Chapter 4: Human Health

Indicators that were selected and included in this chapter were assigned to one of two categories:

- **Category 1** –The indicator has been peer reviewed and is supported by national level data coverage for more than one time period. The supporting data are comparable across the nation and are characterized by sound collection methodologies, data management systems, and quality assurance procedures.
- **Category 2** –The indicator has been peer reviewed, but the supporting data are available only for part of the nation (e.g., multi-state regions or ecoregions), or the indicator has not been measured for more than one time period, or not all the parameters of the indicator have been measured (e.g., data has been collected for birds, but not for plants or insects). The supporting data are comparable across the areas covered, and are characterized by sound collection methodologies, data management systems, and quality assurance procedures.

4.0 Introduction

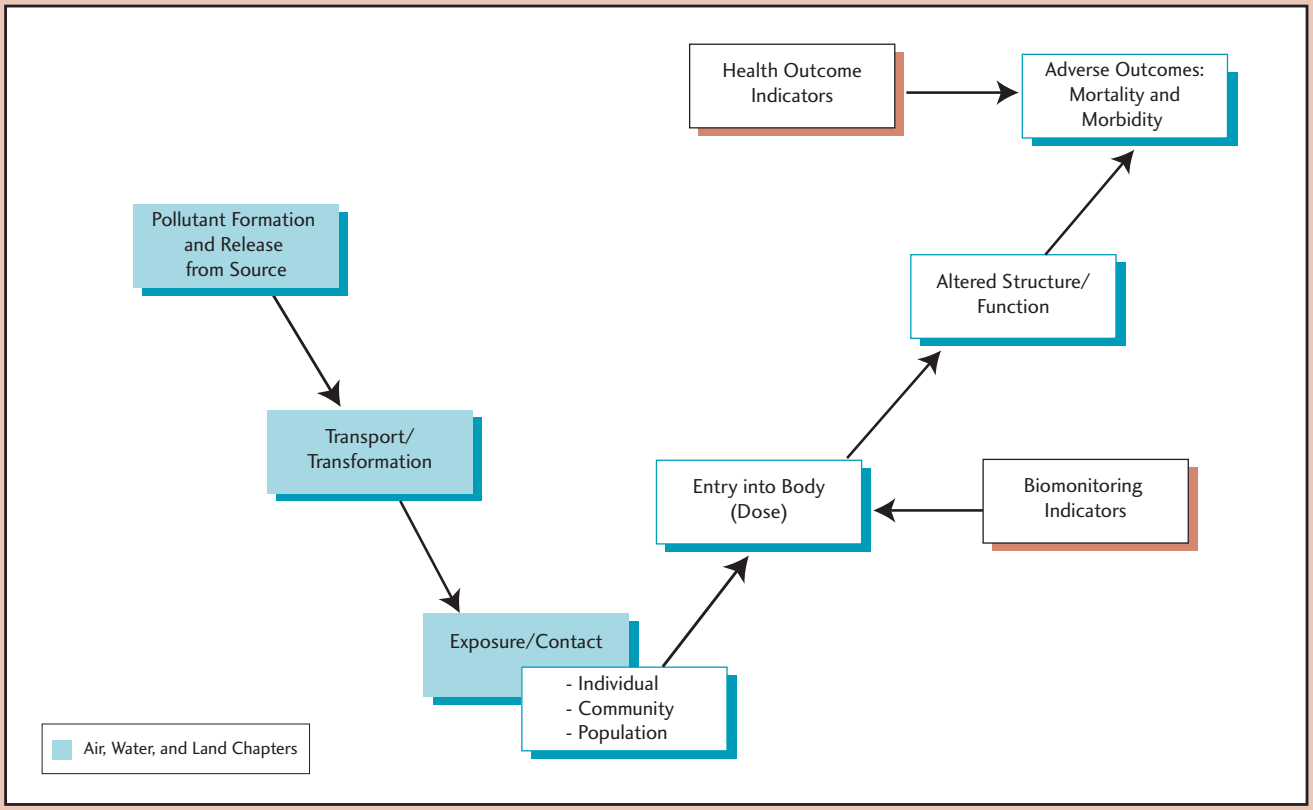
The U.S. Environmental Protection Agency (EPA) is moving in the direction of measuring and assessing human health and ecological outcomes. Traditionally, EPA has used indicators such as decreases in emissions/discharges or decreases in ambient pollutant levels to measure environmental improvement. Health outcome measures complement these traditional approaches by reflecting the actual public health or ecological impacts that result from environmental pollution. By providing a quantitative assessment of these impacts, outcome indicators can strengthen environmental decision-making and enhance EPA's ability to evaluate, prospectively or retrospectively, the success of those decisions.

The key to using outcome-based indicators is a clear understanding of the sequence of events that link changes in environmental conditions to health or ecological outcomes. Exhibit 4-1 depicts this sequence for human health. Each block in the diagram can have indicators associated with it. Indicators for the presence of pollutants or other stressors affecting air, water, and land are covered in Chapters 1 (Cleaner Air), 2 (Purer Water), and 3 (Better Protected Land), respectively, of this report. Indicators for the presence of pollutants in the body and their effects on health (altered structure or function, morbidity, or mortality) are covered in this chapter.

The paradigm depicted in Exhibit 4-1 underlies the science upon which EPA bases its risk assessment process (NRC, 1983). Risk assessments, to a large degree, seek to estimate all linkages depicted in the exhibit. However, understanding the link between human exposure and health outcomes has always been challenging. Decades of research have provided the scientific foundation for understanding how exposure to individual pollutants at elevated levels may affect human health. There is less certainty, however, about the effects of ambient exposures, which typically involve exposure to multiple pollutants at lower levels. Improved understanding of the linkages between these exposures and public health would strengthen EPA's ability to make and evaluate decisions.

The indicators that describe the public health consequences of environmental exposures are called environmental public health indicators (EPHIs). Numerous national and international organizations have recognized the compelling need for EPHIs. The greatest impetus came from a series of reports, by the Pew Environmental

Exhibit 4-1: Environmental public health paradigm



Source: Modified from National Research Council. *Risk Assessment in the Federal Government: Managing the Process*. 1983

Health Commission, which called on "Congress and the White House to protect Americans from chronic diseases—by tracking where and when these health problems occur and possible links to environmental factors." The commission proposed that a Nationwide Health Tracking Network be established to track selected diseases and priority environmental exposures (Pew, 2001). When combined with other information, such as environmental monitoring data and data from toxicological, epidemiological, or clinical studies, EPHIs can be an important key to improving understanding of the relationship between pollution and health outcomes.

Use of Environmental Public Health Indicators

Environmental public health indicators can be used to:

- **Describe** the health status of a population and discover important time trends in disease and exposure frequency. Most, if not all, of the indicators presented in this chapter perform this function.
- **Explain** the occurrence or prevalence of diseases and exposure by helping to identify causal factors for specific diseases or trends. For example, the decline in the lung cancer rate in men has been related to the decline in smoking. For some areas presented in this chapter, the evidence for a relationship is quite strong (e.g., air pollution and pulmonary-cardiovascular related-illnesses). Other areas will require further research to better understand these linkages.
- **Predict** the number of disease occurrences and the distribution of exposure in specific populations. Such predictions could be used, for example, as input for setting priorities and making decisions to protect public health—e.g., establishing cleanup levels for environmental waste sites or regulatory levels for ambient pollutant levels. (Understanding the relationship between exposure and consequent health effects is critical to using indicators for predictive purposes.)
- **Evaluate** policy decisions or interventions. (Again, understanding the relationship between exposure and effect is critical for this use.)

Two types of environmental public health indicators are described in this chapter:

- **Health outcome indicators.** These indicators measure the occurrence in a population of diseases or conditions that are known or believed to be caused to some degree or exacerbated by exposure to environmental pollutants or stressors.
- **Exposure indicators.** While there are four types of exposure indicators (see sidebar), this chapter focuses on biomonitoring indicators, which involve using tests of human fluid and tissue samples to identify the presence of a substance or combination of substances in the human body.

For some of the EPHIs described in this chapter, a strong linkage has been established between environmental exposure and outcome. However, for many of the EPHIs presented, such as the outcome indicator of overall mortality, no linkage between environmental exposure and outcome has been determined. For these, further research would be needed to establish and strengthen any linkages. Similarly, for some EPHIs, the linkage with the source of the pollution is clear (e.g., lead in gasoline), while for others the source or sources are much less certain.

Types of Exposure Indicators

Four approaches can be used to measure or estimate exposure (i.e., direct human contact with a pollutant). No approach is best suited to all pollutants. Different approaches are appropriate to different types of pollutants, and each approach has strengths and weaknesses.

- **Ambient pollutant measurements.** Historically, environmental measurements of ambient pollutant concentrations have generally been used to estimate human exposures. One limitation of ambient measurements is that the presence of a pollutant in the environment does not necessarily mean that anyone has been exposed. Chapters 1 (Cleaner Air), 2 (Purer Water), and 3 (Better Protected Land) provide examples of ambient measurement indicators.
- **Stochastic models of exposure.** This approach combines knowledge of environmental pollutant concentrations with information on people's activities and locations (e.g., time spent working, exercising outdoors, sleeping, shopping) to account for their contact with pollutants. This approach requires knowledge of pollutant levels where people live, work, and play, as well as knowledge of the choices that they make in regard to day-to-day activities.
- **Personal monitoring data.** With personal monitoring, the monitoring device is worn by individuals as they proceed through their normal activities. This approach is most common in workplaces. Personal monitoring data provide valuable insights into the sources of the pollutants to which people are actually exposed. However, a challenge with personal monitoring (as with biomonitoring) is ensuring that sufficient sampling is done to be representative of the population being studied.
- **Biomonitoring data.** Several environmental pollutants, notably heavy metals and some pesticides, can be found in the body. These pollutants or their breakdown products (i.e., metabolites formed when a pollutant is broken down in the body) leave residues that can be measured in human tissue or fluids such as blood or urine. These residues reflect the amount of the pollutant that actually gets into the body, but by themselves they provide no information on how the individual came into contact with the pollutant.

One of the greatest challenges to elucidating the connection between environmental exposure and disease is the fact that exposure to an environmental pollutant or stressor is rarely the sole cause of an adverse health outcome. More generally, individuals are exposed to more than one pollutant at a time, and exposure is just one of several factors that contribute to the disease occurring or to the severity of a preexisting disease. Other factors include, for example, diet, exercise, alcohol consumption, heredity, medications, and whether other diseases are also present. Also, different people have different vulnerabilities, so some may experience effects to certain ambient exposure levels while others may not. All these factors make it difficult to establish a causal relationship between exposure to environmental pollutants and disease outcome except in rare cases, such as some historical occupational exposures, where exposure was unusually high.

This chapter presents a broad spectrum of indicators that can now be used, or could potentially be employed in the future, to assess and track the public health impacts of environmental exposures. These indicators provide an overview of the health and exposure of people in the U.S. and identify the trends of those indicators in the U.S. Specific indicators for exposure and outcomes in children are presented, as children may be especially susceptible to environmental pollutants.

This chapter is organized into six sections:

- **Section 4.1** describes three case studies that illustrate the role of indicators in establishing linkages between effects and outcomes and in evaluating environmental management actions.
- **Section 4.2** compares health measures within the U.S. to these same measures throughout the rest of the world.
- **Section 4.3** discusses outcome indicators and trends for selected diseases that either have a major impact on the health of people in the U.S. or may be caused to some extent by environmental pollution. Exhibit 4-2 lists the key public health questions that are asked in this section and the indicators that are available to help answer these questions.
- **Section 4.4** presents biomonitoring indicators and trends for specific environmental pollutants. The section begins by providing background on biomonitoring indicators and their limitations and data sources. The section then presents biomonitoring indicators for numerous specific pollutants and discusses other important pollutants for which biomonitoring data are not yet available. Exposure information for many of these pollutants is discussed in Chapters 1 (Cleaner Air), 2 (Purer Water), and 3 (Better Protected Land) of this report. The key exposure questions asked in this section and the indicators available to help answer these questions are presented in Exhibit 4-2.
- **Section 4.5** discusses an emerging field that attempts to quantify the overall burden of environmental disease on society.
- **Section 4.6** discusses the key challenges and data gaps for understanding the link between environmental exposure and health outcomes, and some recent government activities to continue and advance the work in this area.

Many federal and state government agencies collect data that underlie environmental public health indicators. Continued effective coordination and collaboration among such agencies will be vital to further the development and use of environmental public health indicators. Key data sources used for this chapter include the:

- **World Health Organization (WHO), World Health Statistics Annual,** a joint effort by the national health and statistical administrations of many countries, the United Nations, and WHO.
- **United Nations, Demographic Yearbook,** a comprehensive collection of international demographic statistics compiled from questionnaires sent annually and monthly to national statistical services and other government offices.
- **National Center for Health Statistics, National Vital Statistics System,** which provides data on births, deaths, marriages, and divorces in the U.S. since 1933.
- **National Center for Health Statistics, National Health Interview Survey (NHIS),** a continuous nationwide survey in which data on personal and demographic characteristics, illnesses, injuries, impairments, chronic conditions, utilization of health resources, and other health topics are collected through personal household interviews.
- **Centers for Disease Control and Prevention, Epidemiology Program Office, National Notifiable Diseases Surveillance System,** which provides weekly provisional information on the occurrence of diseases defined as notifiable (i.e., a disease that health providers must report to state or local public health officials due to its contagiousness, severity, or frequency).
- **National Institutes of Health, National Cancer Institute, Surveillance, Epidemiology, and End Results Program,** which provides data on all residents diagnosed with cancer in 11 geographic areas of the U.S.
- **The EPA's National Human Exposure Assessment Survey (NHEXAS),** a multiday, multimedia study that examined chemical concentrations in indoor air, outdoor air, dust, soil, food, beverages, drinking water, and tap water.
- **National Center for Health Statistics, National Health and Nutrition Examination Survey (NHANES)**, a series of surveys designed to collect data on the health and nutritional status of the U.S. population. Chemicals and their metabolites were measured in blood and urine samples from selected participants.

The chapter is not intended to be exhaustive. Rather, it provides a snapshot, at the national level, of the current U.S. environmental public health indicators and status based on key data sources with sufficiently robust design, quality assurance, and maturity. The chapter does not provide health status information that may be more applicable to certain geographic areas or to subgroups with potentially greater susceptibility to environmental pollution due to such factors as age, genetics, lifestyle, or medical status.

Exhibit 4-2: Human Health - Questions and Indicators

Health Status of the U.S. : Indicators and Trends of Health and Disease

Question	Indicator Name	Category	Section
What are the trends for life expectancy?	Life expectancy	1	4.3.1
What are the trends for cancer, cardiovascular disease, chronic obstructive pulmonary disease and asthma?	Cancer mortality	1	4.3.2
	Cancer incidence	2	4.3.2
	Cardiovascular disease mortality	1	4.3.2
	Cardiovascular disease prevalence	1	4.3.2
	Chronic obstructive pulmonary disease mortality	1	4.3.2
	Asthma mortality	1	4.3.2
	Asthma prevalence	1	4.3.2
What are the trends for gastrointestinal illness?	Cholera prevalence	2	4.3.3
	Cryptosporidiosis prevalence	2	4.3.3
	E. coli O157:H7 prevalence	2	4.3.3
	Hepatitis A prevalence	2	4.3.3
	Salmonellosis prevalence	2	4.3.3
	Shigellosis prevalence	2	4.3.3
	Typhoid fever prevalence	2	4.3.3
What are the trends for children's environmental health issues?	Infant mortality	1	4.3.4
	Low birthweight incidence	1	4.3.4
	Childhood cancer mortality	1	4.3.4
	Childhood cancer incidence	2	4.3.4
	Childhood asthma mortality	1	4.3.4
	Childhood asthma prevalence	1	4.3.4
	Deaths due to birth defects	1	4.3.4
	Birth defect incidence	1	4.3.4

Measuring Exposure to Environmental Pollution: Indicators and Trends

Question	Indicator Name	Category	Section
What is the level of exposure to heavy metals?	Blood lead level	1	4.4.3
	Urine arsenic level	2	4.4.3
	Blood mercury level	1	4.4.3
	Blood cadmium level	1	4.4.3
What is the level of exposure to cotinine?	Blood cotinine level	1	4.4.4
What is the level of exposure to volatile organic compounds?	Blood volatile organic compound levels	1	4.4.5
What is the level of exposure to pesticides?	Urine organophosphate levels to indicate pesticides	1	4.4.6
What is the level of exposure to persistent organic pollutants?	No Category 1 or 2 indicators identified		4.4.7
What are the trends in exposure to environmental pollutants for children?	Blood lead level in children	1	4.4.8
	Blood mercury level in children	1	4.4.8
	Blood cotinine level in children	1	4.4.8
What is the level of exposure to radiation?	No Category 1 or 2 indicators identified		4.4.9
What is the level of exposure to air pollutants?	No Category 1 or 2 indicators identified Also see Cleaner Air chapter		4.4.9
What is the level of exposure to biological pollutants?	No Category 1 or 2 indicators identified		4.4.9
What is the level of exposure to disinfection by-products?	No Category 1 or 2 indicators identified		4.4.9

4.1 Environmental Pollution and Disease: Links Between Exposure and Health Outcomes

Many studies have demonstrated an association between environmental exposure and certain diseases or other health problems. Examples include radon and lung cancer; arsenic and cancer in several organs; lead and nervous system disorders; disease-causing bacteria such as *E. coli* O157:H7 (e.g., in contaminated meat and water) and gastrointestinal illness and death; and particulate matter and aggravation of cardiovascular and respiratory diseases.

As mentioned in Section 4.0, indicators of outcome and exposure can be important tools both for elucidating these links and monitoring the success of environmental management efforts. Indicators are one of several components needed to establish linkage. Other important components include ambient pollutant measures and toxicological, epidemiological, and clinical studies. Three case studies are described in this section to demonstrate how indicators can be used to establish associations between exposure and effect and to evaluate environmental management actions.

Case Study on Waterborne Disease

This case study focuses on the impact of drinking water treatment on the decrease in mortality related to waterborne diseases. It demonstrates the valuable contribution to public health protection that can occur when the link between exposure and health outcomes is successfully made. As the case study describes, officials knew there was a high incidence of gastrointestinal disease, but they were not able to protect human health until they understood what caused these diseases. Based on this connection, officials were able to take effective action to protect public health. They also were able to use an outcome measure (deaths due to typhoid) to evaluate the success of these protective actions.

At the beginning of the 20th century, waterborne diseases such as typhoid fever and cholera were major health threats across the U.S. More than 150 in every 100,000 people died from typhoid fever each year. Deaths due to diarrhea-like illnesses, including typhoid, cholera, and dysentery, represented the third largest cause of death in the nation.

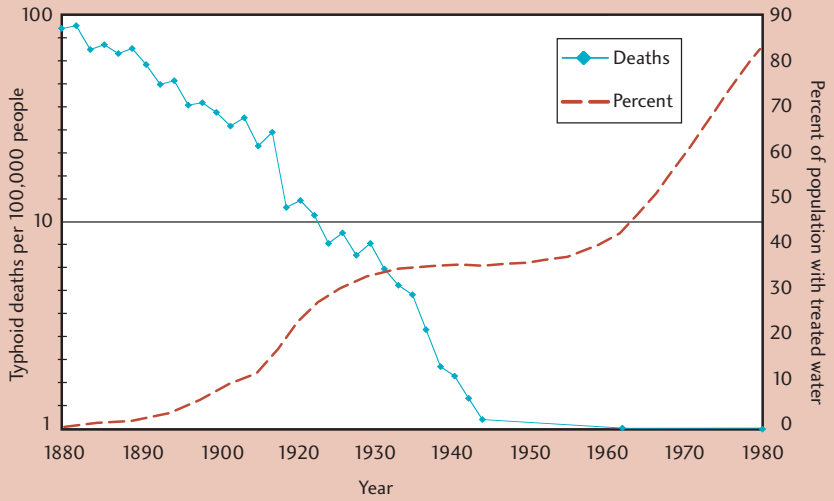
Then scientists identified the bacteria responsible for most diarrhea deaths (typhoid, cholera, and dysentery) and elucidated how these bacteria were transmitted to and among humans. Infected and diseased individuals shed large quantities of microbes in their feces, which flowed into and contaminated major water supplies. The contaminated water was then distributed untreated to communities, which used the water for drinking and other purposes. This created a continuous transmission cycle.

When treatment (filtration and chlorination) of drinking water was initiated to remove pathogens, the number of deaths due to diarrhea diseases dropped dramatically. Deaths due to typhoid fever were tracked throughout the early 20th century, as drinking water treatment was implemented across the country. Exhibit 4-3 shows the percent of the U.S. population that had treated water and the disease rate for typhoid fever from 1880 to 1980.

In this example, the outcome measure was death rates due to typhoid, which was used in conjunction with an environmental process (the number of people getting treated drinking water) to evaluate and promulgate the use of drinking water treatment across the U.S.

Drinking water treatment was one of the great public health success stories of the 20th century (NAE, 2000). It dramatically and significantly reduced death rates from waterborne disease, increasing

Exhibit 4-3: Percent of population with treated water versus typhoid deaths in the United States, 1880-1980



Source: Craun, C.G. *Waterborne Diseases in the United States*. 1986; Whipple, G.C. *Typhoid Fever - Its Causation. Transmission and Prevention.* 1908; Fox, K. National Risk Management Research Laboratory, personal communication, 2003.

life expectancy and reducing infant mortality. Today, public health is protected against new and emerging waterborne microbial contaminants by continual improvements to the drinking water treatment process and continual monitoring of waterborne diseases. Deaths due to cholera, typhoid, and dysentery are so rare in this country that they do not provide valuable information for evaluating the public health impacts of drinking water treatment. Instead, the number of cases of these diseases are tracked to some extent, although reporting is not federally required. Indicators for waterborne disease and other important diseases with actual or potential environmental origins are discussed in Section 4.3.

