when the limb is static but unsupported against gravity. In contrast, the fine tremor associated with mercury poisoning often affects the eyelids, tongue, and outstretched hands. Motor weakness in toxic neuropathies is often found in distal muscles of the arms and legs (Fig. 19-1). Intrinsic muscles of the hands and feet are particularly affected in neuropathies caused by n-hexane, MBK, and acrylamide. Extensor weakness of the forearms is characteristic of lead neuropathy. Impaired coordination is often seen in persons with motor weakness in the extremities; cerebellar pathology need not be present for these manifestations to occur. In summary, distal sensory and motor

impairment characterized by numbness and weakness of the hands and feet is followed by more proximal involvement as the toxic neuropathy develops.

Other Neurologic Manifestations

A wide variety of additional manifestations may be seen that are specific to individual toxins (Table 19-2). Movement disorders that resemble Parkinson disease have been reported in persons exposed to carbon disulfide, carbon monoxide, and manganese; hypotonia, dystonia, and other disorders of locomotion occur in persons with excessive exposure to these substances.

Opsoclonus, a characteristic abnormality of eye movements, can be caused by exposure to chlordecone. It consists of irregular bursts of involuntary, abrupt, rapid jerks of both eyes simultaneously; these movements usually are horizontal, but in severely affected persons they are multidirectional.

Seizures are often seen in workers with acute excessive exposure to industrial toxins. Organochlorine insecticides, such as dichlorodiphenyltrichloroethane (DDT) and chlordane, have been associated with seizures after acute ingestion of a large dose. Seizures are a rare manifestation of lead encephalopathy in adults.

Cranial nerve involvement is uncommon with peripheral neurotoxins. However, trichloroethylene has a predilection for the trigeminal nerves and has been associated with facial numbness and weakness. Carbon disulfide exposure is also associated with cranial neuropathies.

As noted in the case study earlier, an unusual manifestation of neurotoxicity occurred in a group of workers exposed to DMAPN.[1] This substance caused an autonomic neuropathy in the bladder, resulting in urinary retention, urinary hesitancy, and sexual dysfunction. Although symptoms and signs improved after cessation of exposure, some workers had persistent symptoms and signs for at least 2 years after cessation of exposure.

Table 19-2. Other Neurologic Manifestations of Occupational Toxins*

Manifestation	Agent
Ataxic gait	Acrylamide
	Chlordane
	Chlordecone (Kepone)
	DDT
	n-Hexane
	Manganese
	Mercury (especially methyl mercury)
	Methyl n-butyl ketone
	Toluene
Bladder neuropathy	Dimethylaminopropionitrile
Constricted visual fields	Mercury
Cranial neuropathy	Carbon disulfide
	Trichloroethylene
Headache	Lead
	Nickel
Impaired visual acuity	n-Hexane
	Mercury
	Methanol
Increased intracranial pressure	Lead
	Organotin compounds
Myoclonus	Benzene hexachloride
	Mercury
Nystagmus	Mercury
Opsoclonus	Chlordecone (Kepone)
Paraplegia	Organotin compounds
Parkinsonism	Carbon disulfide
	Carbon monoxide
	Manganese
Seizures	Lead
	Organic mercurials
	Organochlorine insecticides
	Organotin compounds
Tremor	Carbon disulfide
	Chlordecone (Kepone)
	DDT
	Manganese
	Mercury

*Includes most but not all of the neurotoxic substances associated with listed conditions.

Diagnosis

Electrophysiologic tests that assess peripheral nerve function, including EMGs and measurements of nerve conduction velocity (NCV), are useful tools for assessing the extent and severity of neurologic disorders in workers exposed to industrial toxins. Noninvasive techniques that measure sensory perception thresholds for vibration and temperature have been developed to monitor diabetic patients for the occurrence of sensory neuropathy; these are also efficient tools to screen individuals who have had significant exposure to neurotoxic agents or have early sensory symptoms. Since EMG and NCV testing assess the function only of large fibers, these tests

of sensory perception threshold, which assess small fiber function, can be valuable in diagnostic evaluations. In addition to detection of toxic neuropathy, these tests may be useful in detection of compression neuropathies, such as carpal tunnel syndrome (Chapter 16).

Electroencephalograms (EEGs) have also been used in the evaluation of workers exposed to neurotoxins, but these tests usually are not as useful as nerve conduction tests. Electroencephalograms may help assess altered states of consciousness of unknown cause. A more promising extension of EEG use is the measurement of cortical-evoked potentials after auditory or visual stimuli, which may be prolonged after exposure to certain chemicals; for example, workers chronically exposed to n-hexane may have prolonged latency of visually evoked responses.

Table 19-3. Behavioral Effects of Occupational Toxins*

Manifestation	Agent
Acute psychosis or marked emotional instability	Carbon disulfide Manganese Toluene (rare)
Acute intoxication	Organic solvents Carbon monoxide
Chronic behavioral symptoms	Acrylamide Arsenic Lead Manganese Mercury Methyl n-butyl ketone Organotin compounds
Chronic toxic encephalopathy	Organic solvents Styrene Lead Carbon disulfide

*Includes most but not all of the neurotoxic substances associated with listed conditions.

BEHAVIORAL DISORDERS

Manifestations

Excessive exposure to industrial toxins may result in behavioral effects ranging from mild symptoms of fatigue to persistent impairment of nervous system function. In view of the nonspecific nature of many behavioral manifestations of neurotoxin exposure, standardized psychometric testing has greatly facilitated the evaluation of these disorders. In general, neurotoxins may affect psychomotor performance, manifested by slowed response time, impaired eye–hand coordination, and diminished concentration ability. Neurotoxins may also cause emotional effects, including irritability, depression, and, at times, emotional liability. Memory and attention may be impaired. Toxins do not usually affect other aspects of cognitive functioning, such as remote memory and fund of general information.

Although few toxins have unique behavioral effects, several substances deserve particular attention (Table 19-3). Carbon disulfide affects all levels of the CNS and may result in bizarre clinical syndromes, including acute psychosis. Neurotoxins may cause both behavioral effects and peripheral neuropathy in the same person.

Most chlorinated hydrocarbon solvents in current use in industry cause a relatively brief "high" after exposure to significantly elevated concentrations in air. Intentional abuse of industrial solvents by individuals desiring these intoxicating effects can cause permanent damage to the PNS and CNS.

Attention to cognitive reserve capacity is important in studying and managing the impact of exposure to neurotoxic chemicals on cognitive function.[2] Cognitive reserve serves as a buffer to manifesting overt symptoms after an insult to the brain. Workers may sustain permanent brain damage due to exposure to toxic substances, but the presence of cognitive reserve may delay appearance of symptoms of clinical significance.[3] Cognitive reserve capacity, like pulmonary reserve capacity, may mask symptoms until later in life, when cognitive reserve diminishes due to aging processes. This phenomenon is relevant to the delayed appearance of symptoms in adults due to toxicity of lead[4] and organic solvents.

Diagnosis

Use of standardized psychometric tests, using measures of memory, intelligence, attention, dexterity, reaction time, personality, and general psychomotor function, helps evaluate exposed

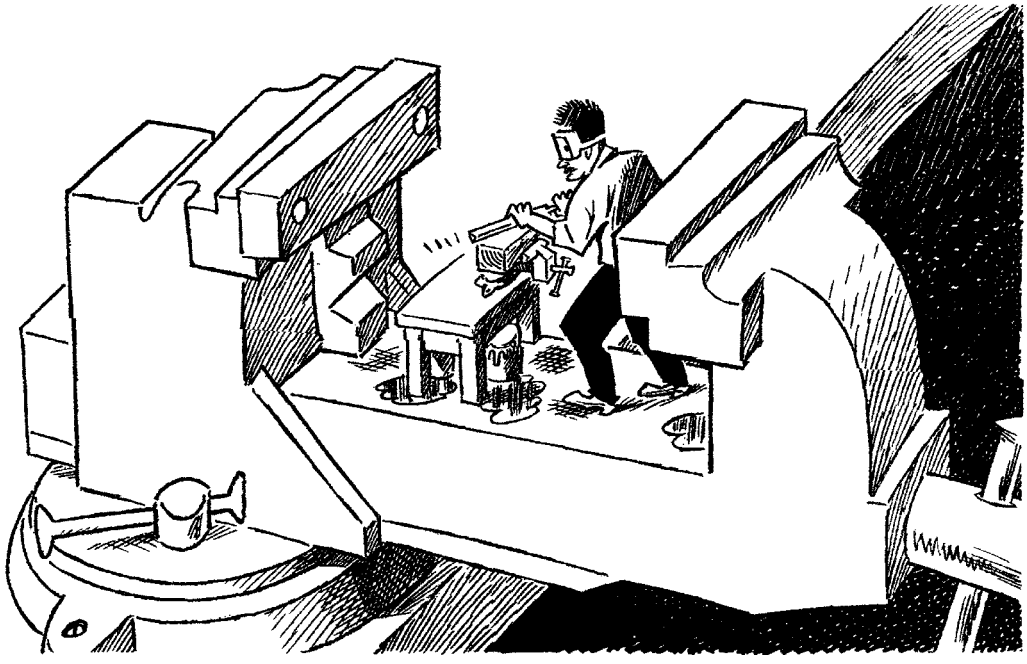
individuals and groups.[5] To facilitate reproducibility in testing of groups and to improve data-handling efficiency, these tests have been adapted for computer administration in epidemiologic and clinical research. Interpretation of test results must consider confounding factors, such as age, education, alcohol consumption, and preexisting neurologic disease. However, the most important feature of the diagnostic process is a carefully obtained occupational history (Chapter 2) that identifies specific neurotoxins and assesses the magnitude and duration of exposure to each. The work history is particularly important in evaluating behavioral disorders, because these conditions are often attributed to factors unrelated to work.

PSYCHIATRIC DISORDERS

There are few epidemiologic studies of the prevalence of psychiatric disorders in the workplace. Occupations associated with higher rates of admission to psychiatric treatment facilities have been identified.[6] For example, health care workers have higher rates of both suicide and admissions to hospitals and mental health centers, although this could be explained by demographic factors or their increased awareness and acceptability of mental health problems. Lawyers, teachers, counselors (except college counselors), and secretaries have elevated rates of major depression.[7]

Employees in low-status positions demonstrate higher rates of psychiatric disorders than those in high-status positions.[8] Workers in low-status positions often experience high job demands with little control over job decisions—characteristics associated with job strain.[9,10] (See Chapter 13.) Although high workload is perceived to be a risk factor for psychiatric illness, it is often difficult to separate workers' perceptions of job characteristics from their negative affective traits; for example, depressed employees may perceive their workload as heavier than healthier workers do. Furthermore, high workload is not consistently associated with increased risk for all psychiatric disorders.

Other psychosocial factors that may contribute to stress and psychiatric disorders among workers include conflicting demands and poor social support, both of which may exert a greater detrimental effect than high workload.[10,11] High social support at work (high levels of support



(Drawing by Nick Thorkelson.)

from colleagues and supervisors coupled with clear and consistent information from supervisors) and skill discretion (job variety and the opportunity to use skills at work) have been shown to decrease absence due to psychiatric illness. Fair decision-making procedures at work may also contribute to a lower incidence of psychiatric disorders.[12]

Chemical Hazards

The causal relation between neurotoxic exposure and psychiatric symptoms is unclear. Although associations between neurotoxicant exposure and psychiatric symptoms have been reported, a major study did not find higher prevalence of depression among workers in occupations expected to have greater chemical exposures, such as construction laborers and metal workers.[7] Because cognitive deficits and various physical symptoms are also associated with exposure, it is possible that recognition of these impairments may lead to reactive depression. Alternatively, psychiatric responses may reflect CNS dysfunction within the frontal, temporal, and limbic regions that is a direct result of exposure. For example, some studies of workers chronically exposed to organic solvent mixtures document increased incidence of mood and anxiety disorders. Brief reactive psychosis has been reported in cases where acute high-level exposures to solvents and some metals have occurred. More studies are needed to better understand the pathogenesis of psychiatric disorders, such as depression, that may result from work-related exposures to neurotoxicants.

Physical and Psychosocial Hazards

Exposure to physical agents in the workplace may lead to psychiatric symptoms. For example, factory workers exposed to high levels of noise have depressive symptoms, such as insomnia, anxiety, and weight loss.[13] Shift workers have low subjective levels of physical health and well-being,[14,15] and they have high rates of alcohol and substance abuse and neuroticism.[16,17] It is not clear whether depression is increased in shift workers.

The proliferation of conditions characterized by nonspecific symptoms has expanded the range of potential mental health concerns within the workplace. For example, some workers who spend much time viewing computer screens have reported hypersensitivity to electromagnetic fields and radiofrequency waves, with eye irritation, headache, fatigue, dermatologic problems, memory impairment, nausea, and mucosal irritation.[18] Factors that may influence nonspecific symptom reporting, in addition to hazardous workplace exposures, include personality style, individual attitudes and belief systems, premorbid psychiatric status, and social pressures, such as employees' perceptions of managers' competence.[19]

Trauma in the Workplace

Traumatic workplace events include serious and fatal injuries and workplace violence, ranging from threats and harassment to murder.[20] These events often have significant economic and psychological consequences. Homicide is the second leading cause of occupational death, after work-related motor vehicle accidents. Certain workplace factors—such as exchanging money, interacting with the public, working at night or in the early morning, delivering goods, and working alone—place employees at high risk for violent attacks.[20]

Controlling the risk of psychological trauma in the workplace presents a challenge. Although there is no specific profile of a violence-prone employee, possible early warning signals include paranoid behavior, desperation over recent personal problems, an inability to accept criticism of job performance, blaming others for problems, and direct or subtle threats of harm.[21] The work environment may increase the risk of workplace violence. For example, a work environment in which the dignity of employees is not respected, such as one with frequent invasions of privacy or high levels of secrecy, or an authoritarian management style may play a role in leading a potentially violent employee to commit a violent act.

A potential consequence of trauma in the workplace is posttraumatic stress disorder (PTSD), which occurs after exposure to a traumatic event involving threatened death or serious injury to self or others, such as witnessing the death of a co-worker. Response to such an

event involves feelings of intense fear, helplessness, and horror. Symptoms of PTSD include intrusive thoughts or dreams and recollections of the trauma, reexperiencing of the trauma, and avoidance of stimuli that arouse recollection of the trauma. Posttraumatic stress disorder in various forms may be involved in chronic pain or posttraumatic head injury syndromes. Posttraumatic stress disorder was first recognized among veterans of the Vietnam War, a unique occupational setting. The extreme traumas of war are not often seen on such a scale within nonmilitary workplaces, although workers in certain occupations, such as police, firefighters, and emergency medical technicians, are at high risk for psychological trauma.[22]

EFFECTS OF SELECTED NEUROTOXINS

Lead

The most commonly encountered workplace substance with clearly recognized neurotoxic effects is lead (see Chapters 11, 20, and 23). The National Institute for Occupational Safety and Health (NIOSH) has estimated that more than 1 million U.S. workers are exposed to lead daily (Fig. 19-2). Outside the workplace, lead exposure in the community impacts the health of millions more through exposure to lead in air, drinking water, dust, and peeling paint.[23] The most common neurologic finding is impaired CNS function, manifested by symptoms of fatigue, irritability, difficulty in concentrating, and inability to perform tasks requiring sustained concentration. These symptoms are associated with abnormalities on standardized neuropsychological testing that indicate impairment of verbal intelligence, memory, and perceptual speed. Symptoms of arm weakness, characteristically affecting extensor muscle groups, are also seen in the early phases of lead toxicity. Often, weakness occurs before abnormalities are seen on nerve conduction testing. After removal from exposure, these symptoms and abnormalities may resolve slowly over weeks to months, the duration depending on their initial intensity and other factors.[24] In children, lead exposure has been shown to have an adverse effect on cognitive function, even at relatively low levels of exposure (<10 μg/dL).[25]

Neurologic abnormalities caused by lead exposure usually occur after hematologic toxicity, as manifested by an elevated zinc protoporphyrin

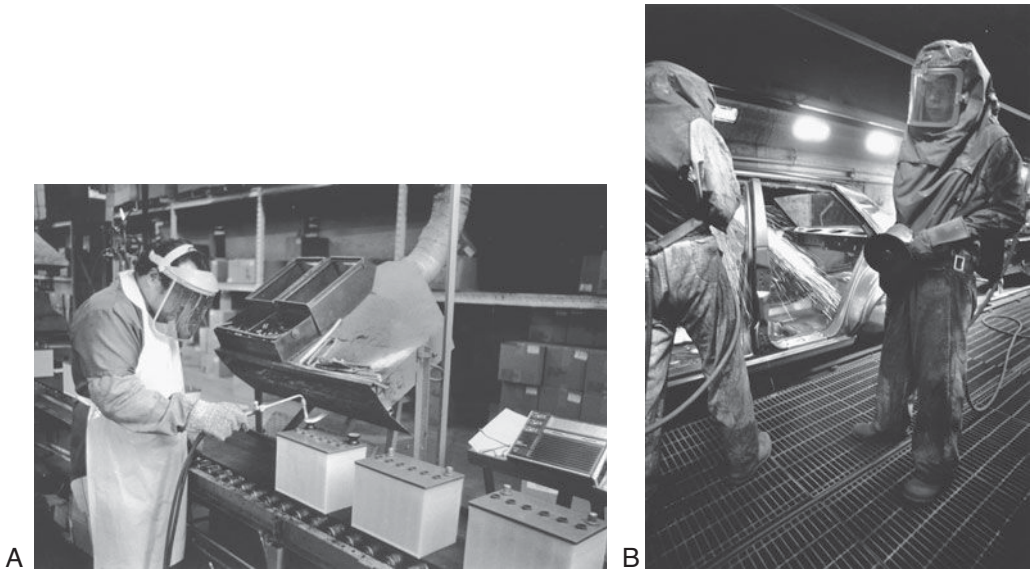


Figure 19-2. Current work practice rules require significant personal and environmental protection in situations of lead exposure: (A) Lead battery worker is protected mainly by local exhaust ventilation. (B) Automobile lead grinder is protected by air-supplied hood and floor exhaust ventilation. (Photographs by Earl Dotter.)

(ZPP) level and a reduced blood hemoglobin concentration. Overt cognitive impairment due to excessive lead exposure may manifest itself after exposure ceases; this latency period between exposure and onset of symptoms occurs as a result of the protective effect of cognitive reserve, as discussed earlier.

Mercury

Although disease as striking as that experienced by Lewis Carroll's Mad Hatter no longer occurs in workplaces in the United States, behavioral effects of exposure to elemental mercury are still seen. Erethism, a set of behavioral symptoms classically associated with mercury toxicity, is characterized by unusual shyness, irritability, and other symptoms. A fine tremor of the hands is associated with mercury poisoning; computer-assisted analysis of EMGs has shown a shift in the frequency of normal forearm tremor as an early manifestation of mercury toxicity. Peripheral neuropathy is not known to occur in elemental mercury poisoning. Measurement of mercury in urine and blood is a useful tool to assess workplace exposure.

Organic mercurials, particularly alkyl mercury compounds such a methylmercury, have a strong affinity for the CNS, and severe neurologic and psychiatric effects have been associated with excessive exposure.[26] The best-described episode of organic mercury poisoning occurred in Minamata, Japan, due to ingestion of fish that had been contaminated by industrial pollution of Minamata Bay with inorganic mercury. Early symptoms of poisoning consisted of distal paresthesias, cerebellar disorders, visual impairment, deafness, and mental disturbances. Sensory deficits occurred, with loss of position sense and impaired two-point discrimination. Some affected individuals had constriction of their visual fields. Mental disturbances were characterized by agitation, alternating with periods of stupor and mutism. The more severely affected patients exhibited dystonic flexion postures.

The devastating and usually irreversible neurologic effects of organic mercury poisoning should be prevented through restriction of mercury use. To that end, the practice of treating seed grain with organic mercurial fungicides has been curtailed by the Environmental Protection Agency (EPA), following outbreaks of neurologic disease in the United States and Iraq among people ingesting food that had been inadvertently contaminated with these fungicides.

Organophosphate Insecticides

Acute organophosphate insecticide poisoning is characterized by the inhibition of acetylcholinesterase, with resultant overactivity of cholinergic components of the autonomic nervous system, inhibition of conduction across myoneural junctions in skeletal muscle, and interference with CNS synaptic transmission. Manifestations of acute toxicity include meiosis (pinpoint pupils), blurring of vision, chest tightness, increased bronchial secretion, and wheezing. Gastrointestinal effects are also seen, including abdominal cramps, nausea, and vomiting. Increased sweating, salivation, and lacrimation are additional characteristic features.

Because organophosphate compounds bind irreversibly to cholinesterase, reactivation of the enzyme system occurs only through synthesis of additional cholinesterase molecules. Therefore, recovery of normal cholinesterase concentrations in red blood cells is slow, and repeated exposure may result in cumulative depression of cholinesterase stores. Recovery after an acute episode of poisoning is usually complete within 7 days unless anoxia has occurred during the acute phase of the episode. Measurement of red blood cell cholinesterase concentrations is valuable during the acute intoxication episode and for surveillance of occupationally exposed workers. Plasma cholinesterase concentrations are of less value in workers because they can be altered by many factors.

A syndrome of delayed neurotoxicity, which develops 8 to 35 days after exposure, has been reported with certain organophosphate compounds. Progressive weakness begins in the distal lower extremities, often with toe and foot drop, followed by finger weakness and wrist drop. Sensory loss is minimal. Deep tendon reflexes are frequently depressed. The disease may progress for 1 to 3 months after onset, and recovery is very slow. There may be psychomotor impairment and abnormal EEG findings.

Evaluation of patients exposed to organophosphate insecticides should include, in addition to

evaluation of manifestations of autonomic nervous system dysfunction, measurement of the red blood cell cholinesterase concentration, which correlates reasonably well with manifestations of clinical toxicity. Migrant workers are at high risk for the acute and chronic effects of exposure to organophosphate insecticides used in agriculture. This population has not been adequately studied and often migrant workers do not receive adequate protection from pesticide exposure.

Organic Solvents

More than 1 million U.S. workers are exposed daily to organic solvents.[27] The most frequently used solvents are toluene, xylene, trichloroethylene, ethanol, methylene chloride, and methyl chloroform (trichloroethane). Although chemically heterogeneous, these compounds are often discussed as a group because of toxicologically similar effects and the high frequency of exposure to various combinations of these substances.

Acute intoxication, with dizziness/lightheadedness or feeling "high," occurs after exposure to excessive concentrations of solvent vapors. Exposure to very high concentrations of solvent vapors or fumes may lead to narcosis with loss of consciousness. Certain psychiatric disorders have been reported following solvent exposure, including somatoform disorder, schizophrenia, and panic disorder.

To facilitate the characterization of persistent health effects of solvent exposure, a nomenclature has been developed by a World Health Organization (WHO) working group[28] and by a workshop of invited experts held in the United States.[29] The mildest form of effect, organic affective syndrome (type 1 solvent health effect), which is typically reversible, is characterized by symptoms of irritability, fatigability, difficulty in concentrating, and loss of interest in daily events. In mild chronic toxic encephalopathy, the symptoms are similar, but abnormalities on neurobehavioral testing are observed. Sustained personality or mood change (type 2a effect) and/or impairment in intellectual function (type 2b effect) may be seen. Type 2 effect may be reversible or may lead to permanent cognitive impairment. Severe chronic toxic encephalopathy (type 3 effect) is characterized by dementia, with global deterioration of memory and other cognitive functions that is usually irreversible. Workers exposed to solvents may exhibit any of these three types of effects, depending on the intensity and duration of their exposure. Workers with prolonged exposure to solvents may develop decreases in reaction time, dexterity, speed, and memory. Relatively few abnormalities have been demonstrated in PNS function; nerve conduction abnormalities have been reported in mixed solvent exposure. Measurement of urinary metabolites, such as hippuric acid in toluene exposure, may be useful in monitoring exposed populations.

Clinical diagnosis of chronic toxic encephalopathy caused by exposure to organic solvents is made by obtaining a careful exposure and clinical history and by performing standardized neurobehavioral tests.[27] Workers exposed to solvent vapors at excessive levels—sufficient to frequently cause acute intoxication—are at increased risk of toxic encephalopathy,[27,30,31] and should be provided a thorough medical and neurobehavioral assessment to determine whether symptoms or signs of neurobehavioral dysfunction are present. They may demonstrate a symptom-free latency period in which chronic symptoms of cognitive impairment, such as memory and attention deficits, are not noted following cessation of exposure for months or even years after exposure ends, only to appear at a later time. This course of illness results from the protective effect of cognitive reserve,[2] which diminishes as a result of the aging process, thereby unmasking the symptomatic manifestations of brain damage caused by neurotoxin exposure during an earlier period.

Carbon Monoxide

Sources of carbon monoxide (CO) that may cause poisoning include exhaust fumes from motor vehicles, inhaled smoke, malfunctioning heating systems, and cleaner fuels, such as propane and methane. Psychological symptoms of CO poisoning include fatigue, apathy, emotional liability accompanied by lowered frustration tolerance, impulsivity, irritability, and, at times, psychosis. Psychological symptoms are often delayed, occurring between 3 and 240 days after

recovery from acute intoxication; approximately 50% to 75% of exposed patients recover from this delayed syndrome within 1 year.[32]

Manganese

Excessive exposure to manganese has long been known to cause profound psychosis ("manganese madness") and/or a movement disorder consistent with parkinsonism.[33,34] Prominent features of the movement disorder, referred to as manganism, include impairment of gait, decreased facial expression, tremor of the hands, and impaired speech. Some improvement has been noted following treatment; however, this benefit has been variable in other studies and is often transient.[35] Significant exposure to manganese occurs in various occupations, including welding; manganese in welding fume constitutes a significant neurotoxic risk if not properly controlled. Recent studies linking manganese exposure from welding and increased rates of parkinsonism deserve particular attention.

MANAGEMENT AND CONTROL OF NEUROLOGIC AND BEHAVIORAL DISORDERS

Management of toxin-induced neurologic problems consists primarily of identification of the offending agent and removal from continued exposure. Probably the best example of a successful public health intervention is the dramatic reduction of lead exposure in the United States, largely due to removal of lead from gasoline. Evidence of lead-related cognitive impairment resulted in major policy change, ultimately protecting children who are most vulnerable to the effects of lead. This use of sound science to shape prudent public health policy can apply by extension to other environmental hazards.[24]

With regard to the workplace, medical management of signs and symptoms may also be of value over the long term. In some instances, removal of the offending agent from the workplace may prevent the development of new cases. Some workers with known exposure may develop mild, early symptoms of neurotoxicity; objective demonstration of functional impairment on standardized tests is essential in the management of individual cases. Workers with evidence of toxin-related symptoms or functional impairment should be removed from exposure until these deficits resolve and exposure in the workplace is terminated.

Prevention of occupationally induced neurologic disorders can be accomplished through workplace medical and environmental control programs. The goal of environmental control is to reduce concentrations of neurotoxic substances in the work environment by various manipulations. Medical strategies designed to reduce neurologic morbidity include preplacement evaluation and periodic medical monitoring. The goal of preplacement evaluation as it relates to neurologic disorders is to avoid placement of workers with preexisting disease, such as peripheral neuropathy, in jobs with exposures that might exacerbate these conditions. Conditions that might impair a worker's ability to perform a job, such as uncontrolled epilepsy in a person operating hazardous machinery, would be grounds for medical exclusion from such jobs.

Periodic medical monitoring programs are becoming more common in industries where neurotoxins are used. An important element of such programs is measurement of the neurotoxic agent in biologic fluids. The most common such application occurs in industries where lead or mercury is used.

Periodic monitoring of lead-exposed workers should include occupational and medical histories; a physical examination with special attention to the nervous system; blood and urine studies to evaluate hematologic and renal effects of lead exposure; and, most importantly, determination of the blood lead level (BLL) and concentrations of zinc protoporphyrin (ZPP). The content of such examinations is mandated by the Occupational Safety and Health Administration (OSHA) standard on occupational exposure to lead, in which specific guidelines are provided for job transfer of workers with elevated BLLs. The standard requires that employers make routine BLL monitoring available to all employees who are exposed to lead at concentrations greater than the action level, regardless of whether respirators are worn. Specific actions must be taken depending on the results of BLL testing. Any worker removed from a job because

of an elevated BLL is protected by the medical removal protection provision of the OSHA lead standard. This provision requires an employer to "maintain the worker's earnings, seniority, and other employment rights and benefits (as though the worker had not been removed) for a period of up to 18 months."

The evaluation of mercury-exposed workers is similar, except that determinations are made of mercury in urine and no enzymatic test, such as the ZPP test, exists to measure the metabolic toxicity of mercury exposure. Workers exposed to cadmium and arsenic should be monitored periodically with urinary determinations of these metals in addition to standard medical evaluations.

Workers chronically exposed to solvents should have periodic medical histories and examinations with attention to the nervous system. Measurement of urinary metabolites of solvents is sometimes helpful as an adjunct to other medical monitoring techniques.

Pesticide-exposed workers, particularly those using organophosphate insecticides, should be periodically evaluated, including with red blood cell cholinesterase levels, to assess their degree of pesticide exposure. Nerve conduction testing is not suitable for routine monitoring of asymptomatic workers.

Treatment of occupational neurologic disease beyond removal of the worker from continued toxic exposure may consist of the administration of drugs designed to remove the offending agent or counteract its effects. Chelating drugs, such as ethylene diamine tetraacetic acid (EDTA), dimethylsuccinic acid (DMSA), and penicillamine, are given as treatment for symptomatic poisoning by lead and other heavy metals. These drugs should not be given prophylactically to lower blood levels of the metal; they have known toxicities, which may add to the toxic effects of the metal and also may increase gastrointestinal absorption of the metal. Individuals should be removed from exposure to the offending agent before initiation of drug therapy.

Atropine, a pharmacologic antagonist of organophosphates, is the drug of choice for treatment of the acute manifestations of organophosphate insecticide poisoning. Repeated doses are given to the point of atropinization, and subsequent doses of the drug may be required since the duration of action of atropine is less than that of organophosphate insecticides. If patients are seen very soon after exposure, oximes can be given to regenerate inhibited cholinesterase enzyme.

Ultimately, prevention of diseases of the nervous system caused by environmental neurotoxins rests on adequate testing of chemicals before their introduction and on environmental measures designed to reduce exposure. The Toxic Substances Control Act (TSCA) addresses the issue of premarket testing, and EPA, which administers TSCA, has specified criteria for neurologic evaluation of chemical substances. Biologic assays of organophosphate compounds have successfully predicted those substances that are neurotoxic to humans. Substances such as n-hexane and MBK, which cause an axonal neuropathy in exposed humans, have been shown to produce similar effects in animals. The neurologic disorder associated with chlordecone was seen in experimental animals several years before it was reported in exposed humans. Therefore, testing of industrial substances by administration of toxins to experimental animals is essential in the identification of substances with neurotoxic potential.

In rare instances, structural similarity alone has proved useful in predicting toxicity. N-hexane and MBK are metabolized to 2,5-hexanedione, which is thought to be responsible for the neurotoxic manifestations of each of these chemicals. Investigation of structure–activity relations therefore may be of value in identifying substances with potential neurotoxicity. In those instances in which neurotoxicity is suspected because of the chemical structure of the compound, animal tests are still required.

MANAGEMENT AND CONTROL OF PSYCHIATRIC DISORDERS

Employee Assistance Programs

Psychiatric disorders often result in time lost from work. In some situations, individuals with mental health problems are more likely to go to work, but they require greater effort to function, suffering from on-the-job impairment.[36]

Therefore, the effects of mental illness may have a more subtle effect on performance. In recognition of the impact that untreated psychiatric disorders have on productivity and morale in the workplace, more than 20,000 U.S. companies offer employee assistance programs (EAPs) to detect and treat employees with psychiatric disorders.[37] Initially, EAPs were developed in response to alcohol-related problems, but it became clear that personal problems beyond substance abuse could also interfere with work performance. Moreover, early detection, before recognition of impaired work performance by a supervisor, is preferable. Therefore, EAPs broadened their scope to include evaluation and referral services for personal problems, such as marital, individual psychiatric, and financial issues. Where broad-based programs are offered, employees are encouraged to seek services on their own or as self-referrals rather than waiting until their job performance suffers. Table 19-4 provides a description of the primary elements of EAPs.

Despite the proliferation of EAPs and widespread claims that EAPs are cost effective, few data are available to address this issue. Evaluation of broad-based EAPs has demonstrated significant improvements in absenteeism, lost time, warnings given to employees, and supervisor ratings of performance after institution of counseling services.[36,37] As managed care has increased, EAP staff members are being asked not only to evaluate and refer troubled employees but also to act as case managers and gatekeepers for utilization of mental health benefits.

Psychiatric Treatment and Productivity

From an organizational perspective, psychiatric disorders impair productivity in several ways, including reducing the number of workers, increasing absenteeism of existing workers, decreasing morale, and reducing quality of work. The annual economic impact of depression in the United States is at least $43.7 billion, including the costs of lost productivity and health care.[38] Although the economic burden of alcoholism is frequently cited, drinking behavior, such as coming to work hung over, in a random undiagnosed sample of employees was also associated with problems at work.[39] The costs of behavioral problems and psychiatric disorders affect not only workers and their families but also managers, employers, and insurance companies.

In sum, psychiatric disorders and associated behavioral problems, such as alcohol consumption, significantly affect productivity—regardless of their cause or their relation to workplace factors and stressors. It appears that when employed persons are treated, their work improves—an encouraging finding that supports the inclusion of psychiatric treatment in health insurance benefits.

Fitness for Duty

When an employee has been out of work because of treatment of a psychiatric disorder, such as depression or anxiety, or a question arises about

Table 19-4. Elements of Employee Assistance Programs (EAPs)

Program type	Internal: EAP staff are company employees
	External: Outside consultant or organization provides EAP
Eligible participants	Employees only
	Employees and eligible dependents
Referral to EAP	Self-refer; voluntary
	Supervisor referral: voluntary or involuntary based on documented poor job performance
Problem type	Substance abuse or dependence
	"Broad brush": substance abuse, psychological, marital, financial, elder care
Service type	800-number telephone evaluation
	One to three evaluation sessions; referral recommendations; crisis intervention
	Short-term treatment (up to 10 sessions)
Supervisor training	Documentation of work performance and referral procedures
	Prevention (stress management skills)

the employee's ability to function on the job, a fitness-for-duty evaluation may be requested. Fitness for duty is defined as the ability of an individual to perform a job, based on the specific job requirements. A detailed understanding of the job duties is required—often a problem, because job descriptions are not necessarily informative or sufficiently behaviorally oriented. Ancillary information, such as interviews with workers in similar positions or with supervisors, may be needed to understand the essential behaviors expected on the job. Fitness for duty can never be based solely on a psychiatric diagnosis; rather, it must be based on a behavioral analysis of the employee's abilities. Past job performance is the best predictor of future job performance. Further, a global assessment of functioning can be useful as a behavioral guide for the individual's current level of functioning and ability to perform daily tasks related to work.[40] Overall, matching of an assessment of the employee's current behavioral functioning with the essential functions required to perform a job, along with consideration of the employee's premorbid level of functioning on the job, yields the best prediction of the employee's fitness for return to the job.

Accommodation in the Workplace

Since the Americans with Disabilities Act (ADA) was passed in 1990, employers have been under increasing pressure to hire and accommodate workers with disabilities, including psychiatric illness. The number of discrimination claims against employers based on emotional or psychiatric impairment has also increased since the passage of the ADA. For example, in 1997, the Equal Employment Opportunity Commission reported that 15% of discrimination claims were related to emotional or psychiatric impairment—the largest category of claims in that year. The need to properly evaluate an individual's ability to perform a job and the ability to make reasonable accommodations is a growing concern among employers in the United States.

The ADA prohibits discrimination based on disability and provides that employers must make "reasonable accommodations" to the disabilities of "qualified" applicants so long as this does not impose "undue hardship." "Qualified" means that the individual can perform the essential functions of the job, except for the disability. "Reasonable accommodation" refers to any modification or adjustment to a job or work environment that allows the qualified employee with the disability to perform the job functions. "Undue hardship" refers to "an action requiring significant difficulty or expense."[41] Employers are not allowed to inquire about a disability before hiring, and the applicant does not have to reveal a psychiatric history at the time of hire. Moreover, if a long-term employee who was previously performing the job develops a psychiatric disorder, the employer is obligated to make accommodations.[42]

Individuals who are hospitalized for psychiatric diagnoses, such as schizophrenia, have low employment rates, often less than 20%.[42] For those who are chronically mentally ill, the best predictors of future work performance seem to be ratings of work adjustment in a sheltered job site, ability to function socially with others, and previous employment history.[43] Therefore, type of psychiatric diagnosis is not as predictive of work capacity as is assessment of objective behavioral performance. Although these findings apply specifically to the psychoses, the same guideline seems to be applicable for any physical or psychiatric illness.

Reasonable accommodations for persons with psychiatric disabilities include analysis of the individual employee's behavioral problems, such as anxiety or sensitivity to criticism, and development of accommodations based on individual needs.[44] For example, when a sensitive employee returns from a hospitalization, the supervisor needs to be trained to offer positive feedback, along with critiques of performance.

A significant number of workers have diagnosable psychiatric conditions, especially depression, and some occupations have higher rates of such disorders that may significantly impact productivity.[45,46] Some occupations appear to place workers at greater risk for traumas that result in psychiatric disorders, such as PTSD. Whatever the causes, psychiatric illness will continue to affect workers and the workplace; therefore, they must be recognized, treated, and accommodated—rather than dismissed or ignored.[47]

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20

Reproductive and Developmental Disorders

Linda M. Frazier and Deborah Barkin Fromer

Hazardous exposures may disrupt reproductive processes in men or women. The challenge is to determine when a particular couple may be at risk, since reproductive problems are common when no toxic exposure can be identified. In the United States, one in seven married couples is involuntarily infertile, and about 1% of all births are conceived using assisted reproductive technologies.[1] Between 10% and 20% of pregnancies end in clinically recognized spontaneous abortion, and rates of earlier, periimplantation loss are even higher. About 3% of newborns have major congenital malformations, and about 10% of school-age children have learning disabilities.[2]

The proportion of adverse reproductive outcomes that is attributable to known reproductive toxicants is thought to be relatively low. This estimate is only partially reassuring. In some developing countries, environmental pollution is substantial and many individuals are occupationally exposed to agents that are known or suspected reproductive toxicants. Hazardous agents and polluting technologies are often used because they are cheaper than safer alternatives. Lack of protective equipment and facilities for washing increases problems such as infertility, birth defects, and congenital brain dysfunction. In developed countries, significant exposures to hazardous chemicals may occur in the workplace or at home through hobbies, household repairs, consumer products, food, or drinking water. Agents such as polybrominated diphenyl ethers (PBDEs) have been banned in Europe but are still used in the United States, resulting in much higher exposure levels.[3] As research methods in reproductive toxicology and epidemiology have become more sensitive, adverse effects have been recognized at lower exposure levels than were previously implicated—and for additional agents.

Childbearing is not the only health outcome that may be harmed by reproductive toxicants. Reproductive hormones are involved in normal functioning of nonreproductive organ systems. Estrogen receptors are present in blood vessels, bone, brain, and heart in both men and women. Androgens influence hematopoiesis, hepatic enzyme function, and calcium exchange in the heart. Research on the genes that regulate these processes may provide insights into reproductive cancers, cardiovascular disease, menopause, osteoporosis, and age-related declines in cognitive function.

Historically, birth defects caused by pharmaceuticals taken during early pregnancy served to break the prevailing belief that the placenta acted as a protective barrier for the fetus. These drugs included thalidomide, which caused limb malformations and other anomalies, and

Box 20-1. DBCP: A Potent Male Reproductive Toxicant

Barry S. Levy

In 1977, a small group of men in a northern California pesticide formulation plant noticed that few of them had recently fathered children. A strong association was found between decreased sperm count and exposure to dibromochloropropane (DBCP), a brominated organochlorine that had been used as a nematocide since the mid-1950s. Testicular biopsies showed the seminiferous tubules to be the site of action and spermatogonia to be the target cell. The relation between reduced sperm count and exposure to DBCP, both in its manufacture and in its use, was confirmed in other studies in the United States and abroad. Follow-up of workers after cessation of exposure showed that spermatogenic function eventually recovered in those less severely affected. However, many of the azoospermic men remained so for many years after cessation of exposure.

Much DBCP was exported by U.S.-based multinational corporations to developing countries. A substantial amount of this pesticide was exported even after DBCP was banned in the United States. Workers exposed to DBCP in developing countries were not informed of its hazards, trained in its use, or provided personal protective equipment to safeguard themselves adequately (Fig. 20-1). In one study of approximately 26,400 DBCP-exposed workers in developing countries who sued U.S. companies, 24% were azoospermic and 40% were oligospermic.

diethylstilbestrol (DES), which caused uterine malformations and vaginal cancer in prenatally exposed women. Mercury and polyhalogenated biphenyl exposures from contaminated food caused severe birth defects, even when the mothers were relatively asymptomatic. In 1977, occupational exposure to dibromochloropropane (DBCP) caused male infertility and alterations in the sex ratio of offspring (see Box 20-1 and Fig. 20-1), demonstrating that reproductive toxicity could affect both women and men.[4]

PRECONCEPTION

Most reproductive studies, whether laboratory studies of experimental animals or epidemiologic studies of men and women, examine the effects of toxicant exposure that begins before conception and continues throughout gestation. Organohalide compounds, such as polychlorinated biphenyls (PCBs), 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), DDT, and PBDEs, cause ongoing exposure because they persist in the fatty tissues of the body.[3,5] If a woman has been heavily exposed to lead in the past, a significant amount is stored in bone and can be released into her bloodstream during pregnancy at levels that affect fetal neurologic development (see Fig. 20-2).[6]

Genetic alterations are a mechanism by which reproductive and developmental processes are harmed. Using bacterial assays, increased mutagenic activity has been detected in the urine of workers exposed to anesthetic gases, chemotherapeutic agents, or epichlorohydrin. Increased frequencies of chromosomal aberrations have been reported in radiation workers and in workers exposed to chemicals such as benzene, styrene, ethylene oxide, epichlorohydrin, arsenic, chromium, and cadmium. While these assays are useful as biological markers of exposure to genotoxicants, they may not predict specific reproductive health effects in individuals.

Structural chromosomal abnormalities in the fetus may have no adverse effects, or they may be associated with birth defects, mental retardation, or other health problems. Numerical chromosomal abnormalities (aneuploidies) are a major cause of spontaneous abortion. For aneuploidies that are compatible with life, infants often suffer physical, behavioral, and intellectual impairments. Both structural and numerical chromosomal abnormalities may originate in either the male or female gamete.[7]

Workplace Exposures among Men

Increased rates of pregnancy loss have been reported in the wives of men who are occupationally exposed to lead, inorganic mercury, organic solvents, pesticides, and other agents (Table 20-1). Male exposure to DBCP or TCDD can cause altered sex ratio in offspring, usually a deficit of male children.[4,8] Certain paternal occupations may increase the risk for congenital malformations, low birthweight, neurodevelopmental disorders, and childhood cancers.

Workplace exposures can be taken into the home on a worker's skin, hair, clothing, or shoes, causing secondary exposure to spouses and children. Mechanisms by which paternally mediated adverse pregnancy outcomes occur include

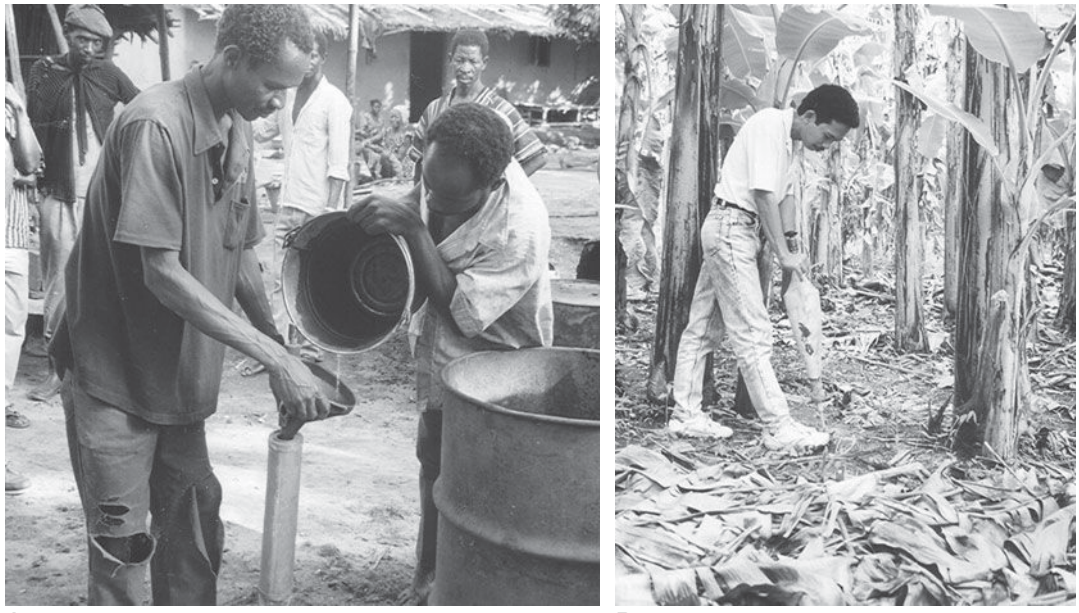

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Figure 20-1. Many workers in developing countries became sterile from exposure to 1,2-dibromo-3-chloropropane (DBCP), even after it was banned in the United States: (A) Simulation of worker pouring DBCP solution, which he has mixed with a stick in a 55-gallon drum, into an applicator. (B) Simulation of worker injecting DBCP solution around the roots of a banana tree. (Photographs by Barry S. Levy.)

Table 20-1. Selected Workplace Exposures with Suspected Effects on Male Reproductive Function

Adverse Effects	Examples*
Decreased libido, hormonal alterations	Lead, mercury, manganese, carbon disulfide, estrogen agonists (such as polychlorinated biphenyls, organohalide pesticides); workers manufacturing oral contraceptives
Spermatotoxicity	Dibromochloropropane (DBCP), lead, carbaryl, toluenediamine and dinitrotoluene, ethylene dibromide, plastics production (styrene and acetone), ethylene glycol monoethyl ether, perchloroethylene, mercury, heat, military radar, chlordecone, bromine, ionizing radiation, carbon disulfide, 2,4-dichlorophenoxy acetic acid (2,4-D), welding
Spontaneous abortion in partner	Solvents, lead, mercury; workers in rubber and petroleum industries
Altered sex ratio in offspring	DBCP, TCDD
Congenital malformations in offspring	Pesticides, chlorphenates, solvents; fire fighters, painters, welders, auto mechanics, motor vehicle drivers, sawmill workers and workers in aircraft, electronics and forestry and logging industries
Low birthweight or preterm birth in offspring	Lead
Neurobehavioral disorders in offspring	Alcohols, cyclophosphamide, ethylene dibromide, lead
Childhood cancer in offspring	Solvents, paints, pesticides, petroleum products; welders, auto mechanics, motor vehicle drivers, machinists and workers in aircraft and electronics industries.

*Some human evidence, albeit limited, has linked each health outcome with the occupations listed and with workplace exposure to at least one of the agents listed; for other agents, animal evidence is available.

Source: Adapted from Paul M, Frazier L. Reproductive disorders. In BS Levy, DH Wegman (eds.). Occupational health: Recognizing and preventing work-related disease and injury (4th ed.). Philadelphia: Lippincott Williams & Wilkins, 2000, pp. 589–603.

Figure 20-2. Adequate control of lead exposure for all workers requires a high level of engineering control and may also require personal protective equipment. Because U.S. law prohibits exclusion of pregnant women from lead work, controls must be sufficient to protect the fetus as well. (*Source*: Saryan LA, Zenz C. Lead and its compounds. In: Occupational medicine (3rd edition). Zenz C, Dickerson OB, Horvath EP, Jr. St. Louis: Mosby Year Book, Inc., 1994, p. 507. Reproduced with permission.)

disruption of sperm production, either directly (by injuring testicular cells) or indirectly (by interfering with the hormonal regulation of spermatogenesis). Sperm cells are vulnerable to genotoxic agents. As long as the stem cell precursors are spared, spermatogenic damage may be reversible over time.

In addition to DBCP, ethylene dibromide, another pesticide, has been associated with post-testicular effects, including decreased sperm velocity, motility, and viability. Occupational exposure verified by urine tests to the organophosphate pesticides ethyl parathion and methamidophos has resulted in aneuploidy in sperm cells, suggesting a genetic mechanism by which paternal exposure to these compounds could cause birth defects.[9]

Other pesticides have also been linked to paternally mediated reproductive problems. Analysis of seasonality showed that miscarriages and birth defects among pesticide applicators were correlated with the spring spraying season.[8] Spontaneous abortion risk was increased two-fold if husbands applied herbicides, but it was increased five-fold if they did not wear protective equipment during application.[10] In another study, measurement of metabolites of DDT in body fat demonstrated a dose–response relationship with birth defects in offspring of men who applied this insecticide in a malaria-control program.[11]

Among the toxic metals, lead has been most intensively studied. Lead may have a direct toxic effect on the gonads and may also impair reproductive endocrine function. Paternal lead exposure has been associated with low birthweight among offspring, mainly when the father's exposure level is high, of long duration, or combined with other exposures, such as solvents. In lead-exposed men, blood lead levels above 40 μg/dL reduce sperm counts and impair sperm motility and morphology.[12] (See Chapters 11 and 19.)

Chemicals have differing potencies for producing adverse effects. The glycol ether 2-methoxyethanol (ethylene glycol monomethyl ether) is an organic solvent that causes testicular atrophy and disruption of the seminiferous tubules; it has been associated with decreased sperm counts in exposed men. A similar solvent, 2-ethoxyethanol (ethylene glycol monoethyl ether, or EGEE), requires a dose 10-fold higher than EGME to cause these effects in animals.

Workplace Exposures among Women

Women's fertility can be impaired by toxic occupational exposures in the periconceptual period (Table 20-2). Women who work in agricultural occupations, especially if they mix and apply herbicides or fungicides, have increased rates of fertility problems. Subfecundity has also been identified among women who work intensively with organic solvents in semiconductor manufacturing facilities, in woodworking workplaces, and in dental offices that handle mercury or

Table 20-2. Selected Workplace Exposures with Suspected Effects on Female Reproductive Function

Adverse Effects	Examples*
Subfecundity	Certain herbicides, fungicides, and organic solvents; mercury; nitrous oxide; agricultural workers, hairdressers, semiconductor manufacture, woodworkers, dental assistants
Menstrual dysfunction	Lead, mercury, shift work, antineoplastic drugs, hairdressers using chemicals, agricultural workers, athletes, dancers.
Spontaneous abortion	Organic solvents such as perchloroethylene, glycol ethers, toluene, xylene, formalin, chloroform, lead, mercury, nitrous oxide, ethylene oxide, antineoplastic drugs, certain pesticides; semiconductor or shoe manufacture workers, laboratory workers, dental assistants, nurses, pharmacists, agricultural workers
Congenital malformations in offspring	Mixed organic solvents, trichloroethylene, halogenated aliphatic solvents, glycol ethers, aliphatic aldehydes or acids, lead, antineoplastic drugs, propellants, dyes, pigments; agricultural workers, hairdressers, housekeepers
Hypertensive disorders of pregnancy	Organic solvents
Low birthweight, preterm birth	Lead, prolonged standing, frequent shift changes. Possibly ethylene oxide, aromatic amines, chlorophenols
Infectious sequelae	Fetal carrier state (hepatitis B, human immunodeficiency virus), fetal morbidity/mortality (rubella, varicella-zoster, human parvovirus B19), serious maternal pneumonia (varicella-zoster)
Contamination of breast milk	Most agents entering the mother's bloodstream can be found in breast milk.
Neurobehavioral disorders in offspring	Lead, mercury, possibly organic solvents

*Some human evidence, albeit limited, has linked each health outcome with the occupations listed and with workplace exposure to at least one of the agents listed; for other agents, animal evidence is available.

nitrous oxide without optimal industrial hygiene measures.[13,14]

Disturbances in ovulation can manifest clinically as menstrual dysfunction. Menstrual disorders have been reported among female agricultural workers, women employed in lead battery plants or exposed to metallic mercury, cosmetologists (who may be exposed to formaldehyde, ethyl acetate, or other chemicals), and shift workers. Antineoplastic drugs disrupt ovarian function not only in cancer patients but also in nurses who handle these drugs without adequate safety precautions.[15]

Exposure to Environmental Pollutants

Doses from exposures to food, water, soil, or air contamination are typically much lower than doses from highly contaminated workplaces. Despite this, adverse reproductive and developmental effects have been linked to certain environmental pollutants, especially those that bioaccumulate (Table 20-3).

Endocrine-disrupting chemicals may affect reproductive hormone function or other hormones, such as thyroid hormone. Potency of endocrine-disrupting substances is determined, in part, by affinity for endocrine receptors. The human estrogens 17-α-estradiol and 17-β-estradiol are used as standards for assessing the estrogen receptor–binding affinity of other pollutants. Human estrogens can themselves be pollutants. After routine wastewater treatment, the effluent contains residual amounts of estradiol, estrone, and other pharmaceuticals that entered the water supply from human and livestock waste.

The estrogen receptor affinities of certain PCBs and bisphenol A are 10,000-fold lower than that of estradiol. Despite this, these agents may affect reproductive endpoints at plausible exposure doses. Women's fertility can be impaired by endometriosis, which can be induced in monkeys by TCDD. Women exposed to TCDD from a large unintentional release in Seveso, Italy, had a doubled risk of endometriosis, although this increase was not statistically

Table 20-3. Selected Environmental Pollutants Associated with Adverse Reproductive and Developmental Effects

Agents	Sources*	Adverse Effects
2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD)	Industrial releases, food chain	Worsened semen parameters, altered sex ratio, immune system disorders, altered timing of puberty, endometriosis, various cancers in laboratory animals
Pesticides such as DDT, dieldrin, mirex, lindane, methoxyclor, and chlordecone	Food chain, household contamination	Early pregnancy loss, male urogenital defects, low birthweight, neurobehavioral disorders, immune system disorders, possibly childhood cancer, earlier menopause
Polychlorinated biphenyls (PCBs)	Fish with high oil content, such as salmon, trout, or mackerel; fatty foods; household contamination	Endometriosis, subfecundity, reduced birthweight, immune system disorders
Polybrominated diphenyl ethers (PBDEs)	Household dust contaminated with flame retardants from textiles, polyurethane foam, and plastics; food chain	Neurobehavioral impairment
Perfluorooctane sulfonate (PFOS), perflurooctanoic acid (PFOA)	Drinking water, household dust contaminated with stain-resisting agents applied to textiles, industrial releases	Neurobehavioral impairment
Human estrogens, bisphenol A	Drinking water contaminated with pharmaceuticals from human and animal waste, food stored in polycarbonate or epoxy resin containers	Reduced male fertility, altered prostate and mammary development, earlier puberty in girls
Phthalates, parabens	Body care products, cosmetics, sunscreen	Worsened semen parameters
Trihalomethanes	Drinking water purified by processes such as chlorination	Spontaneous abortion, low birthweight
Lead	Paint containing lead (used for home interiors in the United States until the 1970s), homes and yards contaminated by leaded gasoline exhaust, industrial releases	Pregnancy-induced hypertension, preterm birth, fetal growth restriction, immune system disorders, neurobehavioral impairment
Arsenic	Drinking water, contaminated dust	Spontaneous abortion, birth defects
Methyl mercury	Large predatory fish, such as shark, swordfish, king mackerel, and tilefish; industrial releases	Birth defects, neurobehavioral impairment
Polycyclic aromatic hydrocarbons, particulates, other air pollutants	Motor vehicles, solid fuel used to heat homes or cook, industrial emissions	Early pregnancy loss, low birthweight, immune system disorders

*Exposure to many of these pollutants may also occur during breastfeeding.

significant. Polychlorinated biphenyls and phthalate esters were measured in women from southern India who had infertility related to endometriosis.[16] Fertile controls without endometriosis had the lowest levels of these compounds and levels increased in stages I through IV of the disease in a statistically significant, dose–response fashion.

When bisphenol A is tested in a pituitary tumor cell assay, it is as potent as estrogen in altering calcium transport and prolactin secretion. It induces these effects at a dose of 0.23 ppt—an exposure level that is lower than the 0.1 to 10 ppb blood and tissue levels in the general population.[17] A primary exposure route for bisphenol A is leaching of the compound into food and beverages from containers manufactured with polycarbonate plastics and epoxy resins. This is of concern because bisphenol A impairs fertility in adult male rodents.

Male fertility can be affected by pollutants through processes that span generations. Exposure of pregnant rats during fetal testicular development to the anti-androgen, vinclozolin,

or the estrogenic pesticide, methoxyclor, reduces sperm production in offspring. These offspring transmit the impairment to subsequent generations through an epigenetic mechanism mediated by DNA methylation.[18] Chemicals can also alter ovarian gene expression.

Sperm counts in men have fallen during the past 40 years in several geographic regions, and rates of hypospadia, cryptorchidism, and testicular cancer have risen. This focused suspicion is on pollutants with estrogenic or anti-androgenic properties that can cause these problems in laboratory animals. Studies from other regions did not confirm these findings, although newer studies have shown that semen indices correlate with biologic concentrations of certain endocrine-disrupting pollutants. Serum TCDD levels were measured in boys 1 year after the 1976 Seveso, Italy, incident; then semen parameters were assessed after the boys reached adulthood.[19] Effects depended upon the developmental window in which exposure occurred. The most pronounced reduction in semen parameters occurred among men who had been exposed during infancy and early childhood (mean age, 6.2 years; median 1976 serum TCDD, 210 ppt). TCDD suppresses androgen action, which is needed to stimulate Sertoli cell proliferation at this period of development. Monobutyl phthalate worsens semen parameters in mice. Men whose urinary concentrations exceed 31.7 ng/mL are three times more likely to have sperm concentrations less than 20 million/mL.[20] More research is needed to clarify the role of many endocrine-disrupting chemicals on male and female fertility.

Pollutants may affect reproductive endpoints by mechanisms other than endocrine disruption. For example, polycyclic aromatic hydrocarbons (PAHs)—products of fossil fuel combustion and tobacco smoke—cause oocyte destruction and ovarian failure in mice.[21]

PREGNANCY

Working women have better pregnancy outcomes than unemployed women, especially in developed countries. This is likely a result of healthier behaviors by women of higher social class, such as avoidance of smoking and alcohol during pregnancy, and the economic benefits derived from working, such as better nutrition and improved access to health care services. On the other hand, certain employment-related exposures increase the risk for adverse pregnancy outcomes (Table 20-2).

Fetal development is rapid during the first weeks of gestation, before a woman knows she is pregnant. The critical period for development of the heart, central nervous system, limbs, and kidneys begins at 3 to 4 weeks gestation. A hazardous exposure can disrupt the complex process of DNA transcription, protein synthesis, and signal transduction, as well as cell division, differentiation, and migration. The second and third trimesters are marked by significant growth of the fetus and by differentiation and maturation of organ systems. Therefore, exposure to toxic agents after the first trimester can still cause problems.

Miscarriage (Spontaneous Abortion)

Hazardous exposures cause early fetal loss in laboratory animal studies. Epidemiologic studies have demonstrated many of these effects in human populations. Embryotoxic exposures include solvents, such as chloroform, methyl alcohol, methylene chloride, and perchloroethylene, used as a dry cleaning agent and as a degreaser. Metals, such as lead and mercury, and anesthetic gases, such as enflurane, halothane, and nitrous oxide, are embryotoxic. Certain pharmaceuticals handled by health care workers, such as antineoplastic agents, pentamidine, ganciclovir, and ribavirin, are embryotoxic in animals. Environmental pollutants such as polybrominated biphenyls (PBBs), PAHs, and the pesticides chlordecone, chlorpyrifos, and lindane cause increased fetal loss (Table 20-3).

Occupational exposure to solvents in dry cleaning, semiconductor or shoe manufacture, and laboratory work has been associated with increased risk for miscarriage. Health care workers may be exposed to anesthetic gases, antineoplastic drugs, or the sterilizing agent ethylene oxide; poorly controlled exposure to each of these agents has been associated with increased miscarriage rates.[22] Even with only moderate

exposure to lead, spontaneous abortion risk is increased. Gold and mercury mines have heavily contaminated many communities with mercury in poorer regions of the world, leading to miscarriage and fetal neurotoxicity.[23]

Women in agricultural families may have an increased risk for miscarriage. While many of the highly toxic chlorinated hydrocarbon pesticides have been banned from use in the United States and Europe, these chemicals persist as environmental pollutants and are still applied widely in developing nations. Higher serum levels of DDT metabolites correlate with higher rates of spontaneous abortion.[24] Studies examining the effects of drinking water contaminants on early fetal loss have conflicting results. However, when exposure levels are high, spontaneous abortion risk is increased, according to studies that examined chlorination by-products (trihalomethanes) and pollutants such as trichloroethylene and arsenic.[25]

Birth Defects

Many of the same exposures that increase risk for miscarriage also increase risk for congenital anomalies.[26] Organic solvent use at work increases risk for birth defects. Case-control studies suggest that women exposed to halogenated aliphatic solvents, glycol ethers, and trichloroethylene may be at special risk for development of orofacial clefts.[27]

Maternal lead exposure may increase the risk for neural tube defects, perhaps by interfering with folate metabolism. Agricultural work by pregnant women has been linked to risk of limb defects. Other maternal exposures that may increase risks for birth defects include aliphatic aldehydes or acids, antineoplastic drugs, propellants, dyes and pigments, working as a hairdresser or housekeeper, and professional application of pesticides in the home. Ionizing radiation exposure may occur among health care workers or flight crews. The most sensitive window of vulnerability for induction of brain defects or mental retardation is approximately 8 to 15 weeks gestation. Substantially higher radiation doses are required to induce birth defects than are received in usual occupational settings. Selected health care workers may be at risk for acquiring infections in the workplace during pregnancy that can lead to congenital anomalies (Table 20-2).

Urogenital malformations in boys have risen in some locations. Estrogenic or anti-androgenic pollutants have been associated with increased rates of these defects, as well as slight reductions in the proportion of male births (altered sex ratio). Although not all studies agree, several studies employing biologic monitoring have confirmed associations between tissue levels of these pollutants and hypospadia or cryptorchidism.[28]

Low Birthweight

Low birthweight may occur as a result of preterm birth or fetal growth restriction. For many infants born with very low birthweight, early delivery has been required because of pregnancy complications, such as placental abruption or eclampsia. Maternal exposure to organic solvents at work has been linked to preeclampsia as well as other causes of preterm birth (Table 20-2). Pregnancy-induced hypertension has been linked to lead exposure from air pollution in the United States. Blood lead levels above 7 to 10 μg/dL increase this risk. A study in a smelter community found that placental lead levels were higher if the infant was preterm.[29]

The risk for having a small-for-gestational-age infant increases in jobs requiring prolonged standing (more than 4 to 6 hours a day), frequent heavy lifting, irregular shifts, or job stress combined with low social support—especially when three or more of these characteristics are present.[30] Modest off-duty exercise among healthy pregnant women does not have this effect. Fetal growth restriction is also associated with intensive exposure to many of the chemicals that increase the risk for spontaneous abortion. Simultaneous exposure may occur to multiple workplace chemicals. Rubber industry exposures include nitrosamines, phthalates, and PAHs. If both parents work in this industry, the birthweight reduction is equivalent to the effect of maternal smoking.[31]

Air pollution has been linked to low birthweight, although the magnitude of risk is small

for low-level exposures and some studies are negative. In the Czech Republic, preterm birth rates increased 18% for every 50 μg/m^3 increase in total suspended particulates and 27% for every 50 μg/m^3 increase in sulfur dioxide.[32] Fetal growth restriction is also increased by prolonged exposure to environmental tobacco smoke, especially if this is combined with exposure to highly polluted outdoor air, as occurred near the World Trade Center following the 9/11 terrorist attack. Many studies show that certain gene polymorphisms reduce the ability to detoxify tobacco smoke and other hazardous chemicals, and this may explain differences in susceptibility to adverse reproductive outcomes from these exposures.

Contamination of drinking water with benzene or chlorinated solvents has been associated with low birthweight in several populations. Some studies show no relationship between birthweight and halogenated by-products from water purification, but trihalomethane levels above regulatory limits (>80 μg/L) increase the risk of fetal growth restriction.[33] Maternal plasma PCB levels are associated with reduced birthweight. Dietary sources of PCBs include fish with high oil content (Table 20-3). Consuming four meals of such fish per month correlates with a 50% increase in maternal PCB levels compared to no intake.[34]

Developmental Immunotoxicity

The fetal immune system develops in a coordinated process involving the thymus, liver, and bone marrow, producing specialized clones of cells primed to respond to foreign antibodies and other threats. Immune system development occurs in stages throughout pregnancy and extends into postnatal life. The impact of toxicant exposure varies according to the timing of exposure. Resulting immune disorders include increased susceptibility to infections; greater risk for allergies, asthma, or childhood leukemia; and autoimmune diseases, such as type 1 diabetes and autoimmune thyroid disorders.[35] The developing immune system is less able to withstand toxic exposures than the adult immune system. For example, atrophy of the fetal thymus is caused by a dose of TCDD one one-hundredth of that needed to cause the same effect in adults. Maternal smoking causes a propensity for asthma among prenatally exposed children, and PCBs reduce infant responses to the vaccine for measles, mumps, and rubella. Other agents damaging the fetal immune system in laboratory animals include mercury, lead, cadmium, DDT, diazinon, chlordane, methoxychlor, and bisphenol A.

Breastfeeding

In 1951, DDT was discovered in breast milk. Since then, many other breast milk contaminants have been documented. Toxic metals such as mercury, lead, cadmium, and arsenic enter breast milk, as do organic solvents. Organochlorine insecticides and fungicides are commonly present, including chlordane, dieldrin, aldrin, DDT, heptachlor, hexachlorobenzene, and hexachlorocyclohexane. Environmental chemicals with high lipid solubility and long half-lives are most likely to be found in appreciable concentrations in breast milk. These include the organochlorine pesticides as well as PCBs, furans, TCDD, and PBDEs. Breastfeeding can deliver significant amounts of environmentally persistent toxic chemicals such as these to the infant, although the benefits of breastfeeding outweighed the risks in some studies. Over the past few decades, levels of these compounds have declined in breast milk in countries where they have been banned or regulated. In Europe, PBDE levels fell during the 1990s, but breast milk levels in the United States, where PBDEs have not been banned, remain 10 to 100 times higher than those in Europe.[3]

LATENT EFFECTS

There are several well-documented examples of prenatal exposures that produce effects that are more prominent in childhood or adulthood than in infancy.

Childhood

Neurobehavioral effects from prenatal toxicant exposures can be detected in newborns, but

important effects may not manifest themselves until later in childhood. Lead exposure is the best documented toxic risk factor for childhood neurobehavioral problems (Chapters 11 and 19). Low-level lead exposure during brain development is associated with childhood problems in memory, learning, and behavior that may persist through adolescence. Studies in laboratory animals demonstrate that gestational exposure without postnatal exposure is sufficient to cause persistent learning and memory deficits.[36] Mercury is also a well-known developmental neurotoxicant. Because mercury bioaccumulates in fish, health agencies have recommended that pregnant and lactating women limit their consumption of fish, especially large predatory species (Table 20-3). Blood mercury levels in about 8% of women of childbearing age in the United States exceed the level recommended by the Environmental Protection Agency (EPA).

Occupational exposure to organic solvents in the prenatal period has been associated with worsened neurobehavioral performance during childhood. For example, 3- to 7-year-old children whose mothers worked with organic solvents during pregnancy scored lower than controls on tests of language and graphomotor skills, and exhibited problem behaviors more often.[37] Deficits in color vision, which in adults have been linked with occupational exposure to organic solvents, are also more common among children whose mothers were exposed to organic solvents during pregnancy.

Environmental exposure to PCBs reduces verbal abilities, processing speed, and visual recognition memory in children.[5] PBDEs produce hyperactivity and impair learning and memory in rodents.[3] Interference with thyroid hormone function by these compounds may contribute to their neurotoxicity. Perfluorooctane sulfonate (PFOS) given prenatally to rodents causes delayed motor development.[38] Reducing air pollution can improve children's neurologic development. When a coal-burning power plant in China was closed, DNA adducts from PAHs fell from 0.32 adducts per 10^8 nucleotides to 0.20 adducts per 10^8 nucleotides, and the proportion of 2-year-olds who had delayed motor development fell from 15% to 5%.[39]

The timing of puberty is altered in humans and laboratory animals exposed to endocrine-disrupting chemicals.[40] Lead and TCDD delay the onset of puberty in girls and boys, whereas higher DDT, PCB, PBDE, and bisphenol A levels are associated with earlier onset of puberty in girls.[17] Adolescents with substantially delayed or accelerated puberty may experience psychosocial problems. Early puberty reduces adult height by prematurely ending long bone growth, and younger age at menarche increases the risk of breast cancer.

The drug DES was the first major agent documented to cause transplacental carcinogenesis in humans—a phenomenon that is well documented in laboratory animals for a variety of carcinogens. The National Toxicology Program (NTP) and the International Agency for Research on Cancer (IARC) have concluded that several pesticides banned from use in developing countries, such as mirex, heptachlor, hexachlorobenzene, and DDT, are carcinogenic. The NTP and IARC are concerned that several pesticides that remain in common use, such as ethylene dibromide and lindane, may be carcinogenic to humans. A prospective study of children found that the rate of childhood cancer was increased nearly two-fold among children from agricultural families whose fathers did not use chemically resistant gloves when handling pesticides.[41]

Adulthood

There is increasing evidence that toxicant exposures during prenatal development cause malignant and nonmalignant disorders that may not become evident until adulthood.[42,43] Drinking water contaminated with arsenic was associated with increased rates of bronchiectasis and lung cancer among prenatally exposed young adults in Argentina. Similar findings were noted in controlled experiments with mice. In utero exposure to estrogenic compounds alters later mammary development in female animals and may increase the risk for breast cancer. In male animals, low-level prenatal exposure to bisphenol A up-regulates estrogen receptors in the prostate and increases precancerous prostate lesions during adulthood. (See Chapter 17.)

Obesity during adulthood may be increased by toxic exposures, such as DDE, a DDT metabolite.[44] In a cohort of families who frequently consumed fish from Lake Michigan, which has been polluted with this insecticide since the 1940s, maternal levels of DDE were available for the pregnancies of 259 women. Their prenatally exposed adult daughters who had DDE levels greater than 2.9 μg/L were 9.2 kg heavier and had body mass indexes (BMIs) that were 2.9 kg/m^2 greater than daughters who were not exposed at this level. Rodent models have also shown that gestational exposures to estrogenic compounds, such as bisphenol A and DES, lead to adult obesity.[43]

Female smokers enter menopause sooner than nonsmokers; when tobacco exposure occurs only during the prenatal period, the risk for early menopause is also increased.[45] Premature ovarian senescence has been demonstrated in animals exposed to TCDD and PCBs during gestation.

Nervous system effects are produced by fetal exposure to many chemicals found in the environment and workplace, leading to concerns these agents may increase the risk for neuropsychiatric disorders that occur with aging. Parkinson disease can be produced in mice by exposing them to a mixture of paraquat and maneb during gestation, then repeating the exposure to one of these agents during adulthood. This suggests that an exposure during development makes the brain more sensitive to toxic insult later in life.[43]

Evaluation and Control of Risk

Reproductive processes are vulnerable in both men and women. Even so, it is often difficult to determine the precise cause of a couple's subfecundity, a child's congenital anomaly, or other health problems for several reasons. Precise assessment of exposures is difficult. There are other contributing health problems. And the research database is neither perfect nor complete. To assess the risk from a potential exposure, four questions are relevant:

1. *What is the agent?*
   The names of chemicals can be found on product labels and material safety data sheets (MSDSs). Tracking these down is laborious, but necessary.
2. *Is exposure actually occurring, and, if so, what are the dose and timing of exposure?*
   Working with an agent, for example, is not necessarily the same as having an internal body exposure to it. How the agent is handled, the protective equipment used, and workplace controls affect exposure level. Toxicant levels can sometimes be measured in blood or body fluids. Inhalational exposures can be estimated with air samples, although skin contact can cause high exposure levels to dermally absorbed compounds even if air levels are low. A birth defect known to develop at gestational week 7 could not have been caused by an acute exposure that occurred only in the third trimester. Spermatogenesis takes about 70 days to complete, so a key period for genotoxic male exposures is thought to be about 2 to 3 months before conception.
3. *Is there evidence to suggest that this agent causes adverse reproductive or developmental effects?*
   All pesticides or all solvents are not the same. A comprehensive literature review or consultation with an expert in reproductive hazard assessment is needed to answer this question.
4. *Given the available evidence, does the agent pose a significant reproductive or developmental risk?*
   Available data can help estimate the degree of risk. A mildly elevated blood lead level during pregnancy, for example, will not typically lead to severe mental retardation. It is important to place the potential exposure in context, considering other concurrent exposures as well as biological risks, such as parental age, poorly controlled diabetes mellitus and other medical problems, medications (such as certain antiepileptic, psychotropic, or anticoagulant drugs), substance use or abuse, and family history of heritable syndromes.

Existing data from animal research and epidemiologic studies converge on certain occupational and environmental exposures that are associated with one or more adverse reproductive or developmental outcomes (Tables 20-1, 20-2, and 20-3). A precautionary approach is

warranted, emphasizing exposure avoidance in the workplace and at home, beginning in the preconception period for both men and women. The following case illustrates this approach.

CASE

A 27-year-old man presented for medical evaluation because of inability to conceive with his wife for 13 months. In his job at an automobile repair shop, he disassembled radiators with an oxygen-acetylene torch and resoldered them with lead-tin solder. There was no special ventilation of his dusty work station, and his work overalls were laundered at home. His 25-year-old wife's infertility work-up revealed no abnormalities. His semen analysis showed a low sperm count of 18 million sperm per milliliter with mildly abnormal motility and morphology. His blood lead level was 63 μg/dL; his wife's blood lead level was 22 μg/dL.

This man had lead poisoning from lead fumes and dust. Home laundering of work clothes laden with lead dust most likely accounted for his wife's elevated blood lead level. Sometimes it is difficult to determine whether a fertility problem is related to a toxicant exposure, although causality is suggested by improvement in sperm indices after remediation of the exposure.

This man is eligible for medical removal from the occupational exposure under the Occupational Safety and Health Administration (OSHA) lead standard. He should not lose wages or benefits because his blood lead level is over 60 μg/dL. An OSHA workplace inspection would identify control measures that could protect all workers. The man should be evaluated by a physician with expertise in treating lead poisoning. The couple should be counseled about the hazards of lead and ways to minimize exposures through safer work practices and prevention of home contamination with workplace lead dust. Although it may seem paradoxical to the couple, contraceptive counseling is important for the time being. Blood lead levels should be normalized in both the man and woman before conception. During pregnancy, the blood lead levels should be kept as low as possible, and they should not exceed 10 μg/dL.

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had organic solvent exposure and both she and her infant lacked certain genes from the cytochrome P450 family that help detoxify chemicals.

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More than 3,000 agents are described, including pharmaceuticals, chemicals, environmental pollutants, food additives, household products, viruses, and genes that cause heritable syndromes or congenital anomalies. This reference book also includes overviews of clinical and experimental teratology.

21

Noise Exposure and Hearing Disorders

Thais C. Morata, David C. Byrne, and Peter M. Rabinowitz

Exposure to unwanted sounds, or noise, is a common on-the-job occurrence. An unfortunate consequence is work-related hearing loss, a condition which has been listed as one of the most prevalent occupational health concerns in the United States for more than 25 years.[1] Hearing loss can be a seriously disabling condition due to the integral role of hearing in human communication. Hearing-impaired individuals often avoid situations in which communication is difficult, rather than risking a misunderstanding and potentially embarrassing mistakes. This tendency leads to isolation, difficulties at work, and possibly adverse psychological consequences. The following scenarios illustrate difficulties associated with noise-induced hearing loss (NIHL):

- Going to restaurants, parties, or other social gatherings becomes a chore, since background noise or music makes conversation difficult, if not impossible.
- Watching television requires the volume to be set very loud, making it irritating or annoying for the rest of the family to be in the same room.
- Working in noisy environments can make communication difficult and increase the risk of workplace injuries due to an inability to hear environmental sounds and warning signals.
- Dealing with tinnitus (the perception of sound in the absence of external acoustic stimuli) becomes an unexpected consequence for some, who expected a hearing loss to result in silence, not an ever-present ringing in their ears.

Greater awareness and improved noise-control strategies are needed for the prevention of these hearing disorders.

PROPERTIES OF SOUND

Sound results from oscillations in pressure in any "elastic" medium, such as air, water, and solids, that effectively couples the sound source with the ear. When transmitted through air, sound is usually described in terms of variations in pressure that alternate above and below the ambient atmospheric pressure. The characteristics of a particular sound depend on the rate at which the sound source vibrates, the amplitude of the vibration, and the properties of the conducting medium. Frequency is an objective description of the rate at which complete cycles of high- and low-pressure regions are produced by a sound source, and it is measured in Hertz (Hz). Subjectively, frequency is often referred to

as "pitch," although there is not an exact correlation between the two terms. Normal human ears respond to a very wide frequency range, approximately from 20 to 20,000 Hz.

A normal healthy human ear is also capable of detecting a remarkable range of sound levels. When the term *level* is used in acoustics, decibel notation is implied. By definition, the decibel (dB) is a dimensionless unit, related to the logarithm of the ratio of a measured quantity to a reference quantity. Decibel notation can cause confusion because it is often associated with different reference quantities. Acoustic intensity, acoustic power, hearing thresholds, electric voltage, electric current, electric power, and sound pressure level may all be expressed in decibels, each having a different reference. The decibel has no meaning unless a reference quantity is specified, or the reference quantity is understood from the context in which it is being used. Sound pressure levels (SPLs) as high as those produced by jet engines (120 dB or greater) are found in some work areas, whereas sound levels approaching the threshold of hearing (approximately 0 dB) are used for audiometric testing.

ASSESSMENT OF NOISE EXPOSURE

The terms *noise* and *sound* are often used interchangeably; however, sound is normally used to describe useful communication or pleasant audible signals, such as music, whereas noise is frequently considered as unpleasant or unwanted sound. Four major factors contribute to the occurrence of negative noise effects: *(a)* the overall noise level, *(b)* the frequency content, *(c)* the duration of exposure, and *(d)* the susceptibility of the individual.

There is a wide variety of measurement instrumentation for conducting noise-exposure assessments. Careful consideration of the objectives of the measurement must be made prior to equipment selection. Sound-level meters and noise dosimeters are the instruments that are used most often. In all circumstances, accurate sound-level measurements require a well-trained operator and calibrated instruments.

A basic sound-level meter consists of a microphone that converts air pressure variations into an electrical signal, an amplifier/filter, an exponential time-averaging circuit, a device to determine the logarithm of the signal, and some type of output display. Some sound-level meters provide only the basic functions, while others are equipped with a very wide range of features, including integration for dose and impulse noise measurement capabilities.

General-purpose sound-level meters are normally equipped with two filters or frequency-weighting networks, designated by the letters A and C. Other frequency-weighting networks, such as B and D, have been developed, but they are not used for industrial noise measurements. Most sound-level meters also will have a linear or flat response setting, which does not apply any correction values—that is, it weights all frequencies equally. The particular weighting network used must always be indicated when sound-level readings are obtained. The A, B, and C weighting curves approximate the response characteristics of the human ear at various sound levels, and, in the earliest sound-level meters, they could be easily produced with a few common electronic components. Empirically, the A-weighting has been found to give a good estimation of the risk potential for hearing damage from exposure to continuous noise. It is the weighting network used for occupational noise exposure measurements.

A noise dosimeter consists of a miniature microphone connected to a small microprocessor-based sound-level meter, which stores the noise data. The microphone is positioned at the top of a worker's shoulder, and the sound-level meter hardware unit is clipped to the wearer's belt or placed in a pocket. Noise dosimeters continuously measure sound levels obtained near a worker's ear, then provide an average value for the exposure throughout the individual's work day. A dosimeter is essentially identical to any other sound-level meter, with the addition of an integrating function that keeps track of the noise level as well as the accumulated exposure time. Dosimeters make it convenient to measure and assess a person's noise exposures, by eliminating the need for the surveyor to follow a worker throughout the work day with a sound-level meter and a stopwatch to assess the worker's exact amount of exposure to different noise levels. Many instruments can continuously log or store noise exposure levels at 1-minute,

10-second, or even 1-second intervals. This noise exposure history information can be saved and analyzed in many ways to help pinpoint periods of high noise levels or other significant occurrences during a work shift.

A worker's daily noise dose can be expressed as a percentage, with 100% constituting the limit of acceptable exposure. A noise dose is usually converted into an 8-hour time-weighted average (TWA). A TWA is a single value for noise level obtained by averaging all of the different sound levels that a worker is exposed to during the work day and normalizing that average to 8 hours. The TWA represents that constant noise level in dBA that has the same severity over 8 hours as the exposure to the actual noise in a work day.

NOISE-INDUCED HEARING LOSS

The Bureau of Labor Statistics has identified noise-induced hearing loss (NIHL) as a leading work-related condition.[1] The reported prevalence of work-related hearing loss varies considerably among occupational groups. With 10 or more years of noise exposure, it is estimated that 8% of the workers exposed to 85 dBA, 22% of the workers exposed to 90 dBA, 38% of the workers exposed to 95 dBA, and 44% of those exposed to 100 dBA will develop hearing impairment.[2] Noise-induced hearing loss is estimated to be among the most common causes of acquired hearing loss. The National Institutes of Health estimates that approximately one-third of all hearing losses can be attributed, at least in part, to noise exposure.[3]

Noise-induced hearing loss is a specific condition with established symptoms and objective findings. The following features characterize cases of NIHL:

1. Irreversible sensorineural (nerve-type) hearing loss that cannot be corrected by conventional medical or surgical procedures, with damage mainly to the cells in the peripheral auditory organ, which are responsible for transforming the sound waves into neural signals.
2. A history of long-term exposure to noise levels—exposure to continuous noise levels greater than 85 dBA for 8 hours a day, or exposure to impact/impulse noise with peaks over 140 dB.
3. Hearing loss that has developed gradually over a period of years, most rapidly during the first 6 to 10 years of exposure—with the rate of loss decreasing as hearing thresholds increase, in contrast to age-related hearing loss.
4. Usually starting in the high frequencies (high-pitched sounds), with most affected persons showing a loss or "notch" in sensitivity at 4,000 Hz. (If high-level noise exposures continue, the loss of hearing generally spreads to adjacent frequencies above and below 4,000 Hz.)
5. An initial hearing loss that may be temporary, after which the original hearing sensitivity is usually restored within a matter of hours. (However, in some cases temporary losses may last for days or weeks. Permanent losses result when these temporary losses do not recover completely.)
6. Reduced word recognition ability that is consistent with the degree of high-frequency hearing loss.
7. No progression if the person is removed from noise exposure.

Hearing loss resulting from hazardous long-term exposure to noise progresses in a fairly well-established, recognizable pattern. Noise-induced hearing loss at the frequencies maximally affected (4,000 and 6,000 Hz) shows a rapid increase over the first 10 years of exposure; the development of the hearing loss then slows and tends to plateau. Hearing loss at frequencies below 4,000 Hz develops at a slower rate, but continuously throughout the entire exposure period.

Noise-induced hearing loss has a gradual onset, and the affected individual might be unaware of any change until significant damage has occurred. Remedial behaviors, such as turning up the radio or television volume or blaming others for not speaking clearly, may conceal initial hearing difficulties. The affected person may be unaware of any hearing problem even when the hearing test indicates decreased hearing ability. In some cases, damage may occur instantaneously, depending upon the noise characteristics

and exposure circumstances. These cases are usually referred to as "acoustic trauma." Generally, impulsive or impact noises are most likely to produce significant losses within short exposure periods, and steady-state continuous noises are responsible for impairments that develop over a long period of time.

Traditionally the mechanism underlying NIHL has been explained as physical trauma causing damage to the cochlea, which contains hair cells responsible for transforming the sound waves into neural signals that are transmitted to the auditory nerve and ultimately to the brain (Fig. 21-1). Hair cells are attached to the basilar membrane, and the stereocilia are in contact with the tectorial membrane. Sound waves lead the basilar membrane to vibrate up and down. The vibration creates a shearing force between the basilar membrane and the tectorial membrane, causing the hair-cell stereocilia to bend back and forth. This leads to internal changes within the hair cells that create electrical signals. Auditory nerve fibers rest below the hair cells and pass these signals on to the brain. Therefore, hair cells respond to sounds by bending of the stereocilia.[4,5]

The most common morphological finding in NIHL is degeneration of the hair cells (mainly the outer rows), which are thought to be the most vulnerable structures of the organ of Corti. The damage of inner hair cells and especially outer hair cells is described as a disarrangement of hairs, fusion of stereocilia, formation of giant hairs that exceed the normal stereocilia in length and thickness, and deformation of cuticular plates. The loss of the outer hair cells induces retrograde degeneration of the efferent fibers, but it has little effect on the afferent cochlear neurons. Therefore, if there were damage to the outer hair cells alone, the lesion would be less obvious—since only rather extensive damage to the inner hair cells causes substantial degeneration of the afferent nerve fibers.

Recently, metabolic processes involving oxidative stress have been shown to contribute to NIHL. The generation of reactive oxygen species, or free radicals, has been associated with cellular injury in different organ systems. Free radicals produce cell damage by binding to macromolecules and producing lipid peroxidation—a basic mechanism of toxicity that is thought to be part of the mechanism of acquired hearing loss. Medical treatments, such as antioxidants, are being sought to prevent or minimize hair cell damage.

OTHER EFFECTS OF NOISE EXPOSURE

Exposure to excessive levels of noise is not restricted to the work environment, and the effects of noise exposure are not restricted solely to decreased hearing. Noise from power tools, powered lawn maintenance equipment, farm equipment, and shooting hobbies (such as skeet, targets, and hunting) or other recreational activities, such as attending music concerts, riding in motorboats, and watching automobile or motorcycle races, are examples of potentially

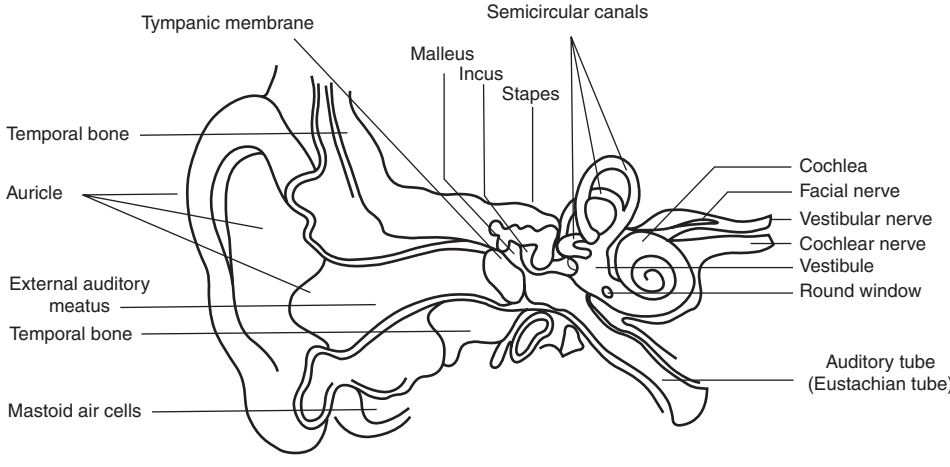

Figure 21-1. Schematic drawing of the ear.

hazardous nonoccupational sources of noise. Additionally, excessive noise exposure is associated with hypertension, ischemic heart disease, respiratory disorders, annoyance, sleep disturbance, and reduced school performance. Noise can also disrupt communication. Some studies have suggested an association between accidents and both occupational noise exposure and/or hearing loss.[6,7]

THE IMPACT OF HEARING DISORDERS

Consequences of hearing disorders range from slight to seriously debilitating. At work, hearing loss can increase difficulties associated with the use of hearing protectors, causing interference with verbal communication and detection of warning signals. In addition, the earnings of workers with severe hearing loss are estimated to be 50% to 70% of those than their peers without hearing impairment.[8]

Hearing loss can have a severe impact on social interaction and family life. Hearing disabilities may have a negative effect on self-image, causing a perception of oneself as abnormal, prematurely old, or as a burden because affected individuals often ask others to repeat what they say. There are several barriers to seeking help and using hearing aids, including cost, pride, denial of a problem, and the stigma attached to deafness. People with hearing difficulty will often try to downplay or conceal its seriousness to minimize the risk of being marginalized, and they may avoid seeking help. People with untreated hearing loss are more likely to report depression and anxiety, and they are less likely to participate in social activities compared to those who wear hearing aids.[9] Unfortunately, less than 20% of the estimated 28 million U.S. residents who could benefit from hearing devices own them, and less than 20% of physicians include hearing testing in regular physical examinations.[10]

INFLUENCE OF OTHER FACTORS ON HEARING LOSS

The incidence and degree of hearing loss vary greatly among groups, partly due to endogenous factors or individual attributes that affect susceptibility, including age, gender, race, blood pressure, and use of certain medications. A common scientific opinion is that hearing loss due to noise exposure should not continue to progress if the person is removed from noise exposure. There is, however, limited knowledge about how noise impairment is influenced by, or interacts with, age-related hearing impairment. The effects of noise and age are challenging to differentiate, but they seem to be additive. Hearing acuity may decline with aging, but a healthy person who has not been exposed to ototraumatic or ototoxic agents may have normal hearing acuity even after age 65. The median hearing level across the frequencies of 1,000, 2,000, 3,000, and 4,000 Hz for 60-year-olds not exposed to noise is 17 dB HL for males and 12 dB HL for females.[11] Young animals are probably more susceptible to the effects of noise. Mice that had been exposed to noise at different ages between 4 and 124 weeks demonstrated differences in their sensitivity to noise exposure; mice exposed to noise when they are young suffer greater hearing impairment, compared to elderly mice.[12] When young mice who have been exposed to noise age, they have been found to have an age-related hearing loss that is more severe than mice of the same age who have not been exposed to noise, suggesting that noise exposure makes mice more sensitive to age-related hearing impairment.

Gender and race also seem to be associated with susceptibility to NIHL. White males have the highest rates of noise-induced hearing loss, and African American females the lowest.

Certain nonacoustic factors in the workplace, which may directly affect hearing or interact with noise, are considered possible contributors to variability in individual susceptibility to NIHL.[13] For example, workers with vibration-induced white finger (VWF) syndrome have a higher rate of hearing loss than workers exposed to similar noise levels, but not to vibration.[14] It is not known if whole-body vibration enhances risk for hearing loss.

Hearing Loss from Chemical Exposures

Since noise is present in most occupational settings, the hearing disorders observed among workers are often attributed to noise exposure

alone, without considering the effects of other agents. The terms "occupational" and "work-related" hearing loss have been used synonymously for NIHL, although this is not always correct, since chemical agents have also been implicated in hearing loss. In several settings, noise coexists with other factors that are potentially dangerous for hearing, so caution should be taken before identifying a hearing loss simply as noise-induced. Moreover, when one considers the possibility that other environmental and occupational factors can affect hearing, current hearing loss prevention initiatives need to be reexamined.

Sensorineural hearing loss is increased in noise-exposed workers who are also exposed to certain chemicals, due to their effects on the auditory system or brain. For example, some metals, solvents, polychlorinated biphenyls (PCBs), pesticides, and asphyxiants (such as carbon monoxide) adversely affect the auditory system, even in the absence of excessive noise.[15] If workers are exposed to these chemicals at sufficiently high concentrations, their hearing may be impaired, even in the absence of exposure to loud noise. Work activities that involve exposure to these agents, often in combination with noise, include manufacturing of metal, leather, and petroleum products; painting; printing; woodworking; construction; furniture making; fueling vehicles and aircraft; degreasing; and firefighting. In addition, hearing loss may occur following consumption of water or fish contaminated with these chemicals.

Exposure to solvents was implicated as a causative factor for hearing loss in a 20-year longitudinal study in a company where 23% of workers in the chemical division had compensable hearing loss, compared to 5% to 8% of company workers not exposed to chemicals. This effect was found despite the lower noise levels in the chemical division (80 to 90 dBA, compared to 95 to 100 dBA elsewhere).[16] Solvents, which can reach the inner ear via the blood, may cause damage to some structures and functions of the inner ear. The onset latency, site of lesion, injury mechanism, and extent of ototoxic damage from these toxins vary due to risk factors such as the type of chemical, interactions with other agents, and exposure level and duration.

Hearing loss from ototoxicity is bilaterally symmetrical and often irreversible. Similar to the effect of noise exposure, a high-frequency "notch" on the audiogram is often present following long-term exposure to ototoxic chemicals, although a wider range of frequencies may be affected, from 2,000 to 8,000 Hz.[15,17]

Ototoxicity of chemicals had been overlooked for a long time because *(a)* workers exposed to ototoxic chemicals are often also exposed to loud noise, and *(b)* audiograms do not identify the cause of hearing loss. (See Box 21-1.) Consequently, it is difficult to perform a differential diagnosis of hearing impairment and assign causation. The nature and severity of ototoxic damage vary according to type of chemical, chemical interactions, and level, duration, and pathway exposure—as well as exposure to excessive noise. The National Institute for Occupational Safety and Health (NIOSH) recommends that hearing-loss prevention programs consider chemical exposures when monitoring for hazards, assessing hearing, and controlling exposures.[18,19] Since 1998, the American Conference of Governmental Industrial Hygienists (ACGIH) has recommended audiograms for workers exposed to toluene, lead, manganese, or n-butyl alcohol.[20]

TINNITUS

Tinnitus is a condition often associated with many forms of hearing loss. It is usually described as "ringing in the ears," but other forms of sound have been reported, such as buzzing, pulsing, hissing, knocking, roaring, whooshing, chirping, whistling, and clicking. Tinnitus can be continuous or intermittent—lasting for minutes to a few hours at a time. It can be a minor annoyance or a serious and nearly intolerable condition. In severe cases, it may interfere with daily activities and sleep. Tinnitus is associated with noise exposure frequently, and also with more than 200 medications as well as dietary, nutritional, hormonal, immunological, and stress factors. (See Box 21-2.)

Although the reported prevalence of work-related tinnitus ranges from 17% to 60% of cases among noise-exposed workers,[21,22] it has attracted relatively little interest. For example, only 13 U.S. states and the United Kingdom, Canada, Australia, Germany, Denmark, and

Box 21-1. Case of Hearing Loss Following Noise and Chemical Exposures

A 41-year-old man came to the occupational medicine clinic for his annual physical examination. He worked in a company that made specialized paints. His job in the paint-mixing rooms was to open and mix the contents of large barrels of solvents, including xylene, toluene, and methyl ethyl ketone in a specified manner, with intermittent use of a loud mixing machine. He did not wear hearing protection because the 8-hour time-weighted average (TWA) of noise exposure during the previous year was 84 dBA. The ventilation in the mixing room had not always been optimal, causing a usually strong solvent smell. He also noted that he often spilled small amounts of solvents on his hands and arms, after which he wiped himself with a rag. He has noted that his hearing had been getting worse, and he was concerned about going deaf. He had no major medical problems and no family history of significant hearing loss.

His physical examination was normal, except for some defatting of his fingertips and apparent hearing difficulty. His audiogram showed a significant hearing loss at high frequencies bilaterally (Fig. 21-2). Compared to his baseline audiogram with the company, he has lost more than 10 dB (as an average over 2,000, 3,000, and 4,000 Hz), and he also had an absolute loss greater than 25 dB at these frequencies. (Therefore, his loss was potentially recordable under the Occupational Safety and Health Administration [OSHA] recordkeeping standards, if it was thought to be due to workplace exposures.) A full audiological evaluation revealed that his hearing loss was sensorineural, and there was no other medical explanation for it.

The physician who evaluated him was faced with several questions concerning this worker:

1. Given that noise exposure, as measured during the previous year, was not excessive, what were possible explanations for the worker's degree of hearing loss?
2. Should his hearing loss be considered a work-related medical condition?
3. What further steps are warranted in evaluating this worker's hearing loss and preventing further hearing loss?

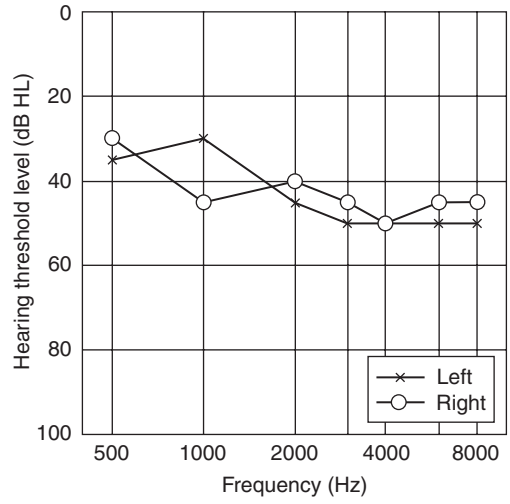

Figure 21-2. Audiogram of a 41-year-old man who worked in paint manufacturing.

This man had been exposed to intermittent noise at work. His noise exposure, as an 8-hour TWA, was below the OSHA action level, but peak exposures from the mixing machine may have been high enough to cause hearing loss over time, even with the 8-hour TWA being less than 85 dBA.

In addition, this worker had simultaneously been exposed to a variety of organic solvents, including xylene and toluene, which are neurotoxic and ototoxic. These solvent exposures in this relatively young man may be *(a)* potentiating the adverse effects of noise on cochlear hair-cell function and survival, and/or *(b)* having a direct independent ototoxic effect. It was necessary to reduce his exposures to both noise and solvents to preserve his hearing.

Sweden provide workers' compensation for tinnitus.[22]

It is likely that there are several mechanisms that cause tinnitus. It likely results from increases in the spontaneous neural activity in the auditory system. The first relay of the primary auditory pathway is in the cochlear nuclei in the brainstem, which tend to develop hyperactivity that might be relayed to higher levels in the brain. Alternatively, heightened activity of some descending pathway or other central mechanism might explain this hyperactivity.

GOVERNMENTAL REGULATIONS

Federal, state, and local governments set and enforce noise standards for aircraft, airports, interstate motor carriers, railroads, medium- and heavy-duty trucks, motorcycles, mopeds, and many commercial, industrial, and residential activities.

The Environmental Protection Agency (EPA) coordinated all federal noise-control activities until 1983. Responsibility of regulating noise was then transferred to state and local governments.

Box 21-2. Case of Noise-Induced Hearing Loss and Tinnitus

A 55-year-old dockworker with tinnitus was evaluated. His job involved working in the hold of freighters, loading and unloading cargo, including steel girders and rods and crates of frozen produce. From a cab, he also operated a loading crane several hours a day. He reported exposure to frequent impact noise from metal striking metal. He noted that, when operating the crane, he had to shout to be able to communicate to a co-worker nearby. He did not wear hearing protection, saying that he needed to hear sounds (such as that of the overhead crane when he was loading), and to shout communication when he was operating the crane. For 1 or 2 hours a day, he operated a forklift in a refrigerated warehouse, where noise from the refrigeration units was so loud that he had to shout to communicate with a co-worker at arm's length. He did not wear hearing protection when he drove the forklift because of the need to hear warning signals and communication from co-workers.

This worker reported that he first noticed tinnitus 15 years before, when his ears rang for several hours after noisy work shifts. Gradually, it became more frequent; by the time of this evaluation, it interfered with his hearing when there was background noise. He reported that many times his hearing decreased after a work shift, and then improved the following day. He also noticed that when he started his car in the morning, the radio seemed excessively loud because he had left the volume turned up the night before.

Recently, he had argued with his wife about the television volume. When she reduced the volume to a level that she preferred, he had difficulty hearing what people were saying, so he increased it. He also had noticed that talking on the phone was difficult for him if there was background noise, and that having a conversation in a bar was also increasingly difficult. He admitted that his friends had kidded him about being "in need of a hearing aid," and that the thought of having a hearing loss had led him to feel depressed.

On physical exam, his blood pressure was 140/88 and he had normal external auditory canals and tympanic membranes. His audiogram is illustrated in Figure 21-3.

This case illustrates many of the clinical aspects of noise-induced hearing loss. The worker reported exposure to occupational noise at or above 85 dBA as indicated by the "shout test." In addition to sources of steady-state noise, such as refrigerator fans and crane motors, he was also exposed to impact noise, including crashes of metal on metal. In addition, he gave a history of recurrent temporary threshold shifts after work shifts, with loss of hearing acuity and tinnitus that improved overnight. Over the years, however, such temporary changes had progressed to permanent hearing loss and tinnitus.

While the symmetric nature of his hearing loss and the "notch" at high frequencies on his audiograms all pointed to the diagnosis of noise-induced hearing loss, he needed to be referred for a full audiological evaluation for other audiological disorders, such as otosclerosis. He may have also been a candidate for a trial of amplification and for tinnitus treatment.

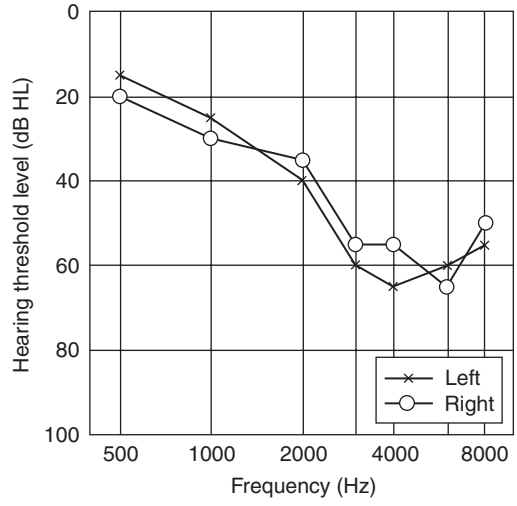

Figure 21-3. Audiogram of a 55-year-old dock worker, illustrative of noise-induced hearing loss.

He needed to protect his hearing if he wished to remain in a noisy area. While noise reduction through engineering controls would have been the ideal way to reduce his exposure, adequate noise reduction may have been difficult, due to the nature of his work. Therefore, hearing protection may have been necessary. Standard earplugs that preferentially attenuate at higher frequencies may have worsened his problem of discriminating speech. Therefore, a "flat-attenuation" earplug may have been more appropriate for him, given his preexisting high-frequency loss.

Another occupational issue facing this man is whether he could safely operate a forklift with his degree of hearing loss. His hearing, with and without amplification, could have been compared to any internal company policies for mobile-equipment operators and to applicable Department of Transportation guidelines. A workplace accommodation for him could have been his having a radio-communication headset that could also provide hearing protection, to allow him both to protect his hearing and to communicate with others while operating the crane.

Finally, his hearing level according to American Academy of Otolaryngology criteria indicated that he had a degree of hearing impairment, which, more likely than not, was due to his occupational noise exposure—although his audiogram also suggested an element of hearing loss due to aging. His history of not wearing hearing protection was not a reason to deny him workers' compensation for his hearing loss.

Although the EPA no longer plays a prominent role in regulating noise, its past standards and regulations remain in effect, and other federal agencies continue to set and enforce noise standards for sources within their regulatory jurisdiction.

Workers in general industry who are exposed to noise levels above 85 dBA are required by the Occupational Safety and Health Administration (OSHA) to be in a hearing conservation program, which includes noise measurement, noise control, periodic audiometric testing, hearing protection, worker education, and record keeping. Twenty-four states, Puerto Rico, and the U.S. Virgin Islands have OSHA-approved state plans and have adopted their own standards and enforcement policies. Most of these state standards are identical to those of federal OSHA. However, some states have adopted different standards or may have different enforcement policies. Most health and safety regulations are designed to keep damage risk within "acceptable limits"—that is, some people are likely to incur a hearing loss even when exposed to less than the maximum daily amount of noise specified in a regulation.

In the construction industry, noise exposures are required to be evaluated and controlled, and hearing protectors must be offered when exposures exceed 85 dBA. Apart from exposure limits, there is no mandatory hearing conservation program for construction workers. However, construction workers may choose to follow the criteria outlined in "Hearing Loss Prevention for Construction and Demolition Workers," developed by the American National Standards Institute. This standard, which helps employers prevent occupational hearing loss among construction and demolition workers with potential exposures to continuous, intermittent, or impulse noise of 85 dBA and or higher, was approved by the American National Standards Institute (ANSI) and the American Society of Safety Engineers in 2007.

Also in 2007, the Federal Railroad Administration issued a final rule entitled "Occupational Noise Exposure for Railroad Operating Employees," which requires railroads to conduct noise monitoring and implement a hearing conservation program for employees whose exposure to cab noise equals or exceeds an 8-hour TWA of 85 dBA. There is no hearing-loss prevention regulation for workers in agriculture, despite their high prevalence of hearing loss, or for workers in the service and public sectors.

Separate from the Occupational Noise Exposure Standard, OSHA's current recordkeeping rule significantly altered the criteria for documenting what constitutes a reportable hearing threshold shift. Work-related hearing loss in either ear is recordable when both of the following occur:

1. An average shift in hearing threshold of 10 dB or greater at 2,000, 3,000, and 4,000 Hz, relative to the audiometric baseline (called a standard threshold shift, or STS)
2. The average hearing level in the same ear is 25 dB or greater at 2,000, 3,000, and 4,000 Hz.

After the recording criteria were revised in 2004, it was anticipated that the number of recordable hearing loss cases would increase in most states,[23] possibly leading to improvements in hearing conservation and noise-control programs. While the revision of the recording criteria has led to some improvement the management of hearing conservation programs, it has not had the expected effects. From 2004 through 2007, about 25,000 cases of recordable hearing shift were reported annually to the Bureau of Labor Statistics.

STRATEGIES FOR IMPROVING HEARING-LOSS PREVENTION IN THE WORKPLACE

The National Occupational Research Agenda (NORA) is a partnership program developed to stimulate innovative research and improved workplace practices. Established in 1996, NORA has become a research framework for NIOSH and the United States. After diverse parties collaborate to identify the most critical issues in workplace safety and health, they work together to develop goals and objectives for addressing these needs. Participation in NORA is broad, including stakeholders from universities, large and small businesses, professional societies, government agencies, and worker organizations.

In 2006, the program entered its second decade with a new sector-based structure to improve the translation of research into practice in workplaces. Within NORA, noise and hearing loss comprise a cross-sector area because noise and hearing loss affect almost all industrial sectors.

Based on extensive experience in hearing conservation, NIOSH has published *Preventing Occupational Hearing Loss—A Practical Guide.*[18] This guide presents attributes of successful hearing-loss prevention programs and identifies responsibilities of management, those who implement hearing-loss prevention programs, and workers affected by noise exposure.

Controlling Hazardous Exposures

Initial steps of hearing-loss prevention programs are hazard assessment and control. Required noise measurements serve as the basis for assessing noise-control alternatives. If employees' daily noise exposures are controlled to levels below a TWA of 85 dBA, a hearing conservation program is not legally required.

Exposure at the NIOSH recommended exposure limit (REL) for occupational noise (85 dBA TWA)[19] for 40 years increases the risk of NIHL by 8%—considerably lower than the 25% increased risk at the current OSHA and Mine Safety and Health Administration (MSHA) permissible exposure limit (PEL) of 90 dBA TWA.

NIOSH previously recommended a 5 dB exchange rate for halving the exposure time when calculating TWAs—that is, starting at the 85 dBA REL for an 8-hour period, for each 5 dB increase in exposure, the permissible exposure time was to be halved. However, since 1998, NIOSH has recommended a 3 dB exchange rate, which is more firmly supported by scientific evidence.[19] The 5 dB exchange rate is still used by OSHA and MSHA.

Whenever there is hazardous noise in a workplace, measures should be taken to reduce noise levels as much as possible to protect exposed workers and to monitor the effectiveness of intervention. The most effective way to prevent NIHL is to remove the noise source from the workplace, such as by engineering controls, or to remove the worker from exposure to hazardous noise.[24]

Unfortunately, hearing protection devices (HPDs) are often adopted in lieu of controlling noise exposure. While relatively inexpensive and easy to use, providing HPDs to control noise exposure is often problematic (Fig. 21-4). In order to achieve the desired noise attenuation, workers must wear HPDs consistently during exposure to noise levels greater than 85 dBA. Workers often find it difficult to do so because HPDs can be uncomfortable and interfere with communication. Consequently, use of HPDs is inconsistent and varies widely. They are usually purchased on the basis of minimum cost and maximum attenuation, often leading to use of uncomfortable devices that overprotect. New electronic hearing protection devices are available that not only protect at appropriate levels but also facilitate communication. Recommendations to increase the use of HPDs include identifying devices that offer adequate attenuation and provide workers with better comfort.

The original rating system developed by the EPA to measure HPD attenuation is recognized as obsolete. Laboratory-derived attenuation values have been shown to fail to predict how HPDs function in the workplace. Therefore, OSHA has instructed its compliance officers to derate the labeled noise-reduction rating (NRR) of HPDs by 50% when enforcing the OSHA Occupational Noise Exposure Standard. NIOSH recommends derating by subtracting from the NRR 25% for earmuffs, 50% for formable earplugs, and 70% for all other earplugs. This *variable* derating scheme, as opposed to OSHA's *fixed* derating scheme, distinguishes among the performance of different types of HPDs. Consensus standards have been developed with new strategies for a more accurate determination of HPD attenuation provided in the field. The latest standards incorporate the variance of both *(a)* the fit of the protector among test subjects, and *(b)* the variance of the HPD's performance over a wide range of noise spectra. These criteria have formed the basis of a proposed revision of EPA's Product Noise Labeling regulation.[25,26] The proposed regulation provides guidance for evaluating and labeling passive HPDs, active noise-reduction devices, and impulsive noise-reduction devices, such as sound restoration (or nonlinear) acoustic protectors.[27]

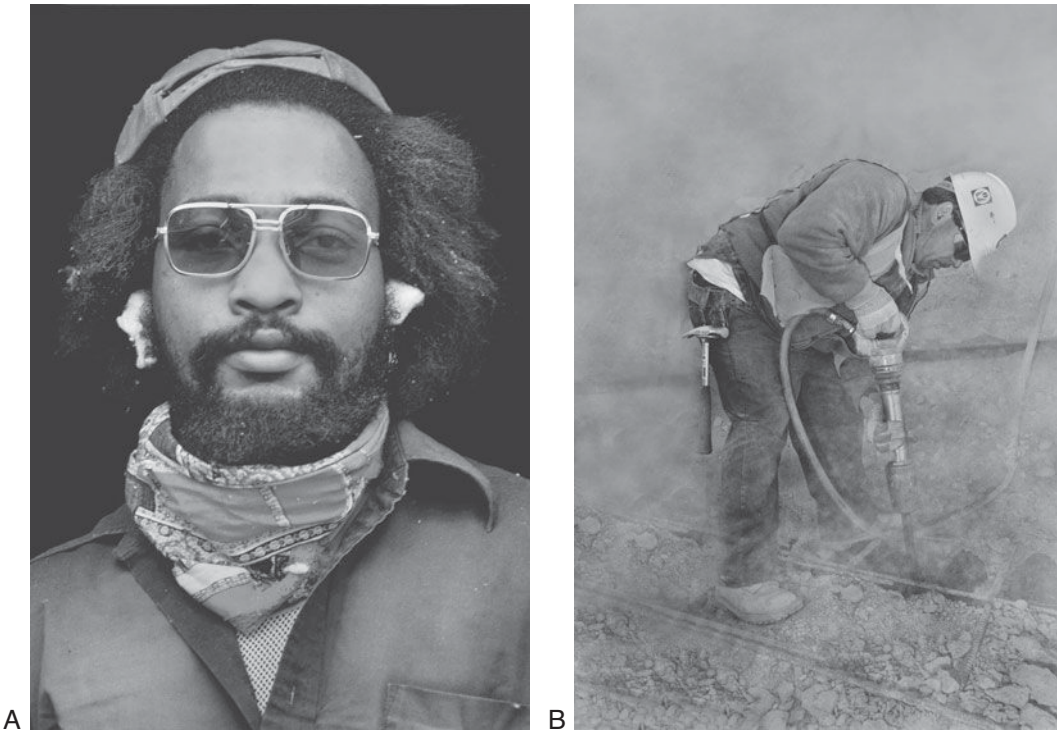

Figure 21-4. Improper and proper hearing protection: (A) Cotton earplugs are ineffective in protecting a worker from loud noise. (B) This jackhammer operator is appropriately wearing earplugs. (Photographs by Earl Dotter.)

Inclusion Criteria for Hearing-Loss Prevention Programs

Federal regulations only consider noise as a risk factor for hearing loss. Preventive strategies that are used to protect workers from noise exposure will not protect workers from incurring a hearing loss due to chemical exposure. When ototoxic chemicals are present in the workplace, hearing loss prevention measures may be needed even where noise exposure does not exceed 85 dBA.

ACGIH advises workers exposed to ototoxins to receive periodic audiograms.[20] The U.S. Army requires that hearing conservation programs consider ototoxic chemical exposures, especially when noise exposure does not exceed permissible or recommended limits.[28] Annual audiograms are recommended for workers whose airborne exposures are at 50% of the most stringent occupational exposure limits for toluene, xylene, *n*-hexane, organic tin, carbon disulfide, mercury, organic lead, hydrogen cyanide, diesel fuel, kerosene, jet fuel, organophosphate pesticides, or chemical-warfare nerve agents—regardless of the noise level. This 50% level, while somewhat arbitrary, ensures data collection from exposure situations below occupational exposure limits. Annual audiograms also are recommended when dermal exposures to these agents result in a systemic dose equivalent to 50% or more of the occupational exposure limit. For workers participating in hearing conservation programs because of excessive noise, reviewers of audiometric data should be alert to possible additive, potentiating, or synergistic effects between noise and ototoxic chemicals, and should, if necessary, initiate reduction of exposure to the noise and/or the chemicals.

Audiometric Monitoring

Audiograms indicate a person's hearing threshold. Results are given in decibels, which indicate

the intensity or loudness a sound has to be for the person to detect it. Thresholds below 25 dB HL are considered normal. NIOSH suggests that *(a)* monitoring audiometry be conducted on noise-exposed workers late in, or at the end of, their daily work shifts; and *(b)* audiometry be repeated immediately after any monitoring audiogram indicates a significant threshold shift.[19] Before conducting retests, workers should be reinstructed and earphones refitted. Those who employ this retest strategy will find a significant reduction in the number of workers called back for a confirmation audiogram—because if the retest audiogram does not show the same shift as the initial audiogram, the retest audiogram becomes the test of record.

By testing workers during their work shifts, one may identify temporary threshold shifts (TTSs). Although the relationship between permanent threshold shifts and TTSs is not completely understood, workers with a TTS are being overexposed to noise. Discovering a TTS and taking action to prevent its recurrence will help protect workers from permanent hearing damage. If annual monitoring audiograms are performed before or at the beginning of work shifts, TTSs from noise exposure on the previous work shifts will have resolved so that any threshold shifts observed will represent permanent shifts in hearing.

Audiometry should be conducted again within 30 days of any monitoring or retest audiogram that continues to show a significant threshold shift. A minimum of 12 hours of quiet should precede the confirmation audiogram to determine whether the shift is a TTS or a permanent threshold shift. Hearing protectors should not be considered as a substitute for a quiet work environment.[19]

The OSHA criterion for the standard threshold shift (a change of 10 dB or more in the average of hearing thresholds at 2,000, 3,000, and 4,000 Hz) may not be the ideal method to identify and prevent permanent NIHL. NIOSH recommends a better criterion for the calculation of significant threshold shift: an increase of 15 dB in the hearing threshold level at any of the test frequencies in either ear (at 500, 1,000, 2,000, 3,000, 4,000, and 6,000 Hz), as determined by two consecutive audiometric tests.[19] This alternative criterion has both high sensitivity and high specificity.

Age Correction

Although many people experience a decrease in hearing acuity with age, others do not. It is not possible to predict who will and who will not develop hearing loss with aging. The median hearing loss attributable to aging for a given age group cannot be generalized to all individuals in that age group. Thus, when calculating significant threshold shifts, age-correcting hearing thresholds will overestimate the expected hearing loss for some people and underestimate it for others.

Unfortunately, the adjustment of audiometric thresholds for aging has become a common practice in workers' compensation litigation. In this application, age corrections reduce the amount of hearing loss attributable to noise exposure, with a consequent reduction in the amount of compensation paid to workers for hearing loss.

Age-correcting audiograms obtained in an occupational hearing-loss prevention program are not recommended.[19] The purpose of the program is to prevent hearing loss. If an audiogram is age-corrected, regardless of the source of the correction values, the time required for a significant threshold shift to be identified will be prolonged. Delaying the identification of a worker with a significant threshold shift is counterproductive to all other hearing conservation efforts.

Accommodating Workers with Hearing Loss

After a confirmation audiogram that indicates a permanent threshold shift, NIOSH recommends a written notification to the worker and a referral to the audiometric manager or professional supervisor for review and determination of probable etiology. This referral should explore all possible causes in addition to occupational noise, including ototoxic chemicals, age-related hearing loss, familial hearing loss, nonoccupational noise exposure, and medical conditions.[19]

Workers with a threshold shift due to causes other than noise should be counseled by audiometric managers and referred to their physicians for evaluation and possible treatment. Appropriate actions should be planned for workers showing a threshold shift that is determined by

the audiometric manager to be due likely to occupational noise. At a minimum, these actions should include reinstruction and refitting of hearing protectors, additional training in worker responsibilities for effective hearing-loss prevention, and/or reassignment to a quieter work area. The professional supervisor should be responsible for identifying whatever changes may be necessary, and for ensuring that they are implemented. According to OSHA's Hearing Conservation Amendment, the professional supervisor of the audiometric testing component of a hearing conservation program must be a licensed or certified audiologist or otolaryngologist, or other physician.

The main factors that enable workers with hearing loss to continue working are ability to cope with the hearing loss, support from management and co-workers, adequate work conditions, psychological support from patient organizations as well as family members and friends, support from medical professionals and programs, and financial and other benefits.[29] A set of guidelines can be used by health professionals for managing the work-related conditions. Important to workers with hearing loss is knowledge about and availability of better hearing protectors and hearing aids, alternative means of obtaining and financing hearing aids, self-acceptance, a quiet work environment, determination and persistence to ask for needed accommodations at work, education of co-workers about hearing loss, and opportunities to communicate information and experiences with other affected workers.

Accommodating Workers with Tinnitus

Several standardized scales exist to evaluate in the interference of tinnitus on the quality of life. Most of them are short and easy to administer. Their use can offer valuable information on how to accommodate workers who suffer from tinnitus.[30] Most important in managing workers with tinnitus is to refer them to otolaryngologists or otologists (ear specialists), who will try to determine its cause by evaluating the auditory system, measuring blood pressure and kidney function, and assessing diet, allergies, and medications. Specialists determine treatment, which may include maskers (electronic devices the size of a hearing aids that use sound to make tinnitus less noticeable), support and counseling, surgery, drug therapy (such as tricyclic antidepressants), diet, psychotherapy, electrical/magnetic stimulation, acupuncture, biofeedback, and hypnosis. They also should explain to patients the pathophysiology of their tinnitus, make recommendations for hearing aids when appropriate, and provide periodic monitoring.[31]

ACKNOWLEDGMENT

This chapter is dedicated to the memory of Dr. Derek E. Dunn.

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FURTHER READING

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Noise and hearing conservation are addressed in this standard, which is applicable to general industry in

the United States. It covers monitoring of noise exposure, audiometric testing, hearing protection, employee training, and record keeping.

Cochlea.org. Available at: http://www.cochlea.org
An educational Web site sponsored by several institutions and companies. It contains sections describing the anatomy, physiology, and pathophysiology of the auditory system.

American Tinnitus Association. Available at: http://www.ata.org
The American Tinnitus Association (ATA) promotes tinnitus awareness, prevention, and treatment. It offers information on prevention programs in schools, urges governmental and private organizations to support hearing conservation, funds research, and facilitates self-help groups.

Suter A. Hearing conservation manual (4th ed.). Milwaukee, WI: Council for Accreditation in Occupational Hearing Conservation.
The fourth edition of the Council for Accreditation in Occupational Hearing Conservation (CAOHC) Hearing Conservation Manual covers all facets of developing a successful hearing-loss prevention program. The manual is designed as a reference text used during Occupational Hearing Conservationist training courses, and it is an excellent resource for practicing professionals.

U.S. Army Center for Health Promotion and Preventive Medicine. Just the facts…occupational ototoxins (ear poisons) and hearing loss. Available at: http://chppm-www.apgea.army.mil/documents/FACT/51-002-0903.pdf
This Web site contains a fact sheet regarding ototoxic chemical exposures and guidelines for hearing conservation developed by the U.S. Army.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

22

Skin Disorders

Loren C. Tapp and Boris D. Lushniak

The skin plays an important role in providing a protective, living barrier between the external environment of the world around us and the internal environment of the human body. As a first-line protective barrier, the cutaneous surface is also subject to the hostile forces of the external environment and, as such, can be directly injured or damaged by these environmental forces.

In general, the causes of environmental and occupational skin disorders can be grouped into the following categories:

1. *Physical insults*: Friction, pressure, trauma, vibration, heat, cold, variations in humidity, radiation (ultraviolet, visible, infrared, and ionizing), and electric current
2. *Biologic causes*: Plants, bacteria, rickettsia, viruses, fungi, protozoa, parasites, and arthropods
3. *Chemical insults*: Water, inorganic acids, alkalis, salts of heavy metals, aliphatic acids, aldehydes, alcohols, esters, hydrocarbons, solvents, metallo-organic compounds, lipids, aromatic and polycyclic compounds, resin monomers, and proteins

These insults are present in every aspect of our environment and can affect the skin in the home setting, during outdoor and leisure activities, while involved in hobbies, and in the work environment. Occupational dermatology is the facet of dermatology that deals with skin diseases whose etiology or aggravation is related to some exposure in the workplace. The role of a health care practitioner involved in occupational dermatology is not only to diagnose and treat patients but also to determine the etiology of occupational skin diseases and make recommendations for their prevention. Making the diagnosis and offering treatment, determining etiology, and recommending preventive measures all can be difficult undertakings.

Environmental and occupational skin diseases can manifest themselves in a variety of ways. This chapter will emphasize skin conditions caused by environmental agents that have a direct effect on the skin. These include irritant contact dermatitis, allergic contact dermatitis, contact urticaria, skin infections, skin cancers, and a large group of miscellaneous skin diseases. Certain common skin diseases, such as atopic dermatitis and psoriasis, are exacerbated by environmental factors, but their etiology remains unclear and they will not be covered here.

CONTACT DERMATITIS

Contact dermatitis is the most common occupational and environmental skin disease.

Epidemiologic data show that contact dermatitis comprises 90% to 95% of all occupational skin diseases.[1,2] Contact dermatitis—both irritant and allergic—is an inflammatory skin condition caused by skin contact with an exogenous agent or agents, with or without a concurrent exposure to a contributory physical agent, such as ultraviolet light. It can result from a nonimmunologic reaction to chemical irritants (irritant contact dermatitis) or from an immunologic reaction to allergens (allergic contact dermatitis). Irritant contact dermatitis is a cutaneous inflammation resulting from a direct cytotoxic effect of a chemical or physical agent, while allergic contact dermatitis is a type IV, delayed or cell-mediated, immune reaction. There are over 57,000 chemicals reported to cause skin irritation, but only 3,700 chemicals are known skin allergens.[3] These are mostly confined to small-molecular-weight chemicals that act as haptens, and usually only a small percentage of people are susceptible to them.

In contact dermatitis, the skin initially turns red and can develop small, oozing vesicles and papules. After several days, crusts and scales form. Stinging, burning, and itching may accompany the skin lesions. With no further contact with the etiologic agent, the dermatitis usually disappears in 1 to 3 weeks. With chronic exposure, deep fissures, scaling, and hyperpigmentation can occur. Exposed areas of the skin, such as hands and forearms, which have the greatest contact with irritants or allergens, are most commonly affected. Over 80% of occupational contact dermatitis involves the hands.[3–5] If the agent gets on clothing, it can induce dermatitis at areas of greatest contact, such as thighs, upper back, armpits, and feet. Dusts can produce dermatitis in areas where the dust accumulates and is held in contact with the skin, such as under the collar and belt line, at the tops of socks or shoes, and in flexural areas, such as the antecubital and popliteal fossae. Mists can produce a dermatitis on the face and anterior neck. Irritants and allergens can be transferred to remote areas of the body, such as the trunk or genitalia, by unwashed hands or from areas of accumulation, such as under rings or interdigital areas. It is often impossible to clinically distinguish irritant contact from allergic contact dermatitis, as both can have a similar appearance and both can be clinically evident as an acute, subacute, or chronic condition (Figs. 22-1, 22-2 and 22-3).

Public Health Importance

Measures of the public health importance of a disease include the absolute number of cases, the incidence rate, the prevalence (rate), the economic impact of the disease, and the prognosis and preventability of the disease.[6]

Specific national data sources on contact dermatitis are limited. In the United States, data from the National Ambulatory Medical Care Survey, a national probability sample survey of nonfederal office-based physicians, showed that in 2006 skin rash was the principal reason for 10.1 million patient visits—1.1% of all visits for that year.[7] Based upon previous surveys, it is estimated that approximately one-half of these visits would have had a diagnosis of contact dermatitis or other eczemas.

In 1988, the National Health Interview Survey (NHIS) included an Occupational Health Supplement, which included questions on dermatitis. Although dated, the Occupational Health Supplement data are the most complete available. The Supplement was scheduled to be repeated in 2010.[8] The survey consisted of personal interviews of people in randomly selected households. For 30,074 people participating in the NHIS, the period prevalence was 11.2% for all dermatitis and 2.8% for contact dermatitis. Projecting these results to the U.S. working population resulted in an estimate of 13.7 million people with dermatitis and 3.1 million people with contact dermatitis.[9]

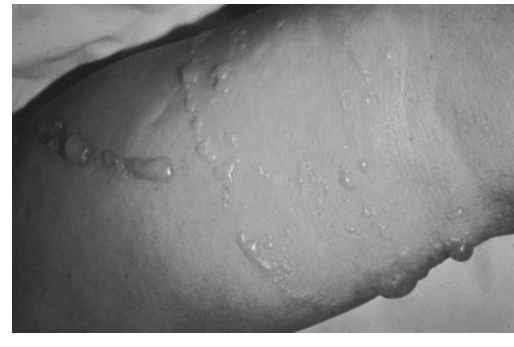

Figure 22-1. Acute contact dermatitis from exposure to ethylene oxide, a strong irritant.

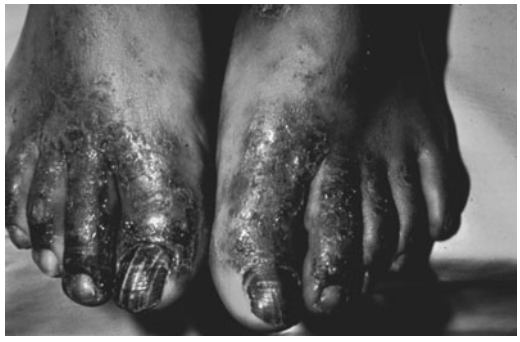

Figure 22-2. Subacute dermatitis from the rubber accelerator mercaptobenzothiazole, which is found in the rubber in a work boot.

Figure 22-3. Chronic dermatitis from exposure to kerosene, a solvent that was used for cleaning the skin.

More information is available on the public health impact of occupational contact dermatitis. Specific national occupational disease and illness data are available from the U.S. Bureau of Labor Statistics (BLS), which conducts annual surveys of approximately 176,000 employers selected to represent all private industries in the United States.[10] All occupational skin diseases or disorders, including contact dermatitis, are tabulated in this survey. BLS data show that occupational skin diseases consistently accounted for 30% to 45% of all cases of occupational illnesses from the 1970s through the mid-1980s, and in recent years accounted for nearly 18% of all occupational illness.[10] A decline in this proportion may be partially related to an increase seen in disorders associated with repeated trauma.

BLS data for occupational skin diseases for 2002 to 2007 are shown in Table 22-1. In 2007, BLS estimated 35,300 cases of occupational skin diseases or disorders in the U.S. workforce.[10] However, because of BLS survey limitations, it has been estimated that the number of actual occupational skin diseases may be 10 to 50 times higher than that reported by the BLS.[11] This increase would potentially raise the number of occupational skin disease cases to between 350,000 and 1.8 million per year. In 2007, BLS data showed an annual incidence rate of 37 cases per 100,000 workers.[10]

Table 22-1. Number and Rate of Occupational Skin Diseases per Year, United States, 2002–2007

Year	Number (in thousands)	Rate (per 100,000)
2002	44.9	51
2003	43.4	49
2004	38.9	44
2005	40.1	44
2006	41.4	45
2007	35.3	37

Source: Bureau of Labor Statistics Annual Survey, 1973–2007.

In 1988, the Occupational Health Supplement of the NHIS indicated that the period prevalence for occupational contact dermatitis occurring in the preceding year was 1.7%. Projecting these results to the U.S. working population resulted in an estimate of almost 1.9 million people with occupational contact dermatitis and a 1-year period prevalence of 1,700 per 100,000 workers for the year.[9] The numbers and rates in the BLS and NHIS surveys are not directly comparable because they rely on different information sources with different ascertainment methods and different case definitions.

The economic impact of a disease can be measured by the direct costs of medical care and workers' compensation or disability payments, and the indirect costs associated with lost work days and loss of productivity. In 1984, the estimated annual direct and indirect costs of occupational skin diseases exceeded $22 million.[11] However, considering that the actual annual incidence may be 10 to 50 times greater than reported in the BLS data, the total annual cost of occupational skin diseases in 1984 may have ranged from about $220 million to $1 billion.[11] (These estimates do not include costs of occupational retraining.)

The Safety and Health Assessment and Research for Prevention (SHARP) program analyzed data from Washington State workers' compensation dermatologic claims and work-related skin diseases reported through SHARP's "sentinel provider network" in the 1993–1997 period. During these 5 years, close to 5,000 claims were accepted for work-related skin disorders and 42,471 lost workdays were reported, costing more than $1.6 million in time loss payments and $1.5 million in medical bills. Comparison with provider network data estimated that compensation data underrepresents the number of work-related skin disorders by more than four-fold.[12]

An analysis of Oregon workers' compensation claims data for 1990 through 1997 estimated the average claim rate of occupational dermatitis to be 5.7 per 100,000 workers. In this 8-year period, 727 workers' compensation claims were filed for occupational dermatitis, of which 611 were determined to be compensable. The total cost of all dermatitis claims was $2.2 million, averaging about $270,000 annually. Oregon claim rates are lower than other states since reporting is not mandatory unless the incident requires 3 or more days of disability leave, and injuries from self-employed workers, such as hairdressers, are not reported. The average cost per claim was $3,552, and the average disability time was 24 days.[13]

A review of 2001 BLS data showed that, of the 38,900 reported cases of occupational skin diseases, 6,051 (16%) resulted in days away from work.[10] The mean time away from work was 3 days, but 19% of lost workday cases had 11 or more days away from work. Of those with days away from work, 78% had a diagnosis of dermatitis. In 2007, of the 35,300 skin disease cases, 5,640 (16%) resulted in days away from work, with a median of 4 days lost.[10] Of these, 64% had dermatitis.

Studies on the prognosis of occupational contact dermatitis stress the importance of primary prevention. A questionnaire survey of 124 patients 5 years after they were initially diagnosed with irritant hand dermatitis found 18% with low, 50% with medium, and 32% with severe hand dermatitis. Severity was measured by self-reported frequency of relapses, frequency of dermatologist visits, and use of topical corticosteroids.[14] A questionnaire survey of 540 patients 1 year after initial diagnosis of occupational hand dermatitis found 41% improved and 25% with persistently severe or aggravated symptoms. Poor prognosis was associated with the presence of atopic dermatitis and being 25 years of age or older. Prognosis was not affected by whether the dermatitis was irritant or allergic. Those with severe occupational hand dermatitis at baseline had a higher risk of taking sick leave and job loss in the following year than those with mild cases. The study found no significant improvement in the disease after the change of job. Severe impairment of quality of life at baseline was a strong predictor of prolonged sick leave, but the presence of depression did not affect prolonged sick leave.[15]

Persistent postoccupational dermatitis (PPOD) can occur following allergic and irritant contact dermatitis. Persistent postoccupational dermatitis begins as a clear-cut occupational contact dermatitis, initially gets better when removed from exposure, but with time the capacity for resolution is lost and persistent dermatitis develops. Predictive factors for PPOD include duration of disease, inability to avoid causative agents, and age.[16] Widespread hand dermatitis on initial examination was found to be the greatest factor for a poor long-term prognosis; other important factors identified include young age at onset of hand dermatitis, history of childhood eczema, and contact allergy.[17] Outcomes may or may not be influenced by leaving the dermatitis-provoking job. In addition, many skin disorders, including contact dermatitis, have been shown to have a significant impact on quality of life.[18–21]

Over the years, there have been changes in the epidemiology of occupational skin diseases. A decrease in the absolute number of cases and the incidence rate in the BLS survey from the 1970s to the early twenty-first century may be attributable to several factors, including changes in industry and industrial practices, increased awareness and preventive measures, and possible underreporting, underrecognition, and misclassification. Still, occupational contact dermatitis remains a relatively common disease with a noteworthy public health impact. These factors, along with the potential chronicity of the disorder, its effect on an individual's vocational and avocational activities, and its preventability make occupational contact dermatitis a disease of public health importance.

Population at Risk and Etiologic Agents

There are many occupations that have unique exposures resulting in occupational contact dermatitis. Total numbers and incidence rates of occupational dermatologic conditions, by major industry division, based on the BLS survey for 2007 are shown in Table 22-2.[10] The greatest number of cases of occupational skin diseases is seen in manufacturing, but the highest incidence rate is seen in the category agriculture, forestry, fishing, and hunting.

In the NHIS, the occupational groups with the highest prevalence of self-reported occupational contact dermatitis included physicians, dentists, nurses, pharmacists, and dieticians (5.6%); public transport attendants, cosmetologists, and other personal service occupations (4.9%); health care therapists, technologists, technicians, and assistants (3.5%); and mechanics and repairers of vehicles, engines, heavy equipment, and machinery (3.5%).[9] Of all accepted workers' compensation claims for occupational contact dermatitis in Oregon, the occupations with highest claim rates were farming, fishing, and forestry workers (18.2%); machine operators and assemblers (16.5%); service-related workers (15.3%); laborers (13.7%); precision production crafts workers (8.0%); and protective services workers (5.7%), followed by technicians and related support workers, transportation and material movers, and professional specialty, administrative support, executive, administrative, and sales employees.[13] Self-employed individuals, such as hairdressers and cosmetologists, are not represented in these claims.

Table 22-2. Number and Incidence of Occupational Skin Diseases, by Industry Sector, 2007

Industry	Number	Incidence per 100,000
Agriculture/forestry/fishing/hunting	1,200	123
Manufacturing	8,800	62
Education and health services	7,700	57
Leisure and hospitality	4,100	46
Construction	2,600	36
Professional and business services	3,600	26
Other services	800	26
Trade/transport/utilities	5,500	24
Information	400	17
Mining	<100	13
Financial activities	600	8
Total	**35,300**	**37**

Source: Bureau of Labor Statistics Survey

The etiology of irritant contact dermatitis is often multifactorial, but the most common skin irritant is wet work, defined as exposure of skin to liquid for more than 2 hours per day, use of occlusive gloves for more than 2 hours per day, or frequent hand cleaning.[22,23] Other common causes of irritant contact dermatitis include soaps and detergents, solvents, food products, cleaning agents, plastics and resins, petroleum products and lubricants, metals, and machine oils and coolants.[22,23] Frictional irritant contact dermatitis can be caused from low humidity, heat, paper, tools, metals, fabrics, plastics, fibrous glass and other particulate dusts, and cardboard, among other causes.[24,25] Causes of allergic contact dermatitis include plants (poison ivy, poison oak, and poison sumac), metallic salts, germicides, plastic resins, rubber additives, and fragrances.[26] The most common skin patch test allergens found to be positive in North American dermatologic patients along with potential sources of exposure are shown in Table 22-3.[27] In health care workers with occupational contact dermatitis who were skin-patch tested, the most common relevant allergens included thiuram mix, carba mix, and glutaraldehyde.[28] The most relevant skin allergens in patients with occupational hand dermatitis who were skin-patch tested included thiuram mix, carba mix, bacitracin, methyldibromoglutaronitrile/phenoxyethanol, formaldehyde, nickel, and cobalt.[29]

Diagnosis

The environmental cause or work-relatedness of contact dermatitis may be difficult to prove. The accuracy of the diagnosis is related to the skill level, experience, and knowledge of the health professional who makes the diagnosis and confirms the relationship with environmental or workplace exposures. Guidelines are available for assessing the work-relatedness of dermatitis, but even with guidelines the diagnosis may be difficult.[2,30] The diagnosis is based on the medical history, including the occupational and environmental history, and physical findings.

Table 22-3. North American Contact Dermatitis Group Patch-Test Results, 2003–2004[27]

Test Substance	Common Sources	Percent Positive
Nickel sulfate 2.5% pet	Metals, jewelry	18.7
Neomycin sulfate 20% pet	Creams, lotions	10.6
Myroxylon pereirae 25% pet		10.6
Fragrance mix 8% pet	Toiletries, scented products	9.1
Quaternium-15 2% pet	Cosmetics, sunscreens	8.9
Sodium gold thiosulfate 0.5% pet	Jewelry, dental products	8.7
Formaldehyde 1% aq	Fabrics, skincare products	8.7
Cobalt chloride 1% pet	Metals, jewelry	8.4
Bacitracin 20% pet	Ointments, creams	7.9
Methyldibromoglutaronitrile/phenoxyethanol 2.5% pet	Biocides, skincare products	6.1
p-Phenylenediamine 1% pet	Hair dyes, leather	4.7
Thiuram mix 1% pet	Rubber, pesticides	4.6
Potassium dichromate 0.25% pet	Cement, leather	4.3
Carba mix 3% pet	Rubber, pesticides	4.0
Diazolidinylurea 1% pet		3.5
Propylene glycol 30% aq	Cosmetics, topical medications	3.3
Imidazolidinylurea 2% pet		2.9
Colophony 20% pet		2.8
Tixocortol-21-pivalate 1% pet		2.7
Diazolidinylurea 1% aq		2.5
Ethylenediamine dihydrochloride 1% pet		2.4

Note: Prevalence of 20 most common positive reactions (*n* varies from 5,106 to 5,145).
aq, in aqueous solution; pet, in petrolatum.

The importance of the patient's history of exposures and disease onset is clear. Standardized questionnaires for surveying work-related skin diseases are available and can be helpful in the workplace.[31] In irritant contact dermatitis, there are no additional confirmatory tests. Patch tests may be used to distinguish allergic contact dermatitis from irritant contact dermatitis.[23] In many instances, allergic contact dermatitis can be confirmed by skin patch tests using specific standardized allergens or, in some circumstances, by provocation tests with nonirritating dilutions of industrial contactants. Irritant contact dermatitis may be overestimated (and allergic contact dermatitis underestimated) due to time, expense, and availability of skin patch testing; physician experience; and the limited availability of allergens in the United States.[32] Skin patch tests should only be conducted by health care professionals trained in conducting and interpreting the tests. Skin patch tests should never be conducted with unknown substances.

The following questions can be used as criteria for determining work-relatedness:

1. Is the clinical appearance consistent with contact dermatitis?
2. Are there workplace exposures to potential cutaneous irritants or allergens?
3. Is the anatomic distribution of dermatitis consistent with cutaneous exposure in relation to the job task?
4. Is the temporal relationship between exposure and onset consistent with contact dermatitis?
5. Are nonoccupational exposures excluded as probable causes?
6. Does dermatitis improve when the person is away from the exposure to the suspected irritant or allergen?
7. Do patch tests or provocation tests identify a probable causal agent?[30]

Treatment and Prevention

Avoiding etiologic irritants and allergens, in addition to wet work, is the first step in any treatment regimen. Dermatitis is treated according to its clinical stage. Acute dermatitis treatment options can include a short course of systemic steroids, topical steroids, and soothing compresses or baths. Antihistamine therapy or use of sedatives may be helpful to decrease pruritus. If secondary infection is present, topical or

systemic antibiotics are indicated. Subacute dermatitis and chronic dermatitis are usually treated with topical steroid therapy and lubrication of the skin. Liberal use of skin moisturizers helps to prevent contact dermatitis by maintaining a healthy skin barrier, and it also helps to repair this barrier if it has been compromised.[23] Potential dangers of long-term use of topical steroids, especially high-potency steroids, include systemic effects and skin atrophy. In addition, contact dermatitis can be caused by ingredients found in topical agents, including antibiotics, fragrances, vehicles, or steroids.

Strategies in the prevention of occupational contact dermatitis include the following:

- Identifying irritants and allergens
- Substituting chemicals that are less irritating or allergenic
- Establishing engineering controls to reduce exposure
- Utilizing personal protective equipment (PPE), such as gloves and special clothing
- Emphasizing personal and occupational hygiene
- Establishing educational programs to increase awareness in the workplace[33]

Chemical changes in industrial materials have been beneficial. For example, the addition of ferrous sulfate to cement to reduce the hexavalent chromium content has been effective in reducing occupational allergic contact dermatitis in Europe. Protective gloves can reduce or eliminate skin exposure to hazardous substances if used correctly, but they may actually cause or worsen hand dermatitis (by permeation and penetration) if selected poorly and used improperly (by contamination).[34] The use of PPE may occlude irritants or allergens next to the skin, and PPE components may directly irritate the skin, so the correct use of PPE is at least as important as their selection.[35] Similarly, the excessive pursuit of personal hygiene in the workplace may actually lead to misuse of soaps and detergents and resulting irritant contact dermatitis. Proper hand-washing methods and adequate moisturizing is valuable in preventing contact dermatitis.[5] The effectiveness of barrier creams is controversial since there are limited data on the protective nature of these topical products during actual working conditions involving high-risk exposures. Educating the workforce about skin care, exposures, and PPE use is an especially important measure in the prevention of occupational contact dermatitis.[36–38]

CONTACT URTICARIA

Urticaria is defined as the transient appearance of elevated, erythematous pruritic wheals or serpiginous exanthem, usually surrounded by an area of erythema. In addition, areas of macular erythema or erythematous papules may also be present. These skin lesions appear and peak in minutes to hours after the etiologic exposure, and individual lesions usually disappear within 24 hours. Urticarial lesions usually involve the trunk and extremities, although they can involve any epidermal or mucosal surface. Large wheal formation, where the edema extends from the dermis into the subcutaneous tissue, is referred to as angioedema. This condition is more commonly seen in the more distensible tissues, such as the eyelids, lips, earlobes, external genitalia, and mucous membranes.

Urticarial lesions can be classified in one or more of the following categories based upon characteristic features:

1. *Duration or chronicity*: Acute or chronic
2. *Clinical distribution of the lesions or the extradermal manifestation*: Localized, generalized, or systemic associated with rhinitis, conjunctivitis, asthma, or anaphylaxis
3. *Etiology*: Idiopathic or cause specific
4. *Routes of exposure*: Direct contact, inhalation, or ingestion
5. *Mechanisms*: Nonimmunologic, immunologic, or idiopathic

Acute urticaria ranges from a single episode to recurrences over a period of less than 6 weeks. Common causes of acute urticaria include insect bites or stings and food or drug allergies. Chronic urticaria occurs daily, or almost daily, over a period longer than 6 weeks. Food, drugs, and infections can also be causes of chronic urticaria. However, in the chronic form, the exact causative agents may never be identified. In most cases of urticaria, the cause is unknown.

Occupational urticaria is presumed or proven to be caused by exposure to one or more substances or physical agents in the workplace. Occupational urticaria may be acute or chronic, localized or generalized, or associated with systemic manifestations, such as asthma. In occupational settings, direct contact with substances, and possibly inhalation, may be the most common routes of exposure inducing urticaria. The pathologic mechanisms may be nonimmunologic, immunologic, or not known.[39] Contact urticaria is defined as urticaria that occurs after direct skin contact with a substance. Another type of immediate skin reaction, "protein contact dermatitis," has clinical features of both immediate and delayed hypersensitivity and is associated with atopy. Pruritis, erythema, and urticarial or vesicular lesions occur within 30 minutes of contact with proteins (fruits, vegetables, spices, plants, grains, enzymes, or animal proteins) followed by eczematous dermatitis. Protein contact dermatitis typically affects the hands.[39–42] Urticarias that result from nonchemical exposures are commonly classified as physical urticarias. These include mechanical urticarias, caused by trauma, pressure, friction, and vibration, and urticaria resulting from local exposure to water or to physical agents, such as cold, heat, and solar radiation.

Public Health Importance

Data specific for environmental and occupational urticaria are limited. In 2007, BLS estimated 35,300 cases of occupational skin diseases or disorders in the U.S. workforce.[10] Further information is available on the 5,640 cases that involved days away from work. Of this subgroup, 80 (1.4%) had urticaria/hives. Their median time away from work was 4 days.

Population at Risk and Etiologic Agents

In general, risk factors for contact urticaria include a history of atopy; a compromise to the barrier function of intact skin due to conditions such as eczema, abrasions, and ulcers; and, in some cases, occupation. Based upon reviews of epidemiologic studies, exposures, and patterns seen in case reports, several occupations may be at higher risk for the development of contact urticaria. These include food handlers, cooks, caterers, and bakers; general health care workers, dental professionals, and pharmaceutical industry workers; animal handlers, such as laboratory workers and veterinarians; and gardeners, florists, woodworkers, and agricultural workers.

For food handlers, cooks, caterers, and bakers, the following foods have been reported to induce contact urticaria: apples, bananas, beans, beer, caraway seeds, carrots, eggs, endives, fish, garlic, grains, kiwi fruit, lettuce, meat (beef, chicken, lamb, liver, pork, and turkey), milk, onions, olives, peaches, potatoes, rice, shellfish, spices, strawberries, and tomatoes.[39,41,43–46] Bakers can develop contact urticaria and other systemic symptoms after exposure to rye, wheat, barley, oat, and buckwheat flours, cinnamon, vanillin, and additive flour enzymes, such as alpha-amylase.[39,41,44]

In health care, dental, and pharmaceutical environments, dermal exposure to a variety of medications or chemical disinfectants can put workers at risk. Exposures that can cause contact urticaria include aminothiazole, bacitracin, benzocaine gel, cefotiam, cephalosporins, chloramine, chloramphenical, chlorhexidine, chlorocresol, ethylene oxide, gentamicin, neomycin, nitrogen mustard, penicillin, pentamidine isethionate, phenothiazines, piperacillin rifamycin, and streptomycin.[39,45,46] Recent studies have found that the increased use of nonpowdered latex gloves and nonlatex gloves in health care settings have resulted in fewer cases of natural rubber latex allergy, at one time an important cause of contact urticaria in health care professionals.[47–49]

Contact urticaria has been found to be caused by animal hair, dander, placenta, saliva, seminal fluid, amniotic fluid, milk, blood, insects, and bacterial and fungal enzymes.[39,50] Slaughterhouse workers, laboratory workers, veterinarians and their staff, and dairy farmers are at risk for developing contact urticaria when exposed to these allergens.

Certain woods and plants can cause contact urticaria. These include the larch, limba, obeche (African maple), and teak woods and plants, such as algae, cacti, chrysanthemum, *Ficus benjamina* (weeping fig), lilies, *Limonium tataricum*, *Phoenix canariensis* (canary palm),

Spathiphyllum walisii (spathe flower), tobacco, tulips, and fungi (shiitake mushrooms). High-risk occupations include agricultural workers, carpenters, florists, gardeners, and woodworkers. Caterpillar hair, insect stings, and moths can also cause contact urticaria in outdoor workers. Agricultural workers may also be exposed to fertilizers and pesticides, some of which can cause contact urticaria.[39]

A variety of industrial chemicals can cause contact urticaria, including the following: acrylic monomers (plastics), polyfunctional aziridine hardener (aziridine reacted with a multifunctional acrylic), aliphatic polyamines (epoxy resins), alkyl-phenol novolac resin, ammonia, castor bean (fertilizers), diethyltoluamide (DEET), formaldehyde (used in clothing, leather, fumigation, and resins), isocyanates, lindane (a parasiticide), paraphenylenediamine, phenylmercuric priopionate (an antibacterial fabric softener), plastic additives (such as butylhydroxytoluene and oleylamide), reactive dyes, sodium sulfide (used in photographs, dyes, and tanning), sulfur dioxide, vinyl pyrrolidone, xylene, and other solvents.[39,45,46] Contact urticaria can occur with exposure to a variety of metal salts, including iridium, nickel, platinum, and rhodium.

Diagnosis and Treatment

The diagnosis of environmental or occupational urticaria is based on the medical and exposure history, physical findings, and in vitro or in vivo testing. Proving etiology or work-relatedness may be difficult. Suggested criteria include the following:

1. Documentation of urticaria by physical examination
2. Exposure to an agent known or presumed to cause urticaria
3. A temporally consistent relationship between exposure and onset of urticaria (usually 30 to 60 minutes)
4. Associated medical symptoms and localization of urticaria consistent with the route of exposure
5. Resolution of the urticaria away from the exposure
6. Exclusion of nonenvironmental or nonoccupational causes
7. Medical testing results indicating allergy to a substance in the environment or workplace. Useful medical tests include the open or closed patch test, prick or scratch test, and tests demonstrating specific IgE to suspect occupational antigens, such as by radioallergosorbent (RAST) assays. Evaluating with both prick and patch testing has been recommended to avoid missing a common accompanying allergic contact dermatitis.[39,44,51]

In cases of environmental or occupational urticaria where a specific causal agent can be identified, the initial treatment is avoidance of the offending agent. First-generation antihistamines, such as diphenhydramine or hydroxyzine, which block H1 receptors, can be employed initially, but they can cause sedation; this may present a safety issue for certain occupations, such as heavy equipment operators. When sedation occurs or presents a safety concern, nonsedating, second-generation antihistamines may be employed. When H1 histamine blockers alone are not sufficient, they may be combined with H2 blockers or doxepin, a tricyclic antidepressant with potent H1 and H2 blocking activity. Doxepin is extremely sedating and should be used cautiously, if at all, when safety concerns arise on the job. Oral corticosteroid therapy may be employed for severe cases of chronic urticaria, especially those associated with angioedema.

Prevention

Strategies in the prevention of environmental and occupational urticaria overlap with those strategies used in the prevention of contact dermatitis and include:

- Identifying allergens
- Substituting chemicals that are nonallergenic
- Establishing engineering controls to reduce exposure
- Utilizing PPE, such as gloves and special clothing
- Emphasizing personal and occupational hygiene
- Establishing educational programs to increase awareness in the workplace (Recommendations for preventing allergic reactions to

natural rubber latex in the workplace have been published by the National Institute for Occupational Safety and Health [NIOSH].)[52]

DERMATOLOGIC INFECTIOUS DISEASES

Environmental or occupational dermatologic infectious diseases are diseases that result from exposure to an infectious agent found in the environment or workplace and have a major manifestation on the skin surface. (Secondarily infected wounds will not be discussed here.) Many environmental and occupational dermatologic infectious diseases not only cause cutaneous signs and symptoms but also systemic effects. Exposure can occur through direct skin contact (epicutaneous), inoculation (percutaneous), or via the respiratory system (inhalational).

Public Health Importance

Epidemiologic data specifically related to environmental or occupational dermatologic infectious diseases are very limited. Other than limited descriptions in case presentations, case studies, and epidemic investigation reports, little is known about the epidemiology of most of these diseases in the United States. In many cases, it is difficult to definitively prove that the disease process is occupationally related. There is limited information on occupational dermatologic infectious diseases in the BLS data. In 2006, there were an estimated 41,400 cases of occupational skin diseases or disorders, or 4.5 per 10,000 workers.[10] Of these, 5,720 resulted in one or more days away from work. Infections of the skin and subcutaneous tissue accounted for 27%, or 1,570 cases (0.2 per 10,000 workers). Most of these cases were listed as cellulitis and abscess (690); the others were included in unspecified diseases (630), not elsewhere classified (180), and pilonidal cyst (40).[11] Median time away from work was 10 days. In 2006, under a separate category of infectious and parasitic diseases, the BLS recorded 2,550 cases, which resulted in at least 1 day away from work.[10] Few diagnoses were listed in this category, but diagnoses with potential skin manifestations included scabies/chiggers/mites (1,330), viral diseases accompanied by exanthem (380), chickenpox (280), herpes zoster (40), dermatophytosis, including athlete's foot and tinea (40), and Lyme disease (20).

Population at Risk and Etiologic Agents

Environmental and occupational dermatologic infectious diseases can be grouped by etiologic agent into the following categories: bacterial, rickettsial, viral, superficial fungal, subcutaneous fungal, systemic fungal, and parasitic.[53] In general, risk of infection can be associated with individual susceptibility, including factors such as immune status and trauma to the skin breaching its protective barrier; the distribution of the pathogen in the environment; and exposure to the pathogen, considering its reservoir, mode of transmission, and conditions in which the pathogen thrives. Reservoirs of the pathogens include people, such as co-workers, clients, patients, or children; animals and animal products; soil and plant materials; ticks and insects; and water and marine life. Conditions in which pathogens can thrive and increase susceptibility include wet conditions, such as wet work, and hot and humid environments. The environmental and occupational dermatologic infectious diseases associated with these sources and conditions are listed in Table 22-4. In addition, laboratory personnel working directly with pathogens are at risk of infection. Recently, there has been concern over possible work-duty exposures for first responders and health care professionals during a bioterrorist attack.

Diagnosis and Treatment

In many cases, it is often difficult to definitively prove the environmental or occupational relatedness of the disease process. Questions to be answered by the clinician include the following:

1. Is the patient's condition a dermatologic infectious disease?
2. Is the organism found in the patient's environment?
3. Was there an opportunity for the person to become infected in the workplace or general environment?
4. What other exposures, such as recreational activities, must be considered?

Table 22-4. Exposures Associated with Dermatologic Infectious Diseases

People, Patients, and Children	Animals and Animal Products	Soil and Plants
Tuberculosis (cutaneous)	Anthrax	Anthrax
Methicillin-resistant *Staphylococcus aureus* infection	Brucellosis	Dermatophytes (geophilic)
Herpetic whitlow	Cat scratch disease	Chromomycosis
Warts	Erysipeloid	Mycetoma
Measles	*Mycobacterium bovis* infection	Sporotrichosis
Rubella	Tularemia	Blastomycosis
Chickenpox	Methicillin-resistant *Staphylococcus aureus* infection	Paracoccidioidomycosis
Herpes zoster (shingles)	Orf	Cutaneous larva migrans
Hand-foot-mouth disease	Milker's nodules	**Wet Work and Hot and Moist Environments**
Erythema infectiosum (fifth disease)	Monkeypox	Candidiasis
Dermatophytes (anthropophilic)	Warts	Dermatophytoses
Scabies	Dermatophytes (zoophilic)	Tinea versicolor
	Mites	
Ticks and Insects		
Lyme disease	**Water, Marine, Fish, and Shellfish Exposures**	
Tularemia	Erysipeloid	
Rocky Mountain spotted fever	*Mycobacterium marinum* granuloma	
Typhus	Tularemia	
Ehrlichiosis	*Vibrio vulnificus* infection	
Leishmaniasis	*Aeromonas hydrophila* infection	
	Photobacterium (Vibrio) damsela infection	
	Vibrio parahaemolyticus infection	
	Pseudomonas aeriginosa infection	
	Warts	
	Cercarial dermatitides	

Diagnosis and treatment are disease-specific and thus beyond the scope of this chapter.

Prevention

Clinicians should view each case of a potential environmental or occupational dermatologic infectious disease from a broader public health perspective as a potential sentinel health event. This recognition and resultant action by clinicians, in appropriate consultation with public health officials, could lead to potential disease prevention in other people. This can only occur with proper diagnosis, a high level of suspicion by the clinician in suspecting environmental or workplace exposures, ultimate confirmation of the association to the exposures that caused the disease, and implementation of measures to reduce these exposures. If successful, this approach would lead to the prevention of relapses and of new cases of dermatologic infectious diseases.

SKIN CANCERS

In 1775, Sir Percival Pott in England first made the link between occupational exposures (soot clinging to skin in chimney sweeps) and skin cancer (squamous cell carcinoma of the scrotum). In 1894, Dr. Paul Unna in Germany drew attention to the association between chronic sun exposure and skin cancers in outdoor workers, such as farmers and sailors.

Skin cancers include melanoma, basal cell carcinoma, and squamous cell carcinoma. Excessive sun exposure is associated with premature skin aging, actinic keratosis (a type of premalignant skin change), and skin cancer.[54] Nonionizing ultraviolet radiation (UVR) from the sun is

the primary cause of skin cancer, in general, and is also the primary cause of occupational skin cancer. The International Agency for Research on Cancer has noted that there is sufficient evidence to establish UVR as a human carcinogen. In addition, a variety of chemical exposures may play a role in the etiology of skin cancers.

Public Health Importance

Melanoma is the least prevalent of the three skin cancers, but it carries the greatest risk of fatality, accounting for 85% of skin cancer deaths in the United States. The American Cancer Society estimated that, for 2008, there would be 62,000 new cases of melanoma and 8,400 deaths due to this disease.[55] Melanoma is likely to be related to excessive sun exposure, although the relationship is complex; it seems to be associated with severe sunburns during childhood. Basal cell carcinoma and squamous cell carcinoma are more clearly related to sun exposure, probably as a result of cumulative, chronic exposure. Basal cell and squamous cell skin cancers are, by far, the most common cancers in the United States, with over 1 million new cases and over 2,200 deaths each year.

Population at Risk and Etiologic Agents

Implicated etiologies for skin cancers include nonionizing radiation from sunlight exposure and other sources of UVR, ionizing radiation, and thermal and chemical stimuli. Outdoor workers may receive up to six to eight times the dose of UVR compared to indoor workers,[56–57] and rates for some skin cancers among outdoor workers have been associated with cumulative UVR exposure.[58] Studies have found an increased risk of skin cancer among agricultural workers, welders, watermen, police officers, physical education teachers, pilots, and cabin attendants.[59] According to the BLS, in 2003, over 6% of the workforce (over 8 million workers) were listed in the following potential outdoor occupations: construction, farm, and forestry workers; fishing workers; gardeners; groundskeepers; mail carriers; amusement/recreation attendants; and surveying and mapping workers. There are likely many more workers occupationally exposed to UVR from sunlight as well as artificial sources, such as welding arcs. In addition, workers exposed to ionizing radiation and chemical agents, such as polycyclic aromatic hydrocarbons, arsenic, alkylating agents, and nitrosamines may be at increased risk. Arsenic intoxication, which can result from ingestion of contaminated well water, has resulted in hyperpigmentation, palmar and plantar arsenical keratoses, and superficial squamous cell and basal cell carcinomas. Other risk factors for skin cancers include fair skin types, fair hair, blue eye color, and having many moles or nevi.

Diagnosis and Treatment

Diagnosis is based upon history, physical findings, and pathology results. Treatment of specific skin cancers, which is beyond the scope of this chapter, depends on the specific type of skin cancer, size, depth, and location of the lesion, and evidence of metastases.

Prevention

The strategies of prevention are primarily based on preventing excessive UVR exposure.[60] This can be accomplished by limiting exposure to sunlight, introducing changes in practices to limit sun exposure during peak UVR hours (10 a.m. to 4 p.m.), wearing UVR-protective clothing and wide-brimmed hats, generously applying broad-spectrum, water-resistant sunscreens that block both UVA and UVB, and wearing UV-blocking sunglasses. Limiting skin exposure to chemicals known to play a role in skin cancer is also important.

In many areas, the National Weather Service, in cooperation with the Environmental Protection Agency, issues daily predictions for UVR exposure. The daily UV Index, reported on a scale from <2 (very low) to 11+ (very high), is part of selected local weather broadcasts and can be used to warn outdoor workers and others of potential high-exposure days, when prevention strategies should be emphasized.

OTHER ENVIRONMENTAL AND OCCUPATIONAL SKIN DISEASES

Many other skin diseases may be related to environmental and occupational exposures (Table 22-5).

Table 22-5. Other Environmental and Occupational Skin Diseases and Examples of Associated Exposures

Condition	Associated Exposures
Hyperkeratoses/calluses/fissuring/blistering	Mechanical trauma
Burns	Heat, electricity, radiation, acids, alkalis
Frostbite/immersion foot, chilblain	Cold, moist environments
Folliculitis/furuncles and acneiform dermatoses	Oils, greases
Chloracne	Chlorinated hydrocarbons
Photodermatitis (phototoxic and photoallergic)	Plants, coal tar, creosote, fragrances
Depigmentation/leukoderma	Phenols, hydroquinones
Hyperpigmentation/occupational melanosis	Coal tar, pitch
Skin discolorations	Silver, gold
Occupational Raynaud disease/vibration white finger	Tools causing hand/arm vibration
Miliaria rubra/prickly heat	Hot, humid work environments
Asteatotic eczema/winter eczema	Cool, dry work environments
Granulomatous dermatoses	Beryllium, zirconium
Ulcerative lesions	Chromium, chemical burns
Connective tissue disorders, such as scleroderma	Silica, vinyl chloride
Nail disorders	Mechanical trauma, contact dermatitis, infections
Alopecia	Chlorbutadine, dimethylamine

Other skin diseases may not be caused by occupational exposures, but they may be exacerbated by such exposures. Examples include lesions of psoriasis produced at sites of skin friction or injury, heat exacerbating rosacea, and wet work initiating dyshidrotic eczema.

CONCLUSION

Environmental and occupational skin diseases include allergic contact dermatitis, irritant contact dermatitis, contact urticaria, a variety of infectious diseases, skin cancers, and other diseases. Thorough investigations of workers with occupational skin diseases can be difficult. Workers should be encouraged to report all potential work-related skin problems to their employers and to their physicians. Because the work-relatedness of skin diseases may be difficult to prove, each person with possible work-related skin problems needs to be fully evaluated by a physician, preferably one familiar with occupational and dermatological conditions. A complete evaluation includes a full medical history, including an occupational and environmental history, and a review of exposures. NIOSH is revamping its skin notations for use in distinguishing between systemic, localized, and sensitizing health effects of dermal chemical exposures.[61] A complete evaluation also includes a physical examination; diagnostic tests, such as skin patch tests to detect causes of allergic contact dermatitis; and follow-up to assess the clinical course of the affected person. Individuals with occupational skin diseases should be protected from exposures to presumed causes or exacerbators of the disease. In some cases of allergic contact dermatitis and contact urticaria, workers may have to be reassigned to areas where exposure is minimal or nonexistent.

Environmental and occupational skin diseases have a major public health impact. They are common, often have a poor prognosis, and result in a substantial economic impact for both affected individuals and society as a whole. Importantly, these diseases are amenable to public health interventions.

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These nine books and book chapters are excellent references on occupational and environmental skin disorders.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health or the U.S. Food and Drug Administration.

23

Cardiovascular Disorders

Kenneth D. Rosenman

Despite a marked decrease in cardiovascular disease (CVD) over the last 30 years, CVD is still extremely common. The U.S. National Heart Lung and Blood Institute estimates that 79.4 million U.S. residents have CVD, including 72 million with hypertension and 16 million with coronary heart disease.[1] Each year, there are an estimated 1.2 million people in the United States who have a myocardial infarction, 700,000 who have a stroke, and 550,000 who develop heart failure.[1] Approximately 32% of all deaths in the United States are caused by CVD.

The etiology of coronary heart disease is multifactorial, including dyslipidemia, cigarette smoking, hypertension, diabetes, and obesity. The etiology of most cases of hypertension is unknown, with less than 10% of cases secondary to known causes. Occupational exposure to specific chemicals and physical and psychological stressors causes CVD. An estimated 5% to 10% of CVD is attributed to occupational and environmental factors, including psychological strain (in jobs with high job demand and low job control), carbon monoxide, lead (Fig. 23-1), and organic solvents.[2] Consideration of occupational and environmental exposures is important in evaluating individuals, such as an auto mechanic with angina pectoris who is exposed to carbon monoxide from the car exhaust. Inadequate consideration of occupational and environmental factors can delay diagnosis and may have fatal consequences.[3,4] Cardiovascular disease is so prevalent that, even if the percentage of CVD attributable to occupational and environmental factors were less than 5% to 10%, many people would still develop CVD due to these factors.

CORONARY ARTERY DISEASE

Work factors may trigger acute CVD events, such as a myocardial infarction or an arrhythmia, or, over prolonged exposure, cause CVD by promoting development of atherosclerosis (see Table 23-1).

Carbon Disulfide

Exposure to carbon disulfide, which is used in manufacturing other chemicals, is usually limited to chemical manufacturing facilities (Fig. 23-2). It causes atherosclerosis by affecting lipoproteins, blood pressure, arterial elastic properties, and oxidative stress.[5] Workers manufacturing viscous rayon, who use carbon disulfide, have increased coronary artery disease. In animal studies, high-fat diets increase the atherosclerotic

Figure 23-1. This worker is exposed to lead by cutting metal that has a lead-based coating.

effect of carbon disulfide. Like the atherosclerotic effect of carbon monoxide, the effect appears reversible with cessation of exposure.[6] Occupational Safety and Health Administration (OSHA) standards do not adequately protect against the development of atherosclerosis.[7]

Other solvents seem not to increase CVD mortality, although some cause arrhythmias. However, a study has found increased CVD among workers exposed to styrene in styrene-butadiene polymer manufacturing facilities.[8]

Carbon Monoxide

Carbon monoxide exposure is produced whenever there is combustion, such as of gasoline, diesel fuel, propane, coal, natural gas, wood, oil, or cigarettes. Carbon monoxide exposure can be fatal when combustion occurs in an enclosed area, such as a gasoline-powered washer used in a basement, a diesel-powered electricity generator used during a blackout, or a propane forklift used in a warehouse. The incidence of carbon monoxide toxicity increases during cold weather when furnaces are used and doors and windows are closed. Carbon monoxide toxicity is therefore more common in colder latitudes. Acute manifestations of coronary artery disease may occur after carbon monoxide exposure. Compared to oxygen, carbon monoxide has 200 times higher affinity for hemoglobin, forming carboxyhemoglobin (COHb), and 50 times higher affinity for myoglobin. In addition, carbon monoxide shifts the oxyhemoglobin curve to the left, which reduces oxygen delivery to tissues, inhibits mitochondrial enzymes, impairs oxygen diffusion into mitochondria, and increases platelet adhesiveness. Animal studies suggest that carbon monoxide has direct effects on cells, with increased oxidant production and lipid peroxygenation, and on the heart, with decreased cardiac output, increased myocardial lactate production, and a decreased threshold for ventricular fibrillation. (See also Chapter 11.)

Carboxyhemoglobin can be measured in either arterial or venous blood or by a new,

Table 23-1. Occupational/Environmental Risk Factors for Cardiovascular Disease

Disease	Risk Factor
Coronary Artery Disease	
Angina/myocardial infarction	Carbon disulfide
	Carbon monoxide
	Decreased lung function
	Lead?
	Nitrates
	Particulates
	Psychological strain (high work demand and low job control)
	Sedentary work
Sudden death	All causes above for angina/ myocardial infarction plus:
	Cold or hot workplaces
	Fluorocarbons
	Solvents
Cardiomyopathy	Arsenic
	Cobalt
	Cold or hot workplaces?
Hypertension	Arsenic?
	Carbon disulfide
	Lead
	Noise
Arrhythmias	Carbon monoxide
	Fluorocarbons
	Solvents
Peripheral Vascular Disease/ Claudication	Arsenic
	Cadmium?
	Carbon monoxide
	Lead?
	Vibration
Cor Pulmonale	Fibrogenic dusts

recently introduced type of pulse oximeter.* Carboxyhemoglobin has a cherry red color, which can be seen in mucous membranes—a specific, but not sensitive, sign. Carboxyhemoglobin is produced endogenously from the normal breakdown of hemoglobin. It is increased due to any cause of hemolysis. Induction of hepatic cytochrome oxidase activity from medications also may increase endogenous COHb production. Normal levels of COHb are less than 1%. Ambient air pollution can increase COHb to 1.7%. Therefore, less than 2% is typically used as a laboratory reference range for COHb.

Workplace standards allow COHb levels as high as 7.4%. Average COHb levels are 4.7% in cigarette smokers, 2.9% in cigar smokers, and 2.2% in pipe smokers. It is unusual for cigarette smokers to have COHb levels greater than 10%. Exposure to environmental (secondhand) tobacco smoke increases COHb levels 0.5% to 1.0%. Alarms on home carbon monoxide detectors, which are based on a concentration-time function, will generally not sound until the concentration of carbon monoxide in the air is at a level that would cause a COHb level of at least 5% to 7%. A summary of the relationship between COHb levels in the blood and carbon monoxide levels in the air is shown in Table 23-2.

Carboxyhemoglobin levels increase with length of exposure. If the concentration of carbon monoxide in the air is constant, an equilibrium will be reached. At rest, this equilibrium is achieved in 8 hours. With exercise and increased pulmonary ventilation, equilibrium is achieved sooner. After exposure to methylene chloride, which is uniquely metabolized to carbon monoxide, COHb levels may reach 10% to 12%.

Treatment of acute carbon monoxide poisoning involves ceasing exposure and decreasing the half-life of COHb (4 to 6 hours on room air) to 40 to 80 minutes by administering 100% oxygen. Treatment with hyperbaric oxygen, which reduces the half-life to 15 to 30 minutes, is especially indicated for pregnant women because fetal COHb is 10 to 15 percentage points greater than maternal COHb and can cause long-term cognitive dysfunction.[9]

Levels of COHb below 30% are associated with headache, nausea, and weakness. Levels of COHb that are 30% and higher are associated with decreased mental alertness and weakness, with increasing likelihood of collapse and coma as levels increase. Myocardial injury, reflected by electrocardiogram (EKG) changes or increases in myocardial enzymes, occurs in over one-third of patients with carbon monoxide poisoning severe enough to be treated with hyperbaric oxygen.[10]

Workplace standards that allow COHb levels up to 7.4% are not protective for individuals

* Oxygen saturation is overestimated by all pulse oximeters in the presence of COHb. In addition, it is overestimated in measurements of arterial blood gases where the value for oxygen saturation is calculated from the arterial oxygen content and the reduction in hemoglobin-binding sites available to oxygen (because of the presence of COHb) is not included in the equation to calculate oxygen saturation.

Figure 23-2. A worker tends machines that spool rayon thread from carbon disulfide. Worker exposure to carbon disulfide was high until this process was enclosed, which reduced worker exposure and, by recycling the carbon disulfide, saved the company a substantial amount of money. (Photograph by Barry S. Levy.)

Table 23-2. Air Levels of Carbon Monoxide Corresponding to a Patient's Carboxyhemoglobin

Carboxyhemoglobin (%)	Concentration of Carbon Monoxide (ppm)	Comments
<1	0	Endogenous production from breakdown of hemoglobin
<2	9	EPA's ambient air 8-hour standard
2.2*	400***	Pipe smoker
2.9*	400***	Cigar smoker
4.7**	400***	Cigarette smoker
5.4	35	EPA's ambient air 1-hour ceiling standard
7.4	50	OSHA's workplace 8-hour TWA standard

*These are average values.
**This is an average value, range 1% to 10%.
***Smoking is intermittent, and inhaled concentration of carbon monoxide over a period of time is less.
EPA, Environmental Protection Agency; OSHA, Occupational Health and Safety Administration; TWA, time-weighted average.

with coronary artery disease. Levels of 2% to 4% can reduce the time to anginal chest pain, ST segment changes on EKG, and claudication in those with coronary artery disease. Levels of 5% and higher can increase cardiac enzymes and arrhythmias.[11] Levels of 6% can increase the frequency of ventricular premature contractions.[12] Levels of 10% and higher can cause myocardial infarctions.

The evidence that carbon monoxide chronically promotes the development of atherosclerosis is not as strong as the evidence that it causes acute adverse CVD effects. Animal studies have shown that carbon monoxide causes increased vascular permeability, arterial wall hypoxia, and platelet adhesiveness—all of which can accelerate atherosclerosis. Epidemiologic studies of firefighters and foundry workers who are

exposed to carbon monoxide have shown increased risk of coronary events while they were employed, which decreased with cessation of exposure. Similarly, studies of cigarette smokers have shown an increased risk of coronary events while smoking that decreases—but not to the risk of nonsmokers—when they quit.

Decreased Lung Function

Decreased forced expiratory volume in 1 second (FEV_1) is a risk for coronary artery disease[13]—a risk that is distinct from the risk of right-sided heart failure (cor pulmonale) due to lung disease. This risk might be due to a hypoxic effect on the heart from lung disease, or substances causing inflammation of both the lungs and the vascular endothelium.

Hot and Cold Temperatures

Working in either a hot or cold environment has triggered acute coronary events in people with underlying coronary artery disease. (See Chapter 12B.) This is consistent with population studies that show that CVD mortality increases during the winter and heat waves.[14,15] Cardiovascular disease mortality above normal during a heat wave is followed by CVD mortality below normal when the heat wave is over, indicating a shift in the time of death in people with significant underlying CVD, without an actual change in the overall mortality rate.

Physiologic changes, such as vasospasm in cold weather or peripheral dehydration and increased blood viscosity in hot weather, coupled with increased physical activity at work, can trigger a coronary event in someone with preexisting coronary atherosclerosis. In addition, endothelial dysfunction appears to increase as ambient temperature increases. There is insufficient evidence that long-term work in a hot or cold environment increases the risk of coronary artery disease.

Lead

A study has shown increased CVD mortality associated with elevated blood lead levels between 5 and 25 μg/dl.[16] This association may be due to increases in blood pressure, homocystine concentration, and/or heart rate variability. (See Chapter 11.)

Nitrates

"Monday morning death" (sudden cardiac death after a weekend away from work exposure) was first described among workers in the explosives manufacturing industry during the 1930s.[17] Death was caused by rebound vasospasm in the coronary arteries from withdrawal from exposure to nitrates. The following chemicals have been identified as having the potential to cause this effect: ammonium and sodium nitrate, ethylene glycol dinitrate, nitroglycerin, dinitroluene, and trinitroluene. "Powder head" is a related phenomenon in which workers develop headache on the weekends away from work due to withdrawal to occupational nitrate exposure. To prevent the headaches, workers placed a few grains of explosive powder in their hat bands throughout weekends in order to maintain exposure to nitrates. (These chemicals can also cause methemoglobinemia.) Increased CVD mortality from myocardial infarction and stroke have been reported in long-term workers in the explosives industry who are exposed to nitrates. This finding may be due to the fact that workers in the explosives industry may develop rebound vasospasm when away from work.

Particulates

Particulate air pollution, which is derived from combustion products, can be toxic to the cardiovascular system. Exposure to fine particulates, mainly from power plants and motor vehicles, is widespread in the general population. Exposure to fine particulates (less than 2.5 μm in aerodynamic diameter, or $PM_{2.5}$) is associated with acute and chronic coronary artery disease.[18,19] Fine particulates cause changes in heart rate variability, arterial dysfunction, and inflammation in old and young people and animals. Specific metals, such as nickel or vanadium, in the fine particulates are thought to play an important role in causing these toxic effects. (See Chapter 6.)

Reducing the levels of fine particulates can have huge benefits. For example, a decrease of

10 μg/m^3 can increase life expectancy by 0.6 years.[18] Ambient air standards in the United States allow fine particulates at a 15 μg/m^3 annual average and a 35 μg/m^3 average in a 24-hour period. Seventeen percent of U.S. residents live in nonattainment areas for the annual standard and 30% live in nonattainment areas for the 24-hour standard. The 15 μg/m^3 annual average is not adequately protective. Since particulate levels are lower indoors, patients with coronary artery disease should reduce their activity and amount of time spent outdoors, depending on the daily concentration of air pollutants. At http://www.airnow.gov, one can determine daily particulate levels throughout the United States and recommendations for outdoor activity, based on an air quality index that ranges from good, to moderate, to unhealthy for sensitive groups, to unhealthy, to very unhealthy, to hazardous.

Workers, such as vehicle mechanics, who are exposed to fine particulates have increased heart rate variability.[20] And workers exposed to diesel exhaust, such as truck drivers or construction workers, have increased risk for coronary artery disease.[21,22]

Psychosocial Factors

Two models have been hypothesized for investigating the role of stress from work on heart disease. The Job Strain Model hypothesizes that work in high-demand jobs with low job-decision latitude increases the risk of heart disease, while high levels of job control negate the negative effects of a demanding job. The Effort-Reward Imbalance Model hypothesizes that work in jobs with high effort, but inadequate reward, increases the risk of heart disease. In the Effort-Reward Imbalance Model, an additional risk factor is overcommitment, characterized by a set of attitudes, behaviors, and emotions reflecting excessive striving in combination with a desire to be approved of and esteemed. One study concluded that 7% to 16% of CVD is secondary to the stress of jobs with high demand and low decision-latitude.[23] Although many studies support job strain as an important CVD risk factor, there continue to be studies that do not find such an association. Studies showing that interventions to decrease job strain and/or increase decision latitude decrease CVD are needed to strengthen the evidence of a causal association.[24] The mechanism of this association may be mediated through known cardiac risk factors. For example, bus drivers who are at increased risk of CVD mortality also have an increased prevalence of hypertension. Their job has a high demand to remain on schedule, but an inability to control traffic or road conditions. Work has begun to examine the interaction between genetic polymorphism and job strain in predicting CVD risk. (See Chapter 14 for a more complete discussion of work stress.)

Part of the problem in the investigation of occupation and heart disease is how occupation is used in studies. Most studies use occupation as a marker of socioeconomic status and, therefore, as a surrogate for lifestyle habits known to be cardiac risks, such as cigarette smoking—rather than as a marker for some psychological or environmental risk factor associated with the occupation.[25]

The metabolic syndrome (abdominal obesity, dyslipidemia, elevated blood pressure, insulin resistance, and a prothrombiotic and proinflammatory state) has been associated with shift work, including in health care workers (see Box 14-2 in Chapter 14).[26] Prospective studies have shown an increased incidence of metabolic syndrome in workers assigned to shift work.[27] However, CVD risk factors may be more frequent in shift workers. In any case, clinicians need to consider CVD risk factors in patients on rotating work shifts, whether shift work causes increased prevalence of CVD risk factors or individuals of a certain socioeconomic status who have a higher prevalence of CVD risk factors are more likely to perform shift work. Further research is needed to evaluate the health effects of different rotating shift schedules. Forward rotating shifts alleviate daytime sleepiness and reduce systolic blood pressure.[28]

Sedentary Work

Studies showing decreased CVD in individuals who exercise regularly have been based on nonwork aerobic activity. This protective effect has not been consistently found in association with physical activity at work.[29] This difference between work and nonwork physical activity has

been attributed to *(a)* the nature of activity at work, which is less likely to be aerobic; and *(b)* an inverse relationship between high physical work activity and decreased time or interest to perform aerobic physical activity outside of work.

Firefighters

Potential CVD risk factors for firefighters include strenuous work in a hot environment, exposure to fine particulates, psychological stress, and exposure to carbon monoxide. The risk of death from coronary heart disease is increased 12 to 136 times during firefighting as compared with nonemergency periods.[30] Previous coronary artery disease, hypertension, and cigarette smoking are all independent predictors of fatal heart attacks during active firefighting.[31] However, career firefighters have not been shown to have more coronary heart disease than other groups of workers.

ARRHYTHMIAS

All types of arrhythmias, including supraventricular and ventricular rhythms, sick sinus syndrome, heart block, and atrioventricular junctional and escape rhythms, have been associated with exposure to carbon monoxide and solvents. Arrhythmias, with or without underlying coronary artery disease, are one of the major causes of sudden cardiac death. There are 2 million individuals hospitalized annually in the United States for whom an arrhythmia is a discharge diagnosis. Approximately half of cardiac deaths occur suddenly.

Solvents are used as carriers for other substances that are insoluble in water, as chemical intermediates in manufacturing or as fuels. Commonly used solvents that can cause arrhythmias include benzene, gasoline, fluorocarbons, tetrachlorethylene, trichloroethylene, trichloromethane, and xylene. (See Chapter 11.)

Exposure to solvents can occur at work, as well as among people abusing commercial or household products to induce intoxication to cause euphoria, delusions, sedation, and hallucinations. Arrhthymias from exposure to solvents have occurred in manufacturing, dry cleaning (with tetrachloroethylene exposure), laboratory-based pathology, and pesticide spraying (with exposure to a pesticide mixed in a solvent carrier).

Animal models, used to assess the ability of specific solvents to directly affect myocardial tissue and to increase sensitivity to catecholamines, have shown the following: *(a)* initiation of an arrhythmia is based on the threshold concentration, not the duration of exposure; *(b)* sensitization to catecholamines continues as long as the solvent is present in the blood, even after the solvent is no longer being inhaled; *(c)* halogenated derivatives of aliphatic hydrocarbons, such as trichloroethane, are more toxic than unsubstituted hydrocarbons, such as ethane; *(d)* combined exposure to noise and fluorocarbons has a greater effect than fluorocarbon exposure alone; and *(e)* solvents have a negative inotropic effect on the heart.

Epidemiologic studies of workers exposed to fluorocarbons have not shown increased arrhythmias under "normal working conditions," a finding consistent with the animal studies that a threshold concentration needs to be reached. Since concentrations of solvents in the blood are the immediate precipitating factor, arrhythmias may occur after exposure has ceased, but before blood level concentrations have fallen below the toxic threshold, such as on the way home from work.

In the absence of an arrhythmia, dizziness, headaches, lightheadedness, and nausea may occur as direct effects of exposure to any solvent. In addition, eye or nose irritation or sore throat, possibly with a reddened mucosa or a runny nose, may be found on physical examination. Abnormalities in kidney and/or liver function may occur as a result of exposure to particular solvents, although the use of many of the solvents with organ-specific toxicity, such as benzene, carbon tetrachloride or chloroform, has been markedly reduced. Measurement of solvents in blood, exhaled air, or as a metabolite in urine is possible, but often not easily available. A history of recurrent skipped heart beats, chest discomfort, syncope, or dyspnea that occurs at work, or shortly after work, can be assessed by continuous EKG monitoring—with assessment of location and activity of any arrhythmias that are detected.

Carbon Monoxide

Decreased oxygen delivery to the heart from exposure to carbon monoxide, especially in individuals with compromised coronary arteries, may cause arrhythmias. (See the earlier discussion of carbon monoxide and Chapter 11.)

CARDIOMYOPATHY

The most common cause of cardiomyopathy is atherosclerosis. However, as described next, cardiomyopathy has occurred in two outbreaks among beer drinkers who had arsenic or cobalt inadvertently added to beer, and cardiomyopathy has been associated with work in hot or cold environments.

Arsenic

Arsenic was inadvertently added to beer in England in the early 1900s, causing many cases of cardiomyopathy.[32] However, no cases of work-related cardiomyopathy have ever been reported due to arsenic exposure.

Cobalt

Hundreds of individuals were reported with cardiomyopathy from ingestion of beer, to which cobalt had been added, in the 1960s from Belgium, Nebraska, Minnesota, and Quebec, with mortality rates up to 22%.[33] Only four work-related cardiomyopathy cases have been reported from cobalt exposure.[34] Long-term work-related exposure to cobalt alters left-ventricle relaxation and filling.[35] In the 1950s, cobalt was prescribed at much higher daily doses (150 mg) for anemia without causing cardiomyopathy, as compared to the 8 mg individuals with cardiomyopathy due to beer were daily ingesting. The interaction among alcohol, the protein-poor diet of heavy beer drinkers, and cobalt, which was the presumed mechanism for the toxicity, may explain why very few cases have been reported due to work exposure. Workers involved in the manufacture or machining of tungsten carbide products are likely to be exposed to cobalt.

Hot or Cold Environment

A study in Japan showed that workers in hot or cold environments were at increased risk for idiopathic dilated cardiomyopathy.[36]

Lead

A reversible interstitial myocarditis has been reported among children with lead poisoning from ingesting paint chips, but not among adults with work exposure to lead.[37]

HYPERTENSION

Lead

Low levels of lead exposure have been associated with an increase in blood pressure. On average, for each doubling of the blood lead level (BLL) below 25 μg/dL, there is a 1 to 2 mm Hg rise in systolic blood pressure and a smaller rise in diastolic blood pressure.[38] With the elimination of lead from gasoline and residential paint in the 1970s, the average BLL of the U.S. population fell to below 5 μg/dL. OSHA allows BLLs as high as 60 μg/dL.

The effect of lead on increasing blood pressure in workers is not as dramatic as might be expected, since above 25 μg/dL the effect of lead on blood pressure levels off. Blood pressure correlates better with chronic measurements of lead exposure, such as X-ray fluorescence measurements of lead in tibial bone, than with BLLs. One study estimated there would be 24,000 fewer myocardial infarctions a year in the United States if BLLs were reduced in half.[39] Adult exposure to lead is most common in the manufacture of car batteries, removal of lead paint from metal structures, removal of lead paint from homes built before 1978, manufacture of brass or bronze (which contain 8% to 10% lead), and the use of lead bullets in indoor firing ranges. (See Chapter 11.)

Noise

Noise exposure is common. Sudden acute exposure to noise increases epinephrine levels, vascular restriction, heart rate, and blood

pressure—although with repeated exposure these acute effects are attenuated. Nevertheless, there is much evidence that occupational and community noise exposure increases blood pressure. For each 5-decibel increase in occupational noise exposure, there is a 0.51 mm Hg increase in systolic blood pressure and a 1.14 increased relative risk for hypertension.[40] The noise level of heavy city traffic is 85 dB, which is the 8-hour time-weighted average (TWA) in the workplace at which OSHA requires institution of a hearing conservation program that includes education, hearing tests (audiometry), and hearing protection. For comparison, the noise level for power tools and chain saws range from 95 to 110 dB and a gun shot is in the range of 140 dB. (See Chapter 21.)

The studies finding an association between noise and blood pressure have been of the following two designs: *(a)* individuals have their blood pressure measured using 24-hour ambulatory equipment and changes are analyzed in relationship to noise levels measured simultaneously;[41] and *(b)* individuals exposed to noise are assessed over a period of years, with the occurrence of hypertension or change in blood pressure measured.[42] The effect of noise on causing hypertension in the community setting (not limited to an 8-hour work day) appears to be even greater with a relative risk in one study of 1.26.[40] No controlled studies have been performed assessing the effect on reducing noise exposure in those with elevated blood pressure.

PERIPHERAL VASCULAR DISEASE AND RAYNAUD DISEASE

Arsenic

Peripheral vascular disease prevalence and CVD mortality are higher in people who live in areas of Chile and Taiwan, where levels of arsenic in drinking water from natural rock sediment range from 0.80 to 1.82 ppm. The Environmental Protection Agency (EPA) standard for levels of arsenic in drinking water in the United States is less than 0.01 ppm (10 ppb). Areas of the Midwest and New England have been above this level, but generally at levels below 0.1 ppm—still well below the levels found in Chile or Taiwan. The increased prevalence of peripheral vascular disease was reversed in a study from Taiwan after the water supply was changed and the arsenic level reduced.[43] Arsenic also causes hyperpigmentation, keratosis of the skin, and skin cancer. The pathological changes seen in individuals living in these communities in Chile or Taiwan were either arterosclerosis or thromboangiitis obliterans. In animal studies, arsenic induces inflammation, increases coagulation, and inhibits nitric oxide synthetase. Long-term exposure to arsenic may increase risk factors for atherosclerosis, including diabetes and hypertension. Raynaud disease (peripheral vasospasm) has been seen in vineyard workers exposed to inorganic arsenic pesticides, which are no longer used.

Cadmium and Lead

Cadmium and lead exposure have been reported in one study to increase the risk for peripheral vascular disease.[44]

Carbon Monoxide

Individuals with peripheral vascular disease who are exposed to carbon monoxide have reduced time to the development of claudication in a controlled exercise study.

Cold

Fish filleters who process large quantities of wet and cold fish have increased risk for Raynaud disease (Fig. 23-3).

Vibration

The use of handheld equipment, such as chain saws, jack hammers, chippers, and grinders—especially when used under cold, wet conditions, increases the incidence of Raynaud disease. Prevalence has been as high as 50% to 80% in some groups of workers. Since this type of equipment is often noisy, there is also an association between Raynaud disease and hearing loss.

Once Raynaud disease develops, just the sound of the vibrating equipment may induce vasospasm. Even the use of equipment with one hand may induce vasospasm in the other hand.

Figure 23-3. Supermarket worker with blanching fingertips of his left hand, demonstrating digital vasospasm due to Raynaud disease. His job included entering a large walk-in freezer to obtain food to stock shelves.

The pathophysiology of Raynaud disease amplifies the effect of norepinephrine constrictor receptors on vascular smooth muscle cells. With chronic exposure to vibration, fibrosis develops and blood viscosity increases (see Chapter 12A).

Preventive measures include the following: *(a)* engineering changes to reduce the need for handheld tools; *(b)* the design of handheld tools that vibrate less; and *(c)* changes in work practices, including ensuring proper maintenance of handheld equipment, taking rest breaks, learning to grasp vibrating tools as lightly as possible, and providing warm clothing and gloves in cold weather.

Vinyl Chloride

In the past, in the manufacture of vinyl chloride, workers who cleaned vinyl chloride reaction kettles developed severe peripheral vascular disease in their fingers with acro-osteolysis, a disorder characterized by destruction of bone in the distal phalanges.

COR PULMONALE

Cor pulmonale is enlargement of the right ventricle secondary to lung disease. Destruction and/or vasoconstruction of pulmonary arterioles from hypoxia may occur from exposure to fibrogenic dusts, such as silica, or occupational causes of obstructive lung disease, such as chlorine gas (see Chapter 18).

IMPAIRMENT/DISABILITY

Heart disease is a common cause for disability in the Social Security Disability Insurance (SSDI) program, in which degree of impairment—and not cause—is used to determine eligibility. In contrast, in state-based workers' compensation systems, compensation for heart disease is uncommon for workers other than police and firefighters. (See Chapter 31.) Most states have "heart laws" for police and firefighters. These laws, which generally predated medical studies on job strain or specific workplace exposures such as particulates, were based on the perceived stress and strain of being a police officer or firefighter. The heart laws for public safety officers in individual states vary, but they are unlike workers' compensation laws that usually require physicians to state that conditions are caused by work within a reasonable degree of medical certainty (more likely than not) for a condition to

be considered work-related. These heart laws presume heart disease in police officers and firefighters *are* work-related, or they require employers to prove cases of heart disease are not related to work or do not allow employers to use evidence of previous heart disease to contest a workers' compensation claim. For other workers, chronic stress and/or strain is usually not considered eligible for workers' compensation; instead, whether an acute cardiac event occurred during a time of unusual work stress or strain, in comparison to the worker's stress or strain outside of work, is required to qualify for compensation.

There is a comprehensive guide to determine fitness to return to work after a myocardial infarction or a cardiac procedure.[45] There are specific medical requirements for airplane pilots and truck drivers that must be met at time of entry into these occupations and periodically thereafter. These medical requirements include evaluation for CVD. None of the guidelines or requirements for fitness to work include consideration of specific workplace exposures, such as carbon monoxide, solvents, or hot or cold environments. Since workers with underlying heart disease are at greater risk from these exposures, clinicians should consider these workplace exposure factors.

Clinicians should also consider the use of various medications in patients with heart disease that would increase their risk of an adverse event. This includes diuretics and beta blockers taken by furnace operators as well as construction and landscape workers on hot summer days, and vasodilators taken by workers in the explosive manufacturing industry.

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SECTION IV

RECOGNITION, ASSESSMENT, AND PREVENTION

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24

Epidemiology

Jennifer M. Cavallari, Ellen A. Eisen, David H. Wegman, and Marie S. O'Neill

Epidemiology, a core science of public health, is the study of the distribution and determinants of disease frequency among populations. While clinical investigations focus on the individual, epidemiological investigations examine populations, with the goal of preventing disease by identifying determinants of disease. The field of environmental and occupational epidemiological research focuses on finding patterns and understanding relationships between environmental or occupational exposures and health consequences. Environmental epidemiology is the study of the health consequences of exposures to hazards in the general environment, both outdoor and indoor, through the environmental media of air, water, soil, or food. The related field of occupational epidemiology is the study of the health consequences of hazardous exposures in the workplace.

In general, studies in epidemiology fall into two categories: *(a)* disease-focused investigations seeking to identify risk factors for related diseases or health outcomes, such as in infectious disease or cardiovascular epidemiology; and *(b)* exposure-focused investigations seeking to identify relationships between exposure factors and diseases or health outcomes, such as in environmental and occupational epidemiology. Regardless of the focus of a given study, the epidemiological principles applied to investigate disease–exposure associations are the same.

The goal of epidemiological studies is to identify and investigate hypotheses about the causal relationships between hazards or exposures and diseases or injuries. An epidemiological investigation begins by defining a research question or hypothesis about the causes or determinants of a disease or injury, or the health consequences of an exposure. The generation of hypotheses is often guided by disease clusters, surveillance data, toxicological information from animal- or laboratory-based studies, workplace investigations, and epidemiological studies. For example, a hypothesis was generated by the report to a state health department of eight workers employed at a microwave popcorn manufacturing plant who had bronchiolitis obliterans, a rare and debilitating lung disease.[1] An epidemiological study, which included medical evaluations and occupational hygiene surveys, found excess rates of lung disease and lung function abnormalities among workers at the plant compared to the general population. Analysis of data from these surveys suggested that inhalation of volatile butter-flavoring ingredients may have been the cause, prompting an industry-wide evaluation of this exposure and pulmonary health effects.

The study hypothesis outlines both the health outcome(s) and the exposure(s) of interest.

The health outcome of interest may be an injury or a disease and may range in clinical severity from preclinical, asymptomatic disease, to physician-diagnosed disease, to death. For example, in studying cardiovascular health effects, epidemiologists may choose to examine *death* from all cardiovascular causes (or, more specifically, from only ischemic heart disease) or *hospital admission* for myocardial infarction. They may also choose to study blood pressure or serum cholesterol levels as biomarkers of risk for cardiovascular disease. A variety of data sources may be used to obtain information on health outcomes, such as death certificates, personal medical records, workplace medical or occupational hygiene records, interviews or questionnaires, or direct measurements of organ functions, such as pulmonary function tests.

Exposure assessment, a core component of environmental and occupational epidemiology, characterizes exposures of interest. It identifies appropriate exposures to monitor, characterizes levels of exposure, and considers variations of exposures in time and space that are specific to a given setting.

After generating a hypothesis about the health outcome(s) and exposure(s) of interest, an epidemiologist identifies the study population. Environmental epidemiology studies are based on groups of people in the general population, who may include infants and older people as well as working-age adults in poorer health than those in the workforce; in contrast, occupational epidemiology studies are based on groups of workers who are usually healthier, on average, than the general population.

All epidemiological investigations utilize standard epidemiological measures to describe and quantify disease occurrence and relationships between exposures and health outcomes. Basic studies may aim to quantify a single measure of risk, incidence, or prevalence. More sophisticated studies may compare measures of disease occurrence or quantify the exposure–health outcome relationship. Identification of appropriate epidemiological measures is dependent upon the study design used to investigate the exposure–health outcome relationship. The "gold standard" in study designs for epidemiological investigations is the cohort study. Additional designs include case-control, longitudinal or panel, and case-crossover studies.

While an epidemiological investigation may seem like a simple exercise in forming a hypothesis, identifying a study population, and choosing the right study design, the challenge is in performing these steps in a way that the results of the study are valid, or free from systematic error. Error, which can be introduced in either a random or systematic manner, may affect interpretation of study results. *Random bias* affects the precision (variability or reproducibility) of measurements in a study. *Systematic error or systematic bias*, which should be evaluated both before a study is begun and later during analysis and interpretation of results, can affect accuracy (proximity to true value).

Data from environmental and occupational epidemiological studies are often used for decision making, such as in a regulatory context using risk assessment, to determine whether an exposure causes a particular health outcome, and, if so, at what level? Decisions about causation require a thorough evaluation of epidemiological studies as well as other research, such as toxicological studies. Some diseases have only an environmental or occupational cause, such as childhood lead poisoning (environmental) and silicosis (occupational). Other diseases, such as carbon monoxide poisoning, can have both environmental and occupational causes. Epidemiology can attempt to determine the relative contribution of environmental and occupational causes to this latter group of diseases.

CHARACTERIZING AND QUANTIFYING EXPOSURE

Assigning an accurate and precise exposure is a critical component of epidemiological investigations. For an accurate assessment of exposure, ideally one would have a thorough evaluation of personal exposures, using techniques of occupational and environmental hygiene (Chapter 26). Ideally, one would evaluate each study participant to characterize the type, duration, frequency, and concentration of that person's exposure. However, assigning exposure to an occupational or environmental hazard can be a challenge for several reasons:

1. Exposures are seldom constant in time or space. Over time, as people move from place to place, their exposure patterns change.

2. In occupational settings, new workers are hired and others leave the workforce.
3. Exposures vary over time due to job transfers, changes in technology and production processes, and use of different materials.
4. Amounts of pollution, noise, and green space in a residential neighborhood can change dramatically, even within a short period of time. These changes may have important health implications for those who live there.
5. Personal exposure levels for relevant time periods are seldom available for individuals, because of the high cost to obtain them or because studies are performed long after exposure has occurred. While personal exposures are ideal, epidemiologists often must choose an alternative way of measuring exposure based on available data and resources.

The choice of an appropriate exposure measure is largely dependent upon the disease of interest and the presumed causal path between exposure and disease. Measures of exposure, or the quantity of a substance external to the body, are often characterized by intensity or concentration as well as duration or time scale. In studies of short-term or acute disorders, current exposures or exposures over short time periods are measured. In contrast, in studies of chronic disease or of the impact of cumulative exposure on an acute disorder, long-term exposures are measured. In situations that have remained fairly constant over time, current exposure may be a reasonable surrogate for past exposure. In situations that are more variable, changes in patterns of exposure over time need to be considered to estimate dose (the amount of substance that has entered the body) to an organ or tissue. There are several degrees of refinement for estimating dose. Dose may be quantified on an organ or tissue basis and, depending on the biological media, may represent exposure over different time periods. For example, lead may be measured in various biological media, including blood and bone. Blood lead levels represent recent lead exposure, while bone lead levels represent cumulative lead exposures over longer time periods.

Potential for Exposure

At its simplest, exposure can be captured as a yes/no measure. A commonly available measure of exposure is employment in a specific industry or a specific job, or residence in a particular geographic area. The potential for exposure is a crude surrogate for exposure; however, if the association between exposure and outcome is sufficiently strong, it can be found, despite measurement error. For example, in a study of shipyard workers, lung cancer was found to be associated with previous asbestos exposure even though fewer than half of the shipyard workers had asbestos exposure.[2] Nevertheless, the magnitude of association between a disease and exposure to a specific agent is often greatly diluted by using such surrogate measures of exposure.

Quantity of Exposure

When more information is available, exposures can be quantified for groups or individuals. Measures of exposure should ideally incorporate the intensity, frequency, and duration of exposure most appropriate to the health outcome being studied. Exposure estimates are improved when formal exposure assessment has been performed, based on either judgments of potential exposure or measurements of actual exposure.

Exposure may vary over time due to changes in the environment or changes in a person's activities. In the workplace, differences in work habits or tasks, seasonal changes in ventilation patterns, and use of personal protective equipment may affect personal exposures.

For studying acute health effects, current exposure estimates alone can be used. But for studying chronic effects, one must either assume exposure has been constant over time or one must integrate current and past exposures to estimate cumulative exposure. In the workplace, a complete occupational history includes documentation of time spent in specific jobs together with information on gaps in employment, such as prolonged sick leaves, periods of layoff, and military leaves (Chapter 3). Estimates of cumulative workplace exposure ideally rely on compilation of current and historical industrial hygiene data and interviews of workers about the history of changes, such as in production processes and

exposure controls. In occupational epidemiology, exposure monitoring can provide historic data on exposure levels for individuals or areas of the workplace over time. These representative measurements, combined with work histories, provide measures of exposure intensity and frequency that can be integrated to estimate cumulative individual exposures. For example, in studies of pulmonary function in the Vermont granite industry, there was a need to account for past exposures, but no measurements were available. Historical exposure, however, was estimated by reopening an old granite shed and operating it without modern exhaust ventilation controls.[3]

To compute cumulative exposure to an occupational hazard, estimated exposure levels are weighted by the number of years in successive jobs and summed over all jobs held by each worker. Exposure that occurred years ago is usually assumed to be biologically equivalent to exposure that occurred last year. Weighting schemes that are more complex should be based on specific biologic hypotheses about the relative importance of different exposure patterns. For example, in silicosis, inhaled silica particles continue to be biologically active from the time of initial exposure, and irreversible changes are believed to accumulate gradually over many years. Exposures in the distant past may be weighted more heavily than those in the recent past for diseases such as silicosis, in which irreversible changes are believed to accumulate gradually over many years.

Often historic exposure data are limited to cruder measures, such as duration of employment or distance of residence from a toxic waste site. Duration of exposure or distance from a source of exposure—rather than integrated measures of duration and intensity—are frequently used as exposure surrogates. Duration of employment can often be documented from payroll or union seniority records.

In contrast, with workplace-based studies, cumulative exposure is rarely estimated in community-based studies because records on environmental exposures with details of concentrations over time and space are rarely available. Often surrogates of cumulative exposure are chosen. For example, in epidemiologic studies of air pollution, chronic health outcomes have been assessed in relation to exposure estimates based on geographic (spatial) variation. The increased lung cancer mortality associated with particles with an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$) is an accurate estimate of relative risk only if residence data are available to enable one to estimate long-term exposure.[4] Assigning long-term exposure to an environmental pollutant, such as particles in the ambient air or disinfection by-products in drinking water, requires knowledge of residential history and/or consumption habits over a long period of time. This information can be obtained from surveys, questionnaires, or public records of vital statistics, water treatment practices, or environmental monitoring.

Quantified continuous exposures—rather than categorical or nonordinal estimates of actual or potential exposures—are advantageous in epidemiological studies because they enable one to estimate exposure–response relationships, which may provide information on the level or quantity of exposure that produces a particular health outcome.

Biological Monitoring

An alternative method of evaluating exposure is biological monitoring to estimate dose—the amount of a substance that has entered the body. Evaluation of individuals with biomarkers—toxic agents or their metabolites in blood, urine, hair, nails, or exhaled air—sometimes improves estimation of dose. Biological monitoring is better than environmental monitoring because it accounts for exposures from multiple routes of absorption: inhalation, skin absorption, and ingestion. For example, the blood lead level has been used in epidemiologic studies of workers and children because it integrates recent absorption from both inhalation and ingestion. Another advantage of biomarkers is that they may reflect exposure over specific time intervals. For example, in studies of the health effects of chronic lead exposure, X-ray fluorescence (XRF) of bone provides a more relevant biomarker of exposure than blood lead level because it estimates the accumulated body burden of lead, reflecting long-term exposure. Although there are no biological monitoring tests for many hazardous substances, biological monitoring is receiving increasing attention. New and improved measures of the

burden of toxic agents on the body will be developed.

MEASURES OF DISEASE OCCURRENCE

Incidence and *prevalence*, two epidemiological measures used to quantify disease occurrence, are standard variables used to measure disease frequencies.

Incidence Proportion and Rate

Incidence measures the occurrence of new cases of disease over a specified time period. The term *risk* is used to describe an individual's probability of developing a disease. On a population level, the average risk in a group for a specific disease is referred to as the *incidence proportion*, or *cumulative incidence*. It is based on the number of new cases occurring during a specified period of time:

$$\text{Incidence proportion} = \frac{\text{Number of new cases}}{\left(\begin{array}{l}\text{Total population at risk}\\ \text{during the specified}\\ \text{time period}\end{array}\right)}$$

For example, an investigation of 1,157 workers in a coated fabrics plant identified 68 cases of peripheral neuropathy over a 1-year period.[5] Only 50 affected workers had onset of this disease during the previous year; 18 of the 68 prevalent cases had onset more than 1 year before. Therefore, the population at risk for development of a new case within the past year was: 1,157 – 18 = 1,139. The 18 workers who already had disease at the beginning of the study period are excluded from both the numerator and denominator, as incidence only refers to the occurrence of new cases. Because the number of new cases in that period was 50, the plant-wide annual incidence proportion was as follows:

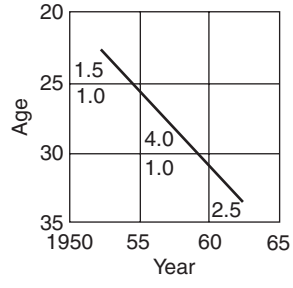
$$\frac{50}{1{,}139} = 4.4 \text{ per } 100 \text{ workers}$$

The *incidence rate* uses the same numerator as the incidence proportion, but a different denominator. The denominator incorporates the concept of person-time, usually expressed in units of person-years. This denominator takes into account not only the number of at-risk persons, but also the length of time during which they were at risk for development of the specific disease. An example of how to calculate the contribution of a single worker to a person-years denominator is illustrated in Figure 24-1. The incidence rate can be thought of as the "speed" at which new cases arise in a population.

$$\text{Incidence rate} = \frac{\text{Number of new cases}}{\text{Sum of person-time at risk}}$$

Prevalence

While the incidence rate assesses the frequency of disease onset, *prevalence* is a measure of disease status. The simplest quantity, known as *point prevalence*, is the ratio between the number



Time period	Age group	Person-years
1950–1954	20–24	1.5
	25–29	1.0
1955–1959	25–29	4.0
	30–34	1.0
1960–1964	30–34	2.5
		10.0

Figure 24-1. Person-years experienced by a worker entering a follow-up program at age 23 years 6 months in mid-1952 and leaving in mid-1962. (*Source*: Adapted from Monson RR. Occupational epidemiology (2nd ed.). Boca Raton, FL: CRC Press, 1989, p. 79.)

of cases present and the size of the population at risk at a single point in time:

$$\text{Point prevalence} = \frac{\text{Number of cases}}{\text{Population at risk}}$$

Unlike incidence, which is based on new cases during a given time period, point prevalence is based on the number of cases at one point in time. To interpret the public health significance of the situation previously described of 68 peripheral neuropathy cases in a coated fabrics plant,[5] one needs a denominator. The total plant population was 1,157. Therefore, the point prevalence was as follows:

$$\frac{68}{1{,}157} = 5.9\%$$

To determine whether this prevalence was excessive, the prevalence in the plant had to be compared with the prevalence in the general population or some other appropriate comparison group. Two limitations of point prevalence are that *(a)* it does not distinguish between old and new cases, and *(b)* it does not take into account the amount of time a person has had a disease. Diseases of short duration may have a low prevalence, even if their incidence rate is high; in contrast, diseases of long duration, even if their incidence rate is low, may have a high prevalence. Prevalence is a reflection of both incidence rate and disease duration. It is best suited for measuring disease burden, and it is poorly suited for studying the causes of disease.

MEASURES OF EFFECT AND ASSOCIATION

The goal of environmental and occupational epidemiology is most often to estimate the effect of the exposure of interest by comparing the occurrence of health effects in an exposed group and a nonexposed group. Quantitative estimates for comparisons include rate ratio and risk ratio, and attributable risk (the difference between rates).

Rate Ratio and Risk Ratio

The *rate ratio* is designed to communicate the relative importance of an exposure by comparing the rate in an exposed population with that in an otherwise comparable nonexposed population. Often the term *relative risk* is broadly used to refer to an incidence rate ratio. This term can also be applied to a risk ratio (a ratio of incidence proportions). Therefore, the terms *rate ratio* or *risk ratio* are preferred to *relative risk*. In its simplest form, a rate ratio is the ratio of two rates while a risk ratio is the ratio of two prevalences (Table 24-1). In the case of the fabrics plant, the suspect neurotoxin was in the print department, so it was possible to create a within-plant comparison. Of the 1,139 disease-free workers in the plant, 169 worked in the print department and 34 of these workers had onset of peripheral neuropathy during the past year, resulting in the following incidence proportion:

$$\frac{34}{169} = 20.1 \text{ per } 100 \text{ workers}$$

Among the remaining 970 workers, there were 16 new cases, resulting in the following incidence proportion during the 1-year period:

$$\frac{16}{970} = 1.6 \text{ per } 100 \text{ workers}$$

Therefore, the incidence risk ratio was as follows:

$$\frac{20.1}{1.6} = 12.6$$

When examining different diseases or the effects of different hazards, relative risks can be compared directly. For example, the relative risk

Table 24-1. Derivation of Risk Ratio and Risk Difference*

	EXPOSURE		
Disease	Present	Absent	Total
Present	a	c	a + c
Absent	b	d	b + d
Total	a + b	c + d	a + b + c + d

**Calculations*:
Exposed disease prevalence = a/(a + b)
Nonexposed disease prevalence = c/(c + d)
Risk ratio (prevalence ratio) = a/(a + b) ÷ c/(c + d)
Risk difference (prevalence difference) = a/(a + b) – c/(c + d)

of lung cancer in heavy smokers compared with nonsmokers is very large (32.4), whereas that for cardiovascular disease is small (1.4). This finding suggests that smoking is more potent as a lung carcinogen than as a cardiotoxic agent.

Rate and Risk Difference

Whereas the risk and rate ratios are measures of the potency of the hazard, the incidence rate and risk differences measure the magnitude of the disease burden in the population that is ascribed to the exposure under study. This concept is particularly useful in studies of an occupational or environmental health hazard when an exposure is generally one of several possible causes of a specific disease. The incidence risk difference (or prevalence difference) is calculated by subtracting the incidence (or prevalence) of the disease in the non-exposed population from that in the exposed population (Table 24-1). Likewise, the incidence rate difference is calculated by subtracting the incidence rate of the disease in the non-exposed population from that in the exposed population. In the coated fabrics plant, the incidence proportion per 100 workers in the unexposed population (1.6) is subtracted from the proportion in the exposed population (20.1) to yield an incidence risk difference of 18.5 per 100 workers—the quantity of disease attributed to exposure.

The concept of risk difference is illustrated by the impact of cigarette smoking on health. Table 24-2 shows that the smoking-attributable risk for lung cancer (2.20 per 1,000) is smaller than the smoking-attributable risk for cardiovascular disease (2.61 per 1,000). The risk difference takes account of both the potency of the disease-causing factor and the magnitude of the disease in the population. Despite the lower relative risk of cardiovascular disease due to smoking, the larger attributable risk indicates that, in a population, reduction of smoking leads to a greater reduction in the occurrence of cardiovascular disease than lung cancer.

Interpreting Rates

Crude Rates

When rates are calculated without consideration of factors such as age or calendar year, they are referred to as *crude rates*. Crude rates can be misleading. For example, if the exposed group includes a high proportion of older people and disease incidence increases with age, then observed differences in crude rates may only reflect differences in age.

Specific Rates

Specific rates are calculated for homogeneous subgroups of a population that are defined by age, gender, or other specific factors. Sometimes an elevated rate of disease exists only in one subgroup, such as people aged 35 to 44 years.

Adjusted Rates

Although specific rates can sometimes provide valuable information, it is cumbersome to compare many specific rates. Methods have been

Table 24-2. Relative and Attributable Risk of Death among British Male Physicians from Selected Causes Associated with Heavy Cigarette Smoking

Cause of Death	DEATH RATE*		Relative Risk	Attributable Risk
	Nonsmokers	Heavy Smokers†		
Lung cancer	0.07	2.27	32.4	2.20
Other cancers	1.91	2.59	1.4	0.68
Chronic bronchitis	0.05	1.06	21.2	1.01
Cardiovascular disease	7.32	9.93	1.4	2.61
All causes	12.06	19.67	1.6	7.61

* Number of deaths per 1,000 per year.
† Smokers of 25 or more cigarettes per day.
Source: Adapted from: Doll R, Hill AB. Mortality in relation to smoking: ten years' observations of British doctors. British Medical Journal 1964; 1: 1399–1410.

Table 24-3. Age Effect on Incidence of Myocardial Infarction in a Hypothetical Population*

	WORKERS <45 YEARS			WORKERS ≥45 YEARS			ALL WORKERS			
Location	Cases	Population at Risk	Age-Specific Incidence Rate	Cases	Population at Risk	Age-Specific Incidence Rate	Cases	Population at Risk	Crude Incidence Rate	Age-Adjusted Incidence Rate†
Factory 1	4	400	10.0	18	600	30.0	22	1,000	22.0	18.0
Factory 2	10	800	12.5	10	200	50.0	20	1,000	20.0	27.5

* The incidence rate is expressed as new myocardial infarctions occurring in a 10-year period of observation per 1,000 population.

† Based on age distribution summed for Factory 1 and Factory 2.

developed for estimating a single summary rate that takes account of differences in the distribution of population characteristics, such as age. Such rates are known as *adjusted rates* or *standardized rates*. Two types of adjustment are commonly used: *direct adjustment*, in which rates in the study population are weighted by person-time in a reference population, and *indirect adjustment*, in which rates in a reference population are weighted by person-time in the study population. These methods are illustrated with examples of adjustment for age in Table 24-3. For a further description of these types of adjustment, see the Appendix at the end of this chapter.

EPIDEMIOLOGIC STUDY DESIGNS

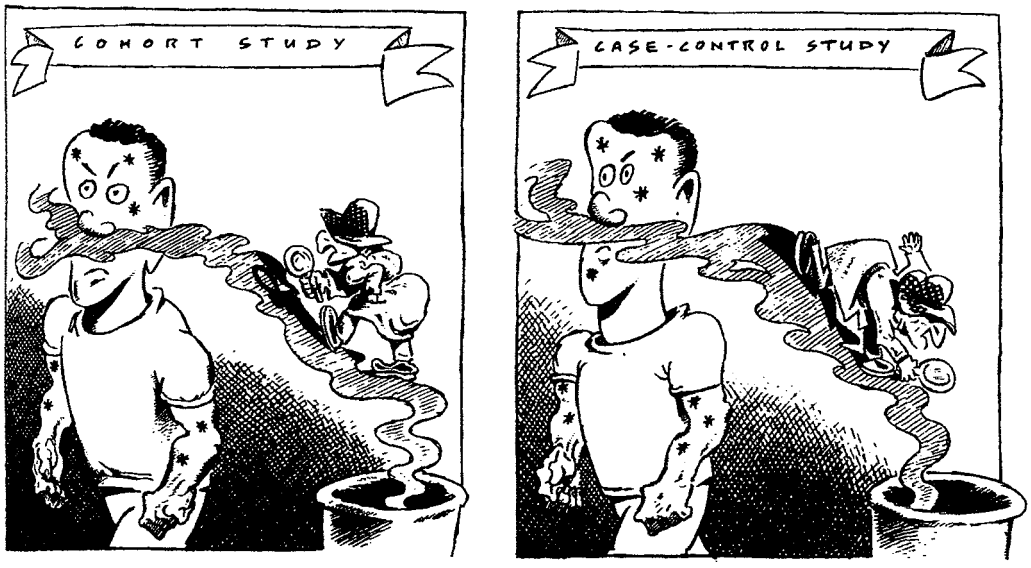
Epidemiologic studies are conducted to measure disease occurrence and to identify associations between exposures and health outcomes. There are several different designs, each with strengths and weaknesses. Cross-sectional, cohort, and case-control designs are those that are most often used. There are additional designs that are unique to the questions posed by occupational and environmental health. Choice of the appropriate study design depends upon several factors, including the research question, data and resources available, and the feasibility of the study.

Cross-Sectional Studies

A cross-sectional study examines the association between exposure and the prevalence of a health outcome at one single point, or short period, in time. The study population includes all subjects in the population of interest who are present at the time of data collection. Study populations may be derived from population surveys, such as the National Health and Nutrition Examination Survey (NHANES); population lists, such as from a driver's license registry or other public records; and lists of labor union members or workers employed in a given industry. Analyses are performed either by comparing prevalence of disease between subgroups defined by exposure status, or by comparing exposure between subgroups defined by disease status. Exposure can be classified *(a)* dichotomously, such as exposed versus nonexposed; *(b)* categorically, such as low, medium, and high; or *(c)* as a continuous measurement. Exposure classification can be based on current or lifetime exposure. The following is an example of a *cross-sectional* study:

The association between environmental lead exposure and blood pressure was investigated in a cross-sectional study based on data from NHANES II, a general health survey based on a representative sample of the U.S. population. The study population was restricted to the white males age 40 through 59. Considering both blood pressure and lead exposure as continuous variables, researchers found that subjects with higher blood lead levels had higher levels of both systolic and diastolic blood pressure. These associations were statistically significant in linear regression models,* after adjusting for known risk factors for high blood pressure, including age, body mass index, nutritional factors, and blood chemistry.[6]

* Linear regression is a statistical approach used to estimate the relationship between dependent variable y and predictor variables x.



(Drawing by Nick Thorkelson.)

Cross-sectional studies are frequently done when investigating risks in the workplace and the general environment. Compared to studies based on other designs, cross-sectional studies generally cost less and take less time to perform. By questionnaire and by direct measurement, they can simultaneously collect information on demographic parameters; exposures; personal habits, such as smoking and diet; past medical history; and current health status.

Cross-sectional studies have several important limitations. They are less appropriate for investigating causal relations because they are based on prevalent, rather than incident, cases of disease. Prevalence is a poor proxy for incidence, especially for diseases of short duration. A related limitation in working populations is that cross-sectional studies oversample workers with long duration of employment and undersample workers with short duration of employment. In addition, it may be difficult to determine whether exposure preceded disease since information on both is obtained at the same time. Occupational cross-sectional investigations based on current workers present at the time of study do not include workers who have left work and those who are absent from work, some of whom may have the diseases being studied. Absence of these workers may result in an underestimate of the association of interest due to the "healthy worker effect" (see section on "Healthy Worker Biases").

Cohort Studies

In a cohort study design, the study population is identified as those who are at risk for developing the disease of interest. These individuals are divided into exposure groups and monitored over time to measure the occurrence of adverse health outcomes. The incidence of adverse health outcomes is observed in the study group and compared with that in a nonexposed, reference group. Cohort studies are either retrospective, in which the cohort is defined at some point in the past and monitored to the present, or prospective, in which the cohort is defined at present and monitored into the future.

Cohort Mortality Studies

Although the cohort design can be used to examine nonfatal outcomes, most occupational cohort studies examine mortality due to specific causes. The most common type of cohort study is the standardized mortality study, in which cause-specific mortality rates of the exposed cohort are compared with those of the general population, which is assumed to be nonexposed. Each comparison results in an approximation of relative

risk, known as the *standardized mortality ratio (SMR)*. If the number of deaths observed in the exposed cohort is equal to the number expected based on death rates in the standard population, the SMR equals 1.0, indicating neither an excess nor a deficit of risk. If the SMR is greater than 1.0, the data suggest an increased risk in the exposed population.

To conduct an SMR study, the following information must be obtained for each member of the cohort: date of birth, date of entry into the cohort, date of leaving the cohort, vital status (alive or dead), and cause of death for those who have died. With these data, one can determine person-years at risk, which take into consideration times when workers entered or left during the study period. This permits a calculation of person-years at risk, adjusting for length of time since entry into the study and for age. An SMR requires personnel records with accurate employment data; if such data on the total population at risk are lacking, the mortality experience can be evaluated by a proportional mortality analysis. The following is an example of a *retrospective cohort mortality* study:

A cohort of autoworkers was studied to examine the relation between exposure to metalworking fluids and specific causes of death.[7] All workers who had ever been employed in one of three Midwestern automobile manufacturing plants for at least 3 years prior to 1984 were included. Subjects were followed for vital status from 1941, the year Social Security records became available, through 1994. By the end of the follow-up period, almost 25% of the 46,400 subjects had died. When the observed number of deaths due to all causes combined was compared to the expected number, there was no obvious excess among white males (SMR = 1.0) and a slight deficit among African American males (SMR = 0.9). For all cancers combined, the SMRs were 1.05 for white males and 0.95 for African American males. When observed deaths due to specific cancers were compared to expected, slight excesses of 1.2 to 1.4 were found for leukemia and cancers of the esophagus, larynx, stomach, and liver among white males and for several of these same cancers, as well as pancreatic cancer, among African American males (Table 24-4). When lifetime exposure to metalworking fluids was evaluated, increasing risk for cancers of the esophagus, larynx, liver, skin, brain, and prostate was associated with increasing exposure.

Cohort Morbidity Studies

Increasingly, the cohort design is being used to study risks associated with nonfatal health outcomes. Retrospective studies can be conducted if information on *past* health status is available, such as in existing medical records or new health surveys. More often, morbidity studies require prospective study designs so that the health

Table 24-4. Standardized Mortality Ratios (SMRs) for Selected Cancers among White and African American Male Autoworkers

	WHITE MALES		AFRICAN AMERICAN MALES	
Cause of Death	Observed	SMR (95% CI)	Observed	SMR (95% CI)
All causes	13105	1.01 (0.99–1.03)	1882	0.86 (0.82–0.90)
All cancers	2,983	1.05 (1.01–1.09)	460	0.95 (0.86–1.04)
Esophageal	83	1.22 (0.97–1.51)	21	0.76 ((0.47–1.16)
Stomach	151	1.16 (0.98–1.36)	28	0.96 (0.63–1.38)
Pancreas	143	0.99 (0.83–1.16)	36	1.50 (1.05–2.07)
Liver	78	1.42 (1.12–1.77)	16	1.31 (0.75–2.13)
Larynx	44	1.16 (0.85–1.56)	11	1.26 (0.63–2.25)
Lung	1,002	1.08 (1.02–1.15)	153	0.95 (0.80–1.11)
Prostate	261	1.06 (0.94–1.20)	55	0.98 (0.74–1.28)
Leukemia	147	1.34 (1.14–1.58)	15	1.28 (0.71–2.10)

Source: Adapted from: Eisen EA, Bardin J, Gore R, et al. Exposure-response models based on extended follow-up of a cohort mortality study in the automobile industry. Scandinavian Journal of Work, Environment & Health 2001; 27: 240–249.

information can be collected directly by performing medical examinations, physiologic tests, or surveys of *current* health status. Longitudinal studies examine change in health status over time.

The following is an example of a *retrospective cohort morbidity* study:

A cohort of approximately 1,000 hospital nurses was studied to examine possible reproductive effects associated with use of sterilizing agents.[8] Questionnaires and medical records were used to collect information retrospectively about both exposure and pregnancy history as far back as 30 years. The frequency of spontaneous abortion among nurses currently using the sterilizing agents was only slightly higher than that for currently nonexposed nurses. A more striking difference was observed when results were stratified according to whether exposure to sterilizing agents had occurred during a previous pregnancy. Among those exposed, the rate of spontaneous abortion was 16%, compared with 6% among the nonexposed. Of the three specific sterilizing agents considered, ethylene oxide showed the strongest association with spontaneous abortion.

The following is an example of a *prospective cohort morbidity* study:

A cohort of 1,022 infants born in the Faroe Islands, an island group situated between the Norwegian Sea and the North Atlantic Ocean, was followed forward in time to investigate the possible neurobehavioral effects of prenatal exposure to methylmercury. The source of exposure was dietary and derived mostly from eating whale meat, a custom in this Nordic community. Batteries of neurophysiologic and neuropsychologic tests were administered to school children at about age 7. The exposure variables were biomarkers, mercury concentration in both cord blood and maternal hair. Mercury concentration in hair of subjects at 12 months of age was also measured. Neurophysiologic testing did not reveal any mercury-related abnormalities, but deficits in language, attention, and memory were related to prenatal exposure to mercury.[9]

Cohort studies have several advantages. First, the study population includes all subjects at risk, rather than a cross-sectional sample. Second, because the population is observed over time, the timing of the exposure relative to the outcome is known. Thus, cohort studies can provide the strongest evidence for a causal relationship. The design is efficient for studying relatively common chronic diseases. Several specific causes of death or disease can be studied in the same cohort study. In addition, detailed exposure assessment can be conducted. However, retrospective cohort studies typically rely on outcomes recorded for other purposes, such as disease diagnosis or cause of death, and thus the endpoint is unlikely to be an early marker of disease. Furthermore, there are two other disadvantages of cohort studies: *(a)* they are often expensive and time-consuming because information ideally should be obtained on all members of the cohort; and *(b)* they usually include little information on lifestyle, socioeconomic status, and other determinants of health status.

Case-Control Studies

Case-control, or case-referent, studies aim to achieve the same goals as cohort studies with a more efficient sampling design. In a case-control study, the investigator examines associations between exposures and a health outcome by comparing exposures of people who developed the health outcome (sometimes called cases) with those of controls, a sample of nonaffected people in the same population. Exposure information is obtained only for cases and controls rather than the whole population as in a cohort study. The case-control design is particularly well suited for infrequent and rare diseases, for cohort studies would have to be very large—and prohibitively expensive—to generate enough cases to study. However, compared to cohort studies, case-control studies often provide poor estimates of exposure.

There are three types of case-control studies: *(a)* those that are nested within cohorts, *(b)* those that are population-based, and *(c)* those that are registry-based. In nested case-control studies, all cases of a specific disease are identified within the cohort, and controls represent a sample of the cohort without the disease. In a mortality study, disease status is generally based on death certificates; in a morbidity study, disease status is generally based on medical records.

In a *population-based* case-control study, all cases occurring in residents of a defined geographic area are included, and controls are selected from the same defined population. In a

registry-based case-control study, reported cases of disease that have begun during a defined time period are identified in a registry, and controls are selected from the same registry. Because registries, such as hospital cancer registries, may not be population-based, controls may be selected from patients with other diseases at the same location as the cases, such as at the same hospital.

The effect measure typically calculated in a case-control study is the odds ratio (OR), which is a ratio of the odds of exposure among the cases compared with the odds of exposure among the controls. In Table 24-1, a/c is the odds of exposure among the cases, and b/d is the odds of exposure among the controls. Odds ratios approximate the incidence rate ratios that are determined in cohort studies. Interpretation of them is similar: When OR = 1, there is no excess or deficit of risk.

A case-control study need not include all cases within a defined population. Valid results may still be obtained when the case group includes only a sample of cases. The two main requirements for a case-control study to be valid are that *(a)* the controls be selected from the population from which cases were identified, and *(b)* both cases and controls be selected without prior knowledge of past exposure history. The following is an example of a *population-based case-control* study:

Non-Hodgkin lymphoma has been associated with agricultural pesticide use in men, but little is known about risks in women. To address this lack of knowledge, National Cancer Institute investigators conducted a population-based case-control study in which cases were defined as incident cases of non-Hodgkin lymphoma among women residing in 66 counties in eastern Nebraska, diagnosed between 1983 and 1986 in area hospitals.[10] Controls were selected from female residents in the same counties using random digit dialing of telephone numbers. The study did not find increased risk due to living or working on a farm. Small risks were observed for women who personally handled insecticides (OR = 1.3) or chlorinated hydrocarbons (OR = 1.7). And women who personally handled organophosphate insecticides had a 4.5-fold increased risk (Table 24-5). Because non-Hodgkin lymphoma is a relatively infrequent disease with a long latency, a case-control study was more feasible than a cohort study. Because exposures occur on farms, each of which employs a small number of workers, a community-based case-control study was more practical than a workplace-based study.

The following is an example of a *nested case-control* study:

The risk of cancer due to exposure to pulsed electromagnetic fields was examined in a series of case-control studies, each focused on a different malignancy, nested in a cohort of electric utility workers.[11] Cases of a particular cancer were diagnosed at any time after entry into the cohort (beginning of employment as an electric utility worker) until the end of follow-up in 1988. Controls were chosen at random from sets of cohort members matched, by age and gender, to each cancer patient. Cumulative exposures were estimated up to the date of cancer diagnosis. Smoking information was obtained from company medical records. The study found no association between exposure to pulsed electromagnetic fields and cancers previously suspected

Table 24-5. Non-Hodgkin Lymphoma, According to Insecticide Use among Women in Eastern Nebraska

	USED ON FARMS			PERSONALLY HANDLED		
Insecticide Class	Cases	OR	95% CI	Cases	OR	95% CI
Any insecticide	56	0.8	0.5–1.3	22	1.3	0.7–2.3
Chlorinated hydrocarbons	20	1.6	0.8–3.1	5	1.7	0.5–5.8
Organophosphates	14	1.2	0.6–2.5	6	4.5	1.1–17.9
Metals	3	1.6	0.3–7.5	0	—	—

OR = odds ratio; 95% CI = 95% confidence interval.

Source: Adapted from: Zahm SH, Weisenburger DD, Saal RC, et al. The role of agricultural pesticide use in the development of non-Hodgkin's lymphoma in women. Archives of Environmental Health 1993; 48: 353–358.

of being associated with magnetic fields. However, the study found an association between cumulative exposure to pulsed electromagnetic fields and lung cancer (after adjusting for cigarette smoking history), with an OR of 3.1 in the highest exposure category.

A case-crossover study, a variation of the case-control study, is used to investigate transient risk factors that may trigger acute events. Each case serves as his or her own control by comparing exposures near the health event to exposures at a time when it did not occur, either before or after. A case-crossover study is designed to investigate whether exposures immediately preceding the health event are different from those that typically occur.[12] The following is an example of a *case-crossover* study:

The association between exposure to vehicular traffic in urban areas and the triggering of a myocardial infarction (MI) was evaluated in a case-crossover study in southern Germany.[13] A total of 691 cases were identified from individuals listed in a regional MI registry for whom *(a)* dates and times of MIs were known, *(b)* survival had been at least 24 hours after the MI, *(c)* the registry's standardized interview had been completed, and *(d)* information had been provided on factors that may have triggered the MI. Patient diaries were used to collect exposure data on activities during the 4 days preceding onset of symptoms. For each individual, the 4 days of data were divided into two periods of traffic exposure: a "case period" for the 6 hours prior to the onset of the MI, and a control period for the 24 to 71 hours prior to the onset of the MI. The study found an association between exposure to traffic and the onset of an MI within 1 hour following exposure (OR, 2.92; 95% CI, 2.22 to 3.83; $p < 0.001$). The MI patient's use of a car was the most common source of exposure to traffic. However, the study also found an association between time spent on public transportation and the onset of an MI 1 hour later.

The principal advantages of a case-control study are its relative simplicity and relatively low cost of studying diseases that do not occur frequently. Case-control studies are useful when multiple exposures are being explored as causes of a specific disease, such as bladder cancer. In contrast, cohort studies are useful when multiple diseases are being explored as being caused by a specific exposure, such as inorganic lead.

Case-control studies are slightly more susceptible to biases than cohort studies are (see section on "Precision and Validity"). For example, if exposure information depends on self-reports, it may be recalled and reported differently by subjects with and without disease. In addition, the need to identify a control group from the same population that generated the cases often presents a challenge.

Ecologic Studies

In an ecologic study, the group, rather than the individual, is the unit of analysis. Ecologic studies require that information is available for both exposure and disease for an entire group, such as a class of students, or the entire population of a community, a state, or a nation. Disease rates and average exposures are compared either between different groups in different places at the same point in time, or within the same group at different points in time. For example, a study in Spain used census-derived data on cancer mortality and information on water source, by municipality, to assess associations between chlorination of drinking water and both stomach and bladder cancer.[14]

Use of ecologic variables is common in environmental epidemiology because high-quality data often exist for a region, but not for individuals in the region. For example, concentrations of air pollutants measured at the city or county level (ecologic measures) may be assigned to all individuals living in that city or county and may be associated with health outcomes in individuals, as was done in a study of daily ozone levels and daily mortality in Mexico City.[15] In a recent validation study, it was found that outdoor particle measurements correlated reasonably well with personal particle exposure over time, supporting their use in longitudinal studies of air pollution and health.[16]

Studies using ecologic variables for both exposure and outcome may be more prone to the *ecologic fallacy*—the inappropriate inference of a causal effect at the individual level from data that are aggregated at the group level. For example, suicide rates were found to be higher in predominantly Catholic countries than in predominantly Protestant countries, suggesting that Catholics had higher suicide rates.[17] While possibly true,

this finding could also be explained by Protestants in predominantly Catholic countries having higher suicide rates. In addition, some other variable unrelated to religion, such as unemployment, might be operating as a causal factor.

Ecologic data are properly interpreted as representing a contextual effect, such as due to living in neighborhoods with little green space or a low literacy rate. Although one needs to interpret ecologic studies with caution, ecologic studies can be used appropriately, such as in *(a)* developing hypotheses for further study, or *(b)* evaluating the effectiveness of an intervention by comparing disease rates in a target population before and after an intervention.

Special Study Designs

Panel Studies

A panel study is similar to a cohort study in which a small group of individuals are followed prospectively over a short time period. Its goal is to investigate short-term health effects of environmental exposures that vary within a brief period. Repeated measures of exposure and health outcomes are obtained during the study period. In environmental epidemiology, panel studies have effectively examined acute health effects of air pollution. For example, a panel of 88 elderly men and women were recruited and, over a 24-hour period, monitored for heart rate variability (HRV).[18] Participants were monitored for 24-hour periods in three different seasons. Data on concentrations of particles in ambient air were available for each of these periods. This panel study found that ambient particulate air pollution was associated with a decline in HRV, indicative of an adverse effect on the heart. In cohorts of workers, panel studies have examined the health effects of workplace exposures over short periods, ranging from a few hours to entire work weeks. In another panel study, that monitored welders over a 24-hour period, an association was found between 4-hour average exposure to particles 2.5 μm or smaller ($PM_{2.5}$) and a decline in HRV.[19] Panel studies can collect much information on each individual studied. However, in studies of long length, participants may leave the study prior to completion.

PRECISION AND VALIDITY

The accuracy of the results from an epidemiological investigation is a reflection of both precision and validity. The precision of an epidemiologic study is determined by the amount of random error (error arising from variability in the data that cannot be readily explained) that may contribute to the study results. Random error often arises due to chance. The precision of a study can be improved by increasing the sample or population size or by modifying the efficiency (reducing random error) of a study.

Careful consideration needs to be given to the validity or lack of bias of a study. The validity of a study is the degree to which the inferences drawn from a study are warranted when one considers the study methods and the characteristics of study participants.

The degree to which the inferences drawn from a study are warranted is determined largely by the absence of bias. Bias, which is systematic error (rather than random error), can be distinguished from precision, which is a random error. Reports of epidemiologic studies should provide sufficient information for readers to understand what potential sources of bias were present and how these biases were addressed. There are three types of bias: selection bias, information bias, and confounding bias.

Selection Bias

Selection bias results from biases in the selection of study participants from the source population. Ideally, study participants represent a random sample of the population of interest. However, if participants are chosen into the study based on both *(a)* their potential for, or level of, exposure, and *(b)* their health outcome status, selection bias may occur. Selection into the population is not a problem for cohort studies if the entire population is included as study participants. But selection bias may occur if there is incomplete participation or if participants have been lost to follow-up, when either situation is related to both exposure and health outcome. Most types of selection bias cannot easily be corrected or controlled in the analysis; they can only be prevented during the design and implementation of the study.

To prevent selection bias in a cohort study, investigators should be kept unaware of ("blinded" from) cohort members' outcome status. Similarly, in case-control studies, investigators should be blinded from the exposure status of cases and controls. In addition, selection of participants should not be influenced by prior knowledge or suspicion of health outcome in a cohort study, or of exposure status in a case-control study. To assess whether they are different than participants who remained in the study, participants who are lost to follow-up or refuse to participate in the study should be evaluated in terms of their potential exposure and health outcome information.

Selection of study participants on either exposure or disease alone may not result in selection bias. For example, an investigation may be limited to workers with only high or low exposure, rather than all workers in an industry or plant. For selection bias to be a potential problem, inclusion in the study must be related to *both* exposure and health outcome.

Information Bias

Information bias, or misclassification, results from errors in the measurement of participants' exposure or health outcome. Unlike selection bias, which arises from subjects not included in the study, information bias arises from participants included in the study. There are two types of misclassification: nondifferential and differential.

Misclassification that is nondifferential is either *(a)* random misassignment of exposure status that occurs regardless of disease status, or *(b)* random misassignment of disease status that occurs regardless of exposure status. Nondifferential misclassification is common in environmental studies, in which there is often little information on subjects' exposures and subjects cannot be well classified into exposure categories. Nondifferential misclassification may occur when the health outcome is broadly defined, obscuring the effect of exposure on occurrence of the etiologically relevant disease. It may also occur when the exposure is broadly defined. For example, a study on the health effects of pesticide exposures in utero may quantify *all* pesticide use, rather than exposure to organophosphate pesticides, which can adversely affect fetal development and growth. This problem is generally worse in retrospective studies because adequate documentation of historical exposures is more difficult. Misclassification generally reduces an estimate of an exposure–disease association or causes an actual health hazard to not be recognized.

Differential misclassification, in which misassignment of exposure is related to disease status or misassignment of disease status is related to exposure, can lead to the perception of a stronger or a weaker association than actually exists. In cohort studies, differential misclassification is commonly prevented by keeping the investigators "blind" to exposure status during collection of outcome information, thereby randomly distributing any errors in collection of information among both exposed and nonexposed groups. In case-control studies, control of differential bias is generally more difficult, because the investigator often knows and the subject always knows disease status when exposure information is being collected. Therefore, in case-control studies, prevention of differential misclassification depends on collecting data as objectively as possible.

Confounding Bias

Confounding, a central issue in performing and interpreting epidemiological studies, occurs when the effect of the exposure of interest is mixed with the effect of another variable, leading to bias. A confounder is both *(a)* predictive of the health outcome in the absence of the exposure, and *(b)* associated with exposure in the population. For example, in a study comparing stomach cancer in coal miners and iron miners, chewing tobacco was considered to be a potential confounder because *(a)* it may be an independent risk factor for stomach cancer, and *(b)* it is used more commonly by coal miners, who are prohibited from smoking in coal mines.

In addition, confounders must not be on the causal pathway between the exposure and disease of interest, or be a part of the pathogenic disease process. For example, a study of coal dust exposures and chronic obstructive lung disease should not control for reduced lung function, which is part of the pathogenic disease process.

Confounding can be controlled either during the design phase of the study or the analysis

of the data. During the design phase, to avoid confounding bias, the population can be restricted to people with or without the potential confounder. For example, a study population may be restricted to include only nonsmokers if smoking is a predictor of the outcome and is also associated with exposure of interest—and would therefore be considered a potential confounder. In case-control studies, matching of study subjects on potential confounders during the design phase can facilitate control of confounding during the analysis. To control confounding in the earlier example of a study of stomach cancer, subjects could be matched on tobacco-chewing habits so that the proportion of tobacco chewers would be the same among cases and controls.

Stratification is the major approach to control of confounding during the analysis phase of cross-sectional, cohort, and case-control studies. A confounder, such as age, is used to define strata, such as 10-year age groups. The exposure–response association is then estimated in each stratum. Stratification, however, becomes problematic as the number of confounders increases, because the strata become too small to allow stable measures of risk. For example, if age, smoking, race, and gender must be controlled for simultaneously, there may be no nonsmoking 40–49-year-old white females in the study population. In this case, stratification becomes an inadequate method of controlling confounding, and mathematical modeling must be used to statistically control confounding.

Healthy Worker Biases

The *healthy worker effect* is used to refer to a bias that arises in occupational epidemiology. This bias results, not from investigator error or oversight, but rather from workers selecting themselves in or out of the study group. Selection bias can occur at two time points, upon hiring into the workforce or upon leaving the workforce. Healthier individuals are more likely to seek and gain employment, which is often called the *healthy hire effect.* Conversely, unhealthy workers are more likely to leave the workplace and healthy workers are more likely to remain in the workplace, creating the *healthy worker survivor effect.* When either selection in or out of the workplace is associated with both health outcome and exposure, selection bias may occur.

As a result of self-selection, employed people are healthier than the general population, which includes older people, people who are chronically ill, and people who are otherwise unfit to obtain and maintain employment. The bias occurs when exposed workers are compared to the unexposed general population. As a result of this bias, studies of illness or death in working populations often find lower rates of chronic diseases than in the general population. In the mortality study of auto workers described previously, the overall SMR for African-American males, expected to be 1.00, was only 0.86. It is rare that an appropriate alternative comparison group, such as another worker population, is available. When possible, healthy worker selection bias is minimized by using a nonexposed comparison group drawn from within the study population.

INTERPRETATION OF EPIDEMIOLOGIC STUDIES

Ultimately, the results from epidemiologic studies can be applied to control or regulate exposures, to identify workers or communities at risk of disease, or to lead to further research (see Box 24-1). Therefore, one must interpret results independently of an epidemiologic study to determine whether it provides evidence that the exposure causes the health effect. The interpretation of epidemiologic studies depends on several factors, including the strength of the association, the validity of the observed association (the extent to which bias is minimized), and supporting evidence for causality as described by Sir Austin Bradford Hill (see discussion later in this section). The strength of an association usually is measured by *(a)* the size of the risk or rate ratios in studies of discrete health outcomes, such as cancer; or *(b)* the magnitude of the difference between groups in studies of physiologic parameters, such as the forced expiratory volume in 1 second (FEV_1). Further evidence of the strength of a causal association is provided by a dose–response relationship, in which the outcome increases with increasing exposure.

When an association appears to be present, its validity must be evaluated by examining for

Box 24-1. Guide for Evaluating Epidemiologic Studies

To assist health professionals in reading, understanding, and critically evaluating epidemiologic studies, the following questions should serve as a useful guide.

Collection of Data

1. What were the objectives of the study? What was the association of interest?
2. What was the primary outcome of interest? Was it accurately measured?
3. What was the primary exposure of interest? Was it accurately measured?
4. What type of study was conducted?
5. What was the study base? Consider the process of subject selection and sample size.
6. Selection bias: Was subject selection based on the outcome or the exposure of interest? Could the selection have differed with respect to other factors of interest? Were these likely to have introduced a substantial bias?
7. Misclassification: Was subject assignment to exposure or disease categories accurate? Were possible misassignments equally likely for all groups? Were these likely to have introduced a substantial bias?
8. Confounding: What provisions, such as study design and subject restrictions, were made to minimize the influence of external factors before analysis of the data?

Analysis of the Data

9. What methods were used to control for confounding bias?
10. What measure of association was reported in the study? Was this appropriate?
11. How was the stability of the measure of association reported in the study?

Interpretation of Data

12. What was the major result of the study?
13. How was the interpretation of this result affected by the previously noted biases?
14. How was the interpretation affected by any nondifferential misclassification?
15. To what larger population may the results of this study be generalized?
16. Did the discussion section adequately address the limitations of the study? Was the final conclusion of the paper a balanced summary of the study findings?

Source: Adapted from: Monson RR. Occupational epidemiology (2nd ed.). Boca Raton, FL: CRC Press, 1989.

selection bias, misclassification, and confounding. Since all studies suffer from some problems with validity, one must judge whether biases could account for the findings of a study.

Results of statistical tests of significance, *p*-values, and/or confidence intervals are usually presented along with estimates of the relative risk. These results contribute to interpretation of studies by providing a measure of stability of the reported associations. Statistical tests guide investigators in deciding whether to reject a null hypothesis.† Rejection is based on a prespecified significance level, defined as the probability that the observed association could have occurred by chance alone (assuming that no effect is expected a priori). For example, a *p*-value of 0.07 indicates that the probability of observing an effect at least as large as the one actually observed is .07, given that no association truly exists. By convention, a significance level of $p < 0.05$ or $p < 0.01$ is most commonly used as the decision rule for rejecting the null hypothesis. A *p*-value can also be interpreted as a continuous measure of the degree of support the data provide for a hypothesis. Confidence intervals provide more information than probability values alone because they provide both *(a)* the range of the magnitude of association consistent with the observed data, and *(b)* a measure of the stability of the estimate of the magnitude of association.

The statistical power of a study to detect a true effect depends on the background prevalence of the disease or exposure, the size of the group studied, the length of follow-up, and the level of statistical significance required. Monitoring of a small cohort for a brief period can yield a falsely negative result. For this reason, when interpreting a negative study, one should examine whether the design precluded a positive finding. For example, a retrospective cohort study of formaldehyde exposure had only 80% power to detect a four-fold risk in nasal cancer mortality—despite having 600,000 person-years of observation—because nasal cancer has a very low background prevalence.[20] Formulas for calculating the

† For example, if the null hypothesis is that no association exists between the exposure and outcome, rejection of the null hypothesis implies that there is an association between exposure and outcome.

statistical power associated with a given sample size are available in standard biostatistics and epidemiology texts.

There has been considerable attention given to the topic of causality. In 1965, Sir Austin Bradford Hill proposed nine "perspectives" for assessing causality in epidemiologic investigations.[21] These perspectives include strength of the association, consistency of findings (within the study and among studies), specificity, temporality, biological gradient (dose–response relationship), coherence, biological plausibility, experimental evidence, and reasoning by analogy. (See Table 25-4 in Chapter 25.)

One epidemiologic study rarely supports causality between exposure and health outcome. Assessment of multiple studies enables one to determine a causal association.

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FURTHER READING

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Well-organized, comprehensive text on epidemiologic approaches specific to environmental studies. Numerous examples guide the reader toward understanding and applying environmental epidemiologic methods.

Beaglehole R, Bonita R, Kjellstrøm T. Basic epidemiology. Geneva: World Health Organization, 1993.
An introductory text on the core ideas underlying general epidemiologic research and useful starting points for more advanced reading. Available worldwide (through the World Health Organization), and a teacher's guide can be obtained for use with the text.

Checkoway H, Pearce NE, Kriebel D. Research methods in occupational epidemiology (2nd ed.). New York: Oxford University Press, 2004.
Very readable, comprehensive text on epidemiologic approaches specific to occupational studies. Numerous examples are provided to guide the reader in understanding both the simple and the complex issues that must be addressed.

Hernberg S. Introduction to occupational epidemiology. Chelsea, MI: Lewis Publishers, 1992.
An excellent introductory text that is well written and illustrated. Aimed at the reader new to occupational epidemiology but somewhat familiar with principles of epidemiology.

Olsen J, Merletti R, Snashall D, Vuylsteek K. Searching for causes of work-related diseases: an introduction to epidemiology at the work site. Oxford, England: Oxford Medical Publications, 1991.
A practical introduction to epidemiology for health professionals with no formal training in the discipline. It is written to assist professionals to better plan and carry out investigation of worksite health problems.

Pagano M, Gauvreau K. Principles of biostatistics. Belmont, CA: Duxbury Press, 1993.
Basic statistics text written in a reasonable fashion with a functional index. Good general reference for statistics.

Rothman KJ. Epidemiology: an introduction. New York: Oxford University Press, 2002.
Introductory epidemiology text for those with little or no background in epidemiology.

Rothman KJ, Greenland S, Lash TL. Modern epidemiology (3rd ed.). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins, 2008.
Probably the best general text on epidemiologic methods designed both for the novice and the expert. Provides principles of epidemiology in substantial detail as well as the quantitative basis for the research methods. Particularly useful as a reference. Good chapters on ecologic studies and on environmental epidemiology.

Steenland K. Case studies in occupational epidemiology. New York: Oxford University Press, 1993.
Provides the reader the opportunity to explore further many of the questions discussed in this chapter through practical and detailed presentation of case studies of various types of epidemiologic studies.

Steenland K, Savitz DA. Topics in environmental epidemiology. New York: Oxford University Press, 1997.
A survey of environmental health issues associated with different environmental media. While examining a number of different problems, the text provides an overview of important methodologic concerns, particularly exposure assessment and statistical methods.

APPENDIX

ADJUSTMENT OF RATES

For purposes of illustration, adjusting for differences in age is examined in detail. Table 24–3 presents a hypothetical problem involving the myocardial infarction experience in two viscose rayon factories. To compare the incidence of myocardial infarction, a summary rate is calculated for each factory. If crude rates were calculated, it would appear that workers in Factory 1 have a slightly greater risk. Comparison of these rates, however, ignores the rather striking difference in age distribution of the populations in the two factories. These can be taken into account by adjusting for age differences by either the direct method or the indirect method.

Direct Adjustment

The principle of direct adjustment is to apply the age-specific rates determined in the study groups

to a set of common age weights, such as a standard age distribution. The selection of the standard is somewhat arbitrary, but often the sum of the specific age groups for the study groups is chosen. In Table 24-3, the standard population consists of 1,200 persons younger than 45 years and 800 persons 45 years or older. The specific rates are applied to this set of weights and then added to create an adjusted rate:

$$\text{Factory 1} = \frac{(.010 \times 1{,}200) + (.030 \times 800)}{2{,}000} = .018$$

$$\text{Factory 2} = \frac{(.0125 \times 1{,}200) + (.050 \times 800)}{2{,}000} = .0275$$

Not only is the magnitude of the rate of myocardial infarction affected by the adjustment procedure, but the rank order is reversed. If another age distribution had been selected as the standard, the standardized rates would change. For example, for 1,500 persons younger than 45 years and 500 age 45 or older, the rate for Factory 1 would become 0.015 and that for factory 2 would become 0.022. Although the absolute magnitudes of the two adjusted rates have no inherent meaning, the relative magnitudes do. While the size of the ratio will change slightly, it will be closely duplicated regardless of the weights. In these two examples of weighting, the ratios of the adjusted rates are 1.53 and 1.47.

Indirect Adjustment

In indirect adjustment, standard rates are applied to the observed weights or the distribution of specific characteristics, such as age, sex, or race, in the study populations. This provides a value for the number of cases (events) that would be expected if the standard rates were operating. The expected number of cases can be compared with the number actually observed for each study group in the form of a ratio. In Table 24–3, assume a national standard rate for myocardial infarction of 1 in 1,000 (0.001) for those younger than 45 years of age and 2 in 1,000 (0.002) for those 45 years or older. The expected number of cases in the two factories would then be as follows:

$$\text{Factory 1} = (.001 \times 400) + (.002 \times 600) = 1.6$$

$$\text{Factory 2} = (.001 \times 800) + (.002 \times 200) = 1.2$$

These expected values are compared with the observed values to calculate a standardized morbidity ratio (SMR), as follows:

$$\text{Factory 1 SMR} = \frac{22}{1.6} = 13.8$$

$$\text{Factory 2 SMR} = \frac{20}{1.2} = 16.7$$

It is tempting to compare the two SMRs and calculate a ratio similar to that calculated for the directly standardized rates. However, a drawback of indirect standardization is that SMRs cannot be compared. Because the age distributions and age-specific rates are significantly different for the two factories, the resulting comparison of the two SMRs would not distinguish differences caused by a different disease incidence rate from differences caused by a different age distribution.

It is reasonable, then, to ask why indirectly standardized rates are used. One reason is that often only one population is being studied, so comparison with the general population experience is convenient and possibly the only reasonable comparison available. Probably of greater importance is the instability of observed rates. In the example presented here, if five rather than two age groups were used and it was also necessary to adjust for both race and sex, then the total number of subdivisions necessary would be $5 \times 2 \times 2 = 20$. With a maximum of 22 cases in either factory, several of the subdivisions would contain no cases and therefore have no reliable rate estimate. Even in the illustration provided, one case more or one case less among the group of younger workers in Factory 1 would have changed the age-specific incidence rate to 12.5 or 7.5, respectively—a very large difference.

25

Toxicology

Robert Laumbach and Michael Gochfeld

Chemical hazards range from very simple molecules, such as carbon tetrachloride, to large complex molecules, such as those found in the toxins of many marine organisms. There are several terms for toxic chemicals: A *toxin* is a poisonous substance of biologic origin, such as animal venoms and many plant chemicals. A *toxicant* is a technical term for a poisonous or hazardous substance, but it is awkward for common usage. *Toxic* is sometimes used as either an adjective, meaning poisonous or hazardous, or as a noun, meaning a chemical with poisonous or hazardous properties. We use the terms *toxic chemical* and *toxic substance* interchangeably. The term *xenobiotic* refers to any chemical substance that is foreign to the body. (See Box 25-1 for definitions.)

Toxic chemicals vary in potency or toxicity, from potent toxins causing profound damage at low doses to less hazardous chemicals that only cause damage at extremely high doses. Chemicals that are highly toxic to one species may be only slightly toxic to another that has mechanisms for detoxifying or eliminating it or is resistant because of a genetic or biochemical factor. Chemicals may cause harm apparent shortly after exposure or harm that may not be apparent until years after exposure.

This chapter covers basic principles of toxicology, including how chemicals enter and move through the body and how they exert pathophysiological effects on target organs. It includes several classifications of chemicals, but it is not a catalogue of toxic effects or a compendium of individual toxic chemicals.

Of the more than 80,000 chemicals, many of which have some use in industry and commerce, few have been adequately tested for toxicity. Even the Occupational Safety and Health Administration (OSHA) hazard communication standard, which requires employers to provide hazard warning container labels and material safety data sheets (MSDSs), is made ineffective if basic information on the chemicals is not available.

All chemicals have properties or characteristics that affect their fate and transport in the environment, the circumstances under which they come in contact with a living organism, the routes of absorption into that organism, and their distribution, metabolism, storage, and excretion after entering the organism. (See Chapter 26 for a description of how chemical exposures are measured in the workplace and ambient environment.)

TOXICOLOGY

Toxicology is the study of the harmful effects of chemicals, including drugs, on living organisms. Toxicologists explore these effects using methods ranging from whole-animal dosing studies to molecular biology.

Box 25-1. Definitions

Aerosol: Either fine liquid droplets or solid particles dispersed in the air. Depending on their size, they may be respirable and may reach the alveoli. The effective aerodynamic diameter is not always the same as the actual droplet or particle size.

Bioavailability: The ability of a substance that enters the body to be liberated from its environmental matrix, especially soil or food, thereby gaining access to enter the bloodstream.

Biotransformation: Intermediary metabolism consisting of metabolic processes that change the structure of a chemical. It may increase (activate) or decrease (detoxify) the harmful properties of a chemical.

Carcinogenicity: The ability of a chemical to cause cancer. Carcinogens can be genotoxic chemicals that damage the nucleic acid, leading to unbridled cell replication (induction), or chemicals that enable induced cells to undergo rapid cell divisions (promotion).

Concentration: The level of a chemical present in an environmental medium or in a body organ or fluid, often expressed on a mass basis, such as micrograms per gram or parts per million, or a volume basis, such as micrograms per liter of water or micrograms per cubic meter of air.

Dose: The amount of a chemical that reaches a target organ or the amount administered (external dose).

Effect dose 50% (ED_{50}): The dose of a chemical that produces a specific effect in 50% of the animals studied.

Exposure: The combination of processes by which chemicals in environmental media (air, water, soil, or food) enter the body (via inhalation, ingestion, or skin absorption) and become distributed to target organs.

Fumes: Very fine solid particles, usually generated when a heated vapor condenses. Many metals form fine fumes. These fine particles readily reach the alveoli.

Lethal dose 50% (LD_{50}): The dose of a chemical that causes death in 50% of the animals studied.

Lipophilic: Pertaining to a chemical (nonpolar) that is much more soluble in organic solvents than in water and can readily move through membranes and concentrate in lipid-rich tissues. (See *Polar and nonpolar compounds.*)

Mechanism: The way in which toxic substances act at the molecular and cellular level to cause morbidity and mortality. Toxic substances include metabolic poisons and cytotoxic poisons that disrupt cell membranes, interfere with chemical reactions, or bind to nucleic acids.

Mutagenicity: The ability of a substance to damage genetic material either by disrupting chromosomal structure or changing the sequence of nucleotides on the nucleic acid molecule.

Pathway: The combination of media and route of exposure, such as ingestion of contaminated soil.

Polar and nonpolar compounds: Polar compounds (such as many inorganic salts) tend to be soluble in water. Nonpolar compounds (many organic compounds) tend to be soluble in organic solvents, such as toluene and lipids, but have very low water solubility. The standard for describing this is the "octanol-to-water partitioning coefficient" (solubility in octanol, divided by solubility in water). Nonpolar compounds pass through the skin and cell membranes more readily than polar compounds. Polar compounds are more readily excreted in urine.

Susceptibility: The vulnerability of an individual or population to be harmed by an agent. It is influenced by many factors, including age, sex, genetic polymorphisms, nutrition, prior exposure, and overall health status.

Teratogenicity: The ability to interfere with normal fetal development, resulting in birth defects.

Threshold: The lowest dose of a chemical that has a detectable effect. For any given chemical, each cellular, biochemical, physiologic, or clinical response may have a threshold. Some effects occur without a known threshold. Since susceptibility varies among animal species and among humans, the threshold is approximated. Thresholds are often used to categorize or rank chemical toxicity.

Toxicity: The intrinsic ability of a substance to harm living cells or processes, organisms, or ecosystems.

Toxicodynamics: The physiologic mechanisms by which toxic substances are absorbed, distributed, metabolized, and excreted and the mechanisms of action on affected target molecules, organelles, and cells.

Toxicokinetics: Quantitative study of time course of absorption, distribution, metabolism, and excretion of a xenobiotic.

Xenobiotic: Any substance foreign to the body, including all synthetic chemicals as well as many pharmaceuticals and essential nutrients.

For a chemical to cause a toxic effect, it must follow a pathway from its source to the site of molecular interaction in the body. Toxic chemicals enter and move through the environment (air, water, soil, and food) at various concentrations until they do one, or likely more, of the following:

- Contact a receptor individual
- Enter the body by inhalation, ingestion, or skin absorption
- Are absorbed into the bloodstream (uptake), reaching a certain concentration
- Undergo metabolism
- Are delivered to target organs and tissues, where they affect some molecular target, causing damage at the genetic, biochemical, cellular, or physiologic level
- Are stored or excreted

Many toxic substances occur naturally, including many metals and their compounds,

while others are anthropogenic or synthetic in origin, created deliberately or inadvertently, such as through human activities. Among the most dangerous toxics are biocides, used deliberately for their toxic effects on certain forms of life. Some biocides are natural in origin, such as the pyrethrins extracted from plants in the daisy family.

The birth of toxicology is often ascribed to Paracelsus (1493–1541), who recognized that a substance that was physiologically ineffective at a very low dose might be toxic at a high dose, and even therapeutic at an intermediate dose.[1] However, around 1500 B.C.E., natural venoms were used for therapeutic purposes. In medieval times, a variety of poisons, many of them of botanical origin, were similarly used. Modern science emerged in the mid-1600s, but it was not until the 1850–1900 period that the chemical industry arose, especially for the development of dyes and paints. In the early twentieth century, modern toxicology emerged, largely due to warfare, pest control, drug development, and food safety.

Toxicology has become a very broad discipline, embracing virtually all aspects of biology and many aspects of chemistry. It has played an important role in the development of pharmaceuticals and pesticides, products in which toxic chemicals are used deliberately in relatively high concentrations. Industrial toxicology focuses on the hazardous properties of raw materials, intermediates, products, and waste products. Occupational exposures to these chemicals are not deliberate, but they may also involve high concentrations. Data generated by toxicologists play important roles in risk assessment and regulation, even for residential and community exposures that are inadvertent and usually occur at relatively low concentrations. Areas within toxicology focus on organs (such as neurotoxicology), functions (such as behavioral toxicology), and organizational levels (such as genetic toxicology). Molecular toxicology is aimed at understanding the most basic level at which xenobiotics interact with organisms, and it also provides biomarkers of exposure and effect.

EXPOSURE

Hazardous materials—including both naturally occurring and anthropogenic (synthetic) chemicals— can be released to the environment, where they become contaminants in environmental media such as air, water, soil, dust, and food. When the human body is exposed to (comes into contact with) chemicals present in an environmental medium, the chemicals can gain access to the body by inhalation, ingestion, dermal absorption, or, rarely, injection. In Table 25-1, an exposure matrix, each cell represents a potential exposure pathway. Those that are highlighted in bold are the main concerns in occupational health. Table 25-2 similarly illustrates pathways that are important in residential or community exposures; in this case, soil ingestion by toddlers often represents the highest intake to a sensitive receptor.

Figure 25-1 illustrates media that are potentially part of an exposure pathway from source to exposure to toxicokinetics in the body, resulting

Table 25-1. Exposure Matrix for Occupational Exposure

	Air	Water	Soil/Dust	Food
Inhalation	Very important for occupational health	Volatiles when cooking or showering	Both workplace and residential	Not a common pathway
Ingestion	Airborne deposition on foods or crops	A major residential pathway	Gardeners and workers who eat at work or without washing	A major residential pathway
Dermal Absorption	A few gases penetrate skin	Important for a few chemicals or mixtures	Some direct contact with workplace chemicals	Not a pathway
Injection	Not a pathway	Not a pathway	Some sharp solid objects can penetrate	Not a pathway

Source: Exposure matrix modified from M. Gochfeld (©). A matrix of routes and media of exposure for risk assessment scenarios. Piscataway, NJ: Environmental and Occupational Health Sciences Institute, 1991.

Table 25-2. Exposure Matrix for Residential or Community Exposure

	Air	Water	Soil/Dust	Food
Inhalation	Important for community air pollution or indoor contaminants	Volatiles when cooking or showering	Fine dust particulates	Not applicable
Ingestion	Airborne deposition on foods or crops	A major residential pathway, particularly with private wells	Toddlers and gardeners	A major residential pathway for garden crops, wildlife, and fish
Dermal Absorption	A few gases penetrate skin	Important for a few chemicals or mixtures. Also for some household chemicals through direct contact	Some direct contact with household chemicals or pesticides	Not a pathway
Injection	Not a pathway	Not a pathway	Some sharp solid objects can penetrate	Not a pathway

Source: Exposure matrix modified from M. Gochfeld (©). A matrix of routes and media of exposure for risk assessment scenarios. Piscataway, NJ: Environmental and Occupational Health Sciences Institute, 1991.

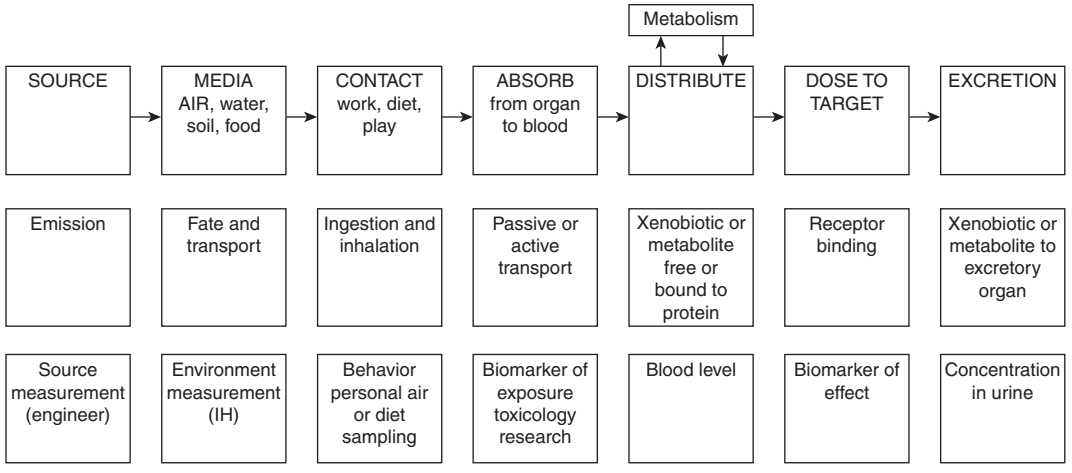

Figure 25-1. An exposure pathway from source through one or more environmental media, to contact, absorption, and distribution in the body, eventually reaching a target organ as well as excretory organs. Top rectangles identify the components of the pathway. Bottom rectangles indicate what can be measured. (Used with permission of the Environmental and Occupational Health Sciences Institute.)

in a dose to the target organ. It is this dose to the target organ that determines the health effect, but many factors intervene, including those governing *(a)* fate and transport in the environment; *(b)* absorption efficiency in the respiratory and gastrointestinal systems and the skin; and *(c)* metabolism, distribution, storage, and excretion. Activity modifies absorption; for example, exercise that increases respiratory rate increases inhalation of contaminants.

Chemicals may exert their toxic effect at the site of contact (skin, eyes, mucous membranes, or lungs), or they may be absorbed into the blood and distributed to target organs, where they cause damage. Some chemicals, such as carbon monoxide, cause damage by affecting blood flow or delivery of oxygen to cells. Some enter cells and interfere with crucial life processes, such as hormone synthesis. Some interfere with cell cycling, disrupting the genetic

activities of the cell and preventing the cell from undergoing normal division. Some kill all cells or only specific ones. Some, which are carcinogens, directly damage the DNA in the cell or interfere with normal regulatory processes of cell division, resulting in cancer.

Assessment of Exposure to Chemicals

Exposure assessment is the discipline that develops and applies measurement techniques and models to quantify human exposure to hazards in the home, community, and workplace. (See Chapter 26.)

The task of measuring the amount or concentration of a substance in an environmental medium, such as by air sampling, is relatively straightforward, requiring appropriate collection and analytic instrumentation. For example, the degree of exposure to an airborne chemical can be determined by sampling the air in an individual's breathing zone near the nose and mouth. However, direct measurement of absorption of the chemical into the body is challenging and usually requires extensive research. Therefore, methods have been developed to infer absorption by measuring biomarkers. For lead, the best biomarker is the blood lead level, which is highly specific and can be influenced only by lead. Similarly, exhaled ethanol, a reflection of recent ethanol intake and current blood alcohol level, forms the basis of the breath-testing of intoxicated motorists.

Many absorbed organic compounds are present only transiently in the bloodstream before being metabolized or excreted. In such cases, measurement of a specific metabolite in urine may be useful for estimating exposure. For example, benzene is rapidly metabolized, so measurement of the blood benzene level is not useful; however, a common metabolite, phenol, can be measured in urine and can be a useful biomarker. One problem, however, is that there are other sources of urinary phenol, including phenol in some cough preparations. Therefore, urinary phenol is a sensitive, but not specific, biomarker of benzene exposure. Serum or urinary cotinine, a breakdown product of nicotine, is a good biomarker of inhalation of cigarette smoke.

ROUTES OF EXPOSURE

Chemicals can enter the body through the skin (dermal absorption), the gastrointestinal tract (ingestion), the respiratory tract (inhalation), and, in some cases, by injection or penetration. Special sites of absorption include the mucous membranes of the eyes, nose, and mouth, and the placenta. However, many chemicals to which humans are exposed do not enter the body.

Crossing Membranes

To enter and move through the body, chemicals must cross various membranes. For example, in the lung a chemical must cross the membrane that separates the air in the alveolus from the blood in the adjacent capillary. Once in the bloodstream, the chemical may enter a metabolic organ, such as the liver; an excretory organ, such as the kidney; or a target organ. In each case, to enter cells, the chemical must again pass through a capillary wall into interstitial space and then through a cell membrane. Cell membranes are not inert "plastic bags," but rather dynamic, fluid structures of lipids and proteins, with different features in different cell types. Some substances pass mainly through membrane pores (as seen under scanning electron microscopes); others are transported by transporter molecules; and some actually remain in the membrane, bound to receptors on the cell surface. Pore transfer often involves passive diffusion along a concentration gradient. Active transport involves processes that use energy to move a substance through a membrane, against a concentration gradient with the aid of transporter molecules. In addition, membranes can be targets for chemicals that cause lipid peroxidation and alter membrane fluidity.

Inhalation

Inhalation is the primary route of exposure in industrial workplaces and in areas with high levels of ambient air pollution. It can also be a serious and occasionally fatal route of exposure in homes, where, for example, carbon monoxide may be a hazard. A typical adult may take about 12 breaths a minute, each with a tidal volume of about 500 ml (of which 150 ml is the "dead space"

of the upper airway). This amounts to 4200 ml (12 × 350 ml), or 4.2 liters per minute (6,000 liters per day) of air per day exchange in the lung—6 cubic meters of air for a resting adult. With activity, both breathing rate and volume increase rapidly, so that 20 cubic meters of air is used to estimate how much airborne contaminant a moderately active person could breathe in a day.

As air passes through the nose, large particles (dusts) are trapped by nasal hairs or may be removed by impaction on the mucous membranes of the nasal passages and pharynx. Smaller particles enter the tracheobronchial tree, where they may land on the ciliated epithelium that lines the walls of larger airways. A thin layer of mucus covers the cilia; particles trapped in this mucus are moved upward by the rhythmic wave-like action of the cilia until they reach the throat, where they are usually swallowed but can also be expectorated. Air contaminants may interfere with this important defense mechanism against inhaled particulates. In vitro experiments show that a puff of tobacco smoke temporarily paralyzes this mucociliary escalator.

The trachea divides into two branches (main stem bronchi) and these divide again and again (about 27 generations) until the alveolar duct ends in the alveolus. Particle size, shape, and density all influence aerodynamic diameter, which determines the efficiency with which particles in air entering the respiratory tract are deposited on various surfaces as they move from the nose and mouth to the distal alveoli. By contrast, pollen grains, which are spherical and about 20 μm in size, are scrubbed out in the nasopharynx and do not enter the lungs.

Particle toxicity may also be related to shape or to intrinsic toxicity. Particles that are deposited in the alveoli are likely to be engulfed by pulmonary macrophages, which secrete enzymes that destroy many kinds of particles. Some particles stimulate the macrophages to secrete cytokines, which invoke a local inflammatory response, followed by formation of a microscopic fibrous area. Recurrent exposure to such substances may lead to progressive fiber deposition, until eventually the elasticity of the lung is compromised by interstitial fibrosis, leading to restrictive lung disease. (See Chapter 18.)

Inhalational exposures to chemical vapors or gases typically exert effects more proximally (in the upper airways) if water soluble, and more distally (in the lower airways or alveoli) if not. Water-soluble chemicals cause irritation of the upper airway, triggering cough as a defense mechanism. Chemicals with low water solubility such as phosgene, do not trigger coughing or reflex bronchoconstriction, and therefore reach the alveoli, where they induce severe inflammation, leading to pulmonary edema—usually delayed 6 to 12 hours after an acute exposure event.

Aerosol Size Definitions

Size of particles, expressed as aerodynamic diameter, generally determines whether they will enter the respiratory tract and, if so, where they will be deposited. The *inhalable fraction* consists mainly of particles with aerodynamic diameters less than 100 μm. The *thoracic fraction* consists of inhaled particles that pass the larynx, mainly smaller than 10 μm. The *respirable fraction*, which may reach the alveoli, consists mainly of particles smaller than 4 μm. The Environmental Protection Agency (EPA) defines particles greater than 10 μm as supercoarse, from 2.5 to 10 μm as coarse, from 0.1 to 2.5 μm as fine, and smaller than 0.1 μm as ultrafine. Engineered particles in the ultrafine range are also referred to as nanoparticles. (See Box 26-2 in Chapter 26.) Particles smaller than 10 μm are designated as the PM_{10} fraction and particles smaller than 2.5 μm as the $PM_{2.5}$ fraction.

Dermal Absorption

The skin is a very effective barrier for many chemicals, but some organic chemicals, such as methylmercury, readily pass through intact skin and enter the bloodstream. The mucous membranes are less effective barriers, and substances can be absorbed in the nasal passages or pharynx. Both the skin and mucous membranes, including those of the eyes, can also be target organs. There are remarkably few data on skin absorption of chemicals. Some nonpolar compounds pass through the skin, while polar compounds do not.

A tragic example of dermal absorption occurred in a chemist, who died after 3 to 5 drops of dimethylmercury were spilled on her latex gloves. Unbeknownst to her—and apparently to the university health and safety officer—dimethylmercury quickly passes through latex

gloves and is readily absorbed through the skin. Just a few drops contained a lethal dose.

Ingestion

Many chemicals enter the body in the food we eat or the water or other liquids we drink. Contamination of drinking water supplies from natural or anthropogenic sources has the potential to affect large populations. Risk assessment assumes a default value of 2 liters per day to represent drinking water intake to a homebound adult. After ingestion, many nonpolar compounds readily pass through the wall of the gastrointestinal tract into the bloodstream. First, they are carried to the liver, where they may undergo metabolic activation or deactivation. From the liver, ingested nutrients and xenobiotics enter the venous circulation and are subsequently distributed throughout the body.

Transplacental Absorption

The placenta is a complex organ, with several cell layers, that provides oxygen and nutrients to the fetus, removes fetal waste products, and maintains pregnancy through hormonal secretion. The placenta maintains active transport for necessary nutrients, such as vitamins, amino acids, calcium, and iron. Xenobiotics pass through the placenta mainly by passive diffusion, unless they are similar in structure to a transported substance. Although it is customary to speak of a placental barrier, there are many infectious agents (especially viruses) and chemicals that readily cross the placenta to reach the fetal circulation. Nonpolar compounds, such as methylmercury and polychlorinated biphenyls (PCBs), readily pass the placenta. Xenobiotics that are bound to proteins or are conjugated are less likely to cross the placenta.

Passage through the Blood–Brain Barrier

The blood–brain barrier, with its low permeability, restricts entrance of many compounds into the brain. It exists because capillary cells in the central nervous system (CNS) form a tight endothelial layer with few pores. There are also tightly wound glial cells that impede passage of chemicals into the brain from the circulation. In addition, the protein concentration in the CNS is lower than in other organ systems, restricting the amount of protein available to bind and transport xenobiotics. The blood–brain barrier is poorly developed at birth, and therefore fetuses and young infants are particularly vulnerable to toxicants that can reach the brain. Methylmercury crosses the blood–brain barrier directly, and inorganic mercury passes through the barrier, by binding to cysteine in the cell membranes.