Case Study on Air Pollution

This case study illustrates how the association between deaths and peak air pollution concentrations was initially discovered by comparing mortality rates and air monitoring data. It also describes how basic research on the health effects of air pollution has helped to establish strong linkages between levels of certain air pollutants and human health effects. These associations have provided sufficient basis for establishing regulations to control the level of pollutants in air. The success of these environmental management efforts can be evaluated by monitoring levels of regulated pollutants in air. However, except for lead (the subject of the third case study below), there are as yet no biomonitoring or outcome indicators that can more directly measure reduced human exposure or outcome on a national level. Nevertheless, a number of potential outcome indicators are discussed that could be available in the future if systems can be set up to track relevant biomonitoring or outcome data with sufficient reliability and coverage at a national level.

Air pollution has been associated with several human health outcomes, including reported symptoms (nose and throat irritation), acute onset or exacerbation of existing disease (e.g., asthma, hospitalizations due to cardiovascular disease), and deaths. The impact of air pollution on health was underscored in London in December of 1952, when a slow-moving area of high pressure came to a halt over the city. Fog developed, and particulate and sulfur pollution began accumulating in the stagnating air mass. Smoke and sulfur dioxide concentrations built up over 3 days. Mortality records showed that deaths increased in a pattern very similar to that of the pollution measurements. (This is illustrated in Exhibit 4-4.) It was estimated that 4,000 extra deaths occurred over a 3- to 4-day period. This was the first quantitative air pollution exposure data with a link to an adverse health outcome (i.e., mortality).

While the London episode highlighted the hazard of extreme air pollution episodes, it was unclear whether health effects were associated with lower concentrations. By the 1970s, the association between respiratory disease and particulate and/or sulfur oxide air pollution had been well established (Dockery and Pope, 1997).

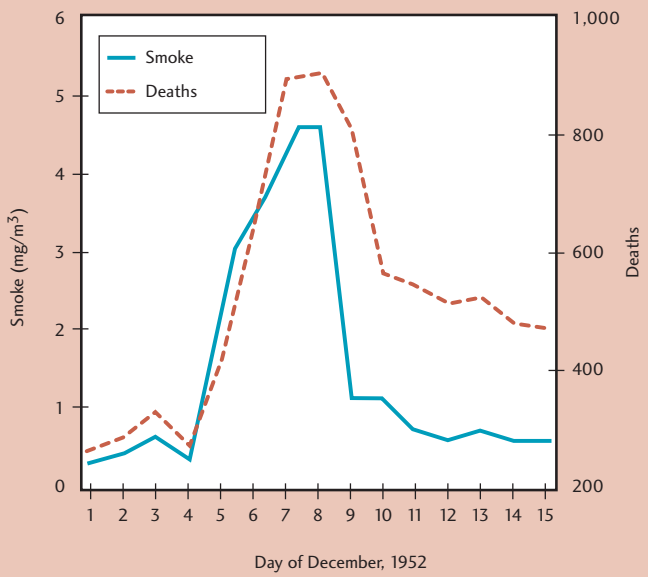
Clinical studies (controlled studies in healthy adult subjects) also provide information about the association between air pollutants and health effects. For example, these studies have demonstrated that ozone causes a number of functional, symptomatic, and inflammatory responses, which tend to increase with an increase in ozone exposure dose (EPA, 1996). Effects of ozone include:

- Decreased pulmonary function, characterized by changes in lung volumes and flow; changes in airway resistance and responsiveness; and respiratory symptoms, such as cough and pain on deep inspiration (EPA, 1996).
- An inflammatory response in the lungs (EPA, 1996).

Based on these types of associations from toxicological, epidemiological, and clinical studies, EPA has established National Ambient Air Quality Standards for six pollutants of concern: ozone, particulate matter, carbon monoxide, lead, nitrogen dioxide, and sulfur dioxide. These standards set limits to protect human health, including the health of "sensitive populations" such as asthmatics, children, and the elderly (EPA, 1999).

Improvements in measuring air pollution and health endpoints, together with advances in analytical techniques, have made it possible to begin to quantitatively evaluate the success of air pollution control measures—such as the National Ambient Air Quality Standards and associated regulations—to protect and improve public health. Though insufficient data were available at the time of this report to develop EPHIs for any criteria pollutants except lead, possible future EPHIs for air pollution include death due to respiratory and cardiovascular disease as well as increased hospital admissions for respiratory and cardiovascular disease.

Exhibit 4-4: London fog episode deaths, 1952



Source: Based on Dockery, D.W. and C.A. Pope. *Outdoor Air I: Particulates.* 1997.

Future EPHIs include:

- **Mortality.** In many countries including the U.S., particulate air pollution has been associated with increased daily mortality from heart and lung diseases (e.g., congestive heart disease, chronic obstructive lung disease). In addition, chronic exposure to air pollution has been linked with increased risk of premature mortality (EPA, April 2002).
- **Hospital admissions.** Hospitalization records are not widely available, and studies have been limited by their availability in communities around the U.S. Nevertheless, many studies have shown that increased admissions for cardiovascular and respiratory diseases are associated with increased pollutant concentrations.

Most recently, subtle changes in the cardiovascular system that can increase a person's risk of heart attack and bring about other cardiovascular effects have been identified as possible EPHIs.

Establishing EPHIs for air pollution and health effects, whether cardiovascular or pulmonary, is still challenged by limits in knowledge of how much air pollution contributes to the risk of both cardiovascular and respiratory disease. Research is still needed to better understand which components of air pollution (i.e., gases, metals, or organics) cause health effects; the extent to which they contribute to risk; and the extent to which other factors (e.g., genetics, lifestyle, age) contribute to risk. Given these limitations, no indicators are presented for any of the six criteria pollutants except lead. A case study on lead is presented below, with further discussion on lead as an indicator provided in Section 4.4.

Case Study on Lead

The third case study concerns lead, a toxic pollutant to which there is human exposure from many different sources. In the previous case studies, outcome indicators were an important key to establishing a linkage between a health effect and its cause. Understanding the cause enabled officials to take action to protect public health. In the case of lead, though it was a known toxin, exposure came from so many sources that it was difficult to know what actions at the national level would effectively reduce lead exposure. Once regulations to do so were put in place, biomonitoring data provided a way to evaluate the success of this environmental management effort in reducing exposure to lead in the U.S.

Lead is a neurotoxic metal that affects areas of the brain that regulate behavior and nerve cell development (NAP, 1993). Its adverse effects range from subtle responses to overt toxicity, depending on how much lead is taken into the body and the age and health status of the person (CDC, 1991).

Currently in the U.S., human exposure to lead may occur in several ways, as listed in Exhibit 4-5. For example:

- Homes built before 1978, commercial buildings, and steel structures may contain deteriorating lead-based paint, which creates lead-contaminated dust (EPA, 1996). An estimated 24 million housing units in the U.S. are at risk for containing some lead paint hazards (U.S. Department of Housing and Urban Development, 2000). Of these, 16 million homes with lead-based paint have children in residence who are younger than 6 years old.
- Other sources of lead exposure include lead-contaminated soil, dust, and drinking water; industrial emissions; and miscellaneous sources (CDC, 1991).

For many years, the largest source of lead in the U.S. environment came from leaded gasoline. Elemental lead was emitted in the exhaust and settled on the ground and in people's homes.

Most lead enters the body via ingestion and inhalation, after which it is absorbed by the bloodstream. Also, lead can cross the placenta, exposing the fetus to lead (EPA, 1996). In adults, most lead poisoning is associated with occupational exposures.

Infants, children, and fetuses are more vulnerable to the effects of lead because their blood-brain barrier is not fully developed (Nadakavukaren, 2000). In addition, ingested lead is more readily absorbed into a child's bloodstream. Children absorb 40 percent of ingested lead into their bloodstreams, while adults absorb only 10 percent. In children, three major organ systems are affected by lead: the nervous system (the brain), the kidney, and the blood-forming organs (NRC, 1993).

Exhibit 4-5: Sources of lead exposure in the United States

Lead-based paint	Homes (built before 1978) Commercial buildings Steel structures (bridges, water towers)
Lead-contaminated soil and dust	Industrial emissions Past leaded gasoline use Deteriorating lead-based paint
Lead-contaminated drinking water	Leaded plumbing solder (now banned)
Miscellaneous	Home hobbies - art, jewelry, fishing weights Use of pewter dishware Cosmetics, traditional medicines Parental occupations

Source: CDC. *Preventing Lead Poisoning in Young Children.* 1991.

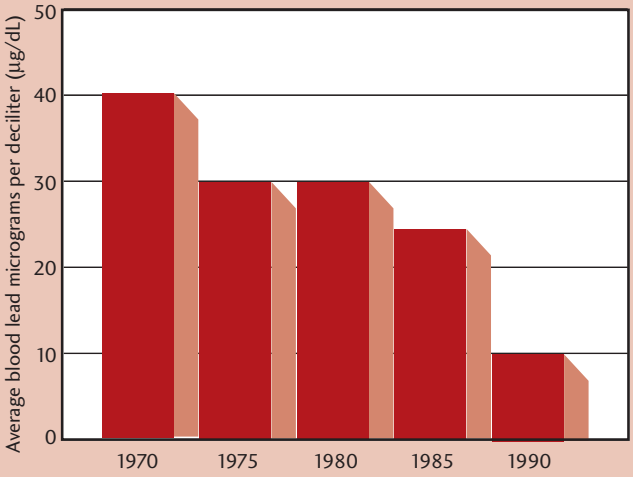
As awareness of the health effects of lead has increased, the CDC has lowered the level considered to be a human health hazard (Exhibit 4-6) (CDC, 1991). In 1970, a blood lead level of 40 micrograms per deciliter (µg/dL) or higher was considered a hazard. Today, 10 µg/dL or higher is considered a hazard (EPA, December 2000). Recent research suggests that blood lead levels less than 10 µg/dL may still produce subtle, subclinical health effects in children (Schmidt, 1999). In 1984, an estimated 6 million children and 400,000 fetuses were exposed to lead at levels that placed them at risk for adverse effects (NAP, 1993). Approximately 4.4 percent of all U.S. children in the 1990s had elevated blood lead levels (NCEH, 1998). As of 1998, an estimated 1 million U.S. children had blood lead levels above 10 µg/dL (NCEH, 1998).

Lead is one of the few pollutants for which biomonitoring and linkage data are sufficient to clearly evaluate environmental management efforts to reduce lead in the environment. The National Center for Health Statistics' National Health and Nutrition Examination Survey (NHANES), a national survey of the health status of the U.S. population, has determined blood lead levels for the U.S. population since the early 1970s. In the 1970s, lead poisoning occurred increasingly in children who did not live in dwellings with lead-based paint, suggesting that another source or sources of lead exposure were of even greater concern than lead paint. Research found that combustion of leaded gasoline was the primary source of lead in the environment. EPA promulgated two regulations:

- One required the availability of unleaded fuel for automobiles designed to meet federal emission standards (e.g., catalytic converters) (EPA, 1973).
- The second required a reduction of the lead content in leaded gasoline (EPA, 1986).

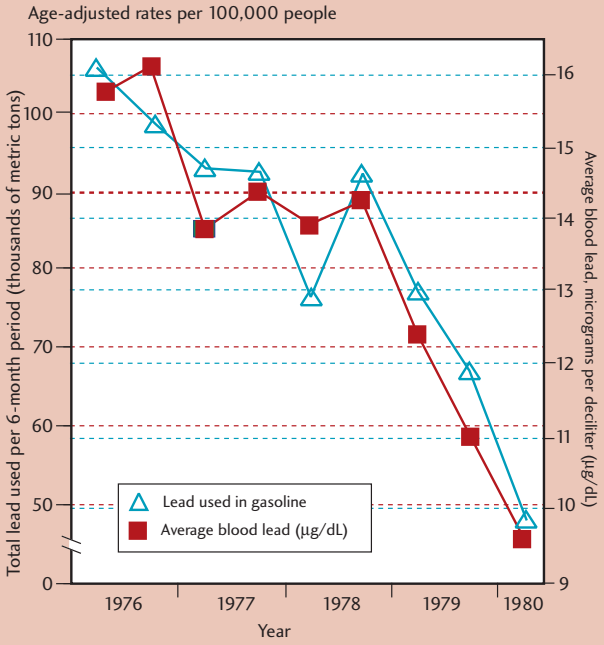
Over the next decade, peak outdoor-air lead concentrations decreased as a result of these controls. Exhibit 4-7 compares the amount of lead used in gasoline production and the average blood lead levels provided by the NHANES from 1976 to 1980. The NHANES survey found a similar decline in children's blood lead levels (Exhibit 4-8). In 1991, a report from the National Academy of Sciences predicted that declining ambient lead levels would reduce the average blood lead level to less than 15 µg/dL. By the late 1990s, the average blood lead level in the U.S. for children was 3 µg/dL (Schmidt, 1999). These data show a demonstrable effect between regulatory actions to control lead and human exposure.

Exhibit 4-6: Blood lead levels considered elevated by the Centers for Disease Control and Prevention and the Public Health Service, 1970-1990



Source: CDC. *Preventing Lead Poisoning in Young Children.* 1991.

Exhibit 4-7: Lead used in gasoline production and average National Health and Nutrition Examination Survey (NHANES) blood lead, United States, 1976-1980

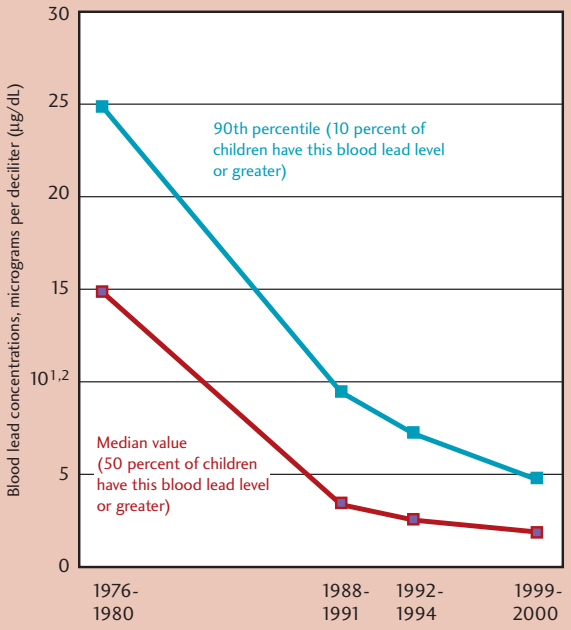


Source: National Research Council. *Measuring Lead Exposures in Infants, Children and Other Sensitive Subpopulations.* 1993.

Elucidating Other Linkages

For all three case studies, the linkage between exposure and disease is fairly strong. Subsequent sections of this chapter describe a number of areas of concern regarding the potential human health impacts of environmental exposure. The linkage in these areas ranges from strong to weak. For example, in some cases outcome indicators are available, but scientists are not yet sure how much of that outcome is contributed by environmental factors. In other cases, biomonitoring indicators are available, but scientists are not sure whether the presence of a contaminant in the body at the levels shown by the indicators causes adverse health effects. These areas are discussed in this chapter, despite relatively weak linkages, because the use of outcome and biomonitoring indicators is a developing area. Understanding of linkages will be strengthened over time as more research is conducted to develop environmental public health indicators and other data that reveal how pollutants contribute to disease.

Exhibit 4-8: Concentration of lead in blood of children age 5 and under, 1976-1980, 1988-1991, 1992-1994, 1999-2000



[1] 10 µg/dL of blood lead has been identified by CDC as elevated, which indicates the need for intervention. (CDC. *Preventing Lead Poisoning in Young Children.* 1991.)

[2] Recent research suggests that blood levels less than 10 µg/dL may still produce subtle, subclinical health effects in children. (Schmidt, C.W. *Poisoning Young Minds.* 1999.)

Source: U.S. Environmental Protection Agency. *America's Children and the Environment-Measures of Contaminants, Body Burdens, and Illnesses, Second Edition.* February 2003. Data from CDC, National Center for Health Statistics, National Health and Nutrition Examination Survey, 1976-2000.

4.2 Health Status of the U.S. Compared to the Rest of the World

Several measures are used worldwide to describe health status. These indicators include life expectancy (i.e., the number of years people can expect to live at birth), the number of infant deaths, and the major causes of deaths.

Collecting and reporting the data necessary to compare these measures between nations is a challenge. Yet, as travel and communications increasingly link the health of nations in the world, the importance of having comparable information has increased. Fortunately, considerable progress has been made to improve the comparability of the necessary data among nations.

In addition to enabling comparisons of health status, the data also can be used to inform U.S. environmental health policy and programs, to focus research efforts, and to provide insights into linkages between environmental factors and health.

Life Expectancy

Life expectancy is the average number of years at birth that a group of infants would live if throughout life they experienced the age-specific death rates present at birth. In 2000, life expectancy at birth for all people in the U.S. was a record 76.9 years (Pastor, et al., 2002). In 1997, the U.S. ranked 19th in terms of life expectancy for both females and males when compared with other countries (Exhibit 4-9). Life expectancy at birth varies widely, both between males and females and between nations. For both sexes, Japan reports the highest life expectancy of all nations, with males expected to live 77.2 years and females expected to live 83.8 years.

Infant Mortality

Infant mortality is a particularly useful measure of health status because it indicates both the current health status of the population and predicts the health of the next generation (NCHS, 2001). Between 1970 and 2000, the infant mortality rate in the U.S. declined from 20.0 to 6.9 per 1,000 live births, the lowest ever recorded in the U.S. (Pastor, et al., 2002; Mannino and Smith, 2001). When compared to other countries, the U.S. ranked 11th in 1960 with regard to infant mortality. In 1998, the U.S. ranked 28th (Exhibit 4-10).

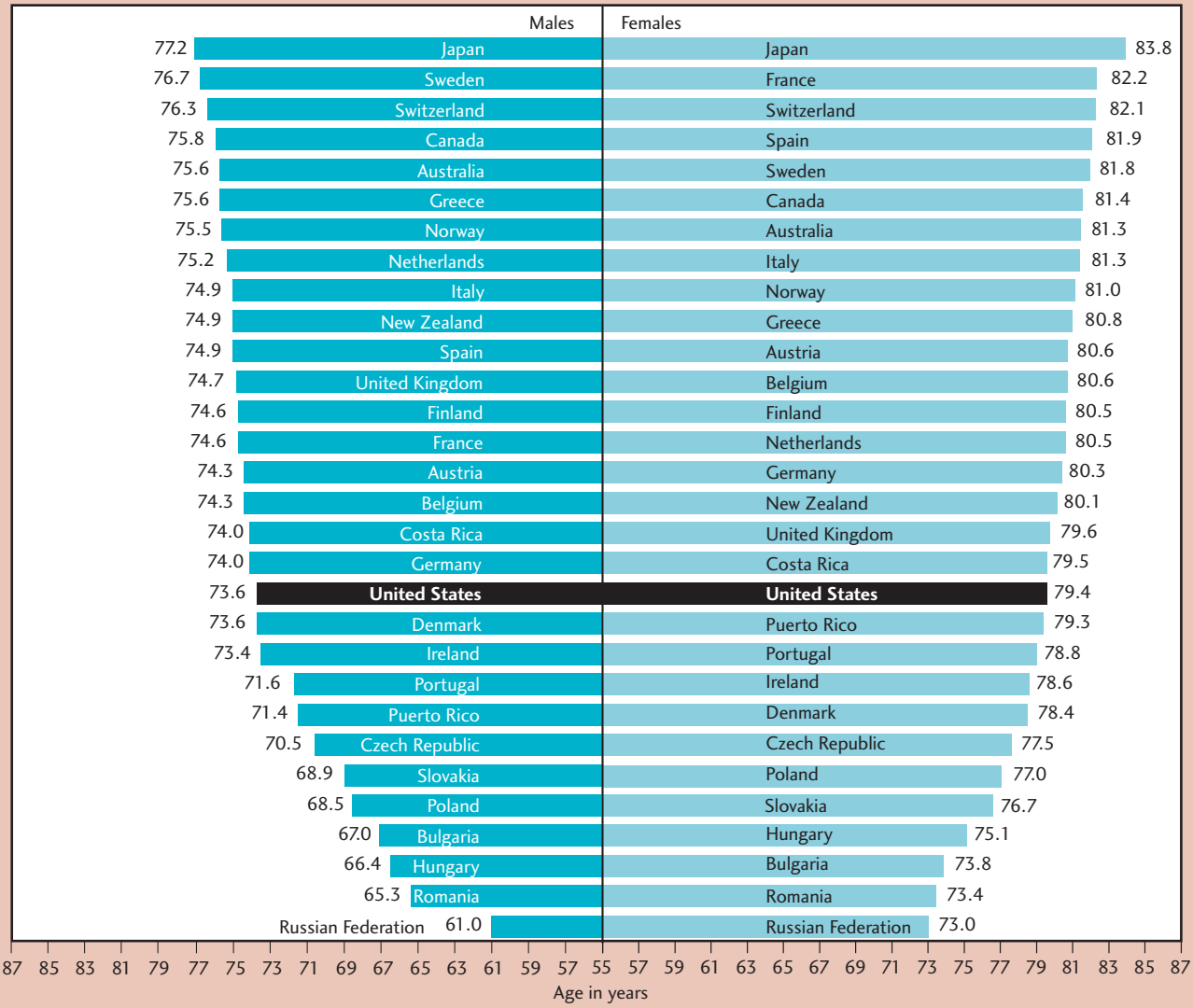
Leading Causes of Death

It is customary to measure the health of a nation by listing the leading causes of death. Comparisons of the 10 leading causes of death in the U.S. and for the world demonstrate that infectious diseases are a major contributor to deaths outside of the U.S. Four of the 10 leading causes of death in the world are infectious diseases (Exhibit 4-11). These diseases account for 20.3 percent of the deaths worldwide. Heart disease is the leading cause of death in the U.S. as well as in the world. While heart disease accounts for nearly one-third of the deaths in the U.S., it accounts for only 12.4 percent of the deaths in the world.