TIME COURSE OF EXPOSURE AND TOXICITY

In addition to the total dose, the time course of exposure to a chemical can profoundly affect the observed toxic effects. Time courses of exposure can range from a one-time, acute, short-term exposure, lasting seconds to hours, to continuous or intermittent, chronic exposure lasting for most of the life span (Fig. 25-2). Various nonspecific terms (acute, subacute, subchronic, and chronic) are used to characterize the duration of exposure in toxicology studies. *Acute exposure* usually refers to less than 1 day, *subacute exposure* to a few days, *subchronic exposure* to 1 to 3 months for rodent studies, and *chronic exposure* for longer, including lifelong exposure. Subchronic exposure may also refer to approximately 10% of the life span. *Lifetime exposure* is about 2 years for rats and 40 to 70 years for human beings. Despite the use of these terms, there is a continuum in the duration and frequency of exposure, both in laboratory experiments on animals and in human life.

Most occupational exposures are intermittent, occurring only during working hours or during particular activities within those working hours. The tradition of averaging exposures over an 8-hour period (as a time-weighted average, TWA) ignores peaks that may exceed thresholds for acute damage. One of the central dogmas of toxicology, Haber's Law, states that dose is a product of concentration times duration of exposure, thereby equating 1 microgram per day for 100 days with 4 micrograms per day for 25 days or, theoretically at least, 100 micrograms per day for 1 day. Haber's Law operates over a very limited range and can be both useful and

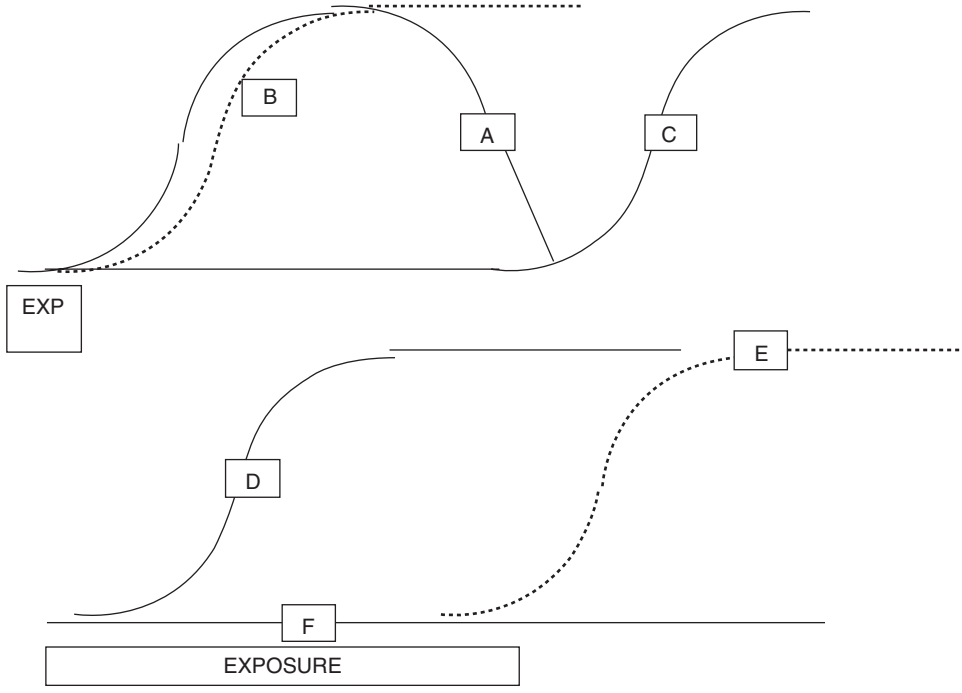

Figure 25-2. Time-course of exposure and response, showing acute versus chronic exposures as well as acute and chronic responses with both short and long latency demonstrated. Curves A, B, and C show responses to a single acute, high-level exposure. (A) An acute self-limited effect; (B) An acute and persistent effect; (C) A long-delayed effect beginning after a long latency following the acute exposure. Curves D, E, and F show response to a chronic, lower-level exposure. (D) A chronic condition arising shortly after onset of exposure, and probably idiosyncratic; (E) A chronic condition beginning after a long period of cumulative exposure; (F) No appreciable response even after long-term, low-level exposure. (Used with permission of the Environmental and Occupational Health Sciences Institute.)

seriously misleading. Consider the analogy of a person who has been prescribed a bottle of 30 pills to take once a day for a month. Taking all the pills on day 1 gets the chore out of the way and according to Haber's Law would be equivalent to one a day for a month, but this could have very different toxic consequences. Taking 10 pills three times a month is not likely to achieve the desired pharmacologic outcome, but 3 pills taken every 3 days might achieve it. Such mixing of frequency and duration is difficult to model, and there are relatively few studies that address this important topic. Therefore, in permissible and recommended occupational exposure limits, the time factor is accounted for by either full-shift, 8-hour TWA standards or 15-minute short-term exposure limits (STELs). The American Conference of Governmental Industrial Hygienists (ACGIH) publishes annual updates of its threshold limit values (TLVs), which are based on an 8-hour TWA, and STELs. The OSHA 8-hour TWA standard for a substance is called the *permissible exposure limit (PEL)*. *Ceiling values* are concentrations that should not be exceeded for any length of time.

The terms *acute* and *chronic* can also refer to the outcome (Fig. 25-2). There is usually a time lag between a dose and its effect, which is referred to as latency. Latency is the period between the start of exposure and the appearance of disease. Latency may change, depending on the techniques used to diagnose the disease. For example, new early diagnostic tests for cancer may result in a shorter apparent latency. Some latencies are only seconds in duration, such as the effects of hydrogen sulfide, which at high concentrations can cause "knockdowns," in which an individual may collapse after as little as one breath. At the other extreme, the latency between start of asbestos exposure and onset of mesothelioma may be

40 years or longer. In general, the latency period for a carcinogen decreases as the cumulative dose of it increases. Long-term chronic exposure may reach a point where the cumulative dose has become sufficient to trigger an adverse health effect. In addition, an acute dose may cause damage that eventually causes an adverse health effect after a long latency. In general, the longer the latency, the more difficult it is to determine that a specific effect was due to a specific exposure.

Reversible, Progressive, and Permanent Effects

A pathophysiological effect caused by a toxic chemical exposure may be reversible, permanent, or progressive. Once the exposure (insult) is removed, many pathophysiological changes have some degree, often complete, of reversibility back to normal structure and function. Reversibility is a function of cumulative dose—that is, a change, such as neurologic or renal damage, may be reversible until it reaches a point where definite structural damage occurs. For example, methylmercury poisoning produces a variety of symptoms and signs, beginning with tingling sensations on the lips and progressing to visual, auditory, and gait impairments, and culminating in blindness, coma, convulsions, and death. The early changes are reversible; but once blindness occurs, complete recovery is not possible. In general, irreversible damage may persist without progressing, or it may progressively worsen as exposure continues. In some cases, damage, such as cancer, may progress even after the exposure has ceased.

Different organ systems have different capacities for repair. Death of a cell is not reversible, but almost all organs are capable of replacing damaged cells by regeneration, which is not always perfect. For example, after viral or toxic hepatitis, the liver regenerates, but the healing often results in cirrhosis, because new liver cells have interposed fibrous tissue that compresses cells and interferes with function. Similarly, after lung injury, the healing process involves formation of fibrous tissue that eventually impairs respiratory function.

When DNA is damaged, there are repair enzymes capable of restoring the genetic material, although not always in the same order as the original. DNA repair mechanisms become less efficient in the elderly, and this is believed to be one of the factors associated with cancer rates increasing with age.

TOXICOKINETICS

Figure 25-3 summarizes the movement of substances from environmental media into and out of various body compartments. *Toxicokinetics* (and *pharmacokinetics*, a related term) is the study of how much and how quickly a xenobiotic undergoes absorption, distribution, metabolism, and excretion. Distribution includes interchanges between blood and target, storage, and excretory organs.

Toxicokinetic studies are used to determine the relationships between systemic exposure and the time course of dose to target organs and elimination from the blood, and to assess relationships between exposures to animals and corresponding exposures to humans. *Toxicodynamics* describes the physiologic mechanisms by which toxic substances are absorbed, distributed, metabolized, and excreted and the mechanisms of action on affected target molecules,

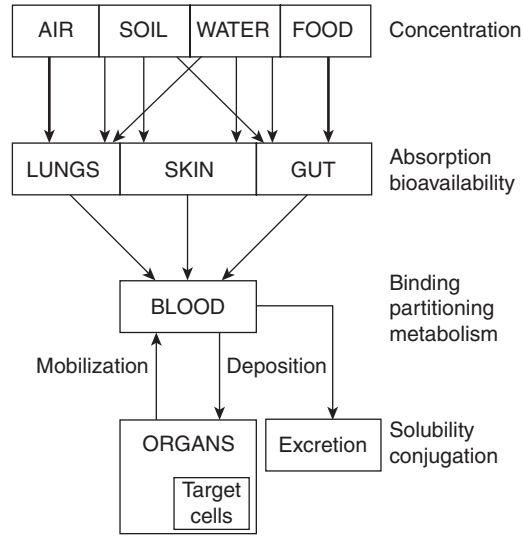

Figure 25-3. A multicompartment model of toxicant distribution showing the relationship among uptake, metabolism, distribution, storage, and excretion. (Used with permission of the Environmental and Occupational Health Sciences Institute.)

organelles, and cells. As researchers provide data on partitioning coefficients and metabolic rates, it becomes possible to develop models that predict how much of a chemical will circulate through each organ, using data on the perfusion rate (amount of blood delivered per minute). These are called *physiologically based pharmacokinetic* (PBPK) models, patterned after models that predict the fate of pharmaceutical agents in the body.

Absorption

Absorption occurs mainly through the lungs, gastrointestinal tract, and skin. When the skin or mucous membranes (mucosa) lining the respiratory or gastrointestinal tract come in contact with a contaminated medium, there is an opportunity for transfer of contaminants across the skin or mucosa into the bloodstream. For every chemical, there is a characteristic absorption pattern across the skin (usually very low) and mucosa (sometimes very high), depending on the chemical and physical attributes of the chemical, including its polarity, solubility, and size. Small, nonpolar compounds tend to be lipophilic and readily pass through membranes. The skin and mucosa prevent the ingress of most large, polar molecules. In addition, there are differences among organs. For example, elemental mercury is volatile at room temperature, and mercury vapor is readily absorbed through the lung; however, the same amount of elemental mercury, if swallowed, would pass through the intestinal tract with negligible absorption. In contrast, if methylmercury were ingested, there could be almost complete absorption.

The superficial linings of the skin, lungs, and gastrointestinal tract form barriers that retard the exchange of water and solutes between the environment and the extracellular and intracellular compartments. Material crosses membranes in various ways, by free diffusion (mainly of polar molecules), through small pores, and by transporter molecules (mainly for polar compounds).

There can be individual variability and variability related to age or other factors. For example, children can absorb about 50% of the lead they ingest, while adults absorb usually less than 10%. Women who have depleted iron stores absorb a much higher proportion of ingested cadmium than men or women with normal iron stores. Transporter molecules are specific carriers of certain toxics, and their presence and efficiency varies among people, in part due to genetic factors. Transporters serve a normal physiologic function; for example, metallothionein proteins regulate movement of zinc, an essential element, through the body. Cadmium, a xenobiotic, strongly binds to metallothionein, which carries it to other organs, including the kidney where it is excreted.

Bioavailability

Whereas absorption for any chemical is a property of the organ, bioavailability refers to the matrix properties, especially of soil and food, which may bind a toxic compound and interfere with absorption. Even a chemical that is readily absorbed in pure form may not be absorbed from a particular environmental medium. For example, soil characteristics may affect binding of some toxic chemicals, altering their bioavailability. In Times Beach, a small town in Missouri, dioxin-contaminated oil was used to suppress dust on dirt roads throughout the town, ultimately leading to an unprecedented buy-out of the entire town by the federal government and relocation of all of its residents in the early 1980s. In Newark, New Jersey, where Agent Orange, an herbicide contaminated with dioxin, had been manufactured at a factory on the Passaic River, leaks and fires had contaminated surface soil and river sediment with dioxin. The relatively sandy soil from Times Beach readily yielded dioxin when fed to animals by gavage (indicating high bioavailability); in contrast, dioxin was bound tenaciously to the oil-contaminated soil from Newark so that little could be extracted and absorbed in the gastrointestinal tract (indicating low bioavailability). Therefore, the amount of absorption of any chemical depends on both *(a)* the absorptive capacity of the contact organ, and *(b)* the bioavailability of the chemical in its environmental medium or matrix.[2]

Transport

Once a xenobiotic has entered the bloodstream, it is transported by the blood to many organs. Chemicals absorbed from the gastrointestinal

tract are carried first to the liver through the portal circulation—sometimes referred to as the *first pass*, where they may undergo metabolic transformation. In contrast, many volatile chemicals absorbed through the lung are delivered immediately to all organs, including the brain and kidneys. Substances can be carried in free form or bound to transporter molecules, which are usually proteins. Once a substance reaches an organ, its transfer from the capillaries of the organ out into the extracellular fluid or into cells is partially governed by how strongly it is bound in the bloodstream.

Metabolism

Metabolism consists of the processes organisms use to handle foreign substances that have been absorbed. It is divided into two phases. Phase I includes a series of transformations, involving mainly oxidation or reduction that make the absorbed compound more water soluble. Phase II involves linking the toxic substance to a molecule, which can make it more water soluble and facilitate its excretion in the urine. A xenobiotic may undergo metabolic alteration in any organ, but this is one of the specialized functions of the liver. Sometimes, the metabolic change reduces the toxicity (*detoxification*); in other cases, it increases it (*bioactivation*). Metabolism may also make a compound more (or occasionally less) water soluble, thereby enhancing the ability of the kidney to eliminate the chemical. Since metabolism is not usually totally effective, there can be a complex situation with both the absorbed chemical and one or more metabolites circulating at the same time. The ratio of the metabolite(s) to the parent compound in the blood is called the *metabolic ratio*, which is sometimes used to measure the efficiency of metabolism in humans.

Intermediary Metabolism (Biotransformation)

Metabolites may have greater or lesser toxicity than the parent compound. While the liver detoxifies some xenobiotics, it metabolically activates many others, usually through an enzymically mediated, oxidative reaction. Some oxidized metabolites, often referred to as *reactive intermediates*, can "attack" cell membranes as well as intracellular membranes and macromolecules.

Phase I reactions include hydrolysis or hydroxylation, leading to epoxide formation and other outcomes. The cytochrome-P450 enzyme systems play major roles in Phase I reactions. Phase II reactions link a metabolite to glucuronic acid, or add an acetyl, methyl, or sulfate radical. Phase II reactions usually increase the hydrophilic nature of the substance, facilitating its excretion in urine.

Metabolic enzymes are found in most tissues, including those at sites of initial contact with chemicals, such as the epithelium lining the respiratory tract and the skin and mucous membranes. The greatest variety and quantity of metabolic enzymes are in the liver. Within cells, these enzymes are found mainly in the microsomal component of the endoplasmic reticulum but also in the cytosol and other organelles. Certain xenobiotic compounds are metabolized by intestinal flora. Many of the metabolic responses begin with an oxidation step (Fig. 25-4).

Cytochrome P-450

Among Phase I metabolic enzymes are *cytochrome P-450s (or P450s)*. These occur in many families in all organisms. An entire subdiscipline has arisen to study the classification of P450s and their variation among species and organs, the different substrates on which they act, their specificity (or lack thereof), and the metabolites they produce. Once known generically as *liver microsomal oxidases* or *mixed function oxidases*, various metabolic functions are now being assigned to specific P450s, a few of which are discussed next.

P450s are found in most tissues, although the greatest amount and variety occur in the liver. They have a general feature of adding oxygen to a substrate, forming a highly reactive epoxide or a less reactive hydroxide. Oxygen can be involved in breaking double bonds, cleaving esters, and in dehalogenation reactions. As new forms of these enzymes have been discovered, they are assigned to major families and subfamilies. Much of the research on P450s has come from the pharmaceutical industry while investigating how drugs are metabolized. For example, the P450 that

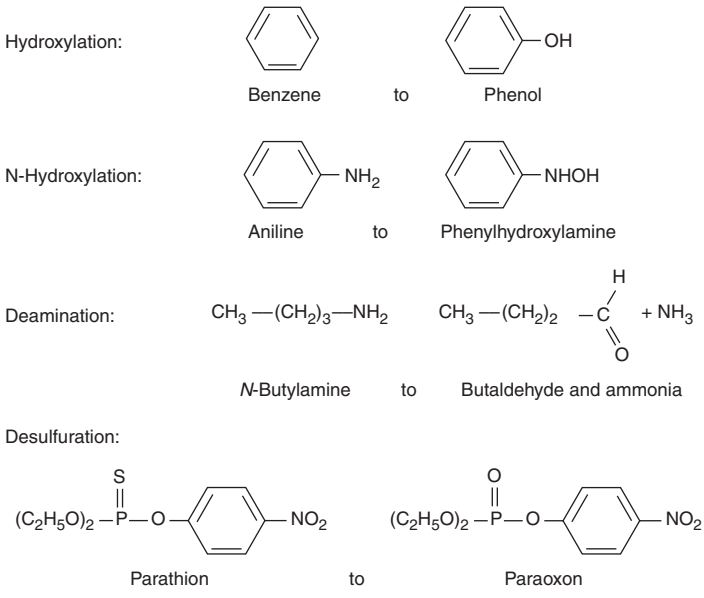

Figure 25-4. Examples of oxidation reactions in metabolism of several common industrial chemicals.

metabolizes caffeine is referred to as P450 1A2 (often abbreviated as CYP1A2). This enzyme also metabolizes some toxic chemicals with very variable efficiency among individuals. Its intrinsic activity can be evaluated by the caffeine breath test. Subjects are given a dose of caffeine labeled with isotopic carbon-13, and the amount of (C-13) carbon dioxide ($^{13}CO_2$) is measured in exhaled breath over a 2-hour period. A high percentage recovery of labeled $^{13}CO_2$ indicates high CYP1A2 activity, and decreased susceptibility to polychlorinated aromatic compounds, such as dioxins and furans. Great interest has focused on CYP2D6 because it is the enzyme that metabolizes the drug debrisoquine; however, it also metabolizes a variety of other xenobiotics.

Tissue Specificity of Enzyme Expression

Variability in P450 expression in different tissues is important, since significant metabolic activation or detoxification may occur in target tissues. Thus, CYP1A2 is expressed in the liver, but not other organs, while CYP1A1 is low in the liver of most mammals, but high in other tissues. Both are induced by polyaromatic hydrocarbons (PAHs) and indoles. Since the two CYPs catalyze different reactions, a single substrate may follow different metabolic pathways in different tissues. This is a rapidly evolving area of research, with important applications to pharmacology and toxicology.

Flavin-Containing Monoxygenases

The flavin-containing monoxygenases (FMOs) represent another family of oxidizing enzymes that are NADPH-dependent. They act on nitrogen-, phosphorus-, or sulfur-containing substrates, such as amines, organophosphates, or thiols. The FMOs have different isoforms that are distributed differently among species and organs. FMO1 occurs at high levels in rat and rabbit liver, with low levels in mouse and human liver; FMO3 occurs at a high concentration in human and mouse liver, but low concentrations in rat and rabbit liver. Liver cells of female mice have higher amounts of FM01 and FMO3 than do male mice.

Examples of Metabolic Activation

An example of the importance of metabolic activation is the case of 1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine (MPTP), an accidental by-product in the production of a designer narcotic. MPTP is oxidized by monoamine oxidase to MPP+, which is transported by the dopamine transporter and concentrates in dopaminergic

neurons of the brain, where it inhibits cellular respiration and causes cell death. An MPTP-containing drug was sold "on the street" and many young adult users ingested dangerous levels and developed irreversible parkinsonism. Since this discovery, toxicologists have used MPTP to create animal models for research on parkinsonism.

Many chlorinated and aromatic hydrocarbons, such as vinyl chloride and trichloroethylene, undergo metabolic activation by formation of a reactive epoxide intermediate. The P450 system also metabolizes the analgesic, acetaminophen, to a quinone, which causes centrilobular necrosis of the liver. Acetaminophen is acted on by prostaglandin H synthase in the kidney, producing a nephrotoxic free radical. In the bladder, the prostaglandin H synthase system metabolizes a variety of aromatic amines into genotoxic metabolites that can induce bladder cancer. In rats, these same organic amines undergo N-hydroxylation in the liver and cause liver cancer.

Phase II Reactions

Phase II reactions include several important conjugation reactions. Metabolites from phase I reactions can undergo conjugation with other molecules, which facilitates their transport and excretion in urine. There are several types of conjugation reactions (Fig. 25-5), among which conjugation to reduced glutathione (GSH) is especially common. This affects a wide range of electrophilic substrates and is accelerated by glutathione-S-transferase (GST) enzymes. Glucuronidation involves connecting the metabolite to glucuronic acid, by various enzymes called glucuronosyltransferases that are found in various mammal tissues. The low-molecular-weight glucuronide complexes are excreted mainly in the urine, although some forms are excreted in the bile.

Polymorphisms at the GST loci result in variable efficiencies of the conjugation reaction. Divalent cations, such as many metals, readily bind with sulfhydryl groups, including GSH. Exposure to mercury increases the activity of several enzymes involved in the synthesis of GSH and the reduction of oxidized glutathione (GSSG). Conversely, acetaminophen depletes GSH levels in liver. Both the depletion and the subsequent hepatotoxicity are inhibited by diallyl sulfone, a metabolite of garlic, which inhibits CYP2E1, the enzyme that activates acetaminophen.

Sulfation is the major means of preparing phenol for excretion. It is also used for alcohols, amines, and other categories of chemicals. Sulfation and acetylation exemplify the sequential phase I and phase II metabolic reactions. Phenol and aniline can be metabolites of other toxins and then be conjugated and excreted. The addition of mercapturic acid (N-acetylcysteine) is a multistep process that proceeds through the addition of glutathione and subsequent

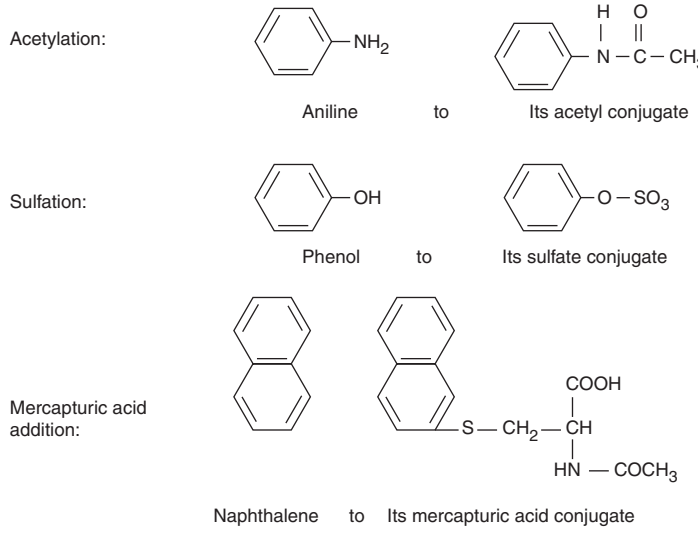

Figure 25-5. Examples of Phase II (conjugation reactions).

cleavage to cysteine derivatives. This reaction is extremely important in handling reactive electrophilic compounds that result from exogenous exposure or endogenous metabolic processes. Polycyclic aromatic hydrocarbons and polyhalogenated hydrocarbons are predominantly excreted in this manner.

N-Acetyl Transferase

Aromatic amines or hydrazines with a nitrogen atom can be metabolized by attaching acetate to the nitrogen (N-acetylation). This is accomplished by N-acetyltransferases (NATs) and serves as a major degradation pathway. There are at least three forms of NAT, and a deficiency in either activity or structure of NAT2 results in slow acetylation of certain drugs, such as isoniazid. This deficiency occurs in about 70% of people in the Middle East, 50% of Europeans, and 20% of Asians.

Sequestration or Storage

A chemical or its metabolite circulating in the bloodstream can be delivered to many organs simultaneously—excretory organs, target organs, or storage organs. Chemicals may be stored for days, months, or decades in storage organs, usually while manifesting little evidence of harm. For example, lead is stored in bone, where it is fairly innocuous; lead exerts its primary toxic effects in the nervous system and other organ systems. Organochlorines, such as polychlorinated biphenyl compounds (PCBs), are stored in fat. They generally do not harm fatty tissue; but if a person mobilizes fat rapidly, massive release of stored PCBs may lead to a potentially harmful, relatively acute, dose to sensitive target organs. Cadmium is stored mainly in the kidney, its primary target organ; even after exposure is terminated, the cadmium is eliminated from the kidney very slowly.

Elimination or Excretion

Once a xenobotic and/or its metabolites are circulating in the bloodstream, they can be delivered to an excretory organ. Excretion is mainly through the urine and feces, but volatile compounds can be excreted primarily in exhaled breath. Many biomarker tests rely on measuring the concentration of a chemical in urine or exhaled breath. Many chemicals, especially those that are lipophilic, are readily transferred to breast milk, posing more of a hazard for an infant than its mother. Some compounds, particularly metals, concentrate in skin or hair, and are lost through the natural sloughing of epidermal cells or hair growth. Substances that are water soluble—or become water soluble—through conjugation are excreted via the kidney; however, they may be toxic to the kidney or bladder because they are concentrated in these organs during urine production. Lipophilic substances or complexes may be secreted into bile and then excreted in the feces; some compounds excreted in the bile—in what is referred to as an enterohepatic cycle—may be reabsorbed in the intestine, thereby retarding elimination and enhancing toxicity.

The bloodstream delivers toxic substances to the renal glomerulus, where most are filtered with water and many other substances, forming the glomerular ultrafiltrate. Only cellular elements—large proteins such as albumin and substances bound to them—escape the filter and remain in the blood. Some of these may be secreted into the renal tubule. As the filtrate leaves the glomerulus and begins to pass down the tubule, the concentration of the toxic substance is similar to its concentration in the bloodstream. However, by the time the filtrate has traversed the tubular system and enters the collecting duct, about 99% of the water has been reabsorbed, so that the toxic substance is about 100 times more concentrated in the urine than in the blood. In this form, it is delivered to the bladder, where it may reside for hours before being eliminated. The liver also plays a prominent role in excretion by producing bile, which may incorporate nonpolar compounds that are not easily excreted by the kidney. Bile carries toxic compounds with it into the intestinal tract.

The rate of excretion of a toxic substance from the body is an important variable. As long as it equals or exceeds the rate of intake, the substance will not build up in the body. Excretion is widely used in biomonitoring. Blood and urine concentrations are measured for many compounds. Hair and fingernails have been used to monitor metals, because they are composed of

keratin, which is rich in sulfhydryl groups that readily bind certain metals, such as mercury. Comparing concentrations in two or more fluids or tissues provides information on the nature of the chemical. Thus, organic mercurial compounds, such as methylmercury, are readily deposited in hair or are excreted in the feces, while inorganic forms of mercury are eliminated mainly in the urine. People who have high blood and urine mercury levels, but low hair mercury levels, probably have been exposed to inorganic, rather than organic, mercury. In contrast, people who have consumed much fish may have high blood and hair mercury, but low urine mercury.

Biological Half-Life

The biological half-life for any compound is the amount of time it takes for half of an absorbed dose to be eliminated from the body. This is not always easy to calculate, particularly if there is ongoing exposure. Unlike the constancy of a radiologic half-life, the elimination of a compound may be biphasic or even triphasic—that is, much of the dose can be eliminated rapidly in a few hours, and the remainder eliminated more slowly over days or weeks. The amount of a toxic substance that is circulating in the bloodstream at any time or the amount delivered to target organs represents a balance between *(a)* uptake and *(b)* elimination or storage. If exposure were terminated, the amount of the toxic substance in the body would gradually decrease. Some substances with short biological half-lives are rapidly excreted, while others with long biological half-lives tend to remain in the body for long periods. Many chemicals have a biphasic or even triphasic elimination pattern, with very rapid elimination for the first few days following exposure, followed by very gradual elimination as the substance is re-released from organs and delivered to the kidney. There can be substantial interindividual variation in the biological half-life of a given chemical; for example, cadmium may have a biological half-life ranging from a few years to many decades.

Delivery to Target Organs

While a chemical is being delivered to storage or excretory organs, some is delivered to *target organs*, where the toxic substance enters cells. The *target organ dose* is the amount of the substance that enters the target organ. This depends on the blood perfusion rate of the organ and the movement of the substance from the blood and ultimately across cell membranes, either by passive diffusion or a variety of active-transport mechanisms. The diffusion rate, following Fick's Principle, is proportional to the concentration gradient, the membrane surface area, and a compound-specific coefficient (which depends on the octanol:water partitioning coefficient, with lipophilic compounds passing membranes more quickly than hydrophilic ones). Theoretically, the diffusion coefficient is inversely related to the cube root of the molecular weight of the compound, with smaller molecules therefore passing through membranes more rapidly than large ones.

Any organ can be a target organ. The lungs (Chapter 18) and the skin (Chapter 22) are frequent target organs because they are in close contact with the environment. The liver and kidneys are also frequent target organs because of the characteristics of their blood supplies and the many metabolic and excretory processes that take place in these organs. Adverse effects of chemical exposures on the liver (hepatotoxicity) include hepatocellular injury, cholestatic injury, fatty liver, granulomatous disease, cirrhosis, and malignancies, including hepatocellular carcinoma and hepatic angiosarcoma. A wide variety of chemicals, including organic solvents, vinyl chloride monomer, arsenic, chlorinated pesticides, and infectious agents can adversely affect the liver. Adverse effects of chemical exposures on the kidneys include proximal tubule dysfunction or damage, immune-mediated glomerulonephritis, end-stage kidney disease, and malignancies of the kidney or bladder. A wide variety of chemicals, including cadmium and other heavy metals, organic solvents, and aniline dyes, can adversely affect the kidneys.

TOXICOLOGIC ENDPOINTS

Health professionals are concerned with identifying and preventing morbidity and mortality endpoints—ranging from skin lesions to death, and involving molecular, biochemical, anatomic, physiologic, behavioral, or other effects.

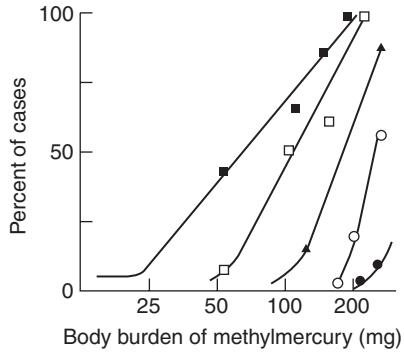

Figure 25-6. A series of dose–response curves for different endpoints of methylmercury toxicity reflecting a major poisoning episode from contaminated grain in Iraq. For each endpoint, a separate dose–response curve can be drawn, and these are nested from the least serious on the left (paresthesias occurring at the lowest dose) to death on the right, compared to the estimated body burden of methylmercury. Solid squares, paresthesias; open squares, ataxia; solid triangles, dysarthria; open circles, deafness; solid circles, death. (Used with permission of the Environmental and Occupational Health Sciences Institute. Based on data from Takizawa Y. Epidemiology of mercury poisoning. In Nriagu J (ed.). The biogeochemistry of mercury in the environment. Amsterdam: Elsevier, 1979.)

For example, Figure 25-6 shows a series of dose–response curves for different endpoints of methylmercury toxicity, reflecting a major poisoning episode in Iraq due to contaminated grain. For each endpoint, a separate dose–response curve can be drawn; these are nested from the least serious on the left, occurring at the lowest dose, to death on the right.

Traditionally, toxicologists have used the LD_{50} as a common primary endpoint for comparing potency of different substances. This is the dose at which 50% of the animals tested will have succumbed. The LD_{50} has also been used to assess the efficacy of antibiotics and pesticides. The potency of chemicals can be ranked on the basis of the LD_{50}. Other endpoints can be quantified the same way, yielding an ED_{50} (the dose which produces a particular effect in 50% of the animals) or an ED_{10} (the dose which produces the effect in 10%). The ED_{10} is sometimes referred to as a benchmark dose. However, most often we are interested in doses that affect only 1% to 10% of the population—the most sensitive people. Many recent studies use a broad range of biochemical, physiological, behavioral, and other endpoints. The ED_{50} and the ED_{10} can be calculated from data generated in animal studies using computer programs.

DOSE–RESPONSE CURVES

The *dose–response curve* describes how any particular response increases in frequency or intensity in an individual or population as the dose increases. Figure 25-7 represents a series of dose–response curves, with dose plotted on the x axis and the response on the y axis. The y axis may be the number of cells killed, the amount of a biomarker released, the number of animals affected, the percent of people with a particular symptom, or the number who die. The most common dose–response curve has a sigmoid shape with three zones: subthreshold, rapid increase, and maximal effect or plateau. If the toxic effect is idiosyncratic, relying on the underlying susceptibility of individuals to a great extent, the sigmoid curve may not be a good representation.

The central portion of the dose–response curve can rise with varying degrees of steepness, reflecting differences in susceptibility of the target population. A chemical that induces the same effect in all people, with little differential susceptibility, will have a much steeper rise than one for which individual responsiveness differs greatly. Dose is usually measured as amount of chemical, such as in milligrams per kilogram of body weight. This works reasonably well for systemic exposures and effects, but it does not adequately describe some toxic phenomena, such as skin sensitization.

Threshold

Theoretically, a *threshold* is the lowest dose at which a toxic substance causes a response, usually in the most susceptible individual. Practically, the ability to establish and precisely estimate a threshold for a particular compound is limited by several factors, including the size of the population sample and the number of different doses used in an experiment. For any given chemical, the threshold may vary, depending on conditions of exposure and individual susceptibility. The threshold can be used as a basis for guidelines or standard setting. Thresholds must be

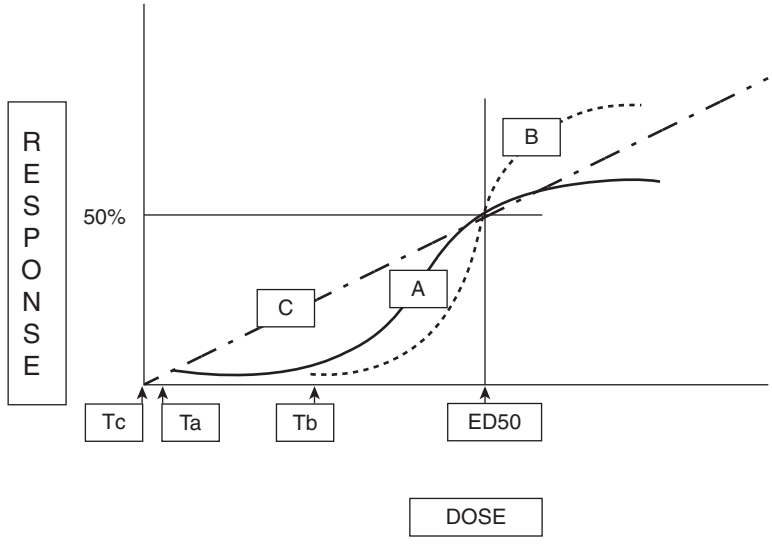

Figure 25-7. Dose–response curves for three hypothetical chemicals. Curves A and B are typical sigmoid curves differing in potency and efficacy. C is a linear, no-threshold curve presumed to be characteristic of the causation of cancer by ionizing radiation. B has a higher threshold than A. Thresholds are indicated by T. ED50 represents the dose corresponding to a 50% response. (Used with permission of the Environmental and Occupational Health Sciences Institute.)

defined in terms of a particular form of a chemical, time course of exposure, target population, and response.

Most toxicological experiments use at least three doses (including a zero dose control), but relatively few studies use as many as five doses. Thus, knowledge of the actual threshold may be crude, since it seldom corresponds exactly to the dose used. Thus, experiments yield a lowest observed adverse effect level (LOAEL), which is the lowest dose above the control value at which the effect is detected. In some studies, this may correspond to the lowest dose tested. A no observed adverse effect level (NOAEL) is the lowest level above the zero control dose at which no effect can be detected. Both LOAELs and NOAELs are used in risk assessment and standard setting.

Although for most chemicals and most responses it is possible to detect a threshold, this is not always possible. Radiation carcinogenesis is generally assumed to follow a nonthreshold pattern. For childhood blood lead levels, there is no detectable NOAEL for neurobehavioral effects; some studies have found that the response slope is steeper in the 5 to 10 μg/dL range than above 10 μg/dL. Since there is no known beneficial role for lead in the body, it is not surprising that any amount of lead may be harmful. Since the threshold is the dose below which no effect is detected, it is possible—and indeed likely—that for lead any apparent threshold was due to the insensitivity of testing, rather than to lack of adverse effect.

Chemical carcinogens vary in their mechanism of action. A major controversy in toxicology is whether the linear nonthreshold model is an appropriate description of the dose–response relationship for chemical carcinogens. At present, this model is used for genotoxic carcinogens. In developing its risk assessments for cancer, the EPA uses a linearized multistage model, to account for cancer arising not only from induction (chemical alteration of the genetic material) but also from factors governing promotion and proliferation.

Hormesis

Chemicals have multiple endpoints with multiple dose–response curves (Fig. 25-7). A special issue in dose–response is hormesis, a phenomenon in which harmful substances, such as radiation, may have some beneficial effects at low doses. This phenomenon of benefit and harm is best exemplified by pharmaceutical agents or

nutrients, such as vitamin A and copper, which not only have a therapeutic (beneficial) but also a toxicologic threshold. Ideally, these two thresholds are far apart. However, where they are close—and therefore a small margin of safety exists—toxicity can occur. Many drugs, for example, have been removed from the market, or never make it to market, because of a low margin of safety.

The concept of hormesis emerged from studies of ionizing radiation, where low doses of radiation have generally been associated with increased longevity. However, the dose–response curve for radiation-induced mutation is independent of any beneficial dose–response curve that might exist, and genetic damage can occur at doses below those postulated to give benefit. Proponents of hormesis argue that dose–response curves are often U-shaped. However, virtually all examples intended to illustrate hormesis use noncomparable endpoints and therefore are not true U-shaped curves. Critics of low-dose extrapolation argue that it ignores hormesis. Hormesis is therefore highly controversial, including in a political context. One must be cautious in interpreting arguments about exposure standards based on hormesis. Hormesis is generally a distraction in discussions of regulation and policy.

BEYOND DOSE–RESPONSE RELATIONSHIPS: PREDICTING TOXICITY IN HUMAN INDIVIDUALS AND POPULATIONS

Dose–response relationships are fundamental to understanding and predicting degree of toxicity of particular toxic substances. A general principle of toxicology relies on the extrapolation of dose–response relationships derived from animal studies to effects on humans. The biochemistry, physiology, and organ structure of humans is very similar to that of other mammals—and to vertebrates in general. Information needed to establish dose–response relationships in human beings comes mainly from animal studies and from epidemiological studies of workers exposed to relatively high levels of toxic substances. For some common industrial chemicals, there is a database of dose–response information based on studies of acute and chronic exposures among workers. To estimate environmental effects, we customarily use data from relatively high workplace exposures, which requires extrapolation down to levels found in communities. For most industrial chemicals, however, data on adverse effects in humans are limited; therefore, we must rely on animal studies.

There are additional challenges in using dose–response information from animal studies to assess the toxicity of exposures to human populations, such as varying susceptibility among individuals. In most animal toxicity studies, genetically homogenous, inbred animal strains are used to reduce variability of responses. But results need to be extrapolated to genetically diverse humans. In human populations, responses to toxicants vary by age, gender, and race/ethnicity, and individuals' genetic backgrounds. In addition, human populations may be exposed to a variety of other chemical substances, and other factors, such as diet, psychosocial stress, and physical activity, may affect responses to toxic substances. In contrast, in dose–response laboratory experiments, environmental conditions are usually strictly controlled so that only the dose of the substance being tested is varied.

Many chronic diseases, such as cancer, heart disease, asthma, and diabetes, appear to result largely from interactions among the person's genetic material and environmental exposures. New so-called "omic" technologies allow for profiling of toxic responses at the levels of gene transcription, protein expression, and metabolism of small molecules. (See page 546.) Ultimately, these technologies may help us to understand complex gene–environment interactions and the effects of multiple stressors.

Interspecies Differences in Response

Although the overall similarities in biochemistry and physiology among animals allow toxicologists to extrapolate among species, the differences among organisms are equally important to understand. Differences in toxic responses between species are well established. These differences have long been exploited in the development of biocidal agents that target specific organisms, such as pesticides, fungicides,

herbicides, and antibiotics. This biological diversity makes it difficult to use results of experiments performed in laboratory animals to predict, with a high degree of certainty, the toxicity of a substance in humans. For the most part, these differences are quantitative (matters of degree) rather than qualitative, but even among similar species, large differences in toxic responses may be observed. For example, the LD_{50} for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD, or dioxin) is more than 1,000 times greater in resistant hamsters than in sensitive guinea pigs. Differences in toxicity may be due to differences in toxicokinetics and/or toxicodynamics. Most of the almost 100,000-fold difference in susceptibility to liver tumors induced by aflatoxin B1 between rats and mice can be explained by differences in metabolism related to expression of cytochrome P450 and glutathione-S-transferase genes.[3] Improved understanding of underlying biological mechanisms will contribute to the validation of extrapolations from animals to humans.

Interindividual Differences in Response

One person may be a heavy cigarette smoker who dies of lung cancer at age 50. Another person may by a heavy smoker who dies at age 90 of other causes. Which biological processes underlie these individual differences in susceptibility to the carcinogenic effects of tobacco smoke? Proteins—primarily enzymes, receptors, and transport proteins—drive toxicokinetic and toxicodyamic processes. The structure, function, and level of expression of these proteins is, in turn, determined largely by the genes that code for these proteins. However, this is not the whole story. Previous, or concurrent, exposures to other chemicals and nonchemical stressors can cause variation in toxicity by several mechanisms.

Genetic Differences

The genomes of individual organisms are the complete sets of genes encoded in the sequence of bases in their DNA. An individual's genetic heritage is determined at conception. The human genome consists of about 3 billion nucleotide base pairs and includes about 20,000 genes. While the amount of genomic variation between individuals is remarkably small—about 0.1% between any two people—these differences underlie the uniqueness of each individual. A polymorphism is a genetic variant that, by convention, occurs in more than 1% of a population. More than 90% of human genetic variants are single nucleotide polymorphisms (SNPs), in which a single nucleotide (A, T, G, or C) differs within a DNA sequence. Other types of genetic variants include insertion or deletion of one or more nucleotides. The extent to which a given SNP or other variant will alter the expression of a gene is highly variable. If a SNP is in a noncoding region of the gene, it may be "silent." If a SNP is in a coding region, and alters the amino acid sequence of the protein, it may change the activity of the protein product. Deletions and other variants may lead to nonfunctional proteins. Variants that do not alter amino acid sequence may affect expression by altering gene splicing or transcription-factor binding.

While some genetic variants cause "simple" genetic diseases, such as Huntington disease (inherited as an autosomal dominant condition), most genetic variants that contribute to disease only cause relatively small increases in risk of "complex" diseases. Sometimes referred to as Mendelian disorders—because high penetrance allows them to be tracked in families, these "simple" genetic diseases account for less than 5% of the human disease burden. Sickle cell anemia is an example of a genetic disease caused by a single base-pair substitution in the gene for the hemoglobin beta subunit, which alters a single amino acid in the protein. In contrast, multiple combinations of genetic variants, each making small contributions to disease risk, are believed to contribute to most of the human disease burden. Furthermore, gene–environment interactions are believed to be important in many diseases, such as asthma, in which individuals with a genetic susceptibility only manifest the disease after some presumed environmental exposure(s).

Polymorphisms in the genes that code for various P450 proteins have been shown to result in different metabolic phenotypes that may increase or decrease risk of toxic responses to particular chemical substances. For example, about 5% to 10% of Caucasians, and a smaller percentage of Asians, have a mutant CYP2D6 that was found

to be inefficient at metabolizing the antihypertensive drug debrisoquine to its hydroxide form. The metabolic ratio, defined as:

$$\text{Metabolic ratio} = \frac{\text{Debrisoquine-OH}}{\text{Debrisoquine}}$$

can serve as a measure of metabolic efficiency. Those who are poor metabolizers of debrisoquine (with a low metabolic ratio) are also poor metabolizers of other substances acted on by CYP2D6.

For example, people whose CYP2D6 genotype makes them poor metabolizers of debrisoquine are at risk of various adverse drug reactions, whereas extensive metabolizers are at increased risk of lung cancer, probably because of carcinogenic metabolites they produce. Therefore, CYP2D6-deficient people are protected from certain environmentally caused cancers, such as lung, bladder, and liver cancer, because of their failure to activate certain pro-carcinogens.

Toxicogenetics and Toxicogenomics

Toxicogenetics is the study of individual genetic differences that lead to different responses to toxicants. *Toxicogenomics* is the study of how exposure to chemical substances affects the expression of genes at the levels of the mRNA transcript (*transcriptomics*), protein (*proteomics*), and metabolites (*metabolomics*). Both of these approaches can contribute to the understanding of individual differences in response to toxicants. Ultimately, these methods may lead to personalized medicine, in which approaches to the prevention, detection, and treatment of disease are tailored to an individual's risk profile and predicted response to interventions. However, reaching this goal will require overcoming the computational and statistical challenges arising from huge quantities of data and enormous numbers of gene–gene and gene–environment interactions.

Over the past decade, new "omic" technologies have been developed that allow for profiling and analysis of biological responses at the genetic, protein, and metabolite levels. Modern genetic methods, such as polymerase chain reaction (PCR), have made it possible to amplify (make very large numbers of copies of) the genes coding for the enzyme, receptor, and transport proteins involved in toxicokinetic and toxicodynamic processes. Modern genetic methods make it possible to study individual genes. With genomic technologies, thousands of genes or gene products can be identified on a single gene chip (microarray). Microarrays consist of thousands of 150–200 micron spots of DNA bound to microscope slides in a known pattern. In *transcriptomics*, similar microarray technology enables the very efficient quantification of mRNA expression of thousands of genes simultaneously. The degree of hybridization of specific mRNA at each spot can be quantified with great sensitivity and specificity. At the level of protein expression, proteomic techniques for separating and identifying proteins are used to analyze the proteome—the proteins present in the cell or tissue at any one time. Finally, metabolomic analysis of thousands of metabolic substrates, products, and cofactors in biological samples can now be achieved using nuclear magnetic resonance (NMR) and mass spectrometry techniques.

Ethical Issues in Genetic Screening

In addition to technical difficulties, the potential application of genetic information to individualized risk assessment faces ethical challenges that are not unprecedented. In 1938, the geneticist J. B. S. Haldane suggested that someday genetic screening might allow identification of hypersusceptible workers—but he emphasized that in the meantime industrial hygiene controls were needed.[4] In 1967, H. E. Stockinger advocated, with limited success, genetic screening of workers for glucose-6-phosphate dehydrogenase (G6PD) deficiency, sickle cell anemia, and alpha-1-antitrypsin deficiency,[5] arguing that workers with these genetic conditions would be hypersusceptiblie to chemicals. Over the ensuing decade, some companies experimented with mandatory or voluntary genetic screening tests; however, like preemployment back X-rays, most were abandoned because of inadequate positive predictive value in the populations screened, high potential for discrimination, and inadequate cost-effectiveness. Genetic screening for susceptibility has resurfaced with completion of the Human Genome Project, and serious ethical and legal issues have been raised. Some states

have legislated that genetic information cannot be used to deny health insurance or treatment, but they have not excluded its use in denying life insurance.

Epigenetics

Every somatic cell of an individual contains the entire genome of the individual. The turning "on" or "off" of specific genes results in the diversity of cell types in the body. Although some of these changes in gene expression are heritable in somatic or germ cell lines, they are not solely governed by the genetic makeup of an individual. *Epigenetics* describes the heritable changes in gene expression that are not coded in the DNA sequence. New discoveries indicate that epigenetic changes may be an important mode of action for toxic substances.

Known molecular mechanisms for epigenetic changes include DNA methylation, histone modification, and expression of noncoding RNAs. These regulatory mechanisms generally involve initiation and maintenance of the silencing of gene expression. Cancer is a disease in which epigenetic changes are well established. (See Chapter 17.) Cancer cells are characterized by both global hypomethylation and regional hypermethylation of tumor suppressor genes. Recently, toxic substances have been shown to cause aberrations in DNA methylation. Cigarette smoke stimulates demethylation of metastatic genes in lung cancer cells. Phenobarbital is a nongenotoxic carcinogen that induces global hypomethylation and regional hypermethylation in rodents. Epigenetic effects of other environmental exposures have recently been discovered. For example, exposure of pregnant Agouti mice to genistein, a phytoestrogen, results in methylation of a regulatory element for a gene that determines coat color, changing the coat color in the offspring. The extent to which epigenetic changes may be reversible is not yet clear.

Interactions between Multiple Exposures

Prior or concurrent exposures to other chemical and nonchemical stressors can lead to complex alterations in the response to a toxic substance. When an individual is exposed simultaneously to two or more chemicals or to a mixture of chemicals, a variety of interactions may occur, both outside and inside the body. These interactions are generally grouped into three categories: *(a)* independence and additivity, *(b)* synergism, and *(c)* antagonism.

If chemical A and chemical B each produce their effects independently of the other chemical, then there is no interaction. There is no interaction when chemicals affect different organs or produce different endpoints. Independence can occur when two chemicals follow different metabolic pathways, bind to different receptors, and do not compete. Additivity occurs when the two chemicals contribute to the same endpoint but show no interaction.

Synergism—a multiplicative effect—is of great practical importance, although there are remarkably few documented examples. In synergism, one chemical enhances the effect of another (or vice-versa), such that their combined effect on some endpoint is greater than would be expected from their independent dose–response curves. Chemical A may enhance the effect of Chemical B, enhance its activation, or interfere with its degradation and elimination. The best documented example of synergism is based on a study of lung cancer in relation to smoking and occupational exposure to asbestos.[6] Smoking increased lung cancer risk about 10 times; asbestos, about 5 times; and both, about 50 times.

Chemical A may reduce the effect of chemical B, if, for example, they compete for the same activating metabolic pathway or for the same receptor. Chemical A may inhibit the uptake of chemical B or its delivery to the target organ. If both A and B are activated by the same pathway, A may saturate the enzyme, preventing B from being activated.

Hypersensitivity

Hypersensitivity is a special case of susceptibility induced by prior exposure to particular substances. Exposure to allergenic compounds will eventually sensitize some workers so that they respond immunologically, developing either dermal or respiratory symptoms. Some allergens, such as chromium, are potent sensitizers, quickly affecting a small proportion of people who contact them. In contrast, a subset of workers who care for research animals in vivariums gradually build up allergic responsiveness that requires them to switch to other species until they become responsive to those as well. A worker who

becomes allergic to a particular chemical at work, will often require relocation, as avoidance of the low levels of exposure that cause hypersensitivity reactions is usually difficult and desensitization therapy is seldom an option. However, if an allergen is encountered only infrequently and predictably, then engineering controls and/or special personal protective equipment may reduce exposure below an individual's response threshold.

Adaptation, Tolerance, and Hardening

At the opposite end of the spectrum are workers who show very little response to a chemical at a dose that would produce symptoms in most co-workers. In some instances, with repeat exposure, physiologic adaptation—the opposite of sensitization—occurs in the metabolism of the chemical or at the target organ. People who build up tolerance to a chemical no longer experience its acute effects, but they may accumulate enough exposure over time for chronic effects to develop. Work hardening is the deliberate process of allowing, or requiring, individuals to work in conditions of gradually increasing exposure so they build up tolerance. This process usually deals with physical stressors, such as extremes of temperature or physically demanding work. Work hardening is an integral part of physical therapy and return-to-work strategies for recovery from musculoskeletal injuries, but it has little if any role in managing exposures to toxic substances in the workplace. Adaptation and tolerance may limit the usefulness of relying heavily on worker's symptoms in evaluating exposures to toxic substances in the workplace.

MECHANISMS OF TOXICITY

Mechanism refers to the manner in which a chemical causes damage, usually at the subcellular or molecular level. Understanding mechanisms is useful in risk assessment, such as in choosing between different extrapolation models for nongenotoxic, as opposed to genotoxic, carcinogens. Toxic substances can interact with different types of macromolecules, such as nucleic acids and proteins. They may bind to receptors, causing overactivation or inhibition of normal activation. The explosion of knowledge in cell and molecular biology, including the mapping of the human genome, gene expression and transcription, cell-cycle regulation, enzyme polymorphisms, cytokines, transcription factors, and cascades of signaling molecules, have greatly increased the opportunity to understand toxicological mechanisms.

Metabolic and Cellular Poisons

Chemicals, such as cyanide, that interfere with cellular respiration are among the oldest known poisons. A chemical may cause enzyme inhibition by binding to a site on an enzyme, altering its three-dimensional structure, and distorting its active site(s) so that it is no longer functional. Some chemicals alter the structure or function of intracellular membranes, such as membranes of the endoplasmic reticulum or the mitochondria. For many toxic substances, swelling of the mitochondria, with loss of detailed structure, is an early histological sign of damage. Other chemicals, such as the hemolysins of certain snake venoms, cause lysis of cells. Some chemicals, especially metals, may bind to the sulfhydryl groups of the cell membrane protein, disrupting its structure and increasing membrane fluidity.

Enzyme Induction

The body does not maintain a complete inventory of all the enzymes that it may need. Some constitutive enzymes are always present, but most enzymes must be induced by introduction of their substrate; it may take up to 24 hours before there is sufficient enzyme to metabolize a xenobiotic completely. Thus, the amount of enzyme in a cell may increase by several orders of magnitude. Some enzyme systems are highly specific and act only on a single substrate; others are nonspecific, catalyzing reactions on a wide range of substrates. Conversely, different substrates vary in their potency at inducing enzymes. Enzyme induction plays an important role in metabolizing xenobiotics. However, sometimes the most important consequence of enzyme induction is the greatly accelerated metabolism of endogenous bioactive compounds.

Enzyme inhibition is a common mode of action for toxic substances. Both cyanide and hydrogen sulfide interfere with the function of

cytochrome oxidase, thereby inhibiting oxidative phosphorylation that is necessary for cellular respiration. Heavy metals, which have a strong affinity for sulfur in proteins, are able to break the disulfide bridges that confer the tertiary structure necessary for normal function. Yet, because of differences in their atomic radius, different metals tend to inhibit different enzymes. For example, several enzymes in the pathway for making hemoglobin, such as delta-aminolevulinic acid dehydratase, are inhibited strongly by lead, but weakly by mercury. Therefore, induction can be accomplished, although with different efficiencies, by a range of substrates. CYP1A2 is induced by a variety of polycyclic aromatic hydrocarbons (PAHs). Before its identity was known, it was referred to as aryl hydrocarbon hydroxylase—its induction triggered by the substance binding to the Ah (aromatic hydrocarbon) receptor. Conversely, a single chemical can be acted upon by more than one of the P450s.

Receptors

Many toxic effects involve the binding of a xenobiotic or metabolite to a receptor, usually on a cell membrane. Receptors vary in their degree of specificity. Advances in receptor biology are proceeding rapidly. Many hormone effects are mediated by attachment of the hormone to a specific receptor. Some endocrine-active substances act by binding to the endogenous receptor without initiating the appropriate response. The effects of TCDD (dioxin) are partially mediated by binding to the Ah receptor. Related substances that bind to the Ah receptor have effects similar to TCDD, but with vastly different dose–response curves due, in part, to their binding affinity. In animal studies, dioxin binds to estrogen receptors in the breast tissue, interfering with normal estrogenic stimuli, and possibly reducing the likelihood of estrogen-stimulated breast cancer.

During normal function, a signal molecule binds to a receptor and initiates a response. After the signal molecule is removed or released from the receptor, another signal molecule may interact with receptor, causing a sustained response. Toxic compounds may bind to the receptor and not release, thereby blocking any further impulses and responses. For example, acetylcholine is a neurotransmitter that is secreted from the presynaptic terminal of parasympathetic nerve cell junctions. It rapidly diffuses across the gap and binds to special receptors on the postsynaptic nerve terminal, initiating a nerve impulse. Acetylcholine is then immediately deactivated by acetylcholinesterase. Some chemicals block the enzyme and others bind irreversibly to the receptor, in both cases preventing further nerve transmission.

Oxidative Stress and Free Radicals

Oxygen is sometimes referred to as highly toxic, because of its ability to alter molecules and change their function. Many bioactivation reactions involve oxidation. Normally, there is a balance between oxidative and antioxidant reactions. Oxidative reactions play important roles in inflammation, aging, carcinogenesis, and other aspects of toxicity. Research is discovering an increasing number of toxicants for which oxidative stress is an important mechanism. For example, chromium increases the formation of superoxide anion and nitric oxide in cells and enhances DNA single-strand breaks.

Toxicologists speak of reactive oxygen species, which are highly reactive molecules containing oxygen. Oxygen can receive an electron and form superoxide anion radical, which can, in turn, react with hydrogen to form hydrogen peroxide, which reacts with free electrons and hydrogen ion to form water and a highly-reactive hydroxyl radical:

$$\text{O-O} + e^- \rightarrow \text{O-O}^{*\cdot} \text{ (superoxide radical)}$$

$$\text{O-O}^{\cdot} + e^- + H^+ \rightarrow \text{H-O-O-H (hydrogen peroxide)}$$

$$\text{H-O-O-H} + e^- + H^+ \rightarrow H_2O + {}^{*\cdot}\text{OH (hydroxyl radical)}$$

In the course of these reactions, the highly reactive free radicals, especially the hydroxyl radical, are available to attack macromolecules, initiating a variety of toxic effects. The superoxide anion radical is formed in many oxidation reactions, where oxygen acts as an electron receptor. In response to the potential harm these reactive oxygen species may cause, the body has evolved antioxidant defenses, including water-soluble vitamin C and lipid-soluble vitamins E and A. Superoxide dismutase, a metalloprotein, and

glutathione-dependent peroxidases, in association with glutathione reductase, serve to scavenge free radicals. Excess nitric oxide production increases intracellular free radicals, enhancing neuronal degradation.

A new area of interest is oxidative damage to proteins through the binding of oxygen to various sites on the protein, forming protein carbonyls. Oxidizers, such as reactive oxygen species and nitric oxide, can bind to proteins, altering their configuration and activity. Much of the toxicity attributed to nitric oxide may be caused by peroxynitrite, which is formed by reaction between nitric oxide and another free radical species, superoxide anion. Peroxynitrite causes oxidative damage to lipids, DNA, and proteins. In general, the amount of oxidation correlates with aging and, in some cases, disease severity. Although oxidation is clearly involved in many chronic disease processes, in most cases it is not clear to what extent it plays a causative role in pathogenesis or is a result of the disease process.

One of the consequences of the formation of free radicals is reaction with unsaturated lipids, including those in cell and organelle membranes, to form lipid peroxides, which, in turn, lead to cell damage and dysfunction. Peroxyl radicals, which are also formed during lipid peroxidation, can react with other unsaturated lipid molecules to initiate additional lipid peroxidation. This chain reaction, initiated by a single free radical, can lead to formation of multiple molecules of peroxide. Some cytotoxicity of chlorinated hydrocarbons, such as carbon tetrachloride, is mediated by peroxidation of membrane lipids, which can be caused by a variety of reactive oxygen species. An active area of research involves identifying naturally occurring and synthetic compounds that interfere with lipid peroxidation.

Effects on Signal Transduction

Cell cycles are regulated by molecules that serve as signals to activate certain genes or receptors that influence the expression of other genes. Signal transduction pathways typically alter gene expression or modify gene products, either enhancing or inhibiting their function. Many endogenous signal chemicals, such as hormones and xenobiotics, can alter gene expression by activating transcription factors, which, in turn, promote the transcription of certain genes.

Adduct Formation via Covalent Binding to Macromolecules

Many chemicals react with and covalently bind to proteins and nucleic acids, forming stable adducts. DNA-repair enzymes may remove DNA adducts, but some adducts persist long enough to cause mutations during DNA replication. Adducts have been linked to cancer induction, and DNA adducts have been investigated as possible markers of exposure to genotoxins or carcinogens. (Interpretation of the frequency of adducts, however, is difficult, partly due to variable rates of DNA repair. Some adducts are repaired within hours, while others persist.) Smokers have higher levels of benzo(a)pyrene adducts to DNA than nonsmokers. DNA-protein cross-linking is promoted by a variety of genotoxic chemicals, including hexavalent chromium. Quantification of specific adducts has improved understanding of toxic mechanisms, but it has not yet proven useful for primary or secondary prevention of disease. In general, further study is needed to validate relationships between adducts and exposure and disease.

Genotoxicity

Genetic damage to germ cells (ova and sperm) may be heritable in offspring. Damage to somatic cells is not heritable, but it may lead to induction of cancer. Various chemicals and ionizing radiation damage nucleic acid directly or interfere with chromosomal replication and cell division. Fortunately, almost all damage to DNA is quickly repaired and only rarely does unrepaired damage go on to become cancer. For example, even in the absence of anthropogenic sources, people are constantly bombarded with ionizing radiation from natural terrestrial and cosmic sources, but almost all damage to DNA is quickly repaired and only a very small amount of the unrepaired damage goes on to become cancer.

Mutagens are substances that cause point mutations (replacement of one base nucleotide with another), chromosomal damage (breakage or translocations), or interference with meiosis, mitosis, or cell division. A variety of tests can

measure chromosomal aberrations, aneuploidy, sister chromatid exchange, translocation, micronucleus formation, glycophorin A, and T-cell receptor genes. New genetic techniques allow sequencing of genes and detection of changes at specific codons. (A codon is a sequence of three nucleotides.)

Genetic analysis can reveal changes, such as GC or AT base pair substitutions, deletions, or duplications at a single gene locus in individuals. The relative frequency of the different mutations is influenced by dose and the conditions of exposure. While GC substitutions are more common in nonsmokers, there is an increased frequency of AT substitutions in smokers. After radiotherapy, there is a substantial increase in rearrangements and deletions that can persist for several years.

Genotoxic chemicals may cause mutation in proteins called proto-oncogenes, producing a mutant oncogene that encodes for a modification of the natural protein product. Some changes, such as that in the Ras proto-oncogene, increase cell susceptibility to cancer. The p21 protein (so-called because it has a Dalton weight of 21 kilos) binds with a receptor on the inner cell membrane and mediates responses to growth factors. Mutation at codon 13 "locks" the protein into the active form such that it no longer responds to other cell signals. With signal transduction impaired, this permanent activation is associated with malignant transformation and proliferation.

Another important phenomenon is the role of tumor suppressor genes, such as p53. Mutant forms of p53 allow unbridled cell proliferation. "Knockout mice," which lack p53, develop cancer at an early age. Some patients with hepatocellular carcinoma have a specific mutation at the 249th codon of the p53. The same mutation also occurs in people exposed to aflatoxin $B_{1,}$ suggesting that the toxin may cause liver cancer by this highly specific mutation.

Carcinogenesis

Carcinogenesis typically involves three steps: initiation, promotion, and proliferation. Many carcinogens exert direct action on DNA and are referred to as genotoxic, but others seem to cause or allow cancer without a direct genotoxic mode of action. *Initiation* is the stage during which genotoxic damage occurs. *Promotion* is the stage during which initiated cells are induced to undergo cell division by certain promoter compounds. *Proliferation* is the stage when unbridled and uncontrolled cell division occurs, accompanied by growth of blood vessels that supply the growing tumor. (See Chapter 17.)

Some scientists believe that there must be a threshold for cancer as there is for other toxicologic reactions. Others argue, on theoretical grounds, that since no threshold (below which no cancer risk exists) has been demonstrated, there is no threshold for cancer. Most scientists are probably undecided about the no-threshold concept for carcinogens or believe that there may be thresholds for some, but not all, carcinogens. In the light of the ongoing controversy, some governmental regulatory agencies have concluded that until it is more certain, it is prudent to act as if there were no threshold for carcinogens. Thus, the application of a no-threshold approach to carcinogens can be viewed as a policy decision, rather than a scientific decision.

Apoptosis

Apoptosis, or programmed cell death, is a necessary part of the life history of most cell types. Activation of apoptosis leads to expression of proteins that prepare the cell to die. This is followed by phagocytosis of apoptotic cells. The important feature of this natural form of cell death, compared with cytotoxicity, is that the former proceeds without invasion of inflammatory cells. Apoptosis is an essential phenomenon during development, allowing the remodeling of tissues. Apoptosis also selectively eliminates cells with damaged DNA and also counters the clonal expansion of neoplastic cells. Inhibition of apoptosis, such as by estrogens, allows mutations to accumulate and tumor proliferation to occur. Hormone-dependent tumors expand when the hormone inhibits apoptosis, while an antiestrogenic drug, such as tamoxifen, allows apoptosis to occur. Conversely, the tumor-promotor phenobarbital inhibits apoptosis. Some chemicals appear to inhibit apoptosis, thereby enhancing the proliferative phase of carcinogenesis. New approaches to cancer chemotherapy focus on exploiting apoptosis to destroy tumor cells.

Immunotoxins

Immunotoxins act by activating or suppressing the immune system. Some alter the expression of immunoglobulins, while others affect lymphocytes. T lymphocytes mature in the thymus and are the main factor in cell-mediated immunity. B lymphocytes are responsible for producing antibodies (humoral immunity). T cells are classified by their surface antigens, and techniques exist for quantifying types and subtypes of different T populations to identify which functions are depressed. Some agents interfere with the production, function, or life span of T and B lymphocytes.

Substances known to interfere with the immune system include *(a)* polyhalogenated aromatic compounds, such as dioxins; *(b)* metals, including mercury; *(c)* pesticides; and *(d)* air pollutants, such as oxides of nitrogen and sulfur. Tobacco smoke has constituents that are immunotoxic. Mercury causes autoimmune damage and glomerulonephritis in rats due to depletion of the $RT6^+$ subpopulation of T-lymphocytes.

Sensitizers or allergens induce an increased immune response. The main target organs are the skin and the respiratory system. Nickel and poison ivy (Rhus) contact dermatitis are common examples of such skin sensitization. (See Chapter 22.) Occupational asthma reflects sensitization of the lung and airways to aerosols. (See Chapter 18.)

Reproductive Effects

The processes of gametogenesis, fertilization, implantation, embryogenesis, and organogenesis, and postnatal development are complex and subject to many environmental insults. Major errors incompatible with life generally result in spontaneous abortion (miscarriage), which can be viewed as a quality-control procedure. All stages are vulnerable to chemical hazards, including the failure to form gametes, such as azoospermia, and formation of abnormal gametes. Dibromochloropropane (DBCP), which causes testicular toxicity, can eliminate or reduce the number of sperm. (See Box 20-1 in Chapter 20.) Many other chemicals, such as lead, have also been implicated in toxicity to the male reproductive system, including interfering with spermatogenesis, semen quality, erection, and libido. The list of chemicals affecting the female reproductive system includes cancer chemotherapeutic agents, other pharmaceuticals, metals, insecticides, and various industrial chemicals. (See Chapter 20.)

Endocrine Disruptors

In the past decade, there has been an intense research and policy focus on endocrine disruption. Endocrine disruptors can mimic hormones, leading to overactive endocrine functions, or can bind with high affinity to endocrine receptors, thereby inhibiting normal endocrine functions.[7] Chemicals can lead to overexpression or underexpression of genes governing hormone or receptor synthesis, and chemicals can exert effects on target tissues influencing their response. The potency of these compounds is influenced by environmental persistence and bioamplification, bioavailability, and binding affinities. Endocrine disruption was well-recognized by the 1970s, when the ability of DDT to alter estrogen metabolism through enzyme induction was described. Although major concerns were then raised regarding the effect of chemicals on human reproductive function, adverse effects in a wide range of animals have since been clearly demonstrated, including effects on development, maturation, and reproduction, as well as cancer.[8]

Many endocrine disruptors occur naturally in vegetables (phytoestrogens), including a group of isoflavonoid and lignin polycyclic compounds. While there is concern about their interference with reproduction and development, their beneficial features are being exploited. One isoflavenoid, coumesterol, antagonizes estrogen during embryonic development, leading to reproductive abnormalities in behavior and hormone function. Others, such as genistein, protect against *(a)* certain hormone-dependent breast cancers by competing with estrogens; and *(b)* other cancers by inhibiting proliferation, differentiation, or the vascular supply. The bioengineered yeast estrogen screen (YES) is being used to screen for the endocrine disruptor action of xenobiotics.

Teratogenesis

Development from conception to birth involves a remarkable sequence of carefully timed interactions among cells through chemical signals,

which results in the differentiation of a few primordial embryonic cells into different tissues and organs. Cells multiply, migrate, connect, and often die, to be replaced by other cells—a necessary part of complex developmental biology. Chemicals may interfere with morphogenesis in varying ways. They may inhibit necessary signals or alter the timing of signals so that cells migrate and differentiate before the appropriate time. The effect of a particular chemical depends on the stage of embryogenesis and fetogenesis, as well as dose. Different chemicals cross the placental barrier with different efficiencies.

Chemicals may have no effect, produce subclinical alterations, or cause major birth defects or fetal death. For example, some effects on the developing nervous system from lead or methylmercury may disrupt the migration, maturation, and connections of nerve cells, leading to a viable infant with cognitive impairment. This impairment may only be apparent when the child has learning difficulties in school or is subjected to psychometric testing. Recognition of this phenomenon has given rise to the field of *behavioral teratology*, which focuses on studying the impact of fetal exposure to lead or alcohol on development and behavior.

Approximately 3% of live births have detectable congenital abnormalities. In general, chemical exposure prior to implantation is likely to be lethal. Exposure during organogenesis begets birth defects or embryolethality. Later in fetal life, there can be intrauterine growth retardation, fetal death, or functional changes that interfere with birth or postnatal development.

TOXICOLOGIC CONSIDERATIONS IN CLINICAL EVALUATION

Occupational and environmental exposures to toxic chemicals occur frequently, and adverse health effects are not rare. Clinicians are frequently confronted by patients who have symptoms that the patients ascribe to, or suspect are caused by, some chemical or event. It is easy for a clinician to be skeptical, especially when histories are complex and exposures uncertain. However, an initial high index of suspicion is often necessary to determine that an illness is caused by occupational or environmental factors. Many chemical exposures have both specific endpoints (such as bone marrow depression due to benzene) and nonspecific endpoints (such as light-headedness due to benzene). Some effects are acute, whereas others develop only after long periods of exposure.

First, one must establish whether the reported symptoms are typical of, or even likely to be associated with, the putative exposure. If not, one is likely to look for a different causal explanation, which may often be a different chemical or biologic agent. "General causation" is the process of determining whether a chemical can cause a given adverse health effect.

Second, one must establish exposure. A careful history can often confirm exposure. If exposure is recent or ongoing, clinical testing may reveal a biomarker of exposure. In addition, environmental measurements can be made to document the presence of the chemical. However, often many months—and many physician visits—have passed before the patient reaches a clinician who understands occupational and environmental disease. (By this time, the trail may be cold and only the history is available to provide information.) The clinician must then determine whether the specific individual's illness was due to this chemical (special causation).

For toxicity due to metals, one can often use chelation to help determine specific causation, given the propensity of metals to bind to sulfhydryl (–SH) groups. A variety of drugs with high concentrations of sulfur can be used to circulate through the body and bind any metal encountered. This can be used in a provocative mode to try to extract stored metal from prior exposures. A baseline urine measurement is obtained; then the chelator is administered (usually intramuscularly or intravenously, although some oral medications, such as DMSA, are now available). Urine is then measured, usually at 12-, 24-, and 72-hour intervals. If there is an excessive body burden of the metal, there should be a rapid increase in excretion of the metal, followed by a gradual return to baseline. Although chelation can be useful, it is more often misused by clinicians, who fail to get a baseline and then compare the concentration in the provoked urine to standard levels for unprovoked urine—resulting in false-positive results.

Biomarkers

A biomarker can be measured, usually in blood or urine, to provide information about exposure or pathophysiologic responses to toxic substances (Table 25-3). There are numerous applications of biomarkers in estimating internal dose. They can be defined as endpoints in a dose–response assessment. Some biomarkers reflect exposure, some reflect damage, and some can identify susceptibility. DNA adducts are biomarkers of exposure to carcinogens or mutagens; the carcinogenic polycyclic aromatic hydrocarbon, benzo[a]pyrene, forms a specific adduct with DNA. Although adduct formation is believed to be part of carcinogenesis, and smokers who have high exposure to benzo[a]pyrene have an increased rate of adduct formation, this marker has not had sufficient predictive value to support widespread use. Familiar biomarkers include blood lead level (a biomarker of exposure) and zinc protoporphyrin (a biomarker of effect from lead exposure). (The Board on Environmental Studies and Toxicology of the National Research Council has a Committee on Biologic Markers, which has published monographs on markers in pulmonary toxicology, reproductive toxicology, urinary toxicology, and immunotoxicology.)

Table 25-3. Illustrative Examples of Biomarkers

Biomarkers of Exposure
Specific chemical agents
Metabolites
DNA adducts
Biomarkers of Effect
Male reproductive disorders
Sperm motility
Semen quality
Müllerian-inhibiting factor
Chromosomal aberrations
Female reproductive disorders
Chorionic gonadotropin assay
Urinary progesterone metabolites
Pulmonary disorders
Pulmonary function testing
Airway reactivity (challenge tests)
Pulmonary cytology
Immunology disorders
Immunoglobulin levels
Lymphocyte ratios (T and B cells)
T-helper cells
T-suppressor cells
Natural killer cells
Lymphocyte functional assays
T-cell-dependent antibody response
Plaque-forming assays
Lymphocyte proliferation tests
Interleukin-2 activity
Specific receptor expression assays
Macrophage/leukocyte respiratory burst response
Lead poisoning
Zinc or erythrocyte protoporphyrin
Delta amino levulinic acid in urine
Delta amino levulinic acid dehydratase activity

CAUSALITY: ENVIRONMENAL CHEMICAL EXPOSURE AND HEALTH EFFECTS

An important role for the clinician, the epidemiologist, and the industrial hygienist is to identify a causal relationship between a chemical exposure and an adverse health effect. Laboratory experiments using sound experimental design can contribute to causality assessment, but health professionals need to make causal inferences with incomplete data. This is especially challenging for conditions with long latency periods or where hazardous substances are part of a mixture. In addition, the health effect may not be specific since many illnesses manifest as a complex of many symptoms or signs, each of which has multiple causes. Simply defining what the health effect is can be time-consuming and frustrating, and establishing specific causation may be impossible.

Establishing a scientific basis for causality often relies on the Bradford Hill "perspectives," guidelines which were presented by Sir Austin Bradford Hill, a British statistician, in the mid-1960s (Table 25-4).[9] These nine items have been widely cited—and often miscited. To establish general causation does not depend on meeting all nine perspectives, only temporality—the cause must precede the effect—is essential. (See also Chapter 24.)

Table 25-4. Bradford Hill's (1965) Perspectives for Establishing a Causal Relationship between an Exposure and a Disease

Criterion	Explanation	Application
Strength	First on his list is "strength of association," "enormous" excess of a disease in a particular industry. Scrotal cancer mortality in chimney sweeps was 200 times greater than other workers. Lung cancer death rate in smokers is 10 times greater than nonsmokers.	Useful. Hill cautioned that even a small relative excess might indicate causation when there is a large exposed population.
Consistency	"Has it been repeatedly observed by different persons, in different places, circumstances and times?"	Important, but Hill cautioned that inconsistency did not refute causation
Specificity	Association of a particular disease with a particular type of work	We must not, however, overemphasize the importance. Hill linked specificity to strength of association. If one outcome showed a much stronger association than others, it could be considered specific.
Temporality	Which comes first, exposure or outcome? Does work cause a disease or do people with a predilection for that kind of work have a susceptibility to the disease?	The only essential standard: that the putative cause precedes the outcome
Biological gradient	Dose–response is helpful if the "association is one which can reveal a biological gradient"	Dose–response is helpful for most toxic reactions, but idiosyncratic sensitivities will not follow the typical dose–response curve
Plausibility	"It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand."	Is it biologically realistic; do we have a known mechanism? But we must take into account that there will always be a first case.
Coherence	"The cause-and-effect interpretation of our data should not seriously conflict with generally known facts…." Also includes histologic verification of preclinical conditions in similarly exposed workers.	Very similar to plausibility
Experiment	Does a preventive action reduce the frequency of the association? "The strongest support for the causation hypothesis may be revealed."	An effective intervention is good evidence.
Analogy	"With the effects of thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy."	Based on what we know, we may accept by analogy, even before definitive evidence accumulates.

However, even when scientists and clinicians believe they understand the probable cause of a work-related condition, courts may require different interpretations for toxic tort cases. In addition, different judicial jurisdictions have different standards of evidence and different criteria for establishing causation and liability. "Reasonable probability" that A caused B is the standard in some jurisdictions, while "more probable than not" is the standard in others. "But for" may require a determination that without exposure to A, health effect B would not have occurred. In many states, workers' compensation requirements are less stringent than those in tort law. In New Jersey, for example, an exposure that causes, aggravates, or accelerates a condition is eligible for workers' compensation. In the future, it seems likely that experts will be asked to estimate attributable risk, so that costs can be assigned to different parties in proportion to their contribution to causation of an illness. Finally, in some cases, causation is assumed unless proven otherwise. For example, the U.S. Congress required the Veterans Administration to give veterans the benefit of the doubt in cases involving herbicide exposure during the Vietnam War. Under this mandate, certain diseases, such as soft tissue sarcoma, in exposed veterans are presumed by the Veterans Administration to be related to herbicide exposure and therefore qualify for compensation.

TOXICITY TESTING

A wide variety of systems and paradigms are used to test chemicals to predict their effects on human health or the environment. Under the Toxic Substances Control Act, new chemicals must undergo extensive safety testing. (See Chapter 30.) It is important to select the appropriate animal model or in vitro test system and to have well-chosen controls, which can include positive controls (those known to readily manifest a specific endpoint). Animal researchers need to choose the appropriate species, genetic strain, gender, and age as well as exposure route. The dosage schedule may be single or multiple. It may be acute, subchronic, or chronic. Duration of a study should be longer than the longest expected latency period. Dosages must be chosen to span the suspected threshold, if one is thought to exist. The route of administration should be relevant to natural conditions of exposure.

Toxicity testing must be subject to good quality assurance and quality control procedures. Quality assurance includes using trained (and, in some cases, certified) personnel, buying and maintaining appropriate equipment and standards, maintaining laboratory hygiene and avoiding cross-contamination, documenting procedures, maintaining records, and participating in interlaboratory testing programs. Quality control refers to laboratory procedures of calibration using blanks and known standards, as well as running replicate samples and analyzing spiked samples (those to which a known quantity of a specific chemical has been added).

Good laboratory practices describe animal care, dosing, and data management. Many commercial laboratories have been found to have faulty laboratory procedures, especially for documenting exposure, side effects, and outcomes.

The National Toxicology Program (NTP), operated by the National Institute of Environmental Health Sciences (NIEHS), sponsors long-term rodent studies to detect the carcinogenic and other toxic properties of chemicals. Chemicals are selected on the basis of data needs of governmental agencies and in response to public input. The standard protocol is two species (rat and mouse), both sexes, and a minimum of 50 individuals for each category, with oral dosing over a 2-year "life span." These 2-year bioassays can provide information on metabolism; genetic, reproductive, and developmental toxicity; and toxic effects on various organ systems. NTP bioassays screen new chemicals for carcinogenic activity and classify them with respect to human carcinogenicity. However, their main application has been to provide tumor incidence data in risk assessment.

ANIMAL WELFARE AND ANIMAL RIGHTS

Animal studies have played an important role in human toxicology and in the development of drugs that are safe for humans and for animals as well. Advances in toxicology have included the exploration of alternative testing procedures, including reducing the number of animals, using animals other than mammals, and developing in vitro techniques. Toxicologists have become increasingly attentive to animal welfare, due to *(a)* the recognition that reducing stress for animals makes them more reliable subjects, and *(b)* pressure from animal-rights activists, who assert that animals have intrinsic rights that, in the extreme, should protect them from any and all use in experimental research. The Animal Welfare Act, administered by the Animal and Plant Health Inspection Service of the U.S. Department of Agriculture, applies to all mammals, except mice and rats. Toxicologists generally agree that experimental animals should not be exposed to unnecessary stress, discomfort, or pain. Researchers using animals must consider animal-care guidelines. Most universities and other animal-testing organizations have animal-use committees that review all research protocols to minimize unnecessary stress and pain, inspect conditions under which animals are kept, and assure availability of veterinary care.

The concern over animal welfare reaches its peak when primates are used. Primates are expensive to acquire and maintain: Most primate studies can afford only a few animals that often live under unnatural and extremely stressful conditions. The capturing of many primates for medical research and industrial applications has had a drastic impact on the survival of several species in the wild.

The assumption that primates are the best models for humans makes sense, when cognitive performance is studied, but it does not take into account great differences in diet, since most primates are herbivorous. Thus, extrapolation from primates to humans is not always more appropriate than extrapolation from other animal models. Statistical interpretation of primate research is thwarted by small sample sizes, and, in some cases, by reuse of the same animals in sequential experiments. Over the past two decades, many primate laboratories have been closed, and, with few exceptions, primate research can be expected to play a diminishing role in future toxicology.

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26

Occupational and Environmental Hygiene

Thomas J. Smith and John D. Meeker

Occupational hygiene (industrial hygiene) is the environmental science of anticipating, recognizing, evaluating, and controlling health hazards in the working environment with the objectives of protecting workers' health and well-being and safeguarding the community at large. It encompasses the prevention of chronic and acute health conditions due to hazards posed by physical, chemical, and biological agents, as well as stress in the occupational environment and concern for the outdoor environment.

Environmental hygiene is the science of anticipating, recognizing, evaluating (including conducting surveillance), and controlling health hazards in the general environment with the objectives of protecting health and well-being for individuals and communities. Environmental hygiene includes the traditional environmental health components of public health—such as sanitation, waste management, and controlling contamination of air, water, and soil—as well as the newer problems of trace residues of hazardous chemicals in food and drinking water (Chapters 8 and 9), global climate change (Chapter 5), and the built environment (Chapter 39).

For example, an occupational hygienist determines the composition and concentrations of air contaminants in a workplace where there have been complaints of eye, nose, and throat irritation, and determines whether the contaminant exposures exceed the Occupational Safety and Health Administration (OSHA) permissible exposure limits or other national limits. If the problem is new and appears to be the result of airborne materials, which might be determined in consultation with a physician or an epidemiologist, then the hygienist would be responsible for selecting *(a)* the techniques used to reduce or eliminate the exposure, such as installing exhaust ventilation around the source of the air contaminants and isolating it from the general work area, and *(b)* performing follow-up sampling to verify that the controls were effective.

Most occupational hygienists have earned either a bachelor's degree in science or engineering or a master of science degree in industrial hygiene. Occupational hygienists tend to specialize in specific technical areas because the scope of the field is wide. Occupational hygienists must work with physicians to develop comprehensive occupational health programs and with epidemiologists to perform research on adverse health effects. It has been traditional to separate occupational hygiene and occupational safety, but the recent trend has been to broaden the training for each discipline to include that of the other. This has led to the specialty of risk management for evaluating and controlling all

types of workplace hazards. At present, occupational hygienists generally do not deal with mechanical hazards or job activities that can cause physical injuries, which are the responsibility of safety specialists. However, it is not uncommon for private companies to have a single individual responsible for both occupational hygiene and safety who has no formal training in either area. In some companies, there has also been a new trend for occupational hygienists to lead or be involved with environmental health and sustainability issues, emergency planning and response, and even security.

Most occupational hygienists work for large companies, consulting firms, or governmental agencies. A small number work for labor unions. For whomever they work, occupational hygienists unfortunately are often located in organizational units where they have little organizational power to bring about necessary changes. For example, hygienists who work for labor unions may be restricted in their access to the workplace for sampling and exposure measurements, which can limit their ability to assess and control hazards.

The closeness of working relationships between occupational hygienists and physicians varies. Some have close collaborative activities with an extensive exchange of information, while others operate with nearly complete independence and have little more than formal contact. A physician who is familiar with the workplace, job activities, and health status of workers in all parts of the process may be very helpful in guiding the occupational hygienist in assessing environmental hazards, and vice versa. Within a framework of multidisciplinary approaches, occupational hygienists and physicians should collaborate with safety specialists, workers in production units, staff members in personnel departments, worker representatives, and delegates of the health and safety committees. Where contact among these groups is minimal, many opportunities are lost for improving the effectiveness of health hazard control and the prevention of adverse effects.

It is important for occupational hygiene to be integrated into an overall program for occupational health without which the effectiveness of intervention strategies may be limited. An effective occupational hygiene program must have good working relationships with the production, personnel, and health and safety departments and strong support by upper management. Core activities of hazard anticipation, recognition, evaluation, and control must be well integrated with the day-to-day activities of the enterprise. There is no single organizational structure that is optimal.

ANTICIPATION, RECOGNITION, EVALUATION, AND CONTROL OF HAZARDS

Formal strategies for workplace assessment have not been well developed. The American Conference of Governmental Industrial Hygienists (ACGIH) monograph on exposure assessment, *A Strategy for Occupational Exposure Assessment* (see Further Reading), is focused on sampling and does not address program management issues. European Union (EU) regulations now require workplace assessment to identify occupational hazards. However, no formal mechanism has been established or validated to demonstrate its consistency. It is expected that EU regulations, when fully implemented, will be beneficial to small and medium-sized enterprises because they will create awareness and an expectation of controlling the problems identified. Although not designed as an occupational health assessment, the EU implemented an ambitious new regulation in 2007, called REACH, which addressed the registration, evaluation, authorization, and restriction of chemical substances. REACH, whose goal is to improve the safe handling of hazardous chemicals, requires manufacturers and importers of chemicals to compile more detailed information on the properties of substances and register this information in a centralized database, which is accessible to professionals and consumers. A requirement of REACH is progressive substitution of the most hazardous chemicals once suitable alternatives are identified.

Anticipation

Anticipation of hazards has become an important responsibility of the occupational hygienist. Anticipation refers to the application and mastery of knowledge that permits the occupational hygienist to foresee the potential for disease

and injury. The occupational hygienist should thus be involved at an early stage in planning of technology, process development, and workplace design. Consider the following example:

An electronics company was developing a new process for making microcomputer chips. The process involved dissolving a photographic masking agent in toluene and then spraying the mask on a large surface covered with chips. The company's occupational hygienist noted that this would expose the workers to potentially high airborne levels of toluene. She suggested they substitute xylene, which has a lower vapor pressure, and modify the process to use smaller amounts of solvent, which would reduce the amount of hazardous waste generated by the process.

It is common that process engineers or industrial researchers will propose using hazardous materials or will not consider the interaction of the worker and the process or machine. Consequently, hygienists can prevent many problems that will be expensive to fix after installation by reviewing early plans and findings of pilot plant experiments.

Identification of hazards may be most easily accomplished using an overview of the production process that describes the complete flow from raw material to final product. Production can be subdivided into its component unit processes. In this stepwise fashion, the processes with hazards can be recognized, worker exposures evaluated, and exposures in nearby areas assessed. Examples of some common unit processes and their hazards are shown in Table 26-1. This general approach and the hazards of a wide range of common industrial processes are discussed in more detail in Burgess' *Recognition of Health Hazards in Industry: A Review of Materials and Processes* (see Further Reading).

This approach can be illustrated by considering a small company that manufactures toolboxes from sheets of steel by a six-step process: *(l)* sheets of steel are cut into the specified shape; *(2)* sharp edges and burrs are removed by grinding; *(3)* sheets are formed into boxes with a sheet metal bender; *(4)* box joints are spot welded; *(5)* boxes are cleaned in a vapor degreaser in preparation for painting; and *(6)* boxes are painted in a spray booth. Production steps 2, 4, 5, and 6 use unit processes with known sources of airborne emissions, and their hazards (Table 26-1) should be evaluated. Exposures of workers involved with steps 1 and 3 may need to be evaluated because they may be located near enough to the operations with hazards to have significant exposure. Additional hazards may also be present depending on the specific processes and equipment used in the various steps. For example, worker exposures to nonionizing radiation and metal fumes may need to be considered if a high-powered laser is used to cut the steel in step 1.

The design of job tasks and an individual's work habits can both have an important influence on exposures. For example, a furnace tender's exposure to metal fumes will depend on the length of tools used to scrape slag away from the tapping hole in the furnace and on the instructions for performing the task. Lack of adequate tools or sufficient operating instructions may cause excessive exposure to fumes emitted by molten materials. Similarly, the furnace tender, who is positioned close to the slag as it runs out of the furnace, may receive a much higher exposure to fume than a co-worker who stands farther away from the molten slag. Therefore, an important part of an evaluation is the observation of work practices used in hazardous unit processes.

Recognition

Recognition of problems in a new or unfamiliar workplace generally requires that the occupational hygienist engage in collection of background information on production layout, processes, and raw materials. This usually begins with a visit to the workplace to become familiar with the production processes and their hazards. These visits are crucial for detecting unique aspects of the workplace that may strongly affect exposures. Information is collected on the following:

- Types, composition, and quantities of substances and materials, including raw materials, intermediate products, and additives
- Design of work processes and tasks
- Emission sources
- Design and capacity of ventilation systems or other control measures

Table 26-1. Common Unit Processes and Associated Hazards by Route of Entry*

Unit Process	Route of Entry and Hazard
Abrasive Blasting	
Surface treatment with high velocity materials, such as sand, steel shot, pecan shells, glass, or aluminum oxide	Inhalation: silica, metal, and paint dust Noise
Acid/Alkali Treatments	
Dipping metal parts in open baths to remove oxides, grease, oil, and dirt Acid pickling (with hydrochloric acid, nitric acid, sulfuric acid, chromic acid, or a mixture of nitric acid and hydrogen fluoride) Molten caustic descaling	Inhalation: acid mist with dissolved metals Skin contact: burns and corrosion, hydrofluoric acid toxicity Inhalation: nitrogen dioxide, acid mists Inhalation: smoke and vapors Skin contact: burns
Blending and Mixing	
Powders and/or liquid are mixed to form products, or undergo reactions	Inhalation: dusts and mists of toxic materials Skin contact: toxic materials
Cleaning	
Application of cleansers, solvents, and strong detergents to clean surfaces and articles; and operation of devices to aid cleaning such as floor washers, waxers, polishers, and vacuums	Inhalation: dust, vapors Skin contact: defatting agents, solvents, strong bases
Crushing and Sizing	
Mechanically reducing the particle size of solids and sorting larger from smaller with screens or cyclones	Inhalation: dusts and mists of toxic materials Noise
Degreasing	
Removing grease, oil, and dirt from metal and plastic with solvents and cleaners Cold solvent washing (clean parts with ketones, cellosolves, and aliphatic, aromatic, and stoddard solvents Vapor degreasers (with trichloroethylene, methyl chloroform, ethylene dichloride, and certain fluorocarbon compounds)	Inhalation: vapors Skin contact: dermatitis and absorption Fire and explosion (if flammable) Metabolic: carbon monoxide formed from methylene chloride Inhalation: vapors; thermal degradation may form phosgene, hydrogen chloride, and chlorine gases Skin contact: dermatitis and absorption
Electroplating	
Coating metals, plastics, and rubber with thin layers of metals, such as copper, chromium, cadmium, gold, or silver	Inhalation: acid mists, hydrogen cyanide, alkali mists, chromium, nickel, cadmium mists Skin contact: acids, alkalis Ingestion: cyanide compounds
Forging	
Deforming hot or cold metal by presses or hammering	Inhalation: hydrocarbons in smokes (hot processes), including polyaromatic hydrocarbons, sulfur dioxide, carbon monoxide, oxides of nitrogen, and metals sprayed on dies, such as lead and molybdenum Heat stress Noise
Furnace Operations	
Melting and refining metals; boilers for steam generation	Inhalation: metal fumes, combustion gases, such as sulfur dioxide and carbon monoxide Noise from burners Heat stress Cataracts from infrared radiation

(*Continued*)

Table 26-1. Common Unit Processes and Associated Hazards by Route of Entry* (Continued)

Unit Process	Route of Entry and Hazard
Grinding, Polishing, and Buffing	
An abrasive is used to remove or shape metal or other material	Inhalation: toxic dusts from both metals and abrasives Vibration from hand tools Noise
Industrial Radiography	
X-ray or gamma ray sources used to examine parts of equipment	Radiation exposure
Machining	
Metals, plastics, or wood are worked or shaped with lathes, drills, planers, or milling machines	Inhalation: airborne particles, cutting oil mists, toxic metals, nitrosamines formed in some water-based cutting oils, endotoxin Skin contact: cutting oils, solvents, sharp chips Noise
Materials Handling and Storage	
Conveyors, forklift trucks are used to move materials to/from storage	Inhalation: carbon monoxide, exhaust particulate, dusts from conveyors, emissions from spills or broken containers
Mining	
Drilling, blasting, mucking to remove loose material, and material transport	Inhalation: silica dust, nitrogen dioxide from blasting, gases from mine can lead to low oxygen levels Explosion hazards from methane and airborne combustible dusts Vibration stress Heat stress Noise
Painting and Spraying	
Applications of liquids to surfaces, for example, paints, pesticides, coatings	Inhalation: solvents as mists and vapors, toxic materials Skin contact: solvents, toxic materials
Repair and Maintenance	
Servicing malfunctioning equipment; cleaning production equipment and control systems	Inhalation: dusts, vapors, and gases from the operation Skin contact: grease, oil, solvents
Quality Control	
Collection of production samples, performance of test procedures that produce emissions	Inhalation: dusts, vapors, and gases Skin contact: solvents
Soldering	
Joining metals with molten lead or silver alloys	Inhalation: lead or cadmium particulate (fumes) and flux fumes
Welding and Metal Cutting	
Joining or cutting metals by heating them to molten or semi-molten state Arc or resistance welding Flame cutting and welding Brazing	Inhalation: metal fumes, toxic gases and materials, flux particulate, etc. Noise: from burner Eye and skin damage from infrared and ultraviolet radiation

* Health hazards may also depend on the toxicity and physical form of the materials used. For further information, see Burgess WA. Recognition of health hazards in industry: a review of materials and processes (2nd ed.). New York: Wiley and Sons, 1995.

Flow visualization with smoke tubes (glass tubes with a packing that produces dense white smoke when air is forced through it) can give qualitative information on effectiveness of local exhaust or process ventilation. Work practices, worker position relative to sources, and task duration can be recorded. Information can be collected on cleaning routines and performance as well as tidiness, which are important determinants of exposure. Consider the following example:

Farmworkers were experiencing episodes of depressed blood cholinesterase levels from organophosphate exposure despite their observing the required waiting times before reentry into sprayed fields and wearing long-sleeved shirts and gloves to prevent skin contact. The pesticide had a very low vapor pressure so there was no significant inhalation exposure. However, it was known that environmental moisture decomposes this type of pesticide. Since the weather was very dry during these episodes, there was concern that the pesticide was not decomposing as rapidly as expected. Consequently, despite the skin protection, there could still be sufficient skin absorption of the pesticide to depress cholinesterase levels. Skin sampling with patches showed that fine dust was sifting through the cloth of the shirt sleeves and depositing much pesticide on workers' arms. The problem was solved by extending the standard reentry times.

If the initial appraisal cannot definitely rule out a hazard, a basic survey has to be performed to provide quantitative information about exposure of workers. Particular account has to be taken of tasks with high exposure. Sources of information are as follows:

- Earlier measurements
- Measurements from comparable installations or work processes
- Reliable calibrations or modeling based on relevant quantitative data
- Air sampling measurements to determine the range of exposures

Sampling may show that sensory impressions underestimate or overestimate exposures; for example, the odor threshold for most solvents is well below the level at which they present a toxic exposure hazard.

If this information is insufficient to enable valid comparisons to be made with the limit values, a full-scale survey must be performed. The full-scale survey examines all phases of workplace activities—both normal activities and abnormal or infrequent ones, such as maintenance, reactor cleaning, or simulation of malfunctions. The survey activities may take several weeks or months in a complex manufacturing or chemical plant.

Evaluation

For the evaluation of recognized or suspected hazards, the hygienist uses techniques based on the nature of the hazards, emission sources, and the routes of environmental contact with the worker. For example, air sampling can show the concentration of toxic particulates, gases, and vapors that workers may inhale; skin wipes can be used to measure the degree of skin contact with toxic materials that may penetrate the skin; biological samples (blood or urine) can sometimes provide data where there are multiple routes of entry; and noise dosimeters record and electronically integrate workplace noise levels to determine total daily exposure. Both acute and chronic exposures should be considered in the evaluation because they may be associated with different types of adverse health effects.

The workplace is not a static environment: Exposures may change by orders of magnitude over short distances from exposure sources, such as welding, and over short time intervals because of intermittent source output or incomplete mixing of air contaminants. In addition, operations and materials used or produced commonly change, as do job titles and definitions. The nature of these changes and their possible effects must be recognized and taken into consideration by the occupational hygienist.

All monitoring programs for both acute and long-term problems should be structured with a clear focus on the individual's sources of exposures and the ultimate objective to estimate dose. Monitoring organized solely around compliance with today's standards will probably be unable to answer tomorrow's questions about hazards

associated with personal exposures. Adverse health effects can be associated with exposures below regulatory standards that were set decades ago. The effects of environmental controls, such as ventilation and personal protective equipment that intervene between the emission source and the worker must also be considered.

The occupational hygienist's decision on whether a hazard is present is based on three sources of information:

1. Scientific literature and various exposure limits, such as *(a)* the threshold limit values (TLVs) of ACGIH,* a set of consensus standards developed by occupational hygienists, toxicologists, and physicians from governmental agencies and academic institutions; *(b)* the World Health Organization (WHO) recommendations†; or *(c)* in some instances, the National Institute for Occupational Safety and Health (NIOSH) proposed health-based recommended exposure limits for select agents to be considered by OSHA in the standard-setting process‡
2. The legal requirements of OSHA (in some cases, these are less stringent than the TLVs because the TLVs have been updated) or regulatory agencies of other countries[1]
3. Problems identified by other health professionals who have examined exposed workers and evaluated their health status

In cases where health effects are present but exposures do not exceed the TLVs, WHO recommendations, or OSHA or other national requirements, prudent hygienists and others may conclude that there is a relationship between adverse health effects and workplace exposures if evidence supports such a relationship. Exposure limits are designed to prevent adverse effects in most exposed workers, but they are not absolute levels below which adverse effects cannot occur. The supporting data for many exposure limits are sometimes insufficient, out of date, or based too much on evidence of acute toxic effects and not enough on evidence of carcinogenicity, mutagenicity, teratogenicity, or other lagged or chronic conditions.

Control

Once a hazard is identified and the extent of the problem evaluated, the hygienist next designs a control strategy or plan to reduce exposure to an acceptable level. Such controls may have two phases, an immediate response with personal protective equipment (PPE) to quickly reduce the hazard, and an engineering follow-up to control the problem more effectively, including:

1. Changing the industrial process or the materials used to eliminate the source of the hazard, such as changing to clean technologies
2. Isolating the source and installing engineering controls, such as ventilation systems
3. Using administrative controls to limit the duration of exposure a worker receives, or, as a final resort, requiring the development of a formal program for the prolonged use of PPE. Administrative controls are less reliable because they depend on enforcement by managers and conscientious application by workers.

In designing control strategies, one should also consider the environmental impact (outside the occupational setting) of emissions, waste, storage, spills, and leaks. Action can be taken at the levels of processes, materials, components, systems, and the entire workplace. Education of workers and supervisors is essential. Both workers and supervisors must understand the nature of hazards and support efforts to control or eliminate them. Implementation of control measures should be supervised and their efficacy evaluated.

Automobile manufacturers have been concerned about the hazards of coolants used in machining and grinding operations. Workers have complained of skin and inhalation problems associated with exposures to liquids splashed on their skin and to airborne mists that they breathe. In the past, controls were implemented

* Can be obtained from: ACGIH, 6500 Glenway Avenue, Building D-5, Cincinnati, OH 45211.

† Can be obtained from WHO, Avenue Appia 20, 1211 Geneva 27, Switzerland.

‡ Can be obtained at http://www.cdc.gov/niosh/pubs/criteria_date_desc_nopubnumbers.html

based on hypotheses about causal factors, without investigations of specific causes for exposures. Some hypotheses were later found to be incorrect and control was incomplete, despite substantial expenditures. Inhalation exposures were only partially controlled by local exhaust ventilation and by enclosure of processes. Exposures were found to be associated with symptoms and reduced pulmonary function. Analysis of coolants revealed that material safety data sheets (MSDSs) were inaccurate, and the machining department did not know about some of the hazardous materials that were being used. Investigations are ongoing to determine what additional controls will be needed to further reduce the exposures, including substitution of components in coolants and better control of microbial contaminants with filtration and other techniques.

This case demonstrates that controlling hazards in large, complex manufacturing operations is very frequently a stepwise process. Control strategies are most effective when based on complete knowledge of the nature of problems.

After hazards are controlled, the hygienist may recommend a hazard surveillance program to ensure that controls remain adequate. Material components of inexpensive raw materials can change without notice, thereby generating problems. This type of environmental surveillance is most effective when performed with a medical surveillance program designed to detect subtle health effects that may occur at low levels of exposure.

The following sections illustrate how assessment and control techniques are utilized. The approach for toxic materials is used as a paradigm, which can also be used for other environmental hazards, including noise, vibration, ionizing and non-ionizing radiation, temperature extremes, poor lighting, and infectious agents.

TOXIC MATERIALS

Exposure Pathways

The health hazard of a given exposure to a toxic material depends on the toxicity of the substance and on the intensity and duration of contact with the substance. Thus, adverse effects can result from chronic low-level exposure to a substance or from a short-term exposure to a dangerously high concentration of it. However, the pharmacologic mechanisms for acute and chronic effects may differ. Occupational hygienists are concerned with both long-term, low-level exposures and brief acute exposures.

In assessing a given hazardous material, the hygienist determines the route of exposure by which workers contact it and by which it may enter their bodies. There are four major routes of exposure: *(1)* direct contact with skin or eyes; *(2)* inhalation, with deposition in the upper or lower respiratory tract; *(3)* inhalation of particulate substances, with deposition in the upper respiratory tract and subsequent ingestion; and *(4)* direct ingestion from eating or drinking. In the workplace, several concurrent routes of exposure may occur for a toxic substance.

Inhalation of airborne particulates, vapors, or gases is, by far, the most common route of hazardous exposure and therefore occupies much of a hygienist's assessment and control activities. Skin absorption may be important if the substance is lipid soluble or the skin's barrier is damaged or otherwise compromised. Ingestion of contaminated food and drink is a problem, especially for particulate and liquid materials, whose degree of risk may depend on the worker's level of awareness of the hazard and personal hygiene habits, and on the availability of adequate facilities for washing and eating at the workplace. Contamination of cigarettes with toxic materials and their subsequent inhalation is also a problem for some substances.

For example, workers handling lead ingots are exposed to a low-level hazard from ingesting small amounts of lead by eating contaminated food or by inhaling small amounts of lead fumes from contaminated cigarettes. However, workers refining lead at temperatures above 800°F are exposed to a serious hazard from inhaling large amounts of lead fume if they work close to unventilated refining kettles for several hours daily. Workers handling liquid nitric acid are exposed to the hazard of direct contact with the liquid on their skin, but they may also be exposed to a respiratory hazard from inhaling acid mist generated by an electroplating process that uses nitric acid. In these two examples, lead and nitric acid cause different types and magnitudes of hazards because their physical forms vary: for

lead, solid material and small-diameter airborne particulates; for nitric acid, liquid and airborne droplets.

Anticipation and Recognition

The first problem the hygienist faces in evaluating an unfamiliar workplace for toxic hazards is identification of toxic materials. In many cases, such as a lead smelter or pesticide manufacturing process, emission sources for toxic materials are clearly evident. But even in these examples some hazards may not be evident without a careful examination of an inventory of the chemicals to be used or in use in the facility, including raw materials, by-products, products, wastes, solvents, cleaners, and special-use materials. Workers in lead smelters can also be exposed to carbon monoxide, arsenic, and cadmium; pesticide workers can be exposed to solvents. Relatively nontoxic chemicals can be contaminated with highly toxic ones; for example, low-toxicity chlorinated hydrocarbons used in weed killers, such as trichlorophenoxyacetic acid (2,4,5-T), may contain highly toxic dioxin, and, in some parts of the world, technical-grade toluene may contain highly toxic benzene. In some cases, toxic materials may not be hazardous to workers because exposures are adequately controlled.

Material safety data sheets, which list the composition of commercial products, are available from manufacturers and can be useful, but they are sometimes too general or out of date. Toxicity data on specific substances can be obtained by literature searches or by searches of toxicity data indices.

Since exposure to toxic substances can occur by contamination of food, drink, or cigarettes, the hygienist determines whether *(a)* eating and drinking facilities are physically separated from the work area, *(b)* facilities for washing are close to eating areas, and *(c)* sufficient time is permitted for workers to use these facilities. Protective clothing and facilities for showering after a work shift should also be provided. Workers' understanding of hazards from toxic materials they are using must also be assessed. Finally, the hygienist determines whether there are rules prohibiting eating, drinking, and smoking in areas with toxic substances, and whether these rules are being enforced.

Evaluation

Measurement Techniques

Direct-reading instruments have sensors that instantaneously detect air concentration of substances and often produce a reading on a dial or digital readout. Most direct-reading instruments can store data for 8-hour time profiles for later retrieval and display. All require careful calibration, adherence to use specifications and conditions, and maintenance to obtain accurate data. The detector tube is another type of direct-reading instrument often used in determining approximate concentrations of air contaminants. This simple device uses a small hand pump to draw air through a bed of reagent in a glass tube that changes color or develops a length of stain that is proportional to the concentration of a given gaseous air contaminant. The conventional tube is suitable for short-term sampling, such as for 10 minutes; however, short-term samples can misrepresent long-term average exposures. Tubes for 8-hour sample collection can measure time-weighted average (TWA) exposures. Detector tubes are manufactured under strict quality control and their degree of measurement uncertainty is specified. Consideration must always be given to interference from other substances, which usually is specified on the tube's data sheets.

Sample collectors that remove substances from the air for analysis in a laboratory may be a less expensive alternative to direct-reading instruments. Personal sampling is a common approach used by hygienists to obtain accurate and precise measurements of workers' exposures. Particulate contaminants are collected with filters, and gases and vapors are collected by solid adsorbents or liquid bubblers. The sampling apparatus is generally simple, consisting of a small air pump usually worn on a worker's belt, connected by tubing to a collector and attached to the worker's shirt at the neck. (Some gas and vapor collectors are passive, using diffusion instead of an air pump to move the contaminant into the sampler.) With the appropriate selection of a gas or particulate collector, or both combined in a sampling train, it is possible to measure the average concentration of an air contaminant in the worker's breathing zone during an 8-hour work shift.

Collection devices for toxic particulates may capture either total dust—that is, all particle sizes that can enter the collector, or only the respirable dust—that is, only particles that can penetrate the terminal airways and alveolar spaces (less than 4 μm). Total particulate samples are collected if the toxic substance causes systemic health problems, as lead and pesticides do. Respirable dust samples are collected if the particulate causes chronic pulmonary disease, such as pneumoconiosis. The type of sampler should be matched to the route of entry, type of effect, and target tissue.

Charcoal and other sorbent material packed into tubes have been the most common collectors for gases and vapors; a small amount of charcoal inside a small glass tube acts as an activated surface that retains nonpolar materials, such as benzene. These collectors are commonly used to measure inhalation exposures to solvents, such as vapor exposures of printers. The specific methods for chemicals are discussed in detail in the *NIOSH Manual of Analytical Methods*.

Passive or badge-type samplers are much more convenient to use for gas and vapor sampling than collectors requiring air pumps, are relatively inexpensive, and have better worker acceptance because they weigh less than pumps. After the sampling period is completed, the cover is replaced on the badge, the total exposure time is noted, and it is sent to a laboratory for analysis. Several passive samplers have well-documented sampling rates. They may surpass active samplers in accuracy. Contamination from liquid splashes during use must be avoided.

Sampling Strategy

The hygienist must design a sampling strategy that takes into account the types of hazards, variations in exposure, routes of exposure, and the uses for the data, such as risk assessment or source evaluation and control. The approach should enable most efficient use of resources.[2] Personal measurements are designed to reflect the accumulation of exposure from a variety of sources that a worker may encounter during a work shift. In some cases, exposure may occur only during certain operations. *Worst-case sampling* is the approach used when it is clear that high emissions from certain activities or sources will occur and it is decided that sampling will only be done during the period of highest exposure. This approach is used by OSHA.Workers in adjacent areas not directly involved with the air contaminant of interest are frequently found to have significant exposures because the air contaminant drifts into their work areas.

Sampling strategies are designed to assess variability in exposures and factors associated with this variability. Variability in exposure levels can be large due to day-to-day variation in work pattern, production rate, and differences in a process. Differences in personal work habits, wind velocity, and wind direction also can cause large variations. The exposed populations should be subdivided into smaller, well-defined groups of workers performing identical or similar tasks. Properly selecting subgroups reduces within-group variability so that measurement resources can be concentrated on the highest exposed groups, although these may be difficult to identify a priori. Single samples are generally avoided because it is difficult to know what one sample value represents. In addition, because workers have different work habits and techniques, there frequently are differences in average exposures among workers.[3] Several replicate samples on workers may indicate how large these differences are and how much the assumption of uniform mean exposure within groups is violated.

In addition to personal sampling, the occupational hygienist also uses *fixed-location sampling*. In this strategy, the sampler is set at a given location that has some useful relationship to a source of exposure. This type of sampling is advantageous because it can enable determination of features of the exposure that would be difficult with personal samples. For example, a large sampler can be used to *(a)* determine the particle size distribution of airborne dust in a work area or *(b)* provide sufficient airborne material for detailed chemical analysis if the composition of the contaminants is not known. These samplers can be very useful for identifying and characterizing sources of exposure and assessing the effectiveness of engineering controls. One must carefully select the sampling location and strategy for fixed-location sampling. In some cases, a combination of personal and fixed-location samples is used to completely describe a situation. For example, personal samples are used to

describe the highly variable exposures of steel workers tending a blast furnace, while fixed-location samples measure exposures to the uniform, well-mixed air levels they experience while waiting in the lunchroom for their next job assignments (for 2 to 4 hours per work shift).

Some large plants use continuous multipoint sampling of gases and vapors with central analysis. The monitors can be linked to alarms, so that rapid action can be taken if concentrations exceed specified limits. Continuous monitoring at stationary sites should be part of a total quality management process when exposures are near the allowable limits.

In some workplaces, the most important route of exposure is skin contact. This route of exposure is difficult to evaluate with environmental sampling because even if the amount of skin contamination can be determined, it is not possible to know how much of the contaminant has already entered the body, or would enter, given sufficient time.[4] Two principal sampling approaches are employed:

1. Cloth patches with impermeable backing can be used to cover given locations of skin, such as the forehead, back of the neck, back of the hands, and forearms, to measure the amount of contamination per unit area that resulted during a period of exposure.
2. Wipe sampling can be performed, in which an area of skin is washed with an appropriate nontoxic solvent to determine the quantity of contamination remaining on the skin after a period of exposure.

Both of these techniques have been used to estimate pesticide exposures of agricultural workers. Addition of a fluorescent whitening agent to the pesticide as a tracer allows visualization of contamination. Additionally, wipe sampling on surfaces can be used as a method to detect and control indiscriminate distribution of toxic materials that workers may contact throughout the workplace. This type of sampling is also useful in estimating the exposure of one person relative to another, or of one area relative to another. It is, however, difficult to know in absolute terms the quantity of contaminant that may penetrate the skin and cause an adverse health effect. Biologic monitoring is probably the best method for determining the intensity of skin exposures to a substance, if a biological monitoring test is available (Chapter 2).[5]

Some nonpolar substances, such as pesticides and solvents, may enter the body both via the respiratory tract and through skin contact. For these substances, both skin contact and air exposure must be evaluated to completely assess the risk. Biologic sampling that integrates these two routes of intake may be a practical necessity. Problems can be associated with biologic monitoring. First, some types of tests detect adverse effects, such as reduced red blood cell cholinesterase in pesticide-exposed workers, but they may show that excessive exposures have already occurred. Second, since there is a complex relationship between exposures and levels of compounds and metabolites in blood, urine, exhaled breath, and other biologic media, proper interpretation of biologic monitoring tests requires knowledge of the toxicokinetics of the specific agent and the temporal variations of a worker's exposure.[6] In many situations, biologic monitoring should only be used to verify that exposures have been controlled. Its use in detecting high exposures should be limited, such as when absorption is primarily through the skin.

It is almost never possible to evaluate ingestion as a route of exposure with sampling. Occasionally, samples of food and drink may be collected to assess the level of contamination; however, this type of exposure is likely to be extremely variable and episodic, so that occasional environmental sampling is usually an ineffective way of assessing exposure.

Exposure measurements on workers performing the same job under similar conditions commonly show substantial variation in mean exposure between workers. These differences are the primary limitation to what can be achieved with exposure controls. There are many reasons why differences might occur. First, individuals have differences in skill, training, and experience, which may lead them to perform a job with different techniques that affect personal exposure. Second, they may have differences in their level of concern about the hazards of the job and take more or fewer precautions to avoid exposure through the use of engineering controls or PPE. Differences among workers on these

factors are generally assigned to "work practices" and dismissed. As a result, there has been little systematic investigation of the nature of these differences, especially the behavioral components, and effective ways for intervening to reduce exposures.

Control

Substitution

Substances and materials that pose risks to impair health and safety should not be used if they can be substituted with something that is safer. Substitution is part of the concept of toxics use reduction and waste management. Toxics use reduction is a formal programmatic approach to examining the materials being used and produced in a workplace, identifying the hazards associated with each, and then developing strategies to reduce the overall burden of toxic materials used or produced. (More information can be found at http://www.turi.org/.) Potential benefits to health and safety have to be balanced against technological and economic consequences. This balance should include product properties, production process, environment, and reliability of supply. For example, toluene or xylene may be inexpensive, less-toxic replacements for benzene being used as a solvent, because they have similar chemical properties and may work as effectively as benzene. They also are less volatile and will produce fewer environmental emissions. Both are readily available commercially and unlikely to be affected by supply shortages. Regular auditing of use of substances and materials helps to identify opportunities for substitution and promotes ongoing toxics use reduction.

Limitation of Release and Build-up of Contamination

If substitution is not possible, then the next step is to attempt to control or limit releases, and to prevent the buildup of toxic materials in the worker's environment (Fig. 26-1). Local exhaust ventilation combined with source isolation will control process emissions. General room ventilation is used to prevent the buildup of hazardous concentrations in the work area from contaminants escaping local exhaust, from spills, or from fugitive emissions (from seals, valves, or pumps). An example of these two ventilation approaches is shown in Figure 26-2.

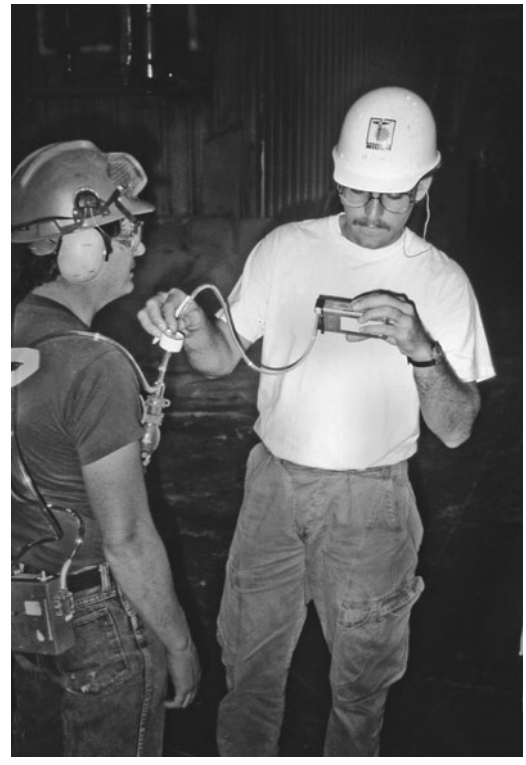

Figure 26-1. A National Institute for Occupational Safety and Health (NIOSH) industrial hygienist checks the airflow rate of a personal sampling pump to measure a worker's formaldehyde exposure in a fiberboard manufacturing plant. (Photograph by Aaron Sussell.)

Local exhaust systems surround the point of emission with a partial or complete enclosure and attempt to capture and remove the emissions before they are released into the worker's breathing zone. Figures 26-3 through 26-5 show several examples of local ventilation systems; various types, with differing degrees of effectiveness, are available. Unfortunately, it is not possible before installation to determine precisely the effectiveness of a particular system, although this is an area of active research. As a result, it is important to measure exposures and evaluate how much control has been achieved after a system is installed. Unless contaminant sources are totally enclosed, collection will only capture a percentage of total emissions. Release of smoke from smoke tubes at the point of contaminant

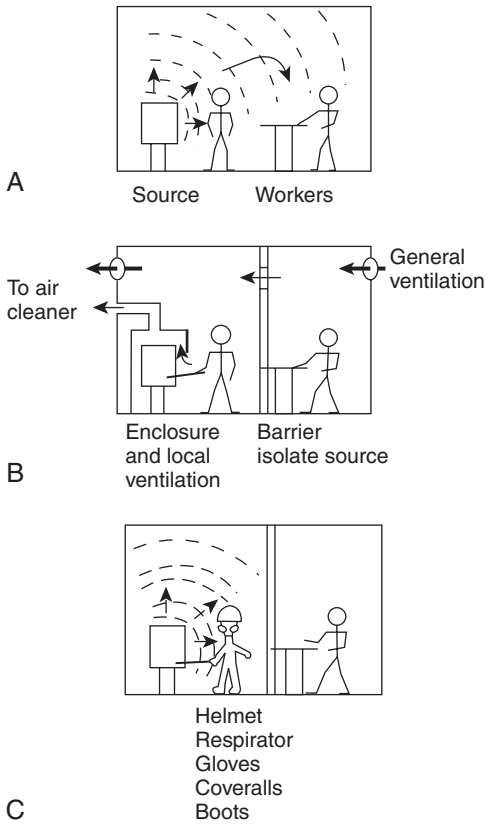

Figure 26-2. Examples of controls for airborne exposures. (A) Workers with primary and secondary exposure to source emissions. (B) Ventilation and source isolation to control exposures. (C) Personal protection and source isolation to control exposures. (Diagrams prepared by T. J. Smith, Harvard School of Public Health, Boston, Massachusetts.)

generation is a useful technique for visualizing the flow of air toward the exhaust. It may reveal if the distance to the exhaust is too large, if there are cross-drafts or strong air disturbances, or if the worker creates wakes, all of which greatly reduce the collection efficiency. A good system may collect 80% to 99%, but a poor system may capture only 50% or less. Careful maintenance must be performed on the system to maintain efficiency. Poor maintenance is probably most responsible for system failures.

The increasing cost of energy has made the practice of ventilating work areas with outside fresh air an increasingly expensive process; much work is being done on the design of systems that can recirculate decontaminated air or use heat exchangers so the heat value is not lost.

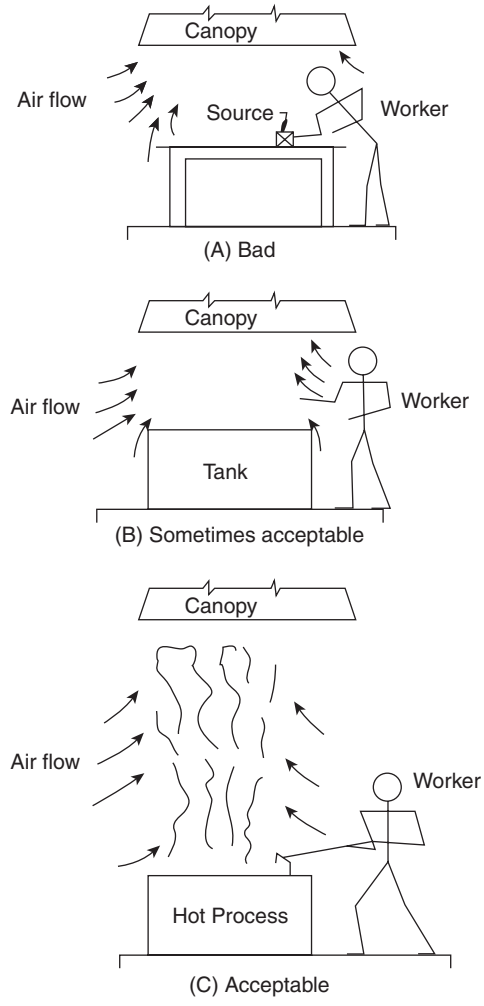

Figure 26-3. The proper use of a canopy hood, which does not allow the air contaminants to be drawn through the worker's breathing zone. The worker's location is crucial. (*Source*: National Institute for Occupational Safety and Health. The industrial environment: its evaluation and control. Washington, DC: NIOSH, 1973; p. 599.)

Limitation of Contact

The third important approach to controlling exposures to toxic materials is to limit worker contact by *(a)* automating processes, *(b)* isolating processes using toxic materials from the remainder of the work area so that the potential for contact with these materials is limited (Fig. 26-6), or *(c)* furnishing workers with PPE, such as respirators (dust masks or gas masks) or hoods or suits with externally supplied air for controlling inhalation of toxic materials.

Figure 26-4. Local exhaust ventilation successfully captures dust produced by stone cutting. (*Source*: W. A. Burgess, Harvard School of Public Health, Boston, Massachusetts.)

Figure 26-5. Electroplating workers are protected by local slot exhaust ventilation. (*Source*: W. A. Burgess, Harvard School of Public Health, Boston, Massachusetts.)

Figure 26-6. A glovebox enclosure system prevents solvent exposure to workers gluing shoes. (Photograph by Barry S. Levy.)

Many people mistakenly think that the use of respirators is a simple and inexpensive way to control exposure to toxic airborne materials. However, there is discomfort in wearing these masks, leading to poor worker acceptance, and a tight seal to the face is needed to achieve good protection, which reduces comfort and leads to variable levels of protection. There are extensive OSHA requirements for an adequate respirator program to ensure that the quality of devices is maintained and that workers are receiving adequate protection. The annual cost of a good respirator program for lead dust exposures is approximately $1,000 or more per worker. Fitting of respirators is extremely important but often neglected; a poorly fitting respirator provides substantially less protection than expected because, even if the respirator filters are highly efficient, air leaks around the edges of the face mask.

The use of rubber gloves and protective clothing does not automatically ensure that workers are protected adequately. Toluene and other aromatic solvents readily penetrate rubber gloves; thus, glove composition must be matched to the chemical nature of the substance. Similarly, long-sleeved shirts or coveralls may not prevent skin contact with toxic dusts because small dust particles can sift through the openings between threads in woven cloth.

A study demonstrated adverse health effects in orchard workers due to pesticide exposures, even though they had been wearing dust masks and long-sleeved work shirts.[7] Despite the shirts, their arms were covered with dust that contained pesticide residues. Even impermeable protective suits are difficult to seal to prevent migration of dust past cuffs and the neck opening. The suits also trap body heat and moisture, resulting in potential heat stress.

To limit contact with hazardous substances, protective clothing should be changed each day and not worn outside the work area, and workers should shower after each work shift. These practices also prevent workers from taking home hazardous materials on their work clothes, skin, and hair, thereby exposing family members and other household contacts.

Exposure can be partially decreased by administrative controls, such as scheduling workers to

spend limited amounts of time in areas with potential exposure, possibly reducing their cumulative exposure below recommended guidelines. While this approach may be effective in certain situations, it *(a)* requires good exposure data to demonstrate its effectiveness and the careful attention of supervisory personnel, *(b)* may be an inefficient use of workers, and *(c)* may be inappropriate for controlling exposures to carcinogens or teratogens.

Ideally, all of these control approaches should be used together to develop an overall control strategy that will address all aspects of exposure to toxic substances in a workplace. Short-term measures, such as extensive use of PPE, may be adopted immediately after a problem is recognized to allow time for developing engineering controls or process modifications that will provide better control over the long term. Despite their undesirable aspects, respirators may be the only effective control device for some exposures, such as those faced by maintenance or cleanup workers. (OSHA policy is to use them only as a last resort.)

An approach that has gained in popularity over the past two decades is the concept of *control banding*, a more generalized approach for controlling hazardous exposures that was developed in the pharmaceutical industry in the 1980s to complement the traditional air sampling and analysis.[8] To assess risk associated with a task or process, control banding combines qualitative and/or semi-quantitative information on the toxicity of an agent and the risk of increased exposures occurring. Based on this risk, each task or process is categorized into hazard "bands" within a facility that encompass broad categories of risk. Each hazard band is associated with a prescribed control strategy that is applied to all tasks or processes in a facility that fall within the band. Bands associated with higher risk are assigned to more stringent control strategies and assessment/reassessment procedures. This approach was designed to reduce time and toxicological expertise required in an occupational health unit in a workplace to assess risk, since hazards could be stratified into broad bands designed to protect workers from agents falling within a given band with a reasonable margin of safety. While there are many examples of successful implementation of this approach, one must properly rate the toxicity of an agent and account for exposure variability to ensure that this control strategy provides an adequate margin of safety.

NOISE PROBLEMS

Occupational exposure to excessive noise is an important problem that is evaluated and controlled, in part, by occupational hygienists (see Chapter 21). Hygienists are trained to measure the intensity and quality of noise, assess its potential for producing damage, and devise means to control noise exposures. Two principal types of workplace noise, continuous and impact, have somewhat different techniques of evaluation and control. *Continuous noise* is produced by high-velocity airflow in compressors, fans, gas burners, and motors. Crushing, drilling, and grinding are important sources of continuous noise because much energy is used in a small space. *Impact noise* results from sharp or explosive inputs of energy into some object or process, such as hammering or pounding on metal or stone, dropping heavy objects, or materials handling.

During the evaluation of a workplace, a hygienist looks for sources of excessive noise, determines which workers are exposed, and then selects an evaluation strategy to clarify the nature and extent of the exposures. If the noise is continuous or almost continuous, a hand-held noise survey meter may be used to determine the noise levels at a worker's location. If the exposure involves impact noises, an electronic instrument that records and averages the high-intensity, but short-duration, pulses is used to characterize the source and exposures.

Typically, workers spend variable amounts of time exposed to noise sources and may work at different distances from noise sources, which will alter their exposures. Exposures may also vary because the output of noise sources may change over time. Therefore, the TWA exposure may not be easy to estimate, even though the sources may present clear potential for overexposure. This problem has been solved by the use of small noise dosimeters worn by workers, which electronically record sound levels and indicate average noise levels during work shifts.

Dosimeters are very useful for describing average exposures. Most modern dosimeters store 8-hour (or longer) time traces, which can be displayed and linked to records of worker activities. A typical noise evaluation will include both assessment of the noise level at the source and dosimeter measurements of workers' noise exposure.

National requirements and TLV guidelines are used by the hygienist to evaluate noise data and decide whether a hazard is present. In addition to the hazard to hearing, a high noise level can significantly interfere with verbal communication, which may create another hazard by interfering with verbal warnings and worker detection of safety hazards, such as moving equipment. The OSHA standard for continuous noise for 8 hours is 90 dBA.* Higher levels are permitted for shorter periods of time. The OSHA standard allows levels of noise exposure that will protect some, but not all workers, from the adverse effects of workplace noise. The TLV for an 8-hour exposure to noise is 85 dBA, which is significantly lower than the 90 dBA OSHA standard for (continuous) noise.

Although techniques exist to obtain an overall TWA of noise exposures received in several different work settings, no techniques exist for assessing the hearing risks of combined exposure to both continuous and impulse noise. Many workers are exposed to both types of noise, such as brass foundry workers who are exposed to continuous noise from gas burners and to impulse noise from brass ingots dropping into metal bins from conveyors.

The strategies for controlling noise are similar to those used for toxic material control:

1. *Substitute*: Use another process or piece of equipment. For example, electrically heated pots for melting metal can be used instead of gas-heated pots to eliminate burner noise.
2. *Prevent or reduce release of noise*: Modify the source to reduce its output, enclose and soundproof the operation, or install mufflers or baffles. For example, noisy air compressors can be fitted with mufflers and placed in soundproofed rooms to control their noise; impact-absorbing materials can be installed to eliminate impulse noise from ingots dropping into a metal bin.
3. *Prevent excessive worker contact*: Provide PPE, such as earplugs or earmuffs, or provide a control booth.

As with toxic materials, the overall strategy to control exposures usually involves separate approaches for various aspects of the problem. It may be necessary to consult an acoustical engineer with advanced evaluation and engineering expertise to address complex noise problems. If engineering controls are not completely effective or are impractical, ear protectors may be required; however, the effectiveness of these devices is limited because sound may also reach the ear by bone conduction. A full-shift exposure above 120 dBA cannot be controlled adequately using earplugs or muffs.

RADIATION PROBLEMS

Radiation hazards are commonly first identified by occupational hygienists, but the responsibility for their evaluation and control overlaps among the occupational hygienist, the health physicist, and the radiation protection officer. (See Chapter 12C.)

Exposure to ionizing radiation can be external (from X-ray machines or radioactive materials) or internal (from radioactive substances in the body). External exposures can be monitored instrumentally by several methods; the type of detector system chosen for a given problem depends on the nature of the ionizing radiation. Personal monitoring is commonly performed with badges of photographic emulsions, thermal luminescent materials, or induced radiation materials that will indicate the cumulative dose during the period the badge is worn. Data from these measurement systems can be used to construct relatively accurate estimates of tissue exposure. If there are also detailed supporting data on worker activities, sources of exposure and points of intervention can also be identified.

* dBA denotes decibels (units of sound intensity on a logarithmic scale) that are summed across frequencies using the "A" scale, which weights frequency in proportion to the human ear's sensitivity to sound (see Chapter 21 for a more detailed explanation).

Non-ionizing radiation is also an external exposure problem. This type of radiation includes a variety of electromagnetic waves, ranging from short-wavelength ultraviolet, to visible and infrared, to long-wavelength microwaves and radiowaves. Exposures to ultraviolet, visible, and infrared radiation are measured with photometers of various types. Microwaves and radiowaves can also be measured by several standardized techniques, but there is uncertainty over the exposure intensities required to produce adverse health effects. Lasers may also provide a source of exposure to varying regions of the non-ionizing radiation spectrum, depending on the power and lasing medium involved in a particular application.

Exposures to radioactive materials can be evaluated with similar methodology to that used for toxic substances. Personal air sampling, surface sampling, and skin contamination measurement can be used to quantify exposures by their route of contact or entry into the body. For example, personal air sampling in uranium mines can measure miners' exposure to respirable radioactive particles that will be deposited in their respiratory tracts. Internal levels of some radionuclides can be detected outside the body and measured directly if they emit sufficient penetrating radiation, such as gamma rays emitted from radioactive cobalt. However, most cannot be detected externally; the quantities of radioactive substances reaching sensitive tissues, such as the bone marrow, usually must be estimated by determining the worker's external exposures and making assumptions about the amount entering the body and being transported to the site(s) of adverse effects.

The Nuclear Regulatory Commission has set standards for allowable ionizing radiation exposures for both external and internal sources. These exposure limits can be used, like TLVs, to decide whether a given exposure presents a health risk. Radiation protection programs have strict requirements about techniques for handling radioactive materials and working with radiation sources. They also require extensive routine exposure monitoring and medical monitoring.

Exposure limits for non-ionizing radiation have been set by OSHA, based on published scientific data, the TLVs developed by the ACGIH, and standards developed by the American National Standards Institute. Equivalent limits have been developed by WHO and several countries (see Further Reading). The eyes and the skin are critical organs to be protected. Standards have been set for the most susceptible organs, such as the eyes. There is concern about reproductive hazards for these agents. Standards also have been developed for lasers based on ophthalmoscopic data and irreversible functional changes in visual responses. As with other types of standards, the numerical limits cannot be treated as absolute and the margin of safety is often uncertain.

Control of external ionizing and non-ionizing radiation exposures is achieved by minimizing the amounts of radiation used, isolating processes, shielding sources, using warning devices, interlocking door and trigger mechanisms to prevent accidental exposures, educating workers and supervisors about the hazards, and, if necessary, requiring use of PPE. Consider the following example:

An industrial X-ray machine used to check castings for flaws is placed in a separate room with extensive lead shielding, and it cannot be triggered when the door to the room is open. The room also has signs warning of a radiation hazard. A red warning light inside the room is lit for 30 seconds before X-rays are released so that a worker inside the room when the door is closed could activate an emergency override switch to prevent operation of the machine. All personnel working near the X-ray machine are required to wear film badges to monitor their accumulated radiation exposure.

Control of internal radiation exposures from radioactive materials is very similar to controls for toxic materials. The objectives are to use minimal amounts of radioactive materials; isolate the work areas; enclose any operations likely to produce airborne emissions; use work procedures that prevent or minimize worker contact with contaminated air or materials; have workers wear PPE to prevent skin contact, eye exposure, or inhalation of materials; monitor environmental contamination levels; and educate workers about the hazards. Careful supervision of work activities and monitoring of

program implementation are required to provide adequate protection.

CONCERNS FOR THE FUTURE

Workplaces in heavy industry have been a concern of hygienists for many years, although this concern is somewhat less at present in the United States and Europe. However, as older technologies have been exported to developing countries, hygienists have been increasingly concerned because these countries seek the economic benefits of heavy industry without adequate resources to ensure worker health and safety.

In some developed countries, there has been increasing concern about the office environment and the health effects associated with energy-efficient, tightly sealed buildings (Chapter 7). There also is concern that, although hazards in large companies have been largely controlled, those in small and midsized companies have not been adequately controlled because these companies have fewer resources.

The scientific basis for occupational hygiene practice has been eroding because of limited research funding and a small number of researchers. Examination of the scientific literature in occupational hygiene shows it to be narrowly focused on limited issues. Internal research funded by companies is often not published because it may aid competitors or may raise liability concerns. Gaining access to workplaces for academic research has also become more difficult. There is a reluctance of employers to examine the hazards of their operations because of concerns about the costs of additional government regulation and legal liability for health claims from previously unrecognized hazards. As a result, there has been little development, updating, and refinement of exposure assessment methods, control technology, intervention strategies, or governmentally enforced exposure limits—despite extensive worldwide development of new materials, production technologies, biomedical and drug manufacturing, and other advances.

Occupational hygiene has not kept up with this development. Some occupational hygienists who are concerned about this situation have begun working to strengthen local research and to develop collaborative international research programs. Joint labor and management research programs supported by company funds have also become increasingly important sources of workplace access and research funding.

ENVIRONMENTAL HYGIENE

Environmental hygiene has much in common with occupational hygiene. There is considerable overlap in basic concepts and approaches for hazard recognition, exposure assessment, and source controls, especially for air pollution. The major difference is that the occupational hygienist works in a prescribed setting and with a generally healthy adult population. In contrast, the environmental hygienist covers the entire environment and all of the population, including young, elderly, and ill people, in addition to healthy adults. Exposures tend to be higher in occupational settings, but not always. Environmental transport of contaminants is usually more extensive in the outdoor environment than the workplace. Residents of an area will have both ambient exposures as well as exposures inside their homes and other buildings.

Source-Transport-Receptor Model

The source-transport-receptor model is a simple, yet powerful, representation of environmental exposures that has been widely used by environmental scientists (Fig. 26-7). The source produces and releases emissions into the environment, such as airborne emissions from a power plant or toxic organic solvents released into groundwater by a waste dump. The source defines the composition and release rate of the emissions. Source sampling is very useful when it can be accomplished. Sometimes there are many small sources that are dispersed, such as individual vehicles in traffic and heating units in homes. Given knowledge about the source, we can identify what types of hazards might be present and what to measure. Environmental processes during transport of emissions include dispersion and dilution, photochemical reactions with sunlight, and removal by rain, adsorption to soil particles, and sedimentation. The main concern is the composition and

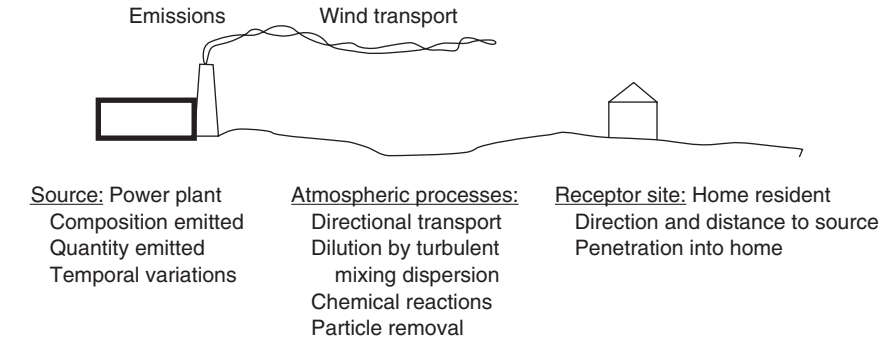

Figure 26-7. Source-transport-receptor model for wind transport of emissions from a power plant to a home.

concentration of emissions reaching the receptor—the exposed person.

Major Problems

Air and water emissions from vehicular traffic, power plants, industrial operations (especially chemical manufacturing and mining), food sanitation, municipal and agricultural wastes, sewage, and solid waste are all major problems (Table 26-2). Usually, one must investigate the characteristics of a given source in detail to understand fully the hazards it presents. There is no simple summary that can adequately describe the complexity and variety of source releases. One should also consider the natural sources that may affect the background levels of an environmental contaminant.

Table 26-2. Major Sources of Environmental Emissions and Releases

Major Sources	Emissions–Contaminated Media	Route of Entry
Natural processes: • Erosion • Vegetation and microbes • Volcanoes • Geothermal springs • Forest and grass fires • Storm runoff and floods • Sea spray • Radon	Airborne dusts, pollen, terpenes, ammonia, methane, sulfur dioxide, hydrogen sulfide Waterborne particles, dissolved carbon and metals, hydrogen sulfide, arsenic Soil contamination with metals, radon	Inhalation and ingestion
Power plants	Airborne ash, nitrogen oxides, metals —Sulfur oxides, arsenic from coal —Sulfur oxides, vanadium from oil Radionuclides in air and water from nuclear power	Inhalation and ingestion
Vehicle emissions: Traffic—cars, trucks, buses Off road—trains, mining Boats and ships	Airborne unburned fuel, nitrogen oxides, PAHs, particles, lead Secondary photochemical smog Waterborne hydrocarbons, lead in street runoff water Soil contamination with hydrocarbons, lead Ship ballast water releases of oil, organisms	Inhalation and ingestion
Home heating emissions - combustion	Particles, hydrocarbons, sulfur oxides, metals	Inhalation
Sewage systems —Human wastes —Small business wastes	Waterborne pathogens, nutrients, toxic chemicals, solvents, metals	Ingestion
Industrial activities —Chemical releases	Airborne contaminants (depend on the specific industry) Wastewater contaminants (depend on the specific industry)	Inhalation and ingestion

PAHs, polynuclear aromatic hydrocarbons.

EXPOSURE CHARACTERIZATION

A variety of methods are used by the Environmental Protection Agency (EPA) and other regulatory agencies to characterize common and dangerous exposures. These methods are based on *(a)* sample collection and analysis, and, more commonly, *(b)* direct-reading instruments. The most important part of an evaluation is the sampling strategy, which specifies how, when, and where samples will be obtained and measurements performed. The goal is to define the magnitude of exposures, which can be highly variable among individuals and across time and space. Thus, a series of measurements must be made to define the distribution of exposures that a person may have. For environmental problems, we generally measure contamination of air, water, food, and soil. There are also measurement techniques for noise, radiation, and other hazards.

Sampling Strategy

One of the most challenging aspects of environmental sampling is ensuring that samples are representative of exposure, since there are so many different sources of variation. Most importantly, time-activity patterns, which dictate one's duration of contact with environmental contaminants in a given microenvironment, can vary greatly within and among individuals—so one needs to consider what conditions may influence the difference between environmental and human exposures. For example, there can be important temporal variations on the scale of minutes, hours, days, weeks, months, seasons, and years, and spatial variations on the scale of feet, yards, hundreds of yards, miles, and more. Contaminants in wells, streams, rivers, and lakes can vary dramatically. So can contaminants in individual food items and batches of food items. As a result, one needs to be clear about the exposure distribution one is attempting to characterize: Is it for healthy adults, children, elderly people, or ill people? For homes along a busy street, for a neighborhood, for a particular time period, or for a certain activity? Do we need to collect personal samples or will fixed-location area samples alone be sufficient? If we wish to define the mean and standard deviation of an exposure, fewer samples are required than if we are trying to define the range or the probability of the highest values (the upper tail of the exposure distribution). If we are looking for exposures that exceed a legal limit, it may be necessary to use a strategy and methods defined by law or regulation. Therefore, the hygienist must have a well-developed set of questions to answer and a sampling strategy that will obtain answers as efficiently as possible.

Sample Collection

Each exposure media has different collection approaches (Table 26-3). Airborne particles may be collected on filters, impacted on surfaces, or electrostatically collected. Nonreactive gases can be collected in bags and bottles. Reactive gases may be collected in liquids using bubblers or treated substrates, such as filters or adsorbents. Vapors, such as gasoline vapors, are gaseous, but their normal state is solid or liquid.

Consequently, they will readily condense on surfaces like the inside of collection bags, whereas gases will not. The preferred method for sampling vapors is with special materials in small holders, such as charcoal in tubes or reactants impregnated on papers or fibrous glass. The collection process may be either active, using a pump to draw air through the collector, or passive, using diffusion to bring the gaseous materials into the collector. Badge-type passive collectors are better accepted by subjects for personal sampling. Automated collectors may be used to collect a long series of air or water samples in a chosen location, or instruments drifting with balloons to follow the removal of power-plant emissions by atmospheric reactions, or floating devices for monitoring ocean water in currents.

Sample Analysis

Sampling media or containers are sent to a laboratory with experience performing the desired analyses. Accredited labs are best. If the data are to be used for determining compliance or legal proceedings, analyses for all regulated substances must follow the method given in regulations. If the measurements are to be used for a research project, where there is no accepted method for the substances of interest, then the investigator

Table 26-3. Examples of Environmental Sampling and Analysis Methods for Common Contaminants

Media and Contaminant	Collection Method Examples	Analytical Method
Air—Metals (lead, cadmium)	Total metals—Membrane filter (0.2 μm pore size) and pump (4 liters per minute)	Acid digestion of filter and sample, atomic absorption (AA) measurement
	Respirable particles—Use cyclone or impactor precollector before filter	Gravimetric analysis for total particle mass
	Direct analysis[a] by specific light scattering instrument for count or mass	
Air—Organic vapors	Hydrocarbon vapors—charcoal in canister	Strip vapors with solvent and gas chromatography (GC) analysis
	Pesticides—Tenax or XAD or other suitable resin	
Air—Reactive gases (ozone, ammonia, sulfur dioxide)	Collect on treated substrate; selective for gas	Specific reaction products; ion chromatography analysis
	Direct analysis by specific electrochemical instrument	
	Direct analysis by specific colorimetric detector tubes	
Air—Carbon monoxide	Collect in bag	Gas sample injected for direct analysis by GC
	Direct analysis by specific electrochemical instrument	
Water—Metals (lead)	Collect sample[b] with acid cleaned polyethylene bottle (1 liter)	Free lead—Acidify water and AA analysis
		Total lead—perchlorate digestion then AA
Water—Microorganisms	Collect water in sterile bottle	Filter water; culture organisms on filter or directly culture water; digest filtered organisms and polymerase chain reaction (PCR) analysis
Food and soil—Pesticides	Collect food or soil sample[c] in chemically clean container	Macerate and prepare[d] food and soil; extract preparation with solvent; GC analysis

[a] Direct analysis is a real-time measurement with a very short averaging time, usually 1 minute or less.
[b] A variety of components may need to be characterized to accurately represent a water contamination problem, such as organisms, suspended materials, or sediments.
[c] Because food and soil are very heterogeneous, careful sampling strategies are needed to collect representative samples that indicate human exposures.
[d]Food and soil are complex matrices and may require extensive preparation to prepare them for analysis.

must work closely with the lab to ensure the validity of the data. Developing new methods to measure an environmental contaminant is a major undertaking, even for highly skilled environmental chemists. It is not unusual for the development and validation process to take several years.

In all cases, there must be detailed quality-control and quality-assurance plans, which should include lab and field blank samples, analysis of standards with each batch of samples, duplicate field samples, replicate analyses, and sometimes spiked blanks or spiked samples to verify that there were no problems occurred during field collection or lab analyses. More information about quality-assurance programs can be found in standard texts on environmental monitoring or publications by regulatory agencies.

DEFINING A HAZARD

Complaints

It is common for residents of an area to complain about odors, dust, and damage to materials from air pollutants. Likewise, they may complain about the odors, color, and opacity of water in ponds, lakes, streams, and rivers, and all of these problems in addition to the taste of drinking water. Any of these complaints may indicate a potential problem. In some cases, the problem is one of aesthetics and, in others, it is a

health risk. If the source of the poor quality can be identified, such as a chemical plant upwind or a sewage treatment plant upriver, then it may be possible to identify what might be contaminating the environment. Often the source is not known, but the odor or other characteristics may be identified. For example, in the past many private wells were contaminated with gasoline leaking from underground storage tanks, and the gasoline odor from the water was notable. Spills or accidental releases are most often noted by people living or passing nearby, and they may be the people most at risk.

Exposure Assessment Study

Where the resources are present, an exposure study may be conducted to define what contaminants are present and determine whether they exceed allowable exposure standards developed by the EPA, WHO, or other agencies. However, it is very difficult to determine what unknown agents are present in sampled media. Unfortunately, there are no general, broad-spectrum methods that can readily determine the components of the black gunk scraped off the bottom of a pond, material obtained from an abandoned barrel left at a waste site, or a substance causing an unpleasant odor in air, water, or food. In general, one must tell laboratory personnel what to measure, so they can choose appropriate methods. If a suitable, sample collection strategy is used, then exposure levels can be compared to exposure standards. When measured levels are less than the standard, it may mean that there is no hazard; however, if there are health complaints related to the exposure, the findings may also mean that the exposure standard has been set too high.

Epidemiologic Study

If there are local health complaints, such as by neighbors of a local industrial plant, then a small study may be conducted by the local health department. A larger, full-scale epidemiological study may also be warranted in some cases. Water contamination problems in Woburn, Massachusetts, starting in the 1950s and ending in 1979 with the closure of contaminated wells, are a good example.[9,10] Excess leukemia in children was found to be associated with residence near the dump. Other community studies have been launched to examine apparent unusual clusters of cases of disease.

CONTROL OF HAZARDS

The same processes used to control occupational exposures and waste emissions are also used in environmental controls. Elimination of the source is the only completely successful control approach. Sometimes it is possible to change the source or limit its output. Personal exposure controls are even less successful for general environmental hazards than they are for occupational hazards. Respirators and protective clothing are impractical and unreasonable for the general public. Although there is an active market for home and personal air cleaners, the amount of protection they can provide is limited; in addition, some of the electrostatic particle collectors will expose people to ozone, a hazardous gas. Therefore, emission controls at the source are the most common and effective way to deal with emissions from large or numerous sources.

EXAMPLES OF MAJOR PROBLEMS

In this section, we consider some of the major types of environmental problems, their sources, transport phenomena, and characterization of exposures at the receptors.

Air Pollution

Daily variation in concentrations of airborne particulate matter less than 10 μm in diameter (PM_{10}) and less than 2.5 μm in diameter ($PM_{2.5}$) have been closely correlated, in many cities worldwide, with daily fluctuations in death rates—especially from respiratory and cardiovascular diseases. These effects occur even at low concentrations (20 to 200 $\mu g/m^3$). Some, but not all, of those who have died have been people with preexisting respiratory and cardiovascular diseases that would not have otherwise been fatal.

Environmental hygienists have been challenged to better define air pollution exposures

associated with adverse health effects in the general population. Time-activity studies have followed the daily lives of children, adults, elderly people, and ill people, while monitoring their exposures. Surprisingly, aside from children playing outdoors and people engaged in outdoor sports, most people in cities spend limited time outdoors during which they are exposed to urban air pollution. Indoor exposures account for much more of their time. Exposures to some general categories of air contaminants, such as oxides of nitrogen (NOx), can be higher inside homes than outdoors because there are sources of NOx indoors, such as gas-fired stoves. However, the outdoor levels of $PM_{2.5}$ permeate indoors. Therefore, even though some people do not spend much time outdoors, their indoor exposure—and their mortality rates—correlate with people who have had much exposure to outdoor $PM_{2.5}$. (See Box 26-1.)

Indoor and outdoor exposures may be qualitatively and quantitatively different if there are differences in strong emission sources in one place or the other. In some instances, aerosols generated inside homes may be less toxic than outdoors. For example, indoor $PM_{2.5}$ comes from reentrainment of dust and dirt tracked into homes or from vacuuming and sweeping, whereas outdoor $PM_{2.5}$ comes from combustion sources and atmospheric reaction products (smog). In that case, higher indoor exposures do not translate to higher risk. Some toxic outdoor air contaminants, such as some gases and small-diameter particles within $PM_{2.5}$, can readily penetrate into homes. However, highly reactive ozone in smog is found at lower concentrations indoors than outdoors because it reacts quickly with surfaces.

Once materials are released to the environment, there are several processes that operate to modify and remove them—processes that differ for particles and gases. Combustion processes can produce dense clouds of nanoparticles (0.01 to 0.1 μm), sometimes visible as smoke. (See Box 26-2.) These rapidly agglomerate into larger particles (~1 μm in diameter). As the particles become larger, they settle out of the air or are removed by impaction on surfaces of buildings and trees. During periods of high humidity, water-soluble particles will absorb water and may grow large enough to be removed by sedimentation and impaction on surfaces. Particles can also be removed by precipitation. Semivolatile vapors of polynuclear aromatic hydrocarbons (PAHs), tars, and greases condense on ambient particles as hot combustion gases cool. Gaseous emissions can also undergo atmospheric reactions. Photochemical reactions of some hydrocarbons can produce nanoparticles. Sulfur dioxide from burning materials containing sulfur, such as coal and fuel oil, can be photochemically oxidized to sulfur trioxide, which reacts with water to form sulfuric acid, which absorbs water to form tiny droplets. Atmospheric ammonia reacts with the sulfuric acid to form ammonium sulfate. Some hydrocarbons will be oxidized to peroxides, aldehydes, and organic acids by ozone and other strong oxidants.

The hydrocarbons and nitrogen oxides released by vehicles in the summer can be

Box 26-1. Assessing Indoor Air Pollution

In many cases, indoor problems cannot be characterized by a generally uniform clinical picture and a specific cause (Chapter 7). These problems result from complex interactions among several factors, including air contaminants (including their odor), temperature, ventilation, air movement, illumination, noise, ergonomics, and psychological and social factors. Emerging complaints about indoor air quality should be assessed and controlled promptly. Specific measures should include the following:

1. Checking whether operational conditions are normal for the HVAC and other systems
2. Determining the nature and magnitude of problem by using a standardized "sick building" questionnaire and/or structured interviews
3. Performing a technical survey to assess risk factors inherent in the use and operation of the building
4. Assessing construction materials, furniture, quality of cleaning, moisture damage and mold growth, temperature, air movement, and carbon dioxide concentration
5. Estimating degree of recirculation of air and possible contamination of intake air
6. Measuring ventilation efficiencies with tracer gas studies
7. Making a detailed assessment of contaminant sources and concentrations
8. Performing clinical examination of affected persons and additional occupational hygiene investigations, such as performing detailed chemical analyses of complex mixtures and assessing individual work habits to guide training interventions

Box 26-2. Nanomaterials: Occupational and Environmental Exposures, Health Effects, and Control Measures

Margaret M. Quinn

Submicrometer particles, which are measured on the nanometer (nm) scale (billionths of a meter), can be more biologically reactive than larger particles of similar chemical composition and may present a greater health risk. Some types in ambient and work environments include aerosols from fossil fuel combustion, diesel exhaust, asphalt fume, flour dust, welding fume, silica, asbestos, and some synthetic fibers. New types of nanoscale particles are being made by applying atomic-scale engineering processes to carbon; metals such as iron, beryllium, chromium, lead, cadmium, manganese, zirconium, titanium, aluminum, silver, and gold; and many other materials. Two of the most common engineered nanoparticles—nanotubes (hollow tubes) and fullerenes (hollow soccer ball–shaped spheres)—are made of complex configurations of carbon atoms whose surfaces can be modified by attaching chemical functional groups or biological molecules, such as antibodies.

The discussion presented here draws on a recent report from the National Institute for Occupational Safety and Health (NIOSH) and other national and international resources (see Further Reading section).

Terminology

The term *ultrafine particle* has been used to describe airborne particles smaller than 100 nm (sometimes less than 500 nm) in diameter. Most of these refer to particles with diameters in the nanometer range that are heterogeneous in composition and are unintentional by-products of processes involving combustion, welding, or use of diesel engines. The term *nanoparticle*, defined as a particle less than 100 nm, is frequently used to describe particles with specific chemical compositions or properties, such as shape, size, or surface characteristics. The two terms are often used to differentiate nanoscale particles that are engineered (nanoparticle) and those that are incidental (ultrafine). However, these designations do not necessarily imply differences relevant to hazard assessment, measurement, or control of exposures.

The International Organization for Standardization Technical Committee 229 (Nanotechnologies) is developing globally recognized nomenclature and terminology for nanomaterials. *Nano-object* is defined as material with one, two, or three external dimensions in the size range from approximately 1 to 100 nm. Subcategories of nano-object are *(a) nanoplate*, a nano-object with one external dimension at the nanoscale; *(b) nanofiber*, a nano-object with two external dimensions at the nanoscale, with a hollow nanofiber termed a *nanotube* and a solid nanofiber termed a *nanorod*; and *(c) nanoparticle*, a nano-object with all three external dimensions at the nanoscale. Nano-objects are commonly incorporated in a larger matrix or substrate referred to as a *nanomaterial*. Nano-objects may be suspended in a gas (as a *nanoaerosol*), suspended in a liquid (as a colloid, or *nanohydrosol*), or embedded in a matrix (as a *nanocomposite*).

Health Effects

The health effects of nanomaterials are uncertain. The toxicity of ultrafine or nanoparticles is greater than that of the same mass of larger particles of similar chemical composition, possibly due to more nanoparticles per unit mass and differences in their physical and chemical properties. Carbon nanotubes may possibly be toxic because their needle-like shape resembles that of asbestos fibers. Introduction of long, multiwalled carbon nanotubes into the abdominal cavities of mice induced inflammation and formation of granulomas. Carbon nanotubes can enter cells and cause release of pro-inflammatory cytokines, leading to oxidative stress and decreased cell viability. It remains unclear, however, how these findings are relevant to workers or others.

The proportion of inhaled nanoparticles likely to deposit in any region of the respiratory tract is 30% to 90%, depending on breathing rate and particle size. Up to half of nanoparticles 10 to 100 nm in size may deposit in the alveolar region, while those smaller than 10 nm are more likely to deposit higher in the respiratory tract. Respiratory deposition of nanoparticles increases with *(a)* exercise due to increase in breathing rate and change from nasal to mouth breathing, and *(b)* in people with asthma, emphysema, and other lung disorders. Nanoparticles in the lungs may enter the blood and migrate elsewhere in the body. Nanoparticles depositing in the nasopharynx may enter the brain via the olfactory nerve.

Air pollution with ultrafine particles is associated with increased morbidity and mortality from respiratory and cardiovascular disorders. Workers exposed to ultrafine particles have decreased lung function, increased respiratory symptoms, and increased risk of chronic obstructive pulmonary disease and lung fibrosis. Some studies have found lung cancer and neurological effects among workers exposed to ultrafine diesel-exhaust particles or welding fumes.

Some studies suggest that nanomaterials could enter the body through the skin during occupational exposure. Little is known about possible adverse effects from the ingestion of nanomaterials.

Safety Hazards: Evaluation and Risk Management

Although insufficient information exists to predict the fire and explosion risk associated with nanoscale powders, nanoscale combustible material could present a higher risk than a similar quantity of coarser material, given its large surface area. Some nanomaterials that are designed to generate heat through chemical reactions could present a fire hazard.

There are no specific exposure limits in the United States for airborne exposures to engineered nanomaterials or to conventional materials in the nanoscale range. Factors affecting exposure to engineered nanomaterials include the amount of material being used and whether the material can be easily dispersed (in the case of a powder) or form airborne sprays or droplets (in the case of suspensions).

(*Continued*)

Box 26-2. Nanomaterials: Occupational and Environmental Exposures, Health Effects, and Control Measures (Continued)

Occupational and environmental assessment of nanomaterials must involve a multifaceted approach in order to measure all relevant characteristics of nanomaterial exposure. NIOSH has developed the Nanoparticle Emission Assessment Technique (NEAT) to qualitatively determine the release of engineered nanomaterials in the workplace.

Currently no single air sampling method is adequate to characterize exposure to nanoscale aerosols. Air sampling should include a combination of sampling and analytic devices and methods, including the following:

- Direct-reading instruments to count and size particles and to yield the distribution of particle sizes
- Transmission electron microscopy (TEM) to determine particle morphology and to provide an estimate of particle size distribution
- Filter-based air samples for chemical analysis and helping to identify exposure sources

Microscopes equipped with energy dispersive X-ray spectroscopy (EDS) can determine the elemental composition of particles.

Given the limited information about the health risks associated with occupational exposure to engineered nanomaterials, appropriate steps should be taken to minimize the risk of worker exposure with a risk management program. Risk management programs for nanomaterials should be seen as an integral part of an overall occupational safety and health program for any company or workplace producing or using nanomaterials or nanoenabled products. The program should be capable of anticipating new and emerging risks to determine whether they are linked to changes in the manufacturing process, equipment, or introduction of new materials.

A risk management program should include "Prevention through Design" techniques, including an evaluation of materials and practices in order to identify whether safer alternatives are available or could be newly designed (see http://www.cdc.gov/niosh/topics/ptd/).

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Useful Web Sites

European Agency for Safety and Health at Work. Available at: http://osha.europa.eu/en/riskobservatory

European Commission, CORDIS: Nanotechnologies and Nanosciences. Available at: http://cordis.europa.eu/nmp/home.html

National Institute for Occupational Safety and Health. Available at: http://www.cdc.gov/niosh/topics/nanotech/default.html

UK Royal Society of Engineering, Nanoscience and Nanotechnology. Available at: http://www.nanotec.org.uk/index.htm

U.S. National Nanotechnology Initiative. Available at: http://www.nano.gov/

Woodrow Wilson Center for International Studies Project on Emerging Nanotechnologies. Available at: http://www.nanotechproject.org/

photochemically converted to intense eye and upper respiratory irritants, especially highly reactive oxidants including ozone, under the action of ultraviolet radiation from the sun. The classic time pattern is as follows: Vehicle emissions during the morning rush hours are converted to afternoon smog, as occurs in Los Angeles, Denver, and Houston, and other large cities. Although emission controls on cars, improved engines and fuel, and catalytic mufflers have dramatically reduced emissions per car over the past three decades, the number of

cars has increased over this period. As a result, the number of smog alerts has decreased, but not proportionally to individual vehicle-emission reductions. (See Chapter 6.)

Common Personal Sources

Although many personal items, such as cars, home heating units, barbeques, and power mowers, are minor sources of pollution, when taken together in large numbers in a city or an area where people are concentrated, they can contribute significantly to air pollution. Areas that have poor air flow, such as valleys, can have major episodes of ambient air pollution. Car and truck traffic accounts for much of the emissions in urban and suburban areas, including particulates containing elemental carbon, oils, grease, unburned fuel, and PAHs, as well as gases containing carbon monoxide, carbon dioxide, oxides of nitrogen, benzene, toluene, xylene, aldehydes, organic acids, and other volatile organic compounds (VOCs).

Biological Sources

Biological hazards include pollens, airborne molds, bacteria (and related by-products, such as endotoxins from gram-negative bacteria), and other infectious agents. Seasonal allergies are a consequence of contact with pollens released by plants. Less familiar are releases by pine trees of terpenes (chemicals found in terpentine, a paint solvent). The haze in the Smoky Mountains in North Carolina is produced by the photochemical reaction of sunlight with the summertime releases of terpenes. This is supplemented by sulfuric acid aerosol from power plants in the Midwest burning high-sulfur coal. Natural decomposition of vegetation and dead animals by microorganisms produces several common air contaminants. Proteins are reduced to ammonia, nitrates, hydrogen sulfide, and sulfates. Organic carbon is ultimately reduced to methane under anaerobic conditions and carbon dioxide when there is sufficient oxygen; both methane and carbon dioxide are greenhouse gases produced in large amounts by natural sources.

Natural Sources

Forest and grass fires, volcanoes, erosion, and sea spray produce large amounts of air contaminants across broad areas. Natural fires are dangerous due to both the destruction they cause and the large amounts of smoke they produce. Outdoor fires release many of the same combustion products that are found in emissions from cars, trucks, and other humanmade sources. Volcanoes release inorganic particles of ash and sulfur dioxide, and produce hazardous conditions in broad areas downwind. The rare catastrophic eruptions, such as of Mount Pinatuba in the Philippines, can inject thousands of tons of ash and sulfur high into the stratosphere, which can remain airborne for a year or more and enhance sunsets worldwide. Erosion produces soil particles, which can be entrained into the air if the soil is dry and dusty. Dust storms scour tons of dust into the air, exposing populations to very high dust levels. Coastal areas receive constant inputs of sea-salt aerosol from breaking waves and the nanoparticles of salt that are formed by bursting bubbles on the ocean.

Industrial Sources

Generally, these are point sources associated with local operations, such as power plants, chemical manufacturing facilities, and petroleum refineries. Most of these are required to meet governmental regulations on emissions. The specific emissions from these operations depend on the specific industry, which can be found in reference books and databases of governmental agencies in the United States and many European countries. Information is widely available for recognized hazards, such as the criteria pollutants defined by the EPA and WHO, but not for many new chemicals and materials. Small enterprises, such as foundries, auto repair, painting, small manufacturing, and recycling operations, can also be sources of local problems. Public health agencies must conduct continuous environmental surveillance to determine whether controls are effective and to detect problems if they are not.

Power plants are one of the most common industrial sources of airborne materials. They burn coal, oil, or natural gas, and some can switch between different fuels. They are major sources of carbon dioxide and oxides of nitrogen, and, depending on their fuel, they may also release large amounts of sulfur oxides (from coal and oil with sulfur); coal burning also releases mercury vapor and fine particles of ash that

contain metals (lead and cadmium in coal, and vanadium in oil). Exposures will depend on wind direction and weather conditions.

Industrial operations run as designed most of the time. Unfortunately, there can be acute situations that result in large emissions. Spills of chemicals, fires, breakdowns of processing equipment, accidental releases, and failures of control systems can all produce massive emissions. In cases where the failure of a key part of the operation, such as a cooling system, will result in an explosion or a chemical release, a special system is usually in place to deal with a potential breakdown. One of the best examples of this is a nuclear power plant, which has back-up systems to maintain the flow of cooling water and control rods to limit the nuclear reaction. The few cases of malfunction are well known. One lesson from the Three Mile Island reactor accident in 1979 is that despite "fail-safe" engineering, unforeseen combinations of failures can still occur. Catastrophic breakdowns and accidents do occur and disaster preparedness to minimize their effects is critical.

Emission Controls

There are well-developed emission controls for nearly all of the common industrial processes that produce airborne waste products. Particles can be collected by filters, cyclone separators, electrostatic precipitators, and gases and can be removed by scrubbers and other specialized devices. Collection efficiency is never 100%. As a result, even if the proportion collected is 98%, as it is in some power-plant electrostatic precipitators, if the amount of particles produced is very large, then the amount that escapes collection can be large. Engineering controls used to minimize emissions must be well designed and carefully maintained to operate effectively. Assessment of these systems must be done by specialists. In general, elimination of waste production is a much more effective control strategy than removal of waste. Changes in processes and raw materials can sometimes eliminate problematic wastes, such as reducing residual amounts of sulfur in fuels.

Water Pollution

Problems with water pollution tend to be more localized than air pollution, although contamination of large watersheds can affect large populations. Water contaminants can take several forms: dissolved substances, such as salts, and gases; suspended particles, such as clay and organic matter that will settle out of the water; colloids, stable suspensions of very small particles that will not settle out; and floating substances, such as oils and grease. Oxygen content is one of the critical dimensions of water quality because it is necessary to sustain plants and animals in the water and sediments. Solubility of oxygen varies inversely with temperature; cold water holds more oxygen than warm water. The exchange of oxygen with the atmosphere is relatively slow, so conditions that either block the surface uptake or rapidly use up dissolved oxygen can create anaerobic conditions. Oil slicks block oxygen uptake. Large amounts of biological debris and organic carbon, such as from bacterial decay of vegetation and sewage, can deplete water of its oxygen. Then decay becomes anaerobic, which limits what can grow and creates odor and other problems.

Bioaccumulation

Accumulation and concentration of toxic chemical contaminants in the food chain is a serious problem for some substances, such as lipid-soluble materials with very low water solubility that are not broken down by natural biochemical processes or organisms in the environment. Biological transport into and out of organisms requires some water solubility, and phase II metabolism of toxic materials, such as adding polar groups like –OH or $–NH_3$ to nonpolar hydrocarbons, increases their water solubility. The classic example of a substance that accumulates in biological systems is DDT. Since DDT has a low order of human toxicity, it was extensively sprayed to control mosquitoes and other insect pests in the 1940s and 1950s. Then, during the 1960s, it was found that populations of avian predators, such as fish-eating ospreys and eagles, were rapidly declining as a result of high losses of their eggs because the shells had become very fragile and easily broken. Rachael Carson brought this story to national attention in 1962 with her book *Silent Spring*. Researchers had found that trace concentrations of DDT in natural waters accumulated in the lipids of tiny plants. Small aquatic animals ate those plants,

collected the lipids from many plants, and concentrated DDT into their own body fat. Small fish ate relatively large amounts of those small animals, further concentrating DDT in their fat. Medium-sized fish ate the small fish, and large fish ate the medium-sized fish. This concentrating process progressed up the food chain, with DDT becoming more concentrated at each step—as much as 10,000-fold relative to the starting concentration in the plants. Finally, at the top of the food chain, the ospreys and eagles ate the large fish, whose fat now contained toxic levels of DDT. People who eat the contaminated fish can also accumulate high levels of DDT, polychlorinated biphenyls (PCBs), and other environmental chemicals.

A high degree of chemical stability and a low rate of metabolism are critical requirements for bioaccumulation. The stability of DDT and some other chemicals has allowed them to spread throughout the world so that even in the most remote places one can find DDT in the body fat of "top predators." Bioaccumulation also occurs for methylmercury, which is formed in aquatic sediments by bacterial methylation of inorganic mercury. Tuna and swordfish accumulate methylmercury and when people eat these fishes they accumulate mercury in body fat, including in highly lipid neurological tissue. PCBs accumulate in fat, and some radioactive materials, such as strontium-90, bioaccumulate in bone.

Common Sources

The major sources of water pollution in developed countries are storm runoff and sanitary waste water from cities, wastes from industries, runoff from agricultural land where fertilizers and pesticides are used, and runoff from concentrated animal-feeding operations. Large industrialized animal-feeding operations can produce as much fecal waste and urine as a small city. Fertilizers as well as animal and human wastes are major stimulants of plant growth in the receiving waters, leading to algae blooms. The bacteria decay of large amounts of organic wastes can strip the oxygen from the water, producing anaerobic conditions that kill fish and aquatic vegetation and lead to the growth of fungi, slug worms, and bacteria. This process has produced large "dead zones" in the Gulf of Mexico and other locations.

While large oil spills are catastrophic when they occur, they are rare. A larger problem is the discharging of contaminated bilge water from ship tanks. After a ship pumps out a liquid cargo, the empty tanks must be refilled with water to stabilize the ship. When the ship prepares to take on another cargo, it must pump out the water to make space for the new cargo. This constant filling and emptying of ship tanks is a major source of water pollution because the bilge water becomes contaminated with oil or other materials previously in the tanks. This procedure is also a common way that aquatic species are transferred from one area to another, where they may not be native.

Industrial waste releases have historically resulted in water-quality problems, from spills and accidental releases as well as routine releases. Specific hazards depend on the industry. Reference books on water pollution and toxic chemicals can help one identify which materials a given industry may commonly release and the problems they cause. Governmental regulations have greatly reduced the problems in areas where they are enforced. However, new industrial processes can produce wastes with unrecognized hazards.

There are natural cleaning processes that can lead to recovery in polluted waterways. Given sufficient time without further pollution, natural processes will remove the organic oxygen demand and lead to the return of plants and organisms that prefer clean, well-oxygenated conditions. Acidic and alkaline wastes will be neutralized. Toxic metal ions will be removed by complexation, in which insoluble complexes are formed and, by sedimentation, deposited. The bottom sediments will remain toxic, but without much effect if they remain undisturbed.

Water Treatment

Drinking water treatment is directed toward removing floating particle contaminants and killing pathogens. Surface waters with minimal contamination are purified by *(a)* addition of chemicals, such as alum or ferric sulfate, that will cause any particles to stick together (flocculation); then *(b)* sedimentation to allow the big particles to deposit on the bottom of large tanks; and then *(c)* filtration through beds of sand, which removes the fine particles. Following filtration, water is disinfected by adding chlorine,

chloramines, or other bactericides until there is free bactericide in the water flowing to the customer. The free bactericide provides some residual protection against pathogens that might be introduced through breaks or problems in the distribution system. In places where there are mineral contaminants, such as iron or hydrogen sulfide, or hardness in the source water, additional chemical treatments can be used.

Drinking water treatment is directed toward bacterial contamination, which may not be effective for viruses or toxic chemicals. Chlorination creates carcinogens by oxidizing some trace organic chemicals found in surface water. However, we believe that the risk from bacterial diseases far outweighs the small added cancer risk. Some alternative treatment methods do not have this risk, but they are more expensive. There is little routine water-quality monitoring for chemical hazards. There are small expensive devices using reverse osmosis that can be used to purify personal drinking water, but they are impractical for wide use in areas where local drinking-water supplies are contaminated.

Sanitary wastewater treatment is also focused on *(a)* the presence of pathogenic bacteria, and *(b)* the large amount of particulate organic matter that requires oxygen to decompose, known as the *biological oxygen demand* (BOD). Biological oxidation in standard sewage treatment plants is the current method of choice. Typically, raw sewage is filtered with coarse screens and passed through settling tanks to remove large particles that form sludge on the bottom of the tanks. The water phase is then aerated, which produces more particles and sludge. The bacteria are mostly collected in the sludge. The sludge is passed to an anaerobic digester, where more of the biological oxygen demand is removed and many of the bacteria are killed. The water continues to a trickle filter that has a bacterial biofilm (slime) on a solid phase to remove much of the remaining soluble organic waste, followed by a sand filter. In some cases, there is a final disinfection tank to minimize releases of viable organisms. These steps constitute tertiary treatment, which can remove almost 100% of suspended solids and approximately 99% of biological oxygen demand. Additional steps, such as pH control, addition of complexing agents, and air stripping, can remove inorganic and volatile hydrocarbons. Many areas in developing countries do not have any sewage treatment or only primary treatment to remove suspended solids.

The beneficial bacteria in digesters are vulnerable to releases of chemical wastes, as illustrated in the following case:

A chrome-plating shop dumped its acid bath directly into a sanitary sewer and shut down the Salem, Massachusetts, municipal treatment plant, with considerable hazard to workers. The strong acid and toxic metals killed the bacteria and produced a major release of hydrogen sulfide from the sludge and the anaerobic sludge digester. Fortunately, the plant workers escaped without harm when they were warned by alarms from sensors detecting high concentrations of hydrogen sulfide. Several weeks were needed to clean out the system and restart the treatment process, during which raw sewage went into Salem Harbor.

Food Contamination and Sanitation

Food animals and plants can become contaminated by ingestion or absorption of hazardous materials. They can also be contaminated externally by contact with contaminants. Bioaccumulation of aquatic contaminants can be a hazard to humans and other animals. Plants can absorb metals, such as lead and cadmium, from the soil. Pesticides can contaminate the surface of food. Many pesticides are biodegradable, but sufficient time and appropriate environmental conditions must occur to reduce pesticide residues to safe levels. Food crops are tested routinely for pesticide levels and other contaminants; there are strict requirements for allowable levels of pesticides in food, but only a tiny fraction of food crops is tested.

Animal carcasses are visually inspected for quality by government inspectors in many countries. However, the frequency and extent of chemical testing of food items is very limited. When testing is done, it is generally focused on detecting extensive contamination, so limited and localized contamination can easily pass undetected. This situation was highlighted by

the testing for mad cow disease in the United States in 2003, when only a few thousand animals were tested out of the millions processed into meat. Testing is expensive and adds to the complexity of the food distribution system for producers. Unless testing is performed by government agencies, it is likely to be very limited.

Contamination of food items by biological agents, such as bacteria and molds, is common and well recognized as a hazard. Most do not produce disease, but some infectious agents may be transmitted by food. Similarly, it is extremely difficult to prevent contamination of food by materials from rodents and insects. Consumers routinely reject food that has visible contamination or has been exposed to insects. Handling and preparing food under sanitary conditions can minimize contamination and subsequent adverse health effects. Many texts extensively discuss foodborne illness and its prevention. (See Chapter 9.)

Consumer Products

Adverse health effects can occur due to exposure to very low levels of chemicals used in consumer products. Biomarkers of exposure to common pesticides and chemicals used in consumer and personal care products can be detected in almost all people living in the United States.[11] Although detection of a chemical in the body does not prove it is causing a toxic effect, even low levels of some chemicals may cause cancer, birth defects, endocrine disruption, and other adverse health effects.

Solid Waste and Land Pollution

For centuries, people have disposed of many types of wastes in landfills, or "dumps." As populations have grown, especially in large cities, the amount of waste has become overwhelming, sparking recycling programs for paper products, plastics, metals, and petroleum products. As our consumer culture has grown, the composition of materials in landfills has broadened and its complexity has increased. Today's new home computer will be tomorrow's toxic waste because of guaranteed obsolescence and the toxic materials it contains. Developing countries are also experiencing these problems, due to increased consumption and due to developed countries sending them toxic materials, such as used electronic equipment, for disposal.

Surface water and groundwater have been contaminated by runoff and leakage from improperly discarded wastes. Often unrecognized are the potential hazards from small amounts of toxic materials in homes, such as cadmium batteries, paint solvents, pesticides, and cleaners. It is easier to recognize industrial waste problems from large producers of waste. In developed countries, strong regulations generally control releases of industrial wastes, and most large industrial companies reclaim and recycle wastes. However, small firms lack the knowledge about hazards and the resources to control them. (See Chapter 10.)

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27

Occupational Ergonomics: Promoting Safety and Health through Work Design

W. Monroe Keyserling

Occupational ergonomics is the study of people at work to understand the complex interrelationships with their work environments (including facilities, equipment, furniture, and tools), job demands, and work methods. All work activities place physical, mental, and psychosocial demands on workers. If these demands are kept within reasonable limits, work performance will likely be satisfactory and workers' health and well-being will likely be maintained. However, if demands are excessive or poorly matched to the capabilities or expectations of workers, errors, injuries, and decrements in physical or mental health may occur.

Some work-related injuries, such as amputations, fractures, and burns, occur suddenly in association with overt events (such as entrapments, falls, collisions, and explosions), where the resulting energy exchange produces immediate and discernible tissue damage (see Chapter 15). Other injuries, including many work-related musculoskeletal disorders (MSDs), are cumulative, resulting from low-level chronic exposures, such as repetitive and/or prolonged motions or awkward postures, and the onset of symptoms can be delayed for years.[1] (See Chapter 16.)

Occupational ergonomists evaluate work demands and corresponding abilities of people to react and cope. The goal of an occupational ergonomics program is to maintain a safe work environment by designing facilities, furniture, machines, tools, and job demands to be compatible with workers' attributes, such as size, strength, aerobic capacity, information-processing capacity, and expectations. A successful ergonomics program should simultaneously improve health and enhance productivity. Conversely, failure to consider ergonomics in the design of work environments and job demands can lead to increased risk of both sudden-onset injuries and chronic MSDs.

ERGONOMIC APPROACHES

Ergonomic approaches can be applied to preventing injuries, MSDs, and excessive fatigue and discomfort (Box 27-1).

Prevention through Design

Prevention through design (PtD) is a process being promoted by the National Institute for Occupational Safety and Health (NIOSH)[2] and

Box 27-1. Ergonomic Approaches to Prevention

Preventing Sudden-Onset Injuries

- Designing a machine guard that allows a worker to operate equipment with smooth, comfortable, and time-efficient motions. This reduces inconveniences introduced by the guard and decreases the likelihood that it will be bypassed or removed, thus exposing the worker to mechanical hazards that may cause serious fatal or disfiguring injuries. A well-designed guard may also eliminate awkward postures that lead to musculoskeletal disorders (MSDs) in vulnerable body parts, such as the lower back, shoulders, and upper extremities.
- Evaluating the mechanics of human gait to determine forces acting between the floor surface and the sole of the shoe. This information is used to determine friction required to reduce the risk of a slip or fall. Falls can also be prevented by eliminating slip and trip hazards, such as puddles of oil on the floor, uneven floor surfaces, and changes in floor elevation. In situations where these hazards cannot be completely eliminated, ergonomic principles can enhance the ability of workers to perceive and react to these hazards by providing good lighting and contrasting surface colors.
- Designing warning signs for hazardous equipment and work locations so that workers take appropriate actions to avoid accidents. Warnings are particularly important for visitors, inexperienced workers, and contract workers, or if the hazards are hidden or subtle.

Preventing Musculoskeletal Disorders Caused by Overexertion or Overuse

- Evaluating lifting tasks to determine biomechanical strain on the lower back, and designing lifting tasks to prevent back disorders.
- Evaluating workstation layouts to discover causes of postural stress, such as torso bending and twisting and overhead work with arms and hands, and implementing changes in workstation layout to eliminate awkward work postures that may lead to MSDs in the trunk and shoulders. Eliminating awkward postures may also reduce fatigue and enhance performance.
- Evaluating highly repetitive, manual assembly-line jobs and developing alternative hand tools and work methods to reduce the risk of cumulative trauma disorders of the upper extremities, such as tendonitis, epicondylitis, tenosynovitis, and carpal tunnel syndrome.

Preventing Excessive Fatigue and Discomfort

- Designing equipment and furniture of a computer workstation and associated tasks so that an operator can use a monitor, mouse, and keyboard for extended periods without experiencing visual fatigue or musculoskeletal discomfort. Discomfort in the back, neck, or upper extremities may be a precursor of potentially disabling problems, such as tendonitis or carpal tunnel syndrome.
- Evaluating the metabolic demands of a job performed in a hot, humid environment to develop a work–rest regimen that prevents heat stress (see Chapter 12B).
- Establishing maximum work times for transportation workers, such as truck drivers and airline pilots, to reduce the risk of drowsiness, performance errors, and accidents caused by sleep deprivation.

other agencies and professional organizations to prevent occupational injuries and illnesses by eliminating, or substantially reducing, hazards early in the job-design process. Prevention through design is intended to cover the entire life cycle of facilities, equipment, and processes, including construction, operation, maintenance, and dismantling and disposal. Ergonomists are an important part of the PtD team and consider a broad range of issues, including the following:

- *Physical ergonomics of production tasks*: Assuring that the physical demands of the job, such as force, posture, and energy expenditure, do not exceed the capability and capacity of workers.
- *Cognitive ergonomics of production tasks*: Assuring that the mental and information-processing demands of the job are matched to capabilities and expectations, thus reducing the likelihood of human error that could lead to injuries and other losses.
- *Ergonomics of maintenance tasks*: This includes *(a)* assuring that there is adequate physical space to safely perform maintenance activities in biomechanically efficient work postures; *(b)* designing tools that allow workers to perform maintenance tasks without exerting excessive force; and *(c)* designing lock-out and energy-control systems so that diagnosis and repair of equipment problems can be performed without risking overt traumatic injuries. In addition, developing robust preventive maintenance programs and standard operating procedures for diagnosis and repair reduces the likelihood of errors that may result in injury and extended production interruptions.

Accommodating Persons with Disabilities

The previous examples focused primarily on preventing situations and workplace exposures that could cause death, injury, discomfort, or fatigue. Ergonomic methods and principles can be used to assist workers with disabilities who may need special accommodations to work safely, effectively, and comfortably, such as the following:

- Fire alarms with strobe lights to warn people who are hearing impaired
- Computers equipped with voice-recognition hardware and software to accommodate persons who have lost the use of their hands, or where traditional data-entry devices (keyboards and mice) cause or aggravate upper-extremity MSDs
- Accommodations for older workers because many human capabilities, such as vision, hearing, balance, aerobic endurance, reaction time, and strength, begin to slowly decline starting at about age 40

Ergonomists involved in designing and implementing workplace modifications to accommodate people who have become disabled work as members of multidisciplinary teams that also include physiatrists, psychologists, physical and occupational therapists, and rehabilitation engineers. These teams establish work environments and job demands that are matched to specific capabilities of disabled workers, allowing them to return to work.

The remainder of this chapter describes several subdisciplines of ergonomics concerned with occupational safety and health.

COGNITIVE ERGONOMICS

Cognitive ergonomics (human factors engineering or engineering psychology) is concerned with the perceptual, information-processing, and psychomotor aspects of work. Engineering psychologists design displays, controls, procedures, software, equipment, warning signs, alarms, and general work environments to improve work performance and reduce human error that can lead to injuries, and production and property losses. Common causes of human error include the following:

1. *Failure to perceive or recognize a hazardous condition or situation*: To react to a dangerous situation, a worker must first perceive that danger exists. Many workplace hazards are not easily perceived through vision, hearing, smell, or touch. Examples include excessive pressure inside a boiler, a fork truck or automatic guided vehicle (AGV) approaching from behind in a noisy factory, uneven flooring in a poorly lit room, and a sudden release of an odorless, colorless toxic gas. These situations require special displays or alarms. Boilers should be equipped with gauges that display internal pressure and audible alarms that activate when pressure exceeds safe limits. A simple display can be enhanced by trend information, indicating the direction and rate of pressure changes over time. To protect pedestrians, fork trucks and AGVs must have beepers and flashing lights that operate when the vehicle moves. Good lighting is required near trip hazards, and alarm systems should sound if toxic gases are released. Warning signs at locations with concealed hazards, such as confined-space entry points, enhance awareness so that appropriate preparation and prevention actions are taken.
2. *Failure in information-processing and/or decision-making processes*: Decision making involves combining new information with existing knowledge to provide a basis for action. Errors can occur if an information-processing load is excessive. For example, during the nuclear power plant accident at Three Mile Island, Pennsylvania, in 1979, operators were required to react to multiple alarms and interpret a complex array of informational displays, making it difficult to prioritize actions required to stabilize the reactor. Information overload resulting from performing multiple concurrent tasks, such as texting or using a mobile phone while operating a vehicle, can cause distraction and loss of situational awareness, potentially resulting in a crash. Decision errors

can also occur if critical information is unavailable, incomplete, or misleading, or if previous training was incorrect or inappropriate for addressing a specific situation. As a result of the terrorist attacks on the World Trade Center, changes were made to training and communication systems to enhance the flow of information among firefighters, police, emergency medical service workers, and other responders.

3. *Failures in motor actions following correct decisions*: Following a decision, it is frequently necessary for a worker to perform a motor action, such as flipping a switch or adjusting a knob to control the status of a system or machine. Problems can occur if required actions exceed motor abilities. For example, the force required to adjust a control valve in a chemical plant should not exceed workers' strength. Errors can occur if controls are not clearly labeled or if manipulation of controls cause unexpected responses. Switches that start potentially dangerous machinery or equipment should be guarded to prevent unintentional activation, by covering them, locking them in the "off" position during maintenance and other activities, or placing them in locations where they cannot be accidentally touched.

WORK PHYSIOLOGY

Physical work, such as walking, carrying, lifting, or gripping occurs as the result of contractions of muscle. Work physiology is the branch of ergonomics concerned with the responses of the cardiovascular system, pulmonary system, and skeletal muscles to the metabolic demands of work. If demands exceed metabolic capacities, workers will likely experience excessive fatigue of a few muscles or their entire bodies.

Localized Muscle Fatigue

Local muscle fatigue is associated with work activities that require a body segment to perform sustained static work, high-intensity work, or highly repetitive work. It may result from sustained awkward posture, such as when automobile mechanics flex their torsos while working in engine compartments, or when electricians elevate their shoulders for prolonged periods when reaching overhead to install wires. In other instances, static work may involve short-duration, forceful exertions, such as using a tire iron to unfreeze a rusted lug nut when changing a tire. Local muscle fatigue may be an early indicator of work-related MSDs. (See Chapter 16.)

When a muscle contracts, internal blood vessels are compressed. Because vascular resistance increases with the level of muscle tension, the blood supply to the working muscle decreases. Without periodic relaxation, the demand for metabolic nutrients and oxygen exceeds the supply, and metabolic wastes accumulate. In the short term, ischemic pain, tremor, and reduced strength may occur; in the long term, injuries may occur.[3,4]

Figure 27-1 shows the relationship between the intensity and duration of a static exertion.[5] A contraction of maximum (100%) intensity can be sustained for about 6 seconds; a contraction of 50% of maximum intensity can be sustained for about 1 minute. To sustain a static contraction indefinitely, muscle tension must be

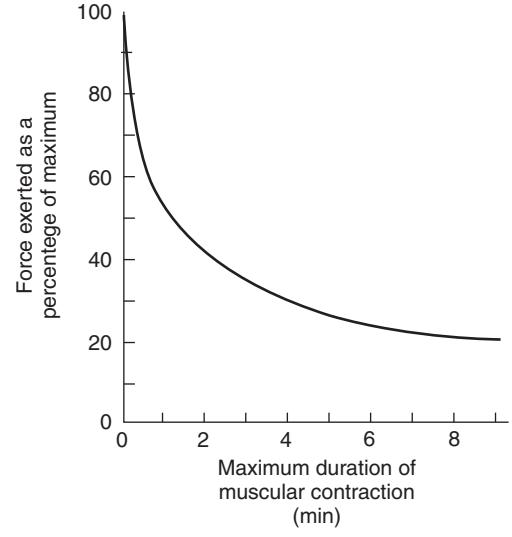

Figure 27-1. Maximum duration of a static muscle contraction for various levels of muscular contraction. (Source: Keyserling WM, Armstrong TJ. Ergonomics. In: Last JM (ed.). Maxcy-Rosenau public health and preventive medicine (12th ed.). Norwalk, CT: Appleton, Century, Crofts, 1986, pp. 734-750.)

kept below 15% of maximum intensity. The endurance curve in Figure 27-1 reflects time to exhaustion. Since workers should not exert themselves to the point of exhaustion, static work demands should stay below the curve. Sustained hand-grip exertions as low as 10% of maximum strength can produce residual muscle fatigue after 24 hours.[6] Work activities should be designed so that static exertions are of limited duration and adequate recovery time is built into the job. Dynamic activities involving cyclical contraction and relaxation of working muscle are generally preferable to static work.

Dynamic Work and Whole-Body Fatigue

Whole-body dynamic work occurs when large skeletal muscle groups repeatedly contract and relax while performing a task. Common examples of dynamic work include walking on a level surface, pedaling a bicycle, climbing stairs, shoveling snow, and carrying a load.

The intensity of whole-body dynamic work is limited by the capacity of the respiratory and cardiovascular systems to deliver adequate supplies of oxygen and glucose to working muscles and remove products of metabolism. Whole-body fatigue occurs when the collective metabolic demands of working muscles throughout the body exceed this capacity. Symptoms of whole-body fatigue include shortness of breath, weakness in working muscles, and a general feeling of tiredness. These symptoms continue and may increase until the work activity is stopped or decreased in intensity.

For extremely short durations of whole-body dynamic activity (generally 4 minutes or less), people can work at intensities equal to their aerobic capacities before a rest break is required. As the duration of work increases, the intensity must decrease. For a 1-hour work period, the average energy expenditure should not exceed 50% of the worker's aerobic capacity. For an 8-hour shift, the average energy expenditure should not exceed 33% of the worker's aerobic capacity.[7]

Aerobic capacity varies considerably within the population. Aerobic capacity peaks in the third decade (20 to 29 years) for both men and women. At age 20, average aerobic capacity is 15 kcal/min for untrained males and 11 kcal/min for untrained females. At age 50, these values decrease to 12 and 8 kcal/min for males and females, respectively. By age 70, the values drop to 9 and 6.5 kcal/min.[8] These are average values for each age-and-gender group and do not reflect the full range of variability among adults. This variability is an important consideration when evaluating ergonomic stress; a job that is relatively easy for a person with high aerobic capacity can be extremely fatiguing for a person with low capacity.

The prevention of whole-body fatigue is accomplished through good work design. The energy demands of a job should be sufficiently low to accommodate the adult working population, including persons with limited aerobic capacity. This can be accomplished by designing the workplace to minimize unnecessary body movements (excessive walking or climbing) and providing mechanical assists, such as hoists or conveyors for handling heavy materials. If these approaches are not feasible, it may be necessary to provide additional rest allowances to prevent excessive fatigue, especially in hot, humid work environments due to the metabolic contribution to heat stress. (See Chapter 12B.)

In establishing metabolic criteria for jobs that involve repetitive manual lifting, NIOSH recommends that the average energy expenditure during an 8-hour work shift should not exceed 3.5 kcal per minute.[9–11] Caution should be practiced when placing persons with low levels of physical fitness on metabolically strenuous jobs.*

To assess the potential for whole-body fatigue for a specific job, one must determine the energy expenditure rate, by one of the following three methods:

1. *Table reference*: One can refer to extensive tables of the energy costs of various work activities. (The text by McArdle, Katch, and Katch cited in the Further Reading

* Aerobic capacity can be determined by measuring oxygen uptake and carbon dioxide production during a stress test. For additional information on measuring or estimating aerobic capacity, see the text by McArdle, Katch, and Katch, cited in the Further Reading section.

section provides tables describing the energy costs of many work tasks.)
2. *Indirect calorimetry*: One can estimate energy expenditure for a specific job by measuring a worker's oxygen uptake while performing the job.[8,12]
3. *Modeling*: One can analyze a job and break it down into fundamental tasks, such as walking, carrying, and lifting. Parameters describing each task can be inserted into equations to predict energy expenditure.[13]

There is no one best method for determining energy expenditure. The selection of a method is often a trade-off between *(a)* the availability of published tables or prediction equations for the specific work activities of interest, and *(b)* the time and expenses associated with data collection for indirect calorimetry, which is necessary to obtain a precise measure of energy expenditure.

BIOMECHANICS

Biomechanics is concerned with the properties of tissue and its response to mechanical stresses. Some injury-causing mechanical stresses in the workplace are caused by overt, sudden-onset events, such as crushed foot bones caused by the impact of a dropped object. The hazards that produce these injuries can usually be controlled through safety engineering techniques. (See Chapter 15.) Other mechanical stresses are more subtle and often do not cause injuries that are immediately perceptible. Work-related overexertion MSDs frequently affect the lower back, neck, shoulders, and upper extremities. They include sprains, strains, tendonitis, bursitis, and carpal tunnel syndrome. Because these disorders can impair mobility, strength, tactile capabilities, and motor control, affected workers may be unable to perform their jobs. Primary risk factors for overexertion injuries and disorders include the following:

- Forceful exertions
- Awkward postures
- Localized mechanical contact stresses
- Vibration
- Temperature extremes[14-18]

All of these risk factors are modified by repetition (frequency of exposure) to these risk factors and duration (total time of exposure to them).

In addition to identifying the presence of these risk factors, ergonomic job analysis evaluates specific job attributes, such as workstation layout, production standards, incentive systems, work organization, and work methods—all of which affect the magnitude, frequency, and duration of worker exposure to these risk factors. This information needs to be obtained to design and implement job modifications effectively.

Forceful Exertions

Whole-body exertions, such as strenuous lifting, pushing, and pulling, can cause back pain and other injuries and disorders (Fig. 27-2). Because the lifting and handling of heavy weights are the most commonly cited activities associated with occupational low back pain, NIOSH[9–11] and the American Conference of Governmental Industrial Hygienists (ACGIH)[19] have issued guidelines for the evaluation and design of jobs that require manual lifting. These guidelines consider factors, such as lift frequency, work duration, workplace geometry, and posture to establish the amount of weight that a worker can lift safely. Factors other than object weight play a significant role in the amount of force that workers can safely exert during lifting and other manual transfer tasks. Due to the effect of long moment arms, handling relatively light loads can stress muscles in the back and shoulder if loads are held at a long horizontal distance in front or to the side of the body (Fig. 27-3).

The following approaches may help reduce the magnitude of forces exerted during whole-body exertions:

- Reducing the weight of an object by decreasing the size of a unit load, such as by placing fewer parts in a tote bin or purchasing smaller bags of powdered or granular materials
- Reducing extended reach postures by removing obstructions that prevent a worker from getting close to the lifted object
- Using mechanical aids, such as conveyors, hoists, conveyors, and articulating arms, to assist a worker and/or eliminate the manual exertion (Fig. 27-4)

Figure 27-2. The load carried by this worker exceeds 50 kg (110 lb). A mechanical assist device, such as an overhead hoist, would reduce the risk of back injuries on this job. (Photograph by W. Monroe Keyserling.)

Forceful exertions of the hands, such as cutting with knives or scissors, tightening screws, "snapping" together electrical connectors, and using the hands or fingers to sand or buff parts, can cause upper-extremity disorders, such as tendonitis and carpal tunnel syndrome.[14–18] Each of the following can increase forces exerted by the finger flexor and extensor muscles and tendons: pinch grips, heavy tools, poorly balanced tools, poorly maintained tools (such as dull knives or scissors), or low friction between a hand and a tool handle. Gloves may increase force requirements of some jobs due to reduced tactile feedback, reduced friction, or resistance of gloves to stretching or compression. Environmental conditions may also increase force requirements; for example, some rubber and plastic materials lose their flexibility when cold and become more difficult to shape or manipulate. The following approaches help reduce the forcefulness of hand exertions:

- Substituting power tools for manual tools: If a power tool is not feasible, redesigning the manual tool to increase mechanical advantage or otherwise decreasing required hand forces
- Suspending heavy tools with "zero-gravity" balance devices
- Treating slippery handles with friction-enhancing coatings to minimize slippage and reduce hand force
- Moving the handle of an off-balance tool closer to the center of gravity or suspending the tool so that off-balance characteristics are minimized
- Using torque-control devices (reaction arms or automatic shut-offs) on power tools, such as air wrenches, nut runners, or screw drivers (Fig. 27-5)
- If high force is required to engage tightly fitting parts, improving quality control to achieve a better fit, and/or using a lubricant to reduce force
- Prewarming rubber and plastic components if these become cold and unmalleable during storage

Awkward Posture

Awkward posture at any joint may cause transient discomfort and fatigue. Prolonged awkward postures may contribute to disabling injuries and disorders of musculoskeletal tissue and/or peripheral nerves. Awkward trunk postures (Fig. 27-3 and 27-6) increase the risk of back injuries.[15,17,20] Raising the elbow above shoulder height or reaching behind the torso can increase the likelihood of MSDs in the neck and shoulders. The workers shown in Figures 27-7A and 27-7B must position their arms awkwardly due to poor workstation layout.

Most awkward postures of the trunk and shoulder result from excessive reach distances, such as bending into bins to place or retrieve parts, reaching overhead to high shelves and conveyors, or reaching overhead or in front of

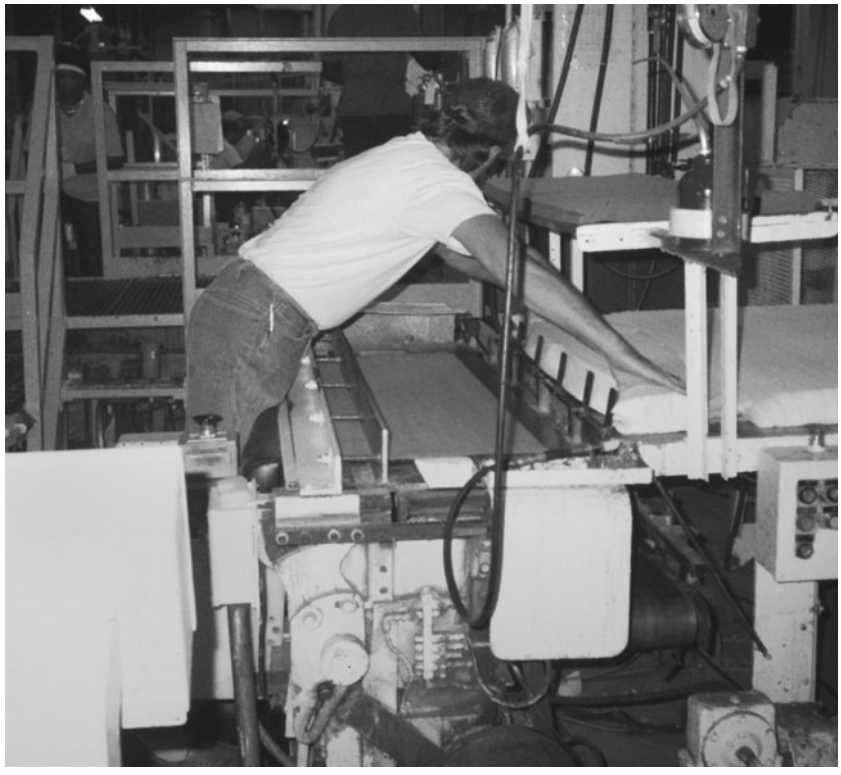

Figure 27-3. Although the lifted load is relatively light (approximately 8 kg), the combination of forward bending and lifting places a high load on the spine, increasing the risk of a back injury. (Photograph by W. Monroe Keyserling.)

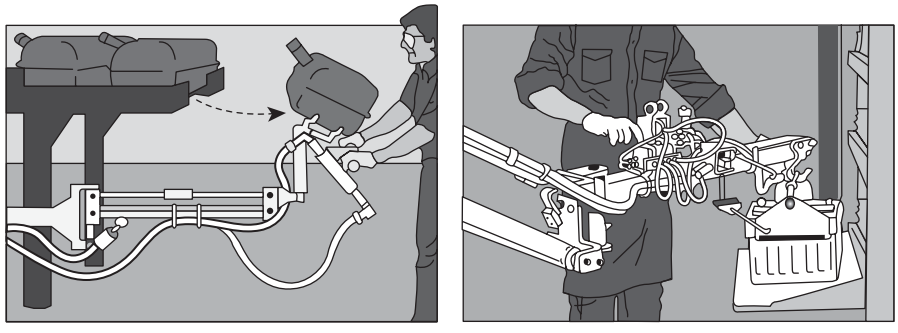

Figure 27-4. Mechanical assist devices can reduce or eliminate forceful exertions during manual materials handling activities such as lifting or carrying. (Courtesy of the University of Michigan and the UAW/Ford Joint National Committee on Health and Safety.) (Source: The University of Michigan Center for Ergonomics. Fitting jobs to people: an ergonomics process. Ann Arbor: The Regents of the University of Michigan, 1991.)

the body to activate machine controls. These postures can be eliminated through improved workstation layout. In general, workers should not reach below knee height or above shoulder height for prolonged periods. Routine forward reaches should be performed with the trunk upright and the upper arms nearly parallel to the trunk. Where possible, workstations and equipment should offer adjustability to accommodate workers of different body sizes—using anthropometry, the

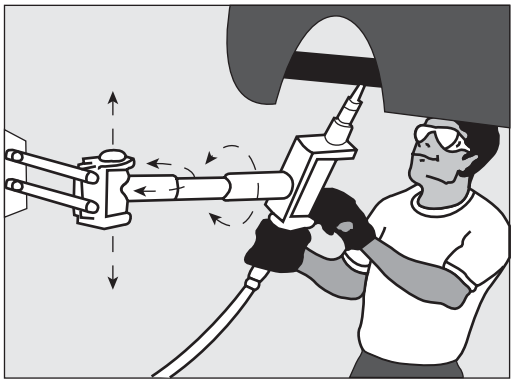

Figure 27-5. Torque control devices can substantially reduce the amount of force exerted when using air wrenches and similar tools. Note that the weight of the tool is also borne by the device, further reducing the force exerted by the worker. (Courtesy of the University of Michigan and the UAW/Ford Joint National Committee on Health and Safety.) (*Source*: The University of Michigan Center for Ergonomics. Fitting jobs to people: an ergonomics process. Ann Arbor: The Regents of the University of Michigan, 1991.)

branch of ergonomics concerned with designing facilities and equipment to accommodate work populations of varying body dimensions. (See the textbook by Pheasant and Haslegrave in the Further Reading section.)

Allowing workers to sit reduces fatigue and discomfort in the legs and feet, and can increase the stability of the upper body, which is necessary for highly precise manual tasks. However, prolonged sitting may lead to back pain. A well-designed work seat, such as one with good lumbar support and adjustability of the seat pan and backrest, enhances comfort and can reduce the risk of MSDs. Layouts that allow workers to alternate between standing and sitting postures are also desirable. Certain jobs, such as working at a moving conveyor line or in a job that requires constant movement among different workstations, cannot be performed while sitting. In situations where workers are required to stand for most of the work shift, anti-fatigue matting and

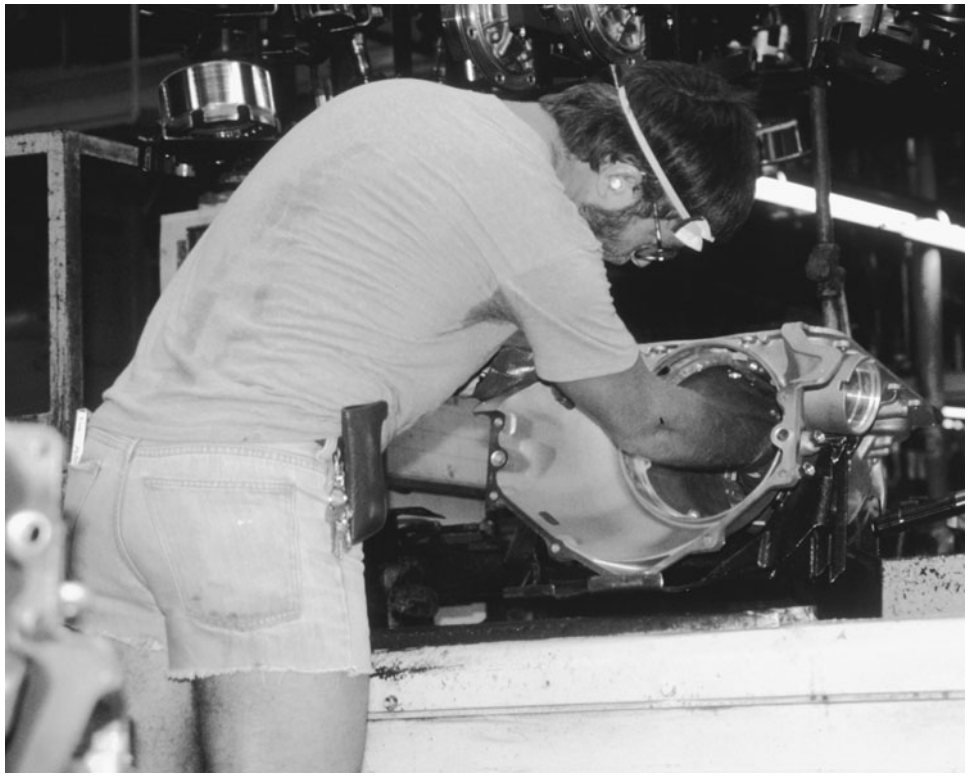

Figure 27-6. This assembly line worker must twist and laterally bend his back in order to see his hands to install a part. (Photograph by W. Monroe Keyserling.)

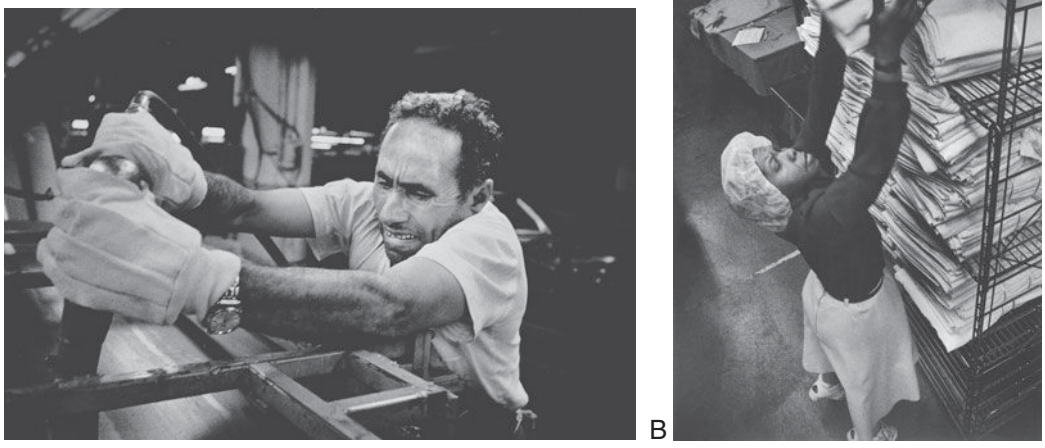

Figure 27-7. Both of these workers are at risk of musculoskeletal disorders: (A) The worker on this job exerts high hand force with elevated shoulders, increasing the risk of shoulder and upper extremity injuries. (B) This laundry worker is at increased risk of upper-extremity and back injuries as a result of a poorly designed work task. (Photographs by Earl Dotter.)

shoe inserts have been shown to enhance comfort in the legs and lower back.[21] In addition, workers should be encouraged and allowed to sit during temporary production interruptions.

Awkward upper-extremity postures can occur at the shoulder, elbow, or wrist. Workers should avoid frequent or prolonged activities that require them to bend their wrist. Jobs that involve precise manipulation to align and position handled parts and materials frequently require substantial wrist deviations (Fig. 27-8). Features of hand tools, such as the shape and orientation of handles, together with workstation layout (location and orientation of work surfaces) strongly influence wrist postures.

Localized Contact Stresses

Local mechanical stresses result from concentrated pressure during contact between body tissues and objects and tools. "Hand hammering" (using the palm as a striking tool) is used in some manufacturing and maintenance tasks as a method for joining two parts. This activity, which can irritate nerves and other parts of the palm, can be avoided by using a mallet. Hand tools with hard, sharp, or small-diameter handles, such as knives, pliers, and scissors, can

Figure 27-8. Garment workers must repetitively use awkward wrist and shoulder postures when operating sewing machines. (Photograph by Earl Dotter.)

irritate nerves and tendons in the palm and fingers. This problem can be controlled by padding and/or increasing the radius of curvature of handles. In some bench-assembly activities and office jobs, contact stresses result from resting forearms or wrists against a sharp, unpadded edge of a work bench. This problem can usually be controlled by rounding or padding the edge, or by providing a support for forearms and wrists.

Seated workstations that produce localized pressure on the posterior knee and thigh can impair circulation, causing swelling and discomfort in the lower legs, ankles, and feet. A common cause of this condition is a work seat that is too high, allowing the lower legs to dangle, producing concentrated compressive forces on tissues where the thighs contact the front edge of the seatpan. Solutions to this problem include adjustable seats and providing a footrest to partially support the weight of the legs.

Vibration

Exposure to whole-body vibration that occurs while driving or riding in motor vehicles, including fork trucks and off-road vehicles, may be a factor that increases the risk of back pain.[15,17] Because driving tasks are usually performed in a seated posture, most drivers are exposed to two back pain risk factors. Driving over rough surfaces for prolonged periods, while sitting in a poorly suspended seat, can increase vibration exposure.

Localized vibration of the upper extremity (segmental vibration) can occur when using powered hand tools, such as screwdrivers, nutrunners, grinders, jackhammers, and chippers. Segmental vibration may contribute to the development of hand-arm vibration syndromes, such as vibration white finger.[15,17] (See Chapter 12A.) Careful selection of proper tools can help to reduce exposure. For example, an air wrench that uses an automatic shut-off system produces less exposure to vibration than a slip-clutch mechanism. Many manufacturers offer a variety of low-vibration hand tools.

Temperature Extremes

Exposure to unusually hot or cold ambient temperatures can produce a variety of adverse health effects. (See Chapter 12B.) In addition to considering the general thermal characteristics (air temperature, air movement, and relative humidity) of the workplace, one should consider temperature extremes that affect the hands. For example, handling extremely hot or cold parts may require the use of special gloves that increase the force requirements of the job. In jobs that involve the use of pneumatic tools, air from high-pressure lines and tool exhaust ports may be directed onto the hands, causing local chilling and reducing manual dexterity and tactile sensitivity. This exposure can be controlled by eliminating leaks and directing exhaust air away from the hands.

Repetitive and Prolonged Activities

Biomechanical and physiological strains experienced by workers are related to cumulative exposure to all the risk factors discussed earlier.[14–17] Because ergonomic risk factors are often related to specific work tasks, jobs that involve high repetition and/or duration, such as driving 5,000 screws a day on an assembly line or continuously performing data entry in an office, typically involve higher exposures than nonrepetitive jobs, such as inspection work in a factory or a supervisory position in an office. Repetitiveness is not a risk factor limited to upper-extremity problems. For example, frequent lifting and repetitive and/or prolonged use of awkward trunk postures increase the risk of back pain.

Repetitiveness can often be measured or estimated using industrial engineering records and other work standards. For example, on an assembly line, repetitiveness is a function of the line speed or the time allowed to complete one unit of work. For a clerk in a bank or insurance office, repetitiveness can be a function of the number of forms processed a day. For a supermarket checker, repetitiveness is a function of the number of items scanned over the course of a work shift. Repetitiveness can also be measured using an observational technique, where the rapidity and intensity of hand motions are compared against benchmarks or a scale with verbal anchors.[22] (A verbal anchor is a word or phrase that describes the speed or urgency of the hand motions required to perform a job.) An ordinal scale with verbal anchors for describing the

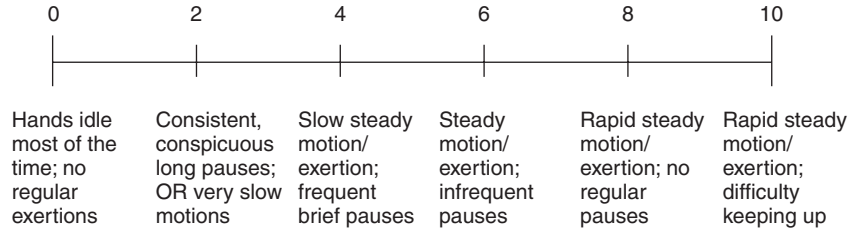

Figure 27-9. Visual analog scale for rating repetition/hand activity with verbal anchors. (Source: Latko WA, Armstrong TJ, Foulke JA, et al. Development and evaluation of an observational method for assessing repetition in hand tasks. American Industrial Hygiene Association Journal 1997; 58: 278-285.)

repetitiveness of hand-intensive work is presented in Figure 27-9. This scale has been incorporated into an ACGIH threshold limit value (TLV) for evaluating worker exposure to repetitive hand activities.[19] Additional techniques for assessing repetition, combined with other risk factors for distal upper-extremity MSDs include the Strain Index[23] and the Rapid Upper Limb Assessment (RULA).[24]

Two approaches to resolving problems of repetition and prolonged exertions are job enrichment and job rotation. The premise behind these approaches is to increase the overall variety of activities performed by a worker to reduce the repetitiveness of any specific stressful activity. While good in theory, these approaches may be very difficult to implement. Job enrichment and job rotation will not be feasible in workplaces where there are no "low-repetition" jobs to combine with the "high-repetition" jobs. Even in situations with a good mix of low- and high-repetition jobs, there may be other factors, such as increased learning time and seniority restrictions, that present significant barriers. In these situations, it may be necessary to establish a participative ergonomics program and to educate management and workers before attempting these interventions.

COMPONENTS OF AN ERGONOMIC PROGRAM

An effective ergonomics program starts with the commitment and involvement of management to provide the organizational resources and motivation to control ergonomic hazards in the workplace. Management must also perform regular reviews and evaluations of the program to assure program goals are met in a deliberate and timely manner. Because ergonomic programs focus on improving the complex interrelationships among workers and their jobs, employee involvement is essential to assuring the success of the program.[25]

An effective program should include the following components:

- Reviews of health and safety records to identify patterns of overexertion injuries and illnesses. In some instances, these records are incomplete, so it is difficult to establish direct links between outcomes and specific work exposures. When this occurs, it is necessary to supplement review of archival records with plant walkthroughs and interviews of workers, supervisors, and/or ergonomic teams to identify specific work locations with excessive exposure to ergonomic risk factors.[26] In addition, record keeping should be improved so that overexertion injuries and disorders can be linked to specific jobs and workstations.
- Training of managers, engineers, and other workers in recognizing and controlling ergonomic risk factors
- Job analysis to identify worker exposures to risk factors that cause overexertion injuries and illnesses
- Job design, and redesign if necessary, to reduce or eliminate ergonomic risk factors
- Medical management of injured workers to improve the possibility of a timely return to work

Limited resources must be directed at those jobs with the greatest ergonomic problems.

One approach for identifying high-hazard jobs is to analyze available medical, insurance, and safety records, such as workers' compensation payments and Occupational Health and Safety Administration (OSHA) logs, for evidence of high rates of overexertion disorders in certain departments, job classifications, or workstations. This approach is called *passive surveillance* because it relies on previously collected information. Passive surveillance may underestimate the true level of cumulative trauma disorders. For example, at small plants that do not have in-plant medical services, workers may seek treatment from their personal physicians. Unless workers request coverage under the workers' compensation system, their complaints and associated treatment may not appear in any company records. *Active surveillance* involves a more aggressive approach to identifying potential problems. It may include employee surveys to identify jobs associated with elevated rates of discomfort in the back, neck, shoulders, and upper extremities. It may also include interviews with supervisors and personnel managers to identify jobs with high turnover. If other employment opportunities are available, workers often seek relief by leaving jobs with unusually high physical stresses before a cumulative trauma disorder develops.

Once high-risk jobs have been identified, the next step is to determine the specific causes of exposure so that corrective actions can be taken. This activity involves job analysis to identify the various risk factors discussed earlier and the development of engineering and/or administrative controls to reduce or eliminate exposures. The appropriateness of an intervention to reduce ergonomic stress will vary among and within facilities. Changes that are practical at one workstation may not be appropriate for others. Alternatives must be evaluated to determine the best strategy for resolving each ergonomic problem. It is also important to recognize that most solutions will require some degree of fine tuning to assure that they are acceptable to workers and accomplish the intended reductions in ergonomic stress. Follow-up job analyses should be performed to assure that solutions are effective and that no new stresses have been introduced. Follow-up health surveillance is also recommended to detect any changes in the pattern of injuries, illnesses, or employee complaints.

CONCLUSION

Occupational ergonomics is a multidisciplinary approach to workplace evaluation and design. The goal of an occupational ergonomics program is to establish and maintain a safe work environment by assuring that physical and cognitive demands are compatible with workers' attributes, capacities, and expectations. Ergonomists collaborate with health professionals, engineers, managers, and workers to identify and ameliorate conditions that can lead to both sudden-onset and chronic injuries. A successful ergonomics program should simultaneously improve safety, health, and productivity.

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Cohen AL, Gjessing CC, Fine LJ, et al. Elements of ergonomics programs: A primer based on workplace evaluations of musculoskeletal disorders. NIOSH Pub. No. 97-117. Cincinnati, OH: NIOSH, 1997.
This document presents general guidance for establishing and managing worksite-based ergonomic programs. Topics include organizational elements of effective ergonomics programs, training for managers and workers, health surveillance, job analysis, engineering interventions to reduce exposure to

ergonomic stress, and medical management. It includes many illustrations, examples, and references.

Kroemer KH, Grandjean, E. Fitting the task to the human: textbook of occupational ergonomics (5th ed.). London: Taylor and Francis, 1997.
A well-written survey text that covers all aspects of ergonomics. Chapters on fatigue, work physiology, anthropometry, biomechanics, and cognitive ergonomics provide an excellent introduction to these topics.

Marras WS, Karwowski W. The occupational ergonomics handbook (2nd ed.). Boca Raton, FL: CRC Press/Taylor & Francis Group, 2006.
This two-volume set provides comprehensive coverage of ergonomics in the work environment, with special emphasis on anthropometry, work physiology, biomechanics, and work-related musculoskeletal disorders. Volume 2 is particularly useful for occupational health professionals, with chapters that provide detailed description and discussion of quantitative assessment tools for performing job analyses to assess risk factors associated with lower-back and upper-extremity disorders.

McArdle WD, Katch FI, Katch VL. Exercise physiology: energy, nutrition, and human performance (6th ed.). Philadelphia: Lippincott Williams & Wilkins, 2006.
This comprehensive textbook covers a wide range of issues in work and exercise physiology. Early chapters cover basic exercise physiology (nutrition, energy conversion during exercise, structure and function of the pulmonary, cardiovascular, and neuromuscular systems) while advanced chapters cover applied topics, such as measurement of human energy expenditure, training for muscle strength and aerobic power, and rehabilitation training programs. Appendices include comprehensive tables of energy expenditure costs of common household, occupational, and recreational activities.

National Institute for Occupational Safety and Health. Applications manual for the revised NIOSH lifting equation. NIOSH Pub. No. 94-110. Cincinnati, OH: NIOSH, 1994.
A "hands-on" users' guide for evaluating work activities that require manual lifting. Numerous examples demonstrate application of the 1991 Revised NIOSH Lifting Equation in a variety of work environments. The guide includes a brief summary of the scientific basis of the 1991 Lifting Equation with references to biomechanical, physiological, psychophysical, and epidemiological research. The text is supplemented with numerous illustrations and examples.

National Research Council. Musculoskeletal disorders in the workplace: low back and upper extremities. Washington, DC: National Academies Press, 2001.
A comprehensive review of the scientific literature on the relationship between work and MSDs of the low back and upper extremities. Major sections include discussions of epidemiology, tissue pathology, biomechanics, and interventions. Summary tables provide descriptive synopses of key studies, and the list of references is extensive.

Pheasant S, Haslegrave C. Bodyspace: anthropometry, ergonomics, and the design of work (3rd ed.). Boca Raton, FL: CRC Press/Taylor & Francis Group, 2005.
This textbook provides comprehensive coverage of the anthropometric aspects of ergonomics. Introductory chapters describe methodologies for measuring and statistically summarizing human body dimensions. This is followed by an excellent presentation of how anthropometric principles are used to design furniture, equipment, and workstations in the home, office, and factory environments. Numerous examples, illustrations, and anthropometric tables make this text an indispensable reference book for both novice ergonomists and experienced ergonomic designers.

Wickens CD, Lee JD, Liu Y, Gordon-Becker SE. An introduction to human factors engineering (2nd ed.). Upper Saddle River, NJ: Pearson Prentice Hall, 2004.
This textbook provides a good introduction to all aspects of ergonomics with special emphasis on cognitive ergonomics. Introductory chapters cover human sensory mechanisms, displays, cognition, decision making, and design of controls. Advanced chapters cover a variety of topics, including human–computer interaction, human factors in transportation, usability testing, stress and work performance, and the role of human error in accidents.

28

Clinical Occupational and Environmental Health Practice

Gary Greenberg and Bonnie Rogers

Occupational and environmental health programs can range from those that are very comprehensive, offering a large array of services to the worker population, to those that provide focused services to meet mandatory regulatory requirements. While each organization will need to determine the types of programs and services that best meet the needs of its employees, the central tenets are as follows:

- To prevent work-related health hazards
- To foster health promotion and prevention strategies
- To facilitate worker job placement and monitor ongoing work compatibility within the context of physical and mental health capabilities
- To provide for health care and rehabilitation for work-related injuries and illnesses and to provide case management and other services
- To assess and monitor the work environment for health hazards and to participate in providing strategy recommendations for prevention and control of risks
- To enhance interdisciplinary collaboration for occupational health care and services
- To engage in decision making to help resolve ethical issues in occupational and environmental health

By applying occupational health principles to the workplace, all employees, including those who are disabled, are placed in jobs according to their abilities to perform the work. This approach also promotes continuing health care and rehabilitation of occupationally ill and injured workers. The achievement of these objectives benefits both employees and employers by improving health, morale, and productivity.

ASSESSING WORK AND THE WORK ENVIRONMENT

An important and practical aspect of occupational and environmental health is the clinician's awareness of work processes and related demands and potential hazards. Since many occupational health evaluations address specific permissions, clinicians need to know the health parameters of concern and to detect health-related situations of potential risk or prior harm.

To provide a well-informed and reality-based judgment regarding the fitness for workers to perform specific tasks, clinicians should routinely

visit workplaces and assess work processes. Such visits provide a huge advantage in determining work limitations and endorsements. Beginning with providing a useful vocabulary specific to the work being performed, workplace visits also instill insight into the pace, protection, and demands of individual assignments and hazards.

From a preventive perspective, experienced clinicians can often identify job elements of specific danger that require protections not yet in place. Ethical worker protection involves a hierarchy of control measures: first, primary prevention strategies, such as substitution and engineering control measures, and, only infrequently, clinical actions for difficult hazards for which control is not immediately achievable by broader measures. Use of personal protective equipment (PPE), such as respirators, should be a back-up method to protect employees only when more effective measures are not available. (See Chapter 26.)

Clinically triggered actions, such as medical evaluation and monitoring, represent secondary prevention and can be late-onset interventions for worker protection. Whether the intention is to select workers for special capability to withstand hazards (such as with preplacement exams for heavy lifting), to tolerate burdensome PPE equipment (such as with respirator exams), or to identify and remove workers harmed by prior exposures (such as with hearing protection programs), clinical and individual interventions are intrinsically less protective than more widespread and anticipatory programs, where exposures are eliminated or distanced from workers.

Questions that provide important recognition of potential hazards include utilizing basic concepts in industrial hygiene and safety. Clinicians need to be knowledgeable about the work situation and its dangers. The insight to perform these critical workplace assessments is central to occupational hygiene expertise. Repeated visits make for even greater awareness.

Questions need to be posed about speed and volume of work, the purpose of PPE, precautions used for workers who bypass safety measures, and the chemical nature of materials in use. Important vocabulary is acquired by asking about job titles and team definitions. Useful topics are ventilation, noise measures, weather stressors, spill management, and break rooms and associated personal hygiene topics, such as hand washing and laundering of work clothes. Inquiry is necessary about routine cleaning operations, the roles of contract laborers (who often face greater hazards and are poorly supervised), and product packaging (a frequent ergonomic nightmare). Managers often expect clinicians visiting the workplace to discuss first-aid preparations and smoking cessation programs, but these subjects should not dominate the visit. Workers' interactions with their jobs require the closest scrutiny and insight.

OCCUPATIONAL HEALTH CLINICAL VISITS

The clinical services provided to workers and employers vary greatly in the occupational clinic. For each visit type, the focus is unique. Completion of a comprehensive occupational and environmental health history is critical to determine the work-relatedness of any presenting problem (Chapter 2). Examples discussed here are most common and include preplacement evaluations, drug testing evaluations, specific work approvals, and evaluations of exposure to biological agents.

Preplacement Evaluations

Preplacement evaluations, formerly termed "pre-employment physicals," were renamed with the enactment of the Americans with Disabilities Act (ADA) in 1990, which prohibits use of medical screenings before employment is offered. Now, these evaluations yield clinical approvals for particular jobs or recommendations for necessary specific work accommodations. The ADA limits which restrictions can be considered disqualifying and which must be accommodated. An important principle is whether a specific prohibited task is an "essential function of the job," and whether accommodating the restriction would involve "undue hardship" to the employer.

Experienced clinicians recognize that nearly all pertinent health and medical information required for clinical assessment is included in the patient's narrative. Most illness is not apparent by physical exam; therefore, the patient's candor is necessary to make diagnoses as asthma,

coronary artery disease, low back pain, carpal tunnel syndrome, and epilepsy. Established and reliable confidentiality rules are required. In addition, workers should be informed by management that later discovery of intentional concealment of medical information in this process will have administrative consequences.

Since the job candidate is not yet a company employee, clinicians should expect a more rigorous and inflexible management response against proposed restrictions. The ADA rules protect workers who would once have been disqualified for having a "tarnished" work approval. When restrictions apply to "nonessential" job aspects, current rules prohibit management from barring individuals from employment, because minimal work modifications may be enough to hire individuals who are otherwise well.

Drug Testing Evaluations

The exception to the restricted-duty nature of the occupational health report is the evaluation of pre-employment drug testing results, usually involving forensic urinary measurement of metabolites from illicit "recreational" drugs. Here, an unexcused positive result requires a report to management that the candidate—not yet an employee—"does not meet the employer's standards for employment." Such a judgment does not involve a diagnosis of addiction, intoxication, or documented safety risk. It is simply an assessment of whether the urinary metabolites are present and whether they can be considered forgivable, based on confidential review of authorized prescriptions or reasonable dietary constituents.

This evaluation is called a "medical review officer" (MRO) assessment and involves explicitly designated physician training and certification, especially for specific federally designated programs, such as those of the U.S. Department of Transportation (DOT). Forensic skills are additional aspects of this process, including assessment of the documentation of the chain of custody of a urine sample, the worker's proof of authorized medications, and assessment of the specimen's biological validity.

The social policy establishing drug testing is also intended to create a deterrent against recreational drug abuse, in addition to screening abusers from positions that are safety-sensitive. Although a clinical license is required, this process is ultimately quite remote from the collaborative development of medical-care plans in nonoccupational clinical settings, and even distinct from the usual protective assessments for occupational and environmental medicine. These activities are unique in medical practice, since the patient's history may be suspect, and external documentation is required to confirm every assertion.

Other occasions for drug testing are similarly accusatory and nonclinical, including mandated randomly timed testing programs for safety-sensitive positions, and postinjury assessment. These programs allow employers' insurers to assume that metabolites are proof of impairment and thus to remove workers' compensation coverage for both indemnity and clinical care. No documentation of neurological, behavioral, or judgment impairment is required or sought.

Specific Work Approvals

Many work assignments require focused clinician assessment to permit a single activity. The most common of these evaluations are respirator exams and DOT certification examinations, both of which are covered by specific federal guidelines and evaluation criteria.

Required medical record management for occupational and environmental medicine evaluations is unique and specific. The Occupational Safety and Health Administration (OSHA) regulation specifies that medical records for employees be preserved and maintained for at least 30 years beyond the duration of employment. With rare exception, job-related evaluations are maintained by the employer (or designated clinical contractor) in permanently available files. Explicit arrangements must distinguish occupational health from other clinical documentation, for essentially permanent availability for both written and radiological records.

Respirator evaluations are narrowly directed at whether the device would itself pose a health hazard to the worker. Employees with underlying pulmonary disease may be unable to cope with the supplemental inspiratory or expiratory demands of the respirator's filter. Some severely

claustrophobic employees are unable to withstand the perceived confinement of the face mask's opacity or compression. Clinicians should also determine the nature of potential hazard for which respirators are needed, and they should recognize that individuals with highly reactive airways who are exposed to even a minor irritant leak may develop serious bronchospasm, resulting in panicky removal of an otherwise protective device.

Although universally performed in the past, spirometry is not required for approval for most respirator users. Based on illness and the respiratory dangers of potential exposures, selective pulmonary flow and volume testing is still more frequent than in most clinical settings, and it helps to provide objective criteria for workers at additional risk for respiratory disease or incapacity for respirator use (Fig. 28-1).

Even facial hair needs to be recognized as a reason that a negative-pressure mask would be inadequate protection from airborne hazards. Facial deformities may also prevent proper respirator sealing and warrant comment about individualized fit-testing to be performed by safety personnel. Positive-pressure respirators are often required for individuals in the categories described earlier, but they represent an increased cost to management over simple inspiratory filters.

DOT requirements for truck drivers of interstate or hazardous loads represent a special set of considerations, governed explicitly by regulatory language and by evolving agency guidelines. Special concerns regarding any medical condition associated with interrupted or reduced operator vigilance, such as coronary artery disease, sleep disorders, cardiac arrhythmias, diabetes, or seizures, dominate the critical, but arcane, debates over such workers. Special training is available and published texts are helpful. (See N. Hartenbaum reference in Further Reading.) Also, many evaluators share opinions in national electronic forums on occupational medicine, such as Occ-Env-Med-L (http://www.archives.occhealthnews.net), which represents an evolving clinical database.

Exposure-specific assessment may be the entire focus for a clinical encounter in an occupational health setting based simply on the worker's employment category. These extremely narrow assessments include mandated evaluations for injury from specific situations.[1] The clinician's findings on a worksite walkthrough may suggest other circumstances, such as ergonomic stress, where medical monitoring can provide early recognition of potential harm. The examples that follow represent a tiny fraction of the selective reasons for clinical monitoring for harm attributable to exposure. (Please consult National Institute for Occupational Safety and Health [NIOSH] guidance documents at: http://www.cdc.gov/niosh and specific OSHA regulations at: http://www.osha.gov for more comprehensive protocols on such monitoring.)

- Noise-exposed workers are monitored annually for personal compliance with hearing protection and for impaired hearing. An OSHA-defined "standard threshold shift" is a specific reportable degree of deterioration.

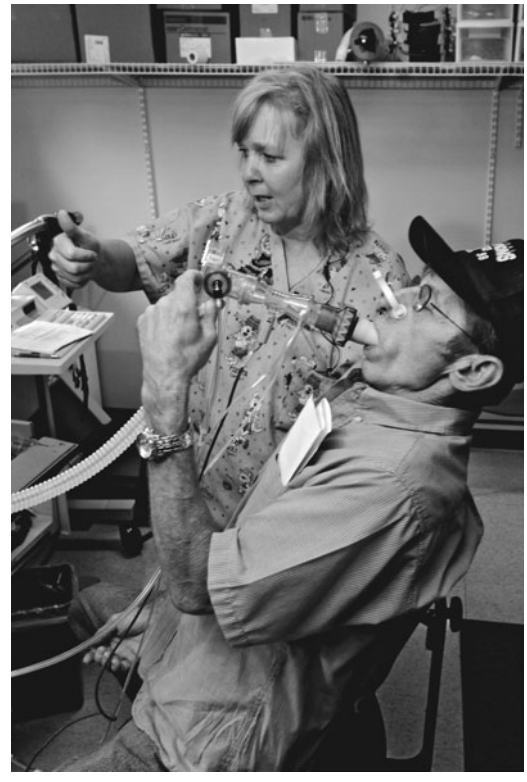

Figure 28-1. Pulmonary function testing requires the tester to elicit a maximal effort by the person being tested. (Photograph by Earl Dotter.)

Regulatory required response to such changes is minimal, but investigations regarding workplace policies and protections are often worthwhile for the individual and workforce as a whole. (See Chapter 21.)

- Lead-exposed workers are required to be monitored for exposure (with blood lead levels) and for metabolic effects (with erythrocyte protoporphyrin levels). Monitoring may detect peripheral nerve or kidney dysfunction, possibly due to lead exposure. Since significant exposure can result from accidental ingestion, worksite personal hygiene (lunchroom hand washing and cigarette contamination) merits discussion. The OSHA lead standard (Lead 1910-1025, accessible at http://www.osha.gov) offers specific regulatory requirements to remove workers from this exposure when findings show increased lead exposure. (See Chapters 11 and 19.)
- Asbestos-exposed workers are routinely provided extreme measures of PPE (clothing and respirators) and other protection (such as closed environments). The most immediate clinical challenge is to monitor these workers' ability to tolerate these devices (including monitoring for heat stress and respirator compliance). Since health risks from asbestos may occur after decades of latency, the required health monitoring for toxic injury is usually premature—and regulatory requirements for clinical monitoring ironically end when exposure does. Nevertheless, chest X-rays and spirometry for restrictive disease are required. Since the dangers of asbestos exposure are greatly amplified by smoking, clinical encounters with these workers should routinely include time spent on tobacco cessation counseling. (See Chapter 18.)
- Cadmium exposure (determined by environmental sampling exceeding a regulatory "action level") requires clinical monitoring for absorbed exposure levels and kidney dysfunction. Blood and urine cadmium levels and urinary β_2 microglobulin (reported per gram of excreted creatinine) are indicators of potential disease. Also important are measures of serum creatinine and inquiry about other risk factors for kidney disease. (See Chapter 16.)

Biological Agent Exposure Evaluations

Each year, an estimated 600,000 to 800,000 health care workers in the United States incur a needlestick injury risk to hepatitis B virus (HBV), hepatitis C virus (HCV), human immunodeficiency virus (HIV), and other virulent pathogens. (See Chapter 13.). Unfortunately, many health care workers still lack knowledge about exposure risk, the availability of immediate remedies, and the urgent need to begin treatment. Occupational health clinicians are in a key position to educate health care workers about the risks and prevalence of bloodborne pathogen exposures in the workplace and measures to take immediately following an exposure.[2] Consider the following case involving exposure to bloodborne pathogens due to needlestick injury.

CASE 1

A hospitalized patient with AIDS became agitated and tried to remove the intravenous (IV) catheters in his arm. Several hospital staff members struggled to restrain him. During the struggle, an IV infusion line was pulled, exposing the connector needle that was inserted into the access port of the IV catheter. A nurse at the scene recovered the connector needle at the end of the IV line and was attempting to reinsert it when the patient kicked her arm, pushing the needle into the hand of a second nurse. The nurse who sustained the needlestick injury tested negative for HIV that day, but tested HIV positive several months later.[3]

In determining intervention, prevention, and control strategies, the health care provider will need to determine several issues: whether an exposure occurred and, if so, what were the circumstances; how best to treat and educate the health care worker; and what follow-up is appropriate. Biological agents of concern include HBV, HCV, and HIV. The health care provider needs an accurate and detailed health history and occupational history, including the events surrounding the exposure. This will include

determining the serostatus of the source case, recognizing issues of informed consent and confidentiality. Testing the exposed individual with appropriate markers/antibody tests for HBV, HCV, and HIV should be done according to recommended (Centers for Disease Control and Prevention) interval schedules. Prophylaxis and treatment should then be offered and given according to U.S. Public Health Service guidelines, such as antiretroviral agents for HIV.

Those at risk of exposure need a clear understanding of the nature of the risk, how best to prevent exposure, important risk factors, modes of transmission, and the need for continued follow-up and treatment. In addition, management must implement systems and strategies to eliminate or mitigate the risk. The health care provider should work in a multidisciplinary team that includes management to determine the most effective approaches to risk reduction.

Reports to Workers and to Management

As for all clinical evaluations, workers must be told, in an explanatory and direct fashion, the results and meaning of their exposure and health effect monitoring. Clinical management and follow-up are required in many circumstances, such as with elevations of blood pressure or liver function tests. The occupational health clinician is responsible for full disclosure of any measurements as well as customized supportive recommendations for personal and ongoing clinical care.

In OSHA-mandated medical surveillance, management must ensure regulatory compliance that screening, or monitoring, is occurring satisfactorily. The clinic needs to provide adequate documentation that these regulations are being followed. Individual clinical reports should reflect simply that each worker has been evaluated according to these requirements (see Fig. 28-2). Usually, individual results are suppressed unless a work-related health consequence has been found, and then rules regarding workers' compensation findings apply (see below).

An aggregate report should also be developed, combining results from similar workers to *(a)* demonstrate and document the number of workers screened and the proportion (or rate) of those with health findings of significance, and *(b)* enumerate those evaluations which required either worker removal or exposure modification. Statistical reports regarding the distribution of numerical results (usually biological exposure indices), including comparison with previous years, and explanation of any changes demonstrate the work of the clinic in improving the overall success of the occupational health and safety program. These aggregate reports are not confidential. They should be discussed with employers' health and safety professionals and also made available to workers and their representatives.

Work-Related Care and Workers' Compensation

Care for workers who have injuries and illnesses that are recognized as being related to work is influenced by state-specific workers' compensation laws and regulations (Chapter 31). This care is affected by whether the worker or the employer selected the site of care. When workers are allowed to choose the site of care, it is usually in the same site as for personal illness—usually because of comfort, confidence, and convenience. When employers choose the site of care, it is much more likely to be provided in a designated occupational medicine practice by health professionals known to management and familiar with the workplace and its activities and policies.

In workers' compensation care, confidentiality rules are suspended. Once an employee files a claim, whether seeking wages or medical care, the employer or its workers' compensation insurer is permitted direct involvement in the review of decisions regarding clinical management, attribution, and specialty referrals. Release of personal health information, even for prior and unrelated illness, is permitted. Reports to management are not required to conceal diagnosis or medical details, and health care providers can choose to abandon the customary, confidential "restricted-duty" reports in favor of simple photocopies of the clinical record. Where permitted by management, reports that voluntarily respect the worker's privacy are preferred, but they are no longer required by regulation.

Often, even when the patient's care involves clinical specialists, employers will utilize their

HEALTH RECOMMENDATION FORM

This clinical evaluation was designed to meet the regulatory requirements for:

❑ Arsenic Exposure	☐ Formaldehyde Exposure
❑ Animal Handler	☐ Hazardous Waste
❑ Blood Borne Pathogen	☐ Lead Exposure
❑ Cholinesterase Inhibitor Use	☐ Noise Exposure
❑ D.O.T. Certification	☐ Respirator Wear

_____
Employee Name

_____
Employer

Baseline Periodic Exit Consult Test/Immunization Only
Visit Type

Drug screen requirements
- ❑ have been met
- ❑ have not been met
- ❑ not applicable

This worker **is / is NOT** medically cleared to wear a protective respirator.

- ☐ **SCBA for protection from toxic exposures**
- ☐ **SCBA for Hazmat and/or MERT**
- ☐ **Air purifying particle mask, negative pressure and PAPR**
- ☐ **Not Applicable**

Additional Notes

- ❑ Tetanus Vaccine Given
- ❑ Tdap(Tetanus, Diphtheria,Pertussis)vaccine given
- ❑ Hep. B Series **initiated / completed** at Duke
- ❑ Hep. B Series **initiated / completed** as reported by employee.
- ❑ Hep. B Series declined
- ❑ Hep. B Titer **is / is not** consistent with immunity
- ❑ Hep. A Series **initiated / completed**
- ❑ Twinrix Series **initiated / completed**
- ❑ Hepatitis C Titer completed at Duke.
- ❑ Tuberculin Skin Test completed.
- ❑ Baseline Audiogram performed
- ❑ Audiogram shows **NO** OSHA STS
- ❑ Audiogram shows a **temporary** OSHA STS. Repeat test in 30 days
- ❑ Audiogram shows a permanent OSHA STS that **is / is not** recordable
- ❑ Blood lead level_____mcg/dl, consistent with levels seen in **exposed / non-exposed** individuals.
- ❑ **Urinary Heavy Metal** or **Cholinesterase** consistent with levels seen in **exposed / non-exposed individuals**

Based on my evaluation, there **is \ is not** a detected medical condition, which would place the examinee, or others at increased health or safety risk as a result of performing his/her work duties. For protection of safety and health, the following accommodations are recommended:

1. _____
2. _____
3. _____

_____	_____	_____	_____
Examination Date	**Report Date**	**Signature**	**Examining Clinician (Printed)**

ISSUES REGARDING COMMUNICATION

Active communication with employers is essential. If these recommendations provided to management seem over- or under-protective (risking either the worker's health or career success), ***please call*** *our office. Often, further discussion will lead to more appropriate accommodations. Our recommendations are intended to allow workers to remain productive while preserving both their health and safety.*

Figure 28-2. Example of a health recommendation form. (Courtesy of the Duke Division of Occupational and Environmental Medicine, Duke University Medical Center, Durham, NC)

customary physicians and nurses to monitor the care and its consequences. Occupational health specialists review determinations of work absences and work restrictions, including those that enable workers to return to specially modified duty.

Tertiary Care Consultations in Occupational and Environmental Health

Occupational and environmental health physicians are also consultants to other specialities

and attorneys on attribution and causation, sometimes about diseases that have resolved or patients already deceased. In legal situations, opposing experts' opinions can be expected. Most of these tertiary consultations regard questions about work-related illnesses, rather than injuries. In these situations, the specialists should provide knowledge and experience in exposure assessment, toxicology, and regulatory matters.

The following guidelines may be helpful in developing a consultation:

- Identify the source of all cited information.
- Consider and report every aspect of the employee's work experience. Exposure histories require an entirely open narrative style, in which much collected information is synthesized and condensed into a smooth, but highly detailed, description of the exposure situation.
- Maintain a textual tone of open impartiality. Make sure that conclusions do not contaminate statements of facts and objective findings. All collected information should be stated in a factual, nonjudgmental tone. Consultations can never include all potentially significant information, so the expert must leave open the opportunity to change opinions if new information, such as correction of misstatements or objective measures of prior estimates, comes to light after the consultation report is filed.
- Disclose the scientific basis for an expert opinion, such as from these varied sources:
  - Textbooks of occupational and environmental medicine
  - Clinical sources related to the specific organ system of concern
  - Medical and scientific journals, many of which can be accessed through the National Library of Medicine (PubMed and ToxLine)
  - The NIOSH database of extracts of texts regarding exposure-related disease (accessible at: http://www2a.cdc.gov/nioshtic-2)
  - The cumulative opinions of Occ-Env-Med-L international forum listserv, including both its present-day members and the collected discussions over 15 years of archived discussion
  - The case database of the Association of Occupational and Environmental Clinics (accessible at: http://www./aoec.org/tools.htm)

Restrictions

Authoring justifiable and protective, health-motivated communications to management without disclosing medical diagnoses can be challenging. The case examples of these communications that follow can be adapted to other clinical settings.

CASE 2

A middle-aged worker in a manufacturing facility with hazardous machinery has type 1 diabetes with poor control of blood glucose. Potential work restrictions and accommodations for this worker that need to be communicated to management might include:

- No unscheduled overtime
- No frequent work shift changes
- No skipped meal breaks
- Needs access to unscheduled snacks during work hours
- No work alone, and co-worker monitoring or contact every 15 minutes
- No work assignment above or adjacent to unprotected high hazards, including electrical, chemical, height, or mechanical dangers
- No operation of vehicles or hazardous powered equipment

If neuropathy, retinopathy, cataracts, peripheral vascular disease, or coronary artery disease have developed, then other restrictions and accommodations may be necessary, including the following:

- No climbing ladders
- Practical vision testing for visually demanding assignments
- No exposure to extremes of temperature
- No strenuous physical activity
- Custom-fit, steel-toed shoes for all non-office work

CASE 3

A young worker entering a new work situation in a chemical packaging plant has mild persistent asthma. Even if this employee's physical exam is normal, work restrictions and accommodations may need to declare the following:

- No exposure to respiratory irritants, such as chlorine, ozone, and smoke
- No work with diisocyanates or where these compounds have recently been used
- No tasks requiring respirators—medical evaluations may be needed to evaluate the need for this restriction
- No work in IDLH (immediate danger to life or health) environments that require respirators

CASE 4

A worker who developed a lumbar strain outside of work may be permitted to work in a modified assignment, with restrictions and accommodations for 5 days, possibly including the following:

- No lifting, carrying, pushing, or pulling of 25 pounds force (not weight)
- No sustained crouching, stooping, or kneeling
- Frequent position changes, including changes from sitting to standing
- Optional posture (employees' choice of sitting or standing) for half of any work hour

Developing worker restrictions and accommodations includes consideration of risk assessment, pathophysiology, familiarity with job demands, and the culture of the employer's worksite.

SENTINEL HEALTH EVENTS: RECOGNIZING THE PUBLIC HEALTH IMPACT OF INDIVIDUAL CASES

Sentinel health events are individual or multiple cases of occupational disease or injury that have significant public health importance. Clinicians need to report these to trigger investigations and intervention measures that are designed to protect a larger population. For example, when a worker is diagnosed with a toxic neuropathy, the clinician needs to report the problem so that the employer can control or eliminate the responsible agent, assess co-workers, and establish a comprehensive plan to deal with the problem.

CASE MANAGEMENT AND SUPERVISED REHABILITATION

Occupational health clinicians often coordinate and monitor care provided by others, especially for patients receiving workers' compensation, to reduce miscommunication, delay, and even fraud. Case management is the combination of planned activities to ensure appropriate use of medical facilities, early and appropriate referrals, and quality of care at controlled or reduced costs.[4] It is the coordination and use of health care resources to return the employee to optimal levels of health. A written plan and protocols should be established to ensure quality of services and to evaluate results. The plan also should outline return-to-work procedures, including establishment of restricted- or modified-duty jobs.

The case manager is often the occupational health nurse but sometimes may be an insurance company employee. The case manager should establish a proactive approach to case management, beginning with assessing worksite safety, identifying potential hazards, and working to prevent or control such hazards in collaboration with other health and safety professionals. The case manager evaluates an employee's health needs and level of function, communicates essential information regarding work status and job requirements to the occupational health care provider, and keeps the employer up to date regarding employee progress or changes in the plan, such as return-to-work dates, and work limitations. The appropriateness of available modified job positions should be considered in collaboration with the occupational health care provider when determining readiness for work.

The primary role of the case manager is to facilitate communication between the occupational

health unit, management, the insurance carrier, the health care provider(s), and the employee. The case manager maintains frequent communication with the employee and all necessary persons but also is an advocate for the employee, facilitating open communication, rehabilitation, and the return-to-work process.

Care monitoring is often achieved by telephone contacts, but sometimes nurses accompany patients on visits to specialists to ensure that treatment and rehabilitation plans are received and acted upon. The patient needs to recognize that the nurse's role is observational and passive and that the employer has complex motives.

HEALTH PROMOTION

The primary goal of health promotion is to maintain health and optimize an individual's health potential. Emphasis is placed on developing positive health behaviors, recognizing personal responsibility for health, and engaging families and communities in health promotion and disease prevention activities (Fig. 28-3). (See Chapter 38.)

Every 10 years, the Department of Health and Human Services provides national objectives for promoting health and preventing disease. Since 1979, the Healthy People project and publications have set and monitored national health objectives to meet a broad range of health needs, encourage collaboration across sectors, guide individuals toward making informed health decisions, and measure the impact of prevention activity. The framework of *Healthy People 2010*[5] provided 28 focus-area objectives, each with targeted objectives. The goal for the occupational safety and health focus area (#20) was to "promote the health and safety of people at work through prevention and early intervention."[5] Preventing occupational disease and injury requires changes in work practices, engineering controls, worker monitoring, workplace surveillance, worker education, and supportive management. Information on Healthy People 2020 is available at: http://www.healthypeople.gov/HP2020/.

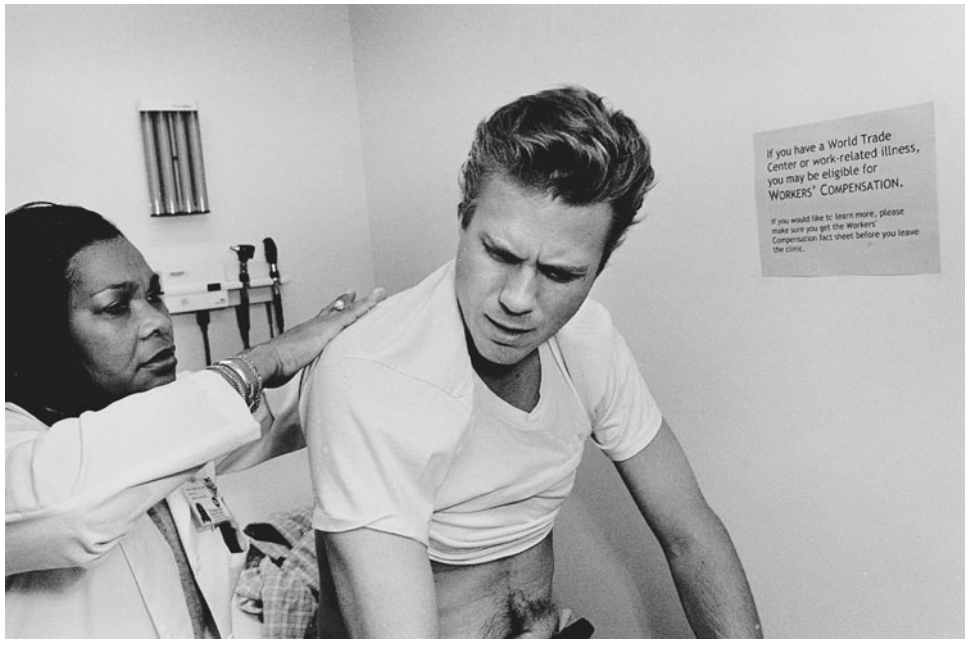

Figure 28-3. Occupational health services provide opportunities for physicians to identify a wide range of health risks, including nonoccupational risks. (Photograph by Earl Dotter.)

Since occupational and environmental health practice is a population-based specialty within public health practice, the goals of health promotion and health protection are inextricably linked to prevention of illness and injury. Occupational and environmental health care providers practice at all three levels of prevention, working to ensure the employee's best interests and health regardless of the source of the provider's compensation for these services.

Primary prevention is intended to prevent illness or injury. Because occupational health is one of the preventive health specialties, primary preventive services distinguish occupational health from other types of health care that address only curative services. Primary prevention, which incorporates both health promotion and health protection, enhances the well-being of individuals, groups of employees, and the company as a whole, by reducing or eliminating hazardous exposures and protecting workers against remaining hazardous exposures and their adverse health effects. Primary preventive services include programs designed to enhance coping skills and good nutrition, knowledge about potential health hazards (both in and outside the workplace), immunizations, and use of safe work practices. Primary preventive strategies include walkthrough assessments of workplaces to identify hazards, to modify work environments in order to reduce hazards, and to supply PPE to workers when hazardous exposures cannot otherwise be controlled. While occupational hygienists, ergonomists, and safety specialists may have important roles in primary prevention, occupational health care providers often work closely with them to identify potential health hazards that require correction.

Secondary prevention is intended to detect illness or injury at a relatively early stage, often before symptoms or clinical signs are noticed. When disease is detected at this early stage, it may be possible to take measures to slow, arrest, or reverse the disease process. For employees with potential work-related exposures, early detection utilizes preplacement examinations, health surveillance, and periodic screening activities to identify illness at the earliest possible stage and eliminate or modify the hazard-producing agent or condition.[6] Because interventions are likely to be both clinically and workplace based, secondary prevention explicitly requires occupational health clinicians to work beyond their clinical role. For example, if results for a worker at a battery manufacturing plant indicate that the employee has significantly elevated blood lead levels, the clinical response is to assess target organ function and determine whether chelation therapy is indicated. However, this case represents a sentinel health event, indicating excessive lead exposure in the workplace. In addition to providing clinical care, the health care provider must contact the employer to report the hazard, alerting the employer to the need for making workplace changes to reduce or eliminate the exposure.

Secondary prevention usually addresses ailments that are not yet symptomatic. These ailments typically are detected through screening examinations, some of which are required by OSHA for workers exposed to specific hazards. OSHA may also invoke the "general duty" clause of the Occupational Safety and Health Act to require medical surveillance for other occupational exposures. (See Chapters 2 and 30.) In addition, secondary prevention includes *(a)* screening for generally nonoccupational medical problems, such as hypertension, and *(b)* referral for counseling and treatment of employees with emotional or mental health problems whose work performance has deteriorated.

Tertiary prevention, which is provided after injury or illness has occurred, is intended to provide for rehabilitation and optimal recovery. Tertiary preventive services include clinical care for occupational injuries and illnesses. Those who provide this care need to plan for the employee's ultimate return to work. Examples of tertiary prevention include rehabilitation in return-to-work programs for workers who have had a myocardial infarction or traumatic injury or transitional duty programs after treatment of a cumulative trauma disorder. Initial return to work may be to a modified or alternative job with reduced demands, or with a graduated return to the original (or similar) job. It may also be necessary to modify the original job to correct ergonomic or other problems that would otherwise lead to reinjury or exacerbation of an illness relapse. Clinicians should coordinate this process with employer representatives to ensure that necessary workplace

changes are made. Tertiary preventive measures also include continued rehabilitation of employees with substance abuse problems, smoking cessation, counseling of employees with chronic obstructive pulmonary disease, and special monitoring for employees with noise-induced hearing loss.

THE MULTIDISCIPLINARY APPROACH

Essential to the success of an effective occupational health and safety program is *(a)* a thorough assessment of the work and work environment to determine potential and actual work-related exposures, and *(b)* continuous monitoring to prevent and control workplace hazards. This is a complex process that requires the knowledge and skills of many disciplines.[7] A multidisciplinary approach to occupational health care is necessary and requires occupational medicine physicians, occupational health nurses, safety specialists, occupational hygienists, ergonomists, and others to collaborate in recognizing, treating, and preventing occupational illness and injury.[7]

There is great diversity among providers of occupational and environmental health services. Physicians are one source of providing occupational and environmental health services, and their training and backgrounds vary. Some physicians who designate their practice as occupational medicine have been trained in occupational medicine residencies; however, most occupational medicine physicians are trained in other specialties. About one in five are board-certified in occupational medicine.

Business and industry have traditionally provided the most employment in clinical occupational medicine. Physicians who work at the upper management level are more involved in questions of policy, whereas those at the worksite level are more involved in clinical duties. Physicians also work for governmental agencies at federal and state levels. Opportunities to practice occupational medicine can also be found in clinical settings in the community. In some cases, occupational medicine clinics exist as independent contractors to sell services to client companies. Some clinical occupational medicine units are in medical centers or medical schools, usually within departments of medicine or family practice. Physicians may perform complex illness and injury management, evaluate patients for insurance companies (such as for disability impairment ratings), and participate in health and medical surveillance programs on a consultative basis, subcontracting with occupational hygiene and clinical laboratory facilities, when appropriate.

Many physicians in other specialties also see patients with occupational illnesses and injuries. For example, patients with work-related injuries and musculoskeletal disorders may be cared for by orthopedic surgeons or specialists in physical medicine and rehabilitation, and patients with occupational asthma may be referred to pulmonologists or allergists. Most cases of work-related injury or illness are seen in the offices of primary care practice physicians or in emergency departments; however, the association between diseases and their work-related causes may be overlooked.

Occupational health care is also provided by occupational health nurses. Although not all nurses working in occupational health are specialty trained, available training curricula include master's degrees in occupational health nursing. Certified occupational health nurses have completed requisite work experience and educational training and have passed a certification examination.[8]

The occupational and environmental health nurse practicing in the clinical setting will need to be familiar with occupational and environmental diseases and injuries within the contexts of nursing practice and process, and the exposures relevant to the workforce population served. For example, a major cause of occupational illnesses among production workers is exposure to toxic chemicals. The occupational and environmental health nurse must be able to assess problems and provide treatment or refer injured or ill workers to the appropriate health care provider, when necessary. Nurses are often the only licensed health professionals at the worksite and often manage the functions of an occupational health clinic, refer employees needing medical management to consulting physicians, and serve as case managers in managed care organizations and insurance companies.

Occupational and environmental hygiene is the environmental science of anticipating, recognizing, evaluating, and controlling health hazards in the work environment with the objectives of protecting workers' health and well-being and safeguarding the community at large. It encompasses the study of hazards posed by physical agents, chemical agents, biologic agents, and stress in the occupational environment as well as concern for the environment outside the workplace. (See Chapter 25.) However, all members of the occupational health and safety team play an integral role in identifying and managing workplace exposures and hazards.

Hazard recognition requires skill in assessing the work and work environment and investigating health risks. (See Box 2-3 in Chapter 2.) A full-scale worksite assessment and walkthrough survey, which is a major component of the hazard recognition process, should be done in a multidisciplinary context.

Safety in the workplace is everyone's responsibility and awareness of safety and health issues is critical to prevention of "accidents." The principal responsibility of the safety professional is to design, implement, and evaluate strategies aimed at preventing and controlling workplace hazards that emphasize training and education of workers about job safety. The emphasis and commitment on safety starts with top management and extends, by example, throughout the organization to managers and other employees.

The establishment of a workplace health and safety committee is vital to transforming health safety ideas into prevention and protection strategies—through policies, procedures, program development, and training and education about health and safety in the workplace. The health and safety committee should include representatives of various levels of management, employees, the physician, the occupational and environmental health nurse, the safety manager, other health care professionals, and, if applicable, union representatives. A health and safety plan with specific goals and objectives should be established within the context of the committee, with input from employees and all levels of management.

Ergonomics is concerned with matching work and job design to fit the capabilities of most workers by adapting the product to fit the user rather than the reverse. (See Chapter 27.) The design of the work environment should be flexible enough to consider the need for individual variation.[9,10] For example, two people with the same height and weight may have a different arm reach or strength, and accommodations for these differences should be available.[7] An effective ergonomic health and medical program should encompass a multidisciplinary approach. It should include early identification, evaluation, treatment, follow-up, rehabilitation, and recording of signs and symptoms by health care providers knowledgeable in these areas and the company's operations, work practices, and light-duty jobs. The ergonomist or other qualified person should analyze the physical procedures used in the performance of each job, including lifting requirements, postures, hand grips, and frequency of repetitive motion. Resolution of ergonomic problems is best accomplished through a team problem-solving approach.

A comprehensive occupational health and safety program is one that looks beyond the specific requirements of the law in addressing general and specific workplace hazards. Doing so requires substantial knowledge in the occupational health sciences as well as an interdisciplinary approach to recognizing and understanding work-related risks and hazards so that effective occupational health and safety services can be delivered.

ETHICS

Ethical problems are encountered by occupational and environmental health professionals in their work. Because the environments in which these health and safety professionals function can be characterized by competing goals and interests and differential power structures, thorny ethical issues are common.

Ethical conflict is nothing new in occupational and environmental health practice. Traditional concerns about maintaining confidentiality of employee health records, hazardous workplace exposures, issues of informed consent, risks and benefits, and dual-duty conflicts have been added to newer concerns about genetic screening, worker literacy and understanding, work organization, and return to work.[7,11,12] The American

Box 28-1. The Association of Occupational and Environmental Clinics Patient Bill of Rights

Our clinic is a member of the Association of Occupational and Environmental Clinics (AOEC). As an AOEC member, we are committed to providing you with quality health care, and to helping you understand the nature of your illness and any risks to your health. Our primary obligation is to you, our patient. We assure you that your medical care will be handled with compassion and strict confidence.

As a patient of our clinic, you have a right to the following:

1. To know and consent to all tests and procedures before they are performed
2. To know the results of all tests and procedures
3. To obtain a copy of your medical records, if requested
4. To obtain a list of contracts and grants that this clinic has with any organizations, such as government agencies, industries and companies, labor unions, or community groups

As an AOEC clinic, we promise to do the following:

1. Maintain your records in strict confidence and not release them to anyone outside this clinic without your express written permission. (Your filing of worker's compensation, health insurance, or legal claims may require you to release your records.)
2. Help you obtain information about worker's compensation, Social Security, disability, and other health and welfare benefits if you request such information.
3. Provide information on your Occupational Safety and Health Administration (OSHA) rights, assist you in getting a workplace inspection, and help you improve health and safety at your worksite.
4. Provide legal testimony of our findings, if necessary.
5. Declare any possible conflict of interest by providing you with a list of our grants and contracts, if requested.
6. Explain the results of all medical tests and procedures performed under our direction.
7. Help you understand the causes of your illness and risks to your health.
8. Work with you to prevent future health problems.

(*Source*: http://www.aoec.org/principles.htm. Accessed on June 16, 2010.)

College of Occupational and Environmental Medicine (ACOEM) has recently developed an updated code of ethics, which is available at: http://www.acoem/org/codeofconduct.aspx.

All occupational and environmental health professionals recognize that patients deserve quality care. The Association of Occupational and Environmental Clinics (AOEC) has developed a patient bill of rights, which can serve as a useful framework for providing quality care (Box 28-1).

CONCLUSION

Clinical occupational and environmental health practice encompasses a wide range of services provided by professionals working in multidisciplinary teams. The primary goals are to reduce hazard exposure and to prevent disease and injury. Provision of services to meet these goals include assessment and monitoring of the workplace and the general environment for health and safety hazards, interventions designed to mitigate or eliminate hazardous exposures, case management for injured and ill individuals, and other measures to promote health and prevent disease and injury.

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FURTHER READING

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29

Risk Communication and Information Dissemination

Paul Schulte, Scott Schneider, and Ray Sinclair

In occupational and environmental health and safety, risk is the product of a hazard and exposure to the hazard. Risk communication is a form of preventive action intended to reduce morbidity and mortality. Risk communication in occupational and environmental health and safety includes dissemination of risk information among workers, employers, labor and environmental specialists, legislators and regulators, members of the general public, researchers, and other decision makers.[1–3] However, the *communication* part of risk communication implies a process broader than dissemination. It signifies an *exchange* of perceptions among stakeholders about workplace risks to worker health and safety. Or it signifies an *exchange* of perceptions among stakeholders about environmental risks to community health (Box 29-1).

There are three objectives for risk communication: autonomy, equity, and efficiency.[4] Under the Occupational Safety and Health Act, workers are guaranteed a safe workplace. However, without effective risk communication, workplace safety is not likely to be achieved. Before workers begin their jobs, they should have more information about the jobs than hours, wages, and benefits. They should also know the job-related risks to their health and safety and the necessary measures to control these risks. This type of information sharing is the only way to ensure that workers have complete information about the risks to which they are exposed. Unfortunately, economic pressures impinge on workers' autonomy to decide on what work they will do.

Concerning the objective of equity, labor-contract law presumes that parties to a contract (employer and employee) have equal information before they agree to the contract. Employer and employee agree on remuneration in return for labor under specific conditions. However, the employer often has more information about the risks associated with job-related materials and processes. The employer may also know more about the resources available to control or abate hazards. Therefore, there is a need for risk communication to balance the contract agreement as much as possible. Workers should understand what they face in their jobs.

Concerning the objective of efficacy, risk communication is necessary for having efficient social systems and economic markets. There are risks in every human endeavor. Deciding which risks are acceptable and which ones are not will be done most efficiently by a society when all stakeholders are engaged in communications about those risks. Risk communication helps to ensure that all expertise is considered and that agreed-upon practices will be widely accepted.

Box 29-1. Environmental Risk Communication

Craig W. Trumbo

Environmental risk communication can be broadly defined as all communicative activities within the information sphere relative to a given hazard that has diffuse boundaries in terms of its demographic, geographic, temporal, and outcome characteristics. Communications range from those that are from one person to another, to those that are generated by the mass media to the general public, and they range from casual communications to those that have a specific purpose. The information sphere encompasses domains of activity, such as news, advertising, policy, regulation, and entertainment.

Environmental risk communication often involves diffuse boundary characteristics that provide the contextualizing attributes of who (demographic), where (geographic), when (temporal), what (outcome), and why (cause and effect). The diffuse nature of these boundary characteristics separates environmental risk communication from occupational health and safety communication, which is typically associated with a narrowly defined population (workers), at a specific place (or specific class of places), within an identified time of exposure (while at work), and with a shared set of outcomes often derived from a single well-identified and casually linked hazard, such as asbestos or noise. In contrast, environmental risks may involve ill-defined or broad (and often shifting) demographics spread over equally ill-defined or broad areas, with no clear beginning or end to exposure times. Outcomes are often uncertain and rarely are they causally linked.

Examples of environmental hazards can be divided between those that are naturally occurring and those that are humanmade. Naturally occurring hazards involve chronic problems, such as solar ultraviolet radiation and radon exposure, and acute problems, such as fires, earthquakes, and extreme weather events. Humanmade environmental hazards include those that are voluntary—mainly risky behaviors, such as smoking, consuming an unhealthy diet, substance abuse, and unsafe sex, and those that are involuntary, such as air and water contamination by chemical or radioactive substances, food contamination, the consequences of new technologies, and climate change.

While the concept of risk is ancient, environmental risk communication has been scientifically studied only since the early 1980s. It has various disciplinary origins and ongoing threads, located in fields such as communication, public health, psychology, sociology, policy studies, and political science.

There are four most salient areas of research on environmental risk communication:

1. The various psychological mechanisms behind the perception of risk, which are relevant to understanding risk-communication audiences and how they process and understand risk messages
2. Sociology and anthropology research on environmental risk as a phenomenon occurring at the intersection of physical, social, and cultural processes
3. How the mass media transmit environmental risk messages to the general public
4. The pragmatic concerns of those charged with communicating risk

The complexity of environmental risk communication extends into practice. While risk communicators may also be individuals or groups engaged in informal or unofficial information transmission, many environmental risk communicators are professional practitioners. There are commonalities across these areas of practice.

Most of the prescribed steps for effective occupational risk communication are also applicable in environmental risk communication:

- Define the audience in terms of its receptivity to the communication, ability to process the information, and capacity to act on recommended behaviors.
- Understand the demographic characteristics of the audience, especially as risk messages can be understood differently, according to age, sex, and racial and ethnic group.
- Consider the language and style of the message.
- Pretest and evaluate the message.
- Consider transmission of message elements through textual, visual, or audio modes.
- Ensure that message content establishes and maintains the credibility of the source, while providing factual information or instructions—and often persuasion to act.
- Consider the channels through which environmental risk messages are sent.

Channel selection and management has become a major challenge for risk communicators because communication technologies have rapidly evolved and function in different ways for different populations or groups. The decline of the U.S. newspaper industry poses a considerable challenge because this avenue for community-level dissemination offered significant efficiencies of scale. The rapid growth of social media poses a considerable challenge since little is yet known about its efficacy in environmental risk communication.

To some degree, risk communication has been developed as a set of persuasive tools used by special interests to serve their needs. Political and economic interests have always had access to the strategies identified here, and they will always apply them toward their vested interests, sometimes risking public harm. Therefore, professional risk communicators should conduct their work with an unbending respect for the public good.

The objective of efficiency should also drive decisions about how much and what kind of risk communication is undertaken.

While the importance of risk communication may seem obvious today, legislated mandates for the creation and dissemination of risk information were necessary in the past. A range of federal legislation and regulations contains such stipulations concerning occupational and environmental health (Table 29-1). Requirements are included in the Occupational Safety and Health Act of 1970 and the Occupational Safety and Health Administration (OSHA) Hazard Communication Standard—perhaps the most direct regulatory expression of occupational risk communication objectives. The Environmental Protection Agency (EPA) also mandates training that encompasses risk communication (Fig. 29-1). In addition, voluntary consensus standards have called for risk communication, such as those of the American National Standards Institute (ANSI); corporate policies, such as those of Responsible Care; and labor and community-organization health and safety policies and practices.[5–7]

A RISK COMMUNICATION MODEL

A model for risk communication is depicted in Figure 29-2. Each stakeholder in the risk communication process is both a sender and a receiver of risk communications (risk-communication messages) as each takes in information, processes it, and sends it to others. Other preventive actions may take place as the understanding of risks and prevention methods increases. Risk communication is only effective if information is received and used by a significant number of stakeholders. The stakeholders in Figure 29-2 are usually engaged simultaneously in risk communication, each providing a unique perspective on new knowledge, attempting to make sense of it.[8,9] Figure 29-2 conceptualizes stakeholders at the individual level; however, at the organizational level there are also stakeholders, such as academic institutions, government agencies, insurance companies, labor unions, professional associations, and non-governmental organizations.

Figure 29-3, which depicts the risk communication process in greater detail, is based on general health-communication models.[10,11] New risk and prevention knowledge may be created by any stakeholder. That information creates a necessity for one or more stakeholders to consider taking—or not taking—preventive actions (top of Fig. 29-3).

Alternatively, and in reverse, the *need* for a preventive action may drive people to seek new information.[12] In these cases, the information is part of the store of information that resides in databases, books, periodicals, collections, certification criteria, training materials, and general understanding.[13] Information is then sought by people who have a specific need for a preventive action and are seeking change in behaviors. These behaviors are influenced by roles and contexts in which the seekers of information operate.[14,15]

If risks and prevention strategies are well-known and widely accepted, then new knowledge provides merely a refinement of prevention practices to make them more effective, requiring minimal additional risk communication. However, new knowledge about risks and controls generally leads to planning activities for risk communication.[16] These activities include an assessment of stakeholders, external factors, and available resources. The assessment of stakeholders involves determining which stakeholders should receive the new information based on their likelihood of contributing to prevention activities. Sometimes the information may be so compelling that employers and workers will take action upon receiving the information. Sometimes insurers, media representatives, government regulators, union officials, and others may be expected to play a role in stimulating preventive actions. External factors, such as the current economic and political climate, should be assessed to inform the rest of the process. These assessments, combined with the importance of the new risk information to the preservation of health and safety, should then be used to decide how many resources to devote to further risk communication. The process may then continue to development and production of risk-communication messages.

Design of messages includes an assessment of stakeholder audiences to ensure that their

Table 29-1. Examples of Legislative and Regulatory Requirements for Disseminating Occupational Health and Safety Information

Legislation	Requirements
Occupational Safety and Health Act	
Public Law 91-596 http://www.osha.gov/pls/oshaweb/ owadisp.show. document?p.table=OSHACT&p.id=2743 U.S. Code Citation: 29 U.S.C. 651 *et seq.* http://frwebgate.access.gpo.gov/cgi-bin/getdoc. cgi?dbname=browse.usc&docid=Cite:+29USC651 Section 20 (a)(7)(d) Research and related activities Section 21 (a) Training and employee education	Requires NIOSH to disseminate information obtained from this research and related activities to employers and employees. Requires the conduct or support of education programs to provide qualified personnel to carry out the purposes of the OSH Act, and information programs on the importance and proper use of safety and health equipment. Also provides for the establishment and supervision of programs for the educationand training of employers and employees in the recognition, avoidance, and prevention of unsafe or unhealthful working conditions in employments covered by the OSH Act.
OSHA regulations	
29 CFR 1910.1200 Hazard communication(e) Written hazard communication program http://trwebgate.access.gpo.gov/cgi-bin/get-cfr.cgi?TITLE=29&PART1910&SECTION=1200&TYPE=PDF	Requires employers to develop programs to make available information on hazardous substances and develop a written hazard communication program. Employers must also show how they will inform workers of hazardous chemicals and the hazards of nonroutine tasks.
Toxic Substances Control Act	
Public Law 94-469 http://www.epa.gov/region5/defs/html/tsca.htm U.S. Code Citation: 15 U.S.C. 2601 *et seq.* http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=browse.usc&docid=Cite:+15USC2601 U.S. Code Citation: 15 U.S.C. 2603 http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=browse.usc&docid=Cite:+15USC2603	Establishes and coordinates a structure for the exchange of research and development results on toxic chemicals among federal, state, and local authorities. Includes ways to facilitate and promote the development of standard data formats, analyses, and consistent testing procedures as part of the research and development exchange structure.
Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)	
Public Law 95-510 http://www.epa.gov/superfund/action/law/cercla.htm U.S. Code Citation: 42 U.S.C. 9601 et seq. http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=browse.usc&docid=Cite:+42USC9601	Training areas under this legislation include the safe packaging, loading, unloading, handling, storing, and transporting of hazardous material and emergency preparedness for responding to an incident involving the transportation of hazardous materials.
Superfund Amendments and Reauthorization Act (SARA)	
Public Law 99-499 http://www.epa.gov/superfund/action/law/sara.htm U.S. Code Citations: 26 U.S.C. 9601 *et seq.* http://frwebgate.access.gov/cgi-bin/getdoc.cgi?dbname=browse.usc&docid=Cite:+42USC9601 42 U.S.C. 9605 et seq. http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=browse.usc&docid=Cite:+42USC9605	Title III of SARA provides a framework for emergency planning and preparedness and requires facilities to provide community groups with information on their inventories of hazardous chemicals and for manufacturers to report releases to the environment.

NIOSH, The National Institute for Occupational Safety and Health; OSHA, The Occupational Safety and Health Administration. (*Source*: Adapted from Reference #2 on page 637.)

Figure 29-1. Environmental Protection Agency–mandated pesticide training. (Photograph by Earl Dotter.)