Cancer Morbidity and Mortality

The age-adjusted cancer mortality rates for all body sites except skin are higher for males than females in all of the countries presented in Exhibit 4-12. There is wide variation among men and women in age-adjusted cancer death rates. Hungary has the highest age-adjusted total cancer (except skin) death rates for both males and females (272.3 and 149.4 per 100,000 people, respectively). The U.S. ranks 16th for males, with an age-adjusted cancer death rate of 161.8 per 100,000, and 10th for females, with an age-adjusted cancer death rate of 116.4 per 100,000. Sweden has the lowest age-adjusted

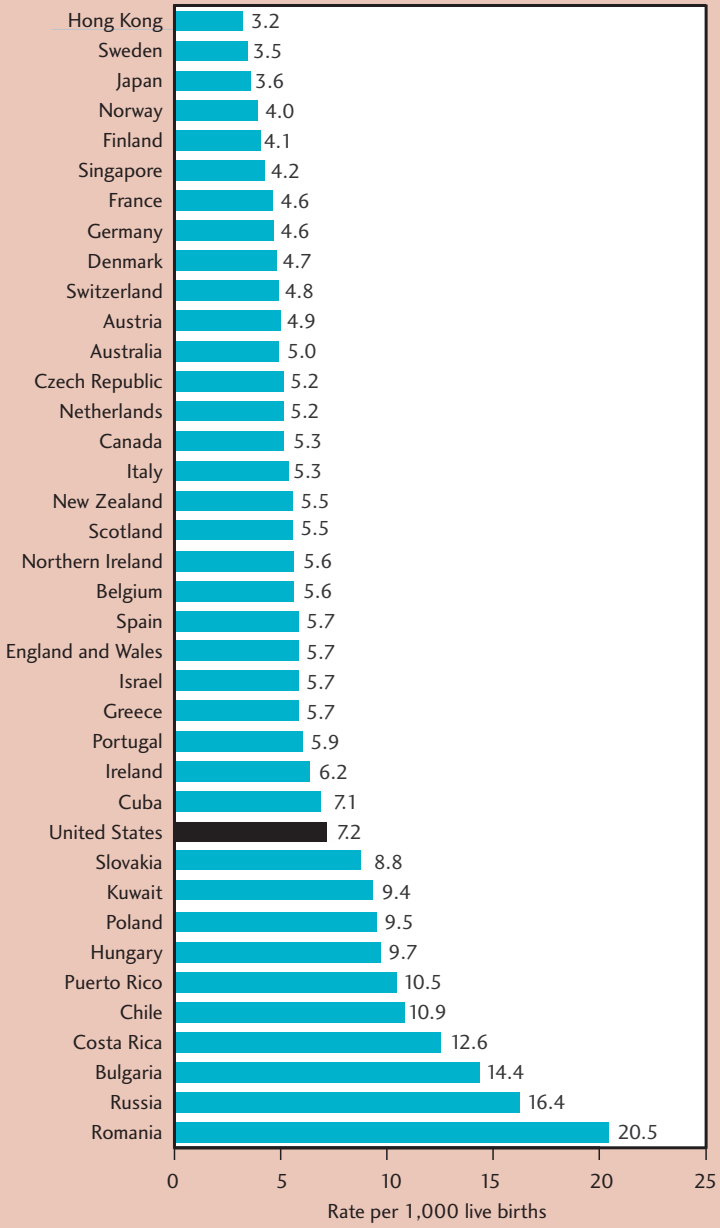
Exhibit 4-9: Life expectancy at birth, according to sex, United States and selected countries, 1997



Note: Rankings are from highest to lowest life expectancy based on the latest data available for countries or geographic areas with at least 1 million people.

Source: Pastor, R.N., et al. *Health. United States, 2002*. 2002.

Exhibit 4-10: Infant mortality rates per 1,000 live births, United States and selected countries, 1998*



* Data for Kuwait, Slovakia, and Spain are for 1996.

Source: Pastor, R.N., et al. *Health. United States,2002*. 2002.

cancer death rate for males, and Greece has the lowest rate for females (137.9 and 81.8 per 100,000, respectively) (United Nations, 2001).

The age-adjusted incidence of cancer for all sites except skin varies widely among different countries (Exhibit 4-13). Hungary reported the highest age-adjusted incidence of cancers for males (405.4 per 100,000 people). New Zealand had the highest age-adjusted cancer incidence rate for females (303.2 per 100,000 people). The U.S. has the third highest age-adjusted cancer incidence rates for both males and females (361.4 and 283.2, respectively). Age-adjusted cancer incidence rates are higher for males than females in each of the countries presented in Exhibit 4-13 except Denmark (GLOBOCAN 2000, 2001).

The varying incidence and mortality rates for cancer between different countries could be due to many factors. Factors related to the economic, social, cultural, psychological, behavioral, and biological mechanisms that influence the onset of cancer may contribute to these differences in rates (NCI, 2002). A portion of these differences might also be attributable to the varying prevalence of certain behavioral risk factors for cancer—such as cigarette smoking, diet, and alcohol consumption—within different countries. The availability and use of certain drugs, such as anticancer and immunosuppressive drugs, may also cause differences in the rates of cancer among different countries. The extent to which early diagnoses and treatment methods are available and utilized could also account for some portion of the variation in cancer rates among different countries, as could variations in methods of classifying and reporting cancer.

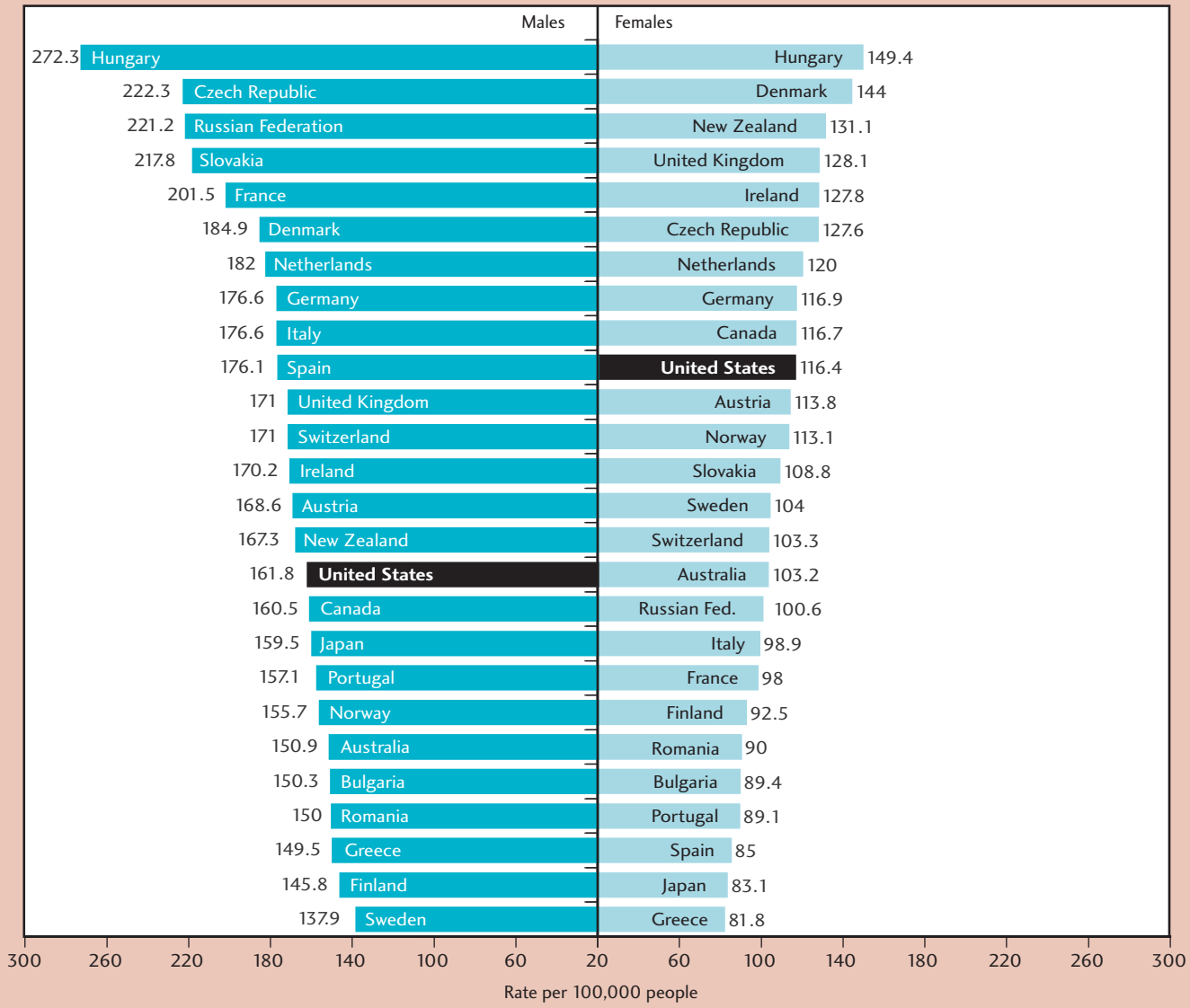
For more on morbidity, mortality, and age-adjusted rates, see Section 4.3.

Exhibit 4-11: Number of deaths and percent of total deaths for 10 leading causes of death, world (including U.S.), 1990, and United States, 1999

Cause of Death	Number of Deaths	Percent of Total Deaths
World (Including U.S.) (1990)		
All causes	50,467,000	100.0
Heart disease	6,260,000	12.4
Stroke	4,381,000	8.7
Lower respiratory infections	4,299,000	8.5
Diarrheal diseases	2,946,000	5.8
Conditions arising during the perinatal period	2,443,000	4.8
Chronic obstructive pulmonary disease	2,211,000	4.4
Tuberculosis	1,960,000	3.9
Measles	1,058,000	2.1
Road traffic accidents	999,000	2.0
Trachea, bronchus and lung cancers	945,000	1.9
All other causes	27,502,000	54.5
United States (1999)		
All causes	2,391,399	100.0
Heart disease	725,192	30.3
Cancer	549,838	23.0
Stroke	167,366	7.0
Chronic lower respiratory diseases	124,181	5.2
Accidents (unintentional injuries)	97,860	4.1
Diabetes mellitus	68,399	2.9
Influenza and pneumonia	63,730	2.7
Alzheimer's disease	44,536	1.9
Nephritis, nephritic syndrome, and nephrosis	35,525	1.5
Septicemia	30,680	1.3
All other causes	484,092	20.2

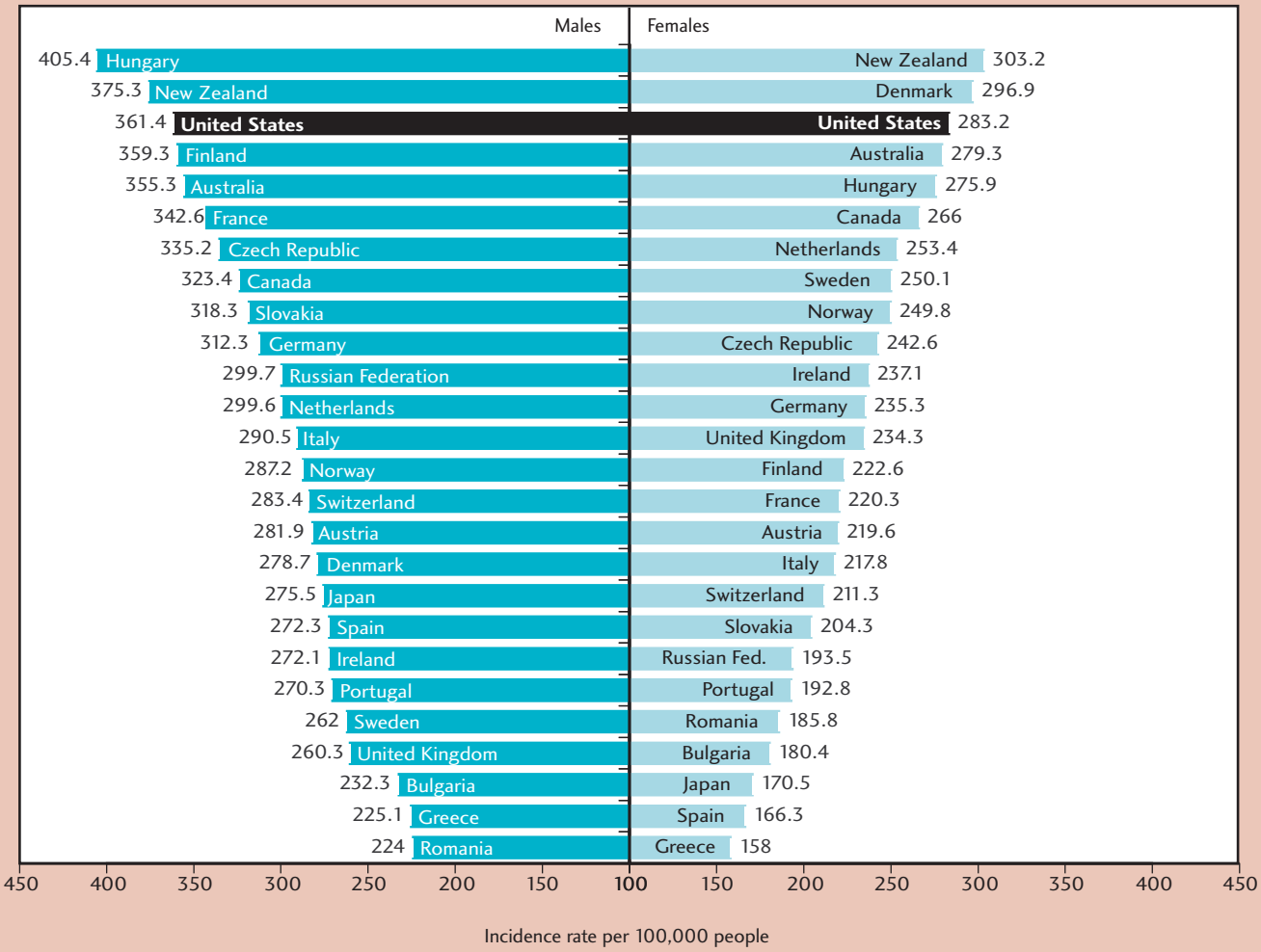
Sources: World Resources Institute, et al. *World Resources 1998-99*. 1998; Anderson, R.N. *Deaths: Leading Causes for 1999*. 2001.

Exhibit 4-12: Age-adjusted cancer mortality rates for all sites except skin, by sex for selected countries, 2000



Source: United Nations. *Demographic Yearbook, 1999*. 2001.

Exhibit 4-13: Age-adjusted cancer incidence rates for all sites except skin, by sex for selected countries, 2000



Source: GLOBOCAN. *Cancer Incidence, Mortality, and Prevalence Worldwide, Version 1.0.* 2001; International Agency for Research on Cancer. *IARC Cancer Base No. 5.* 2001.

4.3 Health Status of the U.S.: Indicators and Trends of Health and Disease

This section identifies key indicators of health outcomes (mortality and disease) in the U.S. and describes trends for these outcomes. These outcomes are featured in this report because they are important measures of the health of people in the U.S., and/or because environmental exposure does or may play a role in contributing to the outcome.

The case study on air pollution, presented earlier in Section 4.1, provides an example of how health outcome data can be used to elucidate the linkage between pollution exposure and health outcomes. In this case study, a comparison between mortality rates and air monitoring data revealed an association between deaths and peak air pollutant concentrations.

Mortality

Overall mortality is a key measure of health in a population. There were more than 2,391,399 deaths in the U.S. in 1999 (Anderson, 2001), a number much larger than the 1,989,841 recorded in 1980. The increase in the number of deaths reflects the increase in the size and the aging of the U.S. population. The age-adjusted death rate for all causes has declined steadily since 1950, from 1,446 per 100,000 people to 876 in 1998. The age-adjusted death rates are higher for men than for women, a relationship that has not changed over the years. Heart disease, cancer, and stroke are the three leading causes of death, accounting for about 60 percent of all deaths.

This section presents trends in life expectancy and in mortality due to cancer, cardiovascular disease, chronic obstructive pulmonary disease, and asthma. It also presents trends in mortality for children, including infant mortality and mortality due to cancer, asthma, and birth defects.

Unless otherwise noted, the death statistics are based on the underlying cause of death and are compiled from death certificates. The underlying cause of death is the disease or injury that is judged to have initiated the events that led to death. The mortality rate is the proportion of the population that dies of a disease. The rate is usually calculated for a calendar year, is often expressed per 100,000 population, and is called the crude death rate.

Morbidity

Morbidity is another measure of health for a population. Morbidity data are often described by using the incidence and prevalence of a disease or condition:

- **Incidence** refers to the number of new cases of a disease or condition in a given time period in a specified population.
- **Prevalence** refers to the total number of persons with a given disease or condition in a specified population in a particular time period.

This section provides information on trends for several diseases, including cancer, cardiovascular disease, asthma, and gastrointestinal illness. It also examines trends in children's environmentally related diseases, including cancer and asthma as well as low birthweight and the incidence of birth defects.

Comparison Across Time, Populations, and Geographic Areas

Incidence, prevalence, and mortality statistics may be used to compare the rates of disease at two or more points in time or across different populations or between different geographic areas. These comparisons are particularly useful to determine whether the populations differ by some factor (often called a risk factor) that is known or suspected of affecting the risk of developing the disease or condition. For example, different populations that are compared can be countries, workers in factories, or states.

In general, disease incidence, prevalence, and mortality increase with age. For this reason, when comparing different populations, the data must often be adjusted to account for the age differences between the populations. The adjusted data, called "age-adjusted rates," are used when appropriate in this chapter.

Perceived Well-Being

Another measure of health, perceived well-being, is discussed briefly here, but is not covered by an indicator. The reporting of health as excellent, very good, good, fair, or poor captures both the physical health of the individual and the emotional aspects of well-being (Kramarow, et al., 1999). In 1999, approximately 90 percent of the population of the U.S. reported that they were in good, very good, or excellent health (Eberhardt, et al., 2001), a slight increase from 89.6 percent in 1991. As might be expected, the percentage of people reporting good-to-excellent health decreases with age. While 95 percent of those 18 to 44 years of age reported good-to-excellent health, only 77 percent of persons 65 years of age and older reported that they were in good-to-excellent health. Also, non-Hispanic African Americans and Hispanics of all ages reported worse health than non-Hispanic Whites (Eberhardt, et al., 2001).

This section addresses five questions:

- What are the trends for life expectancy? (Section 4.3.1)
- What are the trends for cancer, cardiovascular disease, chronic obstructive pulmonary disease, and asthma? (Section 4.3.2)
- What are the trends for gastrointestinal illness? (Section 4.3.3)
- What are the trends for children's environmental health issues? (4.3.4)
- What are the trends for emerging health effects? (Section 4.3.5)

4.3.1 What are the trends for life expectancy?

Life expectancy is the average number of years at birth that a group of infants would live if throughout life they experienced the age-specific death rates present at birth.

Indicator: Life expectancy - Category I

The primary source for data on life expectancy in the U.S. is the National Center for Health Statistics (NCHS). Through its National Vital Statistics System, the NCHS has collected and published data on births, deaths, marriages, and divorces in the U.S. since 1933. U.S. data are for the 50 states and the District of Columbia, unless otherwise specified. Virtually all births and deaths are registered. U.S. Standard Certificates of Live Birth and Death are revised periodically, usually every 10 to 15 years. New versions of the U.S. Standard Certificates of Live Birth and Death are planned for 2003. Most state certificates conform closely in content and arrangement to the standard certificate recommended by NCHS, and all certificates contain a minimum data set specified by NCHS. At the time of birth, the mother provides demographic information on the birth certificate, such as race and ethnicity. Medical and health information is based on hospital records. Demographic information on the death certificate is provided by the funeral director based on information supplied by an informant. A physician, medical examiner, or coroner provides medical certification of cause of death.

What the Data Show

Throughout the 20th century there has been a general improvement in life expectancy at birth in the U.S. (Hoyert, et al., 2001). In 2000, life expectancy at birth reached a record high of 76.9 years, based on preliminary data. In 1999, life expectancy was 76.7 years (Pastor, et al., 2002). This follows 5 consecutive years of improvement and a general upward trend in life expectancy throughout the 20th century.

The gap in life expectancy between males and females widened from 2.0 years to 7.8 years between 1900 and the late 1970s. Now this gap is narrowing, and in 2000 the difference in life expectancy between the sexes was 5.4 years. This improvement was primarily due to a greater reduction in mortality for males from heart disease, cancer, suicide, and homicide. Between 1970 and 1999, life expectancy at birth in the U.S. increased from 67.1 to 73.9 years for males and from 74.7 to 79.4 years for females (Pastor, et al., 2002; Mannino and Smith, 2001).

In 1999, life expectancy at birth for the African American population reached a record high of 71.4 years. In 2000, the difference in life expectancy between the African American and White populations was 5.6 years, based on preliminary data. Based on 1999 data, White females continue to have the highest life expectancy (79.9 years), followed by African American females (74.7 years), White males (74.6 years), and African American males (67.8 years). The narrowing of the gap in life expectancy between Whites and African Americans was largely due to a greater reduction in mortality for African Americans due to homicide, cancer, stroke, and HIV-related disease.

Data Source

National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-25, for information.)

4.3.2 What are the trends for cancer, cardiovascular disease, chronic obstructive pulmonary disease, and asthma?

Several chronic diseases that are important indicators of health are presented in this section. Cardiovascular disease, cancer, and stroke are the three leading causes of death in the U.S., accounting for 60.3 percent of all deaths (Anderson, 2001). Chronic obstructive pulmonary disease, a category of diseases that restrict airflow through parts of the respiratory system, was the fourth leading cause of death in the U.S. in 1999 (Hoyert, et al., 2001). Asthma, a chronic condition characterized by inflammation of the airways and lungs, affected more than 10 million people in the U.S. in 1999 (Mannino, et al., 2002).