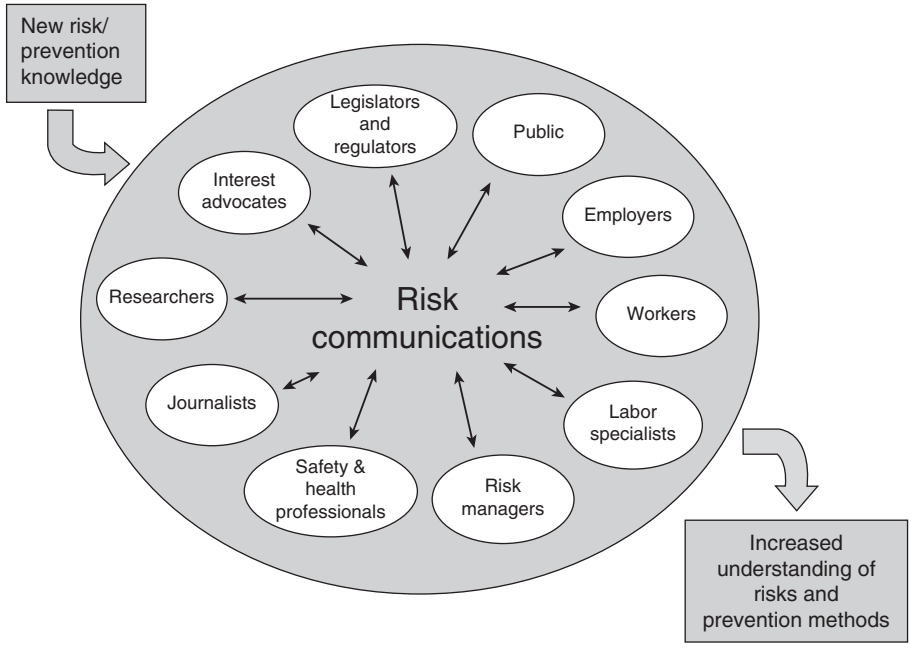


Figure 29-2. Risk communication model.

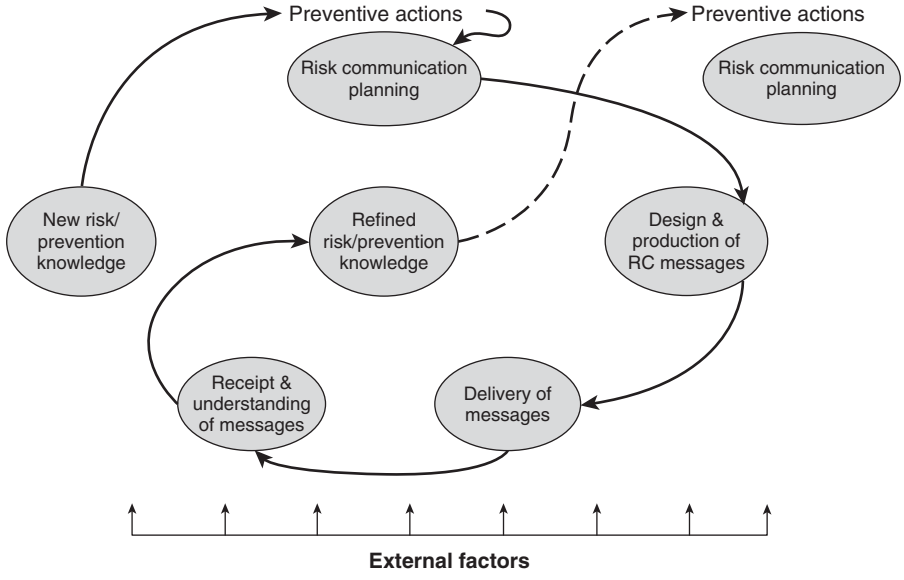


Figure 29-3. The risk communication process.

demographic characteristics, current knowledge, attitudes, biases, and behavior patterns influence the design (Fig. 29-3, right center). Design also includes the selection of message channels (methods to disseminate messages) and the frequency of message transmission through each channel. Message channels include training, print, video, and Internet-based methods.

Message production involves the writing of copy and the preparation of visual and aural elements appropriate to each selected channel. Message delivery involves the transmission of messages to the stakeholder targets. Message delivery should be guided by information about the media access and patterns of use of the intended recipients—which media they use to make decisions about risks and hazard protection, and when and how often they use these media.

The next stage in the process is the receipt and understanding of risk communications by stakeholder audiences (Fig. 29-3, lower left)—often unnecessarily left to chance in occupational and environmental health and safety. Audiences can be asked about the receipt and understanding of disseminated messages. Understanding the message is critical because it is the only basis for achieving the final step in the process: risk and prevention knowledge, which leads to further, and perhaps more tangible, prevention actions by stakeholders (indicated by the dashed-line arrow in Fig. 29-3).

Overall, the model demonstrates that risk communication is the development of a consensus for preventive action among stakeholders based on a shared or agreed-upon interpretation of new risk and prevention knowledge. If external factors are favorable, once information and knowledge is obtained, a process of use and impact occurs.[2,3,17]

Risk communication faces "real-world" barriers.[6,18,19] To understand the flow of risk information, one must also analyze the barriers or external factors that affect the process. For example, information on practices that reduce risks of noise-induced hearing loss has been widely disseminated, but these practices have not been adopted at an appreciable level by many industries. The need for effective programs to prevent hearing loss is not at issue. However, economic, social, and political barriers to the use of this information are significant.[20] Approaches to reducing occupational morbidity and mortality are often complex and often beyond the realm of risk communication. However, a stronger emphasis should be placed on determining additional types of information needed by stakeholders and how data are used by organizations and individuals in making decisions.[21,22]

In each of the stages, there is feedback. Every time information or knowledge is created or interacts with a person or situation, the validity or appropriateness of the information or knowledge can be tested and the possibility of feedback arises. This, in turn, can result in new or modified information and knowledge. This model builds on the one-way, linear, source-message-channel-receiver model.[23] However, it is more likely that all the stages are occurring simultaneously, or at least interacting with or influencing each other.[24] In practice, the boundaries of the science information process are permeable and information flows in all directions.

RISK COMMUNICATORS

Occupational and environmental health and safety communication lies at the nexus of diverse human and organizational interests and a vast array of workplaces and processes. Several types of risk communicators (senders and receivers of messages) are involved. Different audiences require different types of communication, tailored message content and formulations, and specialized means of delivery.[19,21,25] The approach of dividing mass audiences into smaller and more homogeneous segments is known as audience segmentation.[26] The following are observations about nine types of risk communicators in occupational health and safety. Environmental health involves a similar group of risk communicators (Box 29-1).

Workers

In occupational health and safety, workers are the primary focus of risk communication. They bear the risks of the hazards, suffer from exposure to hazards, and benefit from controls used to reduce or eliminate hazards. Workers care about their health and safety. They also care deeply about their families and being able to support them. Traditionally, workers are targeted as receivers of risk information since their understanding of the risks they face and the controls available to them is central to risk communication. Employers, union representatives, government officials, health professionals, journalists, and representatives of advocacy groups and other non-governmental organizations are common sources of risk communication for workers. The information workers receive should be timely, understandable, accurate, actionable, and reliable because the risks that they face are often significant and imminent.

Workers are also important senders of information. They may convey information about ways in which they are exposed and the symptoms that result. Since they often play a role in the development and use of control methods, workers can be sources for information about those controls for all other stakeholders. They may be especially effective sources of communication for co-workers. More experienced workers are often trusted sources of information for those with less experience, especially younger workers. Workers are often very effective communicators when they tell their stories about how they or their friends have been injured or escaped injury.

Employers

Employers are important risk communicators because they have some autonomy to direct or control what occurs in the workplace. They are also able to direct resources to health and safety programs and specific measures for hazard control. In addition, the law and the federal government generally hold them responsible for workplace health and safety (OSH Act of 1970). Because of their importance, employers receive risk information from all other stakeholder groups. Employed workers represent an important source of information for employers. Worker protection should be a partnership between employers and workers. Other important sources of information for employers are insurance companies, trade associations, health and safety professionals, and government officials, as well as conferences and meetings, and the Internet. Employers send risk communications to all stakeholder groups as they explain their policies and practices and share information on worker injuries and illnesses.

Government Agencies

Government agencies at the federal, state, and local level whose missions involve health, environment, and/or labor are senders and receivers

of occupational safety and health information in several ways. They are mandated to attend to the concerns of the public, especially workers and employers. For example, the National Institute for Occupational Safety and Health (NIOSH) is the federal agency responsible for performing research on occupational health and safety and recommending measures to reduce or eliminate workplace exposures and hazards. NIOSH has conducted many "town-hall" meetings across the United States to learn about workplace safety and health concerns and to use the information obtained to guide research. Government agencies receive reports from employers. For example, the Bureau of Labor Statistics receives information from employers on occupational illnesses, injuries, and deaths.

Government agencies are message senders, mainly to workers, employers, and the general public as well as other government agencies and branches of government. OSHA communicates, primarily to employers and workers, rules for workplace safety and health and information on risks and controls (http://www.osha.gov). NIOSH informs the public of its research projects and results through its Web site (http://www.cdc.gov/niosh).

Non-governmental Organizations

Many non-governmental organizations (NGOs), both for-profit and nonprofit, perform risk communication for occupational safety and health. They include unions, trade associations, workers' compensation insurance companies, safety councils, and advocacy groups as well as environmental non-governmental organizations. (See Chapters 32 and 33.) For example, committees on occupational safety and health (COSH groups) are coalitions of labor unions and individual health and safety professionals and activists who work on issues in a special geographic area. Other NGOs focus on health and safety problems in a specific sector, such as the Center for Construction Research and Training.

Non-governmental organizations receive risk information from workers, employers, researchers, government officials, and others. They also serve as message senders to these same stakeholders, adding their perspective or translating risk communications into other channels, such as translating regulations into training materials and research reports into policy proposals. Non-governmental organizations generally do not perform original research or literature reviews to make determinations about hazards and risks and generally do not issue risk management recommendations. However, there are some exceptions; for example, the Center for Construction Research and Training performs all of these activities.

Professional Organizations

Two types of professional organizations are engaged as senders and receivers of risk communications: those associated with particular occupations and those focused on occupational and environmental health and safety. The former—basically trade associations—focus on the health and safety needs of their members, such as in health care, where worker protection is often seen as a dimension of patient safety. For example, the Association of periOperative Registered Nurses provides training materials, position statements, and advocacy materials on issues such as patient lifting, fire safety, noise, and evacuation of surgical smoke. The Society for Mining, Metallurgy, and Exploration provides similar materials for its members, who are mining engineers.

Professional organizations that focus on occupational health and safety issues include, for example, the American Industrial Hygiene Association (AIHA), the American Society of Safety Engineers (ASSE), the American College of Occupational and Environmental Medicine (ACOEM), the American Association of Occupational Health Nurses (AAOHN), and the American Public Health Association (APHA). Members of these organizations include a mixture of academics, consultants, and practitioners who work on identifying and controlling health and safety problems, often within a specific industrial sector. They are interested in scientific research results and especially information on newly identified hazards and effective interventions for controlling these hazards. These organizations can significantly influence workplace conditions by providing information to their members and by issuing policy proposals. They also have committees of experts on specific topics who can act as resources for technical assistance

and development of policy positions for legislative and executive branches of government.

Researchers

Scientific endeavor is the source of much risk information. Researchers work for academic institutions, government agencies, NGOs, insurance companies, and other groups. Researchers communicate to their peers through scientific publications, using narrow, precise, and detailed messages that are grounded in rigorous scientific methods. Science makes progress through careful building of the general body of knowledge by replicable procedures and careful reporting of hypotheses, research methods, and results. Research communications are tailored to meet diverse needs, ranging from solving specific problems to attempting to obtain financial resources in the political arena.[27,28] Scientific publications are also the cornerstone for reviews of important occupational health and environmental safety topics by government agencies, advocacy groups, and other organizations. Other applied research outputs include methods, tests, technologies, and devices. The goal of all these research communications is to continuously improve and promote occupational and environmental health and safety.[18,29,30]

The General Public

Communications of occupational and environmental health and safety information to the general public come mostly from news media organizations, government agencies, and NGOs, and also from other sources (Fig. 29-4). However, information is also passed to the public in a multistage process in which community opinion leaders learn about an issue from news sources and then pass that information on to others—partly because opinion leaders are viewed as trusted sources in their communities.[31] The public participates in risk communication at the community level as message senders by participating in public meetings with government officials, employers, and others.

Journalists

Journalists include print and broadcast reporters, photographers, and videographers. They work for mass-market media, such as daily newspapers and television and radio networks. They also

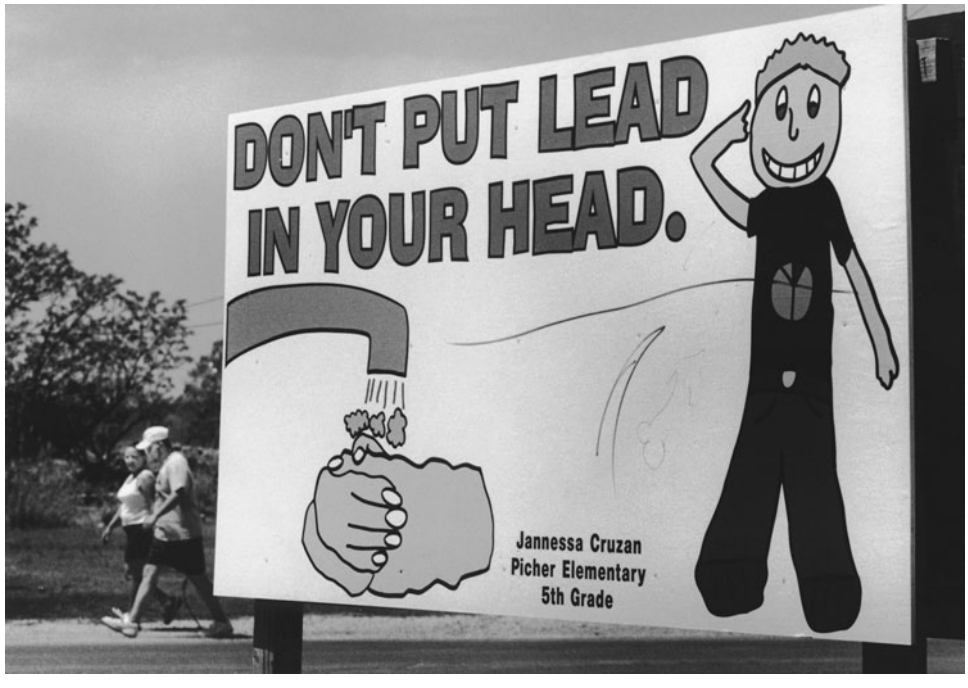
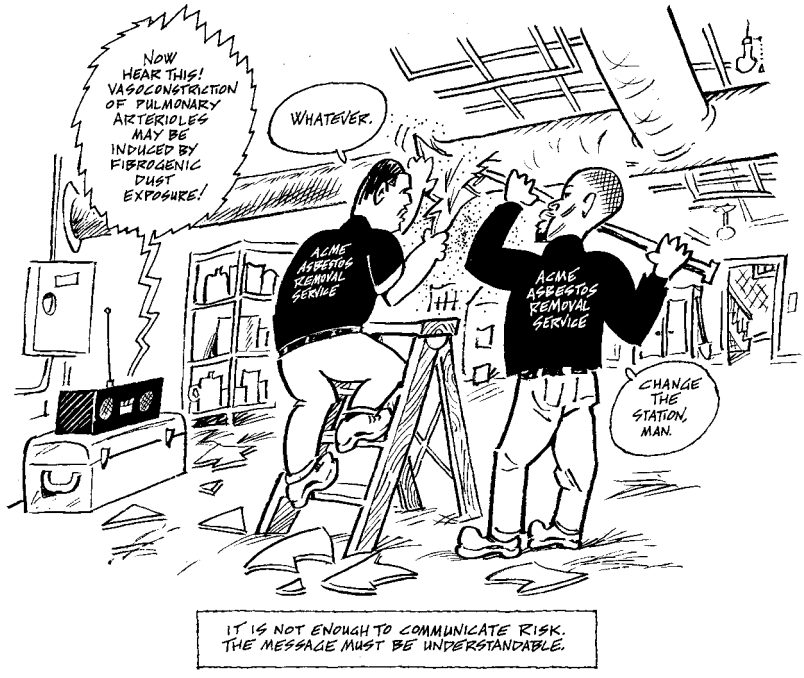


Figure 29-4. Billboard communicates to the general public the risks of lead exposure. (Photograph by Earl Dotter.)



(Drawing by Nick Thorkelson.)

work as freelance providers of content for other media, such as magazines, trade publications, and blogs.

Journalists can amplify risk information and stimulate action. For example, crane collapses in New York and Florida focused public attention on the problem of crane safety and forced OSHA to quickly plan new standards for crane safety (http://www.youtube.com/watch?v=kUVnbJ2Rf5I). In Las Vegas, newspaper attention to construction fatalities forced changes at Nevada OSHA, led to Congressional hearings, and brought nationwide attention to construction safety (http://www.pulitzer.org/archives/8381). Stories about fatalities and foundries in the *New York Times* and on *Frontline* on the Public Broadcasting System created public support for tougher enforcement of OSHA regulations at companies and other entities that often ignore them.

However, the news media infrequently pay attention to occupational health and safety issues. Reporters generally cover established "beats," such as crime, education, sports, and business. Their work is routinized, leaving little time and few resources to develop other sources and content. In addition, newspapers and other print media, which are suffering from drastically reduced revenues, have reduced investigative journalism, in which sufficient time and resources are allocated to study and report in depth on a subject like occupational health and safety.

Although journalists tend to focus on official sources for information, such as government officials and their press offices,[32] they also seek information from unofficial sources involved in an issue, such as workers and community members. Although there are a few journalists who specialize in science issues, many journalists often talk to scientists. However, journalists sometimes may have difficulty understanding scientific concepts and translating scientific information so that it is understandable to wide audiences, but they play a critical role communicating scientific information to wider audiences.[33]

Legislators

Legislators at the federal, state, and local level are targets of occupational health and safety and environmental health information from all other stakeholders. Since they pass laws and allocate public funds, they have a major impact on conditions in workplaces and the general environment. Legislators need accurate information on

risks and the methods and costs of prevention to allocate government resources wisely. Often with the help of aides and communications specialists, legislators communicate about risk issues to their constituents through many message channels.

MESSAGE DESIGN AND PRODUCTION

When risk communication follows routine or informal processes, such as regular reports to government agencies and discussions between employers and employees, little or no planning may be needed. However, in almost all other situations, planning is needed. Planning risk communication should include *(a)* setting objectives; *(b)* deciding which stakeholders should be addressed, depending on their understanding of risks and control measures and their role in prevention activities; *(c)* assessing current status, by in-depth interviews, focus groups, or surveys; and *(d)* determining the resources necessary to achieve the objectives.

When planning is completed, message design and production begins, with five important considerations: audience segmentation, design, style, content, and pretesting.

Audience Segmentation

The nine groups of risk communicators discussed in this chapter represent one level of audience segmentation. Within each group, smaller and more homogeneous segments can be identified. Each segment may require a different strategy and different types of information products. For example, workers may be divided by ethnicity, native language, age, gender, occupation, industry, work experience, and cognitive style. Each of these factors may affect how messages are received and sent. Employers may need to be segmented by these factors as well as the numbers of their employees. In general, the smaller the number of employees, the less expertise is present to evaluate risk information and implement control measures.[34]

Government agencies, NGOs, and professional organizations each have different types of leaders, constituents, missions, resources, and influences. These factors may affect the selection of targets during the segmentation process. Journalists may be segmented by such factors as experience preparing content on a specific topic, such as science, health, labor, business, and environment, as well as the media channels that have previously used their content. When preparing risk communication for peers, even researchers may need to be segmented into groups by their specialties. Occupational and environmental health and safety is an area where many specialties contribute, yet scientists are often so specialized that they have trouble understanding each other.

Message Design

All risk communication should have a compelling message that motivates audiences to listen to the message, understand it, learn from it, and take preventive actions—including passing the message to others. Strategies for communicating with all stakeholder groups should be guided by theories that explain how individuals and organizations receive, understand, and accept new information and how human attitudes and behaviors change.[35] Although much science-based risk information is based on mathematical probabilities, research on human perceptions indicates substantial presence of biases that cause people to assess risk in ways that are decidedly nonmathematical. These biases are stable across a range of situations and groups of people.[36] Perceptual biases exist between technical experts and members of the general public: experts usually underestimate risks, while others (generally with less information) usually overestimate them.[37] Emotional responses can be important too. In certain circumstances, risk communication that attempts to scare people can be effective.[38]

Message Style

Message style is most effective when it is adjusted for each audience segment addressed. For those with low literacy and/or English skills, direct and simple language should be used. Pictures can be used effectively to minimize the amount of text, especially for non-English speakers. "Before" and "after" (or "safe" and "unsafe") illustrations are often effective. Messages should resonate with

the experience of those addressed and have emotional appeal for them. Messages such as "You have a one-in-a-thousand chance of getting cancer from this chemical" lack the emotional impact that may be necessary to retain the audience's attention. The language used in most material safety data sheets (MSDSs), such as "use with adequate ventilation," is too technical and vague for most workers. To resonate with workers, risk communication should use real-world examples or cases studies of hazardous exposure situations that workers face regularly. A companion technique is the use of stories from individuals who have been injured or become ill on the job or who have experience with new health and safety procedures. Properly framed, these stories from peers can have high credibility.

Employers also need concise messages because of their generally heavy schedules and workloads. They trust and want to know about the practices of their peers and competitors. They want case studies with more detail than most workers want, and they want health and safety issues placed into context with all the other aspects of directing a company or some other entity. Messages should use language specific to the trade and industry that demonstrates familiarity with it.

In government, executive-branch officials and legislators need a variety of risk communications from all types of stakeholders to act in the public's interest. Although they will accept and use anecdotal or case-study information, they usually seek objective quantitative data about risk and control. The generalizability of such information helps them choose what appears best for those whom they serve. Communications to government agencies or officials should almost always be formal in tone. Sometimes brevity is essential, such as with legislators. For others, more extensive information is needed, such as reporting to government agencies on workplace injuries, illnesses, and deaths. Risk communication should be consistent with executive agency or legislative priorities—that is, in the right place at the right time. When communications include policy implications, they should be concise and logical, connecting risk and control information with viable policy options. The style used in communications to NGOs and professional associations should be similar to the style used in communicating with government agencies or officials. Such communications should demonstrate understanding of an organization's mission and the population or group served.

Risk communications to the public should be straightforward, simple, and brief. Like other audiences, the public needs to know what the hazards are, how and how many people are affected, and what experts recommend to control the problem. When data about risks of occupational and environmental diseases or injuries are presented, comparisons can be made to risks related to more common hazards, such as smoking or sedentary lifestyle. Stories of affected individuals are often critical to effectively communicating with the public. Journalists also need to use a similar style and have "hooks" with their stories to engage readers to attend to their messages. In communicating to journalists, one should provide them with contact information for stakeholders, especially researchers, workers, employers, community leaders, and government officials.

Message Content

Workers and community members need to know which conditions or exposures are dangerous, what the possible outcomes are, how likely it is that they may become injured or ill, and what preventive measures need to be taken. Messages should be succinct and meaningful. Messages such as "Be careful" lack sufficient specificity. For example, messages may need to tell workers how to report unsafe situations to peers and supervisors so that they will be addressed promptly, without negative consequences for their reporting these situations. Workers should be empowered to make or recommend changes. They should be informed of their rights under the Occupational Safety and Health Act, which include the rights to *(a)* refuse hazardous work until the hazard is corrected and *(b)* bring it to the attention of the supervisor or shop steward—without suffering negative consequences.

Like workers, employers also need reliable information on hazards, potential outcomes, and effective controls. Especially valuable are communications that feature practical controls for hazards and sources of additional information, such as databases containing solutions to health and safety problems. Risk alerts that give case studies, such as "fatal facts," can help

dramatize risks and motivate employer action (http://lni.wa.gov/Safety/Research/Face/Files/ConstructionFalls.pdf). Evaluation of safety controls, return on investment, and productivity impact may help persuade employers to adopt the appropriate controls,[39] especially as costs of interventions increase.

Some employers respond well to messages that view safety as essential for a well-run enterprise and good management practices. If workers believe that safety is only important to management because it saves the company money, then an entire safety program can be undermined. Safety should be integral to an employer's identity and culture, including encouraging workers to voice their concerns and suggestions on health and safety. Large employers, who rely on complex management systems, may find this reasoning to be persuasive; small employers may be more persuaded by evidence that "safety pays."

Researchers often communicate risk information in the style of scientific papers and presentations, following the sequence of background, hypotheses, methods, results, and discussion. Communications from researchers are generally judged to be scientifically acceptable by independent peers who carefully examine the content of these messages. This peer-review process gives other stakeholders some degree of confidence in the validity and reliability of the information, but it may not ensure its validity and reliability.[40]

To some extent, the nature, significance, and seriousness of some occupational and environmental health and safety issues make them compelling topics for journalists because they attract audiences. Occupational safety problems and resultant injuries are often relatively easy to report on because they are visible and because causes and effects are apparent. However, occupational and environmental diseases are infrequently covered in the news media because their causes appear to be unclear, complex, or uninteresting. In addition, with multiple stakeholders involved, journalists may not have access to necessary information or they may get conflicting information from multiple sources. Nevertheless, there have been many impactful stories, especially in newspapers, that have focused the public's attention on occupational and environmental diseases and injuries.

Pretesting

Before messages or informational materials are disseminated, they should be pretested, especially if they are intended for large or low-literacy audiences.[11,41,42] Pretests, which involve exposing a few members of an intended audience to a message and gauging their reactions, are done to identify ways to improve messages before they are broadly disseminated.[11,43,44] Messages may be tested to optimize their concept, clarity, credibility, length, or style. Sometimes different approaches to delivering the same risk information may be attempted. Pretests can reveal unexpected interpretations of a message and cultural or gender-based sensitivities about a message.

Messages may be tested by methods including interviews of individuals, focus groups, and surveys. After it is determined what should be tested, such as attention, motivation, recall, or cultural appropriateness, methods are designed to assess these qualities of the message. Test subjects should be drawn from the intended audience. Biases of those tested should be considered if they have not been randomly selected. Data from pretests should be analyzed to determine how tested messages can be improved.

MESSAGE CHANNELS

Risk messages reach audiences through message channels. For many purposes and audiences, communicators prefer electronic channels, which include television, radio, video, and the Internet, as well as e-mail, blogs, CD-ROMs, DVDs, podcasts, listservs, webinars, and social media, such as Facebook, MySpace, and Twitter. Social media are produced and distributed with an expectation of generating ongoing communications and interaction among pairs and groups of individuals. They are faster and cheaper than other channels, but they may be less reliable. Print channels, which include newspapers, magazines, books, scientific journals, and written reports and policies, are being increasingly adapted to the Internet. While comparatively expensive, live, face-to-face interaction through meetings, seminars, or training programs represents another channel, which can be very effective by facilitating the give-and-take of true communication.

Using the most appropriate channels involves understanding which channels work best for which audiences at which times. When making decisions about channels, risk communicators should also decide on the number of times each channel will be used for the same or a similar message. One use of a channel is almost never sufficient for positive results.

Messages to workers about hazards are best delivered in small brochures and training classes where workers are encouraged to ask questions and to discuss issues. "Toolbox talks," which are training sessions designed for and delivered in work settings – not training rooms, can be effective where training facilities are not available. They are short, practical, delivered by supervisors or peers, and have few or no written materials. Organized workers often highly trust messages that come from their unions.[45] Therefore, training sessions presented jointly by management and a labor union can be especially effective. Training and information dissemination at the worksite is very effective if workers are paid for the time they participate—which conveys respect for them and the importance of the message. Giving workers materials to take home and read on their own, which does not convey the importance of the information, is often ineffective in reaching them with messages. However, messages disseminated through union publications, before or after paid training, may reach family members who can help reinforce these messages.

Photographs and videos can be effective teaching tools, especially when time is allotted for discussion after viewing them. Merely showing a video without discussion is generally ineffective and may indicate to workers that management does not really want to know their opinions or concerns or to clarify issues raised. Video recordings of workers using their own words to describe workplace hazards, traumatic workplace events, or safety procedures that were or were not used can lend an unparalleled level of realism to training experiences. Video storytelling can help dramatize a problem and facilitate meaningful interactions about hazard control. Asking open-ended questions is an effective technique to help get workers talking about their experiences and concerns. Role playing is a useful technique in helping workers confront awkward situations, such as raising safety concerns with a boss. Health professionals can effectively deliver health messages to workers, such as helping them understand the importance of protective equipment, such as earplugs, respirators, and other personal protective equipment.

Employers obtain information from suppliers, trade journals, trade association meetings, and the Internet. Targeting health and safety information to purchasing agents is important since they are responsible for acquiring supplies and equipment with safety features.

Government agencies use official channels to disseminate occupational and environmental health and safety information to the general public, often with a fixed set of procedures. OSHA communicates with other agencies through various mechanisms, including notices of proposed rule making, which government agencies issue when they seek to add, remove, or change a regulation. These notices are published in the *Federal Register*, an official daily publication of federal regulatory activity that government agencies often use to communicate with stakeholders. Many other communications between agencies are done routinely, without notice. For example, state health departments regularly share injury and illness data with NIOSH. All government agencies provide "non-directed" information through postings on agency Web sites and other electronic media. For example, NIOSH has a monthly online newsletter known as *NIOSH eNews* (http://www.cdc.gov/niosh/enews/).

One official channel that NIOSH uses to provide comprehensive review of the scientific literature about a specific hazard, worker exposure to the hazard, risk of disease and injury associated with exposure, and methods to control the hazard is publication of criteria documents. These contain specific preventive recommendations so that, as far as practicable, no employee will suffer diminished function capacity or life expectancy as a result of work. The OSH Act presumes that OSHA will use criteria documents to help protect the lives and livelihoods of workers.

Researchers and members of professional organizations use scientific journals to provide and receive risk communication. One source estimated that at least 155 journals publish occupational health and safety papers. At least 35,000 papers, reports, pamphlets, fact sheets, and other occupational health and safety documents are released each year.[46] Many occupational health

and safety professionals also attend conferences, where they share information through formal presentations, interest group meetings, and networking events.

All stakeholders can be reached by mass-media channels, such as newspapers, magazines, books, and television. Some of the classic books in social criticism have raised concerns for occupational safety and health issues, including Upton Sinclair's *The Jungle*, Paul Brodeur's *Expendable Americans*, and Studs Terkel's *Working*. Fictional television programs have included plot or subplot lines that depict occupational and environmental hazards. Reality programs that focus on dirty, risky, or dangerous occupations can serve to communicate occupational health and safety information. However, they may also give the impression that the risk of work-related illnesses, injuries, and deaths are acceptable and inevitable.

Social Media

Social media, which combine technology, social interaction, and communication to form networks, establish business and personal relationships, and transmit information quickly, include e-mail, instant messaging, blogs, Wikis (collaborative Web sites), podcasts, Internet forums, microblogs (blogs with very short posts), social networking sites, and real simple syndication (RSS) feeds (syndicated Internet content). More information may be moving through social media than through traditional media. Organizational hierarchies and traditional channels of communication and information dissemination are evolving into faster and more complex interactions. The trend toward flatter (more horizontal or less hierarchical) organizations will likely promote use of social media for reaching the objectives of these organizations.

Social media may be used for occupational and environmental health and safety risk communication.[47] Social media can help achieve a socially constructed definition of risk through interactive, multipath communications in which all users are active generators and consumers of information. This can help focus opinions and move people to action. In addition, the use of social media may build communities of occupational and environmental health and safety practice through rapid communication among stakeholders. For example, social media could help government agencies obtain stakeholder views about new hazards or proposed policy changes. Social media may also be useful to researchers in soliciting stakeholder input and feedback on protocols. Because social media provide the real-time feedback on issues, they may be useful as surveillance tools. (See Chapter 3.). Used systematically, they could be used to identify prevalence or incidence of various work-related health conditions and possible control measures. [48]

All message channels have limitations. Use of social media may be problematic for large organizations that are accustomed to closely controlling their communications. Uses of social media must be monitored for adverse consequences, such as inaccurate information attributed to an organization, breached security of restricted information, and damage to an organization's credibility or reputation. Evaluations should be conducted to assess the capacity of social media to efficiently achieve the risk communication objectives of stakeholders.

EVALUATION

Risk communication should include evaluation of activities in the context of stated objectives and money spent. Process measures to consider include the following:

- Exposure of audience members: Did they have an opportunity to get the message?
- Attention to the message: Did they pay attention to it?
- Comprehension: Did they understand it?
- Involvement: Did it resonate with them?
- Acceptance: Did they recognize it as a valid message?
- Actions: What actions, if any, did they take?

If these process measures are positive, then changes in the knowledge, attitudes, skills, behavioral intentions, and/or behaviors of the audience members should be measured.

A larger focus of research in occupational and environmental health and safety should be on how people obtain and use risk information. There has not been sufficient research on the dissemination, adaptation, and use of occupational and environmental health and safety information. Senders of information often have

optimistic assumptions about dissemination of research findings in contrast and what is actually available to, and assimilated by, potential users of that information.[2] This mismatch especially applies to how employees, managers, and organizations obtain and use occupational health and safety information and the dynamics of decision making and stages of occupational change involving such information.[34,49] Nevertheless, promising examples of research on occupational safety and health risk communication and information dissemination can be cited, including approaches that are theory-based and rigorously designed.[50]

There are four output categories, or phases of output, that could be envisioned to track the flow of occupational and environmental health and safety research: immediate, intermediate, penultimate, and ultimate.[2,51,52] Each category is produced by government agencies, corporations, labor unions, trade associations, and NGOs. Each phase of output transforms the prior output to an input, and then disseminates this information as its own output. Various mechanisms exist to monitor and encourage these dissemination and transformation efforts. One method is to first identify representative organizations and institutions at each stage and then to monitor transformation activities and the number of outputs and inputs.[51,52] As outputs move downstream from immediate to ultimate and are absorbed and transformed by recipient organizations, a "dilution" effect may occur with respect to impact and the ability to measure contributions.[52] A one-to-one relationship rarely exists between receipt of an input at one level and a corresponding output at another level. Measuring inputs and outputs of various recipient organizations is potentially useful for monitoring the flow of safety and health research, but this approach does not necessarily indicate the full diffusion path or actual adoption of useful information. The diffusion of innovations theory is an alternative framework for monitoring dissemination of new ideas emanating from a research activity.[53]

ETHICS

Risk communicators are obliged to perform in compliance with ethical standards. A major issue is divided loyalty. Some risk communicators work for an organization, where they are expected to contribute both to the protection of workers and the financial status of the organization, which can create ethical dilemmas.

Other ethical issues concern the use of risk communications in lieu of other more costly or time-consuming interventions. For example, it is not acceptable to tell workers they have to continually avoid a hazard if methods exist to eliminate that hazard. The peer-review system used by researchers for risk communication helps to ensure that research findings are reported truthfully, with transparency about potential conflicts of interest among researchers. However, the system has limitations. The few examples of misconduct have included falsifying research results and plagiarizing the work of others. There are also cases in which peer reviews have not been thorough or critical enough to ensure the scientific rigor and quality of a publication. And, when conflicts of interest are not made clear, the peer-review system fails all of its stakeholders.[40]

CONCLUSION

Risk communication faces the following challenges:

1. Audiences are more diverse than ever before, making it difficult to reach many of them. In addition, today there are fewer intermediaries, such as labor unions and health and safety departments in large corporations, that can help communicators reach out and transmit timely, meaningful information.
2. Communicators and researchers must step outside their usual networks and address more diverse audiences. They must translate complex information so that it will be meaningful for lay audiences. This can be challenging for specialists who have spent years within their disciplines or realm of science.
3. It is increasingly difficult to get the attention of the public and the mass media, especially with downsizing and restructuring of traditional news outlets. Public health

communication competes for attention with economics, politics, sports, and entertainment. Even within public health, occupational and environmental health and safety competes for attention with consumer health, women's health, children's health, elder health, lifestyle health, prevention of infectious disease, and other sets of issues.

4. Occupational and environmental health and safety attracts public consciousness when there are catastrophes, such as mining disasters and crane collapses; scandals, such as flagrant violations of health and safety regulations; and emerging problems that have environmental or consumer health implications, such as hazards in the manufacture of flavorings. It is increasingly difficult to arouse indignation about the "quiet" toll of work-related injuries, illnesses, and deaths that occur throughout the year.
5. It is increasingly challenging to tailor risk communication to address the specific needs and learning styles of many different audiences.

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The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health or the Laborers' Health and Safety Fund of North America.

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Government Regulation of Environmental and Occupational Health and Safety in the United States and the European Union

Nicholas A. Ashford and Charles C. Caldart

The manufacturing, processing, and use of chemicals, materials, tools, machinery, and equipment in industrial, construction, mining, and agricultural workplaces often cause environmental, health, and safety hazards and risks. Occupational and environmental factors cause or exacerbate a wide variety of adverse health effects, placing heavy economic and social burdens on workers, employers, community residents, and taxpayers. In addition, consumer products, pharmaceuticals, and contaminated food present health risks to consumers.

Because voluntary efforts in the unregulated market have not succeeded in reducing the incidence of many of these health effects, the public has demanded government intervention into the activities of the private sector. This intervention takes many regulatory forms, including standard setting, government-imposed liability, pollution-reduction markets, and mandatory disclosure of information. This chapter addresses the major regulatory systems (regimes) designed to protect public health and worker health from chemicals discharged from sources that pollute the air, water, ground, and workplace. (The regulation of hazards posed by consumer products, pharmaceuticals, and contaminated food is beyond the scope of this chapter. See Chapter 9 regarding Food Safety.)

The establishment of standards and other legal requirements in these regulatory regimes has occurred over more than 40 years, a period that has seen changes in the use of scientific and technical information in regulatory initiatives and in legal doctrine—including the manner in which science, economics, and technological capability are viewed by the courts. Concepts of risk assessment, cost–benefit analysis, and technology forcing have evolved through the development of case law and changes in the political environment. Often, changes in one regulatory regime have led to changes in other regulatory regimes.

Standards can be classified in several ways. A *performance standard* specifies a particular outcome, such as a specified emission level above which it is illegal to emit a specified air pollutant; however, it does not specify how that outcome is

to be achieved. In contrast, a *design (specification) standard* specifies a particular technology, such as a catalytic converter, that must be used. Either type of standard can be based on *(a)* a desired level of protection for human health or environmental quality, *(b)* some level of presumed technological feasibility, *(c)* some level of presumed economic feasibility, or *(d)* some balancing of social costs and social benefits. Within each of these options, there is a wide spectrum of possible approaches. For example, a standard based on human health might protect only the average member of the population or, alternatively, the most sensitive individual. A technology-based standard might be based on what is deemed feasible for an entire industry, or on what is deemed feasible for each firm within the industry. Some standards might be based on a combination of these factors, such as both technological and economic feasibility.

Beyond standards are various information-based obligations that can also influence industrial behavior, such as *(a)* the required disclosure of—and retention of, or provision of access to—information on exposure, toxicity, chemical content, and production; and *(b)* required testing or screening of chemical products.

Under several new federal environmental laws, regulation in the United States in the 1970s and 1980s created the national model for *controlling*—rather than preventing—pollution in air, water, waste, and the workplace with an "end-of-pipe" focus. In the United States, the 1946 Administrative Procedure Act gave affected parties the right to participate in administrative rule making and to challenge agency actions in the courts. More specific provisions in new environmental laws expanded these rights, and some gave non-governmental organizations (NGOs) and citizens the right to go to court to enforce environmental standards against those who violate them. Eventually, European countries developed similar approaches to pollution control. Although citizens initially had fewer opportunities to challenge government and industry in court, this situation is changing as the European Union becomes the source of much environmental law for its member states.

As experience with end-of-pipe approaches accumulated, there was widespread recognition that *preventing* pollution—rather than merely controlling it—offered advantages for both the environment and industry. This recognition led, in the United States, to the Pollution Prevention Act of 1990, and in the Europe Union, to specific pollution prevention directives (such as the Integrated Pollution Prevention and Control Directive and the Seveso Directives) and several treaties among member states.

While the safety of food, drugs, and commercial products has been a continuing concern for some in the United States, there has been a renewed call for vigorous regulation of product safety both here and elsewhere after recent experiences with contaminated food, toothpaste, and toys, and with adverse reactions to widely used medications. The European Union has also advocated for stronger regulation to ensure the safety of food, medications, and commercial products.

Differing approaches to the testing and screening of industrial chemicals are found in the Toxic Substances Control Act (TSCA), which was passed by the U.S. Congress in 1976, and the REACH Initiative, a regulation of the European Union that became effective in 2007. Table 30-1 lists selected regulatory initiatives that form the backbone of governmental regulation in the United States and the European Union.

In the United States, exposures to toxic substances in the industrial workplace have been regulated primarily through the Occupational Safety and Health Act (OSH Act) of 1970 and TSCA. These federal laws have remained essentially unchanged since being passed, although serious attempts at reform have been made. Since 1990, sudden and accidental releases of chemicals (chemical accidents), which may affect workers and community residents, have been regulated under both the Clean Air Act and the OSH Act.

The OSH Act established the Occupational Safety and Health Administration (OSHA) in the Department of Labor to enforce compliance with the act, the National Institute for Occupational Safety and Health (NIOSH) (within the Centers for Disease Control and Prevention [CDC]) in the Department of Health and Human Services to perform research and conduct health hazard evaluations, and the independent, quasi-judicial Occupational Safety and Health Review Commission to hear employer

Table 30-1. Selected U.S. and EU Environmental Initiatives

The United States	The European Union
The Occupational Safety and Health Act (OSH Act) 1970	Occupational Health Directives
The Toxic Substances Control Act 1976	The REACH Directive 2006
The Clean Air Act (CAA) 1970, 1977, 1990	The Air Directives 1996, 2008
Water Legislation	
The Clean Water Act (CWA) 1972, 1977, 1987	The Water Directive 2000
The Safe Drinking Water Act 1974, 1986, 1996	
Hazardous Waste	
The Resource Conservation and Recovery Act (part of the Solid Waste Disposal Act) 1970, 1976, 1984	The Waste Directive 1975 Directive on Waste Electrical and Electronic Equipment 1991
Cleanup of Contaminated Land and Water	
The Oil Spill Provisions of the CWA 1972	The Liability Directive 2004
The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) (The "Superfund" Law) 1980, 1986	
Pollution Prevention and Inherent Safety	
The Pollution Prevention Act 1990	The Integrated Pollution Prevention and Control Directive 1996
Safety additions to the CAA and the OSH Act 1990 (workers) and CERCLA 1986 (the community)	The Seveso Directives 1982, 1996
The Safety of Food, Drugs, and Other Consumer Products	
The Consumer Product Safety Act 1972, 2008	Product, Drug, and Food Safety Directives
The Federal Hazardous Substances Act 1960, 2008	
The Food, Drug, and Cosmetic Act 1938, 1958, 1996	
Worker and Community Right-to-Know	
OSH Act Hazard Communication Standard 1983 Environmental Planning and Community Right to Know Act (EPCRA) 1986	Incorporation of the Aarhus Convention into EU Law 2006

and worker appeals of OSHA citations. The evolution of regulatory law under the OSH Act has profoundly influenced other environmental legislation, including the regulation of air, water, and waste, but especially the evolution of TSCA. Within the Environmental Protection Agency (EPA), the Office of Pollution Prevention and Toxic Substances administers TSCA; the Office of Air, Water, and Solid Waste and the Office of Emergency Response regulate media-based pollution; and the Office of Chemical Preparedness and Emergency Response implements the chemical safety provisions of the Clean Air Act.

STANDARD SETTING AND OBLIGATIONS OF EMPLOYERS, MANUFACTURERS, AND USERS OF TOXIC SUBSTANCES

The Occupational Safety and Health Act of 1970

The OSH Act requires OSHA to *(a)* encourage employers and employees to reduce hazards in the workplace and to implement new or improved safety and health programs; *(b)* develop and enforce mandatory job safety and health standards; *(c)* establish separate, but dependent,

responsibilities and rights for employers and employees for the achievement of improved safety and health conditions; *(d)* establish reporting and record-keeping procedures to monitor job-related injuries and illnesses; and *(e)* encourage states to assume the fullest responsibility for establishing and administering their own occupational safety and health programs, which must be "at least as effective" as the federal program.

The coverage of the OSH Act initially extended to all employers and their employees, except self-employed people; family-owned and -operated farms; state, county, and municipal workers; and workplaces already protected by other federal agencies or other federal statutes. In 1979, however, Congress exempted from routine OSHA safety inspections approximately 1.5 million businesses with 10 or fewer employees. (Exceptions to this are allowed if workers claim there are safety violations.) Because federal agencies (except the U.S. Postal Service) are not subject to OSHA regulations and enforcement provisions, each agency is required to establish and maintain its own effective and comprehensive job safety and health program. OSHA provisions do not apply to state and local governments in their role as employers. OSHA requires, however, that any state desiring to gain OSHA support or funding for its own occupational safety and health program must provide a program to cover its state and local government workers that is at least as effective as the OSHA program for private employees.

OSHA can begin standard-setting procedures either on its own or on petition from the Secretary of Health and Human Services, NIOSH, state and local governments, any nationally recognized standards-producing organization, employer or labor representatives, or any other interested person. The standard-setting process involves input from advisory committees and from NIOSH. When OSHA develops plans to propose, amend, or delete a standard, it publishes these intentions in the Federal Register. Subsequently, interested parties have opportunities to present arguments and pertinent evidence in writing or at public hearings.

Under certain conditions, OSHA is authorized to set emergency temporary standards, which take effect immediately, but are to be followed by the establishment of permanent standards within 6 months. To set an emergency temporary standard, OSHA must first determine that workers are in grave danger from exposure to toxic substances or new hazards and are not adequately protected by existing standards. Both emergency temporary and permanent standards can be appealed to the federal courts, but filing an appeals petition does not delay the enforcement of the standard unless a court of appeals specifically orders it. Employers may apply to OSHA for a temporary variance from a standard or regulation if they lack the means to comply readily with it, or for a permanent variance if they can prove that their facilities or methods of operation provide employee protection that is at least as effective as that required by OSHA.

The OSH Act provides two general means of protection for workers: *(a)* a general statutory duty on all employers to provide a safe and healthful workplace; and *(b)* promulgation of specific standards to which specified categories of employers must adhere. The Act imposes on virtually every employer in the private sector a general duty to "furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm." A recognized hazard may be a substance whose likelihood of harm has been the subject of research, giving rise to reasonable suspicion even though an OSHA standard has not been promulgated to protect workers from that harm. Placed on OSHA is the burden of proving that a particular substance is a recognized hazard, that occupational exposure to it results in a likelihood of serious harm, and that a reduction in exposure is necessary to protect workers from that harm. Because standard setting is a slow process, protection of workers through the employer's general duty could be especially important, but it is crucially dependent on the existence of reliable data on health effects and the willingness of a particular OSHA administration to use this statutory duty as a vehicle for protection.

The OSH Act specifically addresses the subject of toxic materials. It states, in Section 6(b)(5), that the Secretary of Labor (through OSHA), in promulgating standards dealing with toxic materials or harmful physical agents, "shall set the standard that most adequately assures, to the

extent feasible, on the basis of the best available evidence that no employee will suffer material impairment of health or functional capacity, even if such employee has a regular exposure to the hazard dealt with by such standard for the period of his working life." These words indicate a specific intent to regulate exposure to those hazards, such as chemical carcinogens and reproductive toxicants, whose effects may not be felt for several years or decades.

In the 1970s, OSHA set Section 6(b)(5) standards for asbestos, vinyl chloride, arsenic, dibromochloropropane (DBCP), coke oven emissions, acrylonitrile, lead, cotton dust, and a group of 14 carcinogens. In the 1980s, OSHA added standards for benzene, ethylene oxide, and formaldehyde, and tightened the standard for asbestos to reflect its status as a carcinogen. In the early 1990s, OSHA set standards for cadmium, bloodborne pathogens, glycol ethers, and confined spaces. The agency also lowered the permissible exposure limit (PEL) for formaldehyde from 1.00 to 0.75 ppm (averaged over an 8-hour period) and issued a process safety management (PSM) rule designed to reduce the incidence of chemical accidents. Standards were established for methylene chloride in 1997, and hexavalent chromium in 2006.

Under Section 6(b), the burden of proving the hazardous nature of a substance is placed on OSHA, as is the burden of establishing that the proposed controls are technologically and economically feasible for the regulated industries. The evolution of case law associated with the handful of standards that OSHA promulgated through this section of the OSH Act has been important for the implementation of the OSH Act and environmental law generally. In reviewing OSHA's hazardous substance standards, the federal circuit courts of appeal squarely addressed the difficult issue of when scientific information is adequate to sustain the statutory requirement that the standards be supported by "substantial evidence" on the record as a whole. They also addressed *(a)* the extent to which economic factors were permitted or required to be considered in the setting of the standards, *(b)* the meaning of feasibility, *(c)* OSHA's technology-forcing authority, *(d)* whether a cost–benefit analysis was required or permitted, and *(e)* the extent of the jurisdiction of the OSH Act in addressing different degrees of risk.

The Toxic Substances Control Act of 1976

TSCA directs the EPA to require data from industry on the production, use, and health and environmental effects of chemicals. TSCA also requires the manufacturer of new chemicals—and existing chemicals put to a significant new use—to file a premanufacturing notification with the EPA, detailing known information about the chemical. In addition, TSCA authorizes the EPA to regulate production and use of those chemicals found to pose an unreasonable risk to human health or the environment. Such regulation may take a variety of forms, such as labeling requirements, tolerance levels, and outright bans on chemical use. The EPA may also order a specific change in chemical process technology, or require repurchase or recall of banned chemicals. In addition, TSCA gives aggrieved parties, including consumers and workers, specific rights to sue to enforce the Act, with the possibility of awards of attorneys' fees. (This feature was not included in the OSH Act.)

The EPA has issued a worker protection standard for asbestos (at the new OSHA limit of 0.2 fibers/cm^3), which applies to state and local government asbestos-abatement workers not covered by OSHA. Although the potential for broader regulation of workplace chemicals exists under TSCA, the EPA has not been aggressive in this area. Between 1977 and 1990, of the 22 TSCA regulatory actions taken on existing chemicals, 15 addressed polychlorinated biphenyls (PCBs), which the EPA has a specific statutory directive to address under TSCA. Only three of the remaining seven regulations—pertaining to asbestos, hexavalent chromium, and metalworking fluids—have a strong occupational exposure component. Although the EPA declared formaldehyde a probable carcinogen and IARC classified it as a definite (Group 1) carcinogen, the EPA chose not to take regulatory action on it, opting instead to defer to OSHA workplace regulations.

Nonetheless, the OSH Act and TSCA together provide potentially comprehensive and effective information-generation and standard-setting authority to protect workers. In particular, the information-generation activities under TSCA can provide the necessary data to establish that a substance is a "recognized hazard" that, even in

the absence of a specific OSHA standard, must be controlled by the employer to meet the OSH Act's general duty to provide a safe and healthful workplace.

The potentially powerful role of more comprehensive TSCA regulation was seriously challenged by the Fifth Circuit Court of Appeals in 1991, when it overturned an omnibus asbestos phase-out rule that was issued under TSCA in 1989. The court ruled that the EPA could not ban a chemical under TSCA without having first determined that other regulatory alternatives that would have been less burdensome to industry would not have eliminated the unreasonable risk. This called for a more comprehensive, detailed, and resource-intensive analysis than the one EPA conducted prior to the promulgation of the asbestos rule. Rightly or wrongly, for more than a decade the EPA has viewed this case (which was not appealed to the U.S. Supreme Court) as a significant impediment to future TSCA standards, and it has generally regarded regulation of chemicals under TSCA—except for PCBs—to be a nearly impossible task for the foreseeable future. Even so, TSCA continues to be important for its surviving authority to require the testing of chemicals and the reporting and retention of information. Under the Obama administration, the EPA has promised to revitalize TSCA.

THE CONTROL OF GRADUAL POLLUTION IN AIR, WATER, AND WASTE IN THE UNITED STATES

The Clean Air Act

Although significant changes were made to the statute in 1977 and 1990, the basic regulatory structure of the Clean Air Act (CAA) was established with the Clean Air Act Amendments of 1970. The CAA regulates both stationary and mobile sources of pollution, taking into account *(a)* the relative contributions of each to specific air pollution problems, and *(b)* the relative capacity of different kinds of sources within each category to reduce their emissions. The recognition that new sources using newer technology might be able to achieve greater emission reductions than old sources with older technology led to distinctions between new and existing sources in the Act's stationary and mobile-source provisions. Although driven by equity considerations regarding the relative financial and technical burdens of pollution reduction, this approach has unwittingly discouraged modernization or replacement of facilities and resulted in the operation of older facilities—especially power plants—beyond their expected useful life. For new sources within each industrial sector, the Act sought to achieve uniformity and to encourage technological innovation through the technology-forcing capability inherent in stringent standards. Court decisions recognizing the EPA's technology-forcing authority under the CAA were greatly influenced by earlier decisions upholding OSHA's technology-forcing approach to worker protection.

Section 109(b)(1) of the CAA directed the EPA to establish primary ambient air quality standards that would protect public health with an adequate margin of safety. As interpreted by the courts and supported by congressional history, these standards are to be established without consideration of economic or technological feasibility. In addition, Section 109(b)(2) mandates the establishment of secondary ambient air quality standards to protect the public welfare within a reasonable time. (See Chapter 6.)

Both the federal government and the states have key roles in protecting the ambient air under the CAA. Ambient air quality (concentration) standards are established by the federal government for a few "criteria" pollutants (identified later in this chapter) designated by the EPA. For each such pollutant, EPA establishes primary and secondary standards as discussed earlier. These ambient standards are to be attained through *(a)* emission limitations placed on individual existing polluters through permits issued by state government as a part of their state implementation plans (SIPs) (in Section 110); *(b)* nationwide emission limitations for new sources, established by the EPA and known as new source performance standards (in Section 111); and *(c)* a combination of federal and state restrictions on mobile sources. An emission standard, in contrast to an ambient concentration standard, is expressed as an emissions rate (mg emitted per 100 kg of product, per hour, day, week, quarter, year, BTU, passenger mile, or other unit of measurement).

The CAA does not establish ambient standards for substances designated as "hazardous air pollutants," but rather requires compliance with nationwide emission limitations set by the EPA. Hazardous air pollutants are those recognized as extraordinarily toxic and eventually regarded as no-threshold or low-threshold pollutants. Initially, these were to be regulated (in Section 112) to protect public health with an ample margin of safety and, as with the primary ambient standards for criteria pollutants, emission standards for hazardous air pollutants were to be established without consideration of economic burden. These pollutants, Congress determined, were sufficiently dangerous to preclude any reliance on atmospheric dispersion and mixing as a means of reducing their ambient concentrations. The reliance on federal emission standards reflected congressional concern with *(a)* "hot spots" of localized intense pollution, and *(b)* the fact that release of these substances often is intermittent, or sudden and accidental, rather than continuous. Ambient concentration standards were considered impractical and of little relevance for the sporadic and idiosyncratic sources of hazardous air pollutants, and uniform federal emission standards were considered necessary. (However, California did establish an ambient standard as a complement to the federal emission limitation on vinyl chloride.)

In the early stages of the implementation of the stationary-source provisions of the CAA, the EPA focused on *(a)* the ambient air quality standards for criteria pollutants, and *(b)* emission standards for new sources of criteria pollutants and for all sources emitting any of seven regulated hazardous air pollutants (discussed later in this chapter). Initially, prior advisory ambient standards were made mandatory for the following criteria pollutants: carbon monoxide, sulfur dioxide, oxides of nitrogen, large particulate matter, and photochemical oxidants. In 1979, the standard for photochemical oxidants was narrowed to cover only ground-level ozone, and it was relaxed from 0.08 ppm to 0.12 ppm, averaged over 1 hour. The standard for coarse particulate matter (inhalable particulates up to 10 μm in diameter, PM_{10}) was adopted in 1987. In 1997, the ozone standard was further revised to 0.08 ppm, and in the same rule making, the particulate standard was altered to place more stringent requirements on smaller (<2.5 μm) respirable particles ($PM_{2.5}$), with a 24-hour limit of 65 mg/m^3. In 2006, the $PM_{2.5}$ limit was further lowered to 35 mg/m^3. A standard for a sixth criteria pollutant—airborne lead—was promulgated in 1978; in 2008, the EPA lowered the permissible airborne lead concentration from 1.5 to 0.15 $\mu g/m^3$. (Current primary air quality standards set under Section 109 are found in Table 6-1 in Chapter 6.) In addition, following a 2007 U.S. Supreme Court decision that the EPA has the authority under the CAA to regulate carbon dioxide, the EPA has indicated its intention to establish national limits on greenhouse emissions from automobiles and to regulate carbon dioxide emissions from power plants.

In Section 112 of the CAA, Congress directed the EPA to set emission standards for hazardous air pollutants at levels that protect public health with an ample margin of safety. It is likely that this directive reflected an early assumption that, although very dangerous, hazardous pollutants exhibited a finite threshold—a non-zero level of exposure below which no harm would occur. As the 1970s progressed, however, there was a growing recognition among scientists that this assumption might be wrong and that for many hazardous pollutants there was no detectable level of exposure below which one could confidently predict that no harmful or irreversible effects (especially cancer or birth defects) would occur.

The EPA was therefore faced with a major challenge. Arguably, given its mandate to protect public health with an ample margin of safety, the EPA was required to ban the emission of several hazardous substances, which would essentially ban the use of these substances in many industries. Seeking to avoid this outcome, the EPA adopted a policy of setting Section 112 emission standards at levels that could be achieved by technologically feasible means. (This was the approach then followed by OSHA in setting standards for exposure to workplace chemicals. For carcinogens, OSHA considered no levels to be safe, and it established control requirements for carcinogens at the limit of technological feasibility.)

Using this approach, the EPA set finite (non-zero) standards for arsenic, asbestos, benzene, beryllium, coke oven emissions, mercury, vinyl

chloride, and radionuclides. The standard-setting process was slow and had to be forced by litigation; it took 4 to 7 years to establish a final standard for each of these substances. Had the EPA continued to set standards for more substances, and had it used the technological feasibility approach to spur the development of cleaner technology, environmental NGOs may well have been content to allow the implementation of Section 112 to proceed in this fashion. However, when the setting of new Section 112 standards stalled during the Reagan administration (1981–1988), the National Resources Defense Council, an environmental advocacy organization, decided to press the issue in court.

NRDC v. EPA, decided by the District of Columbia Circuit Court of Appeals in 1987, placed new limitations on the EPA's approach to regulating hazardous air pollutants by ruling that the EPA must determine an acceptable (usually non-zero) risk level for a hazardous air pollutant prior to setting a Section 112 standard for that pollutant. In reaction to this case and to revitalize the moribund standard-setting process, Congress amended Section 112 in 1990 to specify a two-tiered approach: the initial use of technology-based standards, with residual risks to be addressed later by health-based standards.

In the 1990 CAA amendments, Congress listed 189 substances as hazardous air pollutants and directed the EPA to add other substances to the list if they "present or may present … a threat of adverse human effects (including, but not limited to, substances which are known to be or may be reasonably anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic) or adverse environmental effects whether through ambient concentration, bioaccumulation, deposition or otherwise." (Section 112 of the CAA.)

The EPA was directed to set maximum achievable control technology (MACT) technology-based performance standards over a 10-year period for categories of major stationary sources—defined as those emitting more than 10 tons per year of any single hazardous pollutant or more than 25 tons combined. MACT standards must require the maximum feasible degree of reduction (including a prohibition on emissions, where achievable), but must reflect the cost of achieving emissions reduction and any non-air and environmental impact and energy requirements. MACT standards for new sources must be at least as stringent as those met by the best-performing similar source, and MACT standards for existing sources must be at least as stringent as those met by the average of the best performing 12% of similar sources.

For categories of smaller (area) stationary sources, the EPA is authorized to set standards that are less restrictive than the MACT standard, based either on generally achievable control technology (GACT) or the use of specified management practices. For pollutants with an identifiable health threshold, the EPA is authorized to forgo the technology-based approach and to instead set health-based standards that ensure an ample margin of safety—essentially the original mandate of Section 112. In addition, the EPA was obligated to issue a report on risk, which it issued in 2004. If no new legislation recommended by that report is enacted within 8 years, the EPA must issue such additional regulations as are necessary to protect public health with, in general, an ample margin of safety; specifically for carcinogens, these regulations must ensure that lifetime exposure risks are less than 1 in 1,000,000. The EPA has made substantial progress on establishing MACT and GACT standards, but it has just begun the task of developing risk-based or health-based approaches. The 1990 amendments to the CAA also placed an increased emphasis on toxic air pollutants emitted by mobile sources. In 2007, the EPA issued the Mobile Source Air Toxics regulation, which was designed to lower benzene concentrations in gasoline and restrict automotive emissions of benzene and several other toxic substances.

Water Legislation

The two most important federal statutes regulating water pollution are the Clean Water Act (CWA) and the Safe Drinking Water Act (SDWA). The CWA regulates the discharge of pollutants into navigable surface waters and into smaller waterways and wetlands that are hydrologically connected to navigable waters. The SDWA regulates the level of contaminants in public drinking water supplies.

The Clean Water Act

The CWA had its origins in the Federal Water Pollution Control Act Amendments of 1972. The basic structure of the Act was then established, although it was refined and refocused by the Clean Water Act Amendments of 1977 and by the Water Quality Act Amendments of 1987. The CWA regulates the discharge of pollutants to surface waters from point sources, mainly industrial facilities and municipal sewage treatment plants (known under the Act as publicly owned treatment works, or POTWs). The CWA totally prohibits any discharge of a pollutant from a point source to surface waters unless it is done in conformance with the requirements of the Act. Since 1972, the Act has retained as an explicit national goal the elimination of all point-source discharges to surface waters by 1985. Although the no-discharge goal was not met—and may never be fully attainable (by any date)—it has helped focus implementation of the Act on gradual, but inexorable, pollution reduction as discharge limits are made more stringent over time.

The centerpiece of this pollution reduction scheme is the National Pollutant Discharge Elimination System (NPDES) permit. In theory, all point sources must have an NPDES permit before discharging pollutants to surface waters, but some (mostly smaller ones) still do not. The NPDES permit, which is issued after public notice and an opportunity for comment, is meant to incorporate all requirements of the Act, including applicable discharge limits. Point sources are subject both to limits based on technology and to limits based on water quality, and to the more stringent of the two when they overlap.

The technology-based limits are established by the EPA as national standards. To set these standards for industrial dischargers, the EPA first divided industry into various categories, and then established effluent limits for each category, based on its assessment of what was technologically and economically feasible for point sources within that category. In addition, as required by the Act, the EPA set different standards within each industrial category for *(a)* conventional pollutants (currently biochemical oxygen demand, fecal coliforms, oil and grease, pH, and total suspended solids); *(b)* toxic pollutants (now 129 designated chemical compounds); and *(c)* nonconventional pollutants, such as total phenols.

Recognizing that conventional pollutants usually are amenable to treatment by types of pollution control equipment that have long been used at conventional sewage treatment facilities, standards for conventional pollutants are set according to what can be obtained through the use of the best conventional pollution control technology (BCT), taking into account the reasonableness of the cost. In contrast, standards for toxic and nonconventional pollutants are set according to the EPA's determination of the level of pollution reduction that can be achieved by applying the best available technology economically achievable (BAT). Originally, Congress had directed the EPA to set health-based standards for toxic pollutants, on a pollutant-by-pollutant basis. However, this directive resulted in only a few standards, mostly for pesticide chemicals. The political difficulty of establishing national health-based standards for toxic chemicals led environmental NGOs, in a suit against the EPA to compel regulation, to agree to a schedule for setting technology-based standards for a list of designated toxic pollutants. Congress formally endorsed this approach in 1977 by amending the Act to require the EPA to set BAT standards for all of the toxic pollutants on that list.

Under the CWA, the EPA is to consider both control and process technologies in setting BAT standards, which are to result in reasonable further progress toward the national goal of eliminating the discharge of all pollutants and are to require "the elimination of discharges of all pollutants [where] such elimination is technologically and economically achievable." An individual discharger may obtain a cost waiver from BAT standards for nonconventional pollutants if it cannot afford to comply, but no cost waiver is available from the standards for toxic pollutants. For new industrial sources within an industry category, the EPA is to set standards based on best available demonstrated technology (BADT), which can be more stringent than BAT or BCT because of the greater technological flexibility inherent in the design and construction of a new facility. Although industry-wide costs are to be considered by the EPA in establishing BADT

standards, no waivers are available to individual applicants once the standards are set.

The CWA also imposes technology-based standards on POTWs, based on the limitations that can be met through the application of secondary sewage treatment technology. In essence, this requires an 85% reduction in biochemical oxygen demand and total suspended solids. Since 2000, the Act has also required POTWs to comply with the EPA's combined sewer overflow (CSO) policy, which is designed to eventually terminate or substantially minimize the discharge of untreated or partially treated sewage during periods of high rain or snow melt.

In addition, the CWA imposes limitations on discharges by industrial sources into POTWs. Such discharges are known under the Act as indirect discharges (because the pollutants are not discharged directly to surface waters but rather are discharged indirectly to surface waters through a public sewer system). Limitations on indirect discharges are known under the Act as pretreatment standards, because they have the effect of requiring the indirect discharger to treat its wastewater before discharging it to the POTW for further treatment. The EPA has set national technology-based limitations, known as the categorical *pretreatment standards*, on indirect discharges of toxic pollutants by firms in certain industrial categories. The Act also requires the POTW to set such additional pretreatment limits and requirements as are necessary to ensure the integrity of the sewage treatment process and to prevent the indirectly discharged pollutants from passing through the sewer system and causing a violation of the POTWs discharge permit.

For the first 15 to 20 years of the Act's implementation, the primary focus was the establishment and implementation of the technology-based limits, as discussed earlier. More recently, however, considerably more attention has been given to the Act's system of water quality–based limits, which is equally applicable to industrial sources and POTWs. Since 1972, the CWA has directed states to establish, and periodically revise, ambient (in-stream) water-quality standards for all of the lakes, rivers, streams, bays, and other waterways within their borders, and it has required the EPA to set and revise these standards to the extent that a state declines to do so. In addition, the Act has required, since 1977, that NPDES permits include such additional discharge limits beyond the national technology-based limits as may be necessary to meet the ambient water-quality standards of the waterway in question.

To help call attention to these water-quality requirements, Congress in 1987 added what became known as the "toxic hot spot" provision of the CWA, which directed the EPA and the states to *(a)* identify those bodies of water that were in violation of ambient water-quality standards because of toxic pollution, *(b)* identify those point sources whose discharges of toxic pollutants were contributing to those violations, and *(c)* develop an individual control strategy for each such source—which almost always meant a revision of the source's NPDES permit to add or tighten limits on toxic pollutants. Another provision of the Act that has prompted the addition or tightening of water quality–based discharge limits has been the requirement that the states (and, if they decline, the EPA) calculate a total maximum daily load (TMDL) for all waters that are in violation of ambient water-quality standards. For any particular body of water, the TMDL for a particular pollutant is the total amount of that pollutant that may be discharged to the water body in a day without violating the relevant ambient water-quality standard. When a TMDL is set, it often leads to a tightening of the NPDES permits of those point sources whose discharges are contributing to the particular violation of water-quality standards. Although the TMDL requirement has been in the Act since 1972, the states and the EPA have been slow to implement it. Over the past 15 years or so, however, as a result of several successful suits by environmental NGOs seeking to compel the EPA to set TMDLs in the face of state inaction, the TMDL requirement has received greater attention. As a result, the inclusion of water quality–based limits in NPDES permits has become considerably more commonplace.

The Safe Drinking Water Act

Although some sources of drinking water are also regulated as surface waters under the CWA, the legislation specifically designed to protect the safety of the drinking water delivered to the public from public water systems is the Safe

Drinking Water Act (SDWA). Passed in 1974 after a series of well-publicized stories about the number of potential carcinogens in the Mississippi River water (used as drinking water by the City of New Orleans), the SDWA contains very little that is designed to address sources of drinking-water pollution. Instead, the SDWA directs the EPA to set national health-based goals, known as *maximum contaminant level goals* (MCL goals) for various drinking-water contaminants and to set MCLs that are as close to the MCL goals as is technologically and economically feasible. All public water systems, defined as those with at least 15 service connections or that serve at least 25 people, are required to meet the MCLs.

Over the Act's first 8 years, the EPA set only 23 federal drinking-water standards. Dissatisfied with the pace of implementation, Congress amended the Act in 1986 to spur the EPA into action. It directed the EPA to set standards (MCLs and MCL goals) for 83 specified contaminants within 3 years and to set standards for 25 additional contaminants every 3 years thereafter. Ten years later, with scores of MCLs and MCL goals then on the books, Congress scaled back. In a 1996 compromise endorsed by both environmental NGOs and water suppliers, Congress eliminated the requirement for 25 new standards every 3 years. At the same time, it added provisions that effectively ensured both that the standards that had been set would largely be allowed to remain in place, and that new standards would be far slower in coming—and likely would be relatively weaker because of the addition of a cost–benefit requirement.

Since then, the primary focus of the SDWA program has been bringing public water systems throughout the United States into compliance with the existing standards. Although the MCLs are set at a level deemed to be technologically and economically feasible, many water systems have had difficulty affording the cost of meeting, and monitoring for, the MCLs. To attempt to ameliorate the financial burden on municipal water systems, the SDWA has periodically made federal funds available for technology upgrades and infrastructure improvements. The task, however, remains a daunting one. In 2002, the EPA estimated that approximately $151 billion would be needed over the next 20 years to upgrade 55,000 community water systems in the United States.

Regulation of Hazardous Waste

Broadly speaking, the generation, handling, and disposal of hazardous wastes are regulated by the interaction of two federal statutes. The primary federal law regulating hazardous wastes is the Solid Waste Disposal Act. In 1970, Congress amended the Act with the Resource Conservation and Recovery Act (RCRA), and the law has come to be popularly known by that name. RCRA was given regulatory teeth with a set of 1976 amendments under which the EPA, in 1980, promulgated regulations establishing a cradle-to-grave system for hazardous wastes that tracks the generation, transportation, and disposal of such wastes and establishes standards for their disposal. Initially, however, the EPA's disposal standards were minimal to nonexistent and did little to discourage the landfilling of chemical wastes. This led Congress, in 1984, to pass sweeping amendments to RCRA that *(a)* established a clear federal policy against the landfilling of hazardous wastes unless they have first been treated to reduce their toxicity, and *(b)* gave the EPA a specific timetable by which it had to either set treatment standards for various categories of waste or totally ban the landfilling of such waste. As a result, the EPA has set treatment standards, commonly known as the *land disposal restrictions* (LDRs), for hundreds of types of hazardous wastes. These standards are based on the EPA's assessment of the best demonstrated available technology for treating the waste in question. (See Chapter 10.)

Thus, RCRA directly regulates the handling and disposal of hazardous wastes. And, by establishing a set of requirements that must be followed once hazardous waste is generated, it also indirectly regulates the generation of hazardous wastes. RCRA regulations have increased the cost of disposing of most types of waste 100-fold over the past 30 years. In this way, RCRA has operated as a de facto tax on the generation of hazardous waste.

Another law that acts as an indirect check on hazardous waste generation—and that provides additional incentive to ensure that waste is safely disposed—is the Comprehensive Environmental

Response, Compensation, and Liability Act (CERCLA, also known as the *federal Superfund Law*). Its two primary foci are *(a)* the remediation (cleanup) of hazardous waste contamination resulting from imprudent handling and disposal practices of the past, and *(b)* the recovery of remediation costs from those designated as responsible parties under the Act. CERCLA imposes liability for the costs of remediating a hazardous waste site, both on the owners and operators of the site and on those generators of hazardous waste who sent waste to the site. Because the owners and operators are often business entities that are no longer financially viable, CERCLA liability often falls most heavily on the generators. And CERCLA liability is strict liability, meaning that the exercise of reasonable care by the generator is not a defense. Furthermore, unless the generator can establish a convincing factual basis for distinguishing its waste from all or part of the contamination being remediated, CERCLA liability is joint and several, meaning that each responsible party is potentially liable for the full cost of remediation. As a practical matter, this means that the cost of remediation will be borne by those among the responsible parties who are financially solvent, unless they can prove that the waste they sent to the site did not contribute to the contamination being remediated.

The prudent business entity, then, has a strong financial incentive to take such actions as will minimize the likelihood that it will face CERCLA liability in the future. As the only certain way to avoid such liability is to refrain from generating the waste in the first place, CERCLA does provide a rationale for pollution prevention. In addition, it provides business firms with an incentive to meet—or perhaps to go beyond—RCRA regulations in dealing with wastes that they generate.

Nevertheless, much hazardous waste is still generated in the United States. Some hazardous wastes are not adequately treated and not safely disposed, and some hazardous waste contamination is not being adequately addressed (or addressed at all). RCRA and CERCLA both contain what might reasonably be called loopholes and gaps in coverage. Hazardous waste contamination remains an ongoing issue. For example, the EPA has not taken an aggressive approach toward "E wastes," the discarded electronic components that have become increasingly common in our computer-dominated society. In addition, the most common treatment methodology incorporated into the EPA's RCRA treatment standards is incineration, which has brought with it release of airborne contaminants that has only recently been meaningfully addressed by regulation. There is no question, however, that the United States has made considerable progress since the late 1970s, when disposal of chemical wastes in unlined landfills—at a cost of about $15 per ton—was common practice.

THE CHEMICAL SAFETY PROVISIONS OF THE U.S. CLEAN AIR ACT: OBLIGATIONS IMPOSED BY THE EPA AND OSHA TO PREVENT THE SUDDEN AND ACCIDENTAL RELEASE OF CHEMICALS

Although the first congressional response to the concern generated by the deadly industrial accident in Bhopal, India in 1984, was the Emergency Planning and Community Right to Know Act of 1986, the chemical safety provisions of that law are focused almost solely on mitigation—not on accident prevention. A much greater potential for a direct focus on accident prevention can be found in the 1990 amendments to the Clean Air Act, although that potential has yet to be realized by the EPA and OSHA.

As amended in 1990, Section 112 of the Clean Air Act directs the EPA to *(a)* develop regulations regarding the prevention and detection of accidental chemical releases, and *(b)* publish a list of at least 100 chemical substances (with associated threshold quantities) to be covered by these regulations. The regulations must include requirements for the development of risk-management plans (RMPs) by facilities using any of the regulated substances in amounts above the relevant threshold. Each of these RMPs must include a hazard assessment, an accident prevention program, and an emergency release program. Similarly, Section 304 of the Clean Air Act Amendments of 1990 directed OSHA to promulgate a process safety management (PSM) standard under the OSH Act. Section 112(r) of the revised Clean Air Act also imposes a general

duty on all owners and operators of stationary sources, regardless of the particular identity or quantity of the chemicals used on site. These parties have the following duties:

- To identify hazards that may result from accidental chemical releases, using appropriate hazard assessment techniques
- To design and maintain a safe facility, taking the steps necessary to prevent such releases
- To minimize the consequences of accidental chemical releases that do occur

Thus, firms are now under a general duty to anticipate, prevent, and mitigate accidental releases. In defining the nature of this duty, Section 112(r) specifies that it is a general duty in the same manner and to the same extent as that imposed by Section 5 of the OSH Act. Because Section 112(r) specifically ties its general duty obligation to the general duty clause of the OSH Act, case law interpreting the OSH Act provision should be directly relevant. In the 1987 *General Dynamics* case, the District of Columbia Circuit Court of Appeals held that OSHA standards and the general duty obligation are distinct and independent requirements and that compliance with a standard does not discharge an employer's duty to comply with the general duty obligation. Similarly, compliance with other Clean Air Act chemical safety requirements should not relieve a firm's duty to comply with the Act's general duty clause. In addition, the requirement that owners and operators design and maintain a safe facility would seem to extend the obligation into the area of primary prevention, rather than merely hazard control.

The Clean Air Act also requires each state to establish programs to provide small business with technical assistance in addressing chemical safety. These programs could provide information on alternative technologies, process changes, products, and methods of operation that help reduce emissions to air. However, these state mandates are unfunded and may not be uniformly implemented. Where they are established, linkage with state offices of technical assistance, especially those that provide guidance on pollution prevention, could be particularly beneficial.

Finally, the 1990 CAA amendments established the independent Chemical Safety and Hazard Investigation Board (CSHIB). The Board is to investigate the causes of "accidents," conduct research on prevention, and make recommendations for preventive approaches, much as the Air Transportation Safety Board does concerning airplane safety.

In response to its Clean Air Act mandate, OSHA promulgated a workplace PSM standard in 1992, designed to protect employees working in facilities that use highly hazardous chemicals and those working in facilities with more than 10,000 pounds of flammable liquids or gases present in one location. The list of highly hazardous chemicals in the standard includes acutely toxic, highly flammable, and reactive substances. The PSM standard requires each employer to do the following:

- Compile safety information (including process flow information) on chemicals and processes used in the workplace
- Complete a workplace process hazard analysis every 5 years
- Conduct triennial compliance safety audits, and develop and implement written operating procedures
- Conduct extensive worker training
- Develop and implement plans to maintain the integrity of process equipment
- Perform pre-startup reviews for new (and significantly modified) facilities
- Develop and implement written procedures to manage changes in production methods
- Establish an emergency action plan
- Investigate accidents and near-misses at their facilities

In 1996, the EPA promulgated regulations setting forth requirements for the risk management plans (RMPs) specified in the Clean Air Act. The RMP rule is modeled after the OSHA PSM standard and is estimated to affect about 66,000 facilities. The rule requires a hazard assessment (involving an offsite consequence analysis, including worst-case risk scenarios and compilation of a 5-year accident history), a prevention program to address the hazards identified, and an emergency response program.

In 2002, seeking to achieve more comprehensive control of reactive hazards that could have catastrophic consequences, the Chemical Safety

and Hazard Investigation Board urged OSHA to amend its 1992 PSM standard and the EPA to amend its 1996 RMP regulation. The Board also asked OSHA to define and record information on reactive chemical incidents that it investigates or is required to investigate. These recommendations have largely been ignored. The Board also expressed concern that the material safety data sheets (MSDSs) mandated by OSHA do not adequately identify the reactive potential of chemicals. And although the EPA and OSHA signed a memorandum of understanding on the topic in 1996, the U.S. General Accounting Office (GAO) report in 2001 called for better coordination on chemical safety among the EPA, OSHA, the CSHIB, and other agencies.

In late 2009, legislation was being promoted to require OSHA to prepare or revise MSDSs for the list of chemicals in the PSM standard and to generally strengthen OSHA's approach to chemical safety. Many aspects of chemical safety are not covered by specific workplace standards. Most OSHA standards that apply to chemical safety have their origin in the consensus standards adopted in 1971 under Section 6(a) of the OSH Act and are therefore very outdated. However, the general duty obligation of the OSH Act imposes a continuing duty on employers to seek out technological improvements that would improve safety for workers.

POLLUTION PREVENTION AND INHERENTLY SAFER PRODUCTION IN THE UNITED STATES

End-of-pipe control focuses on *(a)* reducing or collecting the harmful emissions, effluents, or waste from industrial processes, and *(b)* in the case of workers' exposure, on ventilating the workplace or providing personal protective equipment—usually without altering inputs, feedstocks, processes, or final products. Early preoccupation with minimizing air and water pollution often shifted the problem to the hazardous waste stream and/or increased workplace exposure, resulting in what is popularly known as a *media shift*. It also often changed the nature of the hazard by increasing the potential for chemical accidents (sudden and unexpected chemical releases, sometimes with accompanying fires and explosions), thus resulting in what is popularly known as a *problem shift*.

Pollution prevention—what the Europeans call *cleaner production* or *cleaner technology*—received its first political push in the United States in the mid-1980s with the pursuit of *waste minimization*, an economically driven movement that grew out of a recognition that often the best way to avoid the rising costs of treatment and disposal of hazardous wastes is simply to generate less waste. Depending on the context and the time period, pollution prevention has also been known as *elimination of pollution at the source*, *source reduction*, and *toxics use reduction*.

Pollution prevention is not a refined version of pollution control. It involves fundamental changes in production technology: substitution of inputs, redesign and reengineering of processes, and/or reformulation of the final product. It may also require organizational and institutional changes. *Inherent safety*, also known as *primary accident prevention*, is the analogous concept for the prevention of sudden and accidental chemical releases. Inherent safety is a concept similar to—and often a natural extension of—pollution prevention. The common thread linking the two concepts is that they both attempt to prevent the possibility of harm, rather than reduce the probability of harm, by eliminating the problem at its source. The changes necessary for pollution prevention are often associated with improvements in eco-efficiency and energy efficiency. In the context of chemical production, they often involve the exploration of alternative synthetic pathways and green chemistry initiatives. The search for, and identification of, alternative production methods may also promote the development and use of inherently safer production technology, although the minimization of accident potential may require a somewhat different—though not necessarily inconsistent—set of changes.

The Pollution Prevention Act (PPA) of 1990 encourages both pollution prevention and inherent safety through the rubric of *source reduction*—any practice which *(a)* reduces the amount of any hazardous substance, pollutant, or contaminant entering any waste stream or otherwise released into the environment (including fugitive emissions) prior to recycling, treatment, or disposal; and *(b)* reduces the hazards to

public health and the environment associated with the release of such substances, pollutants, or contaminants. Explicitly included within the statutory definition are "equipment or technology modifications, process or procedure modifications, reformulation or redesign of products, substitution of raw materials, and improvements in housekeeping, maintenance, training, or inventory control." Explicitly excluded is any practice that "alters the physical, chemical, or biological characteristics or the volume of a hazardous substance, pollutant, or contaminant through a process or activity which itself is not integral to and necessary for the production of a product or the providing of a service." Therefore, pollution prevention and primary accident prevention both come within the PPA's definition of source reduction. In contrast, recycling or reuse does not meet this definition unless it is done as part of a closed-loop production process—as is often done in the metal-finishing industry when metals are recovered at the end of the process and immediately returned to the beginning of the process.

The PPA states, as the national policy, that pollution is to be addressed in a hierarchical fashion. First, "pollution should be prevented or reduced at the source whenever feasible." Second, "pollution that cannot be prevented should be recycled in an environmentally safe manner, whenever feasible." And, third, "disposal or other release into the environment should be employed only as a last resort." The EPA established the Office of Pollution Prevention, as required by the PPA, but EPA's overall commitment to implementing the PPA has waned considerably since the early 1990s. Neither the Clinton nor Bush administrations wholeheartedly embraced the potential opportunities for fundamental change that the PPA represents, and the EPA's source reduction strategy has largely been allowed to languish.

Nonetheless, many industrial firms have found it in their economic interest to adopt pollution prevention approaches, since they can eliminate the need for waste handling, disposal, and treatment and can save pollution control and abatement costs. However, these incentives may be absent concerning the prevention of chemical accidents, because these events are both rare and not statistically predictable; therefore, rational behavioral changes premised on cost avoidance is largely absent.

THE RIGHT TO KNOW AND INFORMATION-BASED STRATEGIES FOR ENCOURAGING ALTERNATIVE TECHNOLOGY IN THE UNITED STATES

The various media-based environmental laws (regulating waste as well as air and water pollution) incorporate several information-disclosure requirements. For example, under the Clean Air Act and Clean Water Act, pollution sources are required to monitor discharges of pollutants and report the results to the EPA or the state. Similarly, those who generate, transfer, treat, store, or dispose of hazardous waste must maintain records of the types and amounts of wastes involved, and they must supply these records to the appropriate agency. The existence of adequate and accurate information of this nature is essential to the optimal operation of both the command-and-control (or regulatory) approaches to risk reduction and to market-based approaches. Without such information, neither type of policy can succeed.

Beyond the particular informational requirements attached to the various regulatory regimes, however, there is a class of more broadly based information-disclosure requirements, popularly known as *right-to-know laws.* In essence, these laws give workers and community residents general statutory rights to *(a)* be apprised of the substances to which they are—or may be—exposed and *(b)* to obtain information about the hazardous nature of these substances.

These laws have two risk-reduction goals: *(a)* to give information to potentially exposed persons that may enable them to take action to avoid or limit such exposure, and *(b)* to encourage those who create such exposures—the manufacturers and users of toxic chemicals—to take actions to reduce or eliminate these exposures.

Many political and legislative initiatives focusing on the right to know emerged in the United States in the early 1980s, when the direct regulation of toxic substances was being deemphasized by federal agencies. Environment and worker

advocates shifted their attention to information as an area of political action because the setting and enforcement of standards regulating toxic substances had slowed significantly.

Workplace information disclosure and reporting requirements began at the state and local level, and in 1983 were added at the federal level, when OSHA promulgated, under the OSH Act, the comprehensive Hazard Communication Standard. These workplace initiatives preceded the more general community right-to-know requirements that were to come a few years later. Worker right-to-know initiatives greatly influenced the evolution of community right-to-know initiatives. Worker and community right-to-know laws largely focus on scientific information about chemicals: *(a)* the ingredients of chemical products and the specific composition of pollution in air, water, and waste; *(b)* the toxicity and safety hazards posed by the related chemicals, materials, and industrial processes; and *(c)* information related to exposure of various vulnerable groups to harmful substances and processes. The 1976 Toxic Substances Control Act (TSCA) had earlier given statutory authority to the EPA to require firms to test the chemicals they produced or imported—and gave citizens unprecedented access to toxicity and exposure information about industrial chemicals. However, the EPA had barely staffed a bureaucracy to implement TSCA when the Reagan antiregulatory revolution of the 1980s swept government. As a result, the EPA budget for TSCA fell by the wayside. TSCA's informational initiatives have yet to be fully developed.

In 1986, Congress amended the federal Superfund law with the Superfund Amendment and Reauthorization Act of 1986, known as SARA. Beyond strengthening certain provisions governing the cleanup of hazardous waste sites, Congress took, with SARA, a significant step toward reducing the likelihood of new hazardous substance contamination in the future. Title III of SARA created the Emergency Planning and Community Right to Know Act (EPCRA), a comprehensive federal community right-to-know program implemented by the states under guidelines promulgated by the EPA. The central feature of EPCRA is broad public dissemination of information concerning the nature and identity of chemicals used at commercial facilities. EPCRA has four major provisions: emergency planning, emergency release notification, hazardous chemical storage reporting, and the Toxic Chemical Release Inventory (TRI) (Table 30-2).

The implementation of EPCRA began with the creation of state and local bodies to implement it. Section 301 required the governor of each state to appoint a state emergency response commission (SERC), to be staffed by "persons who have technical expertise in the emergency response field." In practice, these state commissions have tended to include representatives from the various environmental, public health, and safety agencies in the state. Each state commission, in turn, was required to divide the state into various local emergency planning districts and to appoint a local emergency planning committee (LEPC) for each of these districts. These state and local entities are responsible for receiving, coordinating, maintaining, and providing access to the various types of information required to be disclosed under the Act.

EPCRA established four principal requirements for reporting information about hazardous chemicals. Section 304 requires all facilities that manufacture, process, use, or store certain extremely hazardous substances in excess of certain quantities to provide emergency notification to the SERC and the LEPC of an unexpected release of one of these substances. Section 311 requires facilities covered by the OSHA Hazard Communication Standard to prepare and submit to the LEPC and the local fire department MSDSs for chemicals covered by the OSHA standard. Under Section 312, many of these same firms are required to prepare and submit to the LEPC an emergency and hazardous substance inventory form that describes the amount and location of certain hazardous chemicals on their premises. Section 313 requires firms in the manufacturing sector to report to EPA annually certain routine releases of hazardous substances. The Toxics Release Inventory (TRI) is the EPA database containing these hazardous release reports. In addition, Section 303 requires certain commercial facilities to cooperate with their respective LEPCs in preparing emergency response plans for dealing with major accidents involving

Table 30-2. EPCRA Chemicals, Reportable Actions, and Reporting Thresholds

	Section 302 Emergency Planning	Section 304 Unexpected Releases	Sections 311/312 Chemicals in storage	Section 313 (TRI) Routine Emissions
Chemicals covered	356 extremely hazardous substances	>1,000 substances	500,000 products with MSDSs* (required under OSHA regulations)	650 toxic chemicals and categories†
Reportable actions and reporting thresholds	Threshold planning quantity 1–10,000 pounds *present on site at any one time* requires notification of the SERC and LEPC within 60 days upon onsite production or receipt of shipment.	Reportable quantity, 1–5,000 pounds, *released at any time within a 24-hour period*; reportable to the SERC and NEPC	TPQ or 500 pounds for Section 302 chemicals; 10,000 pounds *present on site at any one time* for other chemicals; Copy if requested to SERC/LEPC; annual inventory Tier I/Tier II report to SERC/LEPC/local fire department by March 1.	25,000 pounds per year *manufactured or processed*; 10,000 pounds a year *used*; certain persistent bioaccumulative toxics have lower thresholds; annual report to the EPA and the state by July 1.

* MSDSs on hazardous chemicals are maintained by a number of universities and can be accessed through http://www.hazard.com.

† The TRI reporting requirement applies to all federal facilities that have 10 or more full-time employees and those that manufacture (including importing), process, or otherwise use a listed toxic chemical above threshold quantities, and that are in one of the following sectors: Manufacturing (Standard Industrial Classification (SIC) codes 20 through 39), Metal mining (SIC code 10, except for SIC codes 1011,1081, and 1094), Coal mining (SIC code 12, except for 1241 and extraction activities), Electrical utilities that combust coal and/or oil (SIC codes 4911, 4931, and 4939), Resource Conservation and Recovery Act (RCRA), Subtitle C hazardous waste treatment and disposal facilities (SIC code 4953), Chemicals and allied products wholesale distributors (SIC code 5169), Petroleum bulk plants and terminals (SIC code 5171), and Solvent recovery services (SIC code 7389).

EPA, Environmental Protection Agency; EPCRA, Emergency Planning and Community Right to Know Act; MSDSs, material safety data sheets; LEPC, local emergency planning committee; NEPC, national emergency planning committee; OSHA, Occupational Safety and Health Administration; SERC, state emergency planning committee; TRI, Toxics Release Inventory.

Source: The Community Planning and Right-to-Know Act, EPA 550-F-00-004, March 2000.

hazardous chemicals. The applicability of these provisions to any particular facility depends on the amount of the designated chemicals that it uses or stores during any given year.

In 1990, Congress added two more chemical reporting requirements to federal law. The Pollution Prevention Act amended EPCRA to require firms subject to TRI reporting to annually report their source reduction (pollution-prevention) practices and waste management practices. In addition, the 1990 Clean Air Act Amendments directed the EPA and OSHA to issue regulations governing prevention of chemical accidents. Under these regulations, each facility using certain chemicals above specified threshold quantities is required to *(a)* develop a risk management program to identify, evaluate, and manage chemical safety hazards; *(b)* submit a risk management plan (RMP) summarizing its program to the EPA or the state; and *(c)* report accidental chemical releases above specified thresholds. In addition, chemical manufacturers and refineries must file startup, shutdown, and malfunction (SSM) plans with the EPA or state air regulators. Some RMP information is available at http://www.epa.gov/enviro. Worst-case chemical accident scenarios—known as *offsite consequence analyses* (OCAs)—are now available for reading, but not for copying, in locally designated reading rooms.

Taken as a whole, these requirements constitute a broad federal declaration that firms choosing to rely heavily on hazardous chemicals in their production processes may not treat information regarding their use of those chemicals as their private domain. Except for trade-secrecy

protections (relating to specific chemical identity) that generally parallel those available under the OSHA Hazard Communication Standard, there are no statutory restrictions on the disclosure of EPCRA information to the general public.

Beyond such scientific information, however, disseminating (or providing access to) legal and technological information may be even more important for empowering workers and community residents to facilitate a transformation of hazardous industries and their practices. Legal information, in this context, refers to statements (or explanations) of the rights and obligations of producers, employers, consumers, workers, and the general public concerning potential or actual chemical exposures. Technological information includes information regarding *(a)* monitoring technologies; *(b)* options for controlling or minimizing pollution, waste, or chemical accidents; and *(c)* available substitutes or alternative inputs, products, and processes that may prevent pollution, waste generation, and chemical accidents. Disseminating such technological information tends to have a far greater potential to induce technological change than does simply collecting and disseminating scientific information about chemical risks and exposures. (For an extensive discussion of worker and community right to know, see Ashford and Caldart [2008] in the Further Reading section at the end of this chapter.)

ENVIRONMENTAL LAW IN THE EUROPEAN UNION

Protection of the environment and the workplace are major challenges in Europe as well as in the United States. In the past, the European Community was strongly criticized for putting trade and economic development ahead of environmental and worker health and safety considerations. More recently, however, the European Union (EU), recognizing that sound development cannot be based on the depletion of natural resources, the deterioration of the environment and public health, and poor workplace conditions, has developed progressive environmental and labor protections to advance environmental and workplace goals. (The EU is also known as the European Community [EC], especially in its directives, regulations, and other legal documents.)

In the early years of the modern environmental era (1970 to 1980), the United States tended to lead the way, with enforceable emission and effluent standards for industry, mandatory environmental impact assessments for federal projects, and the incorporation of both the precautionary principle and the "polluter pays" principle into law. Since the Reagan antiregulatory revolution of the 1980s, support for the precautionary principle has declined in the United States while Europe has incorporated it into its legal framework. (See Ashford and Hall, 2011.) Now, EU environmental law has gone beyond the United States in some respects and contains initiatives not present in U.S. federal law, such as eco-labeling, restrictions on packaging, and mandatory recycling of vehicles and electronic products. The EU has also signed many multilateral environmental agreements that the United States has neither signed nor ratified, such as the Basel Convention on Hazardous Waste, the Convention on Biological Diversity, and the Biosafety Protocol.

The sources of EU law are the EU Treaties, general principles of law, international obligations, and secondary legislation, comprised of regulations, directives, and decisions. Regulations are directly binding in that they do not need to be implemented by the member states to be transformed into national law. In contrast, directives give member states a specified time to transpose them into national law. The EU also adopts nonbinding conventions and resolutions. The European Union now negotiates international environmental agreements on behalf of all EU countries.

The Regulation of Air, Water, and Waste

There have been extensive changes in the EU's regulation of air, water, and waste over the past decade. The EU approach to air and water pollution generally parallels, and lags behind, that of the U.S. Clean Air Act, Clean Water Act, and Safe Drinking Water Act. Various directives address air pollution, such as the 2008 Directive on Air Quality and Management. A comprehensive

Water Framework Directive replaced seven older directives and now anticipates the development of water-quality concentration limits and polluter-discharge limitations. EU waste regulation goes beyond that of the United States in some areas, such as its Directive on Waste Electrical and Electronic Equipment (WEEE), which requires the producers of electrical and electronic products to finance the collection, treatment, recovery, and environmentally sound disposal of WEEE from households.

The Prevention of Chemical Accidents

One of the most important and well-known EU directives is the EU Directive on Major Accident Hazards of Certain Industrial Activities—the Seveso Directive. First implemented in 1982, it requires member states to ensure that all manufacturers prove to a competent authority that *(a)* major hazards have been identified in their industrial activities; *(b)* appropriate safety measures, including emergency plans, have been adopted; and *(c)* information, training, and safety equipment have been provided to onsite employees. A revised version, the Seveso II Directive, came into effect in 1997. Seveso II introduced new concepts, such as *inherent safety*, and extended the scope of the directive to a broader range of installations. The emphasis on inherent safety (the safety analogue to pollution prevention) as the preferred approach places EU practice ahead of U.S. practice, which continues to emphasize traditional, secondary accident prevention measures. Other updates in Seveso II included the introduction of new requirements for safety management systems, an emphasis on emergency planning and land-use planning, and stronger provisions on inspections performed by member states.

The Integrated Pollution Prevention and Control Directive (IPPC)

The purpose of the European Union's Integrated Pollution Prevention and Control Directive (IPPC), adopted in 1996 and amended four times since, is to provide a high level of environmental protection by *preventing* pollution wherever practicable, and by otherwise reducing (controlling) emissions to air, water, and land from a range of industrial and agricultural sectors and activities. Its implementation is known as the Sevilla Process, named after the EU institution located in Seville, Spain, that establishes the permit conditions.

The IPPC represents a shift in focus in EU environmental law from separate emphases on air pollution, water pollution, and waste—usually employing "end-of-pipe" or secondary prevention approaches—to an integrated emphasis on preventing pollution (and sudden and/or accidental releases) at the source. In this way, it parallels the U.S. approach articulated in the 1990 Pollution Prevention Act.

Unlike many of the very general EU Directives, the IPPC places specific restrictions on member states to ensure that individual firms (in the energy industries, the production and processing of metals, the mineral industry, the chemical industry, waste management, livestock farming, and others defined in Annex I of the directive) comply with operating permits. The basic obligations defined in the permits require each regulated installation to do the following:

- Use the best available pollution-prevention measures and techniques—those that produce the least waste, use the least hazardous substances, and maximize the recovery and recycling of substances generated.
- Prevent all large-scale pollution.
- Prevent, recycle, or dispose of waste in the least polluting way possible.
- Use energy efficiently.
- Ensure accident prevention and damage limitation.
- Return sites to their original state when the activity is over.

In addition, each permit must contain several specific provisions, including the following:

- Emission limit values for polluting substances (with the exception of greenhouse gases if an emissions-trading scheme applies)
- Any required soil, water, and air protection measures

- Waste management measures
- Measures to be taken in exceptional circumstances, such as when leaks, malfunctions, or temporary or permanent stoppages occur
- Minimization of long-distance or transboundary pollution
- Monitoring of releases

Approximately 60,000 installations across the EU were required to operate with IPPC permits by late 2007. (Because it was acknowledged that the implementation of these new and considerably tougher BAT rules on all existing installations in the EU could be expensive, the directive granted the covered installations an 11-year transition period counting from the day the directive became effective.) The permits were to be coordinated in addressing together all waste and pollution streams and were to be based on the concept of best available techniques (BAT) for minimizing pollution from various point sources to achieve a high level of protection of the environment as a whole. In the European context, BAT can include performance requirements that anticipate innovation, and not simply the levels of control achievable by existing technologies (as is generally implied by the related term best available technology in the United States). In many cases, BAT means radical environmental improvements within industries. Sometimes it may be costly for companies to adapt their plants to BAT. Identification of required performance levels achievable by BAT is undertaken by the EU Center in Seville and published in its Best Available Techniques Reference Documents (BREFS). In accordance with the United Nations Aarhus Convention (discussed below), and with appropriate safeguards for commercial and industrial secrecy, this information must be made available to interested parties.

Access to Information and Participatory Rights

The EU has several directives implementing the United Nations Aarhus Convention on Access to Information and Public Participation on Decision Making and Access to Justice in Environmental Matters. The Aarhus Convention enunciates three basic rights:

1. *Access to environmental information*: The right of everyone to receive environmental information that is held by public authorities. This not only includes information on the state of the environment but also on environmental policies and measures taken, and on human health and safety indicators related to the state of the environment. Citizens are entitled to obtain this information within 1 month of the request and without having to say why they require it. In addition, public authorities are obliged, under the Convention, to actively disseminate environmental information in their possession.
2. *Public participation in environmental decision making*: The right to participate from an early stage in environmental decision making. Arrangements are to be made by public authorities to enable citizens and environmental organizations to comment on proposals for projects affecting, or plans and programs relating to, the environment. These comments are to be taken into due account in decision making, and information on the final decisions and the underlying rationale are to be provided to the public.
3. *Access to justice*: The right to challenge, in a court of law, public decisions that have been made without respecting the two aforementioned rights, or in violation of environmental law in general.

In the EU, the initial effort to implement the Aarhus Convention came with the issuance of three EU directives, each of which corresponds to one of these three rights. First, EU Directive 2003/04/EC calls for the creation of lists and registers of environmentally relevant information, preferably using electronic databases. EC Regulation 166/2006, which also implements the Aarhus Convention, established the European Pollutant Release and Transfer Register (PRTR), which harmonizes rules under which the member states are to regularly report information on pollutants to the European Union Commission.

Not only does the Convention create a right of access (if requested), but it also creates a duty to inform.

The Convention's second right anticipates public participation in decision making in a timely manner. The Convention "invites the parties to promote public participation in the preparation of environmental policies as well as standards and legislation that may have a significant effect on the environment." The EU took a first step toward the implementation of this participatory ideal with the promulgation of Directive 2003/35/EC.

The third right, access to justice—through access to the courts and to judicial, or at least governmental, review of decision making, met with considerable initial resistance, because in many European countries with parliamentary governments, regulations and laws are not usually challenged through review in the courts. Eventually, however, the EU adopted this right in Directive 2005/370/EC.

There is an important caveat to this set of directives in the EU, however, as particular member states may choose to limit the extent to which these participatory rights are extended to their citizens. Accordingly, executive branch rule-making may continue to operate behind closed doors, especially in parliamentary systems. Whether the participatory goals of the Aarhus Convention will be realized remains to be seen, and it may ultimately depend on citizen and NGO pressure.

Worker Health and Safety

While occupational health and safety standards set by OSHA in the United States are mandatory, the EU employs both mandatory obligations and standards, such as established for carcinogens and mutagens in Parliamentary & Council Directive 2004/37/EC, and "indicative" standards, such the indicative Occupational Exposure Limits (OELs) established in Council Directive 2000/29/EC and Commission Directive 2006/15/EC. The individual member states implement the indicative OELs through their national laws and have the discretion to, but need not, make them mandatory. The member states may also establish more stringent levels of protection, or they may choose to protect workers from hazards not covered by EU initiatives. The EU establishes a hierarchy of measures to be used by employers to reduce the risk from hazardous substances in the workplace. In order of preference, these are as follows:

- Elimination of the need to use the substance (prevention of exposure)
- Substitution with a less hazardous substance
- Technical and organizational measures to reduce employee contact with, or the air concentration of, the substance
- As a last resort, the use of personal protective equipment by employees

While this is similar to the U.S. preference for engineering controls instead of personal protective equipment, the focus on primary prevention and cleaner and inherently safer technology for worker protection has not been a hallmark of U.S. regulation. In the United States, the emphasis on pollution prevention has been primarily in the environmental—as opposed to occupational—context.

The REACH Initiative

In an effort to obtain more extensive information on the nature of chemicals used within the EU, and to lay the foundation for regulation of these chemicals, the European Commission issued a regulation in 2006 establishing the REACH (*R*egistration, *E*valuation, and *A*uthorization of *Ch*emicals) system. (See also Chapter 26.) REACH is the European corollary to TSCA, which was passed 30 years earlier in the United States. REACH came into force in 2007 and created the European Chemicals Agency (ECHA), which became fully operational 1 year later to administer European chemicals policy. (More information is available at http:/echa.europa.eu.)

The main elements of REACH are implementation of uniform procedures (to be in place by 2012) for the registration and evaluation of new and existing chemicals, transfer of responsibility for producing and assessing chemicals data to industry, expansion of the responsibilities of downstream users, and regulation of chemicals through an authorization process. Chemicals of "very high concern" can be placed on the market

only by explicit authorization. It is expected that animal testing will be kept to a minimum and that alternative testing methods, such as short-term bioassays and structure–activity relationships, will be used instead.

There are an estimated 30,000 chemicals used on a significant scale in the EU, and the requirements for their registration (to be completed over a period of 11 years) depend on the amount produced annually. Generally, the system is three-tiered. All chemicals produced in amounts from 1 to 10 tons per year may be initially registered upon the submission of only minimal toxicological information. A safety assessment report is necessary for substances produced in quantities over 10 tons per year (estimated to be about 15,000 substances). This report must identify the relevant chemical properties and exposure profiles, and it must also identify risk-reduction measures to ensure the safe use of the chemical by the producer through downstream users. In addition, a safety data sheet identifying necessary risk reduction measures must be supplied to, and if necessary modified by, all actors in the supply chain. All substances produced in quantities greater than 100 tons per year (estimated to be about 10,000 substances), and all substances produced in smaller quantities that are suspected of being hazardous (estimated to be about 5,000 substances), will be initially evaluated by the relevant authorities in the member states after registration. ECHA approves evaluations, prioritizes candidates for authorization, and determines whether restrictions or authorization (of use or introduction into commerce) is warranted.

In contrast to the well-defined data requirements for risk assessment, the responsibility for risk management is defined only cursorily and superficially in REACH. Manufacturers and importers must identify and apply the "appropriate" measures to "adequately control" the risks identified in the chemical safety assessment, and must, where suitable, recommend them in the required safety data sheets.

If this risk management element of REACH is to be meaningful, there must be a clear definition of "adequate control" and sanctions for noncompliance. Currently, the point of reference for "adequate control" appears to be the probable no-effect concentration (PNEC) for the environment and the derived no-effect level (DNEL) for human health. (The DNEL is equivalent to the no observed adverse effect level [NOAEL] in U.S. parlance.) However, the sanctions for exceeding these levels are not clear. In addition, the sanctions for failing to identify risks during the registration process are very limited, and they often are insufficient to overcome the producer's incentive to withhold such information.

Chemicals with certain hazardous properties—known as *substances of very high concern*—must be separately authorized. These include *(a)* substances that can cause cancer or mutations or are toxic to reproduction (the so-called CMR-substances); *(b)* those that are persistent, bioaccumulative, and toxic (PBT), or very persistent and very bio-accumulative (vPvB); and *(c)* substances, such as endocrine disrupters, that are identified on a case-by-case basis as causing probable serious effects to humans or the environment of an equivalent concern. For any of these substances, the burden of proof shifts from the authorities to the producers, regardless of the amount of the substance produced. An analysis of alternative substances or technologies—and a substitution plan—must be provided by the firm seeking authorization. In general, an authorization of the chemical for certain uses will be issued if the producer is able to prove either that the risks of these uses can be "adequately controlled" or that their socioeconomic benefits exceed their risks. These relatively broadly worded requirements leave wide discretion to the implementing authorities. In the event that the risk cannot be adequately controlled, an analysis of alternative substances or technologies—and a substitution plan—must be provided by the firm seeking authorization.

A Final Comment

The Barack Obama administration is reversing many of the anti-regulatory initiatives of the George W. Bush administration, and the federal government appears to be recommitting itself to elevating the status of health, safety, and environmental law in the United States. This will doubtless have complementary effects in the EU and on international environmental accords as well. It may also lead to more coherent and

harmonized policies among the industrialized nations.

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31

Legal Remedies

Leslie I. Boden, Peter S. Barth, Neil T. Leifer, David C. Strouss, Emily A. Spieler, and Patricia A. Roche

Physicians often express anxiety or confusion about the treatment of patients with occupational and environmental health problems. This occurs, in part, because questions may arise about the legal responsibility for the consequences of occupational and environmental health problems. Disputes about who carries this burden—employer or worker, manufacturer or consumer, polluter or community member—often involve the physician in providing input about causation or extent of injury. This role, in turn, can interfere with the trust and openness needed to ensure the best health outcome for the patient.

The provision of occupational health services presents particular problems for the treating physician. When treating a patient for a minor illness or injury contracted at home, the physician can deal directly with the patient—both the cause of the problem and its treatment will, in all likelihood, be within the patient's control. Health problems caused at work present greater legal, economic, and social complexities. Neither the patient nor the physician can ignore the external forces that will influence the patient's progress and prognosis. Neither the patient nor the physician has control over the workplace design and the hazards that cause the medical problem. The employer's attitudes and policies regarding workplace hazards and workplace-induced disabilities, the extent of job security that the employer offers during periods of disability, and the availability of monetary benefits all influence the course of the patient's recovery. Ultimately, any successful attempt to deliver health services to working people must consider the roles played by the employer, the employer's representatives (including employer-retained attorneys and physicians), the employer's insurer, job security and job mobility of the patient, and economic prospects for the patient.

As a result, many others—employers, attorneys, insurance company representatives, state and federal government health and safety officials, and workers' compensation agency officials—will be looking over the shoulder of the treating physician when a patient's medical problem is caused by work. The interest of these others causes the special legal problems associated with occupational injuries and illnesses.

Environmentally induced illnesses do not cause as wide a range of outside forces to intrude on the physician–patient relationship. Nonetheless, treating physicians may play a critical role in determining how their patients fare in the legal system. Physicians are often uncomfortable with this responsibility, for which they have not been prepared.

This chapter provides an understanding of the legal and institutional environment in which people with occupational or environmental health conditions are treated. It describes the

ways in which actions by treating physicians can affect their patients' legal rights to financial recovery and how physicians may interact directly with the legal system. The chapter is divided into six parts: *(a)* workers' compensation insurance; *(b)* Social Security programs for the disabled; *(c)* international perspectives on workers' compensation; *(d)* the role of personal injury litigation in providing access to compensation for people with occupational and environmental health conditions; *(e)* laws that protect workers' job security; and *(f)* privacy rights of workers and potential conflicts with the information needs of the employers of injured workers.

These patients' economic and employment status may depend on whether physicians are willing to provide documentation regarding the occupational causation or the degree of impairment resulting from the health problem. The patient may need assistance from the physician to be excused from work and to return to work—and to obtain workers' compensation or disability benefits and medical insurance coverage during the course of treatment. Patients' trust in their physicians—and, therefore, their degree of compliance with medical instructions—will be influenced by the physicians' understanding of the pertinent legal issues and their willingness to provide assistance to the patient. Ignorance of the patients' situations and the legal rules surrounding them, may lead to serious adverse consequences for patients, including discharge from employment.

Physicians' primary ethical and legal obligations are to their patients. The economic interests of other people and entities, including those of the patients' employers, are well protected by the legal system. Physicians who treat individuals with occupationally induced health problems and are concerned about the long-term health of their patients must become familiar with the various legal rules that govern requests for *(a)* information that will be made by others and *(b)* assistance that will come from their patients.

WORKERS' COMPENSATION

Leslie I. Boden

Workers' compensation is a legal system that shifts some of the costs of occupational injury and illness from workers to employers. Workers' compensation laws generally require employers or their insurance companies to reimburse part of injured workers' lost wages and all of their medical and rehabilitation expenses.

Historical Background

Before passage of the first workers' compensation act in 1911, workers generally bore the costs of their work-related injuries. Injured workers and their families were forced to cope with lost wages and medical care and rehabilitation costs. Under the common law,* workers had to prove in a court of law that their injuries were caused by employer negligence to recover these costs.

For several reasons, it was extremely difficult for workers to win such negligence suits. Injured workers had the burden of proof and had to show that their employers were negligent, that their injuries were work-related, and that negligence caused these injuries. To sustain this burden of proof, workers had to hire lawyers and often had to rely on the testimony of fellow workers (who, along with suing workers, might be fired for their part in suits). All of this was enough to deter most workers from bringing suit. In addition, employers had very strong common-law defenses that usually protected them from losing negligence suits when they were brought. In the late nineteenth century, these defenses were widely used, and less than one-third of all employees who brought such negligence suits won any award.

The inability to hold employers responsible for their negligent actions persisted in the face of the high and increasing toll of occupational death and disability at the beginning of the twentieth century. After a disabling injury, workers and their families were left largely to their own resources and to assistance from relatives, friends, and charities.

By 1910, some efforts had been made to provide better means of compensation to injured workers and their families. Some of the larger corporations had established private compensation schemes, and several states and the federal

* The common law is a body of legal principles developed by judicial decisions rather than by legislation. Statutory law can override these judge-made laws.

government had enacted employers' liability acts. These laws retained the basic common law liability scheme, but reduced the role of the common-law defenses.

Most injured workers, however, were not able to take advantage of these changes. There was growing support for a major change in the law from the social reformers of the Progressive Era and from major corporations. These pressures gave rise to the passage of the first workers' compensation law in New York State. Many other states rapidly followed suit, and by 1920 all but eight states had passed similar laws, although most did not cover occupational *disease*. Mississippi, in 1948, was the last state to establish a workers' compensation system.

Description of Workers' Compensation in the United States

Workers' compensation provides income benefits, medical payments, and rehabilitation payments to workers injured on the job as well as benefits to survivors of fatally injured workers. There are 50 state and three federal workers' compensation jurisdictions, each with its own statute and regulations.

While U.S. state and federal systems are different in numerous ways, they have several characteristics in common. Benefit formulas are prescribed by law. Generally, medical care and rehabilitation expenses are fully covered, but lost wages are only partially reimbursed. Employers are legally responsible for paying benefits to injured workers. Some large employers pay these benefits themselves, but most pay yearly premiums to insurers, which process all claims and pay compensation to injured workers. Workers' compensation is a no-fault system. Injured workers do not need to prove that their injuries were caused by employer negligence. In fact, employers are generally required to pay benefits even if the injury is entirely the worker's fault.†

The change to a no-fault system was established to minimize litigation. For a worker to qualify for workers' compensation benefits, only three conditions must be met: *(a)* there must be an injury or illness, *(b)* it must "arise out of and in the course of employment," and *(c)* there must be medical costs, rehabilitation costs, lost wages, impairment, or disfigurement.

Clearly, these conditions are much easier for the injured worker to demonstrate than employer negligence. For example, if a worker falls at work and breaks a leg, all three conditions are easily met. Unusual cases sometimes arise in which the question of the relationship of an injury to employment is difficult to resolve, and there may be questions about when a worker is ready to return to work. Such issues may result in litigation, but they are the exception, not the rule. In most cases, a worker files a claim for compensation with the employer, and the claim is accepted and paid either directly by the employer or by workers' compensation insurance carrier of the employer.

The following case is typical of the events that follow many minor claims for workers' compensation:

A man developed a painful muscle strain while lifting a heavy object at work on Monday afternoon. He went to the plant nurse and described the injury. He was sent home and was unable to return to work until the following Friday morning. On Tuesday, the nurse sent an industrial accident report to the workers' compensation insurance carrier and a copy to the state workers' compensation agency. Three weeks after he returned to work, he received a check from the insurance company covering part of his lost wages—as mandated by state statute. The insurer also covered medical expenses related to the muscle strain.

Workers' compensation provides wider coverage than the common law system did. Generally, injuries and illnesses are considered eligible for compensation if occupational exposure is the sole cause of the disease, is one of several causes of the disease, was aggravated by or aggravates a nonoccupational exposure, or hastens the onset of disability (Table 31-1). Suppose, for example, a worker with preexisting chronic low back pain loses 2 weeks from work as a result of lifting a heavy object at work. In this case, the workers' preexisting condition might just as easily have been aggravated by carrying out the garbage at home, but the fact that the disabling even occurred at work is usually sufficient for compensation to be awarded.

† There are exceptions. As an example, it is not uncommon in the United States and in some other jurisdictions to limit or to deny benefits to workers who are injured as a result of their exposure to alcohol or illegal drugs.

Table 31-1. Likelihood of Compensation, by Source of Preexisting Condition and Source of Ultimate Disability

	SOURCE OF PREEXISTING CONDITION	
Source of Ultimate Disability	Work-Related	Non-Work-Related
Work-related	Compensable	Generally compensable
Non-work-related	Generally compensable	Not compensable

Source: Adapted from Barth PS, Hunt HA. Workers' compensation and work-related illnesses. Cambridge, MA: MIT Press, 1980.

Cases in which an occupational injury or illness becomes disabling as a result of non-work exposures are similar in principle. For example, a worker with nondisabling silicosis may leave a granite quarry job for warehouse work. Without further exposure, the silicosis will probably never become disabling. However, the worker may begin to smoke cigarettes and lose lung function until partial disability results. In most states, this worker should receive compensation from the owner of the granite quarry if the work relationship can be demonstrated.

During the past two decades, several states have passed laws that undermined the long-standing principle of workers' compensation that workers are eligible for benefits even if their disabilities are in part caused by nonwork factors. These laws require that work be a major or predominant cause of the disability or eliminate compensation for the aggravation of a preexisting condition or for a condition related to the aging process.[1]

Some states, including California and Florida, allow disability to be apportioned between occupational and nonoccupational causes. While at first this may seem like a sensible approach, apportionment creates some difficult decisions. Many disabilities are not additively caused by two separable exposures. With silica exposure or cigarette exposure alone, the worker in the above example would probably not have become disabled. Often, as in the case of lung cancer caused by asbestos exposure and smoking, the contribution to disability or death of two factors is many times greater than that of one alone. Such issues make the apportionment of disability very difficult, if not impossible. Nevertheless, in these jurisdictions physicians are often asked to provide an opinion on apportionment. Failure to do so may result in loss of benefits for the disabled worker.

When workers' compensation was introduced, workers gained a swifter, more certain, and less litigious system than existed before. In return, however, covered workers lost their right to sue employers through common law. (See Box 31-1 for situations in which workers with occupational injuries and illnesses can sue.) Awards were also lower than those given by juries in negligence suits: Workers' compensation provides no payments for "pain and suffering" as there might be in a common law settlement. In addition, disability payments under workers' compensation are often much less than lost income, especially for more severe injuries.[2]

The United States does not have a unified workers' compensation law. Each state has its own system with its own standards and idiosyncrasies. In addition, federal systems cover federal employees, longshoremen and harbor workers, and workers employed in the District of Columbia. Except for Texas, all states require employers either to purchase insurance or to demonstrate that they are able to pay any claims that might be made by their employees. In most states, private insurers underwrite workers' compensation insurance paid for by premiums from individual employers. In some states, a nonprofit state workers' compensation fund has been established; the state government therefore acts as an insurance carrier, collecting premiums and disbursing benefits. State funds disburse a higher percentage of premiums in the form of benefits than do private insurance carriers.

Types of Benefits

Most workers' compensation cases involve only medical payments because the time lost from work does not exceed the jurisdiction's waiting period (which varies among states from 3 to 7 days). Once lost work time exceeds the waiting period, the worker is eligible for temporary disability benefits—temporary total disability for each day off work and temporary partial

Box 31-1. When Employers Are Subject to Lawsuits for Workplace Injuries and Illnesses

Generally speaking, workers are barred from suing their employers for injuries covered by workers' compensation laws. However, in most states, injured workers can sue their employer for the following reasons:

- If the employer has not properly purchased workers' compensation insurance coverage.
- If the particular injury or illness is not compensable under the state's workers' compensation law. For example, if a state specifically excludes coverage for a specific occupational diagnosis, such as stress-related mental illness or cumulative trauma disorders, the employer is not shielded from lawsuit. However, the no-fault principles of workers' compensation do not apply. To be successful, the worker must prove that the employer was negligent.
- If the claim is related to a specific employment law. Until relatively recently, employers of occupationally injured employees could pay workers' compensation benefits and discharge the workers, without adverse legal consequences. This practice is often no longer legal. Actions brought by employees alleging violation of various state and federal employment laws are not affected by workers' compensation rules generally, including situations in which workers allege mental injury as a result of illegal discrimination or retaliation.
- In addition, when an injury is the result of intentional—not merely negligent—conduct by the employer, some states allow the worker to bring a common-law action against the employer. Most states still preclude these actions or require the worker to prove that the employer specifically intended to injure the worker—the "specific intent" standard. Under this standard, even employers who intentionally violate health and safety rules or are reckless in their approach to occupational safety are protected by workers' compensation immunity. In contrast, a growing minority of states now allow workers to bring common-law actions against an employer when the employer knowingly allows hazardous conditions to exist that are "substantially certain" to result in serious injury. In these states, an employer's knowledge that conditions were extremely hazardous is relevant to determining whether the employer will be held liable for costs of damages beyond those provided by workers' compensation.

disability benefits when job reassignment or reduced hours result in a reduction in pay. Sometimes, a physician will be called on to fill out a form that indicates whether the worker is ready to return to work and, if so, whether any restrictions (like lifting weight limits) should apply.

When an injured worker has recovered maximally from the injury, the physician may also be asked whether the worker has any remaining (permanent) impairment. If so, the worker may be entitled to permanent disability benefits. Rarely, the injury or illness leaves the worker permanently unable to be gainfully employed, and the worker is eligible for permanent total disability benefits. More commonly, permanent partial disability benefits would be paid. This is the benefit type that varies most from state to state. Some states pay "wage loss" benefits, calculating benefits based on the difference between current and pre-injury earnings. Most states pay benefits based on either impairment or loss of earning capacity. Even within a state, the benefit may be based on one or the other, depending on the circumstances of the case and the part of the body that was injured. Typically, this "bifurcated" method pays impairment benefits when the worker returns to work at or near the pre-injury wage. If this successful return to work is not achieved, then higher loss of earning capacity benefits are paid. Whether benefits are based on impairment or on loss of earning capacity, a physician is typically called upon to render an opinion about the patient's permanent impairment.

The Role of the Physician in Workers' Compensation

Workers' compensation is basically a legal system, not a medical system. The decision points for claims in this complex system are shown in Figure 31-1, if a claim is rejected by the workers' compensation carrier or self-insured employer, it will generally be necessary for the injured worker to hire a lawyer. The worker's lawyer may then bargain with the lawyers for the insurance carrier in an attempt to settle the dispute informally. If this bargaining does not result in agreement, the claim must either be dropped or taken before an administrative board—a quasi-judicial body established by state statute—for a hearing. To the worker or to a physician who may be called to testify in such a hearing, such a proceeding may be indistinguishable from a formal trial: Testimony is recorded, witnesses are sworn, and they are cross-examined.

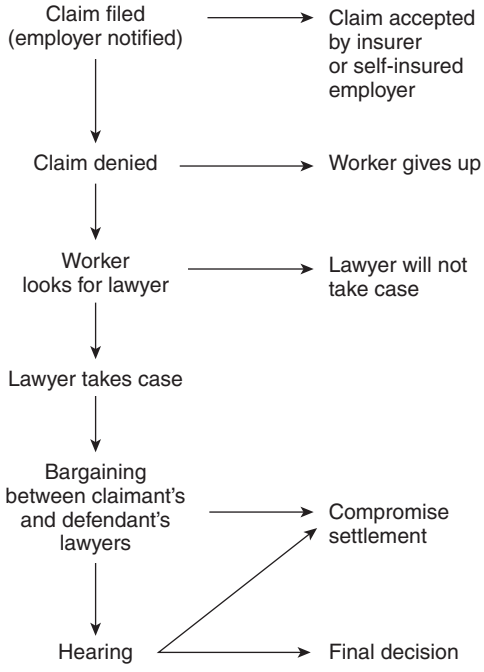

Figure 31-1. Decision points for workers' compensation claims.

As a part of this legal proceeding, medical questions are often raised. There may be disagreement about the degree of disability of a worker, when an injured worker is ready to return to work, or whether a particular injury or illness is work-related. To settle these disputes, physicians may be called on to give their medical opinions about employees' impairment or disability. Most often, physicians provide written opinions, but sometimes they may be called upon to testify. Many physicians do not like to testify, and most are not prepared by their training or experience to assume this role. Their expertise may be challenged; moreover, they may be confused by the different meanings of legal and medical terminology.

In workers' compensation, decisions are based on legal definitions, and the legal distinction between disability and impairment is often unclear to physicians. A physician called in to testify about whether a worker is permanently and totally disabled may understand total disability as a state of physical helplessness and may therefore testify that the injured worker is not totally helpless. However, this standard is not what a workers' compensation board would apply. The term *disability*, as used in workers' compensation proceedings, means that wages have been lost, while *total disability* means that the injured worker loses wages as a result of not being able to perform gainful employment. A relatively small impairment could result in a substantial disability. For example, airline pilots might be barred from working because of a level of visual impairment that might minimally interfere with other aspects of their lives. On the other hand, a worker who has been exposed to silica at work may have substantially reduced pulmonary function and therefore impairment. However, if the worker continues to work at the same job, no wages have been lost and therefore no disability payment is made. Many states, however, offer specified payments for disfigurement or losses of sight, hearing, or limbs, with compensation based on impairment and not on disability.

Although physicians may feel that they have not been trained adequately for their role in workers' compensation, workers do need their support in this area. A lack of assistance may mean unnecessary financial hardship for the victim of an occupational injury or disease.

The best way a physician can help identify a work-related disease or injury is by taking an occupational history. (See Chapter 2.) If the physician suspects a work-related disease or injury, the patient should be informed of the right to receive workers' compensation and the time limits on such claims. (The period for filing a claim generally begins when the patient is informed that the disease is work-related.) Physicians should also suggest the possibility of seeking legal counsel, and they can provide direct help by completing any required reports, including descriptions of the illness or injury and why it is believed to be work-related. The extent of probable disability may be noted. In most states, none of these steps requires testimony before a workers' compensation board; most workers' compensation claims are either paid without contest or settled without a hearing.

In cases involving permanent disability, a medical care provider is typically called on to render an opinion on the worker's impairment or disability. This is not a skill taught in medical school, and how the evaluation is done and written up generally depends on state-specific rules and customs. Providers who provide opinions

about permanent disability without specific training may undermine their patients' entitlement to these benefits. On the other hand, this is a skill that can be learned, and a primary care physician may be the only person willing and able to provide documentation for an employee wishing to file a workers' compensation claim.

Support for valid compensation claims not only assists injured workers but also helps to ensure that employers and their insurance carriers will appropriately shoulder the costs that result from workplace hazards. If these costs are not paid under workers' compensation, they will be borne by workers and their families or by all of us through our share of the costs of third-party medical payments, welfare, Social Security, and other public support programs.

The Adequacy of Workers' Compensation for Occupational Injuries

The fundamental problems of the common law scheme were that litigation was a necessary element of compensation and that it was very difficult for workers to win suits against their employers. Even when workers won negligence suits, payments were made long after they were injured, and one-third or more of each settlement was diverted for legal fees and expenses. Today, workers with minor injuries covered by workers' compensation generally can expect to receive payments promptly and without a contest. In fact, fewer than 10% of all claims for occupational injuries—as opposed to occupational diseases—are contested.

Under workers' compensation, insurance carriers or self-insured employers have the right to contest a claim. A claim may be contested, for example, because the injury is not considered work-related or the claim is for a larger amount than the insurer is willing to pay. However, in most injury cases, the employer or insurance carrier has little incentive to contest because proof of eligibility is easy and the potential gain to the insurer of postponing or eliminating small payments is not enough to offset the legal costs of pursuing a claim.

For expensive injury claims, such as permanent total disability and death claims, insurance companies are much more likely to contest. More than 80% of all workers' compensation claims for chronic occupational diseases,[3] and almost 50% of all injury claims for permanent disability or death, are contested, leading to delays of more than 1 year in settling workers' compensation claims. Even if they do not win, insurers can benefit by contesting a claim. They keep the settlement money temporarily, invest it, and receive investment income until the case is closed and the injured worker paid. Since contesting a claim delays the date of payment, this investment income is an incentive to contest even those cases that the insurer is very likely to lose. The higher the potential settlement, the stronger is the incentive to contest. Claims for permanent disability and death are contested five to ten times more frequently than are claims for temporary disability.

Long delays caused by contests of claims may put great financial pressure on injured workers, who may have high medical costs and substantial lost earnings. This can lead workers to accept settlements that leave them seriously undercompensated. In addition, legal fees, commonly 15% to 20% of cash benefits paid, must be paid by the injured worker.

Aside from the incentives to contest major claims, several other important problems can be cited in the more than 50 workers' compensation systems in the United States. In theory, workers' compensation should cover all employees; however, some states exempt agricultural employees, household workers, and/or state and municipal employees.

The maximum weekly benefit provided varies widely among the jurisdictions: The highest maximum state weekly benefit, on January 1, 2009, was for federal employees ($1,795) and the lowest was for Mississippi workers ($414). Many jurisdictions do not provide for cost-of-living adjustments; a person injured 30 years ago, receiving the maximum weekly benefit rate, and still disabled today may receive disability payments of only $150 to $200 a week. Benefits also do not account for the increased wages that would have been earned if the employee had continued to work.

Recent studies have raised substantial concerns about the adequacy of workers' compensation benefits. A substantial number of workers with occupational injuries may never enter the

Figure 31-2. Injured workers, especially those who experience long-term adverse impact of their injuries—such as this man who lost four fingers of his right hand at work, generally receive inadequate amounts of workers' compensation payments for lost income. (Photograph by Earl Dotter.)

workers' compensation system and, as a result, never receive benefits.[4–6] Even a smaller proportion of chronic occupational disorders are compensated.[7] Many workers receive workers' compensation income benefits that are less than one-half of their lost earnings, especially workers who experience long-term impacts of injuries (Fig. 31-2).[2] Workers with long-term disabilities who do not receive permanent disability benefits do the worst—often receiving benefits that are less than 20% of their injury-related losses.[8]

Workers' Compensation Medical Cost Control

Many states have put in place controls on medical costs, which can affect the level of payments and make it difficult to deliver optimal care. The most common of these controls are as follows:

- Fee schedules that list maximum reimbursement levels for health care services or products
- Limited employee initial choice of medical care provider or limitations on changing medical care providers
- Mandatory bill review for proper charges, generally tied to a fee schedule
- Mandatory utilization review of the necessity and appropriateness of admissions and procedures, length of hospitalization, and consultations by specialists before, during, or after hospitalization
- Managed care programs, such as from health maintenance or preferred provider organizations, that seek to reduce the price and utilization of medical care
- Treatment guidelines designed to reduce the provision of ineffective or harmful medical care

In the 1990s, many states adopted one or more of these methods of containing medical costs. From 1991 to 1997, 13 states added medical fee schedules, and 14 states added hospital payment regulation. In addition, six employee-choice states gave employers and insurers the right to provide managed care where none had done so before 1991. The rate of legislative action in this area slowed after 2000, but it did not stop. For example, California passed major new laws in 2002 and 2003 that reduced allowable medical and pharmaceutical payments, limited the number of visits to chiropractors and physical therapists, and required the use of utilization guidelines.

The importance of these changes goes beyond the impact on medical costs. For example, when they choose the medical provider or managed care organization, employers and insurers have a greater say about the medical treatment and the medical care provider's behavior in litigated cases. In many workers' compensation systems, a medical care provider who has treated a worker furnishes information on *(a)* when the worker is ready to return to work (and temporary disability benefits may be terminated) or *(b)* the worker's level of impairment (affecting permanent

disability benefits). Because choice of medical care provider can affect income benefits, it would be a contentious issue even if all parties agreed it did not affect medical costs.

Medico-Legal Roadblocks to Compensation for Occupational Diseases

The burden of proving that occupational injuries "arose out of and in the course of employment" is usually straightforward. However, workers with occupational illnesses face a different situation (Table 31-2). The workers' compensation system expects a physician to say whether a worker's illness was caused by or aggravated by work. Physicians are asked, "Was this illness caused by workplace conditions?" This is a question for which medical science often does not have a simple answer.

Many aspects of occupational diseases make disabled workers' burden of proof difficult to sustain. Physicians may not realize that their patients may have become ill as a result of workplace exposures. Many physicians are not able to identify occupational diseases because their medical training in this area has been inadequate—many have not even been trained in taking occupational histories. Furthermore, the signs and symptoms of most occupational diseases are not uniquely related to an occupational exposure. Medical and epidemiologic knowledge may be insufficient to distinguish clearly a disease of occupational origin from one of nonoccupational origin. For example, shortness of breath, an important symptom of occupational lung disease, is also associated with other chronic lung diseases (Fig. 31-3).

Table 31-2. Roadblocks to Compensation for Occupational Disease

Limitations of Medical Science	Statutory Limitations	Other Limitations
Difficulty of differential diagnosis	Time limits	Lack of exposure records (duration and intensity)
Lack of epidemiologic and toxicologic studies	Burden of proof	
Multiple causal pathways	Restrictive definitions of disease	
Limitations of physical training		

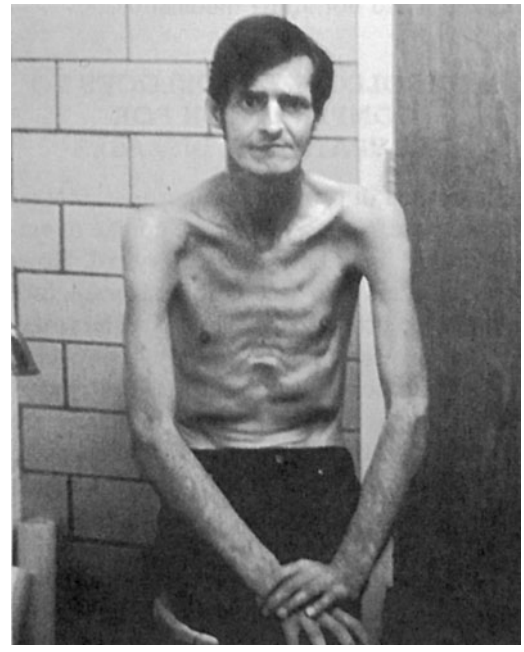

Figure 31-3. Although workers with silicosis, like this rock driller, qualify for workers' compensation, most workers with chronic occupational diseases often find their claims denied. (*Source*: Reprinted from Banks DE, Bauer MA, Castellan RM, Lapp NL. Silicosis in surface coal mine drillers. Thorax 1983; 30: 275.)

Another complicating factor is that a disease may have multiple causes, only one of which is occupational exposure. A worker who smokes and is exposed to ionizing radiation at work may develop lung cancer. Since both cigarette smoke and ionizing radiation are well-established risk factors for lung cancer, it may be impossible to say which of these two factors "caused" the disease. In many cases, occupational disease may develop many years after exposure began, and perhaps many years after exposure ceased. Consequently, memories of events and exposures may be unclear, and records of employment may not be available.

As a result of extended exposure to a hazard, chronic occupational injuries (cumulative trauma disorders), such as carpal tunnel syndrome, noise-induced hearing loss, and chronic

low back pain, may occur. As with chronic occupational diseases, it may be difficult to prove the work-relatedness of these injuries. In addition, records of exposure to occupational hazards are not often kept, so that even when a worker knows the type and duration of exposure, no written evidence of this can be presented.

These aspects of occupational disease mean that many victims do not even suspect that their disease is job-related. For those who do and wish to make a claim, the causal relationship between disease and workplace exposures may be very difficult to establish. These are major reasons why so few claims for compensation for occupational disease are filed. A study of occupational disease in California and Washington revealed that, of the 51 probable cases of occupational respiratory conditions, only one was reported as a workers' compensation claim.

When claims for chronic occupational disease are filed, many are contested by the insurance carrier or self-insured employer. Therefore, payments to disabled workers are delayed and uncertain. Workers with chronic occupational diseases wait much more than 1 year, on average, to receive compensation payments. In addition, administrative and legal costs absorb many of the resources devoted to compensating workers for their occupational diseases.

Establishing Work-Relatedness for Compensation

The burden of proving that disease is occupational in origin lies with workers. They must find physicians who are convinced that their illnesses are occupational in origin or that their illnesses were aggravated or hastened by occupational exposures. Physicians must then be able to convince referees who hear the cases that the diseases are indeed work-related.

The burden of proof might at first seem to be impossible for those diseases that are not uniquely occupational in origin. For example, lung cancer may be caused by smoking, ionizing radiation exposure, possibly by air pollution, or by all of these factors.

Suppose that a worker with lung cancer has smoked cigarettes, has had diagnostic X-rays, and has also been occupationally exposed to ionizing radiation in a uranium mine. Since occupational lung cancer does not have distinctive clinical features, an expert medical witness, using clinical judgment, cannot say that the disease is, without question, occupational in origin—although he or she may be able to say that, more likely than not, it is a cause of the lung cancer. In this case, the legal standard is that there must be a "preponderance of evidence" that the disease is occupational in origin, or the case is unlikely to be settled in favor of the disabled worker. A "preponderance of evidence" means that it is more likely than not (probability greater than 50%) that the illness in question was caused by, aggravated by, or hastened by workplace exposure.

Some workers' compensation laws have been written so that payment of a claim may be denied even though convincing evidence is presented that the illness was caused by or aggravated by the worker's employment. Some states require that a disease not be "an ordinary disease of life." In other words, diseases such as emphysema and hearing loss may not be compensable because they often occur among people with no occupational exposure. More than 20 states have a related requirement that diseases are only compensable if they are "peculiar to" or "characteristic of" a worker's occupation. In some states, compensation may be denied if it is determined that work is not a major or predominant cause of disability or that work aggravated a preexisting condition.

All jurisdictions have a statute of limitations (often 1 or 2 years) on claims for workers' compensation. A 2-year statute of limitations means that the worker must file the claim within 2 years of a given event. A time limit of 2 years after the worker has learned that a disease is work-related imposes no particular hardship on occupational disease victims. In some states, however, the time period begins when the disease becomes symptomatic, even if this takes place before the disease is diagnosed or determined to be work-related. The latter policy for starting the period for the statute of limitations may be a special problem if the worker's physician is not familiar with the occupational disease. The most burdensome statutes require that a claim be filed 1 or 2 years after exposure. Since chronic occupational

diseases commonly do not manifest themselves until 5, 10, 20, or more years after exposure, such rules effectively eliminate the possibility of compensation for workers with these illnesses.

The Problem of Compromise Settlements

A settlement reached outside the courtroom is called a compromise settlement because the amount paid to the injured worker generally is a compromise between the maximum and minimum amounts that the worker could receive in a court decision.

In the face of protracted litigation with uncertain results, a compromise settlement may seem very attractive to an injured worker who may have no wage income for a considerable period and may be facing large medical bills. The injured worker may therefore prefer a small settlement paid immediately to a much larger, but uncertain, settlement that would not be available for 1 or 2 years. Insurers may use their knowledge of the financial pressures on the injured worker to obtain a small settlement; they will thus contest, delaying the time when the case is closed in the hope of obtaining a small compromise settlement.