Indicators

Cancer mortality - Category 1
Cancer incidence - Category 2

The term "cancer" is used to characterize diseases in which abnormal cells divide without control. A cancerous cell loses its ability to regulate its own growth, control cell division, and communicate with other cells. Cancer cells can invade nearby tissues and can spread through the bloodstream and lymphatic system to other parts of the body (NCI, 2003).

What the Data Show

In the U.S., 549,838 people died of cancer in 1999. The death rate was 201.6 per 100,000 people. Cancer accounted for 23 percent of all deaths (Anderson, 2001). Between 1990 and 1998, the age-adjusted death rates for all types of cancer for all persons declined from 173.3 to 161.5 per 100,000 people. The death rate for cancer is highest for non-Hispanic Whites (232.8 per 100,000 people). The death rate for cancer for non-Hispanic African Americans is 185.6 per 100,000 and for Hispanics is 64.6 per 100,000 (Hoyert, et al., 2001). Death rates for different types of cancer show differences across age, gender, and ethnic lines.

Over the past century, the age-adjusted incidence rate for all cancers for all persons decreased from 400.3 per 100,000 people to 395.3. Age-adjusted incidence rates have not declined uniformly over all types of cancer. For example, the incidence of lung cancer for men was 69.8 per 100,000 in 1998, a decline from 81.8 in 1990 and from 76.2 in 1975. For women, the 1998 age-adjusted lung cancer incidence rate of 43.4 per 100,000 people was an increase from 41.6 in 1990 and was nearly 2 times the 1975 rate of 21.5 (Ries, et al., 2001).

Exhibit 4-14 shows the estimated percent change in death and incidence rates according to the type of cancer for men and women of all races, between 1973 and 1998. Notable is the 150.6 percent increase in lung cancer deaths for females between 1973 and 1998. Despite the progress in reducing the number of new cases of some types of cancer, the incidence rates for all types of cancers combined increased 22.4 percent (Ries, et al., 2001).

Data Sources

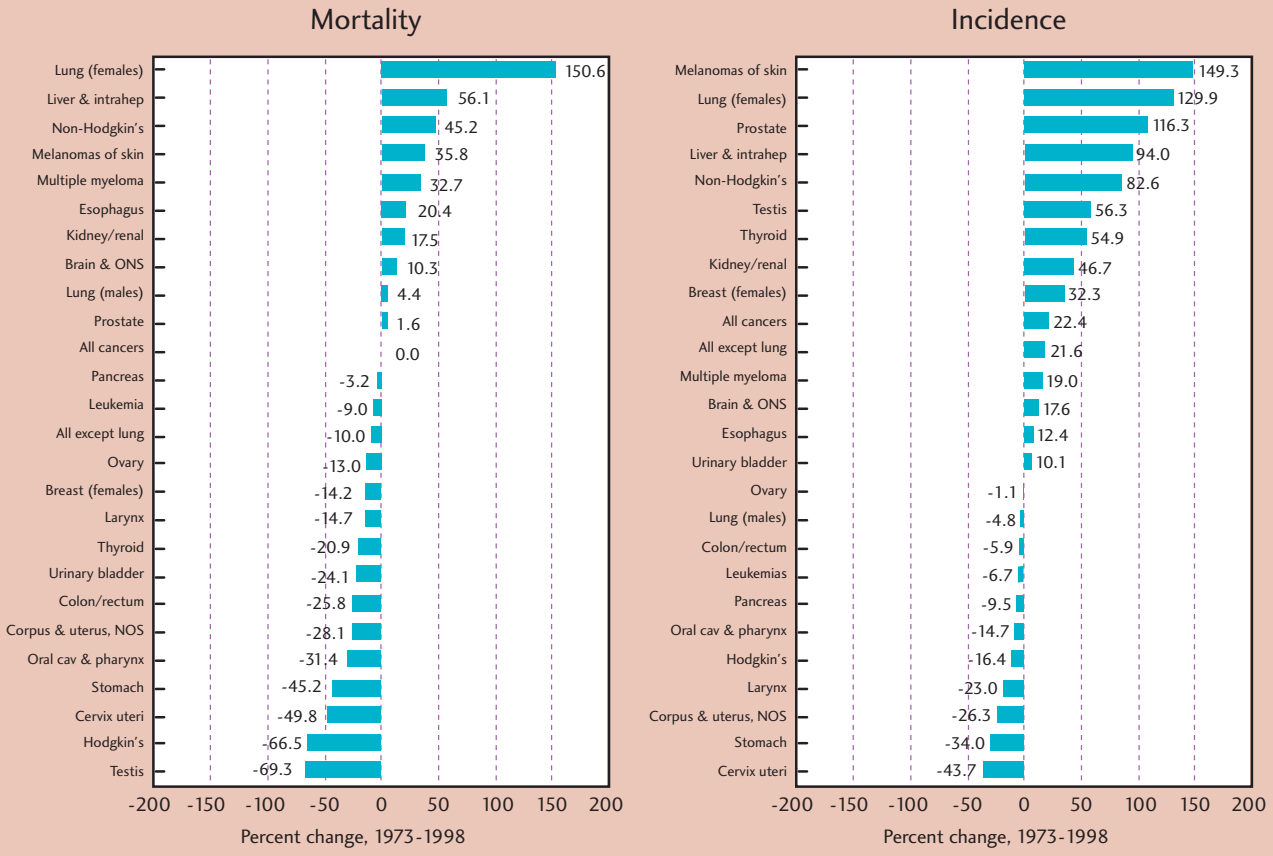
Mortality: National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-25 for more information.)

Incidence: National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-25 for more information.)

Indicators

Cancer mortality - Category 1 (continued)
Cancer incidence - Category 2 (continued)

Exhibit 4-14: Trends in United States cancer mortality rates and Surveillance, Epidemiology and End Results (SEER) incidence (annual percentage change 1973-1998)



NOS = Not otherwise specified
ONS = Other nervous system

Source: Ries, L.A.G., et al. *Surveillance, Epidemiology and End Results (SEER) Cancer Statistics Review, 1973-1998*. 2001.

Indicators

Cardiovascular disease mortality - Category I
Cardiovascular disease prevalence - Category I

The broad category of cardiovascular disease (CVD) includes any disease involving the heart and blood vessels. Coronary heart disease (CHD) and cerebrovascular disease, commonly known as stroke, are the major cardiovascular diseases.

What the Data Show

Because there are several conditions included in the cardiovascular disease category, it is not surprising that the National Heart, Lung, and Blood Institute (NHLBI) estimates that approximately 59.7 million people in the U.S. have some form of CVD (NHLBI, 2000). An estimated 12.2 million people have coronary heart disease and 4.4 million have had a stroke.

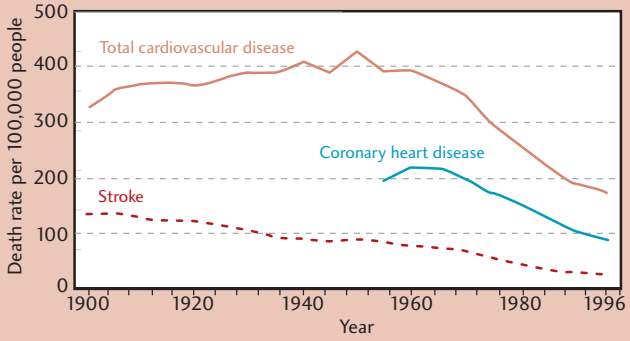
CVD is the leading cause of death for both men and women in the U.S. (AHA, 2001). The age-adjusted death rate for CVD reached a peak in 1950. Between 1950 and 1999, the age-adjusted death rate for CVD declined 60 percent (Exhibit 4-15) (CDC, 1999a). The percentage of all deaths due to CVD increases with age, from 19 percent at 35 to 44 years of age, to 53 percent for people 85 years and older.

The prevalence of cardiovascular disease varies depending upon the age and sex of the individual (Exhibit 4-16). CVD is more prevalent in men than in women until 65 years of age, when the prevalence among women equals that in men. After age 74 years, the prevalence is greater in women than in men. The age-adjusted prevalence of CVD in adults for non-Hispanic Whites is 30.0 percent for men and 23.8 percent for women; for non-Hispanic African Americans it is 40.5 percent for men and 39.6 percent for women.

The death rate for CHD was 195.6 per 100,000 people in 1999 (AHA, 2001). The death rates were lower for White men (249.4 per 100,000 people) than for African American men (272.6) and higher for African American women (192.5) than for White women (152.5) (AHA, 2001).

After age 45, the prevalence of CHD is lower for women than for men at all ages and increases with age for both men and women, peaking after 75 years of age (Exhibit 4-17). The age-adjusted prevalence for CHD for non-Hispanic Whites is 6.9 percent for men and 5.4 percent for women. For non-Hispanic African Americans, the prevalence is 7.1 percent for men and 9.0 percent for women (AHA, 2001).

Exhibit 4-15: Death rates for total cardiovascular disease, coronary heart disease, and stroke, by year, United States, 1900-1996

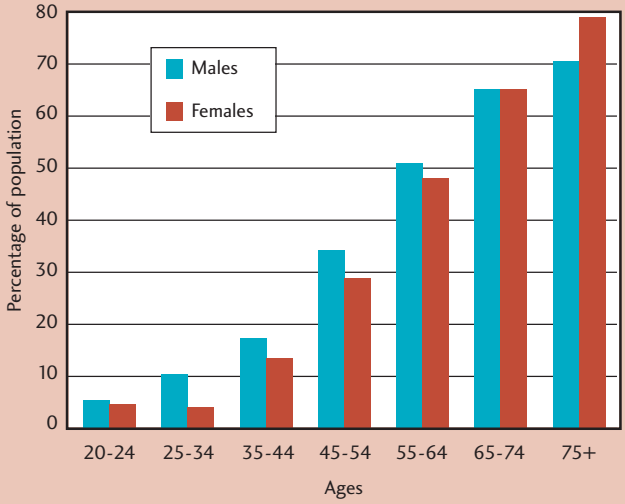


Notes: Rates are per 100,00 people, age adjusted to the 1940 U.S. population.

Diseases are classified according to International Classification of Diseases (ICD) codes in use when the deaths were reported.

Source: CDC. *Decline in Deaths from Heart Disease and Stroke, United States, 1900-1999*. 1999.

Exhibit 4-16: Prevalence of cardiovascular diseases among adults by age and sex, United States, 1988-1994



Source: American Heart Association. 2001 Heart and Stroke Statistical Update. 2001. Data from NHANES III, 1988-1994, the CDC, National Center for Health Statistics, and the American Heart Association.

Indicators

Cardiovascular disease mortality - Category I (continued)
Cardiovascular disease prevalence - Category I (continued)

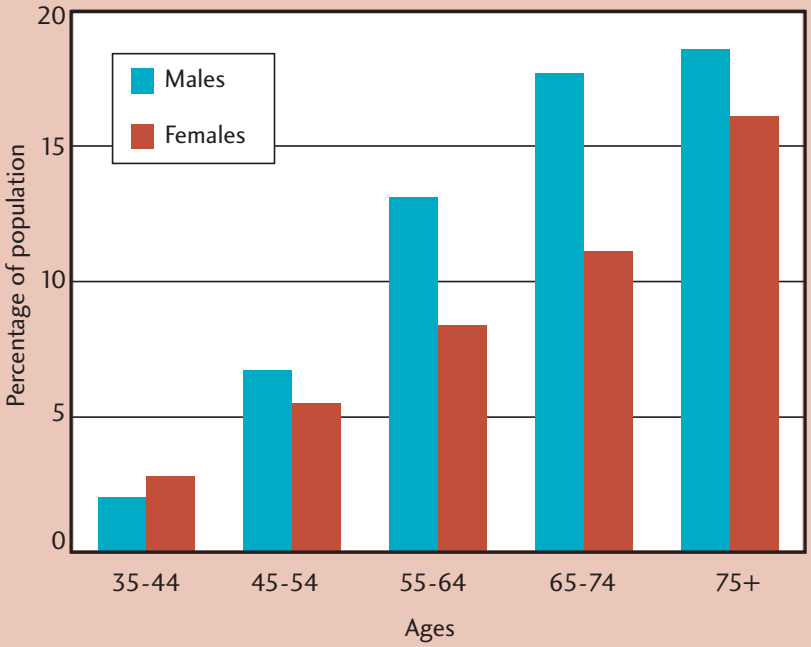
Stroke ranks as the third leading cause of death in the U.S. Stroke accounted for 7.0 percent of total deaths. The death rate for stroke was 61.4 deaths per 100,000 people. The age-adjusted prevalence of stroke is higher for men than for women at all ages. In 1999, there were 167,366 deaths (102,881 were females) attributed to stroke (Anderson, 2001). Death rates for stroke were highest among non-Hispanic Whites (70.8 per 100,000 people), followed by non-Hispanic African Americans (56.6) and Hispanics (18.8).

Data Sources

Mortality: National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-26 for more information.)

Prevalence: NHANES III (1988-1994), National Center for Health Statistics. (See Appendix B, page B-26, for more information.)

Exhibit 4-17: Prevalence of coronary heart disease by age and sex, United States, 1988-1994



Source: American Heart Association. 2001 *Heart and Stroke Statistical Update.* 2001. Data from NHANES III, 1988-1994, the CDC, National Center for Health Statistics, and the American Heart Association.

Indicator Chronic obstructive pulmonary disease mortality - Category I

Chronic obstructive pulmonary disease (COPD), sometimes referred to as chronic lung disease, is a disease that damages lung tissue or restricts airflow through the bronchioles and bronchi (ALA, 2001). Chronic bronchitis and emphysema are the most frequently occurring COPDs.

What the Data Show

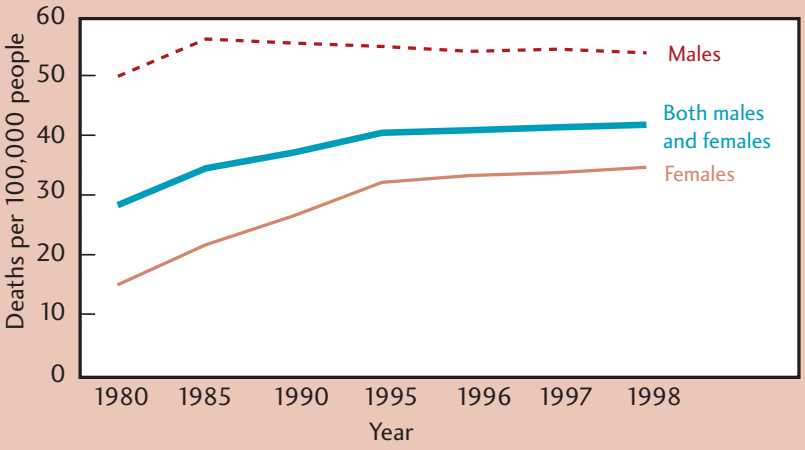
In 1999, COPD was the fourth leading cause of death, accounting for more than 124,181 deaths (5.2 percent of total deaths) (Hoyert, et al., 2001). The age-adjusted death, rate for COPD was 45.8 per 100,000 population. From 1980 to 1998, the age-adjusted death rates for COPD increased from 28.3 to 42.0 per 100,000 population for men and women of all racial and ethnic groups in the U.S. (Eberhardt, et al., 2001). For females, the age-adjusted death rates for COPD increased steadily from 1980 to 1998, from 14.9 per 100,000 population to 34.8 in 1998 (Exhibit 4-18). For males, the age-adjusted death rates rose between 1980 and 1985 from 49.9 to 56.2 per 100,000. From 1990 to 1998, the rate remained generally stable, declining slightly in 1998 to 54.0 per 100,000.

In 1998, the age-adjusted death rate for COPD was highest for White males at 55.4 per 100,000 population, followed by African American males (45.2) and Hispanic males (26.2 per 100,000 population). Among females, White females had the highest rates (36.5) followed by African American females (22.3) and Hispanic females (13.7 per 100,000 population) (Hoyert, et al., 2001).

Data Source

National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-26, for more information.)

Exhibit 4-18: Age-adjusted death rates for chronic obstructive pulmonary disease according to sex, selected years, in the United States 1980-1998



Source: Eberhardt, M.S., et al. *Health, United States, 2001 with Urban and Rural Health Chartbook.* 2001.

Indicators

Asthma mortality - Category I
Asthma prevalence - Category I

Asthma is a chronic respiratory disease characterized by inflammation of the airways and lungs. During an asthma attack the airways that carry air to the lungs are constricted, and as a result less air is able to flow in and out of the lungs (NCHS, 2001). Asthma attacks can cause a multitude of symptoms ranging in severity from mild to life-threatening. These symptoms include wheezing, breathlessness, chest tightness, and coughing (NCHS, 2001).

What the Data Show

In 1999, 4,657 people died from asthma. The age-adjusted death rate was 17.2 per 1,000,000 population. Exhibit 4-19 shows the trends in age-adjusted death rates with asthma as the underlying cause of death.

In 1999, approximately 10.5 million people in the U.S. reported that they had an asthma attack or episode in the preceding 12 months (Mannino, et al., 2002). This included approximately 7.3 million people over 14 years of age. The 1999 age-adjusted prevalence of asthma for people of all ages was 38.4 per 100,000 population in 1999 (Exhibit 4-20). That same year, the prevalence of asthma in adults was highest (42.2 per 100,000) for people 15 to 34 years of age, and lowest (22.1 per 100,000) for those 65 years of age and older. African Americans were more likely to report an asthma episode or attack than other race/ethnic groups, and females were more likely than males to have had an asthma episode or attack. Since 1997, the age-adjusted prevalence of asthma has decreased slightly from 40.7 per 100,000 population to 38.4 per 100,000. Changes in the way asthma data are collected were made in 1997, limiting the ability to compare current data with earlier reports.

There are regional differences in the prevalence of asthma with the highest prevalence in the Northeast (61.8 per 1,000 people) (Adams, et al., 1999). The prevalence in the Midwest was 56.6 per 1,000 people. The prevalence in the South (51.8 per 1,000) was similar to the prevalence in the West (52.9 per 1,000). People who lived in a central city reported a higher number of cases (61.7 per 1,000 people) than those who did not live in the central city (54.9 per 1,000). Those who did not live in a Metropolitan Statistical Area (an urbanized area with at least 50,000 inhabitants) had the lowest prevalence, 46.9 per 1,000 people.

Data Sources

Mortality: National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-27 for more information.)

Prevalence: National Health Interview Survey, Centers for Disease Control and Prevention. (See Appendix B, page B-27, for more information.)

Exhibit 4-19: Annual rate* of deaths with asthma as the underlying cause of death diagnosis, by race, sex, and age group, United States, 1980-1999

	1980	1985	1990	1995	1996	1997	1998	1999
Race								
White	12.9	15.6	17.5	18.8	18.1	17.4	17.0	14.2
Black	27.6	34.8	40.9	46.2	48.0	42.5	44.7	38.7
Other	13.5	16.9	23.6	23.3	27.6	26.6	22.7	20.4
Sex								
Male	14.7	15.9	17.8	17.9	17.7	16.6	16.5	13.1
Female	14.4	19.2	22.1	25.1	25.0	23.7	23.3	20.4
Age Group (years)								
0-4	1.8	1.5	2.0	1.8	2.3	1.9	2.1	1.7
5-14	1.9	2.9	3.2	4.0	4.6	3.4	3.8	3.6
14-34	3.0	4.2	5.0	6.7	6.5	6.1	6.4	5.9
35-64	14.0	17.7	18.8	20.6	20.3	19.0	17.8	15.8
65+	61.8	72.5	87.0	90.8	90.3	86.7	86.9	69.9
Total**	**14.4**	**17.7**	**20.2**	**21.9**	**21.8**	**20.6**	**20.3**	**17.2**

* Per 1,000,000 population. ** Age adjusted to the 2000 U.S. population.

Source: Mannino, D.M., et al. *Surveillance for Athma - United States, 1980-1999*. 2002.

Indicators

Asthma mortality - Category 1 (continued)
Asthma prevalence - Category 1 (continued)

Exhibit 4-20: Estimated annual prevalence of self-reported asthma (1980-1996) or an episode of asthma attack (1997-1999) during the preceding 12 months, by race, sex, and age group, United States, 1980-1999

	1980	1985	1990	1995	1996	1997	1998	1999
	Self-Reported Asthma Prevalence During the Preceding 12 Months					Episode of Asthma or Asthma Attack During the Preceding 12 Months		
Race								
White	31.4	37.0	41.5	54.5	53.6	40.5	37.5	37.6
Black	33.1	38.6	45.8	64.8	65.6	45.4	46.7	42.7
Other	19.9	12.8	40.2	44.4	43.2	34.7	33.7	38.9
Sex								
Male	30.5	33.8	39.1	48.6	43.0	33.0	31.7	31.6
Female	31.9	38.9	44.2	61.1	65.6	47.9	44.4	44.5
Age Group (years)								
0-4	23.0	36.7	44.0	60.5	40.1	41.2	46.4	42.1
5-14	45.1	50.9	63.7	82.0	69.8	60.0	57.8	56.4
15-34	30.0	36.1	37.3	57.8	67.2	44.2	37.5	42.2
35-64	29.9	30.8	38.4	50.1	46.2	37.0	35.7	33.4
65+	31.9	38.6	36.3	39.4	45.5	27.3	28.7	22.1
Total	**31.4**	**38.6**	**41.9**	**55.2**	**54.6**	**40.7**	**39.2**	**38.4**

Data are per 100,000 population, per year.

Source: Mannino, D.M., et al. *Surveillance for Asthma - United States, 1980-1999*. 2002.

4.3.3 What are the trends for gastrointestinal illness?

The human gastrointestinal tract includes the stomach, the large intestine, and the small intestine. Gastrointestinal infections and illnesses are caused by several types of microorganisms—that is, bacteria, protozoa, fungi, and viruses. Food and water contaminated with pathogenic microorganisms are the major environmental source of gastrointestinal illness. A system for reporting food- and waterborne disease outbreaks has been in place for many years in the U.S. This system enables public health officials to investigate and determine the role of food and water in contributing to intestinal illness, and identify actions that may be needed to protect public health. For example, the system tracks the number of waterborne disease outbreaks reported voluntarily by state, territorial, and local public health officials (See box, "Waterborne Disease Outbreaks Associated with Drinking Water 1971-2000"). These data should be interpreted with caution, however, because many factors can influence whether a waterborne disease outbreak is recognized, investigated, and reported. Changes in the number of outbreaks reported could reflect actual changes or simply changes in surveillance and reporting. (For additional information on waterborne disease, see Chapter 2, Purer Water.)

The number of deaths from microorganism-induced gastrointestinal illnesses recently increased in the U.S., after decades of relatively stable death rates (Peterson and Calderon, 2003). The increases were particularly dramatic in young children (less than 6 years of age) and older Americans (more than 65 years of age). Many milder cases of gastrointestinal illnesses go unreported or are not diagnosed, making it difficult to estimate the number of people affected every year. Often, symptoms are not serious enough to warrant a visit to a doctor or hospital, which further contributes to the underestimation of gastrointestinal illness.

Seven notifiable gastrointestinal diseases caused by microorganisms have been chosen as indicators for this report: cholera, cryptosporidiosis, *Escherichia coli* O157:H7, Hepatitis A, salmonellosis, shigellosis, and typhoid fever. The reporting period

includes five years 1997-2001. These include two diseases—cholera and typhoid fever—that are rarely identified in this country. These diseases are nevertheless included because they can be severe illnesses and a sudden increase in their reporting would signal a public health emergency for which prompt action would be needed. In addition to the seven diseases discussed here, a number of other gastrointestinal diseases are caused by microorganisms. These include giardiasis, caused by the pathogen *Giardia*. Giardiasis has become notifiable only as recently as 2002 (CDC, 2003), so no indicator is available at this time.

The primary means of transmission for the seven diseases reported here is oral-fecal. The disease microbes shed in the feces of infected individuals and then can be transmitted to humans through food, water, person-to-person contact, or contact with ill animals. The seven diseases are cholera, cryptosporidiosis, *E. coli* O157:H7, Hepatitis A, salmonellosis, shigellosis, and typhoid fever. Exhibit 4-22 shows the incidence of each for 1997 through 2001.

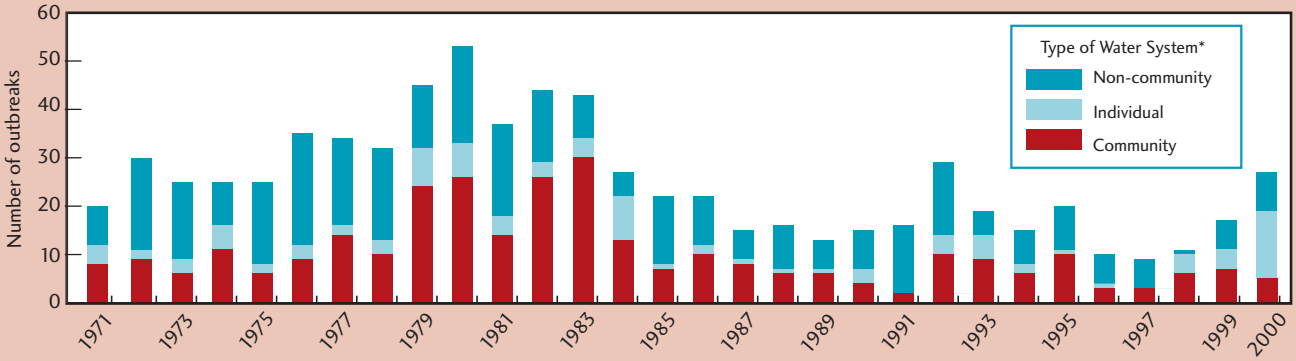
Waterborne Disease Outbreaks Associated with Drinking Water 1971-2000

Since 1971, the Centers for Disease Control and Prevention (CDC), EPA, and the Council of State and Territorial Epidemiologists have maintained a collaborative surveillance system for the occurrences and causes of waterborne-disease outbreaks (WBDO). These data are only a small part of the larger body of information related to drinking water quality in the United States. State, territorial, and local public health agencies are primarily responsible for detecting and investigating WBDOs and voluntarily reporting them to CDC. These data are used to identify types of water systems, their deficiencies, the etiologic agents (e.g., microorganisms and chemicals) associated with outbreaks, and to evaluate current technologies for providing safe drinking water and safe recreational waters. This system reports outbreaks and estimated numbers of people who become ill. It does not provide information on non-outbreak related or endemic levels of waterborne illness. Moreover, the focus is on acute illness. The system does not address chronic illnesses such as cancer, reproductive, or developmental effects. CDC and EPA are collaborating on a series of epidemiology studies to assess the magnitude of non-outbreak waterborne illness associated with consumption of municipal drinking water.

Between 1971 and 2000, there were 751 reported waterborne disease outbreaks associated with drinking water from individual, non-community systems, and community water systems (Exhibit 4-21). During 1999-2000, a total of 44 outbreaks (18 from private wells, 14 from non-community systems, and 12 from community systems) associated with drinking water were reported by 25 states (Craun and Calderon, 2003).

However, these data should be interpreted with caution. Many factors can influence whether a WBDO is recognized and investigated by local, territorial, and state public health agencies. For example, the size of the outbreak, severity of the disease caused by the outbreak, public awareness of the outbreak, whether people seek medical care or report to a local health authority, reporting requirements, routine laboratory testing for organisms, and resources for investigation can all influence the identification and investigation of a WBDO. This system underreports the true number of outbreaks because of the multiple steps required before an outbreak is identified and investigated. Thus, an increase in the number of outbreaks reported could either reflect an actual increase or improved surveillance and reporting at the local and state level.

Exhibit 4-21: Number of reported waterborne disease outbreaks associated with drinking water by year and type of water system, United States, 1971-2000 (n=751)



***Non-community water systems** are systems that either (1) regularly supply water to at least 25 of the same people at least 6 months per year, but not year round (e.g., schools, factories, office buildings, and hospitals that have their own water systems), or (2) provide water in a place where people do not remain for long periods of time (e.g., a gas station or campground).

Individual water systems are not regulated by the Safe Drinking Water Act and serve fewer than 25 persons or 15 service connections, including many private wells.

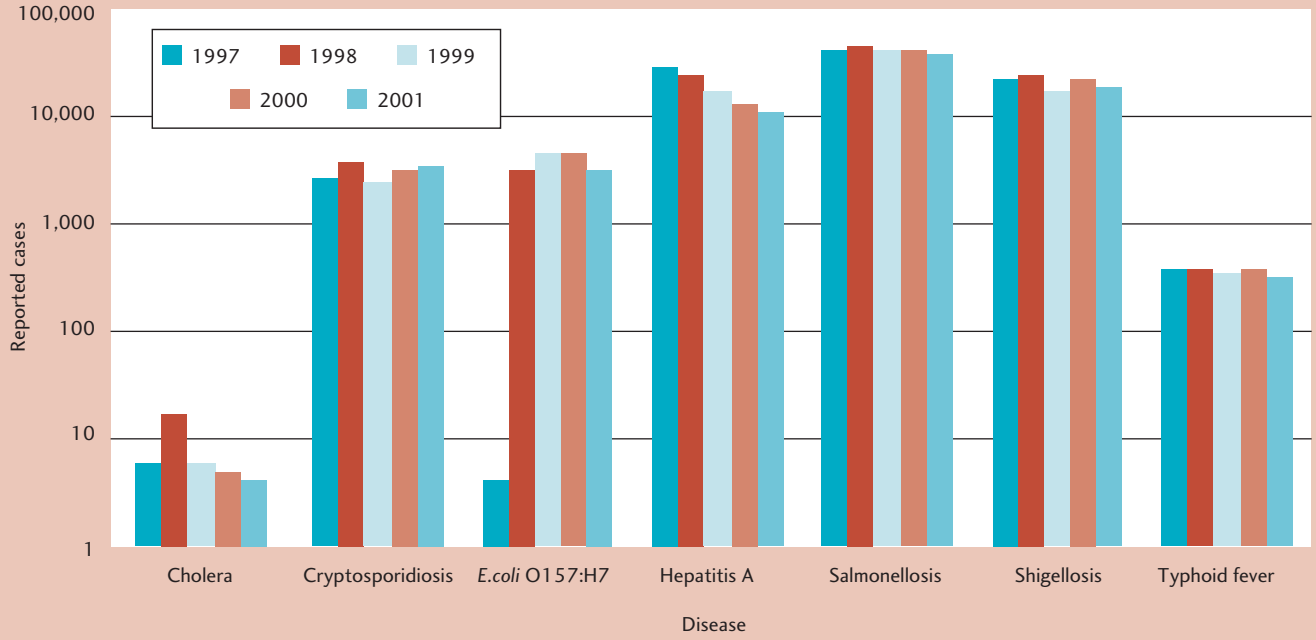
Community water systems provide water to at least 25 of the same people or service connections year round.

Source: Based on data presented in Craun, G.F. and R.L. Calderon. *Waterborne Outbreaks in the United States, 1971-2000.* 2003.

The data source for these seven indicators is the Centers for Disease Control and Prevention, Epidemiology Program Office, National Notifiable Diseases Surveillance System. This system provides weekly provisional information from the Council of State and Territorial Epidemiologists (CSTE) on the occurrence of diseases defined as notifiable. A notifiable disease is one that, when diagnosed, health providers report to state or local public health officials. Notifiable diseases are of public interest because of their contagiousness, severity, or frequency (Pastor, et al., 2002). State epidemiologists report cases of notifiable diseases to CDC, and CDC tabulates and publishes these data in *Morbidity and Mortality Weekly Report (MMWR)* and *Summary of Notifiable Diseases, United States*. Policies for reporting notifiable disease cases can vary by disease or reporting jurisdiction. CSTE and CDC annually review and recommend additions or deletions to the list of nationally notifiable diseases based on the need to respond to emerging priorities. *Reporting nationally notifiable diseases to CDC, however, is voluntary*. Reporting is currently mandated by law or regulation only at the local and state level. Therefore, the list of diseases that are considered notifiable varies slightly by state.

Notifiable disease data are useful for analyzing disease trends and determining relative disease burdens. These data, however, must be interpreted in light of reporting practices. The degree of completeness of data reporting is influenced by many factors such as the diagnostic facilities available; the control measures in effect; public awareness of a specific disease; and the interests, resources, and priorities of state and local officials responsible for disease control and public health surveillance. Finally, factors such as changes in case definitions for public health surveillance, introduction of new diagnostic tests, or discovery of new disease entities can cause changes in disease reporting that are independent of the true incidence of disease.

Exhibit 4-22: Prevalence of reported gastrointestinal diseases, United States, 1997-2001



Sources: CDC. *Notice to Readers: Final 2001 Reports of Notifiable Diseases*. 2002; CDC. *Notice to Readers: Final 2000 Reports of Notifiable Diseases*. 2001; CDC. *Notice to Readers: Final 1999 Reports of Notifiable Diseases*. 2000: CDC. *Notice to Readers: Final 1998 Reports of Notifiable Diseases*. 1999; CDC. *Notice to Readers: Final 1997 Reports of Notifiable Diseases*. 1998.

Indicator Infectious disease prevalence - Cholera - Category 2

Cholera is a diarrhea illness caused by infection of the intestine with the bacterium *Vibrio cholerae*. Infections can often be mild or without symptoms, but can sometimes be severe, and even fatal. Approximately 1 in 20 infected persons has severe disease characterized by severe, watery diarrhea that can lead to dehydration and shock. Without treatment, death can occur within hours (ICTDRN, 2002).

What the Data Show

Very few cases of cholera are reported on an annual basis in the U.S. It is believed most cases are associated with consumption of contaminated seafood or with international travel to areas where cholera is endemic (e.g., South America) (CDC, 2001a).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-27, for more information.)

Indicator Infectious disease prevalence - Cryptosporidiosis - Category 2

Cryptosporidiosis is an illness resulting from infection of the gastrointestinal tract with *Cryptosporidium parvum* and other species of *Cryptosporidium*. This pathogen is excreted by humans, as well as wild and domestic animals, including farm animals; it contaminates water sources via animal feces or domestic sewage. Runoff from agricultural operations into drinking water sources has been one cause of cryptosporidiosis outbreaks (Franzen and Muller, 1999).

Severe diarrhea is the most common symptom. Additional symptoms include gastric pain, fever, nausea, and fatigue (Guerrant, 1997). There is no antibiotic that is effective for treatment of cryptosporidiosis. As a result, a healthy immune system is important in limiting an individual's response to *Cryptosporidium parvum* infection. Cryptosporidiosis can be deadly when contracted by immunocompromised individuals. In extreme cases of cryptosporidiosis, infection can spread beyond the gastrointestinal tract to the gall bladder and biliary tract.

What the Data Show

The occurrence of symptoms or conditions associated with cryptosporidiosis are likely underreported. "We do not know exactly how many cases of cryptosporidiosis actually occur. Many people do not seek medical attention or are not tested for this parasite and so *Cryptosporidium* often goes undetected as the cause of intestinal illness" (CDC, 1998b).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-28, for more information.)

Indicator Infectious disease prevalence - *E. coli* O157:H7 - Category 2

E. coli O157:H7 is one of over 170 strains and many hundred sub-strains of the bacterium *Escherichia coli*. Most strains are harmless and live in the intestines of healthy humans and animals; this strain can cause severe illness. *E. coli* O157:H7 is not a disease itself, but rather a cause of illness. The identifier in the name of the bacterium refers to the specific antigenic markers found on its cell wall and distinguishes it from other types of *E. coli*. Infection often leads to bloody diarrhea and occasionally to kidney failure, particularly in young children (CDC, 2001b). A 1982 outbreak of severe bloody diarrhea was traced to contaminated hamburgers.

What the Data Show

CDC estimates that 73,000 cases of *E. coli* O157:H7 occur annually in the U.S., and that 61 fatal cases occur annually. The illness is often misdiagnosed; therefore, expensive and invasive diagnostic procedures may be performed. Patients who develop severe disease may require prolonged hospitalization, dialysis, and long-term follow-up (CDC, 2001b).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-28, for more information.)

Indicator Infectious disease prevalence - Hepatitis A - Category 2

Hepatitis A virus (HAV) is one of five viruses in the hepatitis group of viruses (A to E) that cause liver disease. Symptoms include jaundice, fatigue, abdominal pain, loss of appetite, nausea, diarrhea, and fever. Adults tend to be more symptomatic than children. HAV is found in the feces of infected people and is usually spread through contaminated food, water, or intimate contact (CDC, 2002d).

What the Data Show

The annual number of reported cases for HAV in the U.S. exceeds 10,000. The estimated number of new infections approaches 100,000 per year. It continues to occur in epidemics both nationwide and in communities. The number of cases is now reaching historic lows and continues to slowly decline, though about one-third of Americans show evidence of past infection (CDC, 2002e).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-28, for more information.)

Indicator Infectious disease prevalence - Salmonellosis - Category 2

Salmonellosis is a disease caused by one of the more than 2,000 strains of the bacterial genus *Salmonella*. Most persons infected with *Salmonella* develop diarrhea, fever, and abdominal cramps 12 to 72 hours after infection. The illness usually lasts 4 to 7 days, and most persons recover without treatment, though antibiotics can be used. In some persons, however, the diarrhea may be so severe that the patient needs to be hospitalized. In these patients, the *Salmonella* infection may spread from the intestines to the bloodstream and then to other body sites. It can cause death unless the person is treated promptly with antibiotics. The elderly, infants, and those with impaired immune systems are more likely to become severely ill from salmonellosis (CDC, 2001f).

What the Data Show

Every year, approximately 40,000 cases of salmonellosis are reported in the U.S. Because many milder cases are not diagnosed or reported, CDC estimates the actual number of infections to be 1.4 million. Salmonellosis is more common in the summer than winter. It is estimated that somewhat more than 500 persons die each year with acute salmonellosis (CDC, 2001f).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-29, for more information.)

Indicator Infectious disease prevalence - Shigellosis - Category 2

Shigellosis is a bacterial disease affecting the intestinal tract. Anyone can get shigellosis, though it is most common in children between the ages of 1 and 14. Most who are infected with *Shigella* develop diarrhea, fever, and stomach cramps starting a day or two after they are exposed to the bacterium. The diarrhea is often bloody. Shigellosis usually resolves in 5 to 7 days. In some persons, especially young children and the elderly, the diarrhea can be so severe that hospitalization is necessary. Some persons who are infected may have no symptoms at all, but may pass the *Shigella* bacteria to others (CDC, 2001g).

What the Data Show

Every year, about 14,000 cases of shigellosis are reported in the U.S. Because many milder cases are not diagnosed or reported, the CDC estimates the actual number of infections to be 448,000. Shigellosis is particularly common and causes recurrent problems in settings where hygiene is poor (CDC, 2001g).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-29, for more information.)

Indicator | Infectious disease prevalence - Typhoid fever - Category 2

Typhoid fever is a life-threatening illness caused by the bacterium *Salmonella typhi*. Typhoid fever is characterized by fever, headache, nausea, and loss of appetite. *Salmonella typhi* lives only in humans. Persons with typhoid fever carry the bacteria in their bloodstream and intestinal tract. In addition, a small number of persons (2 to 5 percent), called carriers, recover from typhoid fever but continue to carry and shed the bacteria. Both ill persons and carriers shed *S. typhi* in their feces and urine (WHO, 1997).

What the Data Show

In the U.S., about 400 *S. typhi* cases occur each year, many of which are acquired while traveling internationally. Typhoid fever is transmitted by eating food or drinking beverages that have been handled by a person who is shedding *S. typhi*, or by consuming water contaminated with *S. typhi* bacteria (CDC, 2001h).

Data Source

National Notifiable Diseases Surveillance System, Centers for Disease Control and Prevention. (See Appendix B, page B-30, for more information.)

4.3.4 What are the trends for children's environmental health issues?

Special consideration must be given to children's health issues because children may be more susceptible to disease and generally may be more vulnerable to their surroundings for many physiological reasons. This section discusses five indicators for children's environmental health issues: infant mortality, low birthweight, childhood cancer, childhood asthma, and birth defects.

Indicator Infant mortality - Category I

Infant mortality in the U.S. is defined as the death of a child before age 1.

What the Data Show

In 1999, a total of 27,937 deaths occurred in infants under 1 year of age (Hoyert, et al., 2001). The infant mortality rate was 7.1 per 1,000 live births, the lowest ever recorded in the U.S. The infant mortality rate for African American infants was 14.6 per 1,000 live births, more than twice the rate for White infants (5.8 per 1,000 live births). The infant mortality rate for Hispanic infants was 5.8 per 1,000 live births. The 10 leading causes of infant deaths account for 67.6 percent of all infant deaths in the U.S. (Exhibit 4-23). Delaware, Maine, Massachusetts, and Utah have the lowest infant mortality rates. Mississippi, Alabama, and Louisiana have the highest (Hoyert, et al., 2001).

Data Source

National Vital Statistics System, Centers for Disease Control and Prevention. (See Appendix B, page B-30, for more information.)

Exhibit 4-23: Number of infant deaths, percent of total deaths, and infant mortality rates for the 10 leading causes of infant death, United States, 1999

Rank	Cause of Death	Deaths	Rate	Percent of Total Deaths
	All causes	27,937	705.6	100.0
1	Congenital malformations, deformations, and chromosomal abnormalities	5,437	138.2	19.6
2	Disorders related to short gestation and low birthweight	4,392	110.9	15.7
3	Sudden Infant Death Syndrome	2,648	66.9	9.5
4	Newborn affected by maternal complications of pregnancy	1,399	35.3	5.0
5	Respiratory distress of newborn	1,110	28.0	4.0
6	Newborn affected by complications of placenta, cord, and membranes	1,025	25.9	3.7
7	Accidents	845	21.3	3.0
8	Bacterial sepsis of newborn	691	17.5	2.5
9	Diseases of the circulatory system	667	16.8	2.4
10	Atelectasis	647	16.3	2.3
	All other causes	9,040	228.3	32.4

Rate is per 100,000 live births in 1999.

Source: Hoyert, D.L., et al. *Deaths: Final Data for 1999*. 2001.

Indicator Low birthweight - Category I

An infant with low birthweight is defined as a full-term infant, born between week 37 and 44 of pregnancy, and weighing 2,500 grams or less at birth. Weight is a critical health measure because low birthweight children are more prone to death and disability than their counterparts.

What the Data Show

The percentage of infants who were born with a low birthweight (weighing less than 2,500 grams) was 7.6 percent in 2000 (Martin, et al., 2002). In 2000, the low birthweight rate for non-Hispanic African Americans (13.1 percent) was twice the rate of that for non-Hispanic Whites (6.6 percent), a relationship that existed for at least the 9 prior years as well (Exhibit 4-24). In 2000, the low birthweight rate for Hispanics was similar to that of non-Hispanic Whites (6.4 and 6.6, respectively). Also shown in Exhibit 4-24 is that non-Hispanic African Americans had the highest proportion of very low birthweight infants (weighing less than 1,500 grams) in 2000, compared with Hispanic and non-Hispanic White populations in the U.S.

Data Source

National Vital Statistics System, Centers for Disease Control and Prevention. (See Appendix B, page B-30, for more information.)