The compromise settlement will usually be paid in a lump sum to the injured worker, in lieu of future payments for lost earnings and medical and rehabilitation costs. Many compromise settlements also release the insurer from future liability: If the worker's condition should change at a later date or if future medical needs or increased costs were inadequately estimated, the insurer would not incur the costs of any increased disability or medical or rehabilitation expenses. The injured worker who has accepted a "compromise and release" settlement may later need additional medical care but not have the resources to pay for that care.

Recommendations of the National Commission

As part of the Occupational Safety and Health Act of 1970, Congress established the National Commission on State Workmen's Compensation Laws to "undertake a comprehensive study and evaluation of state workmen's compensation laws in order to determine if such laws provide an adequate, prompt, and equitable system of compensation." In 1972, the Commission released its report, which described many problems of workers' compensation and made recommendations for improving state workers' compensation systems. This report, still relevant today, included these seven "essential" recommendations:

1. Compulsory coverage: Employees should not lose coverage by agreeing to waive their rights to benefits.
2. No occupational or numerical exemptions to coverage: All workers, including agricultural and domestic workers, should be covered, and all employers, even if they have only one employee, should be covered.
3. Full coverage of work-related diseases: Arbitrary barriers to coverage, such as highly restrictive time limits, occupational disease schedules, and exclusion of "ordinary diseases of life," should be eliminated.
4. Full medical and physical rehabilitation services should be covered without arbitrary limits.
5. Employees should have choice of jurisdiction for filing interstate claims.
6. There should be adequate weekly cash benefits for temporary total disability, permanent total disability, and fatal cases.
7. There should be no arbitrary limits on duration or sum of benefits.

When this report was issued, some state government officials and some federal legislators threatened to establish federal minimum standards for state workers' compensation programs or to supersede them with a national program. In the 1970s and 1980s, many states changed their statutes to follow some or all of the recommendations of the National Commission. By increasing coverage and raising benefits, they substantially improved the value of workers' compensation to injured employees. However, this trend ended in the 1990s, and many states have moved backwards since then.[1] In addition, general changes in coverage have done little to discourage the litigation of occupational disease claims and costly occupational injury claims.

SOCIAL SECURITY DISABILITY INSURANCE AND SUPPLEMENTAL SECURITY INSURANCE

Leslie I. Boden

Social Security Disability Insurance (SSDI) is a federal insurance program: Workers contribute to it through a payroll tax and their future benefits are based on the amount of their contributions. Disabled workers may also apply for and receive Social Security Disability benefits during the same period in which they receive workers' compensation benefits.‡ To be eligible for benefits, workers must fulfill these requirements:

- Be under 65 years of age
- Have worked in covered Social Security employment for an adequate amount of time
- Currently have a qualifying disability, or, if workers have recovered, the period of disability must have lasted at least 12 months and ended less than 12 months before the time they applied for benefits

To establish eligibility for benefits, workers must obtain medical certification of disability. Particular weight is given to the opinion of treating physicians in the Social Security program, especially when their opinions are supported by medically acceptable diagnostic techniques and are not in conflict with other evidence. Primary care physicians who are knowledgeable about the process may therefore strongly influence outcomes for patients.

In the SSDI program, *disability* means that workers are unable to engage in substantial gainful activity as a result of severe, medically determinable physical or mental impairment that has lasted or can be expected to last for 1 year or that will result in death. Workers must be unable to do not only their previous jobs but also any other substantial gainful activity that is available in the national economy. Individuals with severe impairments who have only marginal education and have worked exclusively in jobs involving unskilled physical labor will almost always be considered disabled for the purposes of this program. A person who is currently employed is obviously not eligible for benefits. The extent of disability that is required for eligibility for SSDI benefits is considerably more severe, therefore, than the extent required for eligibility for temporary total or permanent partial disability benefits from workers' compensation programs.

Physicians may determine disability by relying on patients' histories and descriptions of symptoms, including pain. Ultimately, a designated state agency will make the eligibility determination of eligibility, including an evaluation as to whether patients' descriptions of symptoms are consistent with the medical signs, laboratory, and other diagnostic findings.

The Social Security Administration has documents on the Internet that describe the disability determination process and how disability evaluations are done. Several of these documents can be found at http://www.ssa.gov/disability/professionals/publications.htm. One publication, *Disability Evaluation under Social Security*, (also known as the *The Blue Book*), describes the disability evaluation process and provides information about the information needed in medical reports to establish the degree of disability. It also lists specific impairments, by organ system, that are considered severe enough to lead to a determination of total disability. Individuals lacking such impairments may, however, still be eligible for SSDI benefits because they have other totally disabling disabilities. Some people with less severe impairments may still qualify for SSDI benefits because their impairments, combined with their age, education, and labor-market experience, make them unable to engage in substantial gainful activity. For example, a physical impairment that might allow a physician to continue working might be much more disabling for a 60-year-old manual laborer with a fourth-grade education.

SSDI is considerably different from workers' compensation programs. Eligibility for SSDI benefits does not require that any impairment be caused by the patients' work. Eligibility may be based on the patients' total disability resulting from a combination of impairments; physicians therefore should document all health problems of patients, not just those that are job-related. This creates a record-keeping problem for those

‡ The Social Security laws provide that workers cannot receive more than 80% of their pre-injury earnings, including workers' compensation benefits.

patients who have filed simultaneous claims for workers' compensation and SSDI, since only the records related to occupational injuries and diseases should be made available in any litigation over workers' compensation eligibility.

Disabled people who are very poor may qualify for Supplemental Security Income (SSI) benefits. The determination of disability status is the same as for the SSDI program. However, eligibility is not based on payment of Social Security. Instead, SSI is a need-based program based on demonstration of both poverty and disability. Most workers who are severely disabled from occupational injury or disease will meet the requirements of the SSDI program and will not need to meet the need-based requirements of the SSI program.

SOME INTERNATIONAL PERSPECTIVES ON WORKERS' COMPENSATION

Peter S. Barth

In the United States, substantial variation exists, with each state operating under its own statute, practices, and history. Such heterogeneity also characterizes the laws of foreign jurisdictions and, hardly surprisingly, the variety of approaches found is even greater than the variability that exists within the United States. In this section we focus on six significant areas.

The No-Fault Principle and Suing Employers

Workers' compensation programs in the United States provide benefits without regard to fault and generally bar injured workers from suing the employer on grounds of negligence. In jurisdictions outside the United States, the no-fault approach to medical and indemnity benefits for work injuries and illnesses is the norm—that is, workers' compensation benefits are paid regardless of fault. However, lawsuits on grounds of negligence that supplement or replace workers' compensation are not uncommon. Workers' compensation had its origins in Germany and one reason for the adoption of the no-fault approach was to minimize the degree to which workers and employers would be led into disputes. At the time, there was a perceived need for less labor–management contention. The no-fault approach was one of several elements that were believed to promote this in the original statute. Prohibiting lawsuits against employers also has remained a central part of Germany's accident insurance program.

Following the English model, where common-law suits by injured workers are allowed, many of the state governments of Australia allow such suits, in addition to employees' rights to workers' compensation, although they may place significant limits on their use. One such barrier to such suits can be that the injury or disability must be "serious." For example, in the states of Victoria and Tasmania, common–law suits usually cannot be undertaken where the whole person impairment rating is less than 30%. In New South Wales, lawsuits can be undertaken where the impairment rating is 15% or higher. In South Australia and the Northern Territory, lawsuits are not allowed.

In Japan, injured workers can sue their employers. However, damages are not frequently sought for pain and suffering that are the result of a work injury due to employer negligence because awards for damages are small and workers' compensation benefits are considered the appropriate remedy for work injuries.

Several jurisdictions have found that once lawsuits of employers are allowed, it is difficult to eliminate the right to use them. Aside from continuing significant political wrangling in some of the Australian states over the access to this remedy, a good example of this exists in Argentina, where an important legislated reform of the workers' system in the mid-1990s eliminated their use. Fifteen years later, the legislature and the courts have not settled this issue decisively.

The Peoples' Republic of China is also operating in an unsettled area regarding the exclusivity of the workers' compensation remedy. While the statute may appear to bar lawsuits, some courts have allowed them. In addition, in some industrial sectors, including construction, employers are required to purchase privately sold, work-accident insurance for their employees in addition to workers' compensation. This insurance is sold through a government agency.

Insurance Arrangements

In the United States, essentially three types of insurance arrangements exist. In 46 of the 50 states, employers can purchase workers' compensation from private insurers who are licensed to sell such policies that are in accordance with state statutes. In less than half of the states, employers may purchase insurance from state insurance funds, four of which are the exclusive providers of this insurance. In some states where state insurance funds exist side by side with private carriers, these funds act largely as insurers of last resort, while other state funds compete vigorously with their private-sector counterparts. Aside from purchasing insurance from carriers, almost all states allow an employer to self-insure. In every instance, the "privilege" is extended only after an application by the employer is made and reviewed by the state or a body designated for this purpose by the state.

At least three additional types of insurance arrangements exist in other countries that are not found also in the United States. A notable, but somewhat rare, arrangement began in Germany in 1884 and continues to operate. Independent work-accident insurance "institutes" provide coverage to employers, according to the industrial sector in which they operate. These are nonprofit organizations with responsibilities spelled out in federal law. They are governed by boards consisting of employer and worker representatives. In addition to workers' compensation, indemnity benefits, and medical treatment, the institutes have significant roles in *(a)* workplace health and safety research and monitoring programs and *(b)* rehabilitation for injured workers.

An alternative system exists in several of the states in Australia, where employers purchase insurance from "agents" designated by the state to do so. These organizations sell policies, administer claims, and manage the health care and return-to-work issues faced by injured workers, much as insurers do in the United States. However, unlike insurers in the United States these agents do not underwrite the policies and bear no direct risk in offering their insurance. Instead, the state government agency, the Victoria WorkCover Authority (VWA), is responsible for workers' compensation. It sets rates, underwrites insurance, and determines which organizations can work as agents. The agents need not be, and usually are not, insurance carriers. They receive fees from the VWA, based on their performance according to criteria established by the VWA.

In many developing countries as well as Japan and a few other developed countries, the workers' compensation system is administered by the social security agency. This type of approach would be difficult to operate where workers' compensation laws are set and administered at subnational levels, as is the case in the United States, Canada, and Australia. By incorporating the administration of the workers' compensation program with other social insurance programs, this approach eliminates the challenge of having to integrate or coordinate public-sector health care, disability, and old-age and survivors' programs with workers' compensation benefits. This type of integration is most complex where these programs exist at the national level and workers' compensation is based at the state or provincial level.

Compensability or the Coverage of Injuries and Diseases

Important differences exist across states in the United States in the coverage of certain types of injuries and diseases under their workers' compensation programs. As a result, most generalizations about national practices are not useful. However, in comparing the practices in the United States to those in other countries or jurisdictions, three characteristics do appear to stand out, as described next.

The first relates to the compensation for occupational diseases. In most jurisdictions outside the United States, workers' compensation agencies have lists or schedules of covered occupational diseases. These schedules can be the result either of legislation that specifically enumerates diseases or categories of diseases or that enables administrators to do so. Such schedules may include occupations or industries from which exposures occurred and that may be linked to these diseases. When a worker has been diagnosed with a scheduled disease and a history of employment in a listed occupation or industry, the worker is very likely to be covered by

workers' compensation. Questions of proof for compensation are likely to be limited to the diagnosis and any employment history in the listed industry or occupation. Issues that correspond to statutes of limitation may also limit a claimant's ability to secure compensation. In many jurisdictions, these schedules serve as strong presumptions that favor a worker's claim. When a worker's disease is not listed on a schedule, this often serves as a negative presumption of work-relatedness and raises challenging standards of proof for the worker. In some jurisdictions, when a disease does not appear on a list the worker is effectively precluded from being granted compensation. In most developed countries using schedules, a "catch-all" category is indicated for diseases that have not been listed, giving applicants some opportunity for compensation. Nonetheless, the worker or the surviving dependent has a substantial burden of proof that does not occur for workers with scheduled diseases.

A major benefit of the use of schedules is that certain issues need not be litigated for every worker with a listed disease. For example, the etiology of certain types of cancer arising in employees of listed industries need not be disputed on a case-by-case basis. A major disadvantage of the use of schedules is that they are too slow to add diseases or industries that have become well established as related to one's work. Another major disadvantage exists in systems where compensation cannot be granted to claimants with diseases that are not on the schedule. Negative presumptions of work-relatedness also occur in some laws and regulations. For example, in Ontario's statute, "A worker and his or her survivors are not entitled to benefits under the insurance plan for impairment from silicosis unless the worker has been exposed to silica dust for at least two years in his or her employment in Ontario prior to becoming impaired." The use of schedules for occupational disease compensation has largely disappeared in the U.S. state workers' compensation systems, although two federal occupational disease activities, the Black Lung and the Energy Employees Occupational Illness Compensation programs, have explicit presumptions of work-relatedness.

A second difference that is notable between the U.S. state programs and those of some other jurisdictions around the world is the treatment of injuries that may not be the result of accidents. An important example occurs when a worker develops a disabling spinal condition that was not the result of a sudden, unexpected accident. This is an important issue since spinal injuries represent a very significant source of costs in the state workers' compensation programs. In the states, workers are not typically precluded from compensation simply because an injury was not the result of a traumatic incident. In some countries, such conditions are not considered to be injuries—they are regarded as diseases—and may not be compensable. In Germany, compensation is typically limited to claims for injuries resulting from physically damaging events that were sudden and externally affected the body. As a result, if the spinal condition is not considered to be an occupational disease, it is not likely to be compensated. In New South Wales, some spinal injuries are compensable as an "occupational overuse syndrome"—that is, a disease of the musculoskeletal system characterized by discomfort or persistent pain in muscles, tendons, and other soft tissue, with or without physical manifestations.[9]

A third difference regarding the U.S. state systems and most developed countries is the compensation for injuries or death that resulted from accidents while commuting to and from work. Most systems around the world cover such incidents so long as the incident occurred while simply going to or returning from employment, and not for side trips that were made before or after work. A few jurisdictions may have some very small difference in the compensation benefits for commuting accidents. For example, there is a 3-day waiting period before indemnity payments are paid for commuting injuries in Japan, but no such waiting period in the case of injuries occurring on the work premises. In either case, however, the injury will be compensated. In the U.S. state systems, by contrast, the insurer's liability essentially begins and ends at the workplace itself, which may or may not include an employer-provided parking facility. However, employees who are injured or killed while travelling in the course of their work and because of their employment are covered in the U.S. state system.

Health Insurance in the United States and Elsewhere

Workers with compensable injuries and diseases under state workers' compensation programs can expect to have the treatment costs of their conditions paid for by insurers. In some cases, these treatment costs may need to be continued for many years. However, if the injured workers are no longer employed by the same employers, they and their families may not have access to health insurance for conditions unrelated to work injuries. Health insurance in the United States is not a benefit that an employer must provide. Because most health insurance is obtained through the workplace, once workers cease to be employed, health insurance benefits are likely to be terminated. Workers in most cases have the right, under a federal continuation-of-benefits statute, to continue their insurance for a limited period through the employer's group insurance carrier.[10] However, the full cost of the insurance becomes the responsibility of the worker, extension of coverage is only temporary, and the option need not be available to workers of employers with fewer than 20 employees. Injured workers who do not return to work for the same employer face loss of health insurance for themselves and their families. And workers' compensation indemnity benefits do not include the value of the lost health insurance. This issue does not arise in the other developed countries because universal health insurance, independent of the employment relationship, is the norm.

National Differences in Public Disability Insurance

The meaning of "health insurance" in the United States and in other developed countries is also markedly different. In the United States, health insurance means coverage for costs arising from use of the health care system, such as for medical, hospital, and pharmaceutical charges. In most other developed countries, health insurance (or sickness insurance) covers not only these expenses but also income replacement during periods when people are unable to work due to a health condition. (There are five states in the United States that provide some income-replacement benefits for short-term disability for conditions that are not compensable under workers' compensation.) A consequence is that disabled workers need not view workers' compensation as the sole possible source of income support during periods of disability. Workers with a medical condition that prevents them from working need not face an all-or-nothing controversy over compensability for workers' compensation. Instead, income-replacement benefits under sickness or health insurance can approximate or equal benefits that they might have received under workers' compensation. The all-or-nothing choice faced by many U.S. workers under the state workers' compensation programs may account for some of the litigation in some state programs.

Public long-term disability programs covering both occupational and nonoccupational disabilities can also serve to supplement workers' compensation permanent disability benefits. In the United States, public disability programs (SSDI and SSI) only provide benefits to people with permanent and total disabilities, who are considered unable to engage in substantial paid employment for the rest of their lives. Thus, those with permanent conditions that are only partially disabling cannot expect to receive income support if their claims for workers' compensation permanent-disability benefits do not succeed. Some other countries, such as Denmark, Finland, and Ireland, also limit public permanent-disability benefits to totally disabled workers. However, other countries provide benefits to a broader group of disabled workers. For example, France and Italy provide benefits to workers who have lost at least two-thirds of their earning capacity. The threshold in Austria and Norway is 50%.[11] As a consequence, some partially disabled workers who do not receive workers' compensation benefits can rely not only on national health insurance to cover medical expenses but also on public disability benefits to cushion their losses of income.

Permanent Partial Disability Benefits

There is more heterogeneity among U.S. states in methods of providing permanent partial disability benefits than in any aspect of workers' compensation programs. Methods used in

jurisdictions outside the United States also show considerable variability. Some U.S. states use methods that are similar to those in other countries. An interesting approach is found in the new law in the People's Republic of China, where a unique incentive is provided to employers to return injured employees to work (Box 31-2).

Loss of earning capacity to determine permanent disability benefits is used both in the United States and in other countries. For example, Germany and Argentina both rate workers by loss of earning capacity. Unlike their U.S. state counterparts, both countries set a permanent partial disability rating for a period of time, typically 3 years, after which workers are rated again and their indemnity benefits either cease to be paid or are converted into pensions.

An interesting and instructive example of a variant on the bifurcated method can be drawn from British Columbia. Until 2002, it used a "dual system," in which workers with permanent disabilities were typically rated using two methods. Workers were usually rated on both permanent functional impairment (PFI) and loss of earnings (LOE). In 2002, a review found that LOE ratings (and pensions) were almost always found to be higher and that the PFI method rarely determined what the level of pensions would be.[12] As a result of this review, the LOE method is now used only in very limited circumstances, when the impairment method resulted in seriously inadequate benefits—reflecting the government's view that the impairment method provides reasonable and appropriate levels of benefits most of the time.

Yet another form of dual payment system for permanent disability exists in Victoria and other states in Australia, Canadian provinces, and elsewhere. Recognizing that injured workers with permanent impairments sustain both economic losses and noneconomic losses, Victoria pays workers' compensation for both types of losses. This approach overcomes the problem that is caused when a serious impairment does not cause any loss of earnings in a jurisdiction that compensates on the basis of the loss of earnings, but not for the degree of impairment (a "wage-loss-only" jurisdiction). This approach recognizes that the loss of a portion of a finger can end careers for some people, but provide little compensation in an "impairment-method" state. A dual-system approach to permanent partial disability compensation can be found in a few U.S. states, although the impairment benefit is limited both in the amount paid and type of bodily injuries covered.

Box 31-2. Permanent Disability Benefits in the People's Republic of China

In the late 1990s, China developed a pilot workers' compensation program, which has since been widely implemented in China. Its method of compensating permanent-disability cases contains some interesting features. A "working ability determination committee" evaluates the degree of impairment that is based on the "Assessment and Gradation of Disability Caused by Work-Related Injuries and Occupational Diseases." Where there is a permanent disability, the worker is rated between grades 1 to 10, with grade 1 being the most severe impairment. Grades 1 to 4 are impairments that likely would result in a determination of permanent total disability in many jurisdictions in the United States and elsewhere. A lump sum payment is made for permanent disability with a grade 1 impairment resulting in a benefit equivalent to 24 months of pre-injury salary; with a grade 10 impairment, the benefit is equivalent to 6 months of pre-injury salary. The lump sums are subject to a maximum and to a minimum benefit amount.

In addition to the lump sum benefit, workers rated grade 1 to grade 4 are entitled to a disability pension. A grade 1 impairment results in a monthly pension set at 90% of the worker's pre-injury wage; at grade 4, it is set at 75% of the worker's pre-injury wage. These benefits and the lump sums are paid by the regional workers' compensation insurance fund, a public entity. Benefits for workers with grade 5 or 6 are treated differently. If the worker has returned to employment and remains, there is no pension. If the employer has not found appropriate re-employment for the worker, the worker is entitled to a pension of 70% (grade 5) or 60% (grade 6) of the pre-injury wage, and the employer, not the insurance fund, is liable to pay these benefits. There is no pension benefit for individuals rated grades 7 to 10.

Clearly, the system in China is attempting to use this leverage to induce the employer to find employment for seriously injured individuals. Further evidence of this commitment is demonstrated by the rule that if the worker's employment contract is terminated following the disabling injury, workers with a grade 5 to 10 impairment must be paid a disability employment benefit and a work-injury medical care benefit by the employer (not by the insurance fund.) The size of these benefits is determined at the provincial or local government level.

ENVIRONMENTAL AND OCCUPATIONAL TOXIC TORT LITIGATION

Neil T. Leifer and David C. Strouss

During the twentieth century, there was a revolution in mass production. Much of this involved the manufacture and sale of products containing hazardous materials with little or no investigation into the risks they posed to the health and safety of workers, consumers, or others who might be exposed to these products. Concurrent with this industrialization was the growth of the chemical industry and the innovation of synthetics. Lead was used in many products, including paint and gasoline. Asbestos was widely used. Many chemicals were incorporated into manufacturing processes and end products. As a result, there has been significant dispersion of toxic chemicals into workplaces, homes, and ambient environments.

The Development of Environmental and Toxic Tort Litigation

For most of the twentieth century, environmental litigation rarely occurred for personal injuries, mainly because general legal assumptions about injury and causation did not fit the circumstances of environmental and toxic injuries. In the 1960s, advances in medical knowledge about the effects of exposure to toxins changed judicial understanding of these kinds of injuries, allowing for the development of environmental and toxic tort litigation.

Before the 1960s, there had been several legal barriers to lawsuits for environmental and toxic tort injuries. First, under the common law, the courts generally required that there be a privity relationship (a direct connection) between the defendant (the party allegedly causing injury) and the injured party. Since injuries from environmental exposures rarely arose in such contexts, claims for these injuries were barred for lack of privity. In addition, the statute of limitations (the period of time in which an injured party must initiate a civil action), as it then existed, did not anticipate these kinds of injuries. It generally began at the time of exposure, which was then considered to be the time of injury. However, unlike traumatic injury, in which impact and injury occur simultaneously, exposures to environmental toxins often do not manifest injury until years after exposure. Thus, claims for injuries from environmental exposure were often time-barred long before the injury had become manifest. Finally, the concept of legal causation, as it then existed, did not allow for these kinds of claims. During this period, an injured party often was required to prove that the toxic exposure was the sole cause of the injury. However, unlike traumatic injury, in which an injury arose from a solitary event, such as a broken leg from an automobile crash, proof of injury from chemical or toxic exposure was not so obvious. Often, these environmental injuries resulted from prolonged exposure to a variety of products or other circumstances, any one of which could be sufficient to cause the injury, leaving a worker or bystander incapable of proving which actually caused the injury. Thus, claims for injuries from environmental exposure were uncompensated because defendants argued that injured parties could not prove that their products were, in fact, the cause of injuries, or alternatively could not prove how much of each injury was caused by that defendant.

Advances in medicine during this period, including a better understanding of the long-term and synergistic effects of exposure to environmental and occupational toxins, led to modifications and adaptations of these barriers to recovery. The most notable development occurred in the context of asbestos exposure and disease. Asbestos, a naturally occurring mineral that is incombustible, was widely used for many years in insulation and fireproofing materials in many workplaces. Asbestos is also a potent carcinogen with a long latency period between exposure and the onset of disease. The asbestos industry knew for decades of its hazardous properties, but chose not to warn the public, thus exposing millions of people. The seminal case confronting the environmental effects of asbestos and the illogical legal barriers to recovery was decided in Texas in 1973.[13] In the wake of this and other cases decided at about this time, the barriers of access to the courts largely fell and the standards for prosecution of such cases were established. Privity was abolished. The statute of limitations was reinterpreted to begin not at the date of exposure, but when the injured person

knew or should have known of the injury or disease. Finally, injured parties no longer had to prove that a toxic exposure was the sole cause of injury or disease; rather, they only had to show that the exposure was a substantial contributing cause of the injury or disease.

The Elements of Proof in Environmental and Toxic Tort Claims

The basis for liability in any environmental or toxic tort case depends on the circumstances of the case. In cases in which exposure occurs because of a product, the theories of liability known as "products liability" generally apply. In cases in which exposure occurs because of contamination of the environment, such as the contamination of groundwater, soil, or air, the theories of liability in environmental law generally apply.

Products Liability Claims

Environmental or toxic tort injuries litigated in the products liability setting usually arise when someone is exposed to and injured by a pollutant or hazardous substance that is contained in, or is part of, a manufactured product.

In an environmental or toxic tort claim for products liability, an injured person may proceed under a theory of negligence or under a theory of strict products liability. In the case of negligence, the injured person must prove the following:

- The manufacturer or seller of the product owed a duty to exercise reasonable care.
- The manufacturer breached that duty (failed to exercise reasonable care).
- The person was exposed to and injured by the product as a result of the manufacturer's or seller's failure to exercise reasonable care.
- The person has suffered damages as a result of that exposure and injury.

In the case of strict products liability, the first two elements of the negligence claim are essentially satisfied by proof that the product was "unreasonably dangerous." In other words, the focus of the strict products liability case is less on the manufacturer or seller's conduct and more on the condition of the product. However, the distinction becomes somewhat semantic because both require proof of what dangers inherent in a product, such as toxins, were known or knowable by the industry at the time of sale.

The role of physicians and scientists is essential in environmental and toxic tort cases. Often, these cases can only be proven by reference to the scientific literature and the testimony of treating physicians and expert witnesses. These cases are very different from traumatic injury cases in which cause and effect occur simultaneously and can be readily observed. Injury from environmental and toxic exposures is usually far more insidious and complicated. Causation in toxic cases is often less obvious because there frequently is a long latency period between start of exposure and manifestation of disease. In addition, with a few exceptions, most diseases from toxic exposure are not pathognomonic—the presence of the disease does not definitely point to cause. Thus, medical judgments must not only be made concerning the diagnosis but also concerning causation.

Effective proof in an environmental or toxic tort case, therefore, often requires participation of a variety of medical and scientific professionals, frequently including treating physicians, pathologists, toxicologists, specialists in occupational or environmental medicine, epidemiologists, and those in allied fields. The need for and role of these professionals is required because medical and scientific issues tend to permeate every aspect of these cases. For example, in a case of exposure to hazardous fumes in a work environment, the plaintiff may have to prove the following:

- The hazardous nature of the product was known or knowable at the time of exposure.
- The dose and duration of the plaintiff's exposure was sufficient to cause disease.
- The plaintiff has the type of disease associated with exposure to that substance.
- Even after consideration of other factors, exposure probably contributed to the development of the disease.
- The plaintiff has some impairment or loss as a result of the disease.

An occupational physician might be needed to establish general knowledge about the toxicity of the product. An industrial hygienist might be needed to measure or model exposure. An epidemiologist might be needed to testify about the causal association between a specific exposure and a specific disease. And the treating physician might be needed to apply this general knowledge to the specific medical history of the worker.

Environmental Injuries and Tort Claims

Environmental and toxic tort claims also arise in settings that do not involve products. They are typically circumstances in which a person or property has been damaged because of the presence of hazardous or toxic substances in the immediate environment. In the late 1960s, the effects of decades of industrial waste disposal practices began to emerge in the form of groundwater contamination. As residential growth crossed paths with industrialization, contamination of sources of water supplies became a major public heath issue. Ironically, resort to civil action in the courtrooms was triggered not by well-known dumping practices or reporting of contaminants by the Environmental Protection Agency (EPA), but often by unusual patterns or clustering of serious illness. The most well-known case of this type involved the alleged poisoning of children from tainted groundwater in Woburn, Massachusetts.

In 1982, 16 families from Woburn, Massachusetts, filed a lawsuit alleging that cases of leukemia and other illnesses resulted from contamination of drinking water wells. Plaintiffs further alleged that two major defendants disposed of toxic chemicals, including trichloroethylene (TCE) and tetrachloroethylene, that ultimately reached potable water supplies consumed by the families in the case. Their challenge was to present to lay jury members the scientific evidence of groundwater contamination and its adverse health effects in terms that the jury members could understand.

Perhaps the most important lesson from the Woburn case was the enormous difficulty and complexity in proving a groundwater contamination case. The families had to show that the two companies negligently dumped TCE and other toxic solvents that eventually entered groundwater, migrated to city wells, and caused leukemia in children. While the jury found that one of the companies had negligently contaminated the drinking water wells, it also found that the contamination occurred after some of the children developed leukemia. Before the trial continued to the question of medical causation, the parties settled with this company, motivated largely by the uncertainty of establishing causation in light of the jury's prior findings. The partial trial lasted months, cost the families millions of dollars, and involved the testimony of numerous witnesses.

The Woburn case is not unique. Similar lawsuits across the United States include *(a)* the contamination of Anniston, Alabama, where over 22,000 people brought suit for damages caused by 40 years of dumping polychlorinated biphenyls (PCBs) by a chemical manufacturer; and *(b)* widespread contamination of groundwater caused by methyl tert-butyl ether (MTBE) released from underground storage tanks in gas stations and elsewhere.

Medical and scientific experts, ranging from treating physicians to epidemiologists, play key roles in these cases, such as in assessing the concentration and duration of exposure. As the Woburn case demonstrated, these experts must also establish the pathways of such exposures. These experts often perform complex modeling and health assessment of the potentially affected community. Ultimately, experts render opinions, based on review of exposure, health effects, and other data.

Lawsuits against "Third Parties"

While workers' compensation systems bar workers from suing their employers at common law for negligently caused injuries, there is no such bar against suits against third parties. Not surprisingly, workers and their lawyers have sought substantial additional damages from a variety of third parties. Most commonly, people involved in automobile crashes at work have sued the drivers of the other vehicles. In addition, people have frequently sued manufacturers of equipment or substances that contributed or caused their injuries. For example, workers injured by faulty machinery at work have sued manufacturers of

the machinery. Similarly, workers whose lung disease or cancer is related to workplace exposure to asbestos have successfully sued asbestos manufacturers.

These lawsuits always require that the worker show that the third party was negligent—the mere fact of the injury is never enough. In the case of third-party manufacturers of equipment, this negligence is tied to the failure to warn the users of the product (including the injured worker and, in some cases, his or her employer) regarding the safe handling or use of the dangerous machinery or substances. Other third parties who have sometimes been found liable to workers for events surrounding workplace injuries range from workers' compensation insurance carriers (for "bad-faith" dealings regarding the handling of claims or for negligent safety inspections of the workplace) to the Occupational Safety and Health Administration (OSHA) (for failing to cite a flagrant workplace safety hazard). Employers have also sometimes been sued—with varying success—by workers who claimed that their employers were not wearing the "employer hat" when the injury occurred. These lawsuits have involved a wide variety of situations, including when employers made or substantially altered the equipment causing the injury or when employers have negligently provided medical services to employees.

Differences between Lawsuits and Workers' Compensation Claims

It is not surprising that workers attempt to pursue common law litigation instead of, or in addition to, workers' compensation claims. While workers' compensation covers medical care and vocational rehabilitation costs and a fraction of lost earnings, a successful lawsuit will provide full recovery of lost earnings, cover medical care and rehabilitation expenses, pay an additional amount for "pain and suffering," and may pay other benefits as well. A successful lawsuit can provide benefits that are many times those of a successful workers' compensation claim. Moreover, filing a workers' compensation claim does not preclude filing a lawsuit.

But the barriers to successful litigation of lawsuits are also high. Injured workers must prove in most cases that the defendant was negligent; the no-fault rules of workers' compensation do not apply. Lawsuits are tried in the regular civil courts, rather than in specialized workers' compensation administrative courts. Trials in the civil courts are conducted under much more stringent rules of evidence, and proceedings tend to be much longer and require much more work by attorneys. Unlike workers' compensation claims, these cases cannot be pursued without a lawyer.

Challenges to Expert Testimony in Environmental and Toxic Tort Cases

Physicians and research scientists are essential witnesses to show causality (that the chemical in question actually caused the disease) in environmental and toxic tort cases. For example, a patient who believes that contaminated groundwater caused his or her cancer must be able to prove, with medical and scientific evidence, that the contamination caused or significantly contributed to the cause of the cancer, even after accounting for genetics, lifestyle, and other factors. To prove causation, treating physicians and research scientists provide their expert opinions about the cause of the patient's disease.

Expert opinions of physicians and scientists often are decisive in these kinds of cases and, as such, the admissibility of those opinions is often strongly contested. Until 1993, judges relied on a simple standard to determine the admissibility of medical and scientific testimony: whether the evidence was relevant and whether the science itself, including methods, theories, and the scientist's training, was "generally accepted" by the scientific community.[14] The requirement that science be "generally accepted" had the effect of preventing new scientific methods or new facts about the causes of human disease from being used in the courtroom.

In 1993, the United States Supreme Court changed the manner in which judges decide whether scientific evidence and expert testimony can be used in court. In *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, two children with birth defects and their parents sued the manufacturer of the drug Bendectin, alleging that this antinausea medication used during pregnancy caused the children's birth defects. The scientific

literature indicated that the drug was safe for maternal use. The plaintiffs' experts, however, after examining in vitro and animal studies, and reanalyzing and recalculating the published data on Bendectin, found that Bendectin did, in fact, cause birth defects. The trial court rejected the plaintiffs' experts' opinions because they were not based on "generally accepted" scientific methods under the Frye standard. In *Daubert*, the U.S. Supreme Court replaced the "generally accepted" standard with a much more flexible analysis of whether medical opinions and scientific evidence are based on a reliable scientific methodology.

Under the *Daubert* approach, a physician's opinion may be admitted as evidence if it is based on a reliable scientific methodology. The determination takes into account *(a)* whether the subject matter of the science can be, and has been, tested; *(b)* whether the science has been subjected to peer review and publication; *(c)* whether there is a known or potential rate of error and the existence of standards controlling a technique's operation; and *(d)* whether the theory or technique is widely accepted in the field. These are factors for the judge to consider, but the standard is not meant to be used as a checklist. The rationale for such a standard is to allow the judge more flexibility to keep out unreliable science but also to allow introduction into evidence of new medical and scientific theories that may be reliable and well founded but not yet published or widely recognized.

Today in toxic tort cases, industry defendants attempt to discredit the plaintiff's scientific evidence before the trial by arguing that it does not meet the *Daubert* standard. This is known as a *Daubert* challenge. Typically, industry defendants challenge the medical and scientific evidence by arguing that the differential diagnosis or differential etiology method of expert witnesses was unreliable and cannot be proven, expert witnesses were not up to date on current literature, test or study designs were flawed, statistical analyses were inappropriate, and/or expert witnesses were not well qualified. The challenge takes place during pretrial motions, briefs, and hearings where the parties argue extensively about whether the expert testimony meets the *Daubert* criteria.

Physicians or research scientists whose opinions are challenged under *Daubert* must be able to explain to the court the scientific methods that they employed to reach their opinions. These opinions may be based on some type of instrument, test, examination, clinical observation, or other generally reliable method of analysis.

The trial judge's role in a *Daubert* challenge is not to determine whether the jury should believe the expert testimony, but rather to determine whether the expert testimony is reliable science that will assist the jury in deciding questions of fact at the trial. The judge's discretion as to which medical and scientific evidence is "good enough" for the jury has made challenges to the admissibility of scientific evidence much more common and has made it more difficult for injured people to have their "day in court." The judge's rejection of opinions of medical and scientific expert witnesses frequently ends the litigation, as it is virtually impossible for plaintiffs to proceed if the judge decides the necessary medical or scientific evidence cannot be used at trial.

JOB SECURITY FOR WORKERS

Emily A. Spieler

When workers become injured or sick, they confront three important challenges:

1. Those who cannot work need to find a source of income to replace the temporary or permanent loss of wages.
2. Injured workers need health care and assistance in paying for that care.
3. Injured workers need protection of their right to continue to work—the focus of this section.

The worker-patients' economic, physical, and psychological well-being is often dependent on their ability to continue working. Unfortunately, individuals with health impairments have difficulty both in keeping their old jobs and finding new ones. In general, most people without severe health impairments want to continue to work or, if their work was interrupted, to return to work. But an injured worker may or may not be physically capable of returning to the preinjury job, or the job may have been filled and no longer be available. If the old job is unavailable, he or

she may seek a different job with the same employer or a new job with a new employer. Social and private insurance benefits may be limited in both quantity and duration. Employers may resist hiring an applicant with a history of injury or impairment.

Health care providers play a key role in what is often a challenging problem.§ Employers, insurers, and workers look to health care providers to determine not only eligibility for benefits but also to provide opinions regarding the worker's ability to return to work and what work he or she can perform. Employers may informally rely on health care providers to assess the ability of an employee to perform a job.

In addition, a wide range of critical employment laws rely on physicians as gatekeepers, often in ways that may be contrary to the traditional physician–patient relationship. Under these laws, the physician is called upon to assess the capability of the individual to work and to understand the issues involved in certifying the individual for work. These decisions can be complex. For example, if a physician certifies an individual's ability to work, this may result in the termination of workers' compensation or other wage replacement benefits. If an individual will not be able to perform a job that is offered, he or she may be left without both benefits and wage income. And despite the laws that provide job protection for disabled workers, many working people are vulnerable to employer retaliation and discrimination.

Basic Regulation of Employment

Patients are often familiar with the practices of their employers. This knowledge and their own sense of job security will affect the ways in which they will act regarding issues that affect the physician–patient relationship, including if they will be willing to do the following:

- Raise concerns to an employer regarding hazards at work
- Tell an employer that they suffer from health impairments, regardless of whether the impairment is caused or exacerbated by work
- Choose to file for workers' compensation benefits
- Seek (or avoid) a return to work, either at a regular or light-duty job
- Authorize release of medical information to their employers (when the law does not require the release)

The employment-at-will doctrine is at the core of the U.S. legal code governing private-sector employment. Under this doctrine, employers may discharge or discipline an employee without articulating a reason—and an employee can quit without notice. This is most often described as the right of an employer to discharge someone for a good reason, a bad reason, or no reason at all. As a practical matter, this also means that an employer may change the specific elements of the bargain with a worker without notice: If the employee continues to work, he or she is generally deemed to have accepted the new terms.

In the pure application of the doctrine, as it was until the early twentieth century, an employer could legally discharge an employee because of any stigma or prejudice or because the employee was unproductive, too old, African American, an immigrant, a woman, disabled, or the employer disliked the employee. This is no longer true. The terms of this at-will "agreement" can be, and have been, modified by legislation, judicial decisions, individual and union contracts, and public-sector employment rules. Nevertheless, the at-will doctrine remains the default rule for private employment in almost every U.S. jurisdiction: If there is no specific applicable exception to the rule, then the default rule applies and the employee is legally unprotected. In contrast, other developed countries provide broader basic employment protection to most workers, requiring that termination from employment be based on a legitimate reason involving job performance.

In addition to this basic private sector employment rule, a patient's general legal rights at work are affected by answers to the following three questions:

1. *Does he or she work in the private or the public sector?* By and large, public-sector

§ Many of the laws governing worker health will allow for health care providers who are not physicians to give opinions and provide information.

employees (comprising about 22,532,000 workers in 2008—or 17% of the nonfarm workforce) are protected by civil-service and other rules that provide stronger legal protections. These protections, which generally provide a broader range of rights to employees than those available under the at-will doctrine, range from specific entitlements under insurance programs to general protections against discharge.

2. *Is his or her workplace unionized?* Less than 10% of private-sector workers are currently covered by a collective bargaining agreement or union contract. Workers covered by these agreements have two significant advantages. First, they can turn to the union to assist them in evaluating work hazards and alerting employers to their concerns. Second, these agreements almost always provide for protections that are unavailable to private-sector workers who are not covered by union contracts. Collective-bargaining agreements set wages and benefits (including health insurance), establish progressive disciplinary procedures, require employers to have "just cause" to discipline or terminate an employee, and establish rights to job allocation and retention based, at least in part, on seniority.
3. *Where does the patient live and work?* Basic employment protections and the availability and nature of jobs, which vary from state to state, affect attitudes of patients and employers.

Employees have differing views regarding their job security, which will also affect their behavior. They may know the past behavior of their employers toward other employees who have been in similar situations. They may trust—or distrust—their own employers, irrespective of the relevant legal rules. Employees may overstate their general level of legal protection at work.[15,16]

Workers may have difficulty enforcing their legal rights, and attempts to enforce their rights may put them in jeopardy due to inappropriate or illegal retaliation by employers. Legal rights are not self-enforcing. Enforcement of rights can be costly, time-consuming, and cumbersome: enforcement also generally requires medical and other forms of proof. Physicians should be aware that this complex set of legal rules and attitudes justifiably influences the behaviors of their patients.

Specific Legal Protections for Disabled Workers

Eligibility for job protection under an employer-specific disability program or under many disability-based laws will depend on the physician's assessment of whether the patient is capable of performing specific work. To provide useful information to workers, employers, and government agencies, physicians must understand the specific rules relevant to each wage-replacement program and disability-based law. Definitions of disability and the implications of determining disability or impairment vary greatly from one program to another. Physicians must also realize that a determination that an individual is not capable of performing a specific job or specific job functions may result in adverse employment consequences for that person.

Given the extraordinary power in the hands of physicians, more than one opinion will often be sought. For example, if the physician is the individual's treating physician, a determination regarding whether an individual is able to work may only provide the patient with a "foot in the door." Others will still have the opportunity to develop and submit alternative medical data. This adversarial process means, however, that physicians may be called upon to defend their conclusions; to do so, physicians must understand the legal rules associated with a given program. Failure to do so can result in medical reports and testimony that fail to accomplish what the physician intends.

Disability Discrimination

Discrimination and exclusion from the workforce due to disabilities is a continuing problem in the U.S. labor market. Disabled workers face barriers as a result of both their special needs and prejudice or stigma.

The federal Americans with Disabilities Act (ADA), which became effective in 1990, and similar state laws were specifically intended to

encourage employers to employ disabled workers and to provide enforceable employment rights to workers who were subjected to discrimination based on disability. Hailed as great civil-rights advances of the late twentieth century, these laws forbid discrimination by employers against individuals who have disabilities, are incorrectly perceived as being disabled, or have a record of disability.

To qualify for protection, people with disabilities must have a physical or mental impairment that substantially limits one or more major life activities or they must have a record of, or be regarded by the employer as having, such an impairment. Major life activities "include, but are not limited to, caring for oneself, performing manual tasks, seeing, hearing, eating, sleeping, walking, standing, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, communicating, and working.… [A] major life activity also includes the operation of a major bodily function, including but not limited to, functions of the immune system, normal cell growth, digestive, bowel, bladder, neurological, brain, respiratory, circulatory, endocrine, and reproductive functions."[17] If people claim that they are substantially limited in their ability to perform a major life activity of working, they must be unable to perform a broad range of jobs, not simply their own jobs.

The determination of whether an impairment substantially limits a major life activity is made without regard to ameliorative effects of mitigating measures, including medications, assistive technology and prosthetics, hearing aids, or oxygen therapy equipment and supplies. The only exception to this is that the ameliorative effects of ordinary eyeglasses or contact lenses are considered in determining whether an individual's sight is substantially impaired.

This definition, which was amended by Congress in 2008, is quite broad and easily encompasses many individuals with work-related injuries and illnesses. However, temporary or transitory health problems, such as strains and sprains, do not qualify as disabilities under the ADA.

Qualified individuals with disabilities cannot be excluded from work if they can perform the essential functions of a job, with or without reasonable accommodation. Essential job functions include only the fundamental duties of the job. Employers are expected to analyze jobs to determine which duties are essential. Because a job has historically included certain peripheral duties does not mean that the employer can insist that it continue to include those duties.

Disabled employees may not be able to perform these essential functions without some form of accommodation. The ADA requires that employers provide "reasonable accommodation" to qualified disabled employees. The definition of "reasonable accommodation" is quite broad:

> *(a)* making existing facilities used by employees readily accessible to and usable by individuals with disabilities; and *(b)* job restructuring, part–time or modified work schedules, reassignment to a vacant position, acquisition or modification of equipment or devices, appropriate adjustment or modifications of examinations, training materials or policies, the provision of qualified readers or interpreters, and other similar accommodations for individuals with disabilities.[18]

Reasonable accommodation may include modification to the work environment or to the manner in which a job is performed, elimination of nonessential tasks from a job, or transfer of the disabled workers to other vacant jobs that they can perform. The determination of the need for, and the scope of, reasonable accommodation was intended to be made through a flexible, interactive process that involves both the employer and the disabled worker. This process requires the individual assessment of the particular job and the specific limitations of the individual.

After the ADA became effective, disability-rights activists hoped that the courts would act to aggressively expand the rights of disabled employees. Instead, a series of court cases significantly narrowed the possibilities for legal protection for these workers. After an initial ruling that found HIV-positive individuals to be disabled within the meaning of the Act,[19] the courts dismissed these cases at a very high rate, finding that people could not prove that they were "disabled" in a legal sense.[20,21] Individuals with common occupationally caused disabilities, such as those due to musculoskeletal injuries or

respiratory disorders, faced particular resistance in the courts.[22¶]

These pre-2009 court cases created very difficult legal barriers for people with disabilities. They were required to demonstrate that they were sufficiently disabled to be entitled to protection, while still being able to do their jobs. Individuals who suffer from qualifying disabilities also had to demonstrate that they were able perform the "essential functions" of their jobs, with or without "reasonable accommodation." This has frequently been characterized as the "Catch 22"of disability law: If the people were viewed as sufficiently disabled to be members of the protected class, they were often viewed as too disabled to perform their jobs.

In response to the negative rulings in the federal courts, Congress amended the law in 2008, expanding the definition of disability, enumerating the major life activities that must be considered, and instructing the courts to interpret the statute "in favor of broad coverage of individuals." It is still too early to say whether the 2008 amendments will have significant effect on the restrictive interpretations of the original law. No court has, as yet, interpreted the new provisions.

Even if a person is a "qualified disabled person," there are two arguments on which an employer may rely to reject that person from employment. The 2008 amendments did not change these sections of the law. First, if the necessary accommodation would pose an "undue hardship" on the employer, the employer may refuse to accommodate or employ the worker. The interpretation of this provision generally focuses on economic considerations, and health care providers are, therefore, not called upon to provide information to assist in the determination.

Second, and more importantly from the standpoint of occupational health and safety, if workers pose a "direct threat" to their own health and safety or that of their co-workers, they may be excluded from employment. The term "direct threat" means "a significant risk to the health or safety of others that cannot be eliminated by reasonable accommodation."[17]

The determination of whether someone is a "direct threat" often requires input from physicians. The regulatory language is broad, stating that the assessment must be based upon individualized, objective evidence and involve a high probability of imminent and significant risk of substantial harm to the worker or to others. In determining whether to exclude a qualified individual from a job, the employer must consider the duration of the risk, the nature and severity of the potential harm, the likelihood the harm will occur, and the imminence of the potential harm. Future speculative risk, such as an underlying back condition that might worsen over a period of years, would not be sufficient to justify an individual's exclusion from a job.

Employers have successfully argued, however, that there is a tension between the legal obligation to continue the employment of employees who are disabled and the obligation to provide a safe workplace for all employees, as mandated by the Occupational Safety and Health Act (OSH Act; see Chapter 30). In *Chevron U.S.A. Inc. v. Echazabal*, 536 U.S. 73 (2002), a case involving an individual with hepatitis C who sought work in an oil refinery, the U.S. Supreme Court relied on the provisions of the OSH Act to uphold the employer's exclusion of the worker. The Court noted:

> The text of OSHA [the OSH Act] itself says its point is "to assure so far as possible every working man and woman in the Nation safe and healthful working conditions," and Congress specifically obligated an employer to "furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm to his employees." Although there may be an open question whether an employer would actually be liable under OSHA [the OSH Act] for hiring an individual who knowingly consented to the particular dangers the job would pose to him…there is no denying that the employer would be asking for trouble: his decision to hire would put Congress's policy in the ADA, a disabled individual's right to operate on equal terms within the workplace, at loggerheads with the competing policy of OSHA [the OSH Act], to ensure the safety of "each" and "every" worker.

¶ Notably, state courts in several states rejected the federal interpretation of the ADA and interpreted state disability laws more favorably for disabled workers. This is one illustration of employment laws varying from state to state.

While this decision endorses the general principle that employers have an obligation to provide a safe and healthy working environment for each and every worker, it also emphasizes that public policy does not guarantee job security to individual workers with disabilities who are at risk at work. If a worker poses substantial safety risks to self or others, and the employer does not have an open job to which the disabled worker can be reassigned, the employer may terminate the employee.

The disability discrimination statutes initially appeared to create very broad requirements for employers to accommodate the needs of disabled workers through job redesign or reassignment. Despite the historically narrow reading of the ADA by the courts, some disability advocates have maintained that the changing treatment of disabled employees may simply not be mirrored in the visible litigation of claims, but rather is lodged in changing norms that encourage employers to accommodate workers. Employment of people reporting disabilities that affect their ability to work, however, did not rise during the economic boom of the 1990s.[23] Disability discrimination has, in fact, proven to be a remarkably intractable form of exclusion of people from the labor market.

When these interventions are effective, however, the ability of disabled workers to remain in the workforce is substantially enhanced. Job accommodation has unquestionably been shown to successfully extend a worker's working life for a period of years.[24–26] To the extent that a physician can assist an employer and a worker to reach a reasonable accommodation of a patient's disability, the likelihood that the individual will continue a productive life is considerably enhanced.

Employee rights under the ADA are rooted in analyses of the employee's health impairments and functional capacity. Physicians therefore play a critical role in determining whether an employee may be entitled to legal protection. Employees and employers call on physicians to determine whether an individual has a qualifying disability, to assess an individual's functional abilities and limitations in relation to job functions, and to evaluate whether the individual's impairment poses a direct threat to health and safety.

Workers with disabilities may be required by their employers to provide medical documentation about their disability and their ability to work. In writing a report, the physician should be as specific as possible and should be attentive to both the patient's functional capacities and to the functional requirements of the job and the possible hazards. Employees' treating physicians will want to discuss the issues of future injury carefully with patients before supporting a patient's application to return to work. If a health care provider is contacted directly by the employer for information in the context of any of the laws discussed in this chapter, the provider should verify either that the law requires the disclosure or that the patient has given permission for medical information to be disclosed, to avoid violating rules on health care privacy.

The ADA explicitly forbids an employer from making employment decisions based upon medical problems that are unrelated to the applicant's ability to perform the job in question. Preplacement physical examinations can only be performed after the employer has made a conditional offer of employment. An employer can require "fitness-for-duty" medical examinations before returning employees to work after an injury, but these examinations must be limited to job-related inquiries. If, based upon medical examination, the person cannot perform the essential functions of the job, with or without reasonable accommodation, or if the person poses a "direct threat" to self or others, then the employer may refuse to hire or rehire that individual.

Job Security Other Than under the Disability Discrimination Laws

If employees are not "disabled" within the meaning of the disability discrimination laws, they may have remarkably little legally guaranteed job security if they have been injured on the job. On the other hand, informal arrangements can always be made, and physicians can be helpful by providing documentation at the request of employees (or, with appropriate consent, at the request of employers). Many employers and employees are able to work together to reach reasonable decisions regarding when, and if, those employees should be away from work and

when they should return. Some employers will voluntarily provide transfers to workers who would prefer to keep working.

These situations, however, can be complicated if there is not an agreement between employers and employees. In general, injured workers are not legally entitled to temporary or permanent job transfers to accommodate a partial or temporary disability.* This means that employees who prefer to continue to work may be forced to seek nonwage benefits.

If employees leave work, the return-to-work process can be difficult and confusing for them, their employers, and their physicians. Sometimes, workers want to return, but employers do not offer them a job. In other situations, employers offer jobs that workers and their physicians believe do not adequately accommodate the workers' continuing impairments. In general, workers' compensation laws make it illegal for employers to discharge injured workers in retaliation for filing for compensation benefits, but they do not provide broad protection against discharge for lengthy absences, even if the absence is the result of a compensation-related injury. Most states will require workers who are receiving temporary total disability workers' compensation benefits to return to work or lose their benefits if the employer offers them a "light-duty" job. The extent to which there is assurance that the job appropriately accommodates any continuing impairments varies. This situation can put workers and their physicians in difficult positions. A worker's refusal of an offered position may result in termination of workers' compensation benefits; a worker's acceptance of an offered position does not guarantee that any continuing impairments will be appropriately addressed through job accommodations.

There are a few state systems, as well as the federal employees' compensation system, in which people who receive workers' compensation benefits have a legal right to return to work after an absence for a compensable injury. In most of these systems, physicians must certify that workers are fit to return to work. Again, physicians and their patients are confronted with a dilemma. If a physician notes serious functional limitations, this may ensure that the employee will receive appropriate job accommodation. On the other hand, noting these limitations may lead to the opposite result: The lack of availability of "light-duty" jobs—or the refusal to provide one—may leave the patient without a job. If patients do not qualify for protection under disability discrimination laws, then they have no recourse in this type of situation. And even if they are a "qualified person with a disability," employers are not required to create "light-duty" jobs to meet their obligation to provide accommodation.

The goal, in general, is to ensure that disabled workers continue to have an income stream, either through nonwage benefits or appropriate accommodation at work. Because of the interplay between the various laws and systems, workers can be left without either source of income, through no fault of their own.

Leaves of Absence for Employees

Workers may need to be absent from work, sometimes for extended periods, due to health problems that are caused by or exacerbated by their work. The availability of temporary total-disability benefits from workers' compensation does not necessarily guarantee that workers will be welcomed back by the employer when they are ready to return. Larger employers may, however, have their own disability programs, including light duty as well as short- and long-term disability insurance. In situations in which these voluntary benefits are not available, employees have three clear, although limited, rights to time off from work:

1. Under disability discrimination laws, a leave of absence can be considered a reasonable

* There are exceptions to this statement. Under the ADA, qualified workers may in certain circumstances be entitled to job reassignment to a vacant position. Under the Mine Safety and Health Act, coal miners with medical evidence of pneumoconiosis may transfer to a job with guaranteed exposure to less than 1 mg/m^3 of coal dust. Since 1977, the Mine Safety and Health Administration has had broad rights (which have not been implemented) to establish other job transfer programs for miners exposed to toxic substances or harmful physical agents. Federal regulations governing specific hazards under the OSH Act require temporary, but not permanent, job reassignment. For example, the regulation governing occupational exposure to lead provides that workers with elevated blood lead levels may be entitled to a temporary transfer until their lead levels are reduced.

accommodation if a worker has a qualifying disability. This will depend on the specifics of an individual's situation. A person must have a qualifying disability and be able to perform the essential functions of the job; time away from work must not interfere with these principles. Not surprisingly, courts have held that someone who is chronically absent is not protected by the ADA because he or she cannot perform the essential functions of the job. For example, in *Brannon v. Luco Mop Co.*, 521 F.3d 843, (8th Cir. 2008), a case involving an injury at work, the court noted that "regular attendance at work is an essential function of employment." Many other courts have reached the same conclusion.

2. In a few states and in the federal compensation system, injured workers have a right to return to work after collecting workers' compensation benefits.
3. The Family and Medical Leave Act (FMLA) can provide guaranteed job security for workers who are temporarily disabled by occupational injuries. This protection is separate from any benefits a worker may receive through workers' compensation and is also distinct from the prohibitions against discrimination under the ADA. Instead of providing cash benefits or extending protection against discrimination solely to those with substantial impairments, the FMLA simply guarantees that eligible employees will be guaranteed the right to return to work after an absence of up to 12 weeks of continuous leave or an equivalent amount of time of intermittent leave.

FMLA coverage is limited to employers with more than 50 employees and to employees who have worked for a covered employer for a total of 12 months, including at least 1,250 hours of work during the past 12 months. An eligible worker is entitled to leave to care for a family member with a serious health condition or if the employee is suffering from a "serious health condition." If the employer and the employee are covered by the law, the employer is required, once the leave period is concluded, to reinstate the employee to the same or an equivalent job—that is, one in which the pay, benefits, and other terms and conditions of employment are equivalent. (There are some exceptions to this guarantee for high-level managers.) Employers are also required to maintain any preexisting health insurance coverage during the leave period.

The definition of serious health condition includes conditions that are caused or exacerbated by work. The definition is broad, encompassing any illness, injury, impairment, or physical or mental condition that involves inpatient care or continuing treatment by a health care provider. To qualify for FMLA leave, patients must either be unable to do part or all of the essential functions of their jobs for at least three consecutive full calendar days *or* the incapacity must be permanent or long-term and involve subsequent treatment. Thus, both acute and chronic conditions are covered. This definition is much broader than the definition of disability under the ADA.

To be eligible for a leave from work under the FMLA, employees must have medical certification from a physician that they are either unable to work at all or are unable to perform "any of the essential functions" of the position. Leave may be taken by the employee whenever "medically necessary." Employees need not, however, be so incapacitated that they are unable to work at all. An employee may also request intermittent leave, or a reduced work schedule, to accommodate planned medical treatment; the health care provider must then certify that this type of leave is medically necessary and the expected duration and schedule of the leave.

Employees cannot be required to accept light duty rather than take an FMLA-protected leave: Under the FMLA, it is the employee's right to be off work and, then, to return to work, assuming that the physician provides the necessary medical certifications. On the other hand, certification of a serious health condition for the FMLA does not lead to any entitlement to wages or benefits during the period of absence. These wages and benefits are paid only if there is a separate right to them, through workers' compensation, paid sick leave, or short- or long-term disability policies.

Failure of patients to obtain medical certification for leave may result in denial of leave and, in some situations, discharge. Medical certification

is required at three points: *(a)* in person and within 7 days of the initial incapacity to justify the initial leave; *(b)* recertification is necessary after 30 days; and *(c)* the employer may require a "fitness-for-duty" medical determination that certifies that the individual is capable of performing the essential functions of the job before allowing the individual to return to work. As under the ADA, this certification regarding ability to work should be limited to the ability of the patient to perform the tasks in question.

When Can Employees Fight Retaliatory Actions by Employers?

Not all adverse actions taken against employees are the result of employees' functional impairments or disabilities. Sometimes, workers assert that they have been retaliated against because they raised concerns about health and safety or other matters of significant concern. The procedure for challenging retaliation under the OSH Act (Chapter 30) is weak: Time limits are short, the procedure is cumbersome and controlled by the agency, and the remedy is quite limited. In keeping with the continuing viability of the employment-at-will doctrine, there is no general federal law that prohibits retaliatory discharge. In part in reaction to this vacuum, many state courts have developed remedies for individuals who assert that they have been discharged "against public policy." The exact meaning of this concept varies greatly among states. Some states have extended this concept to include situations in which workers have been discharged for activism concerning health and safety concerns.

A worker's valid legal claim against an employer's retaliatory actions does not, however, guarantee success or reinstatement to a job. It can be quite difficult to enforce these rights. Moreover, employers are rarely ordered to employ an individual, even after a lawsuit. The remedy most commonly provided in successful retaliatory discharge claims is monetary compensation, not a job. In general, this monetary compensation fails to pay for the full loss of income of the worker. In fact, reinstatement to jobs is rarely ordered in any kind of employment litigation, including successful claims under the ADA.

CONFIDENTIALITY AND PATIENT INFORMATION

Patricia A. Roche

In connection with all the issues described in this chapter, health care professionals must understand their obligations concerning confidentiality and privacy. Rules governing the privacy and confidentiality of medical information derive from a variety of federal and state laws. Health care providers who strive to understand their legal obligations regarding patient information are advised to consult people who know the applicable state laws, are familiar with federal laws that govern medical information, and understand the relationship between such state and federal laws. They should also recognize that such knowledge, although necessary for achieving compliance with the law, will not satisfactorily resolve all legal questions and ethical dilemmas that arise over information in clinical practice. For example, some laws may permit—-but not require—physicians to disclose some information to certain third parties without the patient's authorization, thus leaving it up to physicians to decide whether to disclose. Medical ethics, rather than the law, will guide such decisions. The following describes some laws applicable to circumstances where a third party requests information from a treating physician about a patient who is involved in a workers' compensation claim.

Duty of Confidentiality

The physician's duty to maintain the confidences of patients is well established as a legal obligation that derives from case law and state statutes. In general, the duty requires that the physician not disclose private information obtained from the patient to anyone who is not directly involved in the patient's treatment without first obtaining the patient's authorization.[27] It does not matter whether the disclosure is made verbally or in writing, but for the duty to apply a physician–patient relationship must be established.† A physician may have a further statutory

† The law does not ordinarily view the relationship between an employee and a physician retained to conduct physical examinations on behalf of an employer as one of patient and physician.

obligation to obtain authorization in writing before disclosing information. And depending upon the specific medical information involved, the process for obtaining valid consent for disclosure as well as the form and content of the written authorization may be dictated by a state statute or regulation.

Case law and state statutes have also established exceptions to the rules requiring patient authorization. For example, all states have mandatory reporting statutes that not only permit, but require, physicians to report certain diseases or conditions to designated public authorities without patient authorization and typically provide immunity from breach-of-confidentiality lawsuits related to such reporting.[27] Depending upon the state, additional exceptions may either permit disclosures, such as to employers, without patient authorization when the patient presents a serious danger to self or others, or may even mandate such disclosures.

If a physician is presented with a subpoena or similar legal process to compel attendance at a judicial proceeding or production of medical records, consultation with a lawyer may be necessary to determine whether the subpoena has been validly issued, if grounds exist to ask the court to "quash" the subpoena, if any testimonial privilege applies to the physician and the information sought, and the legal consequences for failure to obey a valid subpoena.

Confidentiality, Federal Privacy Laws, and Workers' Compensation Claims

When workers' compensation is involved, how much information may be disclosed without patient consent is sometimes addressed in the applicable state statue or by relevant court decisions. Where it is not and the physician is unsure of what information may be disclosed without risking breach of confidentiality, the guiding principle should be to provide the minimal information necessary to achieve the purpose of the disclosure. Specifically, the physician should release only information directly relevant to the workers' compensation case, unless the patient has requested and authorized that additional information be released. The physician should, out of concern for the patient's best interests, consult with the patient before releasing data, even in states where a signed claim for workers'-compensation or occupational-disease benefits operates as an authorization for disclosure of information by a health care provider to the relevant insurer. The physician should recognize that not only is the patient's privacy at issue but also that disclosure may affect employment, access to workers' compensation benefits, or the outcome of a lawsuit.

Physicians hired by an insurer or employer to evaluate the worker's readiness to return to work, degree of disability, or other nontreatment issues are not considered *treating* physicians under the law. If the examining physician has findings outside the purpose of the evaluation, the physician should respect the privacy of the person being examined by disclosing only the required information.

This *minimum necessary* principle is incorporated into several privacy laws, including federal regulations authorized by the Health Insurance Portability and Accountability Act of 1996 (HIPAA) that govern how physicians, health plans, pharmacies, hospitals, and other "covered entities" can use and disclose patients' personal medical information. (Most practicing physicians fit the definition of a "covered entity" used in the regulations, and therefore, are governed by them.) Under these rules, covered entities are required to limit the amount of individually identifiable information that is disclosed without patient authorization. Commonly referred to as the HIPAA privacy rules, these regulations also explicitly address disclosures of health information regarding workers' compensation claims and permit disclosures without the individual's authorization to the extent necessary to comply with laws relating to programs providing benefits for work-related injuries or illnesses. In addition, these regulations permit physicians to rely on a public official's representation that the information requested is the minimum necessary for the intended purpose of the disclosure.

The HIPAA rules set a floor rather than a ceiling for protecting the privacy of medical information. Consequently, when other federal or state laws on medical information provide more protection than the HIPAA rules, the more protective federal or state law governs. For this reason, physicians must become familiar with and understand applicable state laws on

confidentiality and medical information. Most importantly, physicians' legal and ethical obligations to their patients require them to limit disclosure of patient information (other than to the patient) to the minimum necessary to comply with applicable laws and regulations.

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SECTION V

AN INTEGRATED APPROACH TO PREVENTION

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32

The Roles of Labor Unions

Robin Baker, Laura Stock, and Valeria Velazquez

Understanding labor unions is essential for anyone working in the field of occupational and environmental health. Some in this field work directly with a unionized workforce. Others work in government agencies where labor unions are important "stakeholders." Some work on social-justice or environmental campaigns that need labor support to succeed. Others perform research that requires labor participation. The labor movement will influence even those whose work never brings them in direct contact with a union because of labor's significant political and practical effects on occupational and environmental health.

Unions have been the most significant force in the United States advocating for safety protection and decent working conditions not only for their own members but for all American workers (Fig. 32-1). Throughout the twentieth century, unions lobbied in the political arena for health and safety protection, the minimum wage, and child labor laws. Without the labor movement, it is unlikely that the Occupational Safety and Health Administration (OSHA), jobs for occupational health and safety professionals, or an organized voice to effectively promote workers' rights would exist.

Unions have also played important roles in environmental health. In the 1960s, the labor movement supported environmental protection and the civil rights, women's rights, and anti-Vietnam War movements.[1] Later, conflict between some unions and environmental organizations led to a general perception of hostility between these groups. However, workers and environmentalists have increasingly collaborated through "blue-green alliances" that bring together the "blue-collar" working class and "green" environmentalists (Fig. 32-2). The recent push to transition to a "green economy" has heightened interest in ongoing collaboration.

AN INTRODUCTION TO LABOR UNIONS

The role of unions is to represent and pursue the collective interests of workers. These interests include wages, hours, benefits, and conditions of employment, including health and safety. Many unions also consider, as part of their mission, building a better society. Thus, they address issues that extend beyond the workplace, such as peace, justice, and quality of life.

Approximately 15 million Americans belong to a union, representing about 12% of the total U.S. labor force. The number of workers in unions has remained relatively stable, but the percentage of U.S. workers represented by unions has decreased substantially, from a peak of 33% in 1955. As the U.S. economy has changed dramatically, jobs have been lost in sectors that

Figure 32-1. UNITE protest against sweatshop working conditions. (Photograph by Earl Dotter.)

Figure 32-2. Coal workers visiting the site of a coal cleaning plant to learn more about the hazards of its emissions for environmental health. (Photograph by Earl Dotter.)

were highly organized (unionized) and replaced by jobs in sectors where union organizing has been more difficult. Service jobs are increasing as traditional manufacturing jobs are decreasing. Part-time and contract work are growing. The degree of unionization varies significantly from sector to sector, with the highest levels in government (42%) and education (39%) and the lowest in finance and insurance, agriculture, and private social/community services.[2]

The core work of labor is typically carried out at the level of the local union. Local unions ("locals") represent workers in a single workplace or group of workplaces. They may include members in a specific trade or in a range of occupations. Locals are often responsible for negotiating collective bargaining agreements (union contracts) with employers and enforcing these agreements through grievance procedures. Locals are designed to be democratic institutions, governed by officers who are elected by union members. Some members serve as shop stewards, who represent their co-workers and help enforce union contracts. Many locals also hire staff members to help enforce contracts (*union representatives and business agents*) or to expand membership (*organizers*). Locals typically belong to an *international union*, which is comprised of many locals of the same union in the United States and often other countries. (See Box 32-1.)

The American Federation of Labor-Congress of Industrial Organizations (AFL-CIO) and Change to Win are voluntary confederations, comprising a total of more than 63 international unions. The majority of U.S. union members belong to unions affiliated with the AFL-CIO or Change to Win. These confederations engage in political action, lobbying, education, and organizing on behalf of the labor movement as a whole. They bring different unions together, not only on the national level but also within states and local communities. (See Box 32-2 and the labor federations listed in the Appendix.)

BENEFITS OF UNIONIZATION

Workers belong to unions for a variety of reasons. Two important reasons are to have input about their working conditions and to have representation, ensuring that they will be treated fairly at the workplace. Unionized workers generally fare much better in terms of salary and benefits than their nonunionized counterparts. Overall, union workers in the United States earn approximately one-third more than nonunion workers. The wage difference is even greater for minorities and women. Union workers also are more likely than nonunion workers to receive health care, retirement, and short-term disability benefits (Fig. 32-3).[3]

About 60 million (53%) of workers in the United States would choose to have union representation if they could.[4] The U.S. labor movement has been trying to address this need by developing innovative strategies to bring unionization to workers in new and expanding sectors of the economy. In addition to providing professional representation and other services to existing members, unions are seeking to involve both organized and unorganized workers in actively finding solutions to the issues that affect them. Promoting active worker involvement both attracts and retains members.

Many labor leaders have discovered that, in addition to protecting their members, health and safety advocacy can be a dynamic and powerful issue in building unions. Hundreds of thousands of workers go to work each day in pain. Workers navigate through their work shifts, anxious about unsafe working conditions. For many, health and safety is a matter of basic human dignity and respect. In a recent poll, 64% of workers who responded said that workplace safety and health was a leading factor in their decision to join a union; benefits was a leading factor for 60%, wages for 57%, and job security for 54%.[5]

New Approaches for a Changing Workforce

The U.S. workforce of the early twenty-first century is increasingly comprised of women, people of color, and recent immigrants. Workers in these groups, often lacking power and an organized voice, are typically channeled into the lowest paid, most dangerous, and highly stressful jobs.[6] Organized labor is seeking ways to reach out to these workers and to address their special concerns. Unions are taking the lead in

Box 32-1. Glossary of Key Labor Terms

Term	Definition
Affiliate	A union that is a member of a central labor body or federation of unions.
Agency shop	A provision in a contract that requires all nonunion members in a bargaining unit, as a condition of employment, to pay the union a fixed amount for services rendered, such as representation.
Bargaining unit	A group of employees recognized by the employer or designated by an authorized government agency for purposes of collective bargaining.
Building and Construction Trades Council	Unions that represent the construction trades are typically affiliated with the AFL-CIO's Building and Construction Trades Department. There are state and local councils (parallel to the CLCs).
Central Labor Council (CLC)	An organization made up of most local unions in a geographic area, often a county. CLC member locals represent many different trades and international unions. CLCs engage in political action, education, lobbying, and organizing on a local level.
Certification election (representation election)	An election, usually conducted by the National Labor Relations Board or a state board, in which employees in a bargaining unit vote for or against representation by a union. ("Card check" is another way that employees join unions—if a majority sign union cards and the employer recognizes the union, they do not have to go through the often drawn-out and difficult election process.)
Closed shop (union shop)	A provision in a contract that requires the employer to employ only union members.
Collective bargaining agreement/contract	A written agreement between a union and employer. Both parties make offers and counter-offers on the conditions of employment for the purpose of reaching agreement. The process is called collective bargaining and the resulting agreement, signed by both parties, is the collective bargaining agreement or "union contract." The contract addresses matters such as wages, hours, working conditions, and procedures for settling disputes. A contract usually must be ratified by a vote of the union membership.
District council	An organization made up of local unions in a geographical area that belong to the same international union. This council may coordinate bargaining with different employers in the area, or bargaining involving scattered locals having the same employer.
Executive board	The officers who run a local union, district council, or other labor body. On the local level, the executive board is normally elected by the membership. The board typically includes a president, vice president, secretary, treasurer, and trustees.
Grievance	A written complaint by employees or the union that the employer has violated the contract. Grievances are processed through the grievance procedure. The last step of the grievance procedure is usually arbitration, in which a neutral party makes a binding decision.
International union	A large national organization comprised of affiliated local unions in a given industry or in a certain kind of occupation. Most unions in the United States are called "internationals" because they may also represent workers in Canada.
Local union	The basic unit of union organization. A local union has its own bylaws and elected officials. Its jurisdiction may be just one workplace or hundreds, one occupation or many. Local unions may also be called "chapters" or "lodges." Most are affiliated with one of the large international unions.
National Labor Relations Board (NLRB)	An agency of the U.S. government that enforces the Wagner Act and Taft-Hartley Act, which are the basic federal labor relations laws. The Wagner Act is often called the National Labor Relations Act. The NLRB conducts most private-sector certification elections. It decides unfair labor practice charges, including safety-related discharges, failure to provide information, and other safety issues.
Organizer	Organizers may be rank-and-file members or paid union staff members. They work to expand membership in the union.
Steward	The first-line officer of a local union. Stewards are usually rank-and-file workers, elected by union members in a workplace. When workers have complaints or grievances, they usually go first to the steward. In most locals, there is a chief steward and the stewards may constitute a stewards' council.
Unfair labor practice	Action by either an employer or union that violates certain provisions of the labor relations laws, such as refusal to bargain in good faith or retaliation against a worker for union activity.
Union representative (business agent)	Many local unions and councils have their own paid staff members. These union representatives (business agents) perform day-to-day activities of the union, represent members, and help enforce the contract.

Source: Adapted from: Flagler JL. The labor movement in the United States. Minneapolis, MN: Lerner, 1990.

Box 32-2. Names of Major Unions

Unions are generally known by their initials. These can appear to be an overwhelming alphabet soup. However, nearly two-thirds of all U.S. union members belong to one of the ten largest international unions, listed below in order of size (as of 2007). For information about their mission and membership, see the following Web sites:

NEA—National Education Association. http://www.nea.org
SEIU—Service Employees International Union. http://www.seiu.org
AFSCME—American Federation of State, County, and Municipal Employees. http://www.afscme.org
IBT—International Brotherhood of Teamsters. http://www.teamster.org
UFCW—United Food and Commercial Workers. http://www.ufcw.org
AFT—American Federation of Teachers. http://www.aft.org
USW—United Steel, Paper and Forestry, Rubber, Manufacturing, Energy, Allied Industrial and Service Workers International Union. http://www.usw.org
IBEW—International Brotherhood of Electrical Workers. http://www.ibew.org
LIUNA—Laborers' International Union of North America. http://www.liuna.org
IAMAW—International Association of Machinists and Aerospace Workers. http://www.goiam.org

Figure 32-3. Protest by union members that was part of the movement that lead to the passage of the black lung legislation (Coal Mine Safety and Health Act) in 1969. (Photograph by Earl Dotter.)

advocating for equal access to health and safety training and OSHA services. For example, they argue that training programs, publications, and complaint processes should take into account workers' own languages and cultures. Unions have also pushed for new protections against hazards of particular concern to low-wage workers, such as workplace violence, ergonomic hazards, and pesticide exposure. Some unions are now establishing alliances with community organizations that serve immigrant and low-wage workers. For example, unions have joined forces with community groups in campaigns to demand a *living wage* rather than just a *minimum wage*.

Some unions have also helped launch alternative *worker centers* to reach out to underserved workers who are not yet unionized. For example, the Teamsters sponsor the Citizenship Project, a community-based immigrant and workers' rights center in California's Salinas Valley. It offers an array of immigration and naturalization services and other rights-related assistance to agricultural and other workers.

In recent years, some community groups and/or faith-based organizations have created their own worker centers independent of unions. Those centers sometimes take the initiative to address worker health and safety through advocacy, support, and education in the community. For example, the Workplace Project, a center in Long Island, New York, grew out of the struggles of Latino immigrants to respond to nonpayment or underpayment of wages, high rates of workplace injuries, job loss, and other workplace issues. The center has created worker committees for factory workers, day laborers, women working in child care and house cleaning, and maintenance workers. It also offers classes in labor history, health and safety, workers' compensation, organizing, and immigrant and

workers' rights. Recognizing the importance of these community-based centers, in 2006, the AFL-CIO entered into a partnership with the National Day Laborer Organizing Network (NDLON) to promote workers' rights and improve working conditions for all union and nonunion workers, regardless of their immigration status.

A UNION APPROACH TO OCCUPATIONAL HEALTH AND SAFETY

In 1911, a fire broke out at the Triangle Shirtwaist Company on New York's Lower East Side. About 150 employees, almost all of them young immigrant women and girls, perished when the fire swept through the upper floors of the loft building in which they worked. Many burned to death; others jumped and died. The safety exits on the burning floors had been securely locked, allegedly to prevent "loss of goods." The International Ladies Garment Workers Union (ILGWU), along with the rest of organized labor in New York City and around the country, led an outraged response to the tragedy. A state factory investigation committee was formed and paved the way for many long-needed reforms in industrial safety, fire prevention, and child labor protection.[7]

Since the time of the Triangle Shirtwaist Factory fire, U.S. unions have continued to promote worker health and safety through a combination of political action, collective bargaining, technical assistance, and worker education.

Political Action

Unions lobbied throughout the twentieth century for national health and safety standards. By the 1960s, increased interest in occupational safety and health was supported by two parallel political movements. The environmental movement began to question the long-term effects of chemicals on health. The civil rights movement made individuals more aware of their rights. These movements created a climate of reform, which encouraged unions and individual workers and others to advocate for new health and safety laws.

In 1968, labor leaders worked with President Lyndon Johnson's office to propose a government agency to develop and enforce comprehensive national workplace health and safety regulations. A mining disaster that year in Farmington, West Virginia, in which 78 miners died, gave impetus to this campaign. As a result of this tragedy and the advocacy of the United Mine Workers union, the Federal Coal Mine Health and Safety Act was passed in 1969. The Occupational Safety and Health Act (OSH Act) was passed in 1970, extending health and safety rights to nearly all U.S. workers. (The Act excludes those who are self employed, family members working on family farms, workers whose working conditions are covered by other federal agencies, and state and local employees, unless they are covered by their own state OSHA plans.[8]) (See Chapter 30.)

Unions advocate for laws and regulations (standards) that affect wages, hours, and working conditions, including health and safety. International unions have petitioned for OSHA regulations on critical issues, such as bloodborne pathogen exposure, ergonomic hazards, fall protection, and heat-related illness. Proposed OSHA standards are almost always intensely debated at public hearings, in the media, and elsewhere. When adopted, regulations are usually the direct result of concerted union advocacy that has been countered by rigorous opposition from business and industry organizations. Unions mobilize their members to lobby and provide testimony at hearings on proposed OSHA standards, generally giving voice to both organized and unorganized workers' concerns.

Collective Bargaining/Representation

Unions bargain directly with employers for comprehensive agreements aimed at improving working conditions, including health and safety (Box 32-3). This can include *(a)* health and safety committees or representatives, *(b)* the right to refuse unsafe work, *(c)* improvements in the workplace environment, *(d)* personal protective equipment, and *(e)* special safety grievance procedures. Some unions also negotiate for the right of access to employer facilities to

investigate hazards and for the right to accompany government or company personnel when they conduct safety inspections, surveys, and monitoring (Fig. 32-4). When health and safety clauses are included in the contract, unions have a tool for addressing workplace hazards immediately through existing means of contract enforcement, rather than depending on OSHA. The collective bargaining agreement also can address hazards not yet covered by OSHA standards, or fill in the gaps where current standards are inadequate.

Unions also have negotiated for a greater role in occupational health research. For example, the Hotel Employees and Restaurant Employees Union in San Francisco incorporated language about joint labor-management health studies into its contract with the Hotel Multi-Employer Group. The language of the contract allows either party to request a study to assess workstations, workloads, ergonomic issues, and other health and safety problems. The contract calls for joint design of such studies, shared costs, and mutual agreement on researchers and recommendations. Similarly, many of the most important studies of hazards in the auto industry have been conducted as a result of contract language negotiated by the United Auto Workers (UAW).

Effective enforcement of the union contract is just as important as contract language. Unions and their members need to monitor workplaces and to see whether health and safety agreements are being kept. Typically, a worker or union can file a grievance if a violation of the contract is perceived. By including in the union contract a "general duty clause" regarding health and safety in the workplace, a grievance can be filed about almost any unsafe practice or condition. Such a clause states that the employer has a duty to keep the workplace safe.

Besides contract bargaining and enforcement, unions represent members' health and safety concerns in a number of other ways. These can include filing complaints with OSHA or other agencies, organizing direct actions, and mobilizing public support through the media.

Education and Assistance

Unions conduct numerous training programs for their members. Many unions have developed

Box 32-3. Sample Health and Safety Contract Language

General Duty to Protect

The Company is committed to providing a safe and healthy work environment and encourages the active involvement and support of all employees. To achieve this end, the Company will do the following:

- Establish responsibilities of all levels of management and hold them accountable for implementing programs and procedures
- Ensure through proper support and training that all employees are aware of hazards and accept responsibility for working safely
- Ensure that all operations conduct business in compliance with applicable safety and health laws and regulations

Committees

The Parties shall maintain occupational safety and health committees at the national, regional, and establishment levels….Written minutes of each meeting will be maintained and distributed to each committee member and made available to employees upon request.

Research/Studies

Joint studies: At either party's request, a study may be initiated to review workstations, ergonomics of jobs, and other health and safety issues in a particular department. Such a study shall be jointly designed by the parties and conducted by a mutually agreeable expert(s) in the area of occupational health issues. The cost for such a study shall either be equally borne by the parties or funded by other sources, such as foundations or other grants. All implementation issues and recommendations resulting from said study shall be resolved only by mutual agreement of the parties.

Training

Procedures addressing violence in the workplace shall be published and distributed. The School District and the American Federation of Teachers shall be responsible for providing an education and training program. Clerical/technical unit employees shall attend the initial training and shall receive appropriate release time for this and subsequent training. The School District and the American Federation of Teachers shall develop procedures for training new employees.

Source: Adapted from: Collective bargaining for health and safety—a handbook for unions. Berkeley, CA: Labor Occupational Health Program, 2000.

Figure 32-4. The City Center Project in Las Vegas, where the Building Trades Union negotiated for access to investigate and address the high mortality rate among construction workers. (Photograph by Barry S. Levy.)

training manuals, videos, and fact sheets on a wide range of health and safety topics. Some have received funding from federal agencies, such as OSHA or the National Institute for Environmental Health Sciences (NIEHS), to train members about specific issues, such as noise, ionizing radiation, construction safety, ergonomics, hazardous waste, and health care hazards.

Worker training is required under many federal and state health and safety regulations. This is generally the employer's responsibility. However, some union contracts make clear what training the employer and the union must each provide, and how training must be provided. For example, several Canadian Auto Workers contracts specify that union members will deliver all education and training for employees. These members attend 2-week instructor training programs provided by the Canadian Workers Health and Safety Center, funded by the Workplace Safety and Insurance Board of Ontario, to prepare them to assume this training role.[9]

Unions also provide technical assistance to members facing hazards. Some international unions and larger regional bodies have health and safety departments with professional staff, such as industrial hygienists or nurses. Many local unions also work with committees on occupational safety and health (COSH groups)—local coalitions of union members, occupational health and safety professionals, lawyers, and students throughout the United States. They provide advice, training, support, and sometimes materials and equipment. University programs, government agencies, and occupational health clinics also provide technical support to unions and their members, as well as to unorganized workers. (See Box 32-4.)

Applying Union Principles to Health and Safety

Whether through political action, collective bargaining, other forms of representation, or educational programs, unions are guided in their health and safety activities by several basic principles that are the cornerstone of the labor approach to health and safety. The most

Box 32-4. Advocating for Healthy Jobs

Unions have recognized that a healthy job is more than one without hazards than cause injury or illness. It must support a full, productive, and healthy life. Americans spend nearly half of their waking hours at work. The work environment affects workers and their communities in profound ways, impacting stress levels, health care options, emotional well-being, and family life. Many unions push for policies and benefits that support healthy jobs in a broader sense. For example, the Labor Project for Working Families is a coalition of labor and community groups that had successfully lobbied for policies that guarantee paid family leave for all workers in California. Both the AFL-CIO and Change to Win Coalition have placed universal health care at the top of their agendas.

Healthy jobs are those that do the following:

1. Protect against workplace hazards
2. Provide living wages and benefits
3. Limit excessive stress and workload
4. Respect human dignity on the job and are free of harassment and discrimination
5. Offer job security
6. Support healthy lifestyles
7. Support family life
8. Support communities by being environmentally responsible

Source: Labor Occupational Health Program. Healthy jobs call to action. Berkeley: Labor Occupational Health Program, Center for Occupational and Environmental Health, University of California at Berkeley, 2009.

fundamental principle is a commitment to preserving, expanding, and defending workers' rights. These principles are listed in the sections that follow.

The Right to Protection

Unions advocate for safety programs that identify and effectively control hazards. They generally oppose programs that blame workers for injuries and illnesses. Instead of emphasizing unsafe acts, unions demand safe working conditions. They insist on recognition of the fundamental principle of the OSH Act: Employers are responsible for providing a safe and healthful workplace. This principle means that workers have a right to be protected from hazards.

Similarly, unions advocate for the most protective solutions to problems. These usually eliminate hazards at their source, rather than relying on personal protective equipment used by individual workers. For example, eliminating the use of toxic chemicals or installing effective ventilation is preferable, where possible, to relying on the individual use of respirators that are fallible and uncomfortable.

The right to protection also means that workplaces must institute effective injury and illness prevention programs, including such elements as the following: *(a)* systems to identify and control hazards in a timely manner; *(b)* training of all workers on potential hazards they face and skills they need to participate in health and safety activities; and *(c)* mechanisms that allow workers to report symptoms, injuries, and potential hazards without fear of reprisal. When prevention measures fail and workers are injured, unions advocate for timely medical care, compensation for any lost wages, and appropriate return-to-work policies.

The Right to Participate and Act

Unions advocate for the right of workers to participate fully in all aspects of workplace health and safety programs. They also support worker participation in the development and implementation of local, state, and national policies. Worker and union participation takes many forms, including the following: joint labor-management health and safety committees in which unions have equal membership, equal control of the agenda, and an equal leadership role; opportunities for input on new equipment and technology; and a role in setting research agendas and in reviewing and analyzing resulting data.[8] To ensure their ability to participate fully in health and safety activities, workers need an environment in which they can voice concerns and advocate for change without fear of reprisals. To this end, unions have pushed for strong anti-discrimination provisions and strict "whistleblower" protections in both laws and collective bargaining agreements. In addition, unions have fought for the right of workers to refuse hazardous work and have battled to protect and defend workers who exercise this right from discrimination.

The Right to Know

To participate on a full and equal footing with management, workers need access to all relevant

information about the hazards they face on the job. Unions successfully fought for an OSHA Hazard Communication ("right to know") standard that gives workers access to material safety data sheets (MSDSs) that provide information on specific chemicals, chemical labels, records of exposure and medical monitoring, logs of workplace injuries and illnesses, and training about various types of hazards.

Some unions have gone further to demand that workers have the right to understand the information they are given. This right to understand means that information and training must be presented in ways that are accessible to all workers, and that methods and materials must be adapted to take into account differences in language, literacy, culture, and technical expertise.[10] This right is particularly critical given the growing numbers of non-English-speaking workers in the United States, many of whom work in the most dangerous jobs.

A UNION APPROACH TO ENVIRONMENTAL HEALTH

In October 1948, an industrial suburb of Pittsburgh, Pennsylvania, was enveloped in a "killer smog"—the worst recorded industrial air pollution accident in U.S. history. Stagnant air conditions trapped emissions in the town of Donora from a local zinc works and a steel mill. Over a 5-day period, more than 20 people died and thousands more became ill due to sulfur dioxide, zinc, cadmium, and other contaminants in the thick smog. The United Steelworkers union, which represented workers from the mill, joined others in demanding that the two companies be held accountable for poisoning their members and others in the community.[11]

This incident marked the beginning of the U.S. labor movement's involvement in environmental advocacy, long before the rise of the popular environmental movement of the 1960s and 1970s.[1] Unions became involved in advocacy not only for clean air and water but also for conservation measures, such as preservation of wilderness areas for the enjoyment of the working population that had increasing leisure time. Despite its early adoption of environmental principles, labor has a mixed history in working for environmental health, as described in the next section.

Labor Pioneers in Environmental Health

By fighting for the elimination or reduction of hazardous chemicals in the workplace, unions contribute to the protection of the surrounding environment. Workers want a safe, clean environment not just in the workplace but also where they live, play, and send their children to school. As a result, unions have put their political clout behind some important battles for environmental protection. For example, in 1972—just 2 years after labor won the fight for the OSH Act, it helped win passage of the Clean Water Act amendments.

Walter Reuther, president of the UAW, was an early advocate for the environment. Under his leadership, the union created a Department of Conservation and Resource Development in 1967, predating the first Earth Day 3 years later. The UAW lobbied for legislation to control disposal of toxic waste, promote recycling, and establish a national environmental policy.

Tony Mazzocchi, a leader of the Oil, Chemical, and Atomic Workers (OCAW), like Walter Reuther, was a leading labor advocate for environmental protection. The OCAW argued for regulatory protection of the environment, even though its members were among the most vulnerable to job displacement due to new environmental restrictions. He led OCAW to take a visionary role in recognizing the potential catastrophe of leaving environmental pollution unchecked.

Another early leader in labor's advocacy for environmental health was Cesar Chavez of the United Farm Workers Union (UFW). The UFW led campaigns, beginning in the 1960s, to raise awareness of the hazards of pesticides used on food crops. It built a successful alliance to fight for pesticide protection for both consumers and workers (Fig. 32-5).

Jobs versus the Environment

As the post–World War II years of economic growth and security gave way to the economic

Figure 32-5. Cesar Chavez, president of the United Farm Workers Union, leading a campaign to protect farmworkers from the health hazards of pesticides. (Photograph by Earl Dotter.)

decline of the 1980s, tensions between organized labor and the environmental movement grew. Advocacy for environmental protection was increasingly perceived by labor as a threat to union jobs, which already were declining at an alarming rate. In some situations, union jobs were lost as older, environmentally damaging companies went out of business, with little or no concern for displaced workers. In other situations, however, the threat of plant closures was used by companies to enlist labor support in a fight against environmental control. Claims by businesses and industries that regulation would cost jobs were exaggerated or false.[12,13]

As a result of well-publicized conflicts, such as the "timber wars" in the Northwest between loggers and environmental activists, the labor movement developed a reputation for being anti-environmentalist. Similarly, many workers came to view the environmental movement as anti-labor and uncaring about workers. Public opinion generally held that labor would always side with business and industry against environmental protection and in favor of jobs. However, even in the 1980s, the labor movement was never monolithic concerning policies on environmental protection. Those sectors of the labor movement that felt directly threatened by job loss soon learned that, in any event, they were losing jobs to international competition. As a result, a renewed era of labor–environmental collaboration began to develop.

New Alliances

By the dawn of the twenty-first century, strife between the labor and environmental movements started to fade. One survey found that 64% of union leaders in the United States reported their relationships with environmentalists as "good" or "very good"; only 10% identified their relationships as "poor" or "very bad." The worst relations were found in regions, such as the Northwest, where the timber industry is a dominant employer. Elsewhere, labor leaders expressed deep commitment to common concerns with the environmental movement, including restricting use of toxic chemicals, protecting air and water quality, and incorporating environmental standards into international trade agreements.[14]

Important new alliances have been built on the premise of a "just transition" from polluting industries to environmentally friendly jobs.

Beginning in 1996, a coalition of environmental and economic-justice organizations and the OCAW came together to promote dialogue between communities of color affected by polluting industries and workers who rely on those industries for their jobs. This exchange of concerns led to the establishment of the Just Transition Alliance, which promotes policies in local communities to address conflicts between *(a)* maintaining and creating jobs, and *(b)* promoting a clean environment.

"Just transition" principles state that the costs of achieving sustainable development, a healthy economy, and a clean environment should not be borne by workers or by community residents. These principles call for compensation and other reparations, including re-education and training, for both workers and community residents impacted by polluting industries or by the closing of these industries.[15]

More "blue-green" alliances are taking shape, many of them joining in the call for "just transition." These alliances often call for placing equal emphasis on policies that will do the following:

- Protect health of workers
- Protect health of community residents
- Promote sustainable, environmentally friendly employment
- Preserve union representation, maintenance of living standards, workplace safety, and decent working conditions in newly created jobs that replace jobs in polluting industries

Perhaps the most publicized coalitions of labor unions and environmental organizations have been in the area of international trade agreements. Unions and environmental organizations have jointly advocated for the inclusion and enforcement of trade sanctions in such agreements to ensure that global competitors in developing countries maintain adequate standards to protect workers and the environment. At a meeting of the World Trade Organization (WTO) in Seattle in 1999, environmentalists arrived in turtle costumes to protest the WTO ruling against the U.S. Sea Turtle Conservation Act. They marched side by side with many members of labor unions, including the Teamsters union. The news media described the coalition as "turtles and teamsters."[16]

A Green Economy

Alliances among labor and environmental advocates have developed in a variety of fields, including urban development, land use, and energy policy. Construction unions and environmentalists have found common ground by fighting for "smart-growth," rather than "no-growth," policies. "No-growth" policies refer to those that favor limiting any development or expansion in order to support sustainable communities. "Smart–growth" policies advocate for development that avoids urban sprawl, focuses on building in the city center, and promotes sustainable practices in public transportation, housing, jobs, and natural resources preservation.

By jointly advocating for development in already urbanized areas, these alliances have promoted both union construction jobs and control of the health hazards associated with urban sprawl. These hazards include air pollution, heat, motor vehicle crashes, reduced physical activity, decreased water quality, pedestrian injuries and fatalities, and decreased mental health.[17] (See Chapter 39.)

Similarly, the Apollo Alliance has put forward a national sustainable energy policy that would create new high-wage, high-skilled jobs promoting environmental health, or "green-collar jobs." In developing this policy for a more sustainable future, a coalition of top leaders of the labor and environmental movements have joined forces to call for investment in energy-efficient buildings and technologies, such as hybrid cars and solar-powered buildings, and to decrease dependence on oil. Since the beginning of a global economic recession in 2007, even more interest has grown in the creation of "green jobs" to try to stimulate the economy. The labor movement has dedicated significant resources to this cause, such as the AFL-CIO "Center for Green Jobs."[18] The Center assists union leaders in training workers for new careers in a clean-energy economy, educating union members about public policy issues, and providing guidance on training opportunities for green jobs. For example, the Center is exploring opportunities for labor organizations to support projects that create clean

fuels and ultra-efficient vehicles, revitalize manufacturing and rebuild downtown areas in cities, launch "clean-coal" technology, and build a "smarter" electric power grid.

One premise of these alliances is that environmental health advocates who wish to collaborate with the labor movement must address the issue of jobs. At the same time, labor advocates must be more consistent in opposing "job blackmail" (or "job fear"), which can undermine their defense of environmental health. Unions must be committed to countering employers who threaten to move jobs because of environmental regulation. Not only does labor benefit by finding important allies for workers' rights in the environmental movement; it also helps create both sustainable jobs and cleaner, safer community environments for its members. (See Chapter 33.)

TEN TIPS FOR WORKING WITH UNIONS

The first steps toward working effectively with labor unions are understanding both the basic structure and functions of unions and learning about the history of labor's involvement with occupational and environmental health. Much can be learned from the experience of other professionals who have worked with unions. The following are 10 tips for working with unions, based on advice from practicing public health professionals and others.[8,19]

1. Involve the union in your work in as many ways as possible. Unions can be important allies in your efforts to improve health and safety in the workplace and the community. You will avoid difficulties, and gain substantial insight, by working with the union from the start.

Hearing from the unions gives me a complete, more balanced view of the reality of the workplace. Just hearing from management won't give you the whole picture. You need to hear from the "shop floor." It allows me to propose interventions that are likely to work in the real world. (A National Institute for Occupational Safety and Health [NIOSH] researcher)

2. Do not be surprised if a union takes an adversarial stance. It is the job of the union to advocate for its members.

Sometimes I can be frustrated when the union takes a very tough stand. But I have to remind myself that without its support we would have no health and safety regulations, we would have no health and safety programs, we would have no jobs in this field. (An industrial hygienist)

3. Educate yourself about labor. Find out which unions represent the workers with whom you interact. Learn about the union structure. Who are the elected officials, paid staff, executive board members, and shop stewards? Does the union have a designated safety representative? Does it have a community services department? Find out with whom you should communicate. In addition to your local union contacts, many labor unions have staff at national levels who specialize in safety and health. Get to know your union contacts and find out their concerns. Be proactive.

It can be complicated working with unions, knowing who to go to for what. You have to get to know the players. When do you need to talk to elected leaders? When do you want staff and when do you want the steward? When is it a good idea to talk with the international union and when would it be stepping on toes? If I'm not sure, I ask. (A community organizer)

4. Show respect for union leadership. Unions have hierarchies, just like other institutions. Go through proper channels. While you may have good informal contacts with individual union members, be sure to make official requests through the elected leadership.

Despite all of the stereotypes about "big business and big unions," unions are actually one of the few democratic institutions around. Local leaders are elected by the grassroots. It is important to respect these elected leaders. (A union health and safety director)

5. Get to know the rank-and-file membership of the union. While it is important to get the "buy-in" of the union leadership, it is also important to hear directly from the workers. Ask questions and listen carefully.

Sometimes I can't understand a position the union is taking. But if I get out and see for myself and talk with folks, I might understand it better. And sometimes I might get my own point of view across. Someone is always more open to your point of view if you show you are listening, too. (An environmental activist)

6. Visit the workplace. Tour the workplace with a union guide. Arrange to "shadow" a worker through the work day. There is no substitute for seeing working conditions firsthand.

You think you know what the hazards are in a particular industry, but I guarantee that the reality will blow you away! (A public health student)

7. Get to know the collective bargaining agreement at the workplace. Find out what the provisions are for health and safety. Does the contract call for joint labor-management health and safety committees? How are safety and health grievances handled? Are there training or other requirements that go above and beyond what OSHA requires?

Read the union contract. Work with it — it is just as important as what is spelled out in OSHA regulations. (A company safety director)

8. Share information with the union. Keep the union fully informed about the results of all occupational and environmental health investigations. Arrange for union participation in research projects and work with the union to notify workers of the results.

We did a cancer study of our workforce. We had an advisory committee of experts, but, even more importantly, we involved the union. It played a critical role all the way through the process, from designing the study to keeping the workers informed. (An occupational medicine physician)

9. Understand that unions have competing priorities. Health and safety may be important, but so are organizing, negotiating the union contract, representing members, mobilizing politically around local and national elections, and holding union elections. The union may not always be able to work with you on your timetable. Be flexible.

I have learned to expect the unexpected. Just because we have a deadline for our research doesn't mean the union can get to our agenda on our timetable. Most people who work for unions are working long and hard hours, under enormous pressure. If there is a strike, an election, a crisis, then we have to be prepared to wait. (A university researcher)

10. Establish your credibility. If you are an outsider or newcomer, you may be viewed with suspicion until you are known to be a steadfast, ethical advocate for health and safety. Never compromise your integrity.

Make sure the union knows you as a credible professional who is open to hearing concerns, will follow through, and will speak out honestly for health and safety. Go out on a limb. Be a real advocate for health and safety. Establish your track record. (An occupational health nurse)

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FURTHER READING

Center for Construction Research and Training. Available at: http://www.cpwr.com
A Web site of the Building and Construction Trades Department of the AFL-CIO. Has useful information about the building trades union and their work for health and safety. Also provides links to the Center for Construction Research and Training electronic library of information on construction hazards, el COSH.

Delp L, Ottman-Kramer M, Schurman S, Wong K. Teaching for change: popular education and the labor movement. Los Angeles: UCLA Center for Labor Research and Education and George Meany Center for Labor Studies/The National Labor College, 2002.
A collection of articles about the role of popular education. Includes several examples of creative union activities in occupational and environmental health, including participatory research with hotel room cleaners, using theater for worker health and safety, and the activities of the Just Transition Alliance.

Good Jobs First. High road or low road? Job quality in the new green economy. 2009. Available at: http://www.goodjobsfirst.org http://www.aflcio.org and http://www.changetowin.org.
Union Web sites with useful information about the U.S. labor movement and advocacy for health and safety.

Just Transition Alliance. Available at: http://www.jtalliance.org
Addressing collaborative efforts of labor and environmental justice advocates.

Labor Occupational Health Program. Tools of the trade: a health and safety handbook for activists. Berkeley: Labor Occupational Health Program, University of California at Berkeley, 2005.
A practical "how to" resource guide for unions and community groups advocating for worker protection. Contains multiple "real life" examples of union approaches to health and safety.

New Jersey Work Enforcement Council. Available at: http://www.njwec.org
A pioneer in organizing grassroots campaigns to help labor and environmental movements speak with a unified voice.

New York Committee on Occupational Safety and Health (NYCOSH). Available at: http://www.nycosh.org
In addition to news on developments in New York State, the site provides useful nationwide resources on a wide range of health and safety topics. Links available to all of the other COSH groups in the United States.

33

The Roles of Environmental Non-governmental Organizations

Kathleen M. Rest

Humankind has not woven the web of life. We are but one thread within it. Whatever we do to the web, we do to ourselves. All things are bound together. All things connect.

—Chief Seattle, 1855

There are no passengers on Spaceship Earth. We are all crew.

—Marshall McLuhan, 1964

The World Bank defines non-governmental organizations (NGOs) as "private organizations that pursue activities to relieve suffering, promote the interests of the poor, protect the environment, provide basic social services, or undertake community development."[1] They can generally be characterized as voluntary associations, independent of direct government control, nonprofit in nature, and established to pursue a specific mission or for a specific purpose. Non-governmental organizations can be broadly grouped into the following non-exclusive categories: operational, educational and research-focused, and advocacy. Operational NGOs deliver services, provide relief or development aid, and/or design or implement projects on the ground. Educational and research-focused NGOs produce research, analyses, and reports to inform policy processes, as well as information to educate decision makers, professionals, and the public about specific issues. Advocacy NGOs promote or defend specific causes; their campaigns or lobbying efforts target specific policies or changes. Each of the three types of NGOs can operate at the local, state, national, or international level, and some NGOs function across categories.

Non-governmental organizations are significant actors in civil society and world affairs. Influencing government policy, elucidating and changing corporate practices, engaging the citizenry, and otherwise working in the public interest, they have long been major drivers of social change. Environmental NGOs are critical allies for those working in—or advocating for—environmental, occupational, and public health. For an illustrative list of environmental NGOs see http://en.wikipedia.org/wiki/Category:Environmental_organizations.

HISTORICAL CONTEXT*

Accounts of the contemporary U.S. environmental movement often begin with the first

* This section is largely drawn from an exceptional historical account of the U.S. environmental movement: Gottlieb R. Forcing the spring: the transformation of the American environmental movement. Washington, DC: Island Press, 2005.

Earth Day in 1970 and the heady decade of environmental legislation that followed. The 1970s were marked by such landmark legislation as the Clean Air Act, the Clean Water Act, the Safe Drinking Water Act, the Endangered Species Act, and the National Environmental Policy Act. This same period marked the creation of the Environmental Protection Agency, the banning of the pesticide DDT, and the establishment of the Superfund program. These achievements followed on the heels of the rebellion, social unrest, and activism of the 1960s. Rachel Carson's *Silent Spring* (1962) clearly placed environmental concerns—specifically, the widespread use of pesticides and the power of the chemical industry—within the 1960s social agenda, which demanded civil rights and social justice.[2] Citizen and grassroots groups emerged and focused on community and environmental justice issues; existing or new national NGOs focused primarily on legislation, regulation, and the use of the courts to force or implement environmental policies.

Concerns about the natural environment, pollution, and public health, however, have much longer historical roots, as do NGOs associated with other "environmental" issues. In the mid to late 1800s, environmental concerns centered on the aggressive exploitation of natural resources (land, water, forests, and minerals) in the western United States, and on the preservation and protection of resources and places for recreation and/or simply for their existence (such as the wilderness). Some of the oldest environmental NGOs in the United States were established in the late nineteenth century, including the Sierra Club, the Appalachian Mountain Club, and the Audubon Society. Congress established the world's first official national park (Yellowstone) in 1872 and the legislative basis for the national forest system by passing the Forest Reserve Act in 1891.

The early to mid twentieth century marked the emergence of new NGOs and new federal government agencies. Policy debates pitted conservationism as a development strategy against preservationism as a social movement, with value in and of itself. Robert Marshall, an employee of the newly established U.S. Forest Service, an opponent of wilderness-destroying development (including tourism in the national parks) and a vocal advocate for democratizing the wilderness and making it accessible to all, helped establish The Wilderness Society in 1935. Another Forest Service employee, Aldo Leopold, championed a land ethic which "simply enlarges the boundaries of the community to include soils, waters, plants and animals, or collectively: the land."[3] Today, he is considered the father of modern ecology.

By the 1950s, concerns about resource scarcity and population growth gave rise to a technological optimism and a belief in the private sector, which became central to the mission of two important NGOs, both primarily research based: the Conservation Foundation and Resources for the Future.

Largely absent from these early environmental movements and NGOs were concerns about urban environments, pollution, and public health. The historical roots of these issues, however, are noteworthy. Famous for her landmark contributions to occupational health, Alice Hamilton is considered the first great urban and industrial environmentalist in the United States.[4] She and other early social reformers, many of whom were associated with the Settlement House Movement (most famously the Hull House in Chicago), identified and addressed community, consumer, workplace, and environmental problems as conditions of daily life that arose from unhealthy urban and industrial conditions. Conditions such as inadequate garbage collection and sewage disposal, contaminated water supplies, crowded tenements, dangerous workplaces, and polluted air were both sources of—and potential intervention targets for—social and urban public health problems. In the early twentieth century, a public health movement and professionalization of public health advocates emerged, and the U.S. Public Health Service was established in 1912.

Continued industrialization and urban concentration also gave rise to an urban planning movement, components of which had a vision for urban reform. During the 1920s and 1930s, this vision included a concept of "garden cities"—a vision of towns appropriately scaled to accommodate both industry and healthy living.

An era of new environmental challenges began after World War II and the Korean War. Military spending fueled rapid economic growth and

the development of new substances, such as plastics, petrochemicals, and fuel additives, and new technologies, such as nuclear power that was "too cheap to meter." At the same time, the use of advertising and mass marketing expanded and fueled consumption. These changes in production and consumption gave rise to many environmental and public health problems, including air pollution episodes in Los Angeles (1943); Donora, Pennsylvania (1948); and London (the London fog of 1952), which was associated with an estimated 12,000 excess deaths.

By the 1960s, environmental and public health concerns expanded beyond those apparent impacts to encompass worries about more subtle threats to public and ecological health, such as pesticides and hazardous technologies. Rachel Carson, Barry Commoner, and other social theorists who provided the foundation for the contemporary environmental movement bemoaned the divorce of science and technical expertise from a larger public and political framework. They challenged the domination of industry and its imperative to profit at any cost to the environment. Seeing technology as a driving factor in the environmental crisis, they were deeply suspicious of technical expertise—which could be bought—and the assumption that technical solutions were the key to addressing problems. In his influential book *The Closing Circle* (1971), Barry Commoner linked the deteriorating ecosystem to both production choices and the economic system, arguing that new synthetic organic chemicals and petroleum-based products were key drivers of environmental pollution. He also asserted that technical information created a formidable barrier between citizens and legislators.

The 1960s marked the emergence of a New Left, with social movements and organizations that advocated for civil rights; protested the power of the military-industrial complex, the Cold War, and nuclear proliferation; and challenged industry's creation of health and environmental hazards and corporate decisions that adversely affected health and the environment. This gave way to a counterculture movement that advocated for new institutions, such as "underground" newspapers, free universities, free clinics, and food cooperatives; new forms of cultural expression; and alternative lifestyles. Many environmental policy–focused NGOs, with professional staffs and strong advocacy positions, were established during this period, including the Environmental Defense Fund, the Natural Resource Defense Council, Friends of the Earth, the Environmental Policy Center, the League of Conservation Voters, and the Union of Concerned Scientists. Several more broadly focused public interest and consumer-advocacy NGOs, such as Public Citizen and the Health Research Group (both founded by Ralph Nader), were also established. These latter organizations played leadership roles in ensuring strong health, safety, and environmental protections, most notably for automobile safety, safety of drugs and medical devices, and occupational health and safety. A decade of unparalleled environmental progress followed—with 23 major pieces of national environmental legislation in the United States, as well as the Mine Safety and Health Act and the Occupational Safety and Health Act, signed into law.

The antinuclear movement, which also began in the 1970s, arose from a mistrust of the Atomic Energy Commission and a growing concern about thermal pollution, the health effects of low-level radiation emissions, and the safety of nuclear power plants. A new network, Critical Mass, and Friends of the Earth confronted the nuclear power industry with research findings, reports, and mass rallies. Several mainstream environmental NGOs joined the fray, including the Sierra Club and the Environmental Policy Center. Perhaps the most visible, powerful, and militant group opposed to the nuclear power industry to emerge was the Clamshell Alliance, a coalition of activists opposed to the construction of a nuclear power plant in Seabrook, New Hampshire. Its tactics, including civil disobedience and occupation of the site, attracted enormous media attention and became a model for other direct action groups, such as the Abalone Alliance in California, formed to protest the construction of the Diablo Canyon nuclear power plant near San Luis Obispo. (The plant was eventually built despite legal challenges and the arrest of 1,900 Abalone activists at the plant—the largest arrest in the history of the U.S. antinuclear movement.)

Another type of environmental NGO emerged during the 1970s—a type less focused on specific

concerns (such as nuclear power) or geographic issues (such as community-specific toxics problems), and more on bearing witness, protesting, and advocating for social and environmental change. The most successful and long-standing of these NGOs is Greenpeace. (Another is Earth First!, which is dedicated to "deep ecology"—an ecological philosophy that values human and nonhuman life equally—and militant direct action to protect the wilderness.) Founded in Vancouver, British Columbia, in 1971, Greenpeace has focused on transnational issues, such as nuclear testing, killing of baby seals, and commercial whaling. It gained high visibility for dangerous actions, such as sailing vessels into zones where nuclear weapons were tested and other sea-based expeditions.† Greenpeace International, with headquarters in Amsterdam, has more than 40 offices worldwide.

The election of Ronald Reagan as President of the United States in 1980 generated great concern among environmental NGOs and funding organizations, which knew that his "Government-is-the-problem" platform would likely be disastrous for environmental policy and legislation. Directors of 10 mainstream environmental NGOs and representatives of funding organizations convened to plan and coordinate collective positions and actions. This "Group of Ten"—a forerunner of today's "Green Group" of environmental NGO directors—produced *An Environmental Agenda for the Future* (1985)[5] and helped fund an environmental critique of the early years of the Reagan administration.

In 1983, OMBWatch, an NGO focused directly on the activities of the White House Office of Management and Budget (OMB)—a powerful office largely operating beyond the public eye—was founded. Among other things, OMB oversees federal regulation, the federal budget, and the collection and dissemination of information by government agencies. OMB-Watch, with its focus on government transparency and accountability as well as citizen participation, plays a critical role in ensuring that the government is open, responsive, and protective of people's health and safety. OMB-Watch also helps to ensure that government safeguards the environment and honors the public's rights to information and active participation in government processes.

While mainstream NGOs pursued their individual and collective policy agendas—mainly at the national level with their professional staffs and growing budgets—smaller community and grassroots groups emerged that focused directly on the day-to-day problems in local communities and neighborhoods. Toxic substances and hazardous waste often provided the stimulus for these groups. At their core, however, these groups and their networks were concerned with equity, environmental and social justice, citizen participation, and empowerment. These grassroots groups—often established and led by women—mobilized their communities; fought corporate interests; used informational tactics (such as community health surveys) and confrontational tactics (such as blocking waste sites) to demand government action.

The Love Canal Homeowners' Association exemplified this new form of grassroots citizen action. Residents of this lower-middle to middle-class community near Niagara Falls in New York State organized in response to contamination of their community as a result of years of indiscriminate dumping of toxic chemicals by Hooker Chemical Company. They mobilized local residents; performed health surveys; collaborated with outside scientists and environmental NGOs, such as the Environmental Defense Fund; employed attention-grabbing tactics (for example, they once held two Environmental Protection Agency [EPA] officials "hostage" by detaining them for several hours in a Love Canal home that served as the group's headquarters); and successfully confronted local and state government agencies that had been reluctant to act. The federal government eventually bought the residents' homes, and their leader, Lois Gibbs, became a nationally recognized hero of the antitoxics movement. She later established the Citizen's Clearinghouse for Hazardous Waste (CCHW) as a resource for community groups across the country facing similar problems. Now called the Center for Health, Environment & Justice, the organization continues to provide

† One mission resulted in the blowing up of a Greenpeace ship, the Rainbow Warrior, by the French government and the death of a freelance photographer for Greenpeace. See: http://www.greenpeace.org/usa/20-year-anniversary-of-rainbow/timeline-of-events

training, coalition building, and technical assistance to individuals, communities and groups—receiving over 1,500 requests for assistance each year. Many other antitoxics groups were established during this period, notably the National Toxics Campaign Fund, Clean Water Action, Citizens for a Better Environment, and the U.S. Public Interest Research Group (PIRG). PIRG is a state-based network of researchers, organizers, and students dedicated to results-oriented public interest activism; they confront powerful special interests and work on such issues as product safety, prescription drugs, health care reform, tax and budget policy, political corruption, and government reform.

A network of activists dedicated to the dissemination and democratization of information has been supportive to grassroots groups. Perhaps best known among these activists is Peter Montague, whose newsletters *Hazardous Waste News* and *Rachel's Democracy and Health News* became indispensable tools of support and information for local groups addressing toxic substances and social justice.

One of the first environmental NGOs to be run by people of color, WE ACT, was founded in New York City in 1988 to address community struggles concerning a sewage treatment plant in West Harlem. WE ACT is dedicated to building community power to fight environmental racism and improve environmental health, protection, and policy in communities of color and/or of low income. It has become a leading national organization addressing environmental justice in the United States, while continuing its work on local and regional issues.

A pivotal event for environmental justice was the First National People of Color Environmental Leadership Summit held in Washington, DC, in 1991. The Summit's official goal was to reshape and redefine the U.S. environmental movement by building a multiracial environmental movement that would empower and promote self-determination in communities around a central framework of environmental justice. Summit delegates drafted and ratified 17 principles of environmental justice (Box 33-1) and stimulated the creation of a communication network to foster collaboration and sharing of information.

There are thousands more NGOs and grassroots, community organizations in the United States working on environmentally related issues. In Europe, environmental NGOs have special standing and roles as recognized social partners in governmental policy development and decision making.[6] In addition, there are many NGOs that operate internationally, such as Conservation International, the World Wildlife Fund, the Climate Action Network, Oxfam, and the Rainforest Alliance.

STRUCTURE, STAFFING, AND RESOURCES

Most environmental NGOs are nonprofit and tax exempt, operating as 501(c)(3) organizations—an Internal Revenue Service designation that permits tax-deductible contributions, prohibits campaigning for political candidates, and permits limited lobbying activities. Some of these organizations establish 501(c)(4) entities, which permit them to work on political campaigns if this work remains secondary to their primary nonpolitical work.

Most national, mainstream environmental NGOs have professional staff members and substantial financial resources to support their work. Staff members include scientists, lawyers, economists, engineers, technical analysts, public health professionals, policy analysts, lobbyists, field organizers, communications specialists, and specialists in development (fundraising), information technology, finance, and administration. Smaller, grassroots or community groups generally have few paid staff members and rely primarily on work performed by volunteer members and experts.

Many environmental NGOs, such as the Natural Resources Defense Fund, Sierra Club, and the Union of Concerned Scientists, are membership organizations that attract activists and promote activism among their members. Others, such as the World Resources Institute, the WorldWatch Institute, and Rocky Mountain Institute, are environmental "think tanks" that do not engage directly in advocacy or activism. Revenue sources for environmental NGOs include individual members and donors; institutions, such as private foundations, government agencies, and corporations; and sale of products and services, such as books, calendars, and sponsored tours and trips.

Box 33-1. Principles of Environmental Justice
Adopted at the First National People of Color Environmental Leadership Summit, October 24–27, 1991, Washington, DC

Preamble*

We, the people of color, gathered together at this multinational People of Color Environmental Leadership Summit to begin a national and international movement of all peoples of color to fight the destruction and taking of our lands and communities, do hereby re-establish our spiritual interdependence to the sacredness of our Mother Earth; to respect and celebrate each of our cultures, languages and beliefs about the natural world and our roles in healing ourselves; to insure environmental justice; to promote economic alternatives which would contribute to the development of environmentally safe livelihoods; and, to secure our political, economic and cultural liberation that has been denied for over 500 years of colonization and oppression, resulting in the poisoning of our communities and land and the genocide of our peoples, do affirm and adopt these Principles of Environmental Justice:

1. *Environmental justice* affirms the sacredness of Mother Earth, ecological unity and the interdependence of all species, and the right to be free from ecological destruction.
2. *Environmental justice* demands that public policy be based on mutual respect and justice for all peoples, free from any form of discrimination or bias.
3. *Environmental justice* mandates the right to ethical, balanced, and responsible uses of land and renewable resources in the interest of a sustainable planet for humans and other living things.
4. *Environmental justice* calls for universal protection from nuclear testing and the extraction, production, and disposal of toxic/hazardous wastes and poisons that threaten the fundamental right to clean air, land, water, and food.
5. *Environmental justice* affirms the fundamental right to political, economic, cultural, and environmental self-determination of all peoples.
6. *Environmental justice* demands the cessation of the production of all toxins, hazardous wastes, and radioactive materials, and that all past and current producers be held strictly accountable to the people for detoxification and the containment at the point of production.
7. *Environmental justice* demands the right to participate as equal partners at every level of decision-making, including needs assessment, planning, implementation, enforcement, and evaluation.
8. *Environmental justice* affirms the right of all workers to a safe and healthy work environment, without being forced to choose between an unsafe livelihood and unemployment. It also affirms the right of those who work at home to be free from environmental hazards.
9. *Environmental justice* protects the right of victims of environmental injustice to receive full compensation and reparations for damages as well as quality health care.
10. *Environmental justice* considers governmental acts of environmental injustice a violation of international law, the Universal Declaration on Human Rights and the United Nations Convention on Genocide.
11. *Environmental justice* must recognize a special legal and natural relationship of Native Peoples to the U.S. government through treaties, agreements, compacts, and covenants affirming sovereignty and self-determination.
12. *Environmental justice* affirms the need for urban and rural ecological policies to clean up and rebuild our cities and rural areas in balance with nature, honoring the cultural integrity of all our communities, and providing fair access for all to the full range of resources.
13. *Environmental justice* calls for strict enforcement of principles of informed consent, and a halt to the testing of experimental reproductive and medical procedures and vaccinations on people of color.
14. *Environmental justice* opposes the destructive operations of multinational corporations.
15. *Environmental justice* opposes military occupation, repression, and exploitation of lands, peoples and cultures, and other life forms.
16. *Environmental justice* calls for the education of present and future generations which emphasizes social and environmental issues, based on our experience and an appreciation of our diverse cultural perspectives.
17. *Environmental justice* requires that we, as individuals, make personal and consumer choices to consume as little of Mother Earth's resources and to produce as little waste as possible; and make the conscious decision to challenge and reprioritize our lifestyles to insure the health of the natural world for present and future generations.

ISSUES AND STRATEGIES

The range of issues conventionally defined as "environmental" fall into many overlapping categories, such as the following:

- Nature/ecology, including, for example, wilderness, ocean, wildlife, and ecosystem issues
- Industrial/urban, including, for example, toxic substances, air and water pollution, and hazardous waste issues
- Global, including climate change, ozone depletion, and biodiversity issues

Alternatively, environmental issues are sometimes grouped by different types of environments,

such as natural, built, workplace, home, and community environments. They can focus on demographic and societal issues and trends, such as population growth, consumption patterns, and urban sprawl. They are often categorized by environmental threats associated with *(a)* economic sectors, such as energy, agriculture, and industry; *(b)* technologies, such as nanotechnology, nuclear power, and genetic modification of organisms; *(c)* conceptual frameworks, such as sustainability, smart growth, and pollution prevention; or *(d)* human health effects, such as endocrine disruption, cancer, and neurotoxicity. Indeed, there is no paucity of "environmental" subjects.[7] A compendium of topics and issues being addressed by many of the national environmental NGOs in the United States is shown in Table 33-1. Several of these organizations also address such cross-cutting or "framework" issues as environmental law and policy, environmental and social justice, environmental science, and economics.

Broader societal challenges contribute to or are affected by environmental issues. These challenges include, for example, promoting sustainable economic development, creating jobs, promoting social justice, reducing poverty, improving education, protecting consumers, promoting corporate responsibility, engaging citizens in political processes, protecting civil rights, and eliminating racism. Addressing these challenges is often integral to the goals, objectives, and activities of environmental NGOs. Some have criticized the environmental movement for not addressing these broader societal challenges.[8]

Table 33-1. Specific Topics Addressed by Selected NGOs*

- Climate change
- Renewable energy
- Coal
- Mountaintop removal
- Nuclear power, nuclear waste
- Transportation, vehicles, and fuels
- Auto safety
- Food
- Sustainable agriculture
- Forests
- Water
- Oceans
- Fisheries
- Air
- Toxics
- Drug safety
- Occupational health and safety
- Environmental health
- Smart growth
- Recycling
- Living cities
- Parks
- Open spaces
- Wilderness
- Wildlife
- Wetlands
- Environmental justice
- Nanotechnology
- Nuclear weapons
- Space-based weapons
- Scientific integrity
- Endangered species
- Biodiversity

* Issues listed on the homepages of the following NGOs: Natural Resources Defense Council (NRDC), the Environmental Defense Fund (EDF), the Union of Concerned Scientists (UCS), the Sierra Club, the National Wildlife Federation (NWF), Earth Justice, Friends of the Earth, Environment America, Clean Water Action, Physicians for Social Responsibility (PSR), Defenders of Wildlife, Public Citizen, and the Health Research Group.

Environmental NGOs employ a variety of strategies in service of their missions and pursuit of their goals. For example:

- They inform, educate, persuade, and embarrass.
- They bear witness.
- They monitor and serve as watch dogs of government and corporate activities.
- They conduct research and/or otherwise apply their scientific, technical, and analytical expertise.
- They litigate.
- They advocate for or against specific laws, regulations, and other government policies, including those that provide economic incentives and/or subsidies.
- They initiate legislative or regulatory proposals. For example, the Union of Concerned Scientists helped develop and champion the concept of renewable electricity standards. As of August 2010, these requirements were in effect in 28 states and the District of Columbia.
- They mobilize or otherwise engage the public and/or specific constituencies and activist networks to influence policy makers—often by providing information, training, and education.
- They forge alliances and coalitions to magnify their influence on government, corporations, or other powerful institutions.

Climate change is a priority issue for many national environmental NGOs, and they employ many strategies and tactics to establish or strengthen government policy. These include:

- Direct lobbying for national and/or state legislation that caps (or taxes) carbon emissions and for sectoral policies that reduce greenhouse gas emissions, such as renewable

energy requirements for the electricity sector, renewable fuels and fuel economy standards for the transportation sector, efficiency standards for buildings, and policies to protect threatened forests
- Analyzing specific legislative and policy proposals
- Developing reports and analyses on the urgency and impacts of global warming and the possible pathways to significantly reduce emissions (Fig. 33-1)
- Mobilizing citizens and various constituencies, such as scientists, economists, and religious leaders, to pressure their elected officials to take action
- Raising public awareness of the dangers of climate change and the urgent need for action

They also use these tactics to push for government action at the regional, state, and local levels. States are often the incubators for developing and initially implementing and evaluating new government policies. For example, in 2006, California established the most comprehensive, economy-wide program in the United States to reduce greenhouse gas emissions and global warming – the California Global Warming Solutions Act (AB32). This landmark policy is now under threat. Well-financed opponents have organized a referendum to overturn it. The question was on the California ballot in November 2010.

Many environmental NGOs extend their purview beyond government policy making. They seek to influence behaviors, conditions, and decisions of other institutions. In this context, they may focus their attention on, or even engage directly with, corporations. With focused public campaigns, environmental NGOs have exposed corporate practices and behaviors that imperil public health, worker health, and/or the environment. For example, environmental NGOs led campaigns to eliminate McDonald's use of polystyrene "clamshell" packaging. More recently, they persuaded McDonald's to require its poultry suppliers to end their use of antibiotics for growth promotion, as part of a campaign to address the growing threat of antibiotic resistance to human health.

Some environmental NGOs establish partnerships with corporations on specific issues. For example, the Environmental Defense Fund has established partnerships with FedEx to promote its use of hybrid vehicles and with Dupont to address concerns about nanotechnology.

CERES, another nonprofit organization, embodies a different model of corporate engagement. After the Exxon-Valdez oil spill in Alaska in 1989,

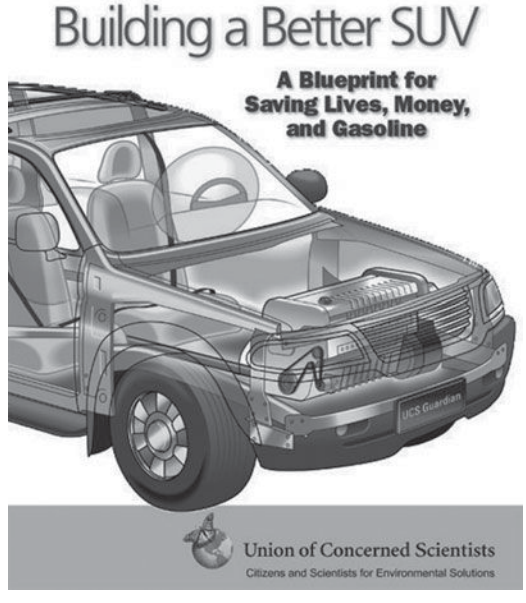

Figure 33-1. Cover of a report of the Union of Concerned Scientists concerning emission of greenhouse gases and climate change.

a group of investors founded CERES, now a national network of investors, environmental NGOs, and other public interest groups. CERES seeks to integrate "sustainability into capital markets for the health of the planet and its people."[9] It created the CERES Principles, a 10-point code for corporate environmental conduct, which has been endorsed by over 50 companies (Box 33-2). It also developed a plan for a sustainable global economy (CERES 20•20) that advocates for *(a)* honest accounting that abolishes the notion that pollution has no costs; *(b)* higher standards of business leadership; *(c)* bold solutions that accelerate "green" or environmentally friendly innovation; and *(d)* smart new policies that reward sustainability.[10]

The Apollo Alliance, a coalition of labor, business, environmental, and community leaders, was formed soon after 9/11. Its mission is to catalyze a clean energy revolution, with high-quality jobs and investments in clean energy technologies. Inspired by the Apollo space program, the Apollo Alliance advocates for a "New Apollo Program" that would invest $500 billion over the next 10 years to "accelerate the development of our vast energy resources, dramatically reduce carbon emissions… and transform America into the global leader of the new green economy." (See http://apolloalliance.org/about/.)

Shareholder activism is another corporate engagement strategy employed by some NGOs and activist investors. Activist shareholders use their equity stake in a corporation to pressure it to adopt corporate policies and practices that are pro-labor and environmentally friendly. The Interfaith Center for Corporate Responsibility

Box 33-2. CERES Principles: A 10-Point Code for Corporate Environmental Conduct

Protection of the Biosphere

We will reduce and make continual progress toward eliminating the release of any substance that may cause environmental damage to the air, water, or the earth or its inhabitants. We will safeguard all habitats affected by our operations and will protect open spaces and wilderness, while preserving biodiversity.

Sustainable Use of Natural Resources

We will make sustainable use of renewable natural resources, such as water, soils, and forests. We will conserve nonrenewable natural resources through efficient use and careful planning.

Reduction and Disposal of Wastes

We will reduce and where possible eliminate waste through source reduction and recycling. All waste will be handled and disposed of through safe and responsible methods.

Energy Conservation

We will conserve energy and improve the energy efficiency of our internal operations and of the goods and services we sell. We will make every effort to use environmentally safe and sustainable energy sources.

Risk Reduction

We will strive to minimize the environmental, health, and safety risks to our employees and the communities in which we operate through safe technologies, facilities, and operating procedures, and by being prepared for emergencies.

Safe Products and Services

We will reduce and where possible eliminate the use, manufacture, or sale of products and services that cause environmental damage or health or safety hazards. We will inform our customers of the environmental impacts of our products or services and try to correct unsafe use.

Environmental Restoration

We will promptly and responsibly correct conditions we have caused that endanger health, safety, or the environment. To the extent feasible, we will redress injuries we have caused to persons or damage we have caused to the environment and will restore the environment.

Informing the Public

We will inform in a timely manner everyone who may be affected by conditions caused by our company that might endanger health, safety, or the environment. We will regularly seek advice and counsel through dialogue with persons in communities near our facilities. We will not take any action against employees for reporting dangerous incidents or conditions to management or to appropriate authorities.

Management Commitment

We will implement these Principles and sustain a process that ensures that the Board of Directors and Chief Executive Officer are fully informed about pertinent environmental issues and are fully responsible for environmental policy. In selecting our Board of Directors, we will consider demonstrated environmental commitment as a factor.

Audits and Reports

We will conduct an annual self-evaluation of our progress in implementing these Principles. We will support the timely creation of generally accepted environmental audit procedures. We will annually complete the Ceres Report, which will be made available to the public.

From: CERES Website, http://www.ceres.org/Page.aspx?pid=416. Accessed on June 13, 2010.

(ICCR) is a coalition of 275 faith-based institutional investors who annually sponsor shareholder resolutions on many social and environmental issues. ICCR has had several significant successes. For example, it convinced a large U.S. automaker to pressure its suppliers to improve working conditions. In collaboration with CERES, it has filed many resolutions that have prompted companies to make climate-related commitments, such as adopting goals to reduce greenhouse gases and to monitor progress toward these goals. ICCR has filed shareholder resolutions to pressure companies to support broad principles of health care reform in the United States. CERES also directs the Investor Network on Climate Risk (http://www.incr.com), an alliance of 80 institutional investors with more than $7 trillion in collective assets.

CRITIQUES AND CHALLENGES FOR THE FUTURE

The historic accomplishments and contributions of environmental NGOs are widely acknowledged. The United States continues to benefit from the epic legislative victories of the 1970s, which would not have occurred without them. Recent critics of the environmental movement and its mainstream players, however, have questioned their relevance, criticized their strategies, and even suggested that the environmental movement is dead.[4,11–12] These critics decry the movement's narrow and arbitrary definitions of environmental problems and solutions. They accuse national environmental NGOs of becoming an integral part of the policy-making processes of the U.S. government, and thus invested in keeping the system intact—rather than promoting realignment of power relationships. They criticize these NGOs for being increasingly willing to compromise and accept incremental changes, rather than the transformative change they see as needed. Friendly critics opine that national environmental NGOs rely too much on outmoded strategies and technical policy solutions when what is needed is a positive and bold vision of the future—one grounded in U.S. values that can be broadly embraced by the public and will help shape and underpin policy decisions. They also criticize national environmental NGOs for failing to connect with and address the day-to-day concerns of disenfranchised communities and populations.

These critics envision a new path forward, in which the environmental movement goes beyond being just one more movement for a special interest. Some advocate for a more ecologically centered movement, with greater appreciation for and focus on the interdependence of people and nature. Others envision the merger of various singly focused "isms" into a broad and progressive social movement, with new institutions and a collective, coherent social justice agenda.

The global and societal problems we face today are enormous and complex. They cannot be defined or solved as single issues. Their solutions are both political and technical. For example, it will take more than solar panels, wind farms, and fuel-efficient vehicles to successfully address global warming. It will require political will to confront the entrenched economic interests of the fossil fuel industry—and its political contributions to elected officials. It will require political will to address issues of global equity and to provide money and technology to help developing countries play their part in solving this global problem. It will also require strong alliances among disparate groups to effectively counter the much more unified and resourced resistance to policies that challenge entrenched economic interests. As of August 2010, the U.S. Senate had not mustered the courage or the political will to pass climate and energy legislation that would cap greenhouse gas emissions and put the United States on a path to avoid the worst impacts of global warming.

In the United States, environmental NGOs have been indispensable partners and leaders in achieving many public health and environmental safeguards. However, any "movement" implies dynamism. Progress—and continued relevance—will require change. Some of this change has begun to occur with the emergence of broad-based coalitions like the Apollo Alliance and the growing activism on college campuses to address such interrelated issues as climate change, poverty, human rights, and sustainable development. All of us—the crew of Spaceship Earth—have an obligation to move these efforts forward by working together, with NGOs and with each other, to protect the environment, promote social justice, and preserve the planet we call home.

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34

Conducting Worksite Investigations

Bruce Bernard

A health care worker developed a severe case of H1N1 influenza, despite being given a surgical mask to use when caring for a patient. She had read on a Web site that N-95 respirators may have prevented her from getting sick. Why were these preventive measures not in place? Who could she get to look into these recommendations?

A poultry processing worker, standing and cutting chicken legs every 3 seconds on an evisceration line, developed eye irritation and a nagging, persistent cough at work, especially when spraying chicken meat. She knew about ergonomic hazards at work, but she wondered whether her cough might be due to some work exposure. She spoke with her line supervisor about this, but she was rebuffed and told to get back to work. She wondered how she could find out whether her symptoms were work-related.

Prisoners used claw hammers to demolish old video display monitors to reclaim recyclable metals. They had heard that the powdery dust covering them might contain heavy metals. Although they were given wrist-length gloves, the dust got on their arms and clothes, and left a gritty taste. They were concerned about thick, grainy mucus coming from their noses due to inhalation of this material.

A dealer at a casino worried about constant cigarette smoke filling the air. A nonsmoker, she was tired of her clothes smelling like smoke, and she wondered whether she was at increased risk of cancer and heart disease as a result. Had not everyone known that cigarette smoke was carcinogenic? What could she do about this exposure?

A man was not surprised when his doctor told him he had hearing loss. He had worked for many years at an animal shelter, where the dogs, kenneled in cement stalls, barked loudly every time he walked by. He had never worn hearing protectors—they had not been offered to any workers. He thought it was time to raise these issues with co-workers and management.

RECOGNITION OF POTENTIAL HAZARDS

There are many reasons why public health practitioners conduct onsite workplace investigations. Most workplaces have fewer than 100 employees and do not have onsite occupational safety and health specialists. Employers often rely on consultants for assistance.

A worker's illness or injury may trigger the need for an onsite workplace investigation to determine its cause and, if work-related, how to correct or control it. Sometimes occupational health and safety specialists are requested to conduct an investigation by employees or managers concerned about workplace exposures or health complaints from employees. Sometimes government agencies request onsite investigations because of increases in injuries or illnesses or newly recognized hazards. Sometimes workers'

compensation or other insurance companies request investigations to make recommendations to companies, when claims for work-related injuries or illnesses have increased.

Other common triggers for workplace investigations include the following:

- Blogs, Web sites, trade publications, insurance communications, or newspapers report that specific occupational injuries or illnesses are due to onsite work processes or tasks.
- Similar workers or workplaces identify specific injuries or illnesses.
- Professional publications or communications call attention to newly recognized occupational hazards.
- Workers begin to report new symptoms after changes in work processes or tasks.

This chapter lays out the general principles of workplace investigations, concerning recognition of potential hazards, preparation for onsite investigations, conducting these investigations, making useful and practical recommendations, and proactively intervening to implement them. After identifying uncontrolled hazards, exposures, or working conditions, the goal is controlling or reducing them to acceptable risk levels—or eliminating them entirely—and then to ensure that periodic reevaluations are done as part of routine operations. Boxes 34-1 through 34-4 describe four different occupational health issues and worksite investigations that address these issues.

IMPORTANCE OF WORKSITE OBSERVATION

There is no substitute for being onsite and witnessing work processes and tasks in "real time." Observation leads to a better understanding of exposures and working conditions, and it assists in developing better strategies for intervention. It helps with formulating recommendations for specific engineering controls, such as local ventilation, and administrative controls, such as

Box 34-1. Silica Exposure among Roofing-Tile Workers

Issue

Exposure to respirable crystalline silica particles places workers at risk for the development of silicosis, an irreversible condition that decreases lung function and increases lung cancer risk. Workers who develop silicosis can have a marked decrease in their quality of life due to difficulty breathing, cough, chest pain, and exercise intolerance. To diagnose silicosis, the treating physician must determine that the patient has a history of work-related exposure to respirable silica, confirmed by either a chest X-ray or a lung biopsy. Without the exposure history, silicosis can be misdiagnosed as another chronic lung ailment, such as emphysema or pulmonary fibrosis due to other causes.

Investigation

The National Institute of Occupational Safety and Health (NIOSH) conducted a site visit in response to a request from a union concerning roofing-tile workers in Arizona. There was concern that employees were exposed to hazardous levels of dust while sawing cement roofing tiles. At the outset, employees were unaware that the tiles contained a high content of silica. The roofing company, which installed about 800 roofs each month, employed about 400 workers; for most of them Spanish was their primary language.