Exhibit 4-24: Percent of live births of very low birthweight and low birthweight, by race and Hispanic origin of mother, United States, 1991-2000

	Very Low Birthweight[1]			Low Birthweight[2]		
	White Non-Hispanic	Black Non-Hispanic	Hispanic[3]	White Non-Hispanic	Black Non-Hispanic	Hispanic[3]
2000	1.14	3.10	1.14	6.6	13.1	6.4
1999	1.15	3.18	1.14	6.6	13.2	6.4
1998	1.15	3.11	1.15	6.6	13.2	6.4
1997	1.12	3.05	1.13	6.5	13.1	6.4
1996	1.08	3.02	1.12	6.4	13.1	6.3
1995	1.04	2.98	1.11	6.2	13.2	6.3
1994	1.01	2.99	1.08	6.1	13.3	6.2
1993	1.00	2.99	1.06	5.9	13.4	6.2
1992	0.94	2.97	1.04	5.7	13.4	6.1
1991	0.94	2.97	1.02	5.7	13.6	6.1

[1] Less than 1,500 grams (3 lb. 4 oz.)

[2] Less than 2,500 grams (5 lb. 8 oz.)

[3] Includes all persons of Hispanic origin of any race.

Source: Martin, J.A., et al. *Births: Final Data for 2000.* 2002.

Indicators

Childhood cancer mortality - Category 1
Childhood cancer incidence - Category 2

Cancer is a disease characterized by uncontrolled growth of cells. A cancerous cell loses its ability to regulate its own growth, control cell division, and communicate with other cells. These cellular changes are complex and occur over a period of time. They may be accelerated in children. Cancer cells can invade nearby tissues and can spread through the bloodstream and lymphatic system to other parts of the body (NCI, 2003). The classification of cancers in children differs from the classification used for adult cancers.

What the Data Show

In 1999, there were nearly 2,200 deaths due to cancer in children and adolescents under 20 years of age (Anderson, 2001). The age-adjusted cancer mortality rates by race and age group are presented in Exhibit 4-25. In 1999, cancer was the third leading cause of death in children 1 to 4 years of age, accounting for 8 percent of the total deaths in this age group (Anderson, 2001). The death rate for cancer in this age group was 2.8 per 100,000 population. For children 5 to 9 years of age, cancer was the second leading cause of death accounting for 14.7 percent of total deaths. The death rate was 2.6 per 100,000 for children 5 to 9 years of age. In older children (15 to 19 years of age), 5.4 percent of total deaths in this age group were due to cancer. Cancer ranked fourth among leading causes of death, with a mortality rate of 3.8 per 100,000 population.

Exhibit 4-26 presents the age-adjusted incidence rates for cancers in children of all races between the ages of 0 and 19 years, 1975 to 1998. There has been an increase in the incidence for all types of childhood cancer since 1975. There also has been a substantial decline in the cancer death rate for children, largely due to improved treatment (EPA, December 2000).

Data Sources

Mortality: National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-31, for more information.)

Incidence: Surveillance, Epidemiology, and End Results Program, National Cancer Institute. (See Appendix B, page B-31, for more information.)

Exhibit 4-25: Age-adjusted Surveillance, Epidemiology and End Results (SEER) childhood cancer (all sites) incidence and United States mortality rates by race and age group, 1994-1998

	Ages 0-14						Ages 0-19					
	Incidence			Mortality			Incidence			Mortality		
Race	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female
All Races	14.4	15.4	13.4	2.7	3.0	2.4	15.9	16.7	15.0	3.0	3.3	2.6
White	14.8	15.6	13.9	2.7	3.0	2.4	16.4	17.2	15.6	3.0	3.4	2.6
Black	12.0	13.0	10.9	2.8	2.9	2.6	12.5	13.3	11.7	3.1	3.2	2.9

Rates are deaths per 100,000 per year and are age adjusted to the 1970 U.S. standard population.

Source: Ries L.A.G., et al. *SEER Cancer Statistics Review, 1973-1988*. 2001

Indicators

Childhood cancer mortality - Category 1 (continued)
Childhood cancer incidence - Category 2 (continued)

Exhibit 4-26: Age-adjusted Surveillance, Epidemiology and End Results (SEER) cancer incidence rates by international classification of childhood cancer (ICCC) selected group and subgroup and year of diagnosis, children 0 to 19 years, 1975-98

	1975-1980	1981-1986	1987-1992	1993-1998
All groups combined	140.0	149.0	157.5	159.1
Leukemia	33.2	36.3	37.6	37.4
Lymphomas and reticuloendothelial neoplasms	24.1	24.9	24.8	23.9
Central nervous system	23.4	24.3	29.6	27.8
Sympathetic nervous system tumors	7.7	8.1	7.6	8.5
Retinoblastoma	2.6	2.7	2.9	3.1
Renal tumors	6.0	6.6	6.3	7.1
Hepatic tumors	1.2	1.5	1.7	1.8
Malignant bone tumors	7.8	9.2	8.9	9.4
Soft tissue sarcomas	10.4	10.9	11.2	11.4
Germ cell, trophoblastic and other gonadal neoplasms	8.6	9.8	11.3	11.7
Carcinomas and other malignant epithelial neoplasms	13.9	13.5	14.6	15.0

Notes: Rates are cases per 1,000,000 per year and are age adjusted to the 1970 U.S. standard population.

Source: Ries, L.A.G., et al. *SEER Cancer Statistics Review, 1973-1998*. 2001.

Indicators

Childhood asthma mortality - Category I
Childhood asthma prevalence - Category I

Asthma is a chronic respiratory disease characterized by inflammation of the airways and lungs. During an asthma attack, the airways that carry air to the lungs are constricted. As a result, less air is able to flow in and out of the lungs (NCHS, 2001). Currently, there are no preventive measures or cure for asthma; however, children and adolescents who have asthma can still lead quality, productive lives if they control their asthma. Asthma can be controlled by taking medication and by avoiding contact with environmental "triggers" for asthma. Environmental triggers include cockroaches, dust mites, furry pets, mold, tobacco smoke, and certain chemicals (CDC, 2002g; CDC, 2003b).

What the Data Show

In 2001, approximately 6 million (9 percent) of U.S. children had asthma, compared to approximately 3.6 percent of children in 1980 (EPA, 2003a).

In 1999, there were 32 deaths due to asthma for children under 5 years of age and 144 deaths for children 5 to 14 years of age (Mannino, et al., 2002). This number is slightly lower than the 189 asthma deaths among children under 15 years of age in 1998.

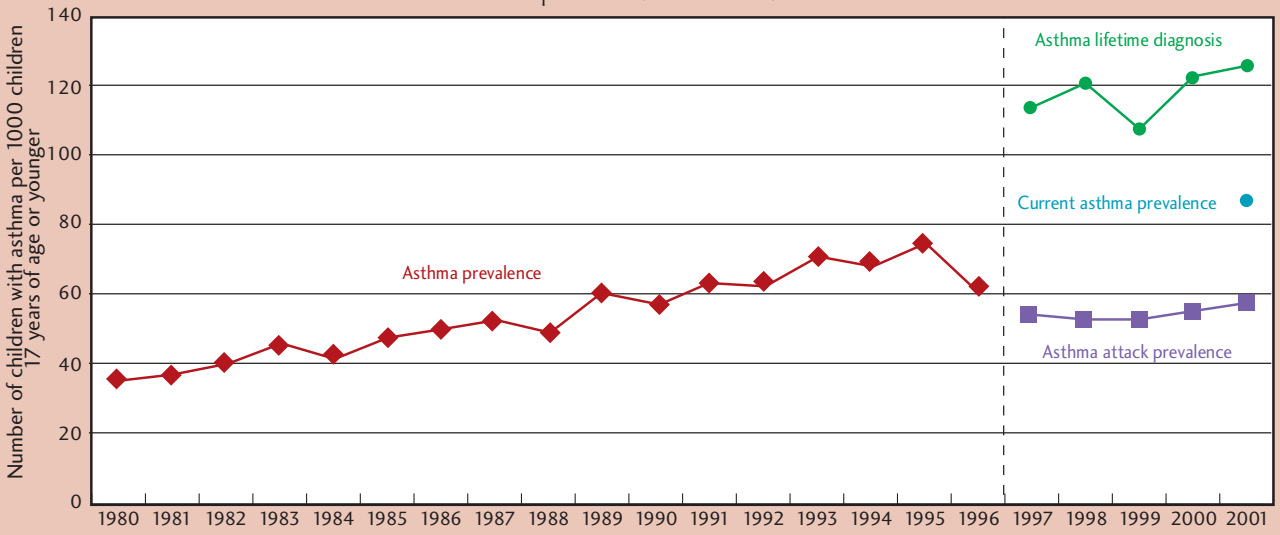
Boys were more likely to have been diagnosed with asthma than girls; the condition was diagnosed in 13 percent of boys compared with 10 percent for girls. Of the 4 million children who reported that they had an asthma attack in the last 12 months, boys were most likely to have had an attack when they were 5 to 11 years of age. Girls were most likely to have had an attack in the previous year at 12 to 17 years of age. Fourteen percent of non-Hispanic African American children had been diagnosed with asthma. The proportion of non-Hispanic White and Hispanic children who had ever been diagnosed with asthma was nearly the same, 11 percent and 10 percent, respectively. Asthma rates in children have increased since 1980, especially for children age 4 and younger and for African-American children (Exhibit 4-27).

Data Sources

Mortality: National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-31, for more information.)

Prevalence: National Health Interview Survey, Centers for Disease Control and Prevention (See Appendix B, page B-32, for more information.)

Exhibit 4-27: Asthma prevalence, 1980-1996, asthma lifetime diagnosis, current asthma, and asthma attack prevalence, 1997-2001, in children



Note: The survey questions for asthma changed in 1997; data before 1997 cannot be directly compared to data in 1997 and later.

Source: Based on and updated from Akinbami, L.J. and K.C. Schoendorf. *Trends in Childhood Asthma: Prevalence, Health Care Utilization and Mortality.* 2002. Data from CDC, National Center for Health Statistics, National Health Interview Surveys, 1980-2001.

Indicators

Deaths due to birth defects - Category I
Birth defect incidence - Category I

Congenital anomalies, or birth defects, are structural defects that are present in the fetus at birth. Because the causes of about 70 percent of all birth defects are unknown, the public continues to be anxious about whether environmental pollutants cause birth defects, developmental disabilities, or other adverse reproductive outcomes. The public also has many questions about whether various occupational hazards, dietary factors, medications, and personal behaviors cause or contribute to birth defects (CDC, 2002c).

What the Data Show

Birth defects (congenital anomalies) are a leading cause of infant deaths, accounting for 5,473 (19.6 percent) of the 27,937 infant deaths in 1999 (Hoyert, et al., 2001). The most frequently occurring types of birth defects were those affecting the heart and the lungs. Because some birth defects are not recognized immediately, they are underreported on the death certificate, so the numbers underestimate the problem (Friis, et al., 1999). Exhibit 4-28 presents the number and rate of live births with congenital anomalies.

Data Source

National Vital Statistics System, National Center for Health Statistics. (See Appendix B, page B-32, for more information.)

Exhibit 4-28: Number and rate of live births with selected congenital anomalies, United States, 2000

Congenital Anomaly (All races)	Number of Congenital Anomalies Reported	Rate
Anencephalus	425	10.7
Spina bifida/Meningocele	822	20.7
Hydrocephalus	940	23.7
Microcephalus	284	7.2
Other central nervous system anomalies	822	20.7
Heart malformations	4,958	124.9
Other circulatory/respiratory anomalies	5,484	138.1
Rectal atresia/stenosis	333	8.4
Tracheo-esophageal fistula/Esophageal atresia	481	12.1
Omphalocele/Gastroschisis	1,180	29.7
Other gastrointestinal anomalies	1,185	29.9
Malformed genitalia	3,344	84.2
Renal agenesis	547	13.8
Other urogenital anomalies	3,943	99.3
Cleft lip/palate	3,259	82.1
Polydactyly/Syndactyly/Adactyly	3,460	87.2
Clubfoot	2,271	57.2
Diaphragmatic hernia	427	10.8
Other musculoskeletal/integumental anomalies	8,614	217.0
Down's syndrome	1,863	46.9
Other chromosomal anomalies	1,575	39.7

Rates are number of live births with specified congenital anomaly per 100,000 live births in specified group.

Note: Of the 4,031,591 live births, there was no response recorded for the congenital anomaly item for 61,744 births.

Source: Martin, J.A., et al. *Births: Final Data for 2000.* 2002.

4.3.5 What are the trends for emerging health effects?

In addition to the diseases reported in the preceding pages, several other diseases are the cause of emerging concern because of their potential impacts on the health of the U.S. population. Information for eight such diseases—diabetes, Alzheimer's disease, Parkinson's disease, renal disease, autism, and three arthropod-borne diseases (Lyme disease, Rocky Mountain spotted fever, and West Nile virus)— is presented in this section. The increasing prevalence of these "emerging" illnesses positions them as potential future candidates for consideration as EPHIs. This will be dependent on their increasing prevalence in the population or a better determination of the role of exposure to environmental pollutants in the onset or exacerbation of these diseases. No specific indicators have been presented for these diseases at this time, but data collected by the CDC, individual states, and other sources illustrate the recent trends in these diseases.

Diabetes

Diabetes is a set of metabolic disorders. Diabetes mellitus (type 2) is the most common form of diabetes and is a disease whereby the body's insulin activity is altered. Insulin is a hormone that signals many biological processes such as the conversion of glucose to glycogen. Glycogen is the form in which food energy is stored in the body. The general symptoms of diabetes are elevated blood glucose levels, excessive thirst, frequent urination, and unexplained weight loss. Heredity, obesity, and age are factors that also contribute to diabetes. Estimates of the prevalence of diabetes vary widely. However, CDC estimates that there are about 11.1 million diagnosed cases of diabetes (CDC, 2002b). In addition to these cases, CDC estimates that there may be about 5.9 million more cases that are undiagnosed (CDC, 2002b). The total of 17 million diagnosed and undiagnosed cases combined amounts to a prevalence of 6.2 percent of the U.S. population. CDC estimates that 1 million new cases of diabetes are diagnosed per year among people aged 20 years and older (CDC, 2002b).

In 1999, diabetes ranked as the sixth leading cause of death in the U.S. There were 68,399 deaths due to diabetes (Hoyert, et al., 2001). The age-adjusted death rates for diabetes increased between 1980 and 1996 from 15.3 to 20.6 per 100,000 people. By 1999, the rate had risen to 25.2 per 100,000 people.

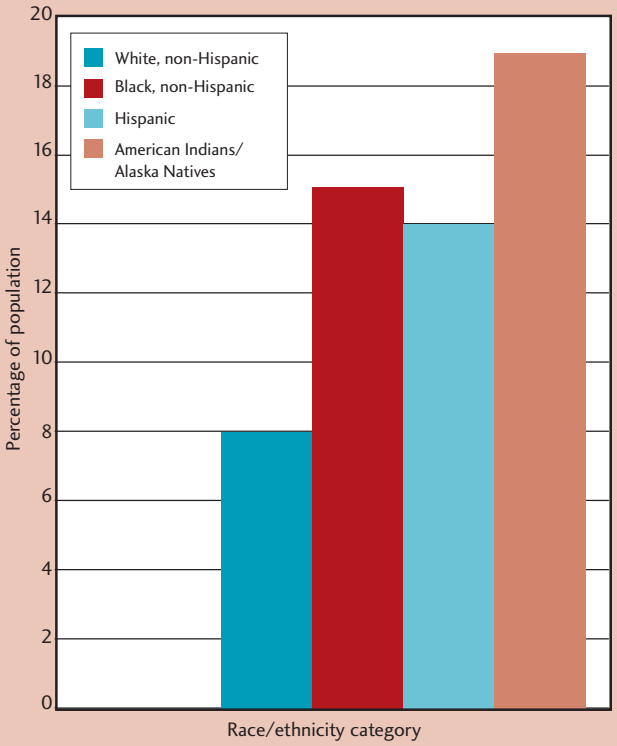
On average, Hispanic Americans are 1.9 times more likely to have diabetes than non-Hispanic Whites of similar age. The risk of diabetes for Mexican Americans and non-Hispanic Blacks is almost twice that for non-Hispanic Whites. Similarly, residents of Puerto Rico are 2.0 times more likely to have diagnosed diabetes than U.S. non-Hispanic Whites. On average, American Indians and Alaska Natives are 2.6 times more likely to have diabetes than non-Hispanic Whites of similar age. Approximately 15 percent of American Indians and Alaska Natives receiving care from the Indian Health Service have diabetes. At the regional level, diabetes is least common among Alaska Natives (5.3 percent) and most common among American Indians in the southeastern U.S. (25.7 percent) and in certain tribes from the Southwest (CDC, 2002b). Exhibit 4-29 shows age-adjusted prevalence data for diabetes in the U.S. by race/ethnicity.

Alzheimer's Disease

Alzheimer's disease is a neurodegenerative disorder. The symptoms of Alzheimer's disease may include dementia, loss of memory, and decreasing physical abilities such as dressing or eating. In the U.S., an estimated 4 million people, mostly elderly, have Alzheimer's disease (Hoyert and Rosenberg, 1999). In 1999, an estimated 354,000 non-institutionalized adults 18 to 64 years of age reported Alzheimer's disease as their main disability (CDC, 2001e).

The death rate due to Alzheimer's disease rose steadily from 1979 to 1996. In 1999, Alzheimer's disease was the eighth leading cause of

Exhibit 4-29: Age-adjusted prevalence of physician-diagnosed diabetes in persons 20 years of age and older, by race/ethnicity, United States, 2000



Source: CDC. *National Diabetes Fact Sheet.* 2002. Data from CDC, National Center of Health Statistics, National Health Interview Survey, 1997-1999 and National Health and Nutrition Examination Survey, 1988-1994.

death in the U.S. (Hoyert, et al., 2001). There were 44,536 deaths attributed to Alzheimer's disease (16.3 deaths per 100,000 population). The death rate for Alzheimer's disease rises sharply with age. In 1999, among people 75 to 84 years of age, there were 15,836 deaths and in this age group Alzheimer's disease ranked as the seventh leading cause of death (Anderson, 2001). The death rate for Alzheimer's disease for this age group was 130.4 per 100,000 population. Among persons 85 years of age and older, there were 24,980 deaths due to Alzheimer's disease for a death rate of 598.3 per 100,000 population.

Death rates for Alzheimer's disease are higher for women than for men and higher for Whites than African Americans (Hoyert, et al., 2001). The 1999 death rates for Alzheimer's disease are highest for White females (25.6 per 100,000), followed by White males (11.4), African American females (9.0), and African American males (4.2). The Alzheimer's disease death rate for Hispanics is 3.1 per 100,000. Hispanic females have a higher death rate (4.3 per 100,000 population) than Hispanic males (2.0 per 100,000). The death rates from Alzheimer's disease are higher in the Northeast and in the Northwest regions of the U.S. (Hoyert and Rosenberg, 1999).

Parkinson's Disease

Parkinson's disease is a neurodegenerative disorder characterized by symptoms such as tremors, muscle rigidity, and changes in walking patterns. The National Institute of Neurological Diseases and Stroke (NINDS) estimates that there are about 500,000 people in the U.S. with Parkinson's disease (NINDS, 2002). The disease mostly affects elderly people and is second only to Alzheimer's disease in the number of older people that are affected (Checkoway and Nelson, 1999). It affects about 0.4 percent of those 40 years of age and older, 1 percent of those older than 65 years, and about 3 percent of those 80 years of age and older. Males are 1.3 times more likely than females to have Parkinson's disease.

A steady increase in the death rate due to Parkinson's disease among people 75 years of age and older has been observed in the U.S. In 1999, there were 14,593 deaths due to Parkinson's disease (Hoyert, et al., 2001). Virtually all of the deaths (14,298) occurred in people 65 years of age and older. The death rate was 5.4 per 100,000 population, with males having a higher death rate than females (6.2 versus 4.5 per 100,000).

The 1999 death rate due to Parkinson's disease was higher for Whites (6.2 per 100,000 people) than for African Americans (1.5 per 100,000) (Hoyert, et al., 2001). The death rate for White males was 7.1 per 100,000 and for White females 5.3 per 100,000. The death rate for African American males was 1.6 and for African American females 1.3 per 100,000. The death rate for Hispanics was 1.2 per 100,000, with Hispanic males having a slightly higher death rate (1.4 per 100,000) than Hispanic females (1.1 per 100,000).

Renal Disease

The kidneys are vital organs and can be seriously affected by a number of primary diseases such as diabetes or hypertension. As these diseases progress, the kidneys may fail to function. Total and permanent kidney failure is called end stage renal (kidney) disease (ESRD). It is estimated that about 424,179 people in the U.S. have ESRD (NIDDK, 2001). Most ESRD occurred in people who have diabetes (150,404 people), hypertension (100,169 people), or glomerulonephritis, a kidney disease (62,119 people).

The U.S. government maintains the U.S. Renal Data System, which provides information on the incidence, prevalence, and mortality for ESRD (CDC, 2000a). Data from this system indicate that there were 89,252 people with ESRD who began treatment in 1999. These cases of ESRD resulted from diabetes for 38,160 people and from hypertension for 23,133 people. Kidney diseases and other primary diseases were responsible for the remainder.

Between 1979 and 1998, the age-adjusted death rates for all types of kidney disease increased, peaking between 1984 and 1988. The age-adjusted death rates for all types of kidney disease are higher among African Americans than among Whites, with African American males having the highest rates during the 1979 to 1998 period.

In 1979, the death rate for total kidney disease was 8.6 per 100,000 people. By 1999, kidney disease had risen to rank as the ninth leading cause of death in the U.S. (Hoyert, et al., 2001). That year there were 35,525 deaths due to all types of kidney disease; 34,719 of them were due to kidney failure. The death rate for kidney disease was 13.0 per 100,000 people; the death rate for kidney failure was 12.7 per 100,000 people (Exhibit 4-30). Death rates for kidney failure were highest for African American females at 19.0 per 100,000, followed by African American males at 17.8 per 100,000.

African Americans and American Indians have higher rates of ESRD than Whites or Asians (AHA, 2001). African Americans represent 32 percent of the patients receiving treatment for ESRD. Recently there has been an increase in ESRD due to diabetes among American Indians and Alaskan Natives (CDC, 2000c). Between 1990 and 1996, the age-adjusted rate of new ESRD treatment among American Indians with diabetes increased 24 percent, from 472 to 584 per 100,000 persons with diabetes.

Autism

Autism is one of several related severe cognitive and neurobehavioral disorders that are classified under the term autistic spectrum disorders. Information about the prevalence of autism in the U.S. is limited, reflecting the use of different diagnostic criteria and a lack of research. First described in the 1940s, autism was thought to affect 2 to 4 children per 10,000 population. Today the prevalence is currently believed to be as high as 1 in 500 children` for all autistic

Exhibit 4-30: Death rates for kidney disease, United States, 1999

	All Races			White			All Other			Black		
Cause of Death	Both Sexes	Male	Female	Both Sexes	Male	Female	Both Sexes	Male	Female	Both Sexes	Male	Female
Nephritis, nephrotic syndrome, nephrosis	13.0	12.8	13.3	12.5	12.4	12.6	15.6	14.8	16.3	19.3	18.2	20.2
Kidney failure	12.7	12.5	13.0	12.2	12.1	12.3	15.2	14.4	16.0	18.9	17.8	19.0
Other	0.3	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.3*	0.4	0.4*	0.4*

Rates are per 100,000 population.

*Figure does not meet the standards of reliability or precision.

Source: Hoyert, et al. *Deaths: Final Data for 1999*. 2001.

spectrum disorders (Iversen, 2000). Currently, autism affects about 400,000 people in the U.S., and occurs about four times more often in boys than in girls.

Researchers have reported that the number of persons with autism is increasing. For example, a recent California Department of Developmental Services (CDDS) report showed an over 200 percent increase in the number of persons entering the regional center service system with autism between 1987 and 1998 (CDDS, 1999). Other states have reported increasing numbers as well (Yazbak, 1999). However, these reports do not necessarily reflect a change in the rate of autism because they do not consider the increase in the total population (Fombonne, 2001).

The number of cases of autism in children in the U.S. has increased over time. The number of children 0 to 21 years old with autism who are also enrolled in federally supported programs for the disabled has grown from 5,000 in 1991 to 79,000 in 2000 (NCES, 2001). This represents an increase from 0.1 to 1.1 percent of all children with disabilities served, or an increase from 0.01 to 0.14 percent of all children in public schools.

Arthropod-Borne Diseases

Certain ticks and mosquitoes (arthropods) can carry bacteria and viruses that cause disease in humans. They acquire the bacteria and viruses when they bite an infected mammal or bird. Arthropod-borne diseases include Lyme disease, Rocky Mountain spotted fever (RMSF), and West Nile virus (WNV).

Lyme Disease

Lyme disease is the most commonly reported arthropod-borne disease in the U.S. (Orloski, et al., 2002). The illness was first described in Europe during the 1800s; however, it was not identified in the U.S. until the early 1970s when a cluster of children with "juvenile rheumatoid arthritis" in Lyme, Connecticut, was reported by their parents (Shapiro and Gerber, 2000). Investigation of the cluster led to the description of Lyme arthritis in 1976 and then to the identification of the causal pathogen. Between 1992 and 1998, there were 88,967 cases of Lyme disease reported to the CDC. The number of cases increased from 9,896 in 1992 to 16,802 in 1998 (Exhibit 4-31).

The incidence of Lyme disease was highest in eight northeastern and mid-Atlantic states and two north central states. These states accounted for 92 percent of the total cases.

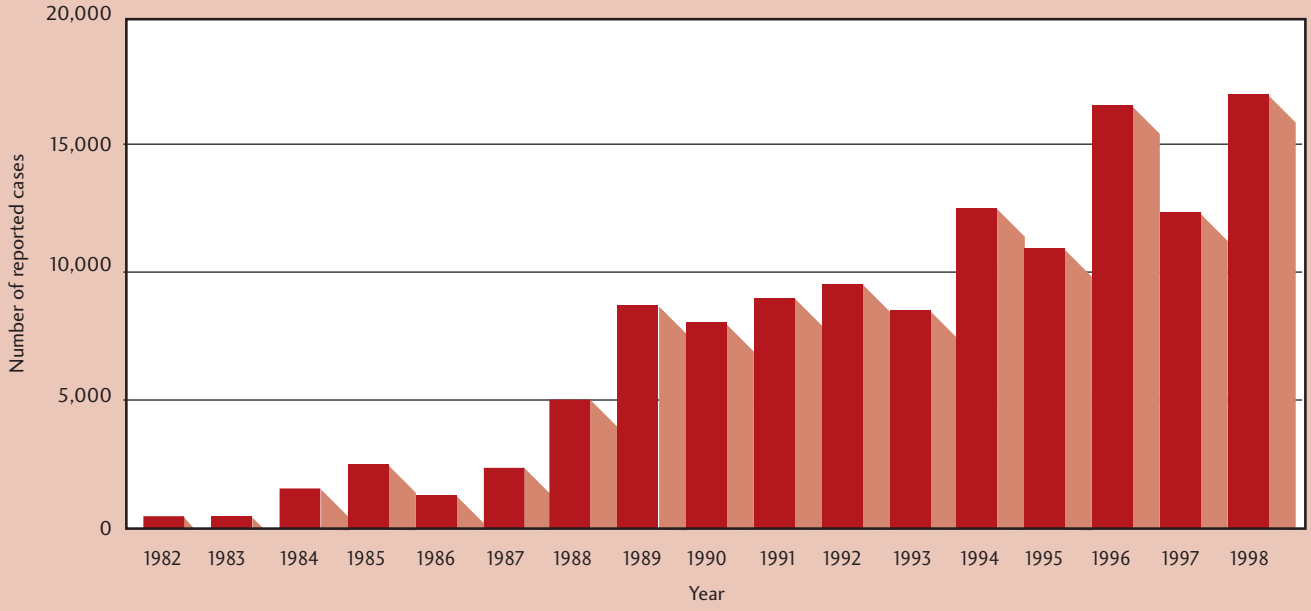
Rocky Mountain Spotted Fever

Although Lyme disease is the most commonly reported tick-borne disease in the U.S., RMSF is the most commonly *fatal* tick-borne disease in the U.S. (Holman, et al., 2001). Physicians first recognized RMSF in the northwestern U.S. during the late 1800s; Howard Ricketts identified the causal pathogen in the early 1900s (Gayle and Ringdahl, 2001; Paddock, et al., 1999). RMSF was the first disease in the U.S. shown to be transmitted by tick bite (Walker, 1998). Although RMSF was first identified in the Rocky Mountain states, fewer than 3 percent of cases were reported from that area between 1993 and 1996. The highest incidence of cases in that time period was found in North Carolina and Oklahoma. These two states accounted for 35 percent of the total cases from 1993 to 1996 (CDC, 2002c). RMSF has been reported throughout the continental U.S. (except in Maine, New Hampshire, and Vermont).

Between 1990 and 1998, there were approximately 4,800 cases of RMSF reported to the CDC (CDC, 2000b). The annual number of cases has varied between 250 and 1,200 cases since 1942, with a peak between 1975 and 1981.

The ratio of the number of deaths due to RMSF compared to the number of cases of the disease is the highest in children under 10

Exhibit 4-31: Number of reported cases of Lyme disease, United States, 1982-1998



Source: Orloski K.A., et al. *Surveillance for Lyme Disease*. 2002.

years of age (2 to 3 percent) and those over 70 years of age (9 percent) (CDC, 2000b).

West Nile Virus

In 1937, WNV, a strain of encephalitis, was first identified as a human pathogen in the West Nile region of Uganda. The pathogen was found in blood taken from a woman during a yellow fever investigation (Rappole, et al., 2000). Since 1937, WNV has been determined to be widespread in many areas of the world, particularly Africa, the Middle East, Europe, Russia, India, and Indonesia (Horga and Fine, 2001).

Cases of WNV were first documented in the U.S. in 1999 (CDC, 2000d). A total of 80 cases in humans were reported in 1999 (62 cases) and 2000 (18 cases). Because severe neurological illness (encephalitis meningitis) occurs in fewer than 1 percent of persons infected, it is thought that a greater number of cases with less severe symptoms may go unreported. Based on this assumption, it is estimated that approximately 2,000 persons may have been infected with WNV during 2000 (CDC, 2000d). The prevalence of the disease in humans is increasing. During 2002 there were 3,989 diagnosed cases in humans (CDC, 2002f). The number of deaths caused by West Nile encephalitis has increased from 7 in 1999 to 259 in 2002 (CDC, 2002f).

4.4 Measuring Exposure to Environmental Pollution: Indicators and Trends

Historically, human exposure to pollutants has been estimated based on:

- Measurements of ambient pollutant concentrations in air, water, or land, combined with:
- Estimates or measurements (through personal monitoring) of the frequency and duration of human contact with the contaminated media.

This approach has provided a valuable foundation for many of the regulatory and non-regulatory actions that have been taken to limit exposure to ambient pollutants. However, ambient measurements do not provide information on the degree to which ambient pollutants actually enter into the body. Another type of indicator—biomonitoring data—can help provide this information. Biomonitoring measures the amount of a pollutant in human tissue or fluids. It provides an important complement to more traditional exposure assessment indicators. National-scale biomonitoring data can be used to:

- Measure and track average body burden resulting from exposure across the entire population to a variety of pollutants.
- Enhance environmental disease prevention efforts by providing an important bridge to understanding the relationships between ambient pollutant concentrations, exposures to these pollutants, and health problems. (The lead case study, discussed earlier in Section 4.1, provides an excellent example of this application.)
- Establish reference ranges to identify people with unusually high exposures or the percentage of the population with pollutant exposures above established levels of concern (CDC, 2003a).

This section focuses primarily on biomonitoring indicators and is divided into ten parts:

- Section 4.4.1 provides background information on biomonitoring indicators—what they are and their limitations.
- Section 4.4.2 describes the major data sources for these indicators.
- Sections 4.4.3 to 4.4.8 describe specific pollutants and the data available to monitor these pollutants, including heavy metals (Section 4.4.3), cotinine (Section 4.4.4), volatile organic compounds (Section 4.4.5), pesticides (Section 4.4.6), and persistent organic pollutants (Section 4.4.7). Section 4.4.8 presents indicators that are available to specifically monitor children's exposure to some of these pollutants. In all, 10 biomonitoring indicators are currently available for tracking trends in human exposure to specific environmental pollutants. Summaries of the data linking exposure to human health effects can be found in ATSDR's toxicological profiles and EPA's criteria documents for these chemicals.
- Section 4.4.9 briefly discusses a number of pollutants—radiation, air pollutants (except for lead), biological pollutants, and disinfection by-products —for which no biomonitoring indicators currently are available or feasible. For these pollutants, traditional exposure assessment will continue to serve as the method for estimating human exposure until biomonitoring indicators become available or feasible.
- Finally, Section 4.4.10 touches on endocrine disruptors—considered an emerging issue.

4.4.1 Biomonitoring Indicators

"Dose" (the amount of a pollutant that enters the body) is often expressed as average daily dose or total potential dose. Once a pollutant crosses the boundary into the body, biological processes act on that contaminant to utilize, remove, or store the contaminant and/or its metabolites. Body burden is the concentration of a contaminant dose that is retained in the human body. Body burden can be estimated from measurements of the contaminant in the blood, urine, or adipose tissue. These measurements provide the basis for biomonitoring indicators.

The buildup of a contaminant in the body (i.e., the level of body burden) depends on a variety of factors, including the nature of the contaminant; the efficacy of the biological removal processes; and the magnitude, timing, frequency, and duration of exposure. Some contaminants, such as lead, are not easily removed and are retained in the body for long periods of time. Other contaminants, such as many volatile organic compounds (VOCs), are rapidly eliminated in exhaled breath or other removal processes.

The level of body burden is usually estimated from the concentration of a contaminant (or its metabolite) measured in the blood, urine, hair, or adipose tissue, and can be used to infer that an exposure occurred. In some cases, the level of body burden associated with a particular contaminant may prove to be an indicator of the person's extent of exposure to that pollutant.

There are a number of potential problems, however, with using body burden as an indicator of exposure. In some cases, several different pollutants may give rise to the same biomarker. Further, most measures of body burden reflect only a "snapshot" in time and many different exposure scenarios can lead to the same concentration measurement. Lastly, the measure gives no information about how the person was exposed.

Nonetheless, national scale measures of body burden are useful indicators of exposure in the population. While such measures do not necessarily provide information about the nature of the exposures, they do represent the average levels of exposure in the population as a whole. Such national scale measures of body burden are often more convenient to obtain than to estimate the exposures by accounting for all of the exposure concentrations and durations for the whole population. As mentioned earlier, body burden (biomonitoring) data are not available for all pollutants of interest to EPA. In such cases, ambient data or exposure measurements and models are used to assess human exposure.

4.4.2 Data Sources for Biomonitoring Indicators

Two primary sources provided data for the biomonitoring indicators presented in this section:

- **The National Health and Nutrition Examination Survey (NHANES)**, conducted by the National Center for Health Statistics (NCHS). Specifically, data were used from the second, third, and fourth surveys (NHANES II; NHANES III; and NHANES IV [1999-2000]).
- **EPA's National Human Exposure Assessment Survey (NHEXAS)**. Specifically, data were used from surveys of three regions: Maryland, EPA Region 5, and Arizona (NHEXAS–MD; NHEXAS–Region 5; and NHEXAS–AZ).

Two others sources of biomonitoring data—autopsy data and tissue registry data—were considered but not used for these indicators. As described below, neither of these sources contains rich biomonitoring data, which significantly limits their usefulness as data sources for human contaminant levels.

National Center for Health Statistics, National Health and Nutrition Examination Survey (NHANES)

NHANES consists of a series of surveys conducted by CDC's NCHS. The survey is designed to collect data on the health of the U.S. population, including information on topics such as nutrition, cardiovascular disease, and exposure to chemicals (CDC, 2001c). The NHANES surveys have been performed over a number of years. The first survey, NHANES I, took place from 1971 through 1975; NHANES II occurred from 1976 through 1980; NHANES III was performed from 1988 through 1994; and the most recent NHANES for which data are available took place in 1999-2000. In this section, the year(s) in which the data were collected are identified in each citation of NHANES.

As part of the survey, blood and urine samples were collected to measure the amounts of certain chemicals thought to be potentially harmful to people. Because of the extensive work involved with laboratory analysis, some chemicals were measured for all people in the survey, while other chemicals were only measured in representative subsamples of people in an age group.

The CDC National Report on Human Exposure to Environmental Chemicals (often referred to as the "CDC Report Card") (CDC, 2001c) summarizes chemical exposure data from NHANES. Information from the CDC report is presented hereafter under the heading "NHANES 1999-2000." To date, this report has been released twice. Data from the first report are updated in the larger, second report. The second report represents the U.S. population over a 2-year period, 1999-2000. Two years of data provide more stable estimates for the total population and are necessary for adequate sample sizes for some subgroup analysis. Future reports will be released every 2 years and will cover data for a 2-year period (e.g., 2001-2002, 2003-2004, 2005-2006).

National Human Exposure Assessment Survey (NHEXAS)

The goal of NHEXAS was to better understand the complete picture of human exposure to toxic chemicals by looking at humans' many exposures to all types of toxic chemicals. NHEXAS was a multiday, multimedia study that examined chemical concentrations in indoor air, outdoor air, dust, soil, food, beverages, drinking water, and tap water. For some contaminants, body burden measurements were obtained from samples of blood, hair, or urine.

Phase 1 of NHEXAS consisted of demonstration and scoping studies in Maryland; Phoenix, Arizona; and EPA Region 5 using probability-based sampling designs. Although the study was conducted in three different regions of the U.S., it was not designed to be nationally representative. The Region 5 study was conducted in Ohio, Michigan, Illinois, Indiana, Wisconsin, and Minnesota and measured metals and VOCs. The Arizona study measured metals, pesticides, and VOCs. The target population for the NHEXAS-MD study consisted of the non-institutionalized permanent residents of households in the city of Baltimore or four counties in Maryland. Samples from select environmental and biological media, as well as questionnaire data, were collected in NHEXAS-MD. The three NHEXAS studies are identified in this section as NHEXAS-AZ, NHEXAS-Region 5, or NHEXAS-MD, to indicate where they were performed.

Autopsy Data

Autopsies can provide important information about deaths resulting from known or suspected environmental or occupational hazards. For example, one of the earliest indications of the rise in lung cancer deaths came from reports that lung cancers were being identified with increasing frequency in autopsies (Hanzlick, 1998).

The value of an autopsy database for body burden and epidemiologic studies has been recognized; however, few such studies have been conducted. This is partly because autopsies are

performed on a non-random sample of deaths and because environmental contaminant levels are typically not measured during an autopsy (Moore, et al., 1996). Also, autopsies are performed on only a small percentage of the U.S. population. In 1980, autopsies were performed in approximately 17 percent of deaths in the U.S. By 1985, the percentage had declined to 14 percent. While nearly all deaths due to homicide and other medico-legal causes were autopsied, autopsies were performed in only 12 percent of all deaths due to natural causes (CDC, 1998a).

Difficulties in accessing autopsy data can limit their usefulness as well. Prior to 1995, the National Center for Health Statistics (NCHS) collected data from death certificates indicating whether an autopsy was performed. Since that time, however, such information is no longer available from the NCHS national mortality statistics databases (Hanzlick, 1998).

Tissue Registry Data

Human tissues are stored for study in many forms including solid organs, organ sections, histology slides, cells, and DNA. Tissue registries are maintained for medical education and biological research, but few studies have been conducted to identify trends in environmental contaminants in tissues using tissue registries. Tissue registry samples and information are not population-based, and at present there is no central database containing information about tissue samples (Eiseman and Haga, 1999).

EPA has conducted one of the most extensive tissue studies. From 1976 to 1987, the EPA conducted the National Human Adipose Tissue Survey (NHATS). NHATS was a national survey that collected adipose tissue samples to monitor exposure to toxic compounds among the general population. Pathologists and medical examiners from 47 metropolitan areas collected samples from autopsies and elected surgeries (Crinnion, 2000; Orban, et al., 1994). Even though the study was a significant biomonitoring effort, data from NHATS are not presented in this report because newer data sources are available.

4.4.3 What is the level of exposure to heavy metals?

Heavy metals are important environmental pollutants because they are related to several adverse health effects when ingested or inhaled. Five metals have been selected for in-depth presentation in this section: chromium, lead, arsenic, mercury, and cadmium. These metals are known to be related to severe adverse health effects and are relatively common in household, work, and school environments. Exhibit 4-32 presents EPA regulatory standards and guidelines for these five metals. Indicators are available for lead, arsenic, mercury, and cadmium and are discussed on the following pages. At present, no indicator is available for chromium, but it is discussed below because human health may be adversely affected by chromium in the environment. (For additional information on heavy metals in the environment, see Chapter 1, Cleaner Air.)

Chromium

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms, but primarily in two valence states: trivalent chromium (III) and hexavalent chromium (VI). Chromium (III) is an essential nutrient and is much less toxic than chromium (VI), which is generally produced by industrial processes. Chromium (III) and chromium (VI) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving (ATSDR, 2001).

In air, chromium compounds are present mostly as fine dust particles that eventually settle over land and water. Chromium can strongly attach to soil and only a small amount can dissolve in water and move deeper in the soil to underground water. Fish do not accumulate much chromium in their bodies from water (ATSDR, 2001).

People can be exposed to chromium by eating food containing chromium (III); breathing contaminated workplace air or experiencing skin contact during use in the workplace; drinking contaminated well water; or living near uncontrolled hazardous waste sites containing chromium or near industries that use chromium (ATSDR, 2001). Although studies have been conducted that measure the amount of chromium in drinking water, ground water, soil, and air, there are no studies that measure the body burden of chromium in human tissue.

Chromium III is an essential nutrient that helps the body use sugar, protein, and fat. An intake of 50-200 µg of chromium (III) per day is recommended for adults. On average, adults in the U.S. take in an estimated 60-80 µg of chromium per day in food. Therefore, many people's diets may not provide enough chromium (III). Without chromium III in the diet, the body loses its ability to use sugars, proteins, and fat properly, which may result in weight loss or decreased growth, improper function of the nervous system, and a diabetic-like condition. Therefore, chromium (III) compounds have been used as dietary supplements and are beneficial if taken in recommended (but not excessive) dosages (ATSDR, 2000). Chronic high exposures to chromium (III), however, may affect the skin, liver, or kidneys (ACGIH, 1991; Rom, 1992).

In general, chromium (VI) is more toxic than chromium III. Breathing in high levels (greater than 2 µg/m^3) of chromium (VI), such as in a compound known as chromic acid or chromium (VI) trioxide, can irritate the nose, causing symptoms such as runny nose, sneezing, itching, nosebleeds, ulcers, and holes in the nasal septum. These effects have primarily occurred in factory workers who make or use chromium (VI) for several months to many years. Long-term exposure to chromium (VI) has been associated with lung cancer in workers exposed to levels in air that were 100 to 1,000 times higher than those found in the natural environment. Lung cancer may occur long after exposure to chromium VI has ended (ATSDR, 2000).

No biomonitoring data are readily available for chromium. Interest is developing in examining chromium as an emerging environmental pollutant.