Site visits were made to four construction sites to obtain samples of cement-tile dust and data on dust composition and particle size. Employees' silica exposures were aggravated by gas-powered leaf blowers used to remove dust and debris from the tiles during installation.

Although investigators brought with them Spanish interpreters, workers' local Mexican dialect made translation difficult. Workers reported that wearing respirators was very difficult because of the heat, and their sweat made proper face seals impossible. Workers were given questionnaires, chest X-rays, and pulmonary function tests to establish baseline evaluations. Most of them had normal lung function; none had moderate or severe impairments. After controlling for smoking, investigators found that decreasing lung function correlated with increasing years of dry-cutting cement tiles. No chest X-rays suggested silicosis. Employees stated that they wore respirators and hearing protectors infrequently. Although some employees reported respiratory symptoms consistent with silica overexposure, none said that they knew the hazards of silica overexposure. More than 75% of employees had been exposed to respirable silica levels above permissible limits. Some had been exposed to levels of total and respirable dust, noise, and carbon monoxide above permissible limits.

Source: National Institute for Occupational Safety and Health. NIOSH health hazard evaluation report (HETA #2003-0209-3015), Diversified Roofing Inc., Phoenix, Arizona. Washington, DC: NIOSH, November 2006.

Box 34-2. Chlorine Exposure among Lifeguards at an Indoor Swimming Resort

Issue

Chlorine is a broad-spectrum, inexpensive disinfectant that is active against most microorganisms, including bacterial spores. Eye and upper respiratory tract irritation, which are frequent symptoms of chlorine exposure, are usually intermittent. Chloramines, which are chlorine-related compounds, are suspected as a major cause of symptoms when chlorine is used as a disinfectant because of the interaction between chlorinated water and nitrogenous material. Exposures to chlorinated compounds may occur in indoor water parks, which have become more popular in the United States and Canada since 2000. To reduce energy costs, some of these facilities recirculate previously heated air, which may contain irritating concentrations of chlorine and chloramines.

Investigation

The National Institute of Occupational Safety and Health (NIOSH) received a request from a local health department to investigate symptoms of eye, nose, and lower respiratory tract irritation among employees at a large indoor water park resort. At the initial site visit, investigators met with representatives of management, lifeguard employees, and the local health department representatives to discuss issues and tour the facility. Industrial hygienists collected area air samples for trichloramine, soluble chlorine compounds, and endotoxin, and they measured air temperature and relative humidity. Investigators performed water-chemistry tests, and collected water samples for *Legionella*, fecal coliform bacteria, mycobacteria, endotoxin, sulfites, and sulfates. They conducted reviews of the designs of the water and ventilation systems.

Medical investigators initially conducted private interviews with employees and then designed a questionnaire survey, which they administered to lifeguards and a comparison group consisting of employees not working in the pool area. The questionnaire covered demographics and workplace information, smoking status, medical history, episodes of pneumonia or chest "flu" with fever and cough since working at the water park, and work-related symptoms within the previous 4 weeks. Participation was voluntary and written informed consent was obtained from the parents of all study participants under age 18. Lifeguards completed an additional questionnaire, at the end of their work shift on days when air sampling for chloramines was done, concerning symptoms experienced at work that day. Health outcomes of interest included the following: *(a)* work-related respiratory symptoms (cough, wheezing, shortness of breath, and chest tightness); *(b)* mucous membrane irritation (cough, sore throat, and eye and nose irritation); *(c)* systemic symptoms (fever, body aches); and *(d)* skin rashes. (Symptoms associated with acute upper respiratory infections were not included.) Symptoms were considered work-related if they occurred during work and improved when away from work. Symptoms of chest "flu" with fever and cough at work and recurrent episodes of pneumonia identified possible cases of hypersensitivity pneumonitis.

Lifeguards had significantly more work-related respiratory symptoms, eye and nose irritation, fever, body aches, and skin rashes in the 4 weeks prior to questionnaire completion than unexposed employees. Lifeguards had significantly more work-related cough and eye irritation when hotel occupancy was high. Trichloramine concentrations, which were similar to those in other indoor swimming pools, were nevertheless at levels reported to cause irritation of mucous membranes. Endotoxin levels in the air in almost all pool areas exceeded the American Conference of Governmental Industrial Hygienists (ACGIH) proposed recommended limit values for endotoxin exposure. Water-chemistry test results met state standards. No *Legionella*, mycobacteria, or fecal coliform bacteria were found in any water sample. Placement of the air-supply diffusers and return-air inlets 30 to 80 feet above deck level made it difficult to provide adequate air movement and mixing at the pool surface and deck levels; it also created the potential for short-circuiting supply air to the exhaust. After the resort made changes to the ventilation system, respiratory irritation among workers and visitors decreased.

Source: Chen L, Dang B, Mueller C, et al. Investigation of employee symptoms at an indoor waterpark. Health hazard evaluation report (HETA 2007-0163-3062), Great Wolf Lodge, Mason, Ohio. Washington, DC: National Institute for Occupational Safety and Health, June 2008.

rotating workers to minimize exposure to hazardous tasks or processes. It also enables a health and safety specialist to recognize hazards or unsafe conditions that may go unnoticed by workers or employers.

Evaluating a workplace often requires a multidisciplinary approach. Input by employees and employers, physicians, engineers, chemists, health physicists, and social scientists may be needed to successfully address hazards and unsafe work conditions. The most successful approaches coordinate many disciplines and incorporate effective communication between employees and employers for recognizing, evaluating, and controlling hazards and unsafe working conditions. A multidisciplinary approach may not be practical for many workplace situations. However, each person evaluating a workplace must be knowledgeable of possible contributions of other professionals in solving problems. For example,

Box 34-3. Environmental Tobacco Smoke Exposure among Casino Dealers

Issue

Longstanding National Institute of Occupational Safety and Health (NIOSH) policy, based on findings of the Surgeon General, states that workers should not be involuntarily exposed to environmental tobacco smoke (ETS), which is associated with increased risk of lung cancer, other respiratory disease, and heart disease. There is no risk-free level of ETS exposure. Nonsmokers exposed to ETS at home and at work increase their risk of developing heart disease by 25% to 30% and lung cancer by 20% to 30%. Almost half of all nonsmoking U.S. residents are regularly exposed to ETS. (See Box 7-1 in Chapter 7.)

Investigation

NIOSH received a request from gaming employees in three casinos in Las Vegas who were concerned that their workplace exposure to ETS was causing acute and chronic disorders. During onsite evaluations that NIOSH performed at the three casinos, casino dealers expressed concern about respiratory disorders associated with ETS exposure. Some dealers were selected for environmental and biological monitoring to determine exposure to a specific tobacco carcinogen, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), and its metabolite, NNAL, the presence of which in urine indicates ETS absorption.

Industrial hygienists performed, in the gaming areas, both full-shift personal breathing-zone sampling and area-air sampling for nicotine, respirable particles, and other components of cigarette smoke. Preshift and postshift urine samples were collected from 114 casino dealers to determine whether NNAL levels increased during work shifts. Casino dealers had measurable airborne levels of ETS (including nicotine, toluene, and hydrocarbons). As evidenced by measurable urinary NNAL, they had absorbed NNK, a carcinogen in ETS. Investigators recommended that casinos prohibit smoking and develop smoking cessation programs for their employees.

Box 34-4. Indoor Air Quality and Cancer

Issue

Almost 70% of workers in the United States are employed in nonindustrial, nonagricultural, indoor work environments. The proportion of National Institute of Occupational Safety and Health (NIOSH) investigation requests since 1972 for indoor environmental quality (IEQ) problems has increased 100-fold, now representing 56% of all requests. Over one-half of these IEQ requests mention mold or water damage as an environmental exposure concern, and about one-fourth mention asthma or cancer as a health concern. (See Chapter 7.)

Investigation

NIOSH received a request from managers of office buildings regarding ongoing employee and union concerns about a possibly higher rate of cancer among current and former employees of the buildings. No cause for these cancers had been identified, but employees were concerned about potential exposure to jet fuel and deicing compounds from the nearby airport, asbestos and water damage in the buildings, and overall poor air quality. The health hazard evaluation (HHE) focused on the employees in two, adjacent three-story brick office buildings, which had been constructed in the early 1960s. NIOSH investigators reviewed reports on asbestos remediation in these buildings, responses to complaints from building occupants, and environmental sampling during the previous 14 years. They evaluated management surveys of cancer diagnoses among building employees. Investigators spoke with representatives from the Ohio Environmental Protection Agency about any past or current environmental contamination issues. They spoke with representatives from the state cancer registry. They visited the site, met with management and union representatives, performed a walk-through survey of the buildings, measured IEQ comfort parameters, and looked for evidence of water damage, water incursion, visible mold, and other problems. They presented cancer findings to employees. They then had a closing conference with management and union representatives. Twenty different types of cancer had been diagnosed among building employees in the previous 25 years, the most common of which were cancers of the breast (17 cases), lung (7 cases), and prostate (4 cases)—the three most common cancers in the United States. Airport runoff of jet fuel and deicing fluid had entered the Rocky River, next to one of the buildings. But these substances are not known to cause cancer, and the river was not a source of drinking water for building occupants. Much of the asbestos in the buildings had been removed, but some was still managed in place and posed no hazard to building occupants. The investigators identified minor IEQ problems, such as water damage to ceiling tiles and walls and poor maintenance of fan coil units, but these problems are not known to be associated with the types of cancer diagnosed among building employees. They found no evidence that reported cancers were associated with work—the numbers and types of cancers were not unusual and there were no significant work-related hazardous exposures. Investigators recommended that management and the union encourage employees to learn about *(a)* known cancer risk factors, *(b)* measures they could take to reduce their risk for preventable cancers, and *(c)* availability of cancer screening programs.

a physician studying a work environment should have not only knowledge of the health effects of specific chemical exposures but also a basic understanding of the relevant chemistry, chemical-sampling techniques, and engineering requirements for control.

To recognize potential hazards at a workplace, one should become familiar with work processes, review a list of possible exposures from chemical and physical agents, consider job activities in work areas of interest, and study possible control measures for exposures or hazards that may be present. One also needs to determine how managers respond to workers' reports of symptoms. Managers' commitment to respond promptly to these reports and investigate potential causative factors provides clues on how they approach workplace problems and how committed they are to prevention.

THE NIOSH HEALTH HAZARD EVALUATION PROGRAM

The National Institute for Occupational Safety and Health (NIOSH), part of the Centers for Disease Control and Prevention (CDC) within the Department of Health and Human Services, is the U.S. federal agency responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses. The NIOSH Health Hazard Evaluation Program responds to requests for workplace evaluations from employees, unions, employers, and other governmental agencies. A health hazard evaluation (HHE) is an investigation of a workplace to assess whether workers are exposed to hazards or harmful conditions. The NIOSH HHE program, using a team consisting of an industrial hygienist and an occupational medicine physician with training in epidemiology, conducts 300 to 400 investigations annually. Through the HHE program, NIOSH identifies current hazards and recommends practical, scientifically valid solutions for reducing exposures, controlling harmful conditions, and preventing disease, injury, and disability. Workplace investigation techniques used by the NIOSH Health Hazard Evaluation Program are described next. They can be adapted for use at most workplaces.

Preparing for a Workplace Investigation

Gathering Information

Investigators develop overall plans, collaboratively determine specific questions to be answered, and plan the investigative strategy. Initial telephone calls obtain information about the workplace problem with the person who requested the investigation, workers, managers, and other people. Initial information is obtained on workplace operations, the materials or chemicals used, hazards present, processes and work tasks, time sequence and duration of existing problems or concerns, previous actions taken to address the problem, recent process or materials changes, and the urgency of the situation. Emergency situations—those that are immediately hazardous to life or health—should be referred immediately to the Occupational Safety and Health Administration (OSHA). Investigators determine whether managers are aware of any potentially work-related health problem at the workplace. If there is a labor union that represents workers who may be affected or exposed, it is informed about the investigation and requested to provide relevant information, if available, on workers' medical care and work tasks.

At the time of the initial telephone call, determine which parties need to be included in the investigation: employers, workers, any worker representatives (from local and national unions), medical care providers, other health professionals, and local and state health department representatives, as deemed appropriate. Critical to a successful investigation is involvement of employees and their representatives, such as union stewards, as well as managers and other employer representatives from the start. Because employees have a unique understanding of job tasks, and working conditions, information gained from them is especially valuable in determining whether hazards exist and assessing them. Involving employees from the start helps to improve the quality of the investigation, minimize oversights, and enable them to fully understand the need for the investigation and gain their cooperation.

Usually, with little background investigation, early clues help determine the scope of necessary work. For example, illness among many workers

in different jobs in various departments likely indicates the need for a full-scale, workplace-wide investigation. Alternately, if suspected problems are confined to isolated tasks or relatively few workers, only a more limited, focused investigation may be necessary.

On the initial phone call, determine what health and safety hazards might be encountered onsite and what personal protective equipment (PPE) members of the investigative team might need during the site visit. If respirators are required onsite, only personnel who have been medically cleared, trained, and fit-tested can use them.

Roles of the Investigative Team

For the industrial hygienist, preparation for a field investigation begins with identifying exposures of concern, determining whether there are appropriate sampling and analytical procedures that will need to be performed, determining analytical chemistry or microbiological services needed, determining proper instruments to be selected, and making an industrial hygiene equipment list. Determining appropriate sampling usually requires being onsite or having enough information beforehand to know exactly what needs to be sampled, where, and why; performing sampling in a rush and obtaining unneeded data points (because "it may be the only opportunity to sample") is rarely fruitful. Preparation for sampling includes arranging for equipment, supplies, and analytical services, and knowing any shipment requirements for hazardous materials.

For the occupational medicine physician, preparation involves searching medical literature, reviewing medical records, and having the diagnostic and examination skills to sort out what may be work-related in the workplace. Medical support staff responsibilities may include designing a study; developing the investigative protocol; obtaining necessary approval from a human subjects review board; preparing consent forms and questionnaires and other data-collection forms; and arranging for field-study materials, personnel, and medical tests.

If biological testing is to be conducted, arrangements need to be made for clerical support, data-collection forms, supplies for venipuncture and collection of urine or other biological samples, as well as forms to request tests not routinely performed by clinical laboratories, such as those for metals, pesticides, volatile organic compounds, polychlorinated biphenyls, furans, dioxins, polycyclic aromatic hydrocarbons, and phthalates. Plans also need to be made for special studies, such as pulmonary function tests, chest X-rays, neurobehavioral tests, and other tests that may require a consultant.

Obtaining Needed Information before the Site Visit

Many manufacturers have technical and other information on their Web sites on product lines, work processes, financial status, and managerial systems. Major unions also have useful information on their Web sites. Information on Web sites also includes research findings, technical experts, and survey instruments.

If the worksite is a manufacturing facility, investigators need to learn about goods produced, chemicals and other substances used, and intermediate products formed in the production processes. Much of this information can be obtained before the site visit through discussions with employees, employers, and technical experts, or on the Internet.

Before the site visit, obtain records on exposure monitoring, purchasing, production, health and safety policies and operating procedures, all of which can help in determining the exposures of most concern. Employee rosters, staffing lists, employee turnover rates, and floor plans may also provide useful information. Reviewing these documents prior to the site visit will help give investigators a better understanding of potential hazardous exposures and company procedures to respond to hazardous situations. The site visit will help to determine whether these procedures are operational. Once background information is obtained, the leader of the investigation assembles an investigative team.

Material safety data sheets (MSDSs) on hazardous substances, which are mandated at manufacturing plants by the OSHA Hazard Communication Standard, can be requested from management. Workplaces in other industries will generally not have MSDSs; however, containers of hazardous substances that they use, such as cleaning products and insecticides, are required to have hazard warning labels that can provide some toxicity information.

OSHA Logs and Other Existing Records

Investigators can request to obtain *(a)* the logs of injuries and illnesses that are required by OSHA, and *(b)* plant medical records, workers' compensation claims, insurance claims, absentee records, and job-transfer applications, all of which can yield useful information on work-related injuries and illnesses. If workers in certain departments or processes have higher rates of health problems than others, especially if they have the same type of injuries or illnesses, this suggests specific areas for investigation. Jobs with increased rates of certain symptoms, such as lightheadedness or concentration problems, may also have higher risks for acute injuries.

In 2004, OSHA mandated access to illness and injury log summaries available at each workplace, so that information could be easily collected during an investigation. OSHA now requires that employers post, in a common area wherever notices to employees are usually posted, a summary of job-related injuries and illnesses. The summary must list the total number of job-related injuries and illnesses that occurred in each year since 2003. OSHA also requires the posting of annual average number of employees and total hours worked during the calendar year, so that workplace incidence rates can be calculated. Companies with no recordable injuries or illnesses must still post the form. All summaries must be certified by a company executive. OSHA also requires employers to make a copy of the summary available to employees who move from worksite to worksite, such as construction workers, and employees who do not regularly report to any specific worksite.

Medical and First-Aid Records

Investigations of suspected work-related injuries and illnesses should also include review of first-aid and medical records to understand the magnitude and seriousness of such problems. The Health Insurance Portability and Accountability Act (HIPAA) requires that *(a)* specific medical-release authorization from individual workers be given before access to their medical records can be obtained, and *(b)* employers and onsite health care providers protect individual health data. Exempt from HIPAA requirements are public health officials, who are authorized by law to have access to individual health information for the purpose of preventing or controlling disease, injury, or disability—including for investigations and interventions. Examination of employee first-aid and medical records may offer leads to jobs or operations that may cause or contribute to other work-related problems.

Specifics of a Workplace Investigation

The Initial Worksite Visit

The primary purposes of the workplace site visit are to *(a)* determine, while onsite, the severity and extent of the problem; *(b)* identify possible causes; *(c)* see if, at an early stage of the investigation, that there may be possible solutions to the problem; and *(d)* ascertain whether further assessment is needed. The initial site visit can be usually completed in 1 or 2 days, but it may take longer, if additional time is needed to complete it without a follow-up visit.

A good way to start the site visit is with a meeting with all those involved, including the facility manager, the chief local union official (or other worker representative if employees are not represented by a union), health care professionals, engineering and maintenance workers familiar with the facility, and consultants who are familiar with the facility. Discuss plans for confidentiality of information from worker interviews and personnel and medical records, and procedures for videotaping, photographing, and other recording. Review personal protective equipment requirements and any other relevant safety procedures to be used during the investigation.

Walkthrough Observational Survey

A walkthrough survey, which can be the most important part of the investigation, should include managers, employees, their representatives, including the person who requested the workplace investigation, unless that person has requested confidentiality or has declined to participate (Fig. 34-1). Usually, the main purposes of the walkthrough survey are to observe facility operations, identify potential hazards, and talk informally to employees, managers, and others about the problem.

Figure 34-1. An industrial hygienist and an occupational medicine physician pause for questions from a worker during a workplace walkthrough survey. (Courtesy of the National Institute for Occupational Safety and Health.)

The walkthrough allows observation of workers performing job tasks, use of PPE or protective clothing, placement of materials, tools, physical layout of the workplace, and the organizational climate. Many potentially hazardous operations can be detected by visual observation during the walkthrough. Using lists obtained beforehand of chemicals, raw materials, products, and by-products assists in identifying hazardous inhalational and skin exposures. Knowledge of fuels used in burning processes assists in identifying air contaminants. Observation of ventilation systems helps to determine needs for improved control measures. The walkthrough can assist in understanding job tasks that place workers in specific jobs at risk and can help determine the need for additional industrial hygiene sampling, worker interviews, and medical testing.

The dirtiest, dustiest operations are not necessarily the most hazardous. For example, dust particles that cannot be seen by the unaided eye can be the most hazardous because they are of respirable size. The absence of a visible dust cloud does not necessarily mean that there is no airborne dust. Odors are not reliable indicators of exposure: Odors might not be detected of vapors and gases present in concentrations considerably above their permissible levels, and ability to detect an odor often decreases as exposure continues.

Workers' Job Tasks

It is important to obtain a list of workers' routine job tasks and requirements in areas of the workplace being investigated. Changes in job requirements or modifications of work techniques or processes may have profoundly affected hazardous exposures. Shift work or overtime work requirements may contribute to prolonged exposure of workers, which may not occur on an 8-hour work schedule.

Most job tasks can be described in terms of *(a)* tools, equipment, and materials used; *(b)* workstation layout and physical environment; *(c)* task demands; and *(d)* organizational climate in which the work is performed. More definitive

procedures for collecting information on job tasks can include the following:

- Videotaping to observe workers performing tasks for a time-activity analysis
- Photographing workstation layout, tools, materials, and chemicals used
- Recording workstation measurements and characteristics of work surfaces, including heights, edges, reach distances, and slip resistance
- Determining perceived exertion of workers

While screening tools, such as checklists have been widely used in many investigations, most have not been scientifically validated. Combining checklist observations with data on symptoms offers a way of reducing uncertainty.

Focusing on Jobs

Jobs associated with the most, or the highest rates of occupational illnesses and injuries, deserve the most attention. Jobs in which recent cases have occurred deserve priority attention. Priority for job analysis and intervention should be given to those jobs in which *(a)* the most people are affected, or *(b)* changes in work exposures or processes are taking place or planned. Jobs associated with workers' complaints of fatigue and discomfort should be ranked next in priority for analysis and intervention. Finally, where screening suggests presence of significant risk factors or exposures for occupational illnesses or injuries, more detailed job analyses should be done. Jobs with higher levels of exposure or multiple risk factors may indicate a need for control.

Selection of Instruments to Evaluate the Work Environment

Industrial hygiene sampling (Fig. 34-2) is sometimes necessary on the initial site visit to determine the range of exposures to begin planning for more definitive sampling (Chapter 26). Direct reading instruments and/or detector tubes are generally used because of their portability and

Figure 34-2. Industrial hygienists collect follow-up samples for a silica exposure among roofers. (Courtesy of the National Institute for Occupational Safety and Health.)

ease of use. In-depth quantitative air sampling is generally not done on the initial site visit.

Interviews

The lead investigator should establish a schedule to interview the following people:

- Managers and other employer representatives
- Workers (Although it is reasonable to interview specific workers at their request or the request of others, it is important to interview a cross section of workers. Group interviews can supplement individual interviews.)
- Union representatives
- Physicians, nurses, and other health and safety personnel
- Representatives of the human resources department

Conducting Symptom Surveys

Symptoms surveys can assist in focusing on specific concerns of workers and in identifying possible work-related disorders that might otherwise go unrecognized. These surveys provide information to narrow the focus of investigation. In addition to questions about workers' job titles and tasks, the location, frequency, duration, and intensity of symptoms will help to determine the focus of the investigation. By definition, symptom surveys rely on self-reports—a potential limitation. An epidemiologist can assist with questionnaire design and data analysis.

Medical examinations: A disadvantage of using OSHA logs or company-based medical information to identify possible cases of work-related injuries or illnesses is the lack of uniform definitions. In the NIOSH HHE program, investigations have included limited physical examinations focused on specific organ systems or parts of the body. Data obtained can help establish the prevalence of work-related conditions and whether any might be related to work. Prevalence data for a comparison group of unexposed or lesser-exposed workers may be helpful. Standardized periodic medical examinations, performed at some workplaces, may provide valuable clues, but they are generally not designed for continuous surveillance.

Integration of data: All those involved in the investigation should meet onsite to discuss and integrate their findings and plan next steps.

Summarizing Onsite Information and Holding a Closing Conference

Hold a closing conference before the initial worksite visit is completed to discuss what has been accomplished. Invite those present at the opening conference and other key employees and managers. New recommendations can be made and previous ones can be modified. Future activities and reports can be discussed.

Activities after the Site Visit

Maintain all records, notes, forms, and other data from the site visit in locked files. Check and decontaminate all sampling equipment. Arrange for laboratory analysis of samples. Review and check analytical results for reliability. Make arrangements for coding, entry, analysis, and storage of medical data.

Follow-up Reports

Within a few days of the initial site visit, write a letter to managers and employees and their representatives, summarizing the findings of the visit. The letter should use clear language and provide a clear understanding of possible health effects associated with the hazards encountered. A telephone conference call can also communicate information on a health hazard—or its absence—and alleviate any misunderstandings or concerns heightened by publicity. It can also facilitate timely implementation of control measures. Any results and recommendations reported by telephone should be included in a subsequent written report. Preparation of the final report to employers and employees should integrate both the industrial hygiene/environmental and medical/epidemiological components of the investigation.

Considering Recommendations of a Worksite Investigation

The occupational health and safety three-tier hierarchy of controls—widely accepted as an intervention strategy for controlling workplace

hazards—is useful in outlining recommendations in the report. The three tiers are as follows:

1. Engineering controls
2. Administrative controls (changes in work practices and management policies)
3. Personal protective equipment (PPE)

Engineering Controls

Recommendations should begin with examination of existing engineering control strategies to determine whether the following are evident:

1. Work is set up to reduce worker exposures.
2. Substitution has been attempted to reduce harmful material exposures.
3. Work operations are isolated or enclosed to reduce worker exposures.
4. Wet methods are being used to reduce generation of dusts.
5. Local exhaust and general ventilation are adequate.
6. Shielding from radiant heat, ultraviolet light, radiation, and other forms of energy is used.
7. Modifying presentation of parts on assembly lines has been attempted.
8. Equipment is height-adjustable, tools are in adequate proximity, and objects handled are of appropriate weight.
9. Appropriate procedures are in place for housekeeping, waste disposal, eating and washing, and use of toilet facilities.

Administrative Controls

Recommendations regarding administrative controls are usually directed to management, because they concern work policies that reduce or prevent exposures. Administrative control recommendations can address issues such as the following:

1. Scheduling shifts and rest breaks
2. Rotating workers in and out of specific jobs
3. Evaluating production quotas and performance standards concerning their impact on workplace stress, work pace, and worker control
4. Providing meaningful light-duty jobs, as deemed appropriate, to allow injured or ill workers to maintain contact with fellow employees and gradual return to normal activities, while providing for specific medical needs
5. Providing periodic training of employees on work risk factors and recordkeeping
6. Implementing medical management and surveillance programs
7. Implementing workplace smoking policies

Most administrative recommendations should be seen as *(a)* temporary measures until engineering controls can be implemented, or *(b)* measures to use when engineering controls are not technically feasible. Since administrative controls do not eliminate hazards, managers must ensure that practices and policies are diligently followed. Administrative controls, such as worker rotation or allowing more rest breaks, are "stop-gap" measures—not permanent solutions.

Personal Protective Equipment

Personal protective equipment measures do not substitute for good engineering or administrative controls or good work practices. Personal protective equipment recommendations should be implemented only with the assurance that these other controls and practices have already been considered. When use of PPE is deemed necessary, appropriate training must be on proper use and maintenance of PPE.

Implementing controls normally consists of *(a)* initial testing of the selected measures, *(b)* modifying these measures based on initial testing, *(c)* implementing them on a large scale, and *(d)* evaluating their effectiveness. By testing and evaluating measures, one can determine whether they achieve the desired outcome and identify any necessary modifications. Workers can provide valuable input into testing and evaluation. Worker acceptance of changes is important to the success of the control measures. Workplace control measures often start by targeting problems clearly identified in the workplace investigation and those problems that appear easiest to solve. Early success can build confidence and experience needed later to solve more complex problems.

Evaluating Effectiveness of Controls

Periodically evaluate implemented controls to determine whether they have reduced hazards and/or decreased injuries or illnesses, and to ensure that control measures have not introduced new risk factors. Follow-up evaluation should occur no sooner than 6 weeks after implementation of control measures to avoid discarding effective control measures that may not have yet demonstrated their benefits. Evaluation may also include a symptom survey and completion of a risk-factor checklist or another job-analysis method. Results of a follow-up symptom survey can be compared with those of the initial symptom survey to determine the effectiveness of control measures in reducing symptoms. (Be aware that some ergonomic control measures lead to changes in work methods, requiring workers to use different muscle groups, which may make them sore during the "break-in" period.)

Proactive Approaches

To this point, the topics outlined in this chapter have represented *reactive* approaches for workplace investigations. In contrast, *proactive* approaches are geared to preventing problems from developing. Proactive measures emphasize designing work tasks and processes to avoid causes of occupational illnesses and injuries. They include design of operations that ensure proper selection and use of tools, job tasks and processes, workstation layouts, and materials that are unlikely to harm workers.

Essential Considerations

Ideally, workplace problems are identified and resolved in the planning process. In addition, general occupational health and safety knowledge, learned from an ongoing health and safety program, can be used to build an approach more oriented to prevention. Management commitment and employee involvement in planning are essential. For example, management can set policy to require health and safety considerations for any equipment to be purchased, and production employees can offer ideas on the basis of their experiences for alleviating potential problems.

Decision makers who are planning new work processes, especially those involved in the design of job tasks, equipment, and workplace layout, must become more aware of health and safety factors and principles. Designers must have appropriate information and guidelines about risk factors for occupational illnesses and injuries and ways to control them. Studying past job designs can help determine what improvements are needed.

Because design strategies try to target the causes of potential occupational illnesses and injuries, engineering approaches are preferred over administrative approaches—they eliminate risk factors instead of only reducing exposure to them.

FURTHER READING

Centers for Disease Control and Prevention. Health hazard evaluations. Available at: http://www.cdc.gov/niosh/hhe/.
This Web site provides the complete guide to the nuts and bolts of the NIOSH Health Hazard Evaluation Program.

National Research Council and Institute of Medicine of the National Academies. The Health Hazard Evaluation Program at NIOSH. Reviews of Research Programs of the National Institute for Occupational Safety and Health. Washington, DC: National Academies Press, 2009.
A useful resource on the NIOSH Health Hazard Evaluation Program.

Occupational Safety and Health Administration. Screening and surveillance: a guide to OSHA standards. OSHA 3162-12R. Washington, DC: U.S. Department of Labor, 2009. Available at: http://www.osha.gov/Publications/osha3162.pdf.
A quick reference for locating and implementing the screening and surveillance requirements of OSHA standards.

The findings and conclusions in this chapter are those of the author and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

35

Responding to Community Environmental Health Concerns

Henry A. Anderson and Henry Nehls-Lowe

Improved health owes less to advances in medical science than to changes in external environment, and to a favorable trend in the standard of living. We are healthier than our ancestors not because of what happens when we become ill but because we do not become ill: and we do not become ill not because of specific protective therapy but because we live in a healthier environment.

—Rene Dubos, *Man Adapting*, winner of the 1969 Pulitzer Prize

Communities have experienced unprecedented advances and challenges in environmental health in the past three decades. Developments have included new chemical contaminants, such as fire retardants and nanoparticles, policies for addressing climate change, a focus on the built environment, and application of health impact assessments and other new tools. Two advances have been extremely valuable in raising and maintaining community awareness about environmental health issues: the evolution of the community right-to-know movement and the development of the Internet and the World Wide Web.

The community right-to-know movement continues to identify environmental health disparities and empower communities to address these disparities by providing easy access to relevant information. It enables communities to know more about their environment, sources of hazardous exposure, and proposals that might adversely affect their health and quality of life. Although implementation of right-to-know policies has been especially valuable to low-income and minority communities, which are often disproportionally affected by environmental decisions, it has benefited all communities. Community members are now sent the results of water-quality testing performed by their municipal water authorities. Many are familiar with the Environmental Protection Agency (EPA) index of air quality (IAQ) that is included in most daily weather reports. They can also review at local libraries data on Superfund site investigations in their communities.

A major strength of the Agency for Toxic Substances and Disease Registry (ATSDR), established by the Superfund law, is its community outreach and support function. Community right-to-know concepts facilitate involvement in developing solutions to environmental problems that are based in communities. Communities can receive grants to hire their own experts and support community-based organizations.

In occupational health, workers have a comparable right to know, which has been embodied in regulations and has led to availability of material safety data sheets (MSDSs) in the workplace. Right-to-know principles have also been

incorporated into community-based participatory epidemiologic research and proposals that a health impact assessment be performed whenever a project requires an environmental impact assessment.

Because of the Internet and the World Wide Web, data and information that were previously only available to scientists and environmental health professionals are now available to everyone. Community members can use the Internet to locate people in other communities who are facing similar challenges. In addition, the Internet facilitates communication and exchange of opinion. Personal communication tools, such as Facebook, Twitter, and blogs, facilitate real-time, interactive communication.

The Internet has also provided a means for government agencies to provide data for community use, initially via static Web pages, such as those of the EPA Toxics Release Inventory (TRI), which became a major source for communities to learn of potential hazardous exposure and developed interactive data query, graphics, and mapping functions. While unfettered access to raw data and a variety of differing interpretations of those data is the hallmark of the Internet, seldom is there a consensus of opinion upon which people can rely and act. Thus, people searching for information on the Internet often come away with more questions than answers. As a result, communities often turn to government agencies for help.

The National Center for Environmental Health at the Centers for Disease Control and Prevention (CDC) has formed a strong partnership with state and local health departments and the EPA to create Environmental Public Health Tracking Programs. These programs establish trusted Web sites that link environmental and health data. People can not only access information at these Web sites but can also manipulate data to create tables and maps personalized to meet their needs.

Communities often are challenged in converting their newly obtained information into action. Before, obtaining information was the bottleneck; now, it is finding someone to interpret data and respond.

Over 40 states have followed the federal example splitting environmental law enforcement away from public health agencies. Therefore, communities interact with environmental regulatory agencies before contacting public health agencies. The public health system, however, remains the primary resource to assist communities in responding to environmental health concerns, especially when enforcement of environmental laws and regulations has not resolved a problem.[1]

In most states, public health service delivery is a tiered system, usually organized around geopolitical boundaries—city, county, region, and state.[2] Typically, a county or large city health department provides primary services. More specialized services are most often provided by a secondary system at the regional or state level. In some instances, the state health department may provide direct services when local agencies cannot. Environmental health at the local level is provided by public health sanitarians, environmental health specialists, and public health nurses with generalist skills; larger agencies may also employ environmental epidemiologists.

Core local activities include investigating infectious disease outbreaks, indoor air complaints, private well or septic system concerns, and factory emissions. Larger county, city, and state health departments usually are able to employ staff members with more specialized skills and training and deploy more sophisticated equipment and laboratory support. The community and local health care practitioners turn to this interrelated system when they have environmental heath concerns that require action.

Responding to community environmental health concerns is labor and resource intensive, not only to perform studies but also to interpret and communicate results and risks so the public can understand them. The funding and staffing of environmental health agencies has not kept pace with the exponentially expanding access to information on the World Wide Web and increasing public awareness of environmental threats to health.

Environmental health practice needs to be both proactive and reactive. However, it has been increasingly difficult to be proactive with the number of public demands quickly growing to react to events and public concerns—often outpacing response capacity. When local personal resources are insufficient, state personnel

and resources need to augment the response. If state personal and resources are overextended—as can happen when responding to a natural disaster, such as a flood, tornado, or hurricane, the state can request assistance from the CDC, which can rapidly deploy field epidemiologists and other environmental health specialists to support the state and local response.

Community concern may begin with *(a)* a newly identified exposure and fear that adverse health effects may result; or *(b)* the perception that some disease may be occurring more frequently than expected because of a suspected exposure. The latter situation may lead to an investigation of a disease cluster.[3] Regardless of whether the initial request for assistance has begun as an exposure concern or a disease concern, when a community suspects the cause is environmental, an environmental health program often leads the investigation.

A fundamental principle of public health is the prevention of disease by avoiding exposures to harmful levels of toxic exposures in the environment. Despite a generally proactive, preventive approach, environmental health agencies typically respond to situations only after hazardous exposures are discovered. Sometimes, environmental health agencies need to address the unintended hazardous consequences of an emergency response to a chemical spill or "accident." Environmental agencies also frequently address long-term exposure of the public to contaminants that are discovered in groundwater, surface water, and surface soils.

EXPOSURE EVALUATION

When the public is known or suspected to be exposed to hazardous chemicals, environmental health agencies must determine the levels of exposures. Exposure evaluations rely on either environmental screening or sampling data. If a chemical spill occurs, the first responders arriving at the scene are typically firefighters or members of hazardous-waste management teams. With professional judgment and field-chemical screening equipment, they determine the level of hazards and actions needed to control them. When environmental health agencies learn that a neighborhood has been contaminated with chemicals from a dumpsite, laboratory tests of environmental media, such as drinking water, soils, sediments, indoor air, or even fish tissue, can determine levels of contaminants. Unfortunately, high costs of laboratory tests do not allow environmental samples to be randomly collected everywhere but force environmental investigations to focus only on places where contamination is suspected. Even when contamination is discovered in environmental media, an evaluation is needed to determine whether people are being exposed and the frequency, duration, and level of their exposure. An exposure evaluation must examine all possible exposure pathways.

RISK ASSESSMENT

When people are exposed to contaminated soil or drinking water, a risk assessment must be performed to determine whether the exposure represents an unacceptable health risk and whether an intervention is needed (Fig. 35-1). A risk assessment examines the exposure, empirical toxicological data of the relevant contaminants, and potentially sensitive populations who may be exposed. For some media, such as groundwater, risk assessments are fairly straightforward because state and federal agencies have established drinking-water standards for many chemicals. These standards take into account children and other sensitive people, and they assume that people ingest 2 liters of contaminated water daily for a lifetime. However, for contaminated soil there is no similar standard, but rather a range of guidelines. These guidelines require investigators to first establish *(a)* how often the public comes in contact with affected soils, *(b)* whether the area is residential or industrial, and *(c)* whether children are visiting the area. For ambient or indoor air, occupational guidelines are suitable for a work setting, but not appropriate for a residential or a commercial setting where occupational health safety practices are not being implemented. The contaminant of concern is often a substance that does not have an exposure guideline for drinking water, residential soil, or workplace air—making the conduct of a risk assessment even more challenging.

Figure 35-1. Holding public meetings with environmental and public health experts is essential to answering the community's questions and concerns. (Courtesy of Wisconsin Division of Public Health.)

EXPOSURE PREVENTION AND INTERVENTION

Once it has been determined that the public is either likely to have, or has, a high risk of being exposed to an unacceptable level of chemical contaminants, actions are needed to prevent or halt the exposure. Interim actions, such as providing bottled water or fencing a contaminated abandoned property, may be sufficient to halt exposures. Bottled water, however, may not be adequate if there are high concentrations in tap water of an organic solvent that can be released to indoor air by washing dishes or showering, and if residents may be exposed by breathing contaminated air. Each exposure intervention must consider and rule out all potential exposure pathways that may exist.

EVACUATION

If a highly contaminated area is discovered in a residential area or a chemical spill is spreading a toxic vapor cloud, the only appropriate action is the relocation or evacuation of those potentially exposed. Evacuation or relocation is costly and stressful for both evacuees and responders. While decisions to evacuate are carefully and thoughtfully made, this is often a time-critical decision made under duress by the responders. Evacuation decisions incorporate a buffer zone that is protective of public safety for those beyond the impacted area. This area can be expanded if the incident worsens or if risk of an explosion or spread of chemical contamination increases. Sometimes, if an airborne chemical plume is quickly dispersing, people in the process of evacuation can receive a significant exposure. In such instances, implementing a "shelter in place" is the best action.

REENTRY

Once an incident is controlled and a cleanup is being planned, decisions must be made about under what conditions it will be safe for people to return to their homes and workplace. These decisions must determine an acceptable level of contamination since total removal of contaminants is usually not feasible. In addition, if there

has been an industrial fire, air deposition of burning by-products onto adjacent properties may affect reentry decisions (Fig. 35-2).

PUBLIC INFORMATION AND RISK COMMUNICATION

As soon as environmental health agencies become involved with a chemical spill or discovery of environmental contamination, they must immediately prepare for and respond to community health questions and concerns about the situation. The risk communication skills of health agency workers are important in effectively conveying health information to the public throughout the incident. (See Chapter 29.)

FOLLOW-UP ACTIONS: HEALTH STUDIES, BIOLOGICAL MONITORING, AND DISEASE TRACKING

If the public was exposed to contaminants at a level that poses an unacceptable health risk, environmental health agencies determine what additional actions need to be taken. For certain chemicals, immediate biological monitoring of exposure can determine whether people have been significantly exposed. For some substances, medical monitoring (screening) may detect previously unrecognized illness among those exposed. Such medical monitoring can be part of longitudinal studies that determine rates of certain symptoms or illnesses over time.[4] Such studies can be expensive and may not be conclusive.

Following are three cases of environmental health actions. Federal, state, and local government agencies can now widely disseminate reports of these actions via the Internet. These reports, though not in peer-reviewed scientific journals, comprise the "gray literature."

Case 1: Mercury Spill at a High School

At 1:15 p.m. on a Friday in January, elemental metallic mercury was spilled from an open-ended glass manometer at the back of a chemistry classroom in a high school. A student attached one end of a rubber hose to the manometer and the other to a pressurized air port. The student then opened the air port, which forced air into the manometer and blew most of the mercury

Figure 35-2. This waste site contains the remains from automobile wrecks and potentially contaminates the soil with heavy metals and other contaminants. (Courtesy of Wisconsin Division of Public Health.)

from the manometer. An estimated 4 tablespoons (60 cc) of mercury spilled onto nearby tables and counters and the floor. Mercury particles were attached to ceiling tiles directly above the manometer. Shortly after the spill, it was discovered by school personnel, who quickly reported it to school administrators, who implemented the school district's emergency procedure plan. Responding to a request by school-district officials, teams of firefighters and hazardous-materials workers arrived at the school. After consulting with emergency responders, the school district hired a hazardous waste cleanup contractor. Also responding to the incident were the Wisconsin Department of Natural Resources, the Wisconsin Department of Health and Family Services (DHFS), and the County Department of Human Services (DCHS).

Immediately after the spill was discovered, the high school science wing was cleared of students and faculty and staff members, and then secured. Soon after, the entire school was locked down and, to prevent the potential spread of mercury contamination, all students and faculty and staff members were not allowed to leave the building until they were screened for mercury contamination. The 70 students who were in chemistry class during or immediately after the spill were sent to locker rooms, where they removed their clothes and shoes and placed them in plastic bags, showered, and dressed in chemically resistant suits. These students were then transported to another school, where they were picked up by their parents and taken home. Their bagged clothes and shoes were transported to the community center. The next day, students and their parents were allowed to pick up their bags of clothes and shoes if it was determined that these bags were free of mercury, based on testing with a mercury analyzer that DHFS had recently obtained. Seven bags had mercury vapor levels so high that their bags of clothes and shoes had to be disposed—in an appropriate manner.

Students and faculty and staff members who had not been in or near the chemistry classroom where the spill occurred were only required to have their shoes screened for mercury before they were allowed to leave the building, to ensure that mercury was not being tracked or carried around the school. Only one person, a science teacher who was in the chemistry classroom when the spill occurred, had elevated mercury vapors coming from her shoes, which were collected and properly disposed. Repeated screening of her clothes did not detect elevated mercury vapors.

The hazardous waste contractors used mercury field-screening meters to screen all indoor areas of the school for elemental mercury vapors. They observed beads of elemental mercury in the chemistry classroom where the mercury had been spilled. They also found elevated mercury vapors in this classroom and in the adjacent hallway and science office. Mercury vapor concentrations at the classroom doorway ranged between 2 and 3 $\mu g/m^3$, and where the spill had occurred between 14 to 22 $\mu g/m^3$. All areas of the school were double-checked to confirm that mercury was elevated only inside of, and very near, the chemistry classroom. The contractors then collected and removed all visible mercury from the classroom and used cleanup methods to appropriately remove mercury residues from the classroom, the adjacent hallway, and selected nearby rooms. They thoroughly ventilated the classroom and science wing. As a precautionary measure, they wet-mopped all flooring throughout the school with a special amalgamating solution to ensure the removal of any mercury residues that may have previously escaped detection. The cleanup staff worked through the night and completed the cleanup by 3:00 p.m. the day after the spill.

Application of Existing Public Health Guidelines and Protocols

ATSDR defines a minimal risk level (MRL) as an "estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects."[5] The ATSDR chronic inhalation MRL for mercury vapor in air is 0.2 $\mu g/m^3$.

Students and faculty and staff members who had been in the chemistry classroom when the spill occurred had inhaled mercury vapors only for a short time. Considering the levels of mercury they had inhaled, it was not likely that this exposure caused any harmful health effects. As a result, it was determined that this exposure posed no apparent human health hazard.

The cleanup goals were ATSDR's suggested action levels for indoor mercury vapors in homes

or businesses with indoor gas regulators. ATSDR recommends that *(a)* after a spill, mercury vapor levels in the breathing zone of a home not exceed 1.0 μg/m^3, and *(b)* at or below this level is acceptable for reoccupancy of any structure. Exceeding the action level of 1 μg/m^3 prompts the need for cleanup or other remedial actions to reduce exposures; exceeding 10 μg/m^3 prompts isolation of residents from exposure and actions to remediate the spill. ATSDR also recommends an action level of 10 μg/m^3 when testing air from a plastic bag in which mercury-contaminated clothes have been placed; in such instances, the bag and its contents must be taken from the owner and appropriately disposed.

Biomonitoring Recommendations and Results

As a further precautionary measure, DHFS and DCHS offered laboratory screening of urine for mercury. Forty-two urine samples were brought to the emergency department at the local hospital by the following Tuesday and sent to the state laboratory for testing. Only one of the urine samples had a mercury level above the detection limit—6 μg/L—but it was still within the "acceptable range." DCHS mailed test results and interpretations to students' parents on Thursday, 6 days after the spill.

Air Sampling before Reentry

As a follow-up activity, air samples were collected from selected locations to provide a final, confirmatory evaluation. A third-party contractor had collected air samples in the high school the day after the spill, by drawing air through glass tubes filled with appropriate sampling media. Tests of these samples did not detect any mercury vapors.

DHFS provided these data to school district officials 2 days after the spill. It had concluded that the data demonstrated that the cleanup had effectively removed all of the spilled mercury. The officials then decided to reopen the high school the next day.

Risk Communication

Communication among all involved parties was critically important throughout the response to the spill. DHFS staff members used their expertise in risk communication to assist school administrators in developing and effectively communicating appropriate health messages. They also assisted the school superintendent and the county health officer in drafting written communications and preparing for a press conference on the evening after the spill to share with parents and media representatives information concerning the situation and prudent actions being taken to address it. They wrote to the superintendent 2 days after the spill to interpret air sampling results and to state that the school was ready to open the next day. They also wrote letters to students' parents the day after the school reopened, informing them why mercury spills are of concern and explaining that low background levels of mercury vapor found in all bags of clothes returned to the students meant that there was no further health concern. They assisted the DCHS health officer in writing letters to parents that interpreted mercury levels in their children's urine specimens. At the school board meeting on the night the school reopened, they presented the public health implications of the incident and answered questions from board members and the public. Since the meeting was televised, they used the opportunity to promote proper removal and disposal of mercury sources, such as oral thermometers and thermostats, from homes and workplaces.

Representatives of the school district, the responding public health agencies (DHFS and DCHS), the hospital emergency department, and the Wisconsin Poison Center all collaborated in the response to the incident. During the response, the poison center received approximately 50 telephone calls from concerned parents. No callers described adverse health effects associated with acute or chronic mercury exposure. School officials and DCHS personnel informed parents that they should seek medical care for their children from their physicians or a hospital emergency department if their children experienced neurologic or other symptoms related to mercury exposure. The emergency department received several inquiries, but it did not diagnose any adverse health effects due to mercury.

Commentary

This case is a traditional "reactive response" to an acute exposure to mercury. Unfortunately, despite replacement programs and regulatory programs to ban use of elemental mercury, such

spills remain common and create more cross-contamination greater than initially apparent. Unusual in this case was the availability of portable direct-read laboratory equipment that allowed rapid exposure assessment and resolution of community concerns. Because of the recognized utility such equipment is gradually becoming standard at the community level. A critical activity involved communicating with all impacted groups—from managers to students.

Case 2: Health Concern at a Hazardous-Waste Remediation Site[6]

How the Problem Began and Progressed

During the summer of 1997, some people residing at a former barrel company in South Milwaukee complained about odors coming from onsite soil treatment activities. Some reported experiencing symptoms when odors were strong. Residents requested that the Wisconsin Department of Health Services (DHS) conduct a public health assessment of site conditions to *(a)* quantify exposure to any contaminants being released and *(b)* evaluate possible adverse health effects.

When the company operated from 1940 to 1964, waste materials—mostly paint-related—were disposed into multiple pits located near the edge of a bluff overlooking Lake Michigan. Leachate from the pits stained the bluff edge and drained to the beach. The site was identified for detailed investigation and remediation. By 1996, condominiums, apartments, and single-family homes had been built over the property.

Three apartment complexes and two condominium complexes—with about 195 residences—were within 100 yards of the property. Another four-condominium complex—with 80 units built in 1968—was on the southwestern corner of the property. Two of these condominium buildings had been built very close to the foundation of one of the previous industrial buildings. A 1952 aerial photograph demonstrated that the 80-unit condominiums were located where drums were once stored. Yet another 8-unit condominium, built in 1982, was located north of these condominiums. Finally, a 24-unit apartment complex, built in 1991, was on the northwest corner of the property. In addition to the 195 apartments adjacent to the property, approximately 210 single-family dwellings were within 300 yards of the property. DHFS estimated that altogether about 1,000 people lived within 300 yards of the property.

Chemical analysis from 1995 of subsurface soil samples revealed the presence of elevated levels of inorganic chemicals, volatile organic compounds (VOCs), and semi-volatile organic compounds (SVOCs). The highest lead level detected was 45,700 mg/kg. In one sample, total VOCs exceeded 7% of soil by weight. The highest level of xylenes found was 36,000 mg/kg.

In 1996, as part of the remediation plan, soils were excavated from these disposal pits, placed on a prepared clay pad, covered with plastic sheeting, and stockpiled on the property. In May 1997, the EPA approved a request for onsite treatment and stabilization of the soil by adding Portland cement to minimize the leaching of lead and to facilitate disposal of the soil as nonhazardous waste.

Soil treatment/stabilization and removal activities were initiated in June 1997 and were scheduled to last about 3 weeks. A canvas tent was erected to house the soil-mixing equipment and minimize release of fugitive dust and VOCs. Soil piles were uncovered and transferred with heavy equipment to a screening device (located outside the mixing tent), which sifted debris from the soil. Soil piles were covered when operations ceased. However, a high water content was found in stockpiled soils, along with much debris, considerably slowing the soil-mixing operation. These and other logistical issues resulted in extending treatment and stabilization activities for 3 months beyond the planned completion date, until September 1997.

Organic vapors from the property were released into the air by onsite soil treatment and stabilization and removal activities. Two environmental consultants used direct-readout instruments to regularly monitor air quality at and near the treatment-area fence (Fig. 35-3). The air monitoring equipment that they used provided information on levels of airborne particulates and total VOCs, percentage of oxygen in the air, and proximity to the lower explosive limit. An organic vapor monitor (OVM) was primarily used to make direct readings of the VOC concentration in air. During soil-treatment activities, OVM readings were reported as high as 352 ppm within the canvas tent, although

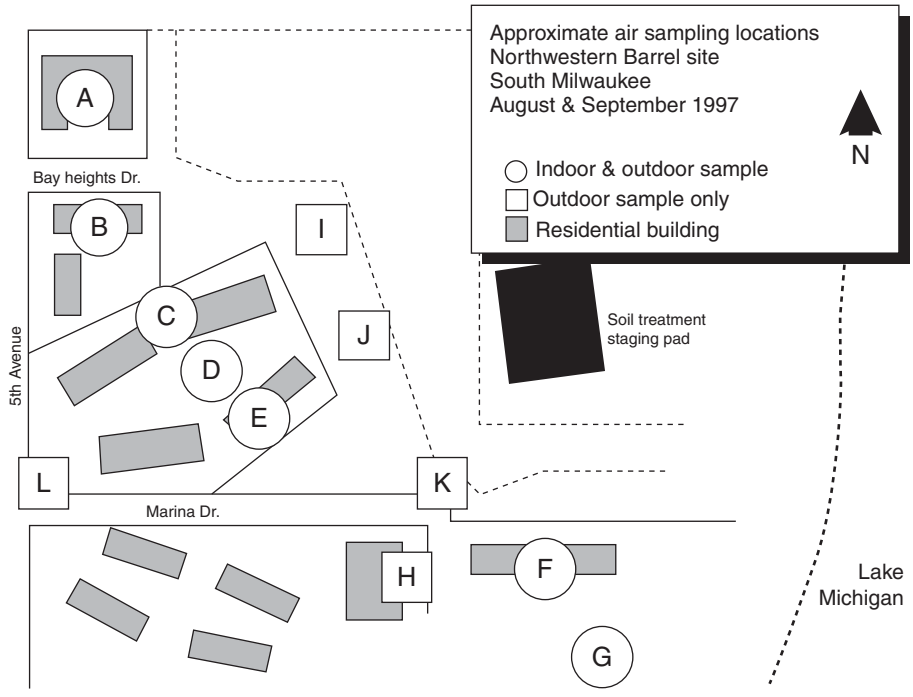

Figure 35-3. Approximate air sampling locations at the Northwestern Barrel site, South Milwaukee, 1997. (Courtesy of Wisconsin Division of Public Health.)

these values often ranged between 30 and 150 ppm. The highest outdoor VOC level of 147 ppm was observed immediately outside the tent. On June 23, total VOCs were measured inside the tent at 157 ppm. Later that day, contractors used compound-specific, hand-held, colormetric Draeger tubes to test air inside the tent and found benzene at 6 ppm (TLV = 0.5 ppm), styrene at 15 ppm (TLV = 20 ppm), and toluene at 125 ppm (TLV = 50 ppm). Onsite workers used appropriate personal protective equipment (PPE) that permitted working in the presence of these vapors. Along the fence line, total VOCs were usually not measurable with the OVM.

These direct readings indicated that fence-line air concentrations of VOCs and particulates were not at acutely hazardous levels. Direct readout meters, such as OVMs, are useful tools when field screening for VOCs. However, there are limitations to such air monitoring instrumentation:

- OVM instruments are not compound specific.
- They may not accurately measure airborne VOCs at concentrations less than 1 ppm.
- OVM use at the property required daily calibration, in accordance with the manufacturer's guidelines.
- Evidence of OVM calibration was to be written on the daily fugitive air emissions monitoring logs, but calibration notations were missing on 31 daily monitoring logs for site activities between June 5 and September 24, 1997.
- Air was monitored only by the environmental consultants during weekdays, but not during evenings or weekends, when there was no work being done at the property.

Listening to and Communicating with the Impacted Community

On certain days between June and September 1997, a number of residents complained about bad odors coming from the soil treatment area at the property, initially on June 29. On that day, the Wisconsin Department of Natural Resources

(DNR) observed that air was stagnant and issued an ozone alert for southeastern Wisconsin. Complaints from nearby residents were also noted in the site air-emissions monitoring log on July 24, July 31, and September 15. Typically, residents complained about air quality when there were light winds from the east or northeast, which carried vapors from the treatment area toward condominiums and apartments, located less than 100 yards to the west.

DHS responded to citizen air-quality complaints by visiting the property several times. During a July 11 visit, a light easterly breeze was blowing from Lake Michigan, across the treatment area, and toward several of the nearby apartment and condominium buildings. Residents again complained about a paint-like odor, which was evident to DHS personnel as they walked around the apartment and condominium areas. VOCs were detected inside and immediately outside the treatment tent, but not on the perimeter of the treatment area.

Some residents reported adverse health effects, including headache, sore throat, lethargy, and burning eyes, especially when they noticed strong odors coming from the treatment area. One resident recalled that he smelled similar odors and experienced similar symptoms when the two pit areas were excavated in 1996. In October 1997, one resident informed DHS that she had developed a respiratory problem shortly after moving into a nearby apartment in mid-June that was diagnosed as asthma. She reported that the severity of her asthma symptoms was temporally associated with odors coming from the treatment area. She was hospitalized twice for asthma during July. She reported that her asthma improved when odors subsided and after onsite soil treatment ended in September.

Investigating Exposure

As a result of reported symptoms, DHS initiated an exposure investigation, evaluating VOC blood levels of residents who complained of poor air quality and symptoms. Before selecting survey participants, residents were asked questions to identify other possible VOC exposures that might confound interpretation of their blood VOC levels. DHS then conducted VOC analysis of air samples collected in and around selected residents' homes, between their homes and the treatment area, and along the perimeter of the treatment area. DHS offered blood VOC testing to approximately 20 people who lived near the property and had complained about odors coming from the property, three of whom agreed to provide blood samples.

Selecting Study Subjects

DHS contacted residents who had air-quality complaints about the property to identify those who experienced health effects associated with airborne contaminants from the property. Participants were informed that blood samples would be collected when wind conditions carried VOC vapors from the treatment area toward their residences. Nearby residents who provided blood samples were selected because *(a)* they planned to be at home for the duration of the day, and *(b)* they did not report a possible alternative confounding exposure to solvents, such as pumping gasoline, smoking cigarettes, or using household solvents.

Blood samples were collected on two separate days approximately 1 week apart. Subjects were interviewed when their blood samples were collected. On August 28, two of the three participants reported they smelled odors coming from the soil treatment area—one said it smelled like paint thinner, and the other, like sweet perfume with a burnt characteristic. Both characterized the odors as weaker than usual. While neither of these people said they had an odor-related illness on August 28, one said he often had headaches on the afternoons of days when he noticed odors coming from the treatment area all day long. The third subject reported often having a sore throat, which she attributed to air coming from the treatment area.

On September 3, a second set of samples was collected from two of the participants when one reported an odor coming from the soil treatment area. This person described the odor as similar to a paint thinner, although weaker than usual. These people did not report any odor-related symptoms on September 3. All of these individuals stated that they were nonsmokers. No subjects reported using any products or materials containing VOCs on the days that samples were obtained. Blood results from these three residents were compared with VOC blood concentrations of non-occupationally exposed

Table 35-1. Residential Blood VOC Sampling (All blood concentrations in μg/L)

			NHANES III BLOOD VOC CONCENTRATIONS			
	Observed Average Blood VOC Concentration		Nonsmokers Only		Smokers and Nonsmokers	
Subject and Chemical	8/28	9/3	50th Percentile (Median)	95th Percentile	50th Percentile (Median)	95th Percentile
Subject 1: Sample Location C						
Benzene	0.04	0.03	0.05	0.09	0.06	0.48
Ethylbenzene	0.04	0.03	0.05	0.23	0.06	0.25
Styrene	0.03	0.01	0.03	0.08	0.04	0.18
Toluene	0.08	0.27	0.21	1.00	0.28	1.50
Xylene (total)	0.19	0.12	0.25	0.97	0.30	1.08
Subject 2: Sample Location C						
Benzene	0.04		0.05	0.09	0.06	0.48
Ethylbenzene	0.01		0.05	0.23	0.06	0.25
Styrene	0.01		0.03	0.08	0.04	0.18
Toluene	0.05		0.21	1.00	0.28	1.50
Xylene (total)	0.09		0.25	0.97	0.30	1.08
Subject 3: Sample Location F						
Benzene	0.03	0.03	0.05	0.09	0.06	0.48
Ethylbenzene	0.12	0.29*	0.05	0.23	0.06	0.25
Styrene	0.05	0.13*	0.03	0.08	0.04	0.18
Toluene	0.10	0.17	0.21	1.00	0.28	1.50
Xylene (total)	0.61	1.68*	0.25	0.97	0.30	1.08

*Exceeds NHANES III 95th percentile for nonsmokers. Outlined blood VOC concentrations exceed NHANES III 95th percentile for smokers and nonsmokers combined.

NHANES III, Third National Health and Nutrition Examination Survey; VOC, volatile organic compound.

participants of the Third National Health and Nutrition Examination Survey (NHANES III) (Table 35-1).

Eliciting Broader Community Health Concerns

As a follow-up, DHS, in November 1997, sent letters and surveys to approximately 240 nearby households, asking residents about their health concerns related to the 1997 soil cleanup at the property. Of the 59 households (25%) that responded, 16 reported illness or symptoms that they thought might be related to contaminants at the site. Generally, people reported combinations of 12 respiratory symptoms, including nose and throat irritation, asthma, nosebleed, increased respiratory infections, wheezing, and coughing. Eight people reported severe or frequent headaches. All respondents reported at least one of the following symptoms: skin rash, lightheadedness, loss of appetite, weight gain, nausea, loss of energy, arthritis, muscle aches, weight loss, and discomfort. Most people who felt ill believed their symptoms were caused by exposure to airborne chemicals. To a lesser extent, people were concerned about soil contact while they were gardening or walking their dogs or their children were playing in soil and water. Twenty people reported outdoor air-quality conditions associated with the property that they believed increased their sensitivity to chemicals. They reported chronic illnesses

affecting their lungs, liver, sinuses, eyes, circulatory system, and kidneys. Two women were concerned about respiratory exposures they had had during pregnancy.

Communication of Public Health Conclusions

Ethylbenzene and xylene levels were higher in the September 3 air samples from Location F (about 100 yards from the soil treatment area) and the fence line than in the August 28 samples. Both fence line samples were south of the treatment area and in between Location F and the treatment area.

Indoor and outdoor air samples taken from residences next to the treatment area showed the presence of relatively low concentrations of site-related VOCs and SVOCs. None of these substances were found in air at concentrations known to cause illness with short- or intermediate-term exposures. The VOCs with the highest offsite concentrations were ethylbenzene (40 ppbv), styrene (85 ppbv), toluene (46 ppbv), and total xylenes (179 ppbv). None of these aromatic VOCs were found at a concentration that exceeded an acute or intermediate minimal risk level of ATSDR.

Nearby residents were exposed to slightly elevated levels of several airborne VOCs during soil treatment at the property. Analysis of blood samples collected from three residents showed that one in Location F on September 3 had high blood concentrations of ethylbenzene, styrene, and xylene. Between August 28 and September 3, his blood concentrations of ethylbenzene and xylene seemed to correlate with indoor and outdoor air concentrations of these substances at Location F.

Commentary

This case is more typical of site-specific community concerns and reflects the problems that can develop from evolving land use in communities. This type of response is typical of the investigations performed by states that are supported by ATSDR funding for site investigations. Biomonitoring was used to demonstrate that exposures were occurring at levels atypical for the general population—a finding that helped determine that the source of the exposure was offsite.

Case 3: Health Complaints from Combustion Emissions Produced by Residential Outdoor Wood-Fired Boilers

Background

Chronic exposure to wood smoke from sources such as wood burning in fireplaces and furnaces, leaf burning, and refuse burning has long been a recognized health hazard and a cause of community complaints. Reactive chemicals contained in smoke particulates and gases are more than just a nuisance, contributing to respiratory and cardiovascular problems. Individuals with asthma and other respiratory sensitivities are especially impacted by environmental smoke. The utilization of wood for residential heating has always been popular, especially in rural areas with a readily available source of inexpensive firewood. The rapid increase in costs of petroleum-based fuel sources along with the appeal of using renewable resources has accelerated the use of wood as a residential fuel. However, an impediment has been the increased risk of house and chimney fires, which has resulted in elevated insurance costs associated with indoor wood heat. The outdoor wood-fired boiler (OWB) is a free-standing heating device with a shed-like exterior containing a metal combustion chamber for a wood fire, which is surrounded by a water tank; the heated water is piped to a nearby building (home) to supply heat. The OWB is operated throughout each day during the heating season. Unlike indoor wood stoves, OWBs are not regulated by mandatory federal emissions standards, although they produce approximately 12 times more particulate smoke than an EPA-certified indoor wood stove. Because OWBs are separated from the home, they do not result in increased home fire insurance. These characteristics have led to a substantial increase in their sales.[7,8] Wisconsin is second nationally in OWB use; more than 27,000 OWBs were sold there between 1990 and 2006. Their popularity is expected to continue.

Public Health Implications

Outdoor wood-fired boilers typically have short stacks that are lower than the rooflines of surrounding structures, so emissions do not disperse as well as emissions from typical indoor

wood stoves, creating a greater potential for localization of harmful emissions in and around adjacent residences.[9,10] Expansion of OWB use into rural developments with greater housing density than the usual isolated "farm house" has led to more neighbor complaints. Few communities have ordinances regulating OWB use and OWBs are not regulated by federal emissions standards. Thus, in most communities, OWBs are invisible until a complaint arises. Between 2005 and mid-2009, local health departments in Wisconsin received at least 150 complaints and the state Bureau of Environmental and Occupational Health (BEOH) has assisted with chronic smoke exposure complaints associated with OWBs.

In addition to the involvement of health officials, complaints are often directed through fire departments, the state environmental agency, and local town officials. The increasing demands on limited state and local environmental health resources led to a systematic impact assessment and development of a remediation strategy.

Investigating Exposure and Quantifying Impact

Modeling of OWB emissions showed that emissions are concentrated close to OWBs[11]; thus neighbors—rather than whole communities—are most likely to be exposed to resultant air contaminants. This finding is consistent with complaints received. Geographic information systems (GIS) can spatially identify neighbors of OWBs for public health investigations. Environmental health professionals from three rural Wisconsin counties were enlisted to perform systematic OWB visual surveillance during their regular travels through residential areas in towns, villages, and unincorporated areas. Outdoor wood-fired boilers identified were located using GIS. Tabular tax assessment data, containing postal addresses, and geospatial tax-parcel or address-point data were combined with the geospatial OWB data to identify the nearest neighbors of the OWBs. Local responses to OWBs have usually involved ordinances that require a minimum distance to the nearest neighbor—often 500 feet. Wisconsin was contemplating recommending a model ordinance that included that distance. Thus, an "exposed" neighbor population within 500 feet of an OWB was chosen and a comparison population 1,000 to 3,000 feet distant. In some instances, OWBs were within 500 feet of each other, creating the potential for overlapping exposures. OWB groups were further defined as a single OWB farther than 500 feet from the nearest OWB, or two or more OWBs within 500 feet of each other or within 500 feet of a common OWB.

A self-administered, mailed questionnaire requested home characteristics, family demographics, and medical history, including symptoms. It was sent to *(a)* residential property owners with mailing addresses in the county within 500 feet of an OWB, and *(b)* an equal number of owners of randomly selected comparison properties between 1,000 and 3,000 feet of an OWB. An enclosed letter stated that an investigation was being conducted to evaluate the health effects from consistent sources of wood smoke, but it did not specifically identify OWBs.

Compiling and Interpreting Study Results

Of 124 OWBs located, 57 (46%) had two or more residences within 100 feet of the OWB. Stack height was inadequate—lower than 2 feet above the closest neighbor's roof line—for 58% of those where a stack height was reported. Analysis with a multivariate logistic regression model found that living 500 feet and downwind of an OWB was associated with a higher rate of symptoms possibly related to an OWB, compared to living between 1,000 and 3,000 feet of an OWB. This analysis also found that a history of asthma, allergies, or heart disease was associated with living within 500 feet of an OWB. When those living upwind, but within 500 feet of an OWB, were excluded from the analysis, a statistically significant linear relationship was found between a score of symptoms possibly related to an OWB and living in proximity to an OWB.

Public Health Action

In this investigation, cooperation between local and state health departments was vital to locating OWBs, obtaining access to county GIS land parcel data, and applying epidemiologic and statistical expertise that was available only at the state agency. Integration of GIS and epidemiology was critical to the success of the investigation—a type

of integration that holds promise for other applications in environmental epidemiology, although agreements on standardization and use of data will be necessary.

This investigation concluded that direction and distance from the OWB mattered. Persons living downwind and within 500 feet of OWBs experienced significantly more symptoms than others living in the community. Living further away from an OWB was protective. These findings were used to support the adoption of a model local ordinance that required a distance of at least 500 feet between an OWB and the nearest residence. It is difficult to separate the nuisance of the odor of wood smoke from its adverse health effects. Analyses produced consistent results when individuals who smelled wood smoke in their homes were excluded, suggesting that the odor of wood smoke was not the trigger for symptoms.

Commentary

This case is a blend of responding to citizen complaints, recognizing the growing trend in the spread of a technology with the potential to cause harm if not managed systematically. This case reflects problems that can develop from unregulated spread of popular technologies.

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FURTHER READING

ATSDR Public Health Assessment Tutorial. Available at: http://www.atsdr.cdc.gov/training/public-health-assessment-overview/html/.
Provides an overview of the health assessment approach used by states and the federal Agency for Toxic Substances and Disease Registry when addressing known or suspected environmental contamination that the public may be exposed to.

CDC/NCEH National Environmental Public Health Tracking Network Portal. Available at: http://ephtracking.cdc.gov/showHome.action
The Tracking Network is a dynamic Web-based tool that tracks and reports environmental hazards and the health problems that may be related to them. It allows scientists, health professionals, and members of the public to see where these hazards and health problems are occurring and how they are changing over time.

USEPA Exposure Assessment Guidance Overview. Available at: http://www.epa.gov/opptintr/exposure/docs/exposurep.htm
A systematic approach to evaluating when people are exposed to chemical contamination.

Morgan MT. Environmental health (3rd ed.). Belmont, CA: Wadsworth/Thomson Learning, 2003.
A comprehensive summary of the practices used in protecting the public environmental health hazards.

Burke R. Hazardous materials chemistry for emergency responders: street chemistry. Boca Raton, FL: CRC Press, 1997.
Useful chemical information and case studies of hazardous material events.

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Addressing Health and Safety Hazards in Specific Industries: Agriculture, Construction, and Health Care

Sherry L. Baron, Andrea L. Steege, Laura S. Welch, and Jane A. Lipscomb

An immigrant farmworker, age 20, was asked to place soil around the perimeter of a tarp covering a field that had been fumigated by injecting methyl bromide gas into the soil. The field was to be used to plant strawberries. It was his first day of work and he was eager to prove that he was a good worker. The ambient temperature was 104°F. After about 4 hours of work, he began to feel nauseous and dizzy. A co-worker told him to drink more water and to take a rest, but he continued to work because he was afraid he would not finish the task. After another hour, he was too dizzy to continue working. He was taken to a clinic in town. At the clinic, the physician asked the worker's supervisor some questions and, after looking up the toxicity of methyl bromide, learned that heat would hasten the volatilization of the gas from the soil. Except for a slightly increased heart rate, the worker's physical examination was normal. Blood tests showed slight electrolyte abnormalities. The doctor diagnosed the worker as having either mild methyl bromide poisoning or heat exhaustion. The doctor called the closest major laboratory, several hours away by car, and found that it would take at least 1 week to determine the worker's blood methyl bromide level. He called the regional poison control center, which told him that there was no specific treatment for mild methyl bromide intoxication. He treated the worker for mild heat exhaustion and had the health educator explain to the worker, in Spanish, the need for frequent rest breaks and good hydration when working in extreme heat and about ways of recognizing and preventing pesticide exposure.*

Some industries pose especially complex challenges for health professionals due to the wide variability of exposures and the high mobility of workers. In these industries, where workers are simultaneously exposed to many different hazards and perform a variety of tasks, it can be difficult to determine which hazard or task is responsible for a worker's health problem. Sometimes, as in the above case, multiple exposures may cause the problem, and determining the responsible exposures may be difficult. While knowledge of the health effects of specific hazards is important, health professionals also need to appreciate the complex factors present in

* Although fictitious, this case was derived from the experience of Dr. Rupali Das, director of the Pesticide Illness Surveillance Program at the Occupational Health Branch of the California Department of Public Health.

hazardous work. In this chapter, we describe three hazardous industries—agriculture, construction, and health care—to address some of these complexities.

Agriculture

Sherry L. Baron and Andrea L. Steege

Worldwide, more people work in agriculture than in any other industry, with most engaged in labor-intensive, small-scale subsistence farming. In the United States, although agricultural production is dominated by larger and more mechanized production, farm work remains one of the most labor-intensive and lowest paid occupations. The broad occupational category of farmworkers includes both family farmers who work on their own farms, and hired farmworkers. Although this chapter focuses on the approximately 2.5 million hired farmworkers, owners of family farms face many of the same categories and combinations of hazards.

In 2002 in the United States, 84% of hired crop farmworkers were Hispanic and 79% had been born in Mexico.[1] One-third of foreign-born farmworkers are recent immigrants, who have worked in the United States for 2 years or less; many are living apart from their families and experiencing social isolation that creates additional stress. Farmworkers are younger (average age, 31) than the general workforce and most (79%) are men. Most have very low literacy, which can have significant impact on their ability to read warning labels or understand safety instructions. Only 22% of farmworkers can read and write English well, and more than half have less than an eighth-grade education. For the many who come from rural areas of Mexico, where an indigenous language is spoken, Spanish is their second and English may be their third language.

Forty-two percent of farmworkers face additional stress because of their need to migrate for work and live temporarily away from home, often in crowded and inadequate housing. Hired farmworkers, on average, work in agriculture for only about 8 months of the year. Due to low wages and extended periods of unemployment, the annual family income for more than half of all hired farmworkers is less than $15,000. Since 53% are not legally authorized to work in the United States, they may be vulnerable to abuse and are unlikely to report mistreatment.

In 1960, Edward R. Murrow's classic television documentary, *Harvest of Shame*, shocked viewers by depicting the deplorable working conditions of farmworkers in the United States. Nonetheless, little attention was paid to improving their health and safety conditions until relatively recently. In 1991, the U.S. Surgeon General convened a national meeting on the health of agricultural workers, and subsequently the National Institute for Occupational Safety and Health (NIOSH) established a network of research centers to improve health and safety of family farmers and hired farmworkers. In 1995, NIOSH convened a special panel to make recommendations regarding the priority occupational health problems for hired farmworkers.[2] This panel selected nine priority health outcomes as a focus for future research and intervention, which are listed in Table 36-1, along with heat-related illness because of the recent recognition of its continuing importance. Although the pace of research on agricultural workers has accelerated, these priority health outcomes are still relevant today. The most common of these occupational health problems are discussed in more detail in the sections that follow.

MUSCULOSKELETAL CONDITIONS

From strawberry pickers harvesting crops in a sustained stooped posture to citrus pickers carrying heavy sacks up ladders while reaching for the next orange, farm work is associated with a variety of musculoskeletal disorders (MSDs). In addition, because one-fifth of farmworkers are paid based on the quantity of crops harvested (piece rate), in many work settings there are economic incentives for them to maintain a rapid, sustained work pace. About one-half of all agricultural injuries (Fig. 36-1) requiring time away from work are musculoskeletal injuries, such as sprains, strains, and injuries causing low back pain. To prevent such injuries, some research centers are developing innovative, low-cost methods of improving the ergonomic design of farm work, such as a redesigned tool to carry potted plants (Fig. 36-2). (See also Chapter 27.)

Table 36-1. Selected Hazards, Health Effects, and Control Strategies in Agriculture

Health Effect	Hazard	Control Strategy
Musculoskeletal disorders	Prolonged stooping, heavy lifting, repetitive movements of the upper extremities during planting, pruning, and harvesting	Ergonomic reengineering of tools and workplace; decrease of weight of loads; job rotation among repetitive and nonrepetitive tasks
Pesticide-related conditions	Mixing, loading, and applying pesticides; working in fields recently sprayed with pesticides; aerial drift of pesticides from adjacent fields; exposure to pesticides in living quarters	Substitution with less toxic substances; adequate protective equipment; training on prevention of pesticide exposures; administrative restrictions on working in fields where exposure may occur
Traumatic injuries	Work-related incidents with tractors and other farm equipment; motor vehicle crashes during transport to and from fields; lacerations from sharp tools for cutting and pruning	Use of roll-over protection systems in tractors; training and enforcement of safe use of equipment; transportation vehicles equipped with personal restraint systems; safe cutting tools
Respiratory conditions	Airborne exposure to allergic and irritant substances, either naturally occurring in the soil and crops or due to chemical substances	Substitution with less toxic materials; use of respirators, if indicated; administrative controls to remove sensitized workers from exposure
Dermatitis	Skin contact with allergic and irritant substances, either naturally occurring in the soil and crops or in fertilizers and pesticides	Substitution with less toxic materials; use of gloves and sleeves, if indicated; administrative controls to remove sensitized workers from exposure
Infectious diseases	Inadequate sanitation facilities; exposure to tuberculosis, sexually transmitted diseases, and other infectious diseases due to living arrangement of migrant workers	Improved sanitation facilities; improved housing facilities; improved medical care screening and treatment services
Cancer	Exposure to chemical substances in pesticides and other agricultural products; prolonged sun exposure	Substitution with less hazardous substances; protective clothing and sunscreen; administrative controls to limit exposure
Eye conditions	Exposure to dusty conditions; foreign bodies from plant material penetrating the eye	Use of protective eyewear; dust control
Mental disorders	Long working hours; inadequate pay; social isolation from family and friends	Improved working and housing conditions; availability of mental health services
Heat-related illness	Exposure to hot, humid environments	Educating employers and workers on the hazards of working in hot environments, implementing heat-stress management measures

(*Source*: Adapted from Reference #2 on page 761.)

PESTICIDE-RELATED ILLNESS

Pesticide-related illness refers to a broad group of health outcomes, including dermatitis, eye injuries, respiratory diseases, and cancer. Although many research studies have been conducted on the toxicology and health effects of pesticides, few of these studies have been directed at hired farmworkers. There is no system in the United States to accurately record the national incidence or prevalence of pesticide-related illnesses that occur in the farm sector, although several states collaborate with NIOSH to identify pesticide poisoning cases through the Sentinel Event Notification System for Occupational Risks (SENSOR) pesticides program. (In addition, California, which employs about one-third of all farmworkers in the United States, operates its own reporting system for occupational pesticide intoxications.) Data from these states provide useful information on the nature of farmworkers' exposures to pesticides (Table 36-2). Most overexposures do not occur to those who are applying pesticides, but instead to workers who are inadvertently exposed to pesticides while performing routine farm tasks, such as harvesting and weeding. These overexposures commonly occur when pesticides being sprayed on one field drift into the breathing zone of farmworkers in nearby fields or when workers handle crops

Figure 36-1. Agricultural workers, like this apple picker, face numerous safety hazards. (Photograph by Earl Dotter.)

with pesticide residues.[3] Although less than one-third of cases of pesticide poisoning lead to lost time from work, given the economic insecurity of most farmworkers, it is difficult to determine whether this reflects the affected workers' need to continue working or the mild severity of most cases, or both.

TRAUMATIC INJURIES

Agriculture is considered one of the most hazardous industries for occupational injuries and deaths. Agriculture has an annual occupational fatality rate in the United States comparable to the mining industry, with about 28 fatalities per 100,000 workers. In 2007, the fatality rate in agriculture was almost twice the rate in both construction and transportation.[4] About one-half of all agricultural fatalities occur as a result of transportation incidents, primarily related to tractors. The adoption of roll-over protective structures (ROPS) on tractors has helped to prevent some fatalities. (See Fig. 15-8 in Chapter 15.)

The annual nonfatal occupational injury rate for farmworkers is about 5.4 injuries per 100 workers.[4] Because of the lack of mandatory workers' compensation coverage for many agricultural workers and their fear of lost wages,

A B

Figure 36-2. (A) Picking up and carrying large potted plants in this manner increases the risk of low back and upper-extremity injuries. (B) This device, used as an ergonomic intervention for nursery workers, reduces the need to bend in order to pick up potted plants; it also has a handle designed to decrease stress on the upper extremities. (Courtesy of University of California Davis.)

Table 36-2. Characteristics of 3,271 Acute Pesticide Poisoning Cases in the Agricultural Industry in Ten States,* 1998–2005

Characteristics	Number (Percent)
Gender	
Male	2,189 (67)
Female	1,054 (32)
Organ Systems Affected	
Nervous system	1,743 (53)
Eye	1,300 (40)
Gastrointestinal tract	1,300 (40)
Skin	1,077 (33)
Respiratory system	1,074 (33)
Cardiovascular system	211 (6)
Severity	
Low	2,848 (87)
Medium	402 (12)
High	20 (0.6)
Factors That Contributed to Pesticide Exposure	
Off-target drift	1,216 (63)
Early reentry into a field	336 (17)
Pesticide use in conflict with label	319 (17)
Unknown	992 (30)

*California, Arizona, Florida, Louisiana, Michigan, New Mexico, New York, Oregon, Texas, and Washington.

Source: Adapted from Calvert G, Karnik J, Mehler L, et al. Acute pesticide poisoning among agricultural workers in the United States, 1998–2005. American Journal of Industrial Medicine 2008; 51: 883–898.

there is probably significant underreporting of work-related injuries. For example, a study in North Carolina, a state that does not have comprehensive workers' compensation for farmworkers, found that 8% of workers reported an injury at work in the previous 3 years. Of the injured workers who considered medical attention necessary, 41% did not receive it within 24 hours and 24% never received it. The most common reason why workers did not receive medical attention was refusal by their supervisors to allow them to leave work or lack of transportation. Medical expenses were paid by employers for only 38% of these injuries.[5]

DERMATITIS

Dermatitis among agricultural workers has been associated with exposures to *(a)* a variety of chemical agents, including pesticides; *(b)* sensitivity to plant materials, such as poison ivy and poison oak; and *(c)* infectious agents. In 2007, agricultural workers in the United States had the highest reported incidence rate of cases of dermatitis—twice that of manufacturing workers. Dermatitis is one of the major health problems associated with pesticide exposure (Table 36-2). A study at clinics in the Midwest migrant stream found that, for men age 20 to 29, dermatitis was the primary cause of clinic visits and, for men age 30 to 44, dermatitis was second only to hypertension. The rate of dermatitis among these farmworkers was 2.5 times that of the general population.[6]

INFECTIOUS DISEASES

The pandemic of H1N1 influenza that began in 2009 and previous preparations for an avian influenza outbreak focus attention on how farmworkers would fare during a communicable disease outbreak. Whether working with livestock or crops, farmworkers may have more obstacles to overcome than the general population because of their substandard living conditions, suboptimal access to medical care, and language and cultural barriers.

Poultry workers may be exposed to avian influenza virus at work.[7] Livestock workers may be exposed to viruses or other infectious agents through animals' respiratory secretions, blood, intestines, or excrement. (See Box 36-1.) Protective measures include seasonal influenza vaccine, training on risk reduction for animal influenza viruses, personal protective equipment and training on its use, sanitary facilities for hand washing, and surveillance for early detection of influenza in both workers and animals.

NIOSH solicited input from stakeholders on recommendations for pandemic influenza preparedness among farmworkers, which focus mainly on their cultural, social, and economic situation.[8] State and local public health officials, in collaboration with farm owners and agricultural extension agents, should include migrant

Box 36-1. Livestock Workers

There are approximately 600,000 hired workers on livestock operations in the United States. In agriculture, injuries related to interaction with animals are second only to machinery-related injuries. A study based on injuries of employees of dairy farms, cattle/livestock raisers, and cattle dealers in the Colorado workers' compensation system between 1997 and 2006 found that livestock-handling injuries were more frequent, more severe, and more costly than injuries involving other tasks. For dairy farmworkers, 48% of livestock-handling claims involved milking, with 21% involving a worker being kicked while milking. Among cattle/livestock raisers and cattle dealers, the most common contributing factor for an injury was horseback riding.[1] These data can be used to focus future safety interventions on specific tasks or locations on the farm.

Although the number of livestock operations has been decreasing in the past 10 years, the number of animals and the animal-to-worker ratio on each operation have been increasing, especially with widespread introduction of combined animal feeding operations (CAFOs). Noise exposure has been noted as a hazard on CAFOs. In swine facilities with many animals in enclosed and confined spaces, noise levels above 85 dBA are common. In addition to injuries and noise, other occupational health concerns on CAFO farms include air emissions, such as hydrogen sulfide, ammonia, volatile organic compounds, particulate matter, and endotoxins.[2]

Zoonotic infections, or zoonoses, threaten the health of livestock workers. When 15% to 45% of people in a community work in a CAFO, human influenza cases can increase by as much as 86% due to animals, workers, and other community members infecting each other. Influenza viruses with pandemic potential may also result after coinfection of a person with both an avian influenza virus and a human seasonal influenza virus—through generation of a reassortment influenza virus that is capable of human transmission.[3]

One preventive approach has been to provide seasonal influenza vaccine to workers to reduce opportunities for their simultaneous infection with an animal (avian or swine) influenza virus and a human influenza virus. Reduced opportunities for dual infections decrease the chances of reassortment and the eventual emergence of a novel influenza virus with pandemic potential.

Another approach is early identification and culling of poultry infected with avian influenza to prevent larger outbreaks among poultry; however, these measures could also create severe financial setbacks to farm owners and farmworkers. The threat of lost income may deter farmworkers from reporting ill poultry to supervisors and animal health officials, leading to further spread of virus. Federal regulations stipulate that farm owners and growers may be compensated for costs associated with culling of poultry for detection of avian influenza subtypes H5 and H7, although protection of workers' wages is not required.

The Centers for Disease Control and Prevention (CDC) provides guidelines for protecting poultry workers, which address personal protective equipment (PPE) and training on its correct use, sanitation, and recognition of influenza in birds and people in the event of an avian influenza outbreak in poultry.[4] Many of these same guidelines, especially for PPE and sanitation, are applicable to other livestock workers.

Along with the shift to CAFOs in the production of animal products, the workforce may also be changing. There are no national U.S. data on demographic and economic characteristics of livestock workers as the National Agricultural Workers Survey provides for crop farmworkers, but there has been an increase in the proportion of Spanish-speaking livestock workers, especially on larger farms in the northeastern United States. Spanish-speaking workers are also younger and work more hours than English-speaking workers.[5]

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clinics, unions, and other farmworker service organizations trusted by farmworkers in preparedness planning. Planning should consider disseminating emergency warnings and public health messages through multiple media and the cultural tone, educational level, and language of the message. It may include two-way information networks (via telephone or trusted messengers) to reach farmworkers in remote rural areas and camps. Because resources of farmworkers are limited, there needs to be planning for provision of food, essential supplies, and transportation during emergencies. Since many farmworkers may live in communal arrangements, alternative housing may be necessary to prevent spread of infection. Finally, differentiation of public-health and emergency-response activities from those of immigration enforcement will be necessary to promote cooperation of farmworkers.[8]

HEAT-RELATED ILLNESS

Heat-related illness and death in farmworkers has received renewed attention recently after several high-profile, heat-related fatalities. In response, both North Carolina and California have enacted laws to protect farmworkers from heat-related illness. From 1992 to 1996, the annual heat-related death rate for crop workers was 0.39 per 100,000, compared to 0.02 per 100,000 for all U.S. civilian workers. In addition, most deaths occurred in foreign-born farmworkers. Therefore, interventions should be both culturally and linguistically appropriate for both workers and their supervisors. In response to these findings, measures to manage heat stress were recommended to agricultural employers,[9] including the following:

1. Providing training to prevent, recognize, and treat heat illness
2. Implementing a heat-acclimatization program
3. Encouraging proper hydration
4. Establishing work/rest schedules in accordance with climate conditions
5. Ensuring access to shade or cooling areas
6. Monitoring the environment and workers during hot conditions
7. Providing prompt medical attention to those showing signs of heat-related illness (See Chapter 12B.)

CHILD LABOR

Agricultural work is one of the most common forms and the most dangerous form of child labor. In 2006 in the United States, more than 29 million youths under age 20 were potentially exposed to agricultural hazards as farm residents, farmworkers, children of migrant or seasonal workers, and visitors to farms (Fig. 36-3).[10] Although many are paid or unpaid children of family farmers, an increasingly important group of hired farmworkers are self-emancipated minors, who are primarily unauthorized recent immigrants living and working away from their families. These workers are especially vulnerable to injury because of their age, their undocumented legal status, and their social isolation from friends and family. (See Chapter 4.)

Figure 36-3. Toddlers play in the rows of a field of green onions while their parents work. (Photograph by David Bacon.)

In 2006, there were about 22,900 injuries to children on farms in the United States. The primary causes of injury were falls and incidents involving animals and farm vehicles. Between 1995 and 2002, there were more than 900 farm deaths in children under age 20, of which 13% were work-related. The most common causes of deaths were machinery incidents (23%), motor vehicles (19%), and drowning (16%).[10]

U.S. child labor laws, which regulate working conditions for minors, have many dual standards that provide lesser protection for children employed in agriculture than children employed in other industries:

- The minimum permissible work age is 14 in agriculture and 16 in other industries.
- Children age 12 or 13 may work in agriculture with the consent of their parents.
- Work tasks that have been designated as hazardous by the federal government can be performed at age 16 in agriculture, but not until age 18 in other industries.

In 1996, a national coalition of organizations issued a National Action Plan entitled "Children and Agriculture: Opportunities for Safety and Health,"[11] which led to special congressional funding to improve research and prevention of child agricultural injuries. One of the major accomplishments of this initiative has been the creation of the *North American Guidelines for Children's Agricultural Tasks*, which, in the absence of laws to restrict hazardous work tasks for youth, created voluntary guidelines to assist adults in assigning age-appropriate tasks to children age 7 to 16. These guidelines primarily focus on educating family farmers and influencing their decisions about which farm tasks their children can safely perform.[12]

FEDERAL REGULATIONS AND HEALTH SERVICE PROGRAMS FOR FARMWORKERS

Historically, federal occupational health laws have been less protective of agricultural workers than other industrial workers. Many Occupational Safety and Health Administration (OSHA) standards, such as the Hazard Communication Standard, explicitly excluded agricultural workers. In addition, OSHA is prohibited from regulating farms with fewer than 11 employees. OSHA regulations targeting agriculture include *(a)* the Field Sanitation Standard, which requires water for drinking and hand washing as well as toilets in the fields; *(b)* the Roll-over Protective Structures (ROPS) for Tractors Used in Agricultural Operations Standard, which requires ROPS in tractors manufactured after 1976; and *(c)* the Migrant and Seasonal Agricultural Worker Protection Act (MSPA), which provides migrant and seasonal farmworkers with protections concerning pay, working conditions, and work-related conditions and requires that temporary labor camps operated by agricultural employers meet state and federal safety and health standards.

Occupational pesticide exposure is the only occupational exposure that is entirely regulated by the Environmental Protection Agency (EPA). In 1992, under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), the EPA promulgated the Worker Protection Standard, a federal regulation that governs use of agricultural pesticides in commercial production. Its worker health and safety provisions require mandatory training programs, enforcement of pesticide reentry intervals, and provision of washing facilities for decontamination. The enforcement of this standard, which is implemented by cooperative agreements between the EPA and state agencies, has been criticized as being inadequate, in part because FIFRA limits penalties against employers.

Under the Migrant Health Act of 1962, the federal government provides support to over 120 community-based and state organizations that offer comprehensive primary care services to address the special needs of hired farmworkers. As a result of this program, a network of migrant health clinics has been created, which improves provision of medical care for these workers. However, significant obstacles remain due to cultural, linguistic, and logistical barriers that result in many farmworkers receiving inadequate or no medical care.

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See the Further Reading and Web Sites section at the end of the chapter.

Construction

Laura S. Welch

Construction workers build, repair, renovate, modify, and demolish structures—including houses, office buildings, churches and temples, factories, hospitals, roads, bridges, tunnels, stadiums, docks, and airports. To understand injury and illness risks in construction work, one must understand the numerous tasks performed by workers in numerous construction trades (Table 36-3).

Construction often must be done in extreme heat or cold, at night, and in windy, rainy, snowy, or foggy weather. Intermittent and seasonal work adds to health and safety risks and to the stress of job insecurity. Episodic employment, frequent change of employer, and continuous change in worksite exposures and ambient conditions make it difficult to document workers' jobs and hazardous exposures. For all of these reasons, there are only limited data on the nature and magnitude of hazardous exposures in the construction industry.

In developed countries, construction is consistently ranked among the most dangerous occupations. In 2007, 24% of all fatal on-the-job injuries in the United States occurred in construction, although construction accounted for only 8% of total employment.[1] For nonfatal injuries there were 2.4 lost-work-day cases per 100 full-time-equivalent construction workers—more than twice the average for all industry. Leading causes of injuries with lost workdays among construction workers were contact with

Table 36-3. Construction Occupations and Tasks

Boilermakers	Construct, assemble, maintain, and repair stationary steam boilers and boiler house auxiliaries. Work involves use of hand and power tools, plumb bobs, levels, wedges, dogs, or turnbuckles. Assist in testing assembled vessels. Direct cleaning of boilers and boiler furnaces. Inspect and repair boiler fittings, such as safety valves, regulators, automatic-control mechanisms, water columns, and auxiliary machines
Brick masons	Lay and bind building materials, such as brick, structural tile, concrete block, cinder block, glass block, and terracotta block, with mortar and other substances to construct or repair walls, partitions, arches, sewers, and other structures
Carpenters	Construct, erect, install, or repair structures and fixtures made of wood, such as concrete forms; building frameworks, including partitions, joists, studding, and rafters; wood stairways; window and door frames; and hardwood floors. May also install cabinets, siding, drywall and batt or roll insulation
Carpet installers	Lay and install carpet from rolls or blocks on floors. Install padding and trim flooring materials
Cement masons and concrete finishers	Smooth and finish surfaces of poured concrete, such as floors, walks, sidewalks, roads, or curbs using a variety of hand and power tools. Align forms for sidewalks, curbs, or gutters; patch voids; use saws to cut expansion joints
Construction laborers	Perform tasks involving physical labor at building, highway, and heavy construction projects, tunnel and shaft excavations, and demolition sites. May operate hand and power tools of all types: air hammers, earth tampers, cement mixers, small mechanical hoists, surveying and measuring equipment, and a variety of other equipment and instruments. May clean and prepare sites, dig trenches, set braces to support the sides of excavations, erect scaffolding, clean up rubble and debris, and remove asbestos, lead, and other hazardous waste materials
Drywall and ceiling tile installers	Apply plasterboard or other wallboard to ceilings or interior walls of buildings. Apply or mount acoustical tiles or blocks, strips, or sheets of shock-absorbing materials to ceilings and walls of buildings to reduce or reflect sound. Materials may be of decorative quality. Include lathers who fasten wooden, metal, or rockboard lath to walls, ceilings, or partitions of buildings to provide support base for plaster, fire-proofing, or acoustical material
Electricians	Install, maintain, and repair electrical wiring, equipment, and fixtures. Ensure that work is in accordance with relevant codes. May install or service street lights, intercom systems, or electrical control systems
Insulation workers	Apply insulating materials to pipes or ductwork, or other mechanical systems to help control and maintain temperature. Also line and cover structures with insulating materials. May work with batt, roll, or blown insulation materials
Operating engineers	Operate one or several types of power construction equipment, such as motor graders, bulldozers, scrapers, compressors, pumps, derricks, shovels, tractors, or front-end loaders to excavate, move, and grade earth, erect structures, or pour concrete or other hard surface pavement. May repair and maintain equipment in addition to other duties
Painters	Paint walls, equipment, buildings, bridges, and other structural surfaces, using brushes, rollers, and spray guns. May remove old paint to prepare surface prior to painting. May mix colors or oils to obtain desired color or consistency
Paperhangers	Cover interior walls and ceilings of rooms with decorative wallpaper or fabric, or attach advertising posters on surfaces, such as walls and billboards. Duties include removing old materials from surface to be papered
Plumbers, pipefitters, and steamfitters	Assemble, install, alter, and repair pipelines or pipe systems that carry water, steam, air, or other liquids or gases. May install heating and cooling equipment and mechanical control systems
Plasterers and stucco masons	Apply interior or exterior plaster, cement, stucco, or similar materials. May also set ornamental plaster
Reinforcing iron and rebar workers	Position and secure steel bars or mesh in concrete forms to reinforce concrete. Use a variety of fasteners, rod-bending machines, rod busters, blowtorches, and hand tools
Roofers	Cover roofs of structures with shingles, slate, asphalt, aluminum, wood, and related materials. May spray roofs, sidings, and walls with material to bind, seal, insulate, or soundproof sections of structures

(*Continued*)

Table 36-3. Construction Occupations and Tasks (Continued)

Sheet-metal workers	Fabricate, assemble, install, and repair sheet-metal products and equipment, such as ducts, control boxes, drainpipes, and furnace casings. Work may involve any of the following: setting up and operating fabricating machines to cut, bend, and straighten sheet metal; shaping metal over anvils, blocks, or forms using hammer; operating soldering and welding equipment to join sheet-metal parts; inspecting, assembling, and smoothing seams and joints of burred surfaces. Includes sheet-metal duct installers who install prefabricated sheet-metal ducts used for heating, air conditioning, or other purposes
Stonemasons	Build stone structures, such as piers, walls, and abutments. Lay walks, curbstones, or special types of masonry for vats, tanks, and floors
Structural iron and steel workers	Raise, place, and unite iron or steel girders, columns, and other structural members to form completed structures or structural frameworks. May erect metal storage tanks and assemble prefabricated metal buildings
Terrazzo workers and finishers	Apply a mixture of cement, sand, pigment, or marble chips to floors, stairways, and cabinet fixtures to fashion durable and decorative surfaces
Tile and marble setters	Apply hard tile, marble, and wood tile to walls, floors, ceilings, and roof decks

Source: Bureau of Labor Statistics. Standard occupational classification manual, 1998 revision. Available at: http://stats.bls.gov/soc/socguide.htm.

objects (35%), falls (23%), and overexertion (18%). Leading specific diagnoses were strains and sprains (35%), cuts and lacerations (11%), fractures (11%), and bruises and contusions (7%).[2]

Construction injuries comprise a disproportionate share of the total costs of occupational injuries in all industries in the United States—almost $13 billion annually. Fatal injuries account for 40% of this cost; nonfatal injuries and illnesses (mainly injuries with lost workdays) account for the rest. On average, the death of a construction worker results in losses of $4 million, and a nonfatal injury with lost workdays costs approximately $42,000. These estimates include *(a)* direct costs, such as payments for hospitals, physicians, and medicines; *(b)* indirect costs, such as wage losses, household production losses, and costs of administering workers' compensation; and *(c)* quality-of-life costs—that is, the value attributed to the pain and suffering that victims and their families experience as a result of injuries or illnesses.[2]

Employers in construction spend more on workers' compensation than employers in any other industry. In 2005, of all employer costs in construction, 5% were spent on workers' compensation—more than double the costs for manufacturing employers and almost three times the average cost for employers in all industries. Workers' compensation insurance premiums for some occupations were much higher than this average. For example, the median insurance premium rate for roofing was $30 for each $100 of payroll. Nevertheless, only 46% of all medical expenses for work-related injuries were paid by workers' compensation—and only 27% among injured Hispanic construction workers. The remaining amount was paid by workers and their families or by other public or private sources, subsidizing workers' compensation medical coverage in the construction industry by at least $734 million annually.

Occupational diseases are also an important cause of morbidity in construction workers. Table 36-4 summarizes diseases that are sentinel health events that may occur in construction workers and specific hazardous exposures that can lead to these diseases. These sentinel health events help to focus attention on intervention and prevention measures. These hazardous exposures include air contaminants, such as wood dust, abrasive blasting dust, gypsum and alkaline dusts, silica, asbestos, lead, diesel exhaust, and welding fumes.

LEAD AND OTHER HEAVY METALS

Lead exposure and resultant toxicity are especially important problems in the construction industry. (See Chapters 11, 19, and 20.) Excessive lead exposure is associated with several

Table 36-4. Sentinel Health Events in Construction

Condition	Industry/Process/Occupation	Agent
Asbestosis	Asbestos industries and utilizers	Asbestos
Bronchitis (acute), pneumonitis, and pulmonary edema due to fumes and vapors	Arc welders, boilermakers	Nitrogen oxides Vanadium pentoxide
Chronic or acute renal failure	Plumbers	Inorganic lead
Contact and allergic dermatitis	Cement masons and finishers, carpenters, floor layers	Adhesives and sealants; irritants (such as cutting oils, phenol, solvents, acids, alkalis, detergents); allergens (such as nickel, chromates, formaldehyde, dyes, rubber products)
Extrinsic asthma	Woodworkers, furniture makers	Red cedar (plicatic acid) and other wood dusts
Histoplasmosis	Bridge maintenance workers	*Histoplasma capsulatum*
Inflammatory and toxic neuropathy	Furniture refinishers, degreasing operations	Hexane
Malignant neoplasm of scrotum	Chimney sweeps	Mineral oil, pitch, tar
Malignant neoplasm of nasal cavities	Woodworkers, cabinet and furniture makers, carpenters	Hardwood and softwood dusts Chlorophenols
Malignant neoplasm of trachea, bronchus, and lung	Asbestos industries and utilizers	Asbestos
Malignant neoplasm of nasopharynx	Carpenter, cabinet maker	Chlorophenols
Malignant neoplasm of larynx	Asbestos industries and utilizers	Asbestos
Mesothelioma (malignancy of peritoneum or pleura)	Asbestos industries and utilizers	Asbestos
Noise effects on inner ear	Occupations with exposure to excessive noise	Excessive noise
Raynaud phenomenon (secondary)	Jackhammer operator, riveter	Whole-body or segmental vibration
Sequoiosis	Red cedar mill workers, woodworkers	Redwood sawdust
Silicosis	Sandblasters	Silica
Silicotuberculosis	Sandblasters	Silica + *Mycobacterium tuberculosis*
Toxic encephalitis	Lead paint removal	Lead
Toxic hepatitis	Fumigators	Methyl bromide

Source: Adapted from: Mullan R, Murthy L: Occupational sentinel health events: an up-dated list for physician recognition and public health surveillance. American Journal of Industrial Medicine 1991; 19: 775–799. Reprinted with permission from: Sullivan P, Moon Bank K, Hearl F, Wagner G. Respiratory risk in the construction industry. In: Ringen K, Englund A, Welch LS, et al. (eds.). Health and safety in construction. State of the Art Reviews in Occupational Medicine 1995; 10: 269–284.

construction tasks.[3] Almost 1 million construction workers in the United States are exposed to lead at work. More than 80% of these workers are involved in commercial or residential remodeling. Before 1993, the OSHA lead standard applied only to general industry, not to construction. In 1992, blood lead levels (BLLs) in bridge construction workers ranged from 51 to 160 μg/dL, with 62% of elevated BLLs involving work in a containment structure. High-risk activities associated with lead dust and fumes among bridge and structural steelworkers include abrasive blasting, sanding, burning, cutting, and welding on steel structures coated with lead paint, while working in containment enclosures. In 1993, the OSHA lead standard was revised, incorporating a presumption of exposure during specific high-risk tasks and requiring

specific protections during these tasks—unless air monitoring demonstrates airborne lead exposure at a concentration below the permissible exposure limit. However, it is important to recognize that even the revised OSHA standard may not fully protect construction workers from lead toxicity. The standard requires monitoring every 2 months; but some tasks, such as burning lead-coated steel, can cause a rapid increase in BLL. Thus, more frequent monitoring and a lower threshold for mandated industrial hygiene inspection or medical removal of workers has been recommended in some circumstances. Elevated BLLs are reported by 32 states to a national lead surveillance (Adult Blood Lead Epidemiology and Surveillance, or ABLES) program. In 2003 and 2004, the construction industry accounted for 17% of the workers with BLLs at or above 25 μg/dL, while construction employment accounted for only about 7% of the total U.S. workforce. Among the top five industries with the most reported cases of BLLs above 25 and 40 μg/dL in this period were building finishing; highway, street, and bridge work; and utilities. These minimal estimates indicated significant lead exposure occurring in the construction industry.

Construction workers can be exposed to manganese and chromium during welding; pipefitters, ironworkers, boilermakers, and sheet-metal workers routinely perform welding and related processes such as arc cutting. This work often occurs in tanks, boilers, or other poorly ventilated settings. Fumes generated during welding contain fine particles from the base metal, the electrodes, fluxes, and the filler rods. In the United States, there are an estimated 410,000 full-time welders and more than 1 million intermittent welders who are exposed to welding fumes. The International Agency for Research on Cancer (IARC) has determined that welding fumes cause cancer. Welders of stainless steel have higher rates of lung cancer than workers who weld using other metals.

Manganese, a known neurotoxin, is a component of nearly all types of steel and many welding rods and wires. Excessive exposure to manganese in other industries, such as manganese mining and smelting, causes symptoms and signs closely resembling those of Parkinson disease. Recent studies of welders suggest the level of manganese exposure in welding fumes can also cause these symptoms and signs.

Metal fumes from stainless steel welding contain hexavalent chromium and nickel, both of which cause lung cancer. OSHA estimates almost 200,000 construction workers in the United States are exposed to airborne hexavalent chromium, and that a substantial proportion of these workers are exposed above the OSHA permissible exposure limit.

NOISE

Construction workers generally have excessive noise exposures and high rates of noise-induced hearing loss. (See Chapter 21.) More than 500,000 construction workers are exposed to potentially hazardous levels of noise. The United States has a different standard for regulation of noise exposure in construction than in general industry; in the construction standard, there is no action level above which a hearing conservation program is required and there are no detailed requirements for training or record keeping. Yet construction work is very noisy. For example, operating engineers, on average, are exposed to noise levels greater than 85 dBA for 49% of their work shifts and greater than 90 dBA for 25% of their work shifts. A laborer using a heavy-duty bulldozer is exposed to 91 to 107 dBA (mean, 99 dBA). Exposure in crane cabs ranges from a mean of 81 dBA in insulated cabs to 97 dBA in noninsulated cabs.[4]

There are places that have reduced noise exposure in construction. For example, British Columbia implemented, in 1987, a specific hearing conservation program in construction. Since that time, reported use of hearing protection by workers has increased from 55% to 85%, and the proportion of 50-to-59-year-old construction workers with hearing impairment has decreased from 36% to 25%. This program demonstrates the feasibility and efficacy of a specific hearing conservation program in the construction industry.

MUSCULOSKELETAL DISORDERS

Soft-tissue musculoskeletal injuries make up a high proportion of all work-related injuries in

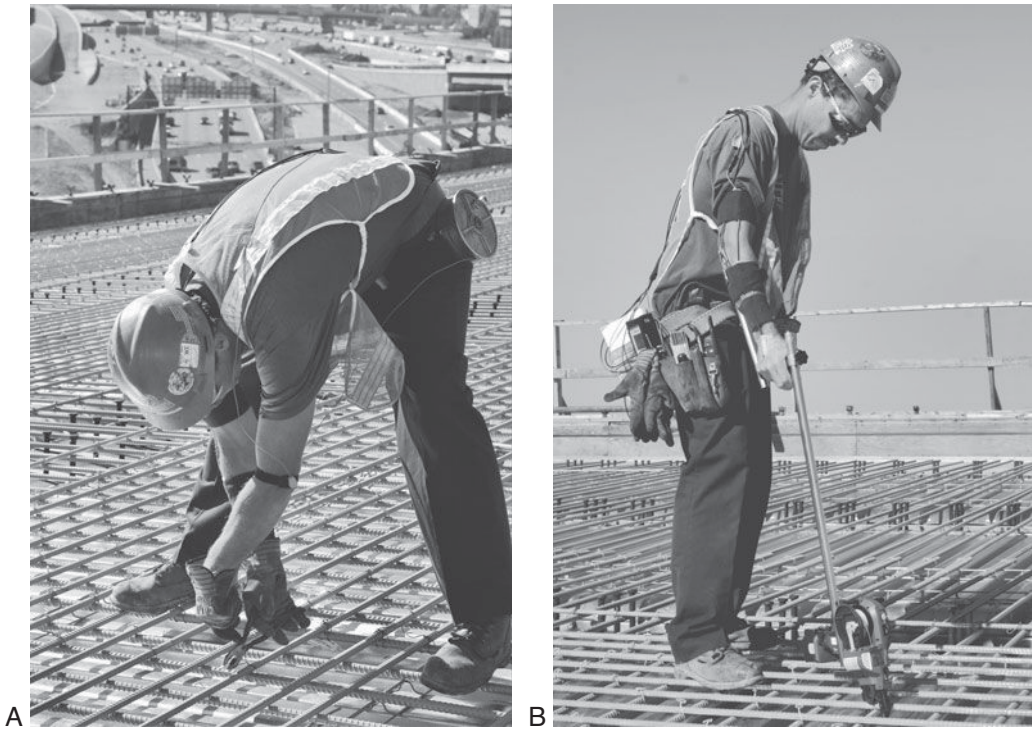

Figure 36-4. (A) Construction workers are at increased risk of upper-extremity and back strain. (B) An ergonomically designed device decreases the upper-extremity and back strain on construction workers who are tieing rebar. (Photographs by Earl Dotter.)

construction (Fig. 36-4).[5] In 2007, there were in the United States an estimated 197,500 injuries and illnesses with lost work days in construction; 35% of these injuries were attributable to strains and sprains and 19% were low back injuries. The rates for these injuries are considerably higher in construction than in all private industry combined.[1,2] Construction workers retire 2 years earlier, on average, than other workers, often because of musculoskeletal conditions, such as arthritis and degenerative disc disease. In a national survey in 1998, 10% of construction workers in the United States reported back pain due to repeated injury at work—twice the rate of all workers. Severe hand discomfort was present in almost 16% of construction workers, compared to 11% of all workers. (See also Chapters 15 and 16.)

Reducing the physical demands on construction workers will require changing the culture of construction, developing new task-specific ergonomic innovations, and promoting participatory ergonomics programs. The construction work environment changes as a project progresses. Both this dynamic nature of the construction process and the project-by-project nature of employment limits employers' incentives to prevent chronic musculoskeletal disorders. Effective interventions to reduce physical demands in construction include decreasing back stress in masons through adjusting work height, eliminating shoulder and neck strain during overhead drilling tasks with a drill support, and various approaches to reduce manual materials handling. Interventions that are more likely to succeed have a perceived relative advantage, are compatible with prevailing norms or practices, can be tried before being fully implemented, and have impacts that are readily observable.[6]

RESPIRATORY DISEASES

Construction workers are exposed to many respiratory hazards, including asbestos, crystalline silica, synthetic vitreous fibers, cadmium, chromates, formaldehyde, resin adhesives, cobalt,

metal fumes, creosote, gasoline, oils, diesel fumes, paint fumes and dusts, pitch, sealers, solvents, wood dusts and wood preservatives, and extremes of temperature.[7] In 2005, the Bureau of Labor Statistics (BLS) reported 1,100 nonfatal work-related respiratory conditions among 7.2 million wage-and-salary construction workers in the private sector in the United States—thought to be a major underestimate. For comparison, the National Center for Health Statistics reported that, in 2000, approximately 20,000 people were hospitalized with asbestosis.

Asbestosis

Asbestos has been recognized as a respiratory hazard for several construction trades. Many construction workers are occupationally exposed to asbestos, especially insulators, plumbers and pipefitters, electricians, and sheet-metal workers. Any construction worker may be at risk for asbestos-induced disease from working near asbestos insulation. Although asbestos is no longer used in new residential or heavy construction, workers may continue to be exposed to previously installed asbestos during maintenance, renovation, addition, or demolition activities.

Silicosis

Occupational exposure to silica can occur among various types of construction workers, including those employed in concrete removal and demolition work, bridge and road construction, tunnel construction, and concrete or granite cutting, sanding, and grinding. Sandblasters are at increased risk from exposure to crystalline silica. Those working nearby on the same construction site may also be at risk from silica-related disease. In the United States, sand containing crystalline silica is still used in abrasive blasting operations for maintenance of structures, preparing surfaces for painting, and forming decorative patterns during installation of building materials; these uses of sand have been banned in many other countries. Silica exposures in the construction industry in the United States continue to exceed recommended limits. Silicosis continues to occur in construction workers worldwide. In addition, silica is a cause of chronic obstructive pulmonary disease (COPD), independent of the presence of radiological evidence of silicosis.

Chronic Obstructive Pulmonary Disease and Asthma

Occupational exposure to the general category of vapors, gases, dusts, and fumes (VGDF) has been have been associated with increased risk of COPD. These exposures, which occur in welding and with use of heavy metals, silica, mineral dust, and adhesives, are common in construction. Chronic obstructive pulmonary disease has been reported among construction workers exposed to asbestos, manmade mineral fibers, and welding fumes; occupations at risk include spray painters, welders, tunnel construction workers, construction painters, and sheet-metal workers. Construction workers can be exposed to allergens, cold, particulates, dusts, fumes, and irritants, all of which can exacerbate asthma. (See also Chapter 18.)

DERMATITIS

Construction workers are exposed to many chemicals that cause irritant or allergic dermatitis (see Chapter 22). Portland cement, which is found in plaster and concrete mixes, is extremely alkaline. Wet plaster also contains slaked lime (calcium hydroxide), which is even more caustic. In addition, Portland cement contains trace amounts of hexavalent chromium, a strong sensitizing agent that causes allergic contact dermatitis in cement workers. Other sensitizing agents include epoxy adhesives, sealants, and chemicals mixed within cement and plaster. Rubber gloves also may cause allergic dermatitis.

For 2005, the BLS reported that skin diseases or disorders accounted for 27% of all occupational illnesses among construction workers. Experts have estimated that the actual number of occupational skin disorders is 10 to 50 times higher than the number BLS reported. One way to prevent allergic contact dermatitis in cement workers is to add ferrous sulfate to hexavalent chromium in cement, a process that forms, when water is added, an insoluble trivalent chromium compound that is not easily absorbed by the skin.

Table 36-5. Epidemiology of Lung Cancer in Construction Workers

Trade	Known Lung Carcinogens
Insulators	Asbestos
Painters and plasterers	Chromium, cadmium, asbestos
Sheet-metal workers	Asbestos, welding fume
Welders	Welding fume, asbestos, hexavalent chromium
Masons	Asbestos, hexavalent chromium, silica
Electricians	Asbestos
Plumbers and pipefitters	Asbestos, welding fume
Roofers	Coal tar, bitumen, polycyclic aromatic hydrocarbons (PAHs)
Carpenters	Wood dust

CANCER

Construction workers are exposed to many carcinogens (Table 36-5). Insulators, painters and plasterers, sheet-metal workers, and other construction workers are at increased risk of lung cancer. Woodworkers, cabinetmakers, furniture makers, and carpenters and joiners have an increased risk of nasal cancer. Workers in many trades have had increased rates of mesothelioma after widespread exposure to asbestos from approximately 1940 to 1980. Given the long latency period for mesothelioma, asbestos-related cases are likely to occur for many years to come. (See Chapter 17.)

REGULATIONS AND HEALTH SERVICES FOR CONSTRUCTION WORKERS

Construction workers are often not covered by the OSHA regulations that cover manufacturing and service sectors. (See Chapter 30.) For example, the standard for noise exposure for the construction industry has no action level above which a hearing conservation program is required, and no detailed requirements for training or record keeping. The rationale for separate OSHA standards for construction is that controls that work in general industry may not work in construction. Therefore, feasibility of a standard must be demonstrated specifically in construction before the standard can be applied to the construction sector. Although this is a reasonable consideration, leaving construction out of a standard until feasibility is demonstrated has led to decades of hazardous exposure for construction workers. Underreporting of injury and illness is prevalent in construction, in part, because the construction industry is comprised mainly of small employers. A legal requirement to report injuries by construction project, which could apply to many small employers, could help to better elucidate and focus more attention on the health and safety problems faced by construction workers.

In the United States, intermittent employment and the high cost of health insurance can leave construction workers and their families without insurance coverage for medical care. In 2005, only 58% of wage-and-salary workers in construction had employment-based health insurance, compared to 75% among all wage-and-salary workers.[2] Because construction is a complex industry, there are proportionately fewer research and prevention activities in construction than in general industry. All of these factors leave the construction industry in great need for improvement in health and safety.

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Health Care

Jane A. Lipscomb

More than 14 million persons (greater than 10% of those employed) in the United States work in health care. About 80% of health care workers are women. The health care sector includes a greater percentage of African Americans and Asians, but a slightly lower percentage of Hispanics, than all industries combined. Some groups within the health care workforce are at increased risk of adverse effects of work-related exposures and demands. For example, registered nurses over the age of 50 are at increased risk of injuries due to the physical demands of patient care. Over the next decade, the number of workers in health care is expected to grow dramatically, with 20% of new jobs in health care. Ironically, health care workers probably confront a greater range of significant workplace hazards than workers in any other sector (Table 36-6), including the following:

- Biologic hazards associated with airborne and bloodborne exposures to infectious agents
- Chemical hazards, especially those found in hospitals, such as waste anesthetic and sterilant gases, hazardous drugs (including antineoplastic medications) and other therapeutic agents, mercury, and industrial-strength disinfectants and cleaning compounds
- Physical hazards, including ionizing and nonionizing radiation
- Safety and ergonomic hazards, which cause a variety of acute injuries and chronic musculoskeletal disorders
- Violence, including physical assaults and threats of assaults
- Psychosocial and organizational factors, including work stress, short staffing, and shift work
- Many health consequences associated with changes in the organization and financing of health care

According to the BLS, in 2007, the occupational injury and illness rate per 100 workers among hospital workers (7.7) was nearly double that of the overall private sector rate (4.2) and higher than rates for workers employed in mining (3.1), manufacturing (5.6), and construction (5.4). Although occupational injury and illness rates have been declining among all industry sectors over the past decade, the decline in the health care sector has been more modest, with the rate ratio for hospital workers compared to all private-industry workers increasing from 1.72 in 2005 to 1.83 in 2007. Workers in nursing homes had an even higher rate of occupational injuries and illnesses—8.8 per 100 full-time workers in 2007.

The health care industry appears to be a decade or more behind other high-risk industries in ensuring safety.[1] There has long been a deep-rooted belief that patient health and safety supersede worker health and safety, and that it is acceptable for health care workers to have less than optimal protection against workplace hazards.[2] For example, infection-control practitioners often promote maximum patient protection while de-emphasizing appropriate measures to prevent worker infections. Both patient and worker hazards arise from the same source—health care practices, products, and materials, and the built environment. Therefore, development of effective approaches to control these hazards requires an integrated approach.[3]

The generation and disposal of biologic, chemical, and radiologic wastes pose risks to communities around health care facilities and beyond, especially if these facilities incinerate their waste on site. The widespread use, and resulting incineration, of plastics containing chlorine compounds, such as polyvinyl chlorine,

Table 36-6. Selected Hazards, Health Effects, and Control Strategies in Health Care

Hazards	Health Effects	Control Strategies
Biological		
Viral (hepatitis B virus, hepatitis C virus)	Acute febrile illness, liver disease, death	Safer needle devices, hepatitis B vaccine
Bacteria (*Mycobacterium tuberculosis*)	Tuberculosis (TB) infection, TB illness, multiple drug resistance, death	Isolation of suspect patients, respirators, ultraviolet light, negative pressure rooms
Natural rubber latex proteins (and rubber chemical additives)	Type I and Type IV immunologic responses; Type I immediate hypersensitivity includes anaphylactic shock	Substitution with low-latex-protein, powderless gloves or nonlatex gloves and supplies
Chemical		
Ethylene oxide	Peripheral neuropathy, cancer, reproductive effects	Substitution, enclosed systems, aeration rooms
Formaldehyde and glutaradehyde	Allergy, nasal cancer Mucous-membrane irritation, sensitization, reproductive effects	Subsititution, local ventilation Substitution, local ventilation
Antineoplastic drugs	Cancer, mutagenicity, reproductive effects	Class 1 ventilation hoods, isolation of patient excreta
Waste anesthetic gases	Hepatotoxicity, neurologic effects, reproductive effects	Scavenging systems, isolation of off-gassing patients
Mercury	Neurologic effects, birth defects	Substitution with electronic thermometers
Physical		
Patient handling	Back pain, injury	Patient handling devices, lifting teams, training
Static postures	Musculoskeletal pain and injury	Rest breaks, exercise, support hose and shoes
Ionizing radiation	Cancer, reproductive effects	Isolation of patients, shielding and maintenance of equipment
Lasers	Eye and skin burns, inhalation of toxic chemical and pathogens, fires	Local exhaust ventilation, equipment maintenance, respirators and face shields
Physical assault	Traumatic injuries, death	Alarm systems, security personnel, training
Psychosocial/Organizational		
Threat of violence, including physical assault	Traumatic injury, death, posttraumatic stress disorder	Training, postassault debriefing
Restructuring	Job strain, exacerbation of musculoskeletal injuries, traumatic injuries, burnout	Acuity-based staffing, employee involvement in restructuring activities; improved safety culture
Additional work stress	Job strain, burnout	Stress prevention and management programs; improved safety culture
Shift work	Gastrointestinal disorders, sleep disorders	Forward, stable, and predictable shift rotation

has the potential to create and release into the atmosphere dioxins, which are highly toxic. Community organizations have successfully advocated for changes, such as phasing out from health care products that contain mercury and bisphenol A (BPA), and reducing incineration of mercury-containing products. In 1998, the American Hospital Association and the EPA signed a memorandum of understanding to prevent release of persistent, bioaccumulative toxic chemicals by hospitals. Now, many hospitals work together with environmental groups to reduce use of chemicals and promote "greener" work practices and environments.

Surveillance of occupational illnesses is even more limited than that of occupational injuries.

In 2005, the incidence of occupational illness among health care workers was 39.9 per 10,000 full-time workers, compared to 26.7 in private industry and 19.6 in the service sector. Nonfatal skin diseases and disorders (7.0 cases per 10,000 workers) and respiratory conditions (5.2) were the most frequently reported illnesses.

MUSCULOSKELETAL DISORDERS

The highest proportion of musculoskeletal disorders (MSDs), which rank second among all work-related injuries, occur among health care workers. Exposures include those involved with lifting, pulling, sliding, turning, and transferring patients; moving equipment; and standing for long periods. Among all categories of workers, hospital and nursing-home workers have the highest number of occupational injuries and illnesses involving lost workdays due to back injuries (Fig. 36-5). (See Chapter 16.)

In 2007, nursing aides, orderlies, and attendants had an MSD rate of 252 cases per 10,000 workers—a rate more than seven times the U.S. average for all occupations. They also reported the most MSD cases involving days away from work (24,340), with a median of 5 days away from work.

Many registered nurses working for at least 1 year have reported neck problems (46%), shoulder problems (35%), and back problems (47%).[4] Almost 80% of nurses experience low back pain during their working lifetime.[5] Nurses reporting highly physically demanding jobs have been five to six times more likely to report a neck, shoulder, or back MSD, as compared with those with less physically demanding jobs. Lifting teams at work, with or without availability of mechanical transfer and lifting devices, have been associated with significantly lower occurrence of back MSDs; nevertheless, many employers still teach body mechanics rather than implementing a comprehensive back injury prevention program. The risk for MSDs is also greater when nurses work shifts longer than 12 hours and on evenings, nights, and weekends.[6]

In the United States, the nursing-home industry spends more than $1 billion annually in workers' compensation premiums, although there is strong evidence that reducing low back load by implementing engineering and administrative controls, such as by safe staffing levels, lifting teams, and use of newer mechanical

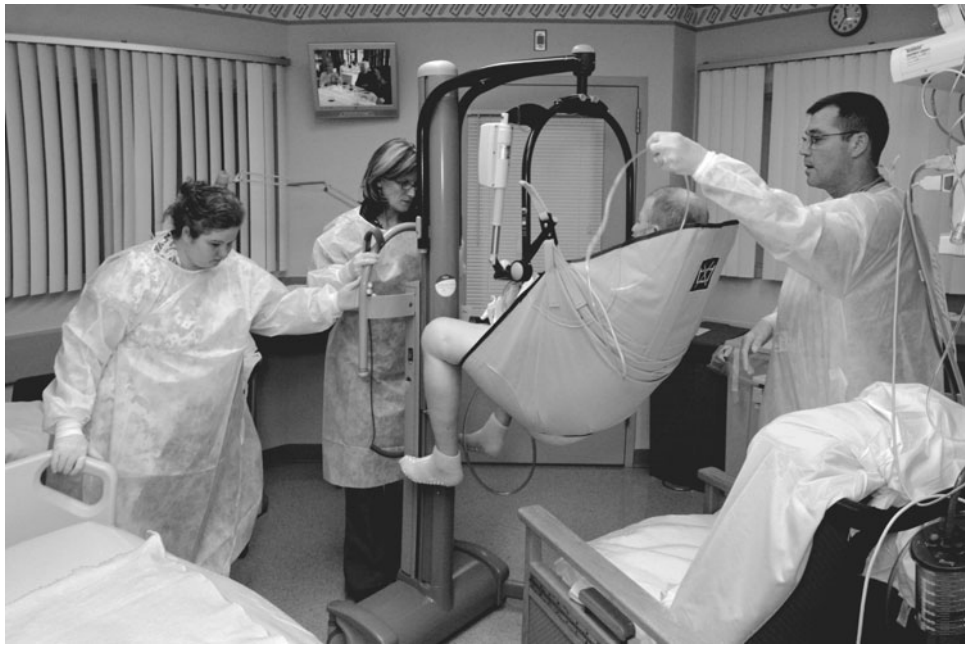

Figure 36-5. Nurses' aides lift a patient by using a mechanical assist device. (Photograph by Earl Dotter.)

patient-handling devices, reduces the risk of injury for both patients and workers. A study that evaluated the effectiveness of a safe resident lifting and movement intervention in nursing homes found that the program reduced the injury rate and workers' compensation costs for injuries due to patient handling, and that the return on investment (ROI) for direct costs of the equipment and training was less than 3 years.[7]

Other health care workers face increased risks for MSDs. For example, laboratory workers are at increased risk for cumulative trauma disorders of the hand and wrist due to repetitive work, such as pipetting. Operating-room workers, who must maintain static postures for long periods of time or hold instruments overhead during long operations, are at increased risks of neck and shoulder pain and injury. Workers who provide health care in patients' homes face risks related to assisting patients who have limited mobility, since few patients have mechanical lifting devices at home and a second person is often not available to assist with patient transfers.

WORKPLACE VIOLENCE

Health care leads other sectors in the incidence of nonfatal workplace assaults. Of all nonfatal assaults against workers resulting in lost work days in the United States, 32% have occurred in the health care sector. In about half of nonfatal assault injuries, patients are the perpetrators. In 2005, the BLS rate of nonfatal assaults among health care workers was 8.8 cases per 10,000—almost four times higher than in the private sector. Among assault victims, 30% were government employees, even though they comprise only 18% of the U.S. workforce.

In each year from 1993 to 1999, 1.7 million incidents of violence occurred in the workplace. Twelve percent of all victims reported physical injuries, half of them requiring medical treatment. Less than half of incidents were reported to the police. Mental health professionals had an annual incidence rate of 68 per 1,000 workers compared to 12 for all workers. Nurses had an annual incidence rate of 22 per 1,000 workers.[8] In a study in a psychiatric facility in Washington State, 73% of staff members reported a minor injury related to an assault by a patient during the previous year; only 43% of those reporting moderate, severe, or disabling injuries due to such assaults had filed for workers' compensation. The survey found an annual assault incidence rate of 437 per 100 employees per year, whereas the reported incidence rate for the hospital was only 35, indicating substantial underreporting of assaults.[9]

Emergency department workers also face an increased risk of injuries from assaults by patients or their family members. Carrying of weapons in emergency departments creates the opportunity for severe or fatal injuries. California and Washington State have enacted standards requiring safeguards for emergency department workers. Since no department in a health care setting is immune from workplace violence, all departments should have violence prevention programs.

Environmental and organizational factors have been associated with patient assaults, including understaffed situations (especially during times of increased activity such as meal times), poor workplace security, unrestricted movement by the public around the facility, and the transport of patients. The presence of security personnel reduces the rate of assault. The rate of assault is increased when *(a)* administrators consider assault to be part of the job, *(b)* there is a high patient-to-worker ratio, and *(c)* work is primarily with patients with psychiatric disorders or with patients who have long hospital stays.

Many psychiatric settings now require that all care providers receive annual training in the management of aggressive patients, but few studies have examined the effectiveness of such training. Those that have done so have generally found improvement in nurses' knowledge, confidence, and safety after taking an aggressive behavior-management program.

Health care workplaces must be made safe for all workers through the use of engineering and administrative controls, such as security alarm systems; adequate staffing; and training.

NEEDLESTICK INJURIES

The most prevalent, least reported, and largely preventable serious risk health care workers face arises from their continuing use of inherently dangerous conventional needles and sharp

devices that lack an engineered injury protection feature. Unsafe needles transmit bloodborne infections to health care workers employed in a wide variety of occupations. Injuries can be dramatically reduced by eliminating unnecessary sharp devices and using sharp devices with engineered injury-protection features. (See Chapter 13.)

Percutaneous injuries continue to frequently occur in health care despite the promulgation in 1991 of the OSHA Bloodborne Pathogen (BBP) Standard. The physical and mental health consequences of transmission of a potentially fatal bloodborne infection have also remained high since then. The requirement under the BBP Standard that hepatitis B vaccine be made available free of charge to health care workers has greatly reduced the consequences of exposure to this pathogen. The advances in the treatment of human immunodeficiency virus (HIV) infection with postexposure prophylaxis has improved the prognosis for health care workers infected with HIV-contaminated blood. There is no vaccine or treatment for hepatitis C virus (HCV), and, therefore, health care workers continue to suffer life-threatening illness following exposure to HCV-contaminated blood. Therefore, all health care workers, not only those working in the acute care setting or those who traditionally handle needles on a regular basis, should receive every available protection from occupational exposure to blood and body fluids.

After a needlestick injury, the risk for the nonimmune health care worker of developing occupationally acquired hepatitis B virus (HBV) infection ranges from 2% to 40%, depending on the hepatitis B surface antigen (HB_sAg) status of the source patient. The risk of transmission from a positive source for HCV is between 3% and 10%,[10] and for HIV, 0.3%.[11] However, the risk of transmission increases if *(a)* the injury is caused by a device visibly contaminated with blood, *(b)* the device is used to puncture the vascular system, or *(c)* the stick causes a deep injury. All of these diseases are associated with significant morbidity and mortality. Only hepatitis B can be prevented by vaccine. Health care, laundry, and housekeeping workers are often engaged in tasks that create a potential for these high-risk needlestick injuries. (See Fig. 1-3 in Chapter 1.)

An estimated 600,000 to 800,000 needlestick injuries occur in the United States annually in health care, half of which are sustained by hospital-based, health care providers. Approximately half of all needlestick injuries are not reported. At an average hospital, workers incur approximately 30 needlestick injuries per 100 beds per year. Fifty-four percent of reported needlestick and sharp-object injuries involve nurses.[12] Annually, percutaneous exposure in the United States leads to an estimated 150 transmissions of HCV among health care workers. The annual death rate in the United States for health care workers from occupational infectious diseases was estimated, for 2002, to be 9 to 29 per 1 million workers, with 75 to 250 deaths from HBV (a number that is expected to markedly decline in future years due to HBV vaccine) and 5 to 10 deaths from HIV, HCV, and tuberculosis (TB) combined.[13]

In the past 20 years in the United States, 57 occupationally acquired HIV infections have been documented among health care workers. Due to reporting difficulties, this number grossly underestimates the number of actual occupationally acquired HIV infections. No new cases occurred between 1999 and 2009; approximately half of all cases occurred before 1991. Of all health care worker infections, 88% have resulted from percutaneous injuries—41% occurring after a procedure, 35% during a procedure, and 20% during disposal of equipment. Unexpected circumstances occurring during or after the procedure accounted for 20% of injuries.

There is a great emotional impact to a health care worker following a needlestick. Drug prophylaxis can be exhausting and debilitating. The emotional threat of having incurred a possible fatal disorder has a profound impact on the daily life of health care workers and their ability to perform their jobs, maintain stable relationships with their co-workers and family members, and have emotional balance. These emotional reactions may be manifest as symptoms of anxiety or posttraumatic stress disorder (PTSD).

Use of conventional sharp devices in health care today has been compared with the use of unguarded machinery many years ago in the industrial workplaces. Safer sharp devices have built-in integrated safety features that prevent needlestick injuries (Fig. 13-3). The term "safer needle device" is broad and includes many different devices, from those that have a protective shield over the needle to those that do not

use needles at all. Needles with integrated safety features are categorized as passive or active. Passive devices offer the greatest protection because the safety feature is automatically engaged after use, without the need for a health care worker to take any additional steps. An example of a passive device is a spring-loaded retractable syringe or self-blunting blood collection device. An example of an active safety mechanism is a sheathing needle that requires the worker to manually engage the safety sheath, frequently using the other hand and potentially resulting in another needlestick.

The passage of the federal Needlestick Safety and Prevention Act in 2000 has afforded health care workers better protection from this unnecessary and potentially fatal hazard. Not only does the Act amend the 1991 BBP Standard to require that safer needles be made available, but it requires employers to solicit the input of front-line health care workers when making decisions to purchase safe needles. While there has been widespread conversion to safety in some device categories (such as phlebotomy needles and intravenous catheters), relatively few safety devices have been used in others (such as laboratory equipment and surgical instruments). Between 1993 and 2001, percutaneous injury rates due to needlesticks in nurses decreased 51%, supporting the use of new technology in reducing percutaneous injury risk.[14]

LATEX ALLERGY

Within the first few years after promulgation of the 1991 BBP Standard, latex allergy, partly attributed to increased use of examination and surgical gloves required by the Standard, began to be reported by nurses and other health care workers. The prevalence of latex allergy among health care workers is estimated to be between 5% and 18%, with atopic workers at greater risk. People with latex allergy are also more likely to develop sensitivity to other allergens, especially certain food items.

Three types of reactions can occur in persons using latex products: irritant (nonallergic) contact dermatitis, allergic contact dermatitis, and latex allergy. The most common reaction to latex products is irritant contact dermatitis, with dry, itchy, irritated areas on the skin, usually of the hands. This reaction is caused by skin irritation from using gloves and possibly by exposure to other workplace products and chemicals. Allergic contact dermatitis (delayed hypersensitivity dermatitis) results from exposure to chemicals added to latex during harvesting, processing, or manufacturing. These chemicals can cause skin reactions similar to those caused by poison ivy.

Latex allergy (immediate hypersensitivity) can be more serious. Certain proteins in latex may cause sensitization. Although the amount of exposure needed to cause sensitization is not known, exposures at even very low levels can trigger allergic reactions in some sensitized individuals. Mild reactions to latex include skin redness, itching (urticaria) or hives, and runny nose, sneezing, itchy eyes, and scratchy throat. More severe reactions include asthma and anaphylaxis.

In 1997, NIOSH recommended the use of powderless, low-protein latex gloves only when protection from infectious agents is needed, and, when latex gloves are used. Substituting nonlatex or powder-free national rubber latex for powdered gloves reduces the incidence of suspected latex allergy and, specifically, latex-related occupational asthma. Hospitals with programs or policies to reduce employee exposure to latex have reported a 40% decrease in latex-related symptoms. A Belgium study found incident cases of definite and probable natural rubber latex-induced occupational asthma markedly decreased from 1999 onward.[15]

CHEMICAL HAZARDS

Health care workers are exposed to *(a)* a wide range of chemical disinfectants, anesthetic waste gases, and hazardous drugs (such as chemotherapetic medications) that are known to cause adverse health effects, and *(b)* others for which there has been inadequate or no testing. The average hospital contains about 300 chemicals—twice the number of the average manufacturing facility. Among disinfectants, formaldehyde is a probable human carcinogen and has been linked to occupational asthma in hospitals. Glutaraldehyde (Cidex), a widely used cold-sterilization solution for disinfecting and cleaning heat-

sensitive instruments, such as endoscopes, and for fixing tissues in histology and pathology labs, is a respiratory irritant and sensitizer. Ethylene oxide, a gas sterilant, is a neurotoxin, carcinogen, and reproductive health hazard, and has been associated with lens opacities among workers responsible for changing ethylene oxide cylinders. Thousands of health care workers were exposed to harmful levels of this gas before the OSHA standard for ethylene oxide was issued in 1984. It continues to be of concern to hospital workers in central supply because of leaks from distribution lines, especially when gas cylinders are being changed. Of particular concern is the fact that the odor threshold for ethylene oxide (260 ppm) is well above the OSHA permissible exposure limit (1.0 ppm) and the NIOSH recommended exposure limit (0.1 ppm), and approaches the immediately dangerous to life and health concentration level. In addition, ethylene oxide is highly flammable and therefore poses a dangerous fire and explosion risk.

Anesthetic agents, used in large amounts in hospitals, are a threat to health care workers when operating room scavenging systems are poorly maintained. Health care workers are also exposed when patients are transferred to the recovery room and exhale anesthesia gases. Specially designed, nonrecirculating, general ventilation systems, with adequate room-air exchange, are necessary in these areas.

Therapeutic agents associated with adverse health effects among workers who handle and administer them include hazardous drugs, such as antineoplastic agents, which are known to cause adverse reproductive effects, cancer, and other adverse effects. Safe handling guidelines have been published by the National Institutes of Health, NIOSH, and OSHA to help control dermal and inhalation exposures associated with the mixing and administration of these drugs. These guidelines state that these drugs should be prepared in a centralized area by trained individuals under a Class II(B) or Class III Biological Safety Cabinet. Proper glove material designated for use with hazardous drugs should be used because most of these substances easily penetrate regular latex gloves. Aerosolized medications pose threats because of how these drugs are administered. One aerosolized drug, ribavirin, is of particular concern because it is a potential human teratogen. Use of aerosolized medication requires use of engineering controls, such as specially designed booths and worker respiratory protection, including compliance with all elements of the OSHA respiratory protection standard.

ORGANIZATION OF WORK

Organization of work refers to management and supervisory practices as well as production processes and their influence on the way work is performed. Perhaps no other single factor influences worker injury and illness rates more than the manner in which work is organized and staffing decisions are made (Fig. 36-6). Few industries in the United States have undergone more sweeping changes in the organization of work over the past two decades than health care. Macro-level changes in the organization of the work of health care delivery have included organizational mergers, downsizing, changes in employment arrangements (such as contract work), job restructuring and redesign, and changes in worker–management relations. Many of these changes accompanied the movement toward managed care, the priority given to cost containment, and conversions from nonprofit to for-profit health care institutions. Health care reform, which is likely to have a major impact on the way in which health care work is organized and performed, provides an opportunity to strengthen worker health and safety protections.

Widespread concern about inadequate nursing staffing levels in health care facilities and its adverse impact on health care errors led to an Institute of Medicine (IOM) report that concluded that the work environment of nurses needs to be substantially transformed to better protect patients from health care errors.[16] The report recommended changes in how nurse staffing levels are established, mandatory limits on nurses' work hours, involvement of nurse leaders in all levels of management, and nursing staff input on decisions about work design and implementation. An earlier IOM report, which concluded that most medical errors result from basic flaws in the way the health system is organized, recommended that health care organizations create environments in which safety is

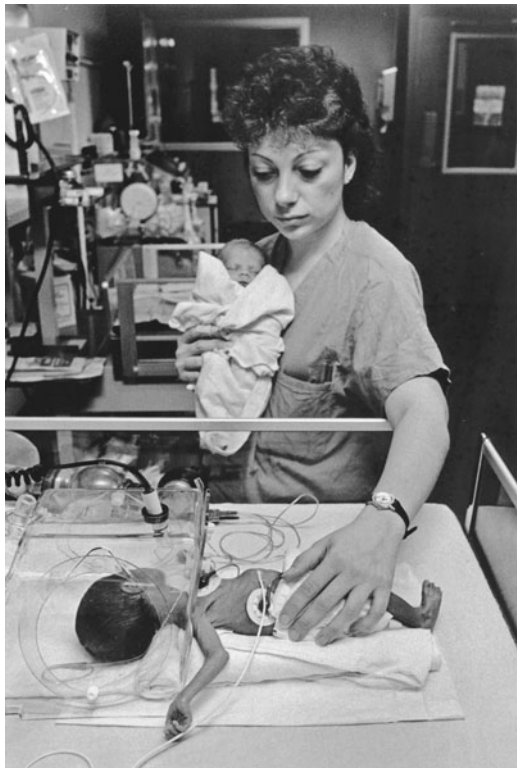

Figure 36-6. A nurse working in the neonatal intensive care unit carries one infant while attending to another. Inadequate staffing can increase nurses' occupational stress. (Photograph by Earl Dotter.)

a top priority and a feature of job design and working conditions.[17]

Despite the increased focus on patient care and nurse staffing, few studies have examined the relationship between organization of work and worker injury and illness. One study, which examined OSHA-200 worker injury and illness logs at many hospitals, found that, when nursing staff was reduced by 9%, a 65% increase in reported injuries and illnesses occurred—largely due to needlestick and back injuries.[18]

LEGISLATIVE AND REGULATORY ACTIONS TO PROTECT HEALTH CARE WORKERS

Legislation, regulations, and voluntary guidelines to protect health care workers have been slow in coming, and inadequate in their coverage. In 1958, the American Medical Association and American Hospital Association issued a joint statement in support of worker health programs in hospitals. In 1977, NIOSH published criteria for effective hospital occupational health programs. In 1982, the CDC published the *Guideline for Infection Control in Hospital Personnel*, which focused on infections transmitted between health care workers and patients—not only health care workers' risks of contracting infectious diseases.[19] CDC guidelines for Blood and Body Fluid Precautions (1983), Universal Precautions (1987),[20] and Standard Precautions (2007),[21] the latter of which combines the major features of Universal Precautions (UP) and Body Substance Isolation (BSI), were published to provide guidance to health care workers.

In 1984, OSHA promulgated its first health care worker–specific standard, covering the use of ethylene oxide. This was followed by the BBP Standard in 1991 and its revision in 2000. OSHA standards addressing tuberculosis and ergonomics were completed, but reversed. At least 10 states have enacted nurse-staffing legislation to protect both patients and workers. Despite claims that the nursing shortage has prevented employers from finding nurses, a California law has had the opposite impact: The wait time for nurses in California to obtain or renew a license increased from weeks to months—evidence that nurses are reentering the field of nursing in response to a more human and patient-friendly environment. Recent legislative efforts, including the passage of safe patient handling laws in nine states and the introduction of legislation in many states and at the federal level, may finally begin to reverse the high rates of MSDs in health care workers. Despite progress in decreasing exposure to bloodborne infections and unsafe patient transfers, it is unlikely that the high rates of occupational injuries and illnesses among health care workers will be reduced without adoption and strong enforcement of new federal regulations addressing the main hazards facing health care workers.

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FURTHER READING AND WEB SITES

American Nurses Association (ANA) Web site. http://www.nursingworld.org.dlwa/osh/.
This Web site offers information on cutting-edge issues of primary concern to U.S. nurses. It contains information on latex allergy, workplace violence, pollution prevention in health care, and other topics. It provides links to relevant Web sites.

Center to Protect Workers' Rights (CPWR). The construction chart book: the U.S. construction industry and its workers (3rd Edition). Silver Spring, MD: CPWR, 2007. Available at: http://www.cpwr.com/publications/page%2049.pdf.
An excellent compendium of statistics related to the safety and health of construction workers.

International Health Care Worker Safety Center, Charlottesville, Virginia. http://www.healthsystem.virginia.edu/internet/epinet.

The Center's Web site contains up-to-date information from its national needlestick injury surveillance program.

National Institute for Occupational Safety and Health. http://www.cdc.gov/niosh/homepage.html.

This NIOSH Web site has special sections for health care, agricultural, and construction workers. Especially useful documents on health care include the following: Violence: Occupational Hazard in Hospitals at http://www.cdc.gov/niosh/2002-101-html; Latex Allergy: A Prevention Guide at http://www.cdc/niosh/93-113.html; http://www.cdc/niosh/02-116pd.html. For agricultural workers and construction workers, there are electronic databases of available materials that are periodically updated: The National Agricultural Safety Database at http://www.cdc.gov/niosh/nasd.html and the Electronic Library of Construction Safety and Health at http://www.cdc.gov/niosh/elcosh.html.

Occupational Safety and Health Administration. Guidelines for preventing workplace violence for health care and social service workers. Washington, DC: NIOSH, 1996. Available at: http://www.osha.gov.

This document provides a succinct discussion of the background of the problem and a detailed description of the critical elements of a violence prevention program. The document provides excellent examples of how to respond to these performance-based guidelines, including a staff assault survey, checklists, and forms.

The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

37

Protecting Disaster Rescue and Recovery Workers

Dori B. Reissman and John Piacentino

This chapter describes temporal phases of disaster and emergency management in terms of worker protection strategies and health surveillance activities. Disaster safety management capacities and coordination plans must be developed before a disaster occurs to deal with the complexities of hazard assessment and control, worker education and training, worker illness and injury surveillance, and access to health care services (when prevention fails). These activities are performed by diverse groups of occupational and environmental health professionals.

INTRODUCTION

Defining Disaster

A *disaster* is defined as a serious disruption in the functioning of society that poses a significant level of threat to life, health, property, or the environment and requires outside assistance to manage or cope with it.[1,2] There are several typologies that are used to classify disasters and crisis events. Disasters may be caused by human action or by forces of nature, including extreme weather events (such as hurricanes, tornados, and cyclones), geological disturbances (such as earthquakes and volanic eruptions), and epidemics of severe infectious diseases (such as severe acute respiratory syndrome [SARS] and pandemic influenza). A *complex humanitarian disaster* is a situation in which populations are displaced due to armed conflict, dramatic political change, or other causes.[3] Technological or industrial disasters may occur from human neglect, error, or by deliberate and harmful actions.

Structural collapse, flooding, and fires are common pathways that may be helpful to consider for contingency planning purposes. Hazard assessment is of prime importance for all types of disasters. Common hazards to anticipate in planning include bulky or sharp debris, fallen trees, downed but energized power lines, structural weakening, displaced wildlife (such as rodents, reptiles, and insects), chemical or gas releases (such as broken pipelines), flooding and mold, and fires with heavy smoke and dust. National planning efforts have also considered terrorism scenarios involving chemical, biological, radioactive, nuclear, or explosive substances that could cause massive disruption or destruction.

Events Influencing National Disaster Policy for Worker Protection

Large-scale and complex disasters, such as the terrorist attacks on September 11, 2001 (9/11) and the deliberate contamination of mail with anthrax spores soon afterward, highlighted the need to organize safety management for disasters and integrate it into the management of incidents.[4–7] On 9/11 in New York City, the World Trade Center towers collapsed as a result of the high-speed impacts of hijacked commercial jet airplanes and the intense heat released as approximately 90,000 liters of jet fuel and materials inside these towers burned.[8] The response efforts were emotionally intensified by the deaths of hundreds of firefighters who had rushed inside to rescue people before the skyscrapers unexpectedly collapsed.[4] Erratic credentialing procedures, supply shortages, disrupted telecommunication systems, faulty instrument readings (due to intense smoke), and the lack of unified authority thwarted efforts to characterize the environmental contamination and better inform efforts to protect workers.[4,5] Inconsistent interpretation of data and advice about safe thresholds emerged, creating confusion, distress, and mistrust of the public institutions managing risk and uncertainties. Shortcomings in on-scene safety information, training, and enforcement made it difficult to control internal perimeters or hazard zones established to differentiate needs for training, personal protective equipment (PPE), and practices for worker protection (see Fig. 37-1).[5]

Soon after 9/11, anthrax spores were mailed to two U.S. senators and several broadcast and print journalists, leading to public health and forensic investigations in several states, large-scale preventive measures, and widespread fear.[9] Confusion ensued over divergent methodologies and authorities for measuring and interpreting environmental and occupational exposure to anthrax spores. In 2003, worker health and safety was again highlighted as SARS emerged, threatened health care workers in southeast Asia and Canada, and challenged research scientists, government officials, health care providers, and health care facilitiy managers to implement appropriate infection-control and respiratory-protection practices.[10] These events led to a number of developments in national policy for emergency preparedness and response, including the National Response Framework and Worker Safety and Health Support Annex.[1]

Description of Emergency Response Workers

Typically, the definition of emergency response workers has included firefighters, emergency medical services workers, and law enforcement personnel. However, depending on the nature and scale of the incident, additional workers contribute to the emergency response activities and include many types of professionals and skilled laborers, including electric utility workers restoring power, and road crews freeing up transportation routes blocked by fallen trees and other debris (Table 37-1).[11] Essential services are those that maintain community operations and minimize disruption from loss of power, sanitation, potable water, safe food and medicine, and needed services (such as transportation). Workers providing essential services during a severe infectious epidemic are likely to function as emergency responders and include health care providers, health care support personnel, behavioral health and social service personnel, and public health investigators and representatives of non-governmental faith-based or civic organizations who directly assist affected people. Those affected may include persons in quarantine or isolation, dependents of essential-service workers, and others, such as children, older people, medically fragile and institutionalized persons, and pet or service animals.

Federal Response to Disasters

The National Response Framework (NRF) provides a guide for how federal assets are brought to bear in response to disasters and other nationally declared emergencies.[1] The NRF is scalable and aligns key federal coordinating roles and responsibilities for emergency response, which operate in partnership with other levels of government, non-governmental organizations, and the private sector.[12] The NRF has a Worker Safety and Health Support Annex, coordinated by the Occupational Safety and Health Administration (OSHA).[13] Expert technical assistance can be called upon from many disciplines, including

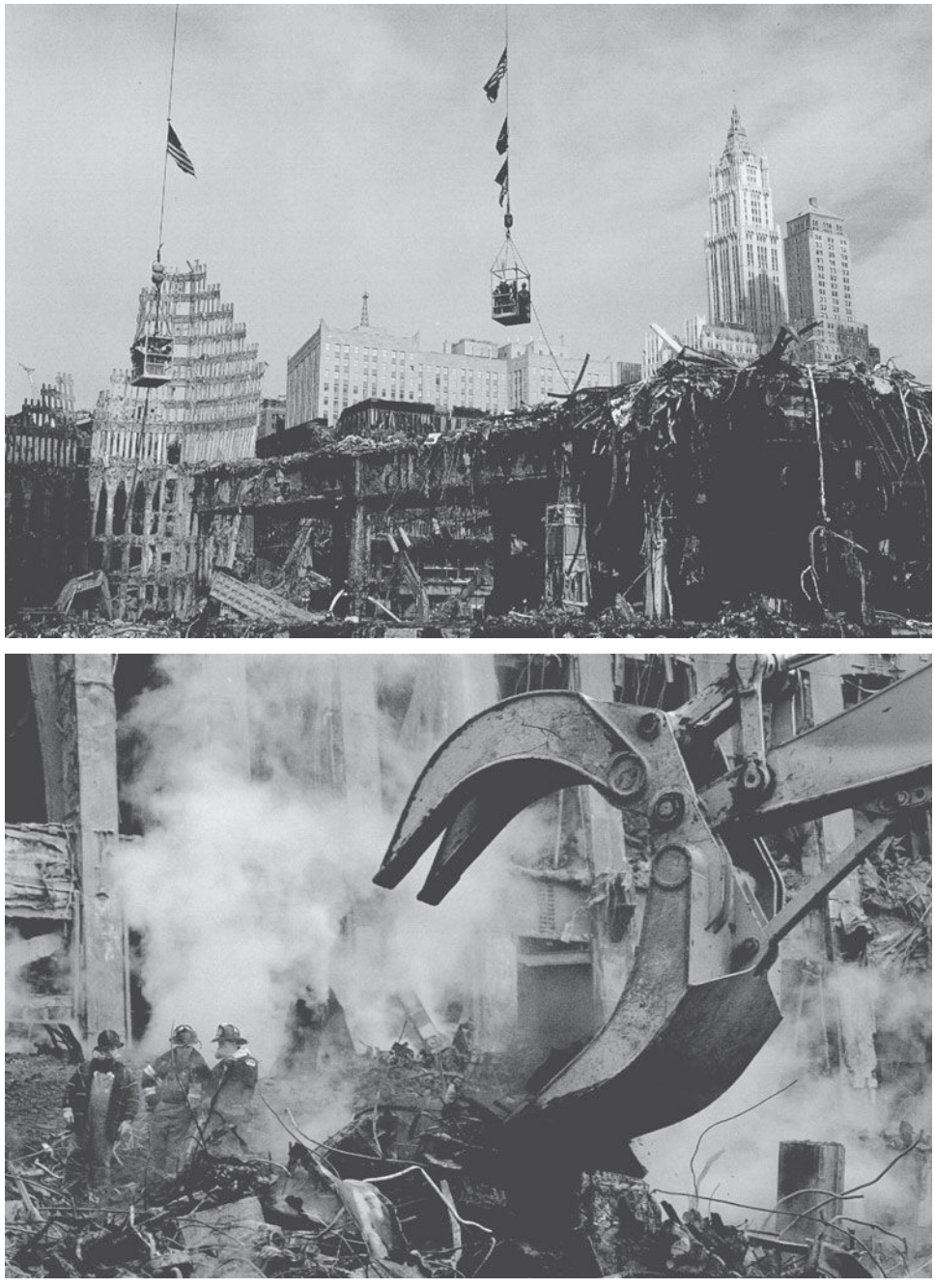

Figure 37-1. (A and B) Recovery workers at the World Trade Center site in September 2001. (Photographs by Earl Dotter.)

industrial hygiene, sanitary engineering, occupational medicine and nursing, and other safety and health professionals. The scope of activities contained in this annex addresses capabilities to identify, characterize, and control health and safety hazards; the use of PPE; on-scene worker risk communication; training; and medical evaluation. Technical assistance can also be requested with regard to medical surveillance as required by regulations, evaluating the need for long-term

Table 37-1. Job Functions of World Trade Center Responders

Traditional
Emergency service workers
Federal disaster responders
Firefighters
Law enforcement personnel
Urban search and rescue (USAR)
Nontraditional Workers
Building cleaners
Building trades workers
Civil service workers
Counselors
Engineers
Environmental assessment workers
Media representatives
Mortuary workers
Nonemergency health care workers
Pastoral care workers
Public officials
Sanitation workers
Transport workers
Veterinarians
Volunteers

Source: Reproduced from Moline J, Herbert R, Levin S, et al. WTC medical monitoring and treatment program: comprehensive health care response in aftermath of disaster. Mount Sinai Journal of Medicine 2008; 75: 67–75.

medical surveillance, identifying appropriate immunizations and prophylaxis regimens, and supporting the psychological resiliency of emergency response workers. Unfortunately, there are still operational challenges in implementing the functions guided by this annex, including when to activate this annex, which may delay hazard characterization and coordination of worker protection assets.[13–18]

DISASTER SAFETY MANAGEMENT

Before a disaster occurs, it is essential to build the capacities and flexibility required to protect workers responding to a disaster. Collectively, these functions and capabilities have been termed *disaster safety management*.[5] As workers from many disciplines and organizational entities are likely to become involved in disaster response and recovery, it is essential to coordinate the following core tasks[5,13,19]:

- Identifying and monitoring site-specific hazards (scientifically measuring and interpreting findings)
- Determining and implementing site-specific exposure control strategies
- Monitoring and reporting health and injury surveillance for disaster workers
- Facilitating worker protection education and training on scene (just in time)
- Ensuring appropriate site access controls and credentialing, especially for unaffiliated volunteers
- Ensuring adequate supply and effective use of PPE
- Ensuring access to appropriate medical and behavioral health care services

Effective worker protection policy requires coordination and cooperation among all entities involved. It is essential to have appropriately qualified and experienced occupational safety and health personnel assigned to these tasks. Safety and health plans need to be professionally designed, monitored, and tailored to specific worksite conditions. Controlling exposures to occupational hazards is the fundamental method of protecting workers. Disaster safety management needs to provide ongoing real-time guidance to disaster incident leadership at all levels of government and public works, health care, and business sectors to ensure robust standards are met to reliably protect responders. Capacity building occurs both within and among organizations (Table 37-2).

Occupational Health Surveillance

In its broadest sense, *occupational health surveillance* refers to the "systematic collection, analysis, and interpretation" of health and exposure data for the purposes of identifying cases of occupational illness or disease, monitoring trends of occupational illness or injury, and monitoring exposure to workplace hazards.[20] Surveillance also includes the timely dissemination of information to people who can implement effective prevention and control measures.

Table 37-2. Building Emergency Response Capacity

Individual	Organizational and Community
• Medical evaluation (Physical and mental health) – Fitness for duty – Medical baseline • Attainment of credentials • Education • Training exercises • Fitting of PPE ensembles • Pre-mission briefing	• Establishment of personnel tracking system • Identification of health and safety officers • Training exercises • Selection and fitting of PPE ensembles • Communication and coordination with local, state, and federal emergency response entities

PPE, personal protective equipment.

Table 37-3. Public Health and Medical Services (Emergency Support Function #8) from the National Response Framework

• Assessment of public health/medical needs • Health surveillance • Medical care personnel • Health/medical/veterinary equipment and supplies • Patient evacuation • Patient care • Safety and security of drugs, biologics, and medical devices • Blood and blood products • Food safety and security • Agriculture safety and security	• All-hazard public health and medical consultation, technical assistance, and support • Behavioral health care • Public health and medical information • Vector control • Potable water/wastewater and solid waste disposal • Mass fatality management, victim identification, and decontaminating remains • Veterinary medical support

It is a critical function in protecting emergency response workers and includes both hazard surveillance and medical surveillance activities. Hazard surveillance, medical surveillance, and screening are also critical functions of the Worker Safety and Health Support Annex. So is the function responsible for public health and medical activities (Emergency Support Function #8) of the National Response Framework (Table 37-3).[13] (See Chapter 3.)

Hazard Surveillance

Environmental sampling and monitoring are critical functions during disaster response, recovery, and remediation phases of a disaster. The goal of hazard surveillance is to characterize work-related exposures to prevent or control (limit) responder exposures. A good archival source of information about prior disasters and associated release of toxic substances is the Hazardous Substances Emergency Events Surveillance System (HSEES), maintained by the Agency for Toxic Substances and Disease Registry (ATSDR).[21,22] In more defined workplace settings, personal exposure monitoring can be conducted to better assess individual exposures, but this is often not practical in a disaster setting. This hazard information is utilized to develop strategies to limit responder exposures and inform medical surveillance activities.

Medical Surveillance

Medical surveillance is the timely analysis of health information to identify cases of occupational injury or illness or a change in biological function among workers. Early identification enables one to evaluate workplace controls or identify opportunities for workplace interventions to prevent occupational illness or disease.[20,23] Examples of medical surveillance activities include the review of OSHA-required injury and illness logs from emergency response operations or review of health data from predeployment, intradeployment (such as monitoring for heat stress or fatigue), and postdeployment medical evaluations.

Medical Screening

Medical screening is an initial examination conducted to detect unrecognized disease or organ dysfunction before an individual would normally seek medical care.[20,23] The primary purpose of medical screening is to limit disease progression by providing early treatment or limiting continued hazard exposure. Results can be considered individually or aggregated to create an understanding of novel health effects or novel relationships between exposures and health effects.[24,25]

Varying Roles of Occupational and Environmental Safety and Health Professionals

The occupational or environmental safety and health professional may assume various roles in protecting emergency response workers and/or the general public (Box 37-1). Roles often differ by specific profession (such as physician, nurse,

Box 37-1. Public and Environmental Health Issues in Disasters

Emergency management and public health share a philosophy of preventing injury and illness whenever possible. In terms of disaster preparedness, a helpful approach is to analyze the systems or pathways of the goods, services, utilities, and linkages that allow a community (or larger population unit) to function. Some experts have termed this a *lifeline vulnerability assessment*.[1] A community is able to function on a daily basis because of its lifelines, albeit the essential infrastructure.

Emergency planners should anticipate the likely disaster hazards that might occur within their jurisdictions, accounting for risks imposed by local industry, commercial transportation routing, population density, economic diversity, land-use, and regional weather or geological patterns. The types of hazards include dangerous weather (hurricanes or tornados), earthquakes, flooding (rainfall exceeding river capacity, or levee or dam breach), power loss, fire, widespread infectious disease (such as influenza), and industrial or commercial transportation incidents (such as chemical spills and explosions involving toxic materials).

Carbon monoxide poisoning can occur from power generators used within enclosed spaces, such as for heating. There are risks of injury from debris strewn about or falling from unprotected heights, or the structural integrity of affected buildings. Downed wires may still be energized, posing threats of electrocution. Wild or stray animals, reptiles, or poisonous insects may be a threat. There are risks of serious infection from improper wound care or poor sanitation. In addition, there are risks associated with exposure to extremes of temperature.

Risk communication is a critical function in disaster response (Chapter 29). Information needs to be disseminated to and understood by all persons attempting to gain access to, or remain within, a disaster-stricken area. Guidance should be widely disseminated about proper methods of sanitizing contaminated surfaces, handling flooded ventilation systems, addressing mold problems, recognizing allergies (such as to mold), handling of dangerous power tools (such as chain saws), appropriately using personal protective gear, and disinfecting private well water.

Within the first days to weeks of a disaster, regardless of whether people are living in their primary residences or in shelters, they need access to potable water, safe food, essential medicines, health care, housing, electrical power, and proper sanitation. Special populations, such as frail elderly people, children, and rurally isolated people must be considered. Surveys of the community will be required to ascertain the adequacy of these resources and to identify additional needs for outreach services to assist those less able to mobilize from their homes. Needs assessments are typically conducted within days to weeks of a disaster and involve door-to-door surveys, and often a population sampling method. Assessments need to be tied to the provision of services, as discussed later.

There are additional systems that need to be evaluated. Health and safety monitoring within makeshift housing (shelters), evacuation centers, temporary medical facilities, and schools can indicate the magnitude of the disaster impact and can affect how and if people can access necessary resources. Sheltering facilities can be evaluated for potential problems with water or food supply, improper food handling or sanitation practices, and decisions regarding high-occupancy spacing and child safety. Such inspections can identify immediate health and safety threats as well as potential catalysts for problems, including supervision of children and housing and visitation for pets. Appropriate public health communication requires an understanding of the characteristics of the underlying population, in conjunction with the environmental conditions. Staff members of facilities will likely also be experiencing stress from the event itself and from the occupancy demands within the facility. Providing risk communication and staff training via a Site Safety Officer or overriding organization (such as the Red Cross or Salvation Army) can help to clarify roles and tasks, cross-training, and control of staff work/rest cycles.

Targeted health screening and disease surveillance measures will be needed to monitor population health and to help optimally direct scarce resources. This screening or assessment must be connected to clinical services able to identify and stabilize chronic or newly emerging illnesses, injuries, and mental or behavioral health problems. The event scenario and potential human exposure(s) will inform the content and duration of such health screening and surveillance activities. Shorter term health monitoring in high-occupancy facilities will assist in early detection and mitigation of infectious disease outbreaks. Exposure to certain chemicals or ionizing radiation may require longer term monitoring for latent disease or adverse reproductive impacts.

Reference

1. Johnston D, Becker J, and Cousins J. Lifelines and urban resilience. In: D Paton and D Johnston (eds.) Disaster resilience: An integrated approach. Springfield IL: Charles C. Thomas Publisher, 2006, pp. 40–65.

industrial hygienist, or sanitary engineer), credentialing, and experience. Various roles include scientific exposure assessment and site characterization, design and implementation of hazard control strategies, worker safety education and training, health surveillance, direct patient care, and medical monitoring. In addition, these professionals may provide indirect support to workers through technical consultation, team leadership, or as an official liaison to the disaster

safety management infrastructure at varying management levels in the field or at headquarters. Such roles require a working knowledge of data collection and analytical techniques (such as root cause, fault tree analysis, failure mode and effect analysis, and basic statistics) to identify high-risk safety conditions, operations, and practices and develop trend analysis and statistical reports. Knowledge and experience would also be required to manage and lead other safety and health professional staff members, which would include human resource–related activities, work/rest scheduling, task management, conflict negotiation, and other skills pertaining to interpersonal relations, team leadership, and psychological resiliency on scene and post event.

Under the National Response Framework, the Department of Health and Human Services coordinates federal support for public health and medical activities during a disaster (Emergency Support Function #8 [ESF8]; Table 37-3).[1] Environmental and occupational health and safety professionals may also be asked to handle a variety of functions listed in Table 37-4, especially as they pertain to temporary housing (including shelters) and work facilities to ensure safe food, water, waste disposal, shower/wash stations, and vermin control.[19] Understanding the variety and interrelationship of roles will help to ensure coordination during all phases of emergency management (preparedness, response, and recovery).

Table 37-4. Emergency Response Activities Designed to Protect Worker Safety and Health during the Deployment Period

- Deployment of an appropriately credentialed health and safety officer
- Establishment of site security and control (perimeter control and designated hazard zones)
- Establishment of evacuation routes and procedures
- Registration of emergency response workers
- Site characterization (potential exposures) and job hazard analysis (personal and environmental sampling and investigation, and statistical reporting)
- Design, implementation, and evaluation/continual refinement and communication about the site-specific health and safety plan (HASP)
- Official enforcement of the HASP (compliance)
- Establishment and maintenance of OSHA 300 log to report injuries and illnesses
- Monitor crew shift length for adequacy of rest, hydration, and nutrition
- Treatment for any emerging physical or mental health problems
- Monitoring for trends with health and injury surveillance
- Review and approve the medical plan, ensuring health care services (ICS 206)
- Maintain Unit/Activity Log (ICS 214) for OSHA 300 injury/illness reporting
- Supervision of other safety personnel
- Familiarization with process to coordinate with overall disaster response management

OSHA Standards Applicable to Emergency Response and Preparedness

OSHA has identified several health and safety standards relevant to emergency preparedness and response activities.[26] Providing a complete list of applicable OSHA standards is impractical. However, many of these standards have particular importance to health care professionals given their requirements for medical evaluation and/or medical consultation. Substance-specific standards (with medical surveillance components) may also apply depending on whether the incident involves the release of a toxic industrial chemical.

Knowledge of the regulatory requirements for medical evaluation and consultation is essential to effectively protecting worker safety and health. Medical evaluations of emergency response and recovery workers should at least meet applicable regulatory requirements. For example, under the Hazardous Waste Operations and Emergency Response (HAZWOPER) standard, the content of the medical evaluation consists of a medical and work history with special emphasis on symptoms related to the handling of hazardous substances and health hazards, and to fitness for duty including the ability to wear any required PPE under conditions that may be expected at the worksite (such as temperature extremes).[27] This standard also describes the frequency of the medical evaluation, such as prior to assignment or as soon as possible upon notification by an employee that the employee has developed signs or symptoms indicating possible overexposure to hazardous substances or health hazards. In addition to developing and conducting medical evaluations, occupational safety and health professionals may also have a role in training workers. For example, under the OSHA Bloodborne

Pathogens Standard, training is required for workers with exposure to blood or other potentially infectious materials.[28]

The regulatory requirements for emergency preparedness and response represent the minimum required to protect workers. Many of the applicable OSHA standards were not designed with consideration of the emergency response environment. For example, although emergency response operations have been characterized as noisy, use of hearing protection could impact the ability of emergency responders to communicate with one another.[4,29] Not all responders are equally covered by OSHA standards. For example, state and local government employees performing emergency response, such as firefighters and law enforcement, are not covered by OSHA regulations in jurisdictions without a State Occupational Safety and Health Plan. Finally, additional considerations, such as mental and behavioral health needs, may also be necessary to more fully meet the medical needs of emergency response and recovery workers.

Hazard Control and the Site-Specific Worker Health and Safety Plan

The OSHA HAZWOPER standard (29 CFR 1910.120) requires a written site-specific worker health and safety plan (HASP) for engaging in any of the following activities:[27]

- Cleanup operations conducted at uncontrolled hazardous waste sites—as required by a governmental body, whether federal, state, or local—or other work involving hazardous substances
- Corrective actions involving hazardous waste cleanup operations at sites covered by the Resource Conservation and Recovery Act (RCRA)—actions that manage environmental contaminants released into soil, groundwater, surface water, and air[30]
- Voluntary cleanup operations at sites recognized by federal, state, local, or other governmental bodies as uncontrolled hazardous waste sites
- Operations involving hazardous wastes that are conducted at treatment, storage, and disposal facilities regulated by RCRA (40 CFR 264 and 265), or by agencies under agreement with the Environmental Protection Agency (EPA) to implement RCRA regulations
- Emergency response operations for either the release or threatened release of hazardous substances, regardless of the location of the hazard

The written HASP is important for ensuring a systematic approach to employee health and safety during hazardous waste cleanup. The HASP must detail the site's health and safety hazards, job tasks and operations, and the specific control measures used to safeguard employees. The key elements of the HASP include safety and health risk or hazard analysis and site-specific requirements for training, PPE, medical surveillance, air monitoring, site control, decontamination, emergency response plan, confined space entry program, and a spills containment program. Initial site characterization and analysis must be performed by a qualified safety and health professional in order to choose and justify engineering controls, work practices, and PPE. A medical surveillance program, which includes baseline, periodic, and exit exams, may be required for workers potentially exposed to hazardous substances. (Further guidance on the content of these medical evaluations is covered later in this chapter.) Electronic software has been developed to assist qualified health and safety professionals to appropriately prepare a written HASP ("eHASP2", March 2006, found at URL: http://www.osha.gov/dep/etools/ehasp/index.html). Given the bioterrorist activities in 2001, a HASP template was developed to help guide cleanup operations for buildings contaminated with anthrax spores from bioterrorism (http://www.osha.gov/dep/anthrax/hasp/index.html).

TIME PHASES FOR EMERGENCY AND DISASTER RESPONSE ACTIVITIES

The National Response Framework organizes emergency and disaster response activities around three functional time phases: *preparedness*, *response*, and *recovery* (Table 37-5).[1]

Table 37-5. Disaster Safety Management Life Cycle

Preparation	• Educate, train, and equip workers (anticipated hazards and command/control operations) • Establish medical baseline and readiness to deploy • Exercise and evaluate systems
Response	• Assess the situation (identify hazards and control strategies) • Deploy resources and capabilities • Coordinate activities and functions • Demobilize assets (people and equipment)
Recovery	• Short term (identify needs and provide resources) • Long term (epidemiological study and medical surveillance)

1. *Preparedness*: The preparedness phase includes activities related to building capacity in order to effectively respond to an emergency. Planning, training, acquiring equipment, and evaluating the effectiveness of emergency response capabilities through training exercises are all a part of building capacity.
2. *Response*: The response phase begins once an incident has occurred. During this phase, emergency response personnel are deployed to the site of the incident in order to mitigate its effect on life, the environment, property, the economy, and society overall. Responders engaged in immediate rescue operations tend to take greater risks in the chaotic disaster work setting, in the service of saving or sustaining the lives of those directly affected. Little time is available to assess the hazards before rescue efforts begin.
3. *Recovery*: The transition between response and recovery operations may not always be clear. In general, recovery operations begin once immediate lifesaving activities are complete and potentially life-threatening hazards are stabilized. The short-term recovery phase focuses on returning the area involved in the disaster to a functional state of self-sufficiency, with attention given to helping individuals, households, businesses, and critical infrastructure meet basic needs. Depending on the incident, the long-term recovery phase may last for months or even years.

These time phases are useful for characterizing and coordinating the major functional elements of disaster management, such as transportation, communication, and public health and medical services (Tables 37-3 and 37-4). Deployment typically refers to mobilizing assets (resources and capabilities) to help manage the disaster. However, organizational entities define their response in accordance with their oversight authorities and when their assets are deployed. Therefore, the phase terminology is sometimes difficult to apply as it is confounded by rules governing the use of different fiscal accountability mechanisms and the sociopolitical landscape of an event. The assets typically include skilled personnel and specialized equipment. The assets are safely returned as early as possible in the response phase termed *demobilization* to enable resource tracking and accountability for both resources and provisions guiding mutual aid and assistance. The term *mitigation* is also used in the field of emergency management to mean the preventive measures taken to reduce the vulnerability of a community (or workplace) to disaster consequences. Mitigation is part of the disaster cycle juxtaposed between recovery and preparedness. Examples of interventions include public and worker education, legislation, engineering design codes and standards (such as for buildings, roadways, bridges, and motorized vehicles), organizational policies, land-use management, and program-evaluation research.

Workers are involved in all of these phases. The charge to the occupational safety and health professional is to protect the health and safety of the workers participating in the emergency response, recovery, remediation, and cleanup activities. Depending on the activity, the occupational safety and health professional may have worker protection responsibilities to an individual worker (such as direct patient care) or to an organization (such as population-based medical monitoring or health surveillance). There are many aspects involved in disaster safety management, requiring diverse skill sets. Qualifications will differ with respect to the tasks required, and expertise from several disciplines will need to be coordinated to ensure that workers are protected.

Individual (Worker) and Organizational (Employer) Preparedness

The workplace can be significantly harmed by a disaster, an act of terrorism,* or other traumatic incident.** The scope and scale of anticipated events may require a thorough systems-level vulnerability analysis, from raw material supply chains to market delivery of final product or services, including disruption in transportation routes required for workers to be on the job.[31] Preparedness encompasses the period of time before a worker or an organization is engaged in emergency response activities. At the individual level, a worker must acquire and maintain appropriate education, training, and certification (credentials) specific to his or her potential deployment+ roles. Medical, emotional, and cognitive readiness are important dimensions of workforce health protection planning.

At the level of organizational preparedness, activities focus on ensuring that qualified workers are ready and appropriately outfitted with personal protective ensembles for deployment. Activities are also designed to build an integrated response capacity within an organization and within a community (Table 37-2). Effective disaster safety management requires appropriate infrastructure and interagency planning and coordination before the emergency arises. Predeployment and preparedness activities shape and influence the overall success of the emergency response efforts, and, in turn, the disaster recovery process.

While the field of emergency management encourages an all-hazards approach to planning, it is not uncommon to organize the core plan around the more likely emergency events, such as an industrial explosion or fire, a facility collapse, violence by a disgruntled employee, or a natural disaster that destroys or disrupts business operations, such as flooding, hurricane-strength winds, or an earthquake. The advent of infectious disease epidemics, such as SARS or H1N1 (swine) influenza, place additional life demands on workers who must care for ill or dependent family members, especially if schools are closed by public health officials to slow down the spread of infection. While business continuity is a key component of such planning, worker safety and health must be incorporated into strategic thinking. The cost of not doing this can be quite high, including loss of specialized workers with a need to recruit and train new staff members; interim loss of productivity; workers' compensation costs for job-related injury, illness, or disability; and other potentially cascading organizational effects, such as loss of morale.

The day-to-day stress in the workplace can impact health from more cumulative factors within worksites, such as how a job is designed, organizational structure, management style, management and co-worker commitment to safe work practices (safety climate), and the availability of adequate resources and support to achieve the mission.[32] Disasters and large-scale emergencies also create competing life demands on a worker to ensure the safety and welfare of loved ones. Disasters erode normally protective supports, increase the risks of diverse problems, and tend to amplify preexisting problems of social injustice and inequality within affected populations.[33] The disaster may provoke new social problems for residents and local business employers by separating families, disrupting social support networks, and compromising critical infrastructure, such as providing essential goods and services (water, food, medical care, power, and housing/facilities).

Integrating Psychological and Behavioral Risk Management

Worker safety and health practitioners must integrate psychological and behavioral risk management strategies into crisis and contingency planning; these require knowledge of the psychological and behavioral consequences of

* *Terrorism* is a policy or ideology of violence intended to intimidate or cause terror as a means of exerting pressure on decision-making by a governing body (an ideological goal). The term *terror* is largely used to indicate clandestine, often low-intensity violence that targets (or disregards the safety of) civilians and generates public fear.

** A *traumatic incident* is a situation that is perceived as personally threatening to one or more persons. Individual perception of what constitutes a traumatic incident may vary from person to person. Typical examples include witnessing murder or serious injuries, and hostage situations.

+ *Deployment* refers to activation of an emergency response role or function, and strategic movement of assets (responders and their logistical support) to achieve the mission.

Table 37-6. Lessons Learned from Prior Disaster Response to Anticipate Psychological and Behavioral Health Hazards and Service Needs

- It is difficult to prepare responders for everything they might encounter.
- Even seasoned responders can face situations and issues that cause uneasiness and distress.
- It is not unusual for responders to be asked to work outside their areas of expertise.
- Concerns about family members and friends rank high on responders' lists of priorities.
- Timely, accurate, and candid information should be shared to facilitate decision making.
- Managers, at every level, need to consider the health, safety, and resiliency of workers on the job as part of situation awareness and for staged planning. This implies the need for occupational health and wellness monitoring.
- Resiliency is an integral component of occupational safety and health, which requires preplanning to maximize worker recovery.
- Self-care plans and peer-support activities are essential to mission completion.
- Everything possible should be done to safeguard responders' physical and emotional health.
- Responders do not need to face response challenges alone. They may share their experiences with buddies, teammates, family members, and colleagues.
- It is especially difficult for responders to maintain emotional distance when they witness the deaths of children.
- Organizational differences among groups of responders and cultural differences between victims and responders can impede the timely and efficient provision of emergency services.
- Individuals may be thrust into leadership roles for which they have had little to no formal training.

Sources: See references 11, 34, 36, 38, and 46.

the disaster, terrorism, or other traumatic incident (Table 37-6).[34,35] Traumatic incidents and disaster exposure can increase the risk of distress reactions or dysfunction, behaviors increasing risk to health and safety, or psychiatric illness. It is helpful to anticipate needs for psychological interventions within the workplace and do the homework required to ensure that potential techniques are based on empirically defensible or evidence-based practices and are conducted by qualified individuals.[36,37] Interventions include leadership initiatives, administrative policies, and enhancing services by partnering with other organizations that can help provide and/or train others to provide psychosocial support services as needed, such as stress, anger, and grief management, and crisis intervention counseling.[34,36,38]

Predeployment Medical Evaluation

The medical evaluation of emergency response workers, which ensures workforce protection and contributes to the overall success of emergency response efforts, is an integral part of preparing workers for emergency response activities. This includes an assessment of both mental and physical health. The purposes of the medical evaluation during the predeployment period are to assess the readiness (fitness for duty) of the responder to be deployed and to establish a medical baseline for future comparisons.[35,39] The fitness-for-duty determination should take into account whether the worker will be able to perform the essential job functions without posing a threat to self, others, or the mission at hand. This determination should be made in compliance with medical and legal standards as outlined in the Americans with Disabilities Act.[40]

Some emergency response workers may already be enrolled in medical surveillance programs as a part of their routine employment. A national standard exists for the initial and annual medical evaluation of fire service personnel providing rescue, fire suppression, emergency medical services, hazardous materials response, and other services.[41] The National Fire Protection Agency Standard on Comprehensive Occupational Medical Program for Fire Departments (NFPA 1582) describes a comprehensive medical evaluation for members and recommends an initial and annual evaluation. Additional medical evaluations are recommended after an occupational injury, illness, exposure, or extended absence from work. In addition, some states have outlined medical evaluations for law enforcement personnel.[42] Finally, some emergency response workers may wear respirators as part of a PPE ensemble to carry out routine job functions, such as medical personnel caring for potentially infective patients.

Prior participation in a medical surveillance program may be a useful source of information for establishing a medical baseline and even making a fitness-for-duty determination. However, depending on the date and content of the evaluation, this information may not be accurate or sufficient to accomplish the goals of the predeployment medical evaluation. Moreover, some emergency response workers may not have received any prior medical evaluation.

The timing and content of the predeployment medical evaluation is guided by knowledge of the anticipated emergency response environment. This environment may be characterized by multiple hazards and a variety of job functions. In addition, workers may be donning a new or modified PPE ensemble and may require additional screening to address the potential for heat stress or other potential complications. Finally, depending on the hazards encountered or recommendations for PPE, regulatory requirements for predeployment medical evaluations may apply.

Conducting a medical evaluation immediately prior to deployment may present logistical difficulties and could even delay worker deployment. Despite the variety of hazards, job functions, PPE ensembles, and regulatory requirements, the content and timing of the predeployment medical evaluation should be sufficient to make a fitness-for-duty determination and establish a medical baseline.

A fitness-for-duty determination states whether a worker has a medical condition (physical or mental) that is likely to interfere with the anticipated disaster or emergency response activities, or pose a threat to self or others. To make this determination, attention should be paid to the following clinical elements:

- Active symptoms or complaints
- Current and past medical and psychiatric conditions (likelihood of episodic exacerbation)
- Functional limitations
- Current medications and treatment regimens (state of medical control and risk of decompensation)
- Immune status (required vaccinations)
- Misuse of nonprescribed substances, including nicotine, alcohol, and street drugs
- Contraindications to postexposure prophylaxis or treatment regimens

Active Symptoms and Complaints

Workers who are actively symptomatic may not be able to be deployed given the potential for medical or psychiatric decompensation, communicability, functional limitations, or any undue risk when placed in the disaster or emergency response environment (and away from usual supports).

Medical and Psychiatric Conditions

The identification of current or past medical conditions, including pregnancy, and psychiatric conditions may not represent an immediate contraindication to deployment. However, consideration should be given to the potential for medical or psychiatric decompensation or episodic exacerbation, once subjected to the anticipated emergency response environment (including long work hours and disrupted sleep and nutritional patterns). In addition, restricted duties may be indicated if a chronic illness, such as hypertension, coronary artery disease, depression, anxiety, and diabetes, is poorly controlled, or if a potential responder lacks psychological flexibility and stress tolerance (coping) or other factors that could impact on the well-being of the worker or team and mission success. Such considerations are especially critical in chaotic, extreme, or austere environments where outside help may not be immediately available or access to water, food, or electrical power is limited.

Functional Limitation

Functional limitation, such as decreased range of motion, loss of strength, or poor cardiovascular fitness should be identified during the medical evaluation, and any work restrictions should be noted as part of the overall fitness-for-duty determination. It is also important to assess for limitations imposed by predeployment fatigue and cognitive impairments (memory or critical thinking skills) that could jeopardize the responder, others, or the mission at hand. For example, problems with thinking or memory may arise from specific psychiatric disorders or symptoms may arise from misuse of chemical substances or noncompliance with therapeutic regimens.

Current Medications and Treatment Regimens

Side effects, storage requirements, adequacy of supply, and time-sensitive dosing should all be considered when making the fitness-for-duty determination. In addition, some routine treatment regimens may be impacted by the response environment, such as continuous positive airway pressure (CPAP) machines or needs

for refrigeration and limited access to electricity. Given that emergency response can involve PPE ensembles, medication regimens (such as diuretics and anticholinergics) should be evaluated for their potential to contribute to the risk for heat stress.

Immunizations and Postexposure Prophylaxis

Effective vaccination is an essential element of workforce protection. Tetanus and hepatitis B vaccination is currently recommended by the Centers for Disease Control and Prevention (CDC) for emergency response workers. Immunization against hepatitis A, typhoid, cholera, meningococcal disease, and rabies is not recommended for disaster responders in the United States.[43,44] Predeployment medical evaluation should also assess for contraindications to likely postexposure prophylaxis or treatment regimens, such as allergy to an effective antibiotic in the context of a suspected release of anthrax spores. Prior knowledge of contraindications to postexposure treatment or prophylaxis regimens will be helpful to limit the potential for adverse side effects.

Substance Misuse or Dependence

Safety and health can be compromised by smoking, nicotine, alcohol intoxication or withdrawal, or the use of other illicit substances. The predeployment medical evaluation provides an opportunity to identify and intervene with such health-risk behaviors.

It is important for the predeployment medical evaluation to contain sufficient information to establish a medical baseline for future comparison. Standard elements include a comprehensive medical history and physical examination. Special attention should be paid to the occupational history, including prior job duties, exposures, PPE ensembles, length of employment, and prior occupational injury or illness. Biological monitoring and extensive testing should be targeted to likely or anticipated hazards during deployment(s).

Emergency Deployment

Emergency deployment encompasses the time period when a worker is actively engaged in responding to an incident. Very little time may be available for preparation of personnel prior to deployment (usually a week or less). Individuals preparing to deploy should receive a pre-mission health and safety briefing that includes guidance about anticipated hazards (physical, chemical, biological, and interpersonal/psychological). Predeployment situational awareness should be provided via updated reports and/or materials designed for rapid dissemination. Depending on the nature of the incident, the Worker Safety and Health Annex of the National Response Framework may be activated.[13] During the deployment period, characterization of the hazard environment begins. For example, environmental and personal sampling may be employed to understand the composition and concentration of hazardous materials released in a dust cloud after structural collapse, and/or structures may be evaluated for instability. Given that the predeployment medical evaluation is based on an understanding of likely or anticipated hazards, it is important for the occupational safety and health professional to gather information about any hazards to which the deployed worker may be exposed. This information will be an essential consideration when designing and implementing a medical screening program for use during the postdeployment period.

Similarly, selections of PPE are also made prospectively. Unanticipated hazards may not be adequately addressed by preselected PPE ensembles, thus resulting in risk to emergency response workers. In addition, most PPE is designed to function during a short-term incident. However, workers responding to the World Trade Center incident reported equipment failure and fatigue and heat exhaustion due to sustained PPE use during the extended response period.[4] Disaster response activities designed to protect workers are listed in Table 37-4. Emergency response personnel are often required to work extended hours in high-risk environments, where alertness and attention to detail are absolute requirements for safe work practices. Elevated stress and fatigue can lead to faulty decision making, unsafe work behaviors, and increased exposures to health hazards.[45] Optimally, the responder has emergency plans and systems in place to handle concerns about the safety and welfare of family members and other loved ones to avoid

both fractured attention on the job and increased likelihood of accidents, improper work practices, and poor decision making.[46]

Demobilization

Demobilization is a process that provides closure to the deployment period for both the responder and the organization. At the individual level, it includes the exit interview for the responder and verifies information collected about possible exposures, health or injury events, and follow-up contact information. Information is shared regarding mission successes and challenges for operational continuity (lessons learned), potential health effects, and available health services and resources. Deployment-related illness and injury should be aggregated and analyzed with respect to geography, process, and time to help inform the need for medical surveillance or epidemiologic investigation. For the organizational perspective, demobilization focuses on the withdrawal of deployed assets, including workers and their equipment. Similar efforts can be implemented organization-wide to absorb health and safety performance lessons to aid a continual quality improvement process and to further demonstrate the interest and commitment of organizational managers and leaders.

Postdeployment

The postdeployment period begins once responders return to their routine work and extends forward in time as long as is practicable to understand health and safety impacts from disaster response work. Characterizing the health and safety impacts from disaster response work is generally accomplished through hazard surveillance during the response, combined with follow-up assessment and clinical services, such as physical examinations, medical screening, and psychological resiliency services. Responders should be kept abreast of the availability of these resources, as well as any entitlements or legal rights. Prior responders are encouraged to report and answer surveys regarding emerging adverse health events potentially related to deployment. Under the Worker Safety and Health Support Annex of the National Response Framework, OSHA has the responsibility to coordinate the federal response in "providing technical assistance, advice, and support for medical surveillance and monitoring as required by regulation (e.g., asbestos and lead) and evaluating the need for longer term epidemiological follow up and medical monitoring of response and recovery workers."[13]

The need for medical screening or more extensive health surveillance may be triggered through hazard surveillance activities conducted during the response as well as the emergence of concerning health effects in significant numbers of responders. Although it is not possible to describe a medical screening program suitable for all types of emergency response operations, the design and implementation of such a program should adhere to some standard elements[20]:

- Assessment of workplace hazards
- Identification of target-organ toxicities for each hazard
- Selection of a screening test for each health effect
- Development of action criteria
- Standardization of the testing process
- Performance of testing
- Interpretation of test results
- Test confirmation
- Determination of work status
- Notification
- Diagnostic evaluation
- Evaluation and control of exposure
- Recordkeeping

The nature of the emergency response environment presents several challenges to successfully designing and implementing an effective medical screening program, as described next.

Assessment of Workplace Hazards

The hazards of an emergency response may not yet be fully characterized or effectively communicated to clinical occupational safety and health professionals. Given that medical screening is often administered to workers without symptoms or at a point when overt disease is not fully recognized, it is essential that the screening program address likely health effects. Assessments of likely health effects are most reliable when based on knowledge of the hazardous exposure.

Identification of Target Organ Toxicities, Selection of Screening Tests, and Development of Action Criteria

Even when hazards have been characterized, they may be diverse and complex. For example, hazard surveillance of the region impacted by hurricanes Katrina, Rita, and Wilma in 2005 identified exposures of noise, dust, asbestos, silica, formaldehyde, and carbon monoxide (see Fig. 37-2).[29] The variety of hazardous exposures of emergency response workers can lead to an overly broad medical screening evaluation with a consequent decrease in the program's performance with respect to specificity and positive predictive value. Thus, care should be taken to identify target organ toxicities, select appropriate screening tests, and develop action criteria for test results.

Standardization of the Testing Process, Performance, Interpretation of Test Results, and Test Confirmation

Emergency response and recovery operations involve the coordination of multiple worker populations, employers, and organizational authorities. Demobilization of workers from the emergency response environment can include the dispersion of response workers over a large geographic area and/or the distribution of workers among various employers. Differences in geographic distribution and employers can give rise to differences in access to postdeployment medical screening as well as differences in medical personnel performing testing. Despite these logistical difficulties, it is important to standardize the elements of the testing process to maximize its utility to the medical surveillance program and the affected workers.

Determination of Work Status, Notification, and Diagnostic Evaluation

The decentralization of emergency response workers can also lead to variations in clinical evaluations, such as effectively addressing work status, performing diagnostic evaluations, and notifying workers of clinical results and injury and illness trends. It can also impede the effective

Figure 37-2. A team consisting of a National Institute for Occupational Safety and Health physician and an industrial hygienist survey work conditions inside the New Orleans Superdome during the Centers for Disease Control and Prevention disaster response after Hurricane Katrina in September 2005. (Photograph by Aaron Sussell.)

communication of hazard surveillance data. Again, despite these logistical difficulties, it is important to standardize, to the extent feasible, determinations of work status, notification, and diagnostic evaluation.

Evaluation and Control of Exposure, and Recordkeeping

The chaotic nature of the emergency response environment, the diversity of responding entities, and the redistribution (dispersion) of the worker population postdeployment make accurate evaluation and control of exposures and recordkeeping especially difficult. Basic requirements, such as knowing who worked at the site, how long they worked there, what they did, and whether they wore PPE, are essential to correlating workplace exposures with occupational injury and illness and communicating health and safety information to responding organizations and workers.

Recognition of these challenges is helpful when developing or attempting to develop a postdeployment medical screening program. For example, individuals charged with developing medical screening programs should attempt to acquire relevant hazard surveillance information. This information may come from federal, state, or local emergency response agencies; questionnaires administered to workers; or preincident hazard vulnerability assessments. When attempting to address incidents with multiple hazards, it is important to emphasize an approach that will clarify worker exposures. Detailed questionnaires or an effective interview process can help to limit the number of considered hazards. Finally, the establishment of a site roster, along with timely review of site records can help identify who was at the worksite, keep track of the duration of the emergency response activities, and provide an accurate description of job duties, exposures, and PPE ensembles.

Workers engaged in emergency response and recovery activities should receive some level of medical assessment during the postdeployment period. Depending on the nature of the incident, the medical assessment may be a simple questionnaire designed to clarify and record self-reported hazard exposures, injuries, or illnesses; job duties; length of deployment; and utilization of PPE ensembles. The assessment may need to be a more comprehensive medical evaluation with a physical examination, medical testing, and additional history elements. The medical baseline established during the predeployment medical evaluation is fundamental to allowing comparisons in health status before and after emergency response operations.

CONCLUSION

Occupational and environmental health and safety professionals are essential to protecting the health and safety of workers participating in emergency response, recovery, remediation, and cleanup activities. They are involved in all phases of the disaster safety management life cycle, from building response capacity during preparation to conducting occupational health surveillance during recovery. Therefore, it is important to appreciate the diversity of functions performed by health and safety professionals and the characteristics of the emergency response environment to ensure effective coordination. Ultimately, this will lead to increased protection of workers and help ensure the success of the overall response.

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The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

38

Implementing Programs and Policies for a Healthy Workforce

Martin G. Cherniack and Laura Punnett

Occupational medicine has infrequently addressed chronic diseases that are not directly related to work. Yet health problems that accompany normal aging, such as cardiovascular disease (CVD), degenerative osteoarthritis, and depression, may also be influenced by the psychosocial work environment and other job exposures.[1–3] Occupational medicine has not yet tapped the full potential of using the workplace as a venue for prevention of chronic nonoccupational diseases and for promotion of overall health and well-being.

The concept of Working Life involves designing and implementing workplace programs to prevent chronic diseases or their complications.[4] The continuum of approaches encompasses those that are traditionally part of workplace health promotion as well as those of occupational health, although it is the genuine integration of these that makes the whole greater than the sum of its parts. Working Life programs may include the following:

1. Worksite health promotion and disease management programs that promote healthy lifestyles or encourage compliance with appropriate clinical treatment, including direct provision of medical care, screening, and clinical services
2. Programs that mitigate or prevent chronic disease processes influenced directly by work, such as ergonomics programs and reorganization of work processes to reduce job stress
3. Programs that address aspects of work affecting quality of life, such as government or employer accommodations for sickness and family leave, child welfare and child care, continuing education, transportation, and housing

HISTORICAL CONCEPTS OF WORKING LIFE

The Working Life concept is not new to the occupational health field in Great Britain and the United States. In 1830, the British physician Charles Turner Thackrah published a masterpiece of occupational medicine that described the effects of urban industrial life on the newly developed working-class population of Leeds, England.[5] Thackrah's world has very little in common with the urbanized workforce of developed countries today. However, his areas of interest affirmed the linkages between occupational medicine and both general medical care and public health. His concerns included controlling dust-related diseases in mines and

tuberculosis in milliners, as well as child development in working families.

In the United States, there was much public health activism in the early twentieth century that emphasized the relationship among work, socioeconomic status, and morbidity and mortality. Both Alice Hamilton[6] and Isaac Rubinow[7] discussed the high rates of death and disease in working families compared to more affluent families, who could afford a healthy lifestyle, including good nutrition, access to medical care, safe and healthful working conditions, and adequate leisure time.

The simplest component of work organization is working hours. Some of the earliest interventions by occupational health physicians in England and the United States involved limitations on the work week and prohibitions of child labor. Over the past 100 years, the average work week has been reduced dramatically. However, the most recent pattern has been a gradual increase of working hours. In 1900, the average work week in manufacturing in the United States was 53 hours. During the Great Depression, the average work week for production workers in manufacturing decreased to 35 hours. During World War II, it increased to 45 hours. From 1977 to 1997, the average work week for full-time workers increased from 44 to 47 hours. There were further increases until 2007, when an economic recession began.

In Sweden in the 1970s and 1980s, there was a broad-based effort to alter work organization and work role based on principles of health and quality of life and reflective of social democratic traditions. This has differed from the United States, where market theory presumes the moral hazard of generous social welfare interventions and the prerogatives of corporations within workplaces. It also differs from some liberal and Marxist traditions, which presume a necessary relationship among technology, division of labor, and work roles. Work structures—and therefore work-life accommodations—should be similar at equivalent levels of development in industrial countries. The Scandinavian model has put less emphasis on deterministic laws and a greater emphasis on managerial choice in designing work organization as well as work and nonwork relationships. Much of this was reflected in the Swedish Work Environment Act of 1978, which placed equal emphasis on the physical and psychological health of the workforce and involved labor organizations in the design of work conditions.[8] In Denmark, similar programs addressing quality of work life were influenced by the European Health and Safety Directive of 1989.[9]

In 2003, Sweden and Denmark had high ratings for quality of work life and for workers' self-assessment of their impact on decision making. These and other Scandinavian countries also ranked high in other priorities, including training opportunities, avenues for professional development, and job security. However, Sweden, in particular, has since experienced dramatic reduction of working-life initiatives and elimination of its federal occupational health research institution.

The European Union (EU) has introduced a cross-national emphasis on common concerns, such as disability and employment as well as aging of the workforce. For example, it has established more generous retirement and disability benefits and an earlier age of retirement than in the United States. These policies are thought to account for the higher unemployment in Europe among older age groups: 45% of adults age 55 to 64 in the EU are employed, compared to 59% in the United States. In the EU, only 9% of people age 65 to 69 work outside the home, compared to 30% in the United States. In addition, 20% of EU workers leave employment because of "working life" issues, such as illness and family responsibilities.

The EU also focuses on transnational health and safety regulations. For example, its physical hazards directive emphasizes transnational standardization of tools, exposure standards, and disease surveillance, although management of work-related diseases and injuries is left to the individual companies or national worker-protection programs. In contrast, in the United States, weaker governmental protections and employer provision of health insurance has led to more investment in Working Life programs by the private sector than by government.

Work–life (or work–family) imbalance refers to the adverse effects of work demands and stress on family life and health.[10] In the United States, it assumes a sustainable equilibrium, at least partially controlled by workers who can personally

recalibrate this equilibrium. However, in Europe, it has been presumed that stability in working populations can only be maintained by legal protections and interventions by a welfare system. These differing traditions are reflected in legislative protections. In the United States, there is no a federal law requiring paid sick days or paid maternity leave. (However, the pending Healthy Families Act would require employers with more than 15 workers to provide their workers with 1 week of sick leave annually.) In contrast, Norway provides 96 weeks and Denmark 52 weeks of paid maternity leave.

The European Foundation for the Improvement of Living and Working Conditions ("Eurofound") conducts surveys and makes policy recommendation throughout the EU to develop a balance between family life, personal commitments, and working time.[11]

The Eurofound has identified three key areas for policy development: career and employment status, health and well-being at work, and career development. Work–life balance features the reconciling of work–family conflicts, especially with progressive policies concerning child care, working time, and parental leave.

Because economic level, technology dissemination, and divisions of labor are somewhat parallel in countries with similar levels of industrial development, there are some common themes between Europe and the United States in the concept of Working Life. The situation in the United States has some particular emphases or distortions because of the more limited role of government in determining working conditions and work–life considerations, such as child care. Employers also play a greater role in the subsidization of health care. Therefore, there has been a particular emphasis on more individualized aspects of work–life balance, such as flexible working hours, especially in companies where retention of a skilled workforce is paramount. There has also been an emphasis on health promotion and lifestyle changes that may improve health status and lower costs of medical care.

In the United States, the concept of Workplace Health Promotion (WHP) encompasses two core approaches that are sometimes in conflict. The more common approach envisions health and the prevention of chronic disease as largely a function of individual behavior and adverse lifestyle choices or dispositions. The focus is on individual responsibility and lifestyle change—diet improvement, exercise, weight loss, and smoking cessation (Fig. 38-1). The workplace is seen as a venue where programs can be developed and implemented, and wage-and-benefits systems can be the source of incentives. Typical services may include influenza immunizations, blood pressure and serum lipid measurements, and disease-awareness events. Assessment and tracking services may be offered onsite by vendors, or online. Sometimes WHP may include employee assistance program (EAP) services, disease management (such as for medication compliance), and cancer screening services.

A second approach to WHP envisions health as a reflection of individual behavior, social and family factors, and environmental conditions, including work quality and reward. Some factors are therefore outside individual control, requiring strategies that involve the nature of work and social considerations, such as family responsibilities. There may be an emphasis on organizational relationships at work, job stress, accommodations for older workers, and roles for employees in making health decisions. It is understood that the workplace influences health, such as by the physical and psychosocial organization and design of work, with particular attention to job stress, cardiovascular disease, and depression.

Most WHP practice in the United States has been based on the first of these two approaches, the individualized perspective. However, limited personal resources often affect the capacity to make lifestyle changes, especially for low-wage workers. Organizational culture and managerial initiatives on health are complicated and their success is influenced by both organizational size and available resources. The term *culture of health* is sometimes used to describe WHP initiatives that go beyond a focus on individual workers.

In practical terms, these two approaches to Working Life and behavior change are contained in the unifying concept of productivity. The concept does not, however, bridge the apparent distinctions, as it has meant a variety of things, including units of output/cost and the skill and decision-making qualities of a workforce. A general scheme is detailed in Figure 38-2.

Figure 38-1. Workers exposed to pesticides and other occupational hazards may also face serious health risks from lifestyle factors, such as smoking. (Photograph by Earl Dotter.)

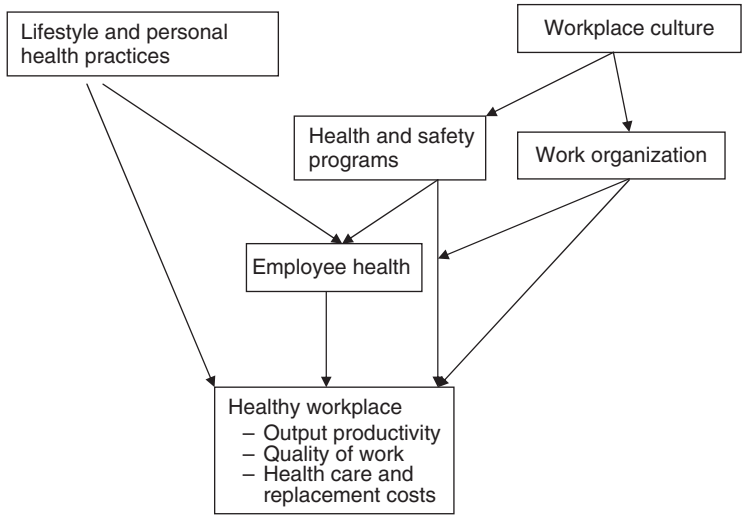

Figure 38-2. Working life and the productivity argument. (*Source*: Martin Cherniack.)

WHP in the United States and WHP in the European Union share the common feature of having evolved functionally and episodically, rather than by legislative act. The motivations and approaches have substantial differences, however. In the United States, a main motivation has been high health-care costs and their potential assumption by employers. In the European Union, the concern has been primarily with workability and, accordingly, the high public costs of disability and unavailability of the workforce in prime working years. U.S. approaches have emphasized lifestyle and individual health practices, whereas the European

approach has focused on work organization and the work environment. In the European Union, employers are relatively more sheltered from direct health-care costs. In the United States, a more contingent workforce is deemed acceptable, but high health-care costs are not.

THEORIES RELATED TO INTEGRATION OF WORK AND NONWORK LIFE

The Sociobiological Model

A social gradient predicts morbidity and mortality.[12] The effect is not limited to all-cause mortality; it includes many chronic diseases, such as cardiovascular and cerebrovascular disorders. Occupation and Working Life represent a major component of the health gradient observed with social hierarchy, education, and income.[13] During midlife, social inequalities exert their greatest effect on health.[14] Possible explanations for this finding include the extent of social network and support, high demand and low control at work, imbalance between workplace effort and reward, and traumatic life events, such as unemployment.[15–19]

Incorporating these concepts, some experts oppose the conventional sociological view of fixed external constructs with the concept of the "psychosocial environment," meaning the social and structural range of opportunities available to the individual to meet core human needs: well-being, productivity, and positive sense of self.[20] They take the perspective of health self-efficacy—that a positive experience of self relies on one's social environment.[21] In this context, work and nonwork life are critical components of a communitarian whole. While this form of analysis may appear "soft" compared with the biomedical exposure-based perspective of traditional occupational medicine, stress effects on the individual worker involve both intrinsic physiologic components and extrinsic combinations of factors that cannot be physically measured directly.

At the core of these models are the two major stress models, the Demand-Control Model[22] and the Effort-Reward Imbalance Model[23] (see Chapter 14). Their application to the broader concept of Working Life also has come from the expansion of the model of high demand and low control to home life. Low control at home predicts coronary artery disease in women (although not in men); it appears to result from a lack of material and psychological resources.[24] A more conventional way of looking at work–family conflict may underestimate important interactive effects, with women's representation in the workforce being amplified in work where there are significant time demands, limited economic rewards, and limited job control.

Stages of Life

Working Life has been indirectly addressed in epidemiology through the Stages of Life concept,[25] which refers to biological, behavioral, and psychosocial influences that occur over the life course, including past-generational and genetic patterns. A key concept is that there are critical periods, after which there may be irreversible adverse health effects. Although the major emphasis has been on early childhood exposures, there is also a "chain of events" concept that addresses critical adult-life stages. For example, job loss can lead to family strain, physical abuse, and divorce. Adverse events in critical or physiologically sensitive periods may be linked, either additively or by triggering a critical health event. The quality of health risks and composition of working life demands vary in different age groups. In younger workers, child-care and financial concerns may predominate. Later in life, family responsibilities may shift to elder care and personal management of chronic diseases. Implicit in this concept is the interaction among broad national and social trends, the workplace and the family, and individual health.

Aging and Work

Because clinical expression of chronic diseases begins to increase in the fourth decade of life and because the workforce is aging, there has been an increased public health focus on work and aging. The fraction of U.S. workers older than 55 is increasing from 13% in 2000 and 17% in 2010 to a predicted 19% in 2050.[26] In 2010, 48% of the U.S. workforce was female, with the

largest aggregate increase in the 55–64 age group (which will increase by 25% by 2020).

The term *age management* explains adaptive accommodations and corrections for aging workers in the contemporary industrial workplace. It presumes that control of personal, organizational, and biomechanical risk factors in the workplace can accommodate age-related changes in physical function. The combination of longevity and altered economic conditions has made older workers (age 55 and older) the most challenging aspect of workforce management. Critical elements of the Working Life concept are preservation of physical and mental capabilities in older workers and necessary accommodations to changes in physiological functioning and the greater need for chronic disease management.

As workers age, clinical symptoms increase and their functional capabilities decline. For example, as people age they lose muscle strength and have an increased prevalence of musculoskeletal disorders. However, progression of diminished function with age is not uniform. In one study,[27] among Finnish municipal workers older than 45, exercise capacity—a strong predictor of sustained employment—declined by more than 20% over 5 years, compared to an expected annual decline between age 30 and 70 of less than 1%. Over 10 years, one subgroup experienced a 50% decline in isometric trunk strength and spine mobility after age 45—more than four times the comparison rate. Trunk strength and function appeared to be especially predictive of work capacity in the fifth and sixth decade. Targeting interventions to workers with the most marked decline in musculoskeletal function mirrors community health interventions where the greatest yield comes from addressing problems in those at highest risk.

WORKING LIFE IN RELATION TO OCCUPATIONAL HEALTH AND SAFETY

A focus on a healthy workforce and worker well-being requires a different perspective on employee health and safety than that of traditional occupational health and safety protection programs (Table 38-1).

For a long time, there has been a separation between those concerned with control of hazardous occupational exposures and resultant health risks and those concerned with reduction of individual and community health risks primarily through behavioral change.[28,29] Labor unions and occupational health specialists sometimes regard workplace-based health promotion programs as a diversion of scarce resources from investment in safer equipment and work processes. By emphasizing a focus on a few high-risk workers, the WHP approach can distract attention from the occupational health needs of all workers.[30–32]

In contrast, some employers and WHP advocates attribute most morbidity, such as musculoskeletal disorders, to individual worker characteristics, such as obesity and lack of physical fitness, or to nonwork activities and

Table 38-1. Some of the Differences between Traditional Measures and Integrated Approaches to Employee Health and Safety

Program Theme	Traditional Measures	Integrated Approaches
Hazard control	Unitary performance standards	Accommodation to age, gender, and anthropometry
Productivity	Absenteeism, workers' compensation costs	Performance, well-being
Implementation	Problem-focused, solution-driven measures	Iterative, participatory
Cost metrics	Medical costs	Economic outcomes
Health care model	Treatment focused/group health	Prevention/behavior focus
Medical model	Occurrence of individual diseases	Group risks
Health metrics	Morbidity and costs	Positive health markers
Interventions	Single-risk focused	Multiple-risk focused
Management systems	Segregated programs	Integrated programs

Source: Institute of Medicine. Committee to Assess Worksite Preventive Health Program Needs for NASA Employees. Integrating employee health: a model program for NASA. Washington, DC: Institute of Medicine, National Academies Press, 2005.

community exposures. This perspective recognizes that *(a)* risk factors for chronic disease and measures to maintain high function and effectiveness of workers are not confined to the work week; and *(b)* a high functioning safety culture cannot eliminate all injuries or chronic diseases.

Work organization and Working Life issues also affect the treatment of individuals with chronic disease. The issues are not simple, however. The effect size of the significant differences in CVD associated with cholesterol control are still mediated by socioeconomic position—and presumably its work hierarchical components.[20] There are several countries where lipid levels are not altered by the socioeconomic gradient.[33] In the United States, extensive pharmacological intervention with statin drugs has had a greater effect on reducing serum cholesterol than in the United Kingdom.[34]

Company size and profitability are also important factors. The largest—often multinational—employers have been the most effective in reducing safety hazards, often because they have more resources available. These employers often have the best Working Life policies and programs, such as flexible employment hours, as well as onsite WHP services, exercise facilities, and healthful food. The richest and most technically innovative employers and sectors may attract highly talented workers for whom lifestyle programs are important. However, large multinational employers often relocate many workers on short notice, suggesting that their dominant concern may be short-term performance rather than long-term employee health.

In many larger companies, WHP activities, administered either through vendors or company programs, have replaced traditional occupational health functions, such as those of occupational health nurses. For example, the provision of services such as vaccinations, hypertension screening, and health counseling is often performed by insurance company personnel, human resources department employees, and/or health-and-fitness contractors.

Different conditions may apply for smaller employers or employers chiefly engaged in the manufacturing sector, where hiring and termination are less fluid and where the focus is on retention of healthy workers throughout their working lives. For these smaller employers, more utilitarian goals may be more suitable, such as reduction in risks for musculoskeletal disorders, cardiovascular disease, and diabetes, and provision for the impacts of major life events. Therefore, choices of employers that stimulate and engage workers in the short term may differ from those that reduce health risks of workers over decades.

WORKPLACE HEALTH PROMOTION IN THE UNITED STATES

Since an estimated 300,000 to 500,000 deaths occur in the United States annually due to obesity and inactivity, and 400,000 due to smoking,[35] core WHP programs typically focus on physical inactivity, overweight/obesity, and smoking cessation. Many programs also address stress, mental health problems, and substance abuse; coronary artery disease, cerebrovascular disease, and hypertension; diabetes; osteoarthritis; and malignancies related to lifestyle factors, such as lung cancer.

A 2006 survey found that 62% of responding U.S. employers offered wellness initiatives and 15% planned to do so in the near future.[36] Many respondents were providing education, decision-support tools, integrated disease management, and coaching to employees.

Numerous companies and unions have developed their own WHP programs. For example, General Motors and the United Automobile Workers have collaborated on the Lifesteps program, which has involved more than 40% of eligible households.[37] In the 1980s, IBM offered "A Plan for Life," which featured tracking of blood pressure, serum lipids, body mass index, and cigarette smoking. A voluntary health assessment survey was coupled with courses offered either onsite or offsite with tuition assistance. One-half of the course participants stopped smoking, compared to one-third of those who did not participate in any courses.

Pitney-Bowes, notable for its emphasis on workplace design and ergonomics, has added to its occupational medicine program with full-scale primary care clinics and some specialty services at its largest sites to reduce medical costs, improve worker access to medical care, and improve patient compliance with prescribed medications.

The company has also increased compliance by reducing requirements for use of generic drugs and by establishing parity of mental health benefits. It has dramatically reduced costs, mainly by more effective utilization.

Some private companies have attempted to standardize WHP programs through use of a uniform checklist format, as reflected by draft standards and measures for the *Wellness & Health Promotion Product Suite* from the National Center for Quality Assurance.[38] For example, there has been widespread use of the health risk assessment (HRA) to assess the health of individual workers and persuade them to alter their behaviors. The HRA consists of a questionnaire, a risk-projection calculator, and a short narrative of health recommendations.

One reason for the particular interest in WHP in U.S. workplaces is the cost of employer-sponsored plans that provide access to health care. Workers with at least three of the common behavioral risk factors, such as smoking, inactivity, obesity, and alcoholism, are 50% more likely to be absent from work than employees without equivalent risk factors.[39] However, there is much variation in how WHP programs affect worker health[40] and uncertainty about whether they reduce health care costs over the long term. Health risk assessments without individual health counseling or coaching generally have not led to changes in health-related behaviors.[41] A review of 42 epidemiological studies with a total of over 500,000 participating workers who were followed for an average of 3.6 years found that, in almost half of the studies, worker absenteeism decreased by an average of 30% and health care costs decreased by an average of 22%.[42] Other researchers have challenged the quality of studies that have evaluated WHP outcomes and argued that costs can be decreased more with targeted disease-management programs.[43] In general, WHP programs have been more successful in sustaining smoking cessation and blood pressure control than other lifestyle changes.

Several programs have attained high participation rates by providing monetary incentives, which have ranged from $20 to complete surveys and HRA forms to several thousand dollars if health goals are met.[44] Provisions of the Health Insurance Portability and Accountability Act (HIPAA) restrict the use of health status as a determinant of incentives or penalties; incentives are legal if they are tied to physiological goals as part of an overall employee policy. Some argue that incentives are a necessary part of a successful WHP program.[45] Because participation rates in WHP programs can reflect different levels of involvement and are not a surrogate for successful changes in health behavior, measures of performance and health and/or work outcomes, such as reduced lost work time, are also important and are sometimes characterized as development or refinement of baseline WHP programs.

In general, WHP outcomes can be divided into three related categories: participation, quantitative outcome targets, and achievement of individual outcomes. Two examples are shown in Table 38-2. The setting of outcome targets is a critical means of assessing effectiveness and is highly tied to the use of incentives. Quantitative targets are based on national norms, which have been set by the National Institutes of Health (NIH) or consensus health organizations. They leave less to interpretation and avoid the costs of individual counseling. In contrast, if incentives are used, there is a tendency to enrich people who are fit already and to further discourage the involvement of those who are least healthy by setting unreachable goals and increasing penalties. In addition, even modest success in one lifestyle behavior is often a "gateway" change that will encourage other results. There are also more refined outcome

Table 38-2. Illustrative Examples of Workplace Health Promotion Outcomes

	Participation	Quantitative Outcome Target	Achievement of Individual Outcomes
Weight loss	Completing health risk assessment Attending weight-loss group	Body mass index <25	10% decrease in baseline weight
Physical activity	Completing health risk assessment Purchasing gym membership	150 minutes per week	Walking program

considerations. For example, moderate overweight (body mass index = 25–29), which does not increase cardiovascular disease risk without other risk factors, may be a achievable goal for obese people. In addition, people who participate in weight-loss programs who do not reach desired weight-loss outcomes are also less likely to increase their weight than nonparticipants. Given the difficulties in evaluating health promotion programs, some have suggested use of less formalized qualitative methods of assessment, such as focus groups and interviews.[46]

The efficacy of establishing programs in selected settings or for a short term needs to be balanced against long-term effectiveness in achieving program goals and desired outcomes. The RE-AIM list of criteria[47] can assist in setting priorities for public health interventions:

*R*each a high proportion and representative sample of employees
*E*ffectiveness in achieving intended outcomes with minimal negative effects
*A*doption by a high proportion and representative sample of workplaces
*I*mplementation with fidelity to original model while minimizing unintended consequences
*M*aintenance of program for at least 6 months

Regarding public policy in the United States, national legislation has focused on insurance premiums. Some individual states have provided a mixture of incentives and assisted services.[48] In particular, certain states have developed cooperative programs with the Centers for Disease Control and Prevention (CDC). For example, Maine and the CDC have developed WHP programs on physical activity, nutrition, tobacco, cancer control, asthma control, cardiovascular health, diabetes prevention and control, and breast and cervical health.

OTHER PROGRAMMATIC INITIATIVES

Participatory Ergonomics

Participatory ergonomics (PE) programs engage teams of workers in designing workstations and in participating in other work-process interventions in ways that will directly affect their jobs.[49] Workers can be similarly engaged in the design of WHP programs.[50] The process resembles the European "health circles," in which workers are engaged as subject-matter experts in changing organizational structure to improve physical and psychosocial working conditions, with positive impacts on employee health, well-being, and absenteeism.[51–52] A successful PE program includes the following:

1. Identifying problems by passive and active surveillance
2. Identifying possible solutions
3. Evaluating solutions, and piloting and improving approaches
4. Implementing tested solutions
5. Evaluating effects and assessing costs
6. Developing long-term sustainable programs with an iterative approach, including identification of the next problem focus, involvement of medical management, and diffusion to new departments and facilities

Enlarging the PE program to cover WHP areas involves three steps that mirror successful occupational health and safety committees:[50]

1. Identifying key leaders or "champions"
2. Communicating the implementation plan and projects throughout all tiers of an organization, with specific messages to different levels of management and workers
3. Designing and implementing training programs customized for each level of the organization

Different strategies are needed to engage managers and employees because WHP is more popular among managers and more acceptable to salaried than hourly workers. However, the largest obstacle to PE programs has often been inadequate commitment by management. An effective steering committee should include all key workplace groups, including midlevel managers and supervisors, recognizing that immediate supervisors approve and arrange release time for members of a design team.[53]

Lifestyle and Work Organization Options

Employers can offer flexible working arrangements in the form of part-time, casual, and telecommuting work. More proactive employers can provide compulsory leave and a strict maximum on work hours, and they can foster an environment that encourages employees not to continue working after regular work hours.

Work–life integration varies by country and by occupation. In general, only highly skilled workers have work–life benefits in their contracts, whereas unskilled workers almost always have only bare-minimum legal requirements in their contracts. Compared to the United States, the EU has gone much further in assuring work–life balance, such as with laws on parental leave and nondiscrimination against part-time workers. However, a study of companies in Great Britain found that less than 5% offered flexible work hours or leave policies beyond national requirements, although more than 25% offered stress counseling.[54]

In the United States, there is some support for parental leave. According to the Family and Medical Leave Act, any "eligible" employee is entitled to 12 weeks of leave for needs of an immediate family member (spouse, children, or parents) or medical reasons during a 12-month period.[55]

Initiatives by Non-governmental Organizations

The National Center for Quality Assurance (NCQA) is a non-governmental organization that has pursued national standardization of workplaces and vendors through wellness and health promotion standards. It relies on established checklists and assessment tools and has established a scoring system based on a set of universal criteria, such as engagement of employers or plan sponsors, privacy and confidentiality, encouragement of wellness and prevention, health appraisal, self-management tools, health coaching, and measurement of effectiveness. The NCQA approach begins with the presumption that, without strong management commitment and central decision making, programs will be ineffective. It focuses on a limited set of recognized adverse health behaviors—smoking, insufficient exercise, and overeating—associated with the most prevalent chronic diseases. It is oriented toward large companies and organizations that can either directly provide resources or pay for services of outside vendors. It excludes most Working Life program components that involve the quality of specific work or a specific work organization.

The American Heart Association (AHA) has endorsed worksite wellness programs to reduce morbidity and lower health care costs.[56] Its program includes public-access Web sites with PowerPoint presentations and supportive materials. Primary program components include tobacco cessation; physical activity and exercise; stress management and stress reduction; screening for hypertension, increased body mass index, hypercholesterolemia, diabetes, and mental health problems; nutrition education; weight management; and cardiovascular disease prevention.

The AHA supports the use of instruments for readiness to change and motivational assessment. Its program, which is developed for all people, regardless of age, gender, or ethnic, cultural, or intellectual capacity, focuses on health behaviors—but not working conditions.

The AHA recommends the following steps to promote cardiovascular wellness in the workplace:

- The president or chief executive officer appoints an internal wellness program coordinator or wellness promotion staff member to be available to employees for consultation.
- The employer identifies procedures and develops activation plans to handle health emergencies of employees at work.
- The employer offers training in cardiopulmonary resuscitation (CPR), first aid, and/or the use of an automatic external defibrillator (AED).
- The employer offers a confidential hotline for alcohol and other substance abuse problems.
- The worksite is defined by supportive social and physical environments that facilitate healthy lifestyle choices.

While the AHA aims to reduce heart attacks and strokes, its overall program focuses on broader behavior change of workers.

INTEGRATION OF WORKPLACE HEALTH PROMOTION WITH OCCUPATIONAL HEALTH

Barriers to successfully integrated health promotion and disease prevention programs include the multifactorial nature of health, the long-term evolution of chronic diseases, the greatest risks being present in lower socioeconomic groups, the long-term commitment necessary for sustainability, and job insecurity of many workers.

The Institute of Medicine proposed an integrated employee health program for NASA.[28] Its recommendations focused heavily on integration of worksite health promotion and occupational health and safety service delivery and incentives.

There is a potential contradiction in WHP programs, because of the differing nature of individualized and group-based programs. Successful community-based health programs that advance behavior change emphasize individual responsibility but also group and individual involvement in setting goals and implementing programs. The external imposition of goals and incentives, even if implemented at the individual level, represents a variation of this approach, with less established outcomes. The integration of occupational health and safety with health promotion as part of the Working Life concept should represent an effort to increase the level of ownership and control of health programs by the active workforce.

One of the most comprehensive analyses of the Working Life concept emerged from the Robert Wood Johnson Foundation's Commission to Build a Healthier America.[57] The report noted the importance of health status to secure employment, the greater impacts of health problems on workers of lower socioeconomic status (SES), and the highly adverse health effects of job loss. It addressed the conventional occupational health focus on the physical conditions of work, as well as the psychosocial aspects and the organization of work. The report provided a work-centered perspective on health that differs from conventional WHP and individual behavioral profiles. Key issues included the following:

1. *Work schedules*: Important considerations include shift work, overtime work, and the time consequences of holding multiple jobs. Sleep deprivation and impacts on family relations are key themes. The flexible hours and telecommuting of higher paid employees are different than the time needs of low-SES workers.
2. *Commuting*: Out of 134 million U.S. workers outside the home, 120 million commute to work. Time duration of commuting is directly related to low back pain, reduced leisure exercise, and obesity.
3. *Work–life balance*: Since 1970, family work time has increased by 11 hours per week. The report recommended flextime, schedule variation, and breastfeeding policies.
4. *Control at work, demands, and decision latitude*: The Commission noted that employees at different organizational levels have marked disparities in opportunities for skill utilization and self-efficacy (the belief in one's ability to perform a task), which may explain much of the SES difference in health status. This was extended into the concept of "organizational justice."

The report also addressed racial and gender discrimination, the need for a supportive work environment, income and other work-related resources, leave time, elder care responsibilities, and health insurance (see Fig. 38-3).

In 2004, the National Institute for Occupational Safety and Health (NIOSH) launched the WorkLife Initiative (WLI), which seeks to establish effective workplace programs to sustain and improve worker health.[58] It focuses on identifying and supporting comprehensive approaches to reduce workplace hazards and promote worker health and well-being. The premise of this initiative is that comprehensive practices and policies that consider both the physical work environment and the organizational work environment, while also addressing personal health risks of individuals, are more effective in preventing disease and promoting health and safety than each approach taken separately. Another key goal is

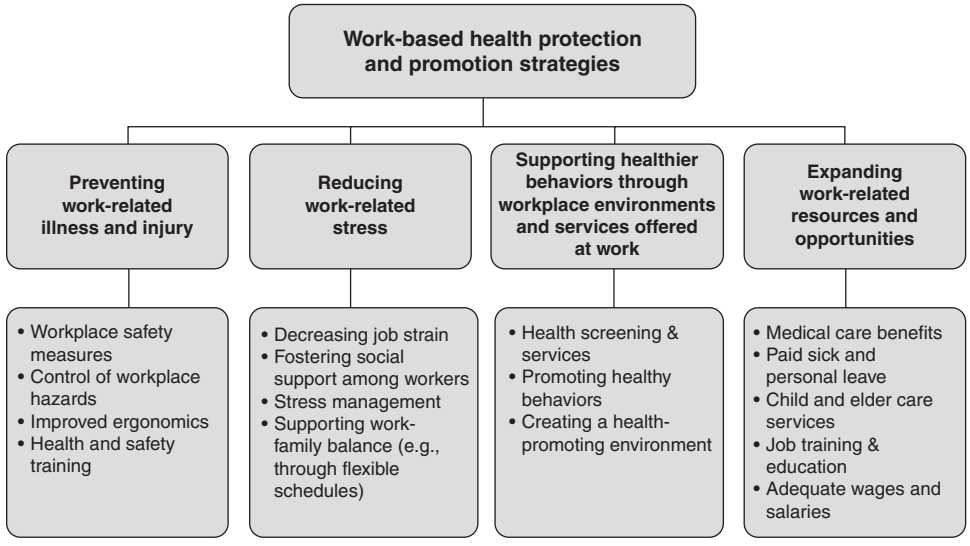

Figure 38-3. Work-based strategies to improve health. (*Source*: Robert Wood Johnson Foundation: Commission to Build a Healthier America. Issue Brief 4: Work and Health, 2008.)

overcoming the many translational issues that now prevent state-of-the-art research findings on occupational health and safety, health promotion, and chronic disease prevention from promptly benefiting workers—regardless of workplace size, work sector, or region. The WLI also recognizes the need to develop a culture of work–life balance. Specific recommendations on program content and policies are available at: http://www.cdc.gov/niosh/worklife/essentials.html.

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39

Addressing the Built Environment and Health

Richard J. Jackson

As we think about environment and health, we often focus on the microscopic—bioaccumulating chemicals or infectious organisms, and the macroscopic—the polar ice caps and the effects of war. But there is another environment that surrounds us and profoundly shapes our health nearly every minute of the day: the built environment—not just our homes and our highways, but also our parks and watersides and all the other places where we spend most of our lives. The built environment is so pervasive and people are so adaptable that we scarcely notice it. Historically, our notion of the "adverse effects" of the built environment has brought visions of slums and crowded cities, visions that have seemed to vanish with major infrastructure improvements, such as water systems and air conditioning, and abundant cars and superhighways.

It is critical, however, to understand that how and what we build affects the quality of air and water, the occurrence of acute and chronic disease, social well-being and prosperity, and the health and well-being of generations to follow. In the nineteenth century, the United States improved the built environment by supplying clean water, safe food, home heat, and lighting; by creating transit; by reducing crowding; by removing biological and industrial waste; and by developing healthier cities and trolley-car suburbs. In the twentieth century, thanks to cheap and abundant fossil fuel and unparalleled prosperity, the United States was rebuilt for automobiles (Figs. 39-1 through 39-3). As the population grew, small towns became cities. Farmlands and forests were turned into subdivisions, industrial parks, and highways. Many public transit systems were abandoned. Schools became larger and more distant. Commutes became longer and traffic congestion became overwhelming. Freshwater became increasingly more precious and the atmosphere hotter and more polluted. Our bodies became less fit and fatter. And our social and mental stress increased. In the twenty-first century, we must develop communities that once again foster and protect health.

For the first 125 years or so of U.S. history, cities were the nation's economic and cultural engine, but they were often squalid and dangerous places. Thomas Jefferson viewed cities as "pestilential to the moral, the health, and the liberties of man."[1] Cities were frequently affected by epidemics, such as yellow fever and cholera. Nearly every family lost a child to infectious illnesses, such as measles and diphtheria, or lost friends and loved ones to tuberculosis. Household and animal wastes accumulated in the streets. Local water supplies were polluted. And crowding—especially of the poor—was the norm.

Figure 39-1. Urban sprawl in the Phoenix metropolitan area. (Photograph by Barry S. Levy.)

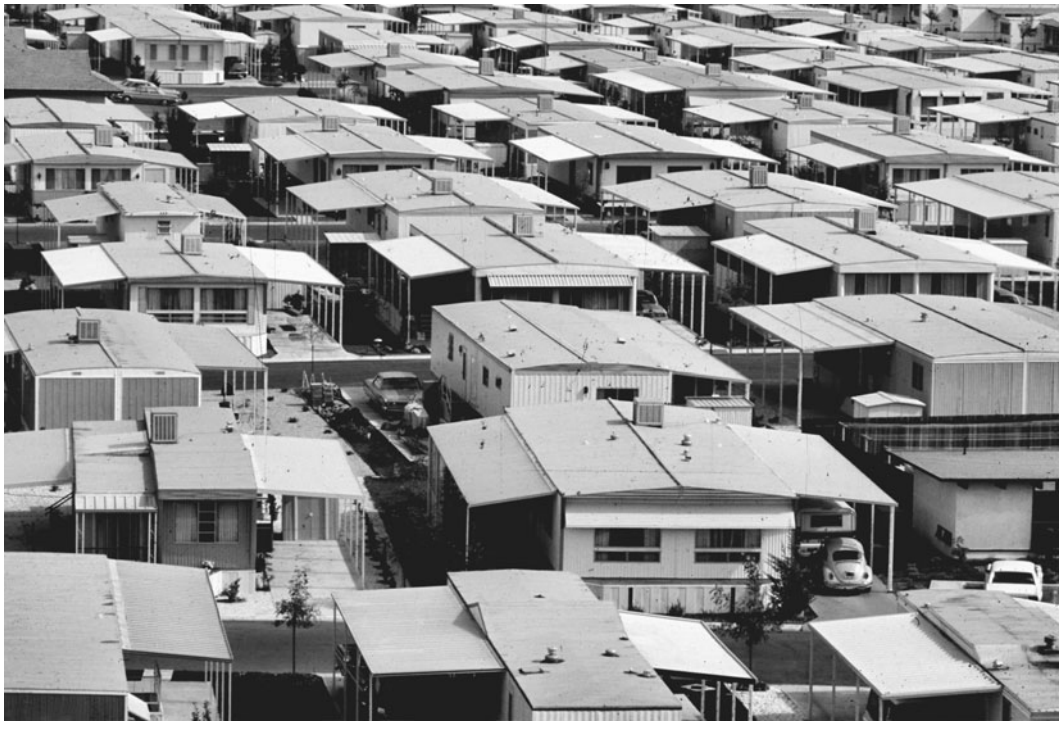

Figure 39-2. Mobile home park in San Jose, California. (Photograph by Earl Dotter.)

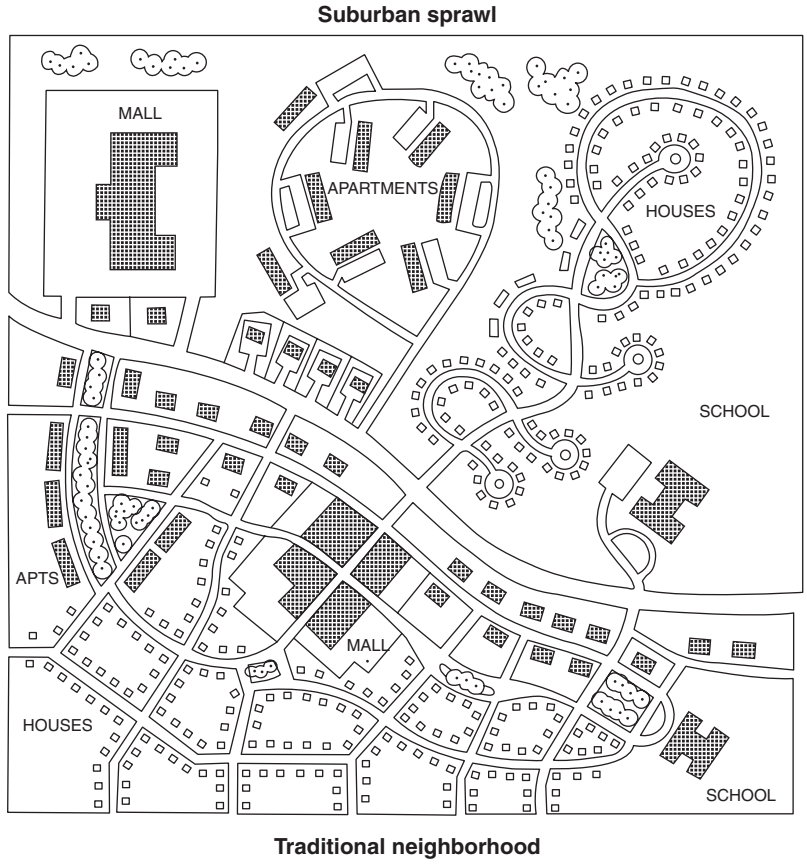

Figure 39-3. A schematic representation of streets and land use in a sprawling neighborhood and in a traditional neighborhood. Sprawling communities are characterized by being dispersed, dependent on automobiles, and having a low density, all of which makes walking and certain socializing challenging, in contrast to "traditional" neighborhoods, where shops and services are interspersed with residential areas and street networks are arranged in a highly connected grid. Sprawl necessitates the use of automobiles to travel between destinations and increases the risk of vehicle crashes, injuries, air pollution, and sedentary lifestyle. (Copyright Andres Duany. Printed with permission.)

"Noxious trades," including cloth dying, slaughtering, and tanning, frequent in cities, were located next to housing.[2] Industrial pollution, such as from gas works and steel mills, brought jobs, but it also made living in cities nearly unbearable. The leading causes of death were infectious diseases that were easily transmitted (Fig. 39-4). Human waste, collected in privies and open trenches, bred flies and attracted pests. Housing was crowded, filthy, and poorly ventilated. More than 30% of all deaths were among children under age 5, with pneumonia, tuberculosis, and gastrointestinal infections the main causes.[3]

Public health leaders began to develop partnerships with business leaders and social reformers to change the built environment in ways that focused largely on hygiene and sanitation. Clean water was brought in from rural areas to enable populations to discontinue use of contaminated wells.

It is easy to imagine the delight of the prospect of a detached home away from the unsafe city. The idea of a solo ride from work to a clean and uncrowded home in a comfortable air-conditioned vehicle would have been unimaginable. Understandably, the country welcomed the era of cheap fuel and abundant cars. Much of this progress came with health benefits; 25 of the 30 years added to the average life span of U.S. residents in the twentieth century (from 47 to 77 years) came from improvements that were primarily environmental health measures.

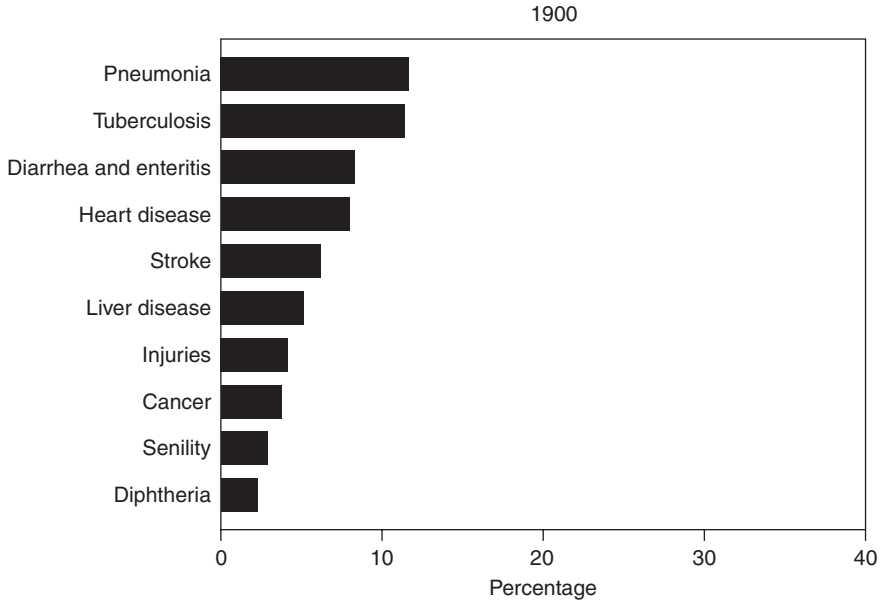

Figure 39-4. The 10 leading causes of death as a percentage of all deaths, United States, 1900. (*Source*: Centers for Disease Control and Prevention. Achievements in public health, 1900–1999—control of infectious disease. Morbidity and Mortality Weekly Report 1999; 48(29): 621–629. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4829a1.htm#fig2. Accessed on November 4, 2009.)

Zoning evolved to protect neighborhoods from nuisances. In the 1926 lawsuit, *Village of Euclid vs. Ambler Realty*, the Village of Euclid, Ohio, assigned zones to land owned by Ambler Realty, which sued the Village for restraint of trade and the reduction of property value. The U.S. Supreme Court decided that the community had the right to zone for residential, commercial, industrial, and other uses—which became a precedent for increasing homogeny of communities in the United States. Zoning served well to prevent, for example, a tannery being operated next to residences, but, over time, it led to extreme exclusion of many uses from neighborhoods.

THE AUTOMOTIVE AND HOUSING TRANSFORMATION

The country's embrace of the automobile, which began early in the twentieth century, coincided with—and was fueled by—a boom in housing. Cars made new places accessible, but they required roads, stimulating governments to develop partnerships with builders. From 1906 to 1911, the Long Island Motor Parkway was built, greatly enabling habitation of the area. Automotive "destinations" were developed, including, in 1922, the first U.S. shopping mall: Kansas City's Country Club Plaza. During the Great Depression, with homelessness and unemployment increasing, the federal government made housing a national priority. After World War II, soldiers returned home to start new families, with education made accessible by the GI Bill and housing made accessible through Veterans Administration loans. Planned communities and subdivisions sprang up across the country, further fueling a housing boom that raised homeownership from 44% in 1940 to 62% in 1960.[4]

This huge increase in housing required more roads, and car ownership was marketed as family destiny and part of the "American dream." The oil, rubber, asphalt, construction, automobile, home-finance, and other industries lobbied aggressively for a national highway system, which led to the Interstate Highway Act in 1956. Revenues for building this highway system came from gasoline taxes, which could not be used for

other purposes—not even public transit. Every city wanted to be connected to the highway system; any town not connected feared, often correctly, that it would wither away.

What happened to cities? The results were mixed. Hartford, Connecticut, home to Frederick Law Olmsted, the father of Landscape Architecture, had a pleasant downtown on the Connecticut River. In the late 1800s, the prosperity of Hartford drew luminaries such as Mark Twain. But by the 1950s, it struggled to maintain its tax base, as wealthy citizens moved to new suburbs that were accessible by car. The building of interstate highway I-91 along the river's west side dealt Hartford a devastating blow, destroying housing and neighborhoods, and cutting off the city and its residents from the river, an environmental and social amenity. Construction of I-84 through the city resulted in loss of irreplaceable buildings. And the huge interchange of I-91 and I-84 paved over the historical original city center and crippled the potential growth of the downtown area.

Cities were not only damaged directly by highways, but they also bore the brunt of the dispersal of higher income workers and the tax base to suburbia. Federal grants and loans tended to devalue racially mixed and minority neighborhoods, exacerbating poor health status there. Developers sought to maximize land investments and rapid returns on investment by building as many homes as possible—often cheaply—on distant and less expensive land, preferably served by highways. To cut costs, these homes were often built with minimal energy efficiency and with little access to desirable amenities, such as sidewalks, parks, and nearby schools. Young families in car-dominated areas felt their children were safer in dead-end cul-de-sacs.

As urban tax revenues and services decreased, cities needed to support more low-income and minority populations, which led to further attrition of tax revenues. One particularly detrimental effect of this was the closing of many fire stations in inner cities, which greatly contributed to increasing blight. The move of the higher income population to suburban areas had a series of environmental impacts. With more people commuting long distances to work, the home became a place of refuge and recreation. For some, three-car garages became the norm in new homes with game rooms and home theaters. With a garage that opened automatically, air conditioning, a large color TV, and a lawn service, one could live in a suburban home for years without meeting one's neighbors. From 1950 to 2000, the average number of residents in a household in the United States decreased from 3.4 to 2.6.[5] Over a similar period, the average usable area in a new home increased from 983 to 2,349 square feet.[6] These changes were abetted by policies that used tax revenues to pay for the highways that commuters used, and by a mortgage-interest tax deduction that reduced the pain of large mortgage payments. Taxpayers in large homes with 37% marginal tax rates, in effect, had larger tax deductions than people less well off who had a 15% marginal tax rate.

Changes in housing have also impacted energy use. While energy use increases in a larger building, energy efficiency increases per unit of area, especially in multistory buildings. Detached housing is less energy-efficient than contiguous housing, but throughout the twentieth century, U.S. builders moved away from contiguous (row) houses in favor of single-family homes. Since energy efficiency represents long-term costs carried by the homeowner, there was little incentive for developers to add such amenities. In addition, more energy is lost the further it is transmitted. About 7% of all electricity produced in the United States is used to move the electricity through transmission lines. Distant buildings also require more water, sewage, transport, and highway investments.

What happened to the countryside? As home building increased land valuations, taxes on agricultural land became very high and unsupportable. Prime agricultural land was lost, leading formerly self-sufficient agricultural counties to become housing and commercial land, with food and other goods brought from increasingly longer distances.

EFFECTS OF THE BUILT STRUCTURES ON THE OVERALL ENVIRONMENT

Roads supported housing sprawl, generating growing demands for services, causing increased driving and highway use, and requiring more

road surfaces. Today, roads in the United States feel crowded and congested because they are. From 1961 to 2004, the number of vehicles in the United States increased from 74 to 247 million (Fig. 39-5), and the number of licensed drivers from 87 to 199 million.[7,8] The annual average miles driven by U.S. drivers increased from 10,043 to 14,908.[9] Since each car requires a significant amount of pavement in the form of roads and parking places, more than 60,000 square miles of land have been covered with asphalt and concrete—an area equal to the size of Georgia.[10]

EFFECTS OF THE BUILT ENVIRONMENT ON AIR QUALITY

Increased population growth, land development, and urbanization have led to the loss of natural land and habitats. Removal of trees has caused adverse health effects. More than aesthetic amenities, trees remove carbon dioxide and produce oxygen, and cool the local environment by as much as 45°F.[11] Increasing the amount of vegetative canopy reduces ozone formation in the near-ground atmosphere. The burning of automotive fuel inherently generates heat and disperses particulate matter, oxides of nitrogen, sulfur oxides, and volatile organic compounds, which, in the presence of sunlight and heat, lead to the formation of ozone. The higher the heat of combustion, the more oxides of nitrogen are produced. The more sulfur in fuel, the more sulfur oxides are produced. Incomplete fuel combustion and evaporation from fuel tanks and during refueling increase volatile organic compounds. Catalytic convertors reduce, but do not eliminate, these emissions. Filters can reduce, but do not eliminate particulates. Ozone is a highly reactive molecule and causes bronchial inflammation, asthma, pulmonary impairment, and other adverse effects. Children, people who are exercising, and people with impaired immunity are most susceptible to these effects. While the soot emitted from an old diesel truck is unsightly and unwanted, the smaller and near-invisible particles generated by newer cars can increase the risk of coronary heart disease, including myocardial infarction.

Over the past 100 years, U.S. cities have become hotter. In Los Angeles, for example, annual maximum daytime temperatures have increased by 1.7°C (3.1°F) and minimum (nighttime) temperatures have increased by 4.0°C (7.2°F).[12] Heat presents significant health risks, most notably heat stroke. Elevated body temperature can cause death. A heat wave in Chicago in 1995 caused over 700 deaths, mainly

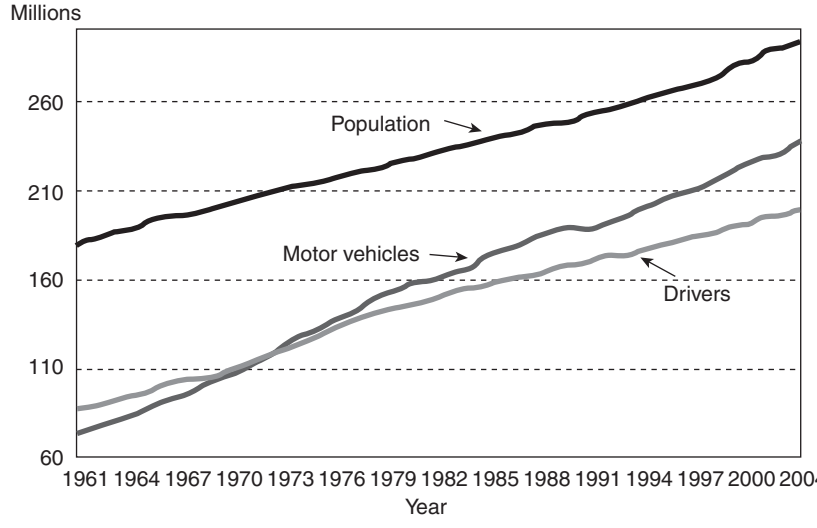

Figure 39-5. U.S. population, number of drivers, and number of motor vehicles from 1961 through 2004. (*Source*: Federal Highway Administration Highway Statistics, 2004. Available at: http://www.fhwa.dot.gov/policy/ohim/hs04/htm/dlchrt.htm. Accessed on November 4, 2009.)

among people without access to air conditioning who lived alone.[13] Most multiunit structures in Chicago were built more for cold winters than for prolonged summer heat waves. Windows were small and frequently inoperable. Roofs were flat and had heat-absorbing black tops. And use of fans in overheated rooms *increased* risk of heat stroke by creating virtual convection ovens. Inadequate nighttime cooling was exacerbated by the loss of shade and by reduction of evaporation of groundwater into the atmosphere via trees and other plants, a process known as evapotranspiration.

THE BUILT ENVIRONMENT AND CLIMATE CHANGE

Ambient temperatures have increased from rising levels of greenhouse gases (GHGs), including water vapor, carbon dioxide, methane, and other gases. (See Chapter 5.) From 1850 to 2005, the atmospheric carbon dioxide level increased from 280 to 379 ppm.[14] It is now rising at an increasing rate.[15] Planetary temperature levels have followed, increasing about 0.74°C over the past 100 years.

The burning of fossil fuels has accounted for about 80% of the increase in carbon dioxide and deforestation for about 20%. The built environment can accelerate deforestation. Most carbon dioxide emissions in the United States are related to buildings, and one-third is related to transportation. Building design that improves energy efficiency decreases waste of fossil fuels, and urban design, transit systems, and bulk shipping all reduce the use of fuel for transportation. Energy inefficient homes are those with large "footprints," noncontiguous design, poorly insulated walls and windows, inadequate daylighting, and outdated illumination, heating and air-conditioning systems, and appliances.

THE BUILT ENVIRONMENT AND EFFECTS ON WATER QUANTITY AND QUALITY

Freshwater is essential for the survival of individuals, and entire civilizations and their economies. Human habitations were sited—and survived—based on water availability. Great cities flourished when large amounts of water were delivered and removed, along with wastes. Reservoirs, aqueducts, and water-removal systems have been essential to the growth and prosperity of communities—for potable water, hygiene and sanitation, and agriculture. In the United States, about one-half of drinking water comes from surface sources, and the remainder comes from groundwater wells.

The built environment profoundly affects water quality. The classic cholera outbreak in London in 1854, traced by John Snow to the Broad Street pump, illustrates the dangers of pit toilets and ponded waste overlying groundwater sources of drinking water. In general, the more layers of soil and the longer the residence time of water in soil, the better the water quality.

The built environment also affects water quantity. Moving water long distances is expensive in human labor and use of fossil fuels. For soil moisture and groundwater to be recharged, rainwater and riverwater must percolate into the soil. Percolation also reduces the risk of flooding from storms; the more water captured onsite, the better. The "base flow" of a stream is the water that flows long after a rainstorm; it is the water that has been allowed to penetrate the earth and to seep slowly out to watercourses. As structures and paving cover the landscape, runoff of rainwater increases. In a forest, typically only 10% of rainwater runs offsite; in an urban area, about 55% runs offsite.[16] Sometimes the health effects of paving streets have late-appearing and indirect effects. Paving over cobblestones of streets in lower Manhattan led to death of street trees, and loss of the shade and cooling they produced.

To guard against disease, people in rural areas should carefully manage their water and waste systems. When human habitations become sprawled out and distant from each other, costly infrastructure for water supply and septic removal is required. To reduce these costs, many localities supply potable water through common systems, but require onsite treatment of sewage by septic systems. These systems require homeowner diligence, but thorough inspection and maintenance often occur only when the home is sold. Between 5% and 40% of septic systems may fail, causing groundwater contamination.[17]

Water also provides an important source of recreation. People need respite on hot days and often seek park or shore lands for play and relaxation. Unfortunately, in many parts of the United States, these places are inaccessible, especially for poor people. New York City, for example, has 578 miles of waterfront, but only a fraction of it is accessible to pedestrians. (See also Chapter 8.)

THE ADVERSE EFFECTS OF BUILDINGS ON HEALTH

People have always sought out safe and secure dwelling places that afford climate protection, water, ventilation, and adequate light. The movement to assure decent housing for poor and working people, especially children, did not come out of advanced understanding of disease processes, but from an appreciation of the elements of a decent quality of life. Science has since validated these basic human insights. Inadequate ventilation in buildings contributes to higher air levels of moisture, mold, smoke, volatile organic chemicals, radon, bacteria, allergens, and other noxious agents. (See Chapter 7.) Poorly insulated buildings do not mitigate outdoor heat and cold extremes, and are hazardous to health. Structure, insulation, and windows can attenuate noxious outdoor noise. Inadequate light, including daylight, presents safety risks. All of this makes reading, studying, and socialization more challenging, and is linked to depression. Buildings with inadequate exits and fire resistance are less safe, as are buildings with poor structural integrity for storms and earthquakes. Buildings with narrow, inaccessible, and sometimes-locked stairways discourage the climbing of stairs, which can be a healthful physical activity.

THE BUILT ENVIRONMENT, ENERGY, AND HEALTH

Structures that are single and unattached, one-story tall, and poorly designed, built, and insulated are less energy efficient than dense, multistory, well-built structures. The longer the distance that energy—gas, oil, coal, or electricity—must be transported, the more expensive it becomes. The greater the distance that water and food, building materials, and other supplies must be transported, the more costly they become. The further people must travel, the greater the cost of their work, school, or play. As more capital is directed to pay for inefficient use of space, less is available for education, health care, and other beneficial uses. The costs of transport are unlikely to decrease as fossil fuel supplies become more expensive, especially when all associated environmental, social, political, and health consequences as part of these costs are considered. The pressures of a growing global population and the depletion of natural resources will further increase demand. Renewable energy sources, such as solar and wind power, will counter some of these effects, especially as startup costs decrease and new energy technologies are brought into large-scale production. However, it is difficult to imagine resource and energy costs decreasing very much over the next two decades.

THE BUILT ENVIRONMENT AND INJURIES

Virtually everyone knows someone who has been impacted by a car crash. The likelihood that any U.S. resident will ultimately die in a car crash is about 1 in 85.[18] Unintentional injuries are the leading cause of death for Americans age 1 to 44, with motor vehicles responsible for the largest percentage of these fatalities.[19] Every year, nearly 40,000 Americans are killed and at least 2.3 million are injured in motor vehicle crashes.[20] However, the per-mile car-related death rate in the United States decreased from 1966 to 2008, from 5.5 to 1.3 deaths per 100 million miles traveled, primarily due to laws restricting speed and alcohol use, and laws requiring seat belts for vehicle passengers and helmets for cyclists.[20,21] Still, the annual cost associated with motor vehicle injuries and deaths is about $230 billion.[22] While there has been major progress, average driving speeds and the number of motor vehicles have increased as enforcement has declined. The risk of car crashes increases dramatically with speed, doubling for each increase of 3 miles per hour above 35 miles per hour.[23]

Roadways are important to safety, and many roadways in the United States are designed and

Table 39-1. Lifetime Risk of Death in the United States, by Type of Transit

Type of Transit	Lifetime Risk of Death
Car	1 in 85
Aircraft	1 in 5,862
Bus	1 in 106,665
Train	1 in 225,879

Source: National Safety Council. Odds of death due to injury, United States, 2005. Available at: http://www.nsc.org/news_resources/injury_and_death_statistics/Documents/Odds%20of%20Dying.pdf. Accessed on November 4, 2009.

built to expedite movement of police cars, fire vehicles, and ambulances. Streets are wide, curves are rounded, and speed bumps minimized—all to enable rapid movement of emergency vehicles. Reducing injuries and deaths due to car crashes will require more use of improved public transit, lowering average speed, and better enforcement of laws and regulations. Table 39-1 illustrates the lifetime risks of death in the United States by car and other forms of transit.

Public transit is viable when there is population density around transit nodes sufficient to support its use. "Complete streets" are designed and operated to ensure safety and accessibility for pedestrians, cyclists, and public transit riders, and serve to calm traffic. Features that make streets safer and more appealing to people on foot and bicycle include adequate sidewalks, bike routes, pedestrian amenities, trees, and storefronts.

THE BUILT ENVIRONMENT AND CHRONIC DISEASE

Obesity is a major risk factor for chronic disease.[24] From 1998 to 2008, the cost of medical treatment for obesity-related disease in the United States increased from $74 to over $147 billion annually. From 1998 to 2006, the prevalence of obesity in the United States increased 37%. Obesity now accounts for more than 9% of medical expenses in the United States.[25]

People who live in low-density areas are likely to drive more, walk less, and be overweight. Areas that have higher concentrations of fast-food restaurants have higher rates of obesity. Children whose schools are close to fast-food restaurants are more likely to eat high-calorie foods that are low in nutrients.

In the past, physical activity was woven into the activities of daily life. Getting to school or work, shopping for food, and cleaning one's home all required physical activity. Few U.S. residents were obese and most would never have imagined joining a fitness club simply to exercise on machines. Fitness was taken somewhat for granted and many working people longed to be free of backbreaking labor. While lives of U.S. residents are now inundated with labor-saving devices, fast food, and instantaneous communication, we work harder, eat less healthy food, and seem to have less free time than ever before. Although fitness is essential to our health, only 34% of children engage in recommended levels of physical activity.[26] In a study of thousands of nurses followed over 25 years, nurses who were not physically active had a 20% to 25% higher rate of mortality than those who were.[27]

The built environment has significant impacts on physical activity. People who live in denser areas use more public transit and have more physical activity than those who do not.[28,29] Those who live near parks and also live close to the places where they travel frequently are more likely to walk or bicycle there.[30,31] Children who live close to schools are more likely to bicycle or walk to school.[32] Living in areas with walkable green spaces lengthens the life of older people in urban areas, independent of their age, gender, marital status, baseline functional status, and socioeconomic status.[33]

THE BUILT ENVIRONMENT AND MENTAL HEALTH

> We drive up and down the gruesome, tragic suburban boulevards of commerce, and we're overwhelmed at the fantastic, awesome, stupefying ugliness of absolutely everything in sight—the fry pits, the big-box stores, the office units, the lube joints, the carpet warehouses, the parking lagoons, the jive plastic townhouse clusters, the uproar of signs, the highway itself clogged with cars—as though the whole thing had been designed by some diabolical force bent on making human beings miserable.

> And naturally this experience can make us feel glum about the nature and future of civilization.
>
> —James Howard Kunstler, *Home from Nowhere*

In the United States, the most prevalent mental disorder is depression, and the second-most prescribed group of medications is antidepressants. The costs of depression are substantial with loss of lives, livelihoods, happiness, and productivity. Stress and anxiety often coexist with and amplify depression. The treatment of mild to moderate depression includes medications, such as selective serotonin reuptake inhibitors (SSRIs) and counseling, including cognitive therapy. Nonclinical interventions for milder forms of depression, such as exercise, relate very much to the built environment. Exercise increases serotonin and alleviates depression. Contact and support from loved ones helps alleviate acute depression, such as depression associated with loss of a loved one. Contact with nature and water revitalizes body and mind.

If one designed a depression-inducing environment, its key features would include prevention of exercise, removal of interesting and pleasing natural features, and creation of social isolation and anxiety. Much of the built environment developed in the mid to late twentieth century succeeded in creating all these features. The built environment was designed for automobiles—not for salving the human mind and fostering relationships. Many U.S. residents spend hours commuting and driving long distances on high-speed, crowded roads, and many people report that their driving to and from work is more stressful than work itself.

EFFECTS ON FINANCIAL, SOCIAL, AND CULTURAL CAPITAL

In the United States, the average family income devoted to transportation has increased from about 12% in 1960 to 18% in 2007.[34] Families of lower income face an even greater burden as they allocate 30% of their average family income to transport-related expenses.[35] In some cities with substantial sprawl, the portion is much higher.[36] Most travel is not done for pleasure, but to meet work needs and other needs of life. A need for affordable housing is a major factor. There is a maxim in the real-estate business that people drive (away from large cities) until they reach places where they can qualify for a mortgage. While some people seek a distant house in suburbia because they want or need a larger home with a lawn on a cul-de-sac, the primary determinant of where someone chooses to live is the cost of housing—and homes further away from urban centers generally cost less. This distance, however, creates perverse outcomes. Often children must be driven to schools and other activities, and many arrive home well before their commuting parents. The cost of a car is substantial, averaging $7,000 to $11,000 annually. If this amount were applied to mortgage payments, the homeowner would be building assets over time rather than consuming them.[37] In addition, subdivision neighborhoods built by developers tend to be economically homogenous and to lack economic engines. Children growing up in such places may have limited exposure to the lessons of living in a diverse community.

"Social capital" is the glue that holds communities together. People are healthier and live longer in areas with high social capital. After disasters, these communities recover more quickly. Long commute times to and from work and school reduce the level of community engagement and social capital. In a recent rating of best American cities, one of the measures was the level of cultural liveliness. A high prevalence of small local restaurants was recognized as an asset compared to an abundance of franchise restaurants. A vibrant cultural scene attracts most creative and productive workers, especially young workers, to an area.[38] Cities with lively cultural opportunities tend to generate more wealth during good times and to resist economic downturns better. Built environments can therefore enhance cultural life or stifle it.

EFFECTS ON VULNERABLE POPULATIONS

Children

While the built environment can adversely affect all people, children are especially vulnerable. Children eat, drink, and breathe three to four times as much per unit of body weight than do

adults. Children face greater hazards from air and water pollution, chemicals in food, and other environmental toxins. Because they have many years of life ahead of them, these adverse impacts last for a long time.

Air pollutants, especially ozone and particulate matter, cause both short- and long-term respiratory impairment in children on days when ozone levels peak, children's school absenteeism, respiratory medication use, emergency department visits, and hospital admissions all increase. When children play outside, reside, or attend school in areas with heavy vehicle traffic, they are exposed to higher levels of air pollutants.

However, restricting children's exposure to the environment—built or natural—is also damaging. The built environment restricts children's opportunities for physical activity and contributes to childhood obesity.[39] Children who do not have opportunities for regular physical activity, such as walking or biking to school, are at risk for becoming overweight and developing diabetes, psychosocial problems, and other disorders. Young people spend much time in or near cars. Motor vehicle crashes are the leading cause of death for U.S. children age 3 to 14—as passengers, pedestrians, and bicyclists.[40] On average, eight children are killed and over 900 are injured daily in motor vehicle crashes in the United States.[41]

Restricting children's exposure to the natural environment increases their risk of depression, isolation, and attention deficit hyperactivity disorder. There is a direct link between the lack of nature in children's lives—"nature-deficit disorder"—and childhood obesity, attention disorders, and depression; exposure to nature is essential for the healthy physical and emotional development of children.[42]

Older People

Older people, too, face health risks due to the built environment. Most U.S. communities are designed for driving, with few alternatives for older people who do not drive. Walking to stores, medical facilities, community centers, and places of recreation requires a safe pedestrian environment and a densely developed community, which is rare in much of the United States. As a result, many older people have little physical activity and social interaction, and become physically and socially isolated.

People with Physical Disabilities

The built environment must be designed to provide access to all community members, regardless of their level of physical ability. All too often, design of public transit systems, sidewalks, and crossing signals overlooks the unique needs of people with physical disabilities. People in wheelchairs require sidewalks that are wide and level, and have appropriate cuts in the curb. People with visual impairment require signals that indicate safe crossing times at intersections.[43] In addition, traffic signals must be timed to allow people with disabilities sufficient time to safely cross streets.

Women

In sprawling communities, children and adults rely heavily on automobiles for transportation. Mothers, in particular, spend much time driving children to and from school, play, and athletic practice, and driving older relatives to clinics and elsewhere. In 1995, the average married woman with school-age children in the United States made more than five automobile trips daily, 21% more than the average man.[44] Driving is a significant source of stress, and more time spent in cars leads to greater risk of crashes and more exposure to air pollutants. Low-density, sprawling communities do not encourage people, especially women who care for young children, elderly parents, and other family members, to perform their daily responsibilities by walking or bicycling.

Poor People and People of Color

Poor people and people of color have suffered the effects of systematic discrimination for many years. Loan and insurance policies created by the Federal Housing Administration, beginning in 1934, favored whites who purchased single-family homes, rather than members of racial and ethnic minorities who lived in multifamily, urban dwellings. For decades, these policies led to the migration of white, middle class people out of inner cities, leading to racial segregation. With them, went opportunities for employment, leading to economic segregation. Health indicators for people of color living in inner cities remain markedly worse than those of white people. A 1990 study conducted in Harlem in

New York City, where at the time 96% of residents were black, found that life expectancy was lower than in Bangladesh. Harlem residents were found to die at three times the rate of white U.S. residents—mainly due to high rates of cardiovascular disease, cirrhosis, cancer, and homicide.[45] In addition, members of minority groups are more likely to live in areas with air pollution,[46] and black and Hispanic children have higher rates of asthma than do white children.[47] Exposure to air pollutants is concentrated disproportionately among the poor and people of color. (See Chapters 4 and 33.)

DEVELOPING HEALTHY COMMUNITIES

All of this may feel very bleak. But nature maintains sustainability and enormous vitality by recycling. A forest, for example, does not create waste. It is critical that we, too, increase the sustainability and vitality of built-environment systems. We must move to reduce extraction and exploitation of limited natural resources and use innovative design to develop the built environment in concert with nature. In addition to sustaining our environment, we must sustain our rich cultural diversity. Many of the adverse health impacts of the built environment disproportionately affect vulnerable groups, including children, the elderly, the poor, and members of racial and ethnic minorities. We must meet the needs of these people and design and redevelop our communities to foster economic, social, and ethnic diversity—for the benefit of all people.

By 2100, the United States population is projected to nearly double, to 570 million.[48] To accommodate this growth in ways that are healthy, socially just, and environmentally stable, we need to design communities where people are able to move about safely on foot, bicycle, and public transit, and where community members engage in the continuous process of healthy community development. It is unsustainable and irresponsible to continue using development practices that harm the public's health and that encourage people to drive cars, which emit nearly 20 pounds of carbon dioxide for each gallon of gasoline used.[49] (See the discussion of climate change in Chapter 5.)

Public transit options that are clean, affordable, and accepted and utilized by local communities reduce use of fossil fuel and emissions. A major opportunity for healthy redevelopment of communities lies in the design of streets that have dense, mixed land-use, and are accessible and safe for all types of transit. These "complete streets" enable safe access for all users, including pedestrians, bicyclists, motorists, and transit riders of all ages and abilities.[50] Establishing and maintaining adequate bike lanes and paths promotes an alternative to automobile transport, and it enables people to get physical activity safely while moving about their communities. Bicycle paths encourage exercise, especially if they link to retail stores, schools, and public transit systems.

Urban redevelopment can promote health by decreasing the number of vacant properties and redesigning failed infrastructure projects. Redevelopment provides the opportunity to reduce urban blight, and it enables communities to have affordable housing, parks, schools, playgrounds, and other public facilities. The Embarcadero Freeway in San Francisco was originally intended to connect the Bay Bridge with the Golden Gate Bridge. It was partially constructed, but it was never completed. (If it had been fully completed, it would have cut the downtown area off from the waterfront.) Traffic—more than 100,000 vehicles per day—routinely ran through local neighborhoods. The partially constructed freeway was severely damaged in an earthquake in 1989, and, while the city weighed reconstruction options, the number of riders on public transit increased by 15%. City officials elected not to rebuild the freeway and instead, in 2002, redeveloped the area as a dynamic multiuse boulevard, accessible by car and public transit, with bike lines, a waterfront promenade, a redeveloped historic building, and a new public plaza that hosts a farmers' market several times a week. Dense commercial development has lined nearby streets, housing in the area has increased by 51%, and jobs have increased by 23%.[51]

Another opportunity to enhance health and well-being through the built environment lies in improving architecture. Given that buildings account for 50% of the production of greenhouse gases in the United States,[52] the American

Institute of Architects (AIA) has asked its members to develop carbon-neutral buildings. Structures that are beautiful, quiet, and have good ventilation and lighting contribute to their surrounding areas and to the health of their occupants. Buildings with physically attractive and easily accessed stairways save electricity (because of less elevator use) and increase physical activity and socialization.

Schools are a critically important element in the shift to healthier built environments. The number of schools in the United States decreased from 262,000 in 1930 to 91,000 in 2009, while average school size increased.[53] New schools are now more often located on large sites on inexpensive land, distant from the neighborhoods they serve. For many years, state and local governments had "minimum acreage standards," which mandated that communities build schools on sites that met specific size requirements, depending on the type of school and the number of students. Though these standards are no longer nationally mandated, 27 states still have similar requirements.[53] Consequently, many schools are not located within walking or biking distance for students. Safety issues are a major concern for parents, who consistently cite traffic danger as a reason why they do not allow their children to bike or walk to school.[54] Children who are sedentary are at increased risk for obesity, diabetes, and cardiovascular disease. In addition, the decrease in walking and bicycling has had damaging effects on traffic congestion and air quality around schools.[55] A federal government program addresses these issues, by providing funding and technical support to community programs that promote children safely and routinely walking and bicycling to school.[56]

Parks and green spaces are also essential parts of a healthy environment. Trees and other plants filter pollutants in the air and water, mitigate wind, reduce solar heat gain, stabilize soil to prevent or reduce erosion, create animal habitats, help filter and absorb stormwater runoff, and may help mitigate carbon emissions.[57] Parks are, by nature, public and recreational, and they provide opportunities for social interaction and community building. Parks that are well admired and frequented are community assets that can spur economic growth nearby; homes located close to parks often have higher resale value than those more distant.

The built environment has significant impacts on water quality and availability, which are likely to intensify as global temperatures increase. Illustrative is the situation in Los Angeles, where rainfall is very limited and more than half of the water used by the area's 13 million residents comes from distant locations, such as the Colorado River. Transporting water to Los Angeles costs much money and energy. Since the city is largely paved with impermeable materials, there are few places where rainwater can soak into the ground. Instead, it flows across hard surfaces, collecting oil, pesticides, animal waste, and garbage before it flows into storm drains and other channels that lead to the ocean, squandering rainfall and polluting rivers, streams, and the ocean. A local organization advocates for residents to collect rainwater in order to reduce the need to import water, decrease runoff, and supply more water during dry months.[58]

The availability of food is another issue linking health with the built environment. Throughout the United States, urban farms and community gardens are producing more local food. In 2009, San Francisco initiated an audit of all unused city land that could be developed into gardens or small-scale farms to create jobs and promote local production of fresh food. Another program in the area, which integrates science with gardening, uses school gardens to teach students about the importance of good nutrition, while producing the ingredients for student lunches, eliminating food transportation costs, and ensuring the freshness of food.

CONCLUSION

The World Health Organization (WHO) defines health as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity." The quality of our built environment affects our health and quality of life.

Academia has a major role to play in developing healthy built environments. Academic courses and further research are needed to expand the body of knowledge on the relationship between the built environment and public health. Research on economics and health and

development policy is especially important. We must develop metrics and methods to account for all of the financial, environmental, social, and health costs associated with development strategies. Our focus must shift away from getting a rapid return on investments to developing sustainable systems. Using a *Health Impact Assessment (HIA)* enables community planners to apply public health analysis to the built environment. An HIA is a "combination of procedures, methods, and tools by which a policy, program, or project may be judged as to its potential effects on the health of a population, and the distribution of those effects within the population."[59] By explicitly incorporating health into design and planning decisions, an HIA can be utilized to anticipate and quantify the health impacts of a particular development strategy.

This is a critical period for humankind and for our planet, which requires informed, holistic, and strong decision making. Health practitioners, planners, politicians, and community members have an obligation to demonstrate leadership in developing and implementing sustainable approaches to the challenges in our natural and built environments.

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Appendix: Selected Non-governmental Organizations

Professional Organizations

Occupational and Environmental Medicine

American College of Occupational and Environmental Medicine. http://www.acoem.org.
An association of physicians and other health care professionals who specialize in occupational and environmental medicine. It is dedicated to promoting the health of workers through preventive medicine, clinical care, research, and education.

Association of Occupational and Environmental Clinics (AOEC). http:// www.aoec.org
A nonprofit association of over 60 clinics and over 250 occupational and environmental health professionals that is dedicated to improving the provision of occupational and environmental health care through information sharing and research. Through AOEC, one can often identify physicians or other health care professionals with specific clinical or research expertise for patient referral or telephone consultation with the treating physician.

American Thoracic Society. http://www.thoracic.org
A professional society of physicians, research scientists, nurses, and other allied health care professionals in such specialties as pulmonology, critical care, sleep medicine, infectious disease, pediatrics, and environmental and occupational medicine. Programs developed by its Environmental & Occupational Health Assembly are featured on its Web site. The Assembly has issued valuable guiding statements on such subjects as air pollution, silica, agricultural hazards, asbestos, workplace asthma and airway disease, indoor air, and respiratory protection.

Occupational Health Nursing

American Association of Occupational Health Nurses (AAOHN). http://www.aaohn.org
The mission of AAOHN is to advance the profession of occupational and environmental health nursing through education and research, professional practice/ethics, communications, governmental issues, and alliances.

Industrial Hygiene

American Conference of Governmental Industrial Hygienists (ACGIH). http://www.acgih.org
A member-based organization that advances occupational and environmental health through publications, including annual editions of exposure guidelines and work practice guides, and other activities.

American Industrial Hygiene Association (AIHA). http://www.aiha.org
An association that serves the needs of occupational and environmental health and safety professionals practicing industrial hygiene in industry, government, labor, academic institutions, and independent organizations.

Safety

American Society of Safety Engineers (ASSE). http://www.asse.org
The oldest and largest professional safety society that is committed to protecting people, property, and the environment. It provides key information and action on occupational safety, health, and environmental issues and practices.

Ergonomics

Ergonomics Society. http://www.ergonomics.org.uk
A professional society for ergonomic specialists that is based in the United Kingdom.

International Ergonomics Association. http://www.iea.cc
A federation of ergonomics and human factor societies throughout the world whose mission is to elaborate and advance ergonomic science and practice and to expand its scope of application and contribution to society.

Toxicology

Society of Toxicology. http://www.toxicology.org
A professional and scholarly organization of scientists from academic institutions, government, and industry representing the great variety of scientists who practice toxicology in the United States and elsewhere. It promotes the acquisition and utilization of knowledge in toxicology, aids in the protection of public health, and facilitates interaction among disciplines.

Epidemiology

American College of Epidemiology (ACE). http://www.acepidemiology.org.
An organization that advocates for policies and actions that enhance the science and practice of epidemiology, promotes the professional development of epidemiologists through educational initiatives, recognizes excellence in epidemiology, and develops and maintains an active membership base of both fellows and members representing all aspects of epidemiology.

International Epidemiological Association. http://www.IEAWeb.org
An organization that aims to facilitate communication among those engaged in research and teaching in epidemiology throughout the world, and to engage in the development and use of epidemiologic methods in all fields of health, including social, community, and preventive medicine, and health administration.

International Society for Environmental Epidemiology. http://www.iseepi.org
An international organization that provides a variety of forums for discussions, critical reviews, collaborations, and education on issues of environmental exposures and their human health effects.

Society for Epidemiologic Research (SER). http://www.epiresearch.org
An organization that promotes research in epidemiology through meetings, publications, and other activities.

Occupational Health Psychology

American Psychological Association. http://www.apa.org.
The Work, Stress and Health Office of APA promotes research, training, practice, and policy to examine the impact of the changing organization of work on stress, health, safety, and productivity in the workplace.

Public Health and Preventive Medicine

American Public Health Association (APHA). http://www.apha.org.
The professional association for public health workers in a wide variety of disciplines and work settings, including occupational and environmental health. Its primary sections devoted to this field are the Occupational Health and Safety Section and the Environment Section.

American College of Preventive Medicine. http://www.acpm.org
A national professional society for physicians committed to disease prevention and health promotion.

Association for Prevention Teaching and Research (APTR). http://www.atpm.org
The professional organization for the academic, medical, and public health community dedicated to prevention, research, and education.

Multidisciplinary International Organizations

International Commission on Occupational Health (ICOH). http://www.icohweb.org
An international non-governmental professional society that aims to foster the scientific progress, knowledge, and development of occupational health and safety in all its aspects. Its numerous scientific committees hold regular symposia, and publish scientific monographs on important issues.

International Union for Health Promotion and Education (IUHPE). http://www.iuhpe.org/
An international professional network that operates through global working groups on select cross-cutting topics. Initiatives focus on health promotion effectiveness, social determinants of health and health inequalities, capacity building, education and training in health promotion, and non-communicable diseases.

Student Organization

American Medical Student Association. http://www.amsa.org
A student-governed, national organization that represents the concerns of physicians-in-training. Its action committees and interest groups help expose medical students to information on subjects not generally covered in traditional curricula. Its Community and Environmental Health Action Committee helps medical and premedical students educate their schools and communities about important public health issues, enables medical and premedical students to influence debate about and achieve progress related to these issues, empowers chapters to develop and execute local public health projects, and provides guidance and training in public health and public health careers.

Labor Organizations

Major U.S. Labor Federations

American Federation of Labor - Congress of Industrial Organizations (AFL-CIO). http://www.aflcio.org
The Change to Win Federation (CTW). http://www.changetowin.org

See Box 32-2 in Chapter 32 for a list of the 10 largest international unions.

COSH Groups

National Council for Occupational Safety and Health. http://www.coshnetwork.org
A federation of local and statewide Committee on Safety and Health (COSH) groups, which are coalitions of labor unions, occupational health and safety professionals, and labor and community activists who are committed to promoting worker health and safety through training, education, and advocacy.

National Construction Center

Center for Construction Research and Training. http://www.cpwr.com
Formerly known as the Center to Protect Workers' Rights, this non-profit organization created by the Building and Construction Trades Department, AFL-CIO, focuses on applied research, training, and service to the construction industry. This organization is an excellent source of information about health and technology related to the construction industry.

Worker Centers

Community-based worker centers address issues, such as discrimination and wage fraud, for disenfranchised worker populations, including immigrants and workers in low-wage jobs or in the informal work sector, such as day laborers. Two national networks that provide information about worker centers are the Interfaith Worker Justice network of workers' centers (http://www.iwj.org) and the National Day Labor Organizing Network (NDLON) (http://www.ndlon.org).

Environmental Non-Governmental Organizations)

See http://en.wikipedia.org/wiki/Category:Environmental_organizations for a list of illustrative environmental non-governmental organizations.

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