Exhibit 4-32: United States federal standards and criteria for five heavy metals

Heavy Metal	Drinking Water Maximum Contaminant Level (MCL)[1]	Ground Water Cleanup Level[2]	Air Standards
Lead	0.015 mg/L	0.015 mg/L	1.5 µg/L[3]
Arsenic	0.01 mg/L	0.01 mg/L	Not Applicable[4]
Mercury	0.002 mg/L	0.002 mg/L	Not Applicable[5]
Chromium	0.1 mg/L	0.1 mg/L	Not Applicable[4]
Cadmium	0.005 mg/L	0.005 mg/L	Not Applicable[4]

1. MCLs are regulatory standards developed pursuant to the Federal Safe Drinking Water Act (SDWA).
2. A groundwater cleanup level is most often the MCL (per the Comprehensive Environmental Response, Compensation, and Liability Act [CERCLA] [also known as Superfund] and Resource Conservation and Recovery Act [RCRA] guidance) for the particular contaminant. Groundwater cleanup levels are established by EPA and states on a case-by-case basis for Superfund site clean-ups and corrective actions at RCRA solid and hazardous waste management.
3. This standard is a quarterly average. Lead is a criteria air pollutant (under the Clean Air Act) and therefore has a health-based standard.
4. This heavy metal is not a criteria air pollutant and thus there is not a health-based standard. Air pollution standards for this heavy metal are technology-based standards, not health-based standards. For example, the emission standard for arsenic is that which is achievable with the best available technology (BAT) for treating arsenic air emissions. In addition, the BAT for arsenic emissions varies across industry sectors and thus emission standards for arsenic also vary across industry sectors.

Source: EPA. *Current Drinking Water Standards.* 2002; EPA. EPA. *Handbook of Groundwater Policies for RCRA Corrective Action.* 2000; EPA. *National Air Quality and Emissions Trends Report 1999.* 2001.

Indicator Blood lead level - Category 1

Lead is a naturally occurring metal found in small amounts in rock and soil. Lead has been used industrially in the production of gasoline, ceramic products, paints, and solder. Lead-based paint and lead-contaminated dust from paint are the primary sources of lead exposure in the home. The body burden of lead can be measured as the amount of lead in blood or the amount of lead in urine. The health effects of lead are discussed in Section 4.1 of this chapter.

What the Data Show

NHANES 1999-2000. The mean blood lead levels for adults are illustrated in Exhibit 4-33. The mean blood lead level for all males in the survey was 2.0 micrograms per deciliter (µg/dL) and 1.4 µg/dL for all females. The mean blood lead level for non-Hispanic African Americans was 1.9 µg/dL. The mean blood lead level for Mexican Americans was 1.8 µg/dL (CDC, 2001c).

NHANES III (1988-1994). Blood lead levels of people were surveyed in two separate phases of NHANES III. The data collected during Phase 2 (1991 through 1994) indicated that the U.S. population's exposure to lead was decreasing.

NHEXAS-Region 5. Blood lead levels for 165 participants were obtained during NHEXAS-Region 5. Lead levels in blood were detectable for about 94 percent of the population; most of the individuals had lead levels well below 10 µg/dL. The mean blood lead level of the participants was 2.18 µg/dL (Clayton, et al., 1999).

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-33, for more information.)

Exhibit 4-33: Geometric mean and selected percentiles of total blood lead concentrations (in µg/dL) for the United States population, aged 1 year and older, by selected demographic groups, National Health and Nutrition Examination Survey (NHANES), 1999-2000

			Selected Percentiles				
	Sample Size	Geometric Mean	10th	25th	50th	75th	90th
Total, Age 1 and older	7,970	1.7	0.8	1.0	1.6	2.4	3.8
Sex							
Male	3,913	2.0	0.8	1.3	1.8	2.9	4.4
Female	4,057	1.4	0.6	0.8	1.3	1.9	3.0
Race/Ethnicity							
Black, non-Hispanic	1,842	1.9	0.7	1.1	1.7	2.8	4.2
Mexican American	2,743	1.8	0.8	1.2	1.8	2.7	4.2
White, non-Hispanic*	2,715	1.6	0.6	1.0	1.6	2.4	3.6
Age Group							
1-5 years	723	2.2	1.0**	1.4	2.2	3.3	4.8**
6-11 years	905	1.5	0.7	0.9	1.3	2.0	3.3
12-19 years	2,135	1.1	0.4	0.8	1.0	1.4	2.3
20 years and older	4,207	1.8	0.7	1.0	1.7	2.5	3.9

* Includes other racial/ethnic groups.

Source: CDC. *Second National Report on Human Exposure to Environmental Chemicals.* 2003.

Indicator Urine arsenic level - Category 2

Arsenic occurs in rock, soil, water, air, plants, and animals. Exposure occurs when arsenic is further released into the environment through erosion, volcanic action, forest fires, or human actions. Human activities involve its use in wood preservatives, dyes, paints, paper production, and cement manufacturing. Arsenic mining is also a source of human exposure (EPA, 2001a).

Inorganic arsenic has been recognized as a human poison since ancient times, and large oral doses (above 60,000 ppb in food or water) can produce death. Lower levels of inorganic arsenic (ranging from about 300 to 30,000 ppb in food, water, or pharmaceuticals) may cause symptoms such as stomach ache, nausea, vomiting, and diarrhea. Inorganic arsenic is a multi-site human carcinogen. Populations with exposures above several hundred ppb are reported to have increased risks of skin, bladder, and lung cancer. The U.S. Department of Health and Human Services (USDHHS) has determined that inorganic arsenic is a known carcinogen. The International Agency for Research on Cancer (IARC) had determined that inorganic arsenic is carcinogenic to humans. Both the EPA and the National Toxicology Program (NTP) have classified inorganic arsenic as a known human carcinogen (ATSDR, 2001).

A large number of adverse noncarcinogenic effects have been reported in humans. The most prominent are changes in the skin, (e.g., hyperpigmentation and keratoses). Other effects that have been reported include alterations in gastrointestinal, cardiovascular, hematological, pulmonary, neurological, immunological, and reproductive developmental function (NRC, 1999).

Children who are exposed to arsenic may have many of the same effects as adults, including irritation of the stomach and intestines, blood vessel damage, skin changes, and reduced nerve function. Thus, all health effects observed in adults are of potential concern in children (ATSDR, 2001).

What the Data Show

NHEXAS-Region 5. Arsenic levels in urine were measured for approximately 202 participants during NHEXAS-Region 5. The mean urine arsenic level was 29.32 micrograms per liter (µg/L), while the median urine arsenic level was 3.65 µg/L. The mean level is much higher than the median level, indicating that the distribution is highly skewed to the higher values (Clayton, et al., 1999).

NHANES. Future NHANES studies will include arsenic. Therefore, NHANES will serve as the biomonitoring data source for arsenic. When NHANES becomes the indicator data source for arsenic, the indicator will become a Category 1 indicator.

Data Source

NHEXAS, Environmental Protection Agency. (See Appendix B, page B-33, for more information.)

Indicator Blood mercury level - Category 1

Mercury is a naturally occurring metal that is widespread and persistent in the environment. It is found in elemental form and in various organic compounds and complexes. Methylmercury (one organic form of mercury) can accumulate up the food chain in aquatic systems and lead to high concentrations of methylmercury in predatory fish. Consumption of contaminated fish is the major source of human exposure to methylmercury in the U.S. (NRC, 2000).

Methylmercury is rapidly absorbed from the gastrointestinal tract and readily enters the brain, where it accumulates and is slowly converted to inorganic mercury. A spectrum of adverse health effects has been observed following methylmercury exposure, with the severity depending largely on the magnitude of the dose. The most severe effects reported in humans were seen following high-dose poisoning episodes in Japan and Iraq. The fetus is considered much more sensitive than the adult. Prenatal exposures interfere with the growth and migration of neurons and have the potential to cause irreversible damage to the developing central nervous system. Infants exposed in utero during the Japan and Iraqi episodes were born with severe disabilities, such as mental retardation, seizure disorders, cerebral palsy, blindness, and deafness. Chronic low-dose prenatal methylmercury exposure from maternal consumption of fish has been associated with more subtle end points of neurotoxicity (e.g., IQ deficits, abnormal muscle tone, decrements in motor function, attention and visuospatial performance) (NRC, 2000).

The human health effects of mercury are diverse and depend upon the forms of mercury encountered and the severity and length of exposure. Large acute exposures to elemental mercury vapor can result in lung damage. Lower dose or chronic inhalation may affect the nervous system, resulting in symptoms such as weakness, fatigue, weight loss, gastrointestinal problems, and behavioral and personality changes. Organic mercury is more toxic than inorganic and elemental mercury (CDC, 2001c). Health effects of organic mercury include vision changes, sensory changes in the limbs, cognitive disturbances, dermatitis, and muscle deterioration. The developing nervous system of the fetus and infants is susceptible to the effects of methylmercury (CDC, 2003).

What the Data Show

NHANES 1999-2000. The blood mercury level reported in NHANES is total blood mercury, including both organic and inorganic mercury. Mercury levels were measured in blood and urine during NHANES 1999-2000 for 705 children aged 1-5 years, and 1,709 adult females aged 16-49. The mean blood mercury level for males and females aged 1-5 years was 0.34 micrograms per liter (µg/L), and the mean blood mercury level for adult females was 1.02 µg/L.

NHEXAS-Region 5. Mercury concentrations in human hair were measured for 182 participants during NHEXAS-Region 5. The mean mercury level in hair, annualized for seasonality, was 287 ppb. More people in older age categories have high levels of mercury in their hair. This increase in mercury level was found not to be an effect of income level (Pellizari, et al., 1999).

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-33, for more information.)

Exhibit 4-34: Geometric mean and selected percentiles of blood mercury concentrations (in µg/L) for males and females aged 1-5 years and females aged 16 to 49 years in the U.S. population, National Health and Nutrition Examination Survey (NHANES), 1999-2000

			Selected Percentiles				
	Sample Size	Geometric Mean	10th	25th	50th	75th	90th
Age Group and Sex							
Males/Females 1-5 years	705	0.3	<LOD	<LOD	0.3	0.5	1.4
Males	387	0.3	<LOD	<LOD	0.2	0.5	1.1
Females	318	0.3	<LOD	<LOD	0.2	0.8	1.6
Females 16-49 years	1709	1.0	0.2	0.4	0.9	2.0	4.9
Race/Ethnicity (females 16-49 only)							
Mexican Americans	579	0.8	0.2	0.4	0.9	1.4	2.6
Non-Hispanic blacks	370	1.4	0.3	0.6	1.3	2.6	4.8
Non-Hispanic whites	588	0.9	<LOD	0.4	0.9	1.9	5.0

<LOD means less than the limit of detection, which is 0.14 µg/L.

Source: CDC. *Second National Report on Human Exposure to Environmental Chemicals*. 2002.

Indicator Blood cadmium level - Category 1

Elemental cadmium is a metal that is usually found in nature combined with other elements such as oxygen, chlorine, or sulfur. Cadmium enters the environment from the weathering of rocks and minerals that contain cadmium. Exposure to cadmium can occur in occupations such as mining or electroplating, where cadmium is used or produced. Cadmium exposure can also occur from exposure to cigarette smoke (CDC, 2001c).

Cadmium and its compounds are toxic. Once absorbed into the human body, cadmium can remain for decades. Exposure to cadmium for many years may result in cadmium accumulation in the kidneys and serious kidney damage. Chronic ingestion of cadmium has resulted in osteomalacia, a bone disorder similar to rickets. Acute airborne exposure, as occurs from welding on cadmium-alloy metals, can result in swelling (edema) and scarring (fibrosis) of the lungs (CDC, 2003).

What the Data Show

NHANES 1999-2000. This survey measured blood cadmium levels in people 1 year and older, and urine cadmium levels in a sample of people 6 years and older. Recent advances in analytical chemistry have made it possible to measure cadmium in very small amounts in blood and urine. Finding a measurable amount of cadmium in the blood or urine does not mean that the level of cadmium causes an adverse health effect (CDC, 2001c). The blood cadmium biomonitoring measurements are similar among males and females as well as among the racial or ethnic groups sampled. Exhibit 4-35 shows that blood levels were higher among people 20 years of age or older than for people younger than 20 years of age (CDC, 2001c). The mean urine cadmium level was 0.3 µg/L (CDC, 2001c).

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-34, for more information.)

Exhibit 4-35: Geometric mean and selected percentiles of blood cadmium concentrations (in µg/L) for the United States population, aged 1 year and older, by selected demographic groups, National Health and Nutrition Examination Survey (NHANES), 1999-2000

			Selected Percentiles				
	Sample Size	Geometric Mean	10th	25th	50th	75th	90th
Total, Age 1 and Older	7,970	0.4	<LOD	<LOD	0.3	0.6	1.0
Sex							
Male	3,913	0.4	<LOD	<LOD	0.4	0.6	1.0
Female	4,057	0.4	<LOD	<LOD	0.3	0.6	1.0
Race/Ethnicity							
Black, non-Hispanic	1,842	0.4	<LOD	<LOD	0.3	0.6	1.0
Mexican American	2,743	0.4	<LOD	<LOD	0.4	0.4	0.7
White, non-Hispanic*	2,715	0.4	<LOD	<LOD	0.4	0.5	1.0
Age Group							
1-5 years	723	**	<LOD	<LOD	<LOD	0.3	0.4
6-11 years	905	**	<LOD	<LOD	<LOD	0.3	0.4
12-19 years	2,135	0.3	<LOD	<LOD	0.3	0.3	0.8
20+ years	4,207	0.5	<LOD	<LOD	0.4	0.6	1.0

* Includes other racial/ethnic groups. <LOD= Less than the limit of detection of the analytical method.

Source: CDC. *Second National Report on Human Exposure to Environmental Chemicals*. 2003.

4.4.4 What is the level of exposure to cotinine?

Environmental tobacco smoke (ETS) is a dynamic, complex mixture of more than 4,000 chemicals found in both vapor and particle phases. Many of these chemicals are known toxic or carcinogenic agents (ALA, et al., 1994). The EPA has classified ETS as a known human carcinogen and estimates that it is responsible for approximately 3,000 lung cancer deaths per year among non-smokers in the U.S. (EPA, NCEA, December 1992).

Cotinine is a major metabolic product of nicotine and is currently regarded as the best biomarker for exposure of active smokers and non-smokers to ETS. Measuring cotinine is preferred over measuring nicotine because, although both are specific for exposure to tobacco, cotinine remains in the body much longer than nicotine.

Indicator: Blood cotinine level - Category I

Cotinine can be measured in blood, urine, saliva, and hair. Non-smokers exposed to ETS have cotinine levels of less than 1 nanogram per milliliter (ng/mL), with heavy exposure to ETS producing levels in the 1 to 15 ng/mL range. Active smokers almost always have levels higher than 15 ng/mL (CDC, 2001c).

What the Data Show

NHANES 1999-2000. Exhibit 4-36 presents data for the U.S. non-smoking population aged 3 years and older. Males have higher levels than females, and people aged 20 years and older have lower levels than those younger than 20 years of age. Levels for non-Hispanic African Americans are higher than for other ethnic groups (CDC, 2001c).

NHANES III (1988-1991). As part of NHANES III, CDC determined that the median level of cotinine among non-smokers in the U.S. was 0.20 ng/mL (Pirkle, et al., 1996, in CDC, 2001c). Results from NHANES 1999-2000 show that the median cotinine level has decreased to less than 0.050 ng/mL—more than a 75 percent decrease from NHANES III to NHANES 1999-2000 (CDC, 2001c). NHANES III (1988-1991) provided the first evidence from a national study that serum cotinine levels are higher among Black smokers than among White or Mexican American smokers at all levels of cigarette smoking (Caraballo, et al., 1998).

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-34, for more information.)

Exhibit 4-36: Selected percentiles of serum cotinine concentrations (in ng/mL) for the United States non-smoking population, aged 3 years and older, National Health and Nutrition Examination Survey (NHANES), 1999-2000

		Selected Percentiles				
	Sample Size	10th	25th	50th	75th	90th
Total, Age 3 years and Older	5,999	<LOD	<LOD	0.06	0.24	1.02
Sex						
Male	2,789	<LOD	<LOD	0.08	0.30	1.20
Female	3,210	<LOD	<LOD	<LOD	0.18	0.85
Race/Ethnicity						
Black, non-Hispanic*	1,333	<LOD	<LOD	0.13	0.50	1.43
Mexican American	2,242	<LOD	<LOD	<LOD	0.14	0.51
White, non-Hispanic**	1,949	<LOD	<LOD	0.05	0.21	0.95
Age Group						
3-11 years	1,174	<LOD	<LOD	0.11	0.50	1.88
12-19 years	1,773	<LOD	<LOD	0.11	0.54	1.65
20+ years	3,052	<LOD	<LOD	<LOD	0.17	0.63

* Research in progress to determine whether levels for black, non-Hispanic people may be affected by biological factors.
** Includes other racial/ethnic groups.
<LOD= Less than the limit of detection of 0.05 ng/mL in serum.

Source: CDC. *Second National Report on Human Exposure to Environmental Chemicals.* 2003.

4.4.5 What is the level of exposure to volatile organic compounds?

In addition to the health effects attributed to VOCs themselves, VOCs are also chemical compounds that contribute significantly to the formation of ground-level ozone (smog) when released to the air. Exposure to ground-level ozone can damage lung tissue and cause serious respiratory illness. (For additional information on VOCs in the environment, see Chapter 1, Cleaner Air.)

Indicator Blood VOC levels - Category I

Biomonitoring data for volatile compounds are difficult to obtain because these compounds do not persist for very long in the body. For this reason, biomonitoring data are indicative of recent exposure only. Only relatively older sources of data, NHEXAS and NHANES III, are available for the body burden of VOCs.

What the Data Show

NHEXAS-Region 5. Blood levels of four VOCs were obtained for participants in NHEXAS-Region 5. The four compounds were benzene, chloroform, tetrachloroethylene (PERC), and trichloroethylene (TCE). The mean level of benzene measured in blood was 0.07 µg/L. The mean level of chloroform was 0.07 µ/L. The mean level of PERC was 0.21 µg/L. The mean level of TCE was below the limit of detection (Clayton, et al., 1999).

NHANES III (1988-1994). Blood samples were analyzed for the presence of VOCs during NHANES III. NHANES III was conducted on a nationwide probability sample of approximately 33,994 persons aged 2 months or older. Of these, an exposure questionnaire was administered and blood samples analyzed for VOCs in a convenience sample of 1,018 adult participants aged 20 to 59 years. Toluene, styrene, and benzene were present in the blood of more than 75 percent of the participants. Analysis of this and other data collected during NHANES III shows a strong association between lifetime cigarette smoking and toluene, benzene, and styrene levels (Churchill and Kaye, 2001).

Data Source

NHANES III (1988-1994), National Center for Health Statistics. (See Appendix B, page B-34, for more information.)

4.4.6 What is the level of exposure to pesticides?

Organophosphate pesticides account for about half of the insecticides used in the U.S. Organophosphate pesticides are active against a broad spectrum of insects and are used on food crops as well as in residential and commercial buildings and on ornamental plants and lawns. Exposure to these pesticides occurs primarily from ingestion of food products or from residential use (CDC, 2001c).

The mechanism of toxicity of the organophosphate pesticides is to inhibit the enzyme that breaks down acetylcholine, which transfers nerve impulses between nerve cells or from a nerve cell to other types of cells, such as muscle cells. This leads to a buildup of acetylcholine, which overstimulates muscles, causing symptoms such as weakness and paralysis (CDC, 2001c). (For additional information on pesticides in the environment, see Chapter 1, Cleaner Air; Chapter 2, Purer Water; and Chapter 3, Better Protected Land.)

Indicator Urine organophosphate levels to indicate pesticides - Category I

Pesticides biomonitoring data are obtained by measuring the chemicals that pesticides are broken down into in the body. Measurement of these pesticide metabolites reflects exposure to pesticides that has occurred predominantly in the last few days (CDC, 2001c). The reason is that these metabolites persist within the body for only a short time.

Presently, national biomonitoring data are available primarily for organophosphate pesticides. Future studies may provide additional indicators for non-organophosphate pesticides, such as carbamates and persistent pesticides.

What the Data Show

NHANES 1999-2000. Urine levels of organophosphate pesticide metabolites were measured in a subsample of NHANES participants 6 through 59 years of age who were selected to be representative of the U.S. population. Finding a measurable amount of one or more metabolites in urine does not mean that the level of the organophosphate causes an adverse health effect. Whether organophosphate pesticides at the levels of metabolites reported during NHANES 1999-2000 are a cause for health concern is not known (CDC, 2001c). Exhibit 4-37 shows the amount of each metabolite in urine reported in NHANES 1999-2000.

NHEXAS-MD. Urine levels of metabolites of some common pesticides were measured during NHEXAS-MD. 1-naphthol (1NAP) is a urinary metabolite of both carbaryl and naphthalene. The mean urine level of 1NAP measured for 338 participants was 33.7 µg/L. 3,5,6-trichloro-2-pyridinol (TCPY) is the major metabolite in urine of the pesticides chlorpyrifos, chlorpyrifos-methyl, and triclopyr. The mean urine level of TCPY measured for 346 participants was 6.8 µg/L. Malathion dicarboxylic acid (MDA) is a principal metabolite of malathion, an organophosphate pesticide used against insects. The mean urine level for MDA measured during NHEXAS-MD was below the level of detection. Atrazine mercapturate (AM) is a urinary metabolite of atrazine, a widely used herbicide in the U.S. The mean urine level for AM measured during NHEXAS-MD was below the level of detection (MacIntosh, et al., 1999).

Exhibit 4-37: Geometric mean and selected percentiles of selected pesticide metabolite urine concentrations and creatinine-adjusted levels for the United States population aged 6-59 years, National Health and Nutrition Examination Survey (NHANES), 1999-2000

	Sample Size	Geometric Mean	Selected Percentiles (95% confidence interval) 10th	25th	50th	75th	90th
Dimethylphosphate							
µg/L of urine	1,949	NC	< LOD	< LOD	0.74	2.80	7.90
µg/g of creatinine*	1,949	NC	< LOD	< LOD	0.81	2.93	8.46
Dimethylthiophosphate							
µg/L of urine	1,948	1.82	< LOD	< LOD	2.70	10.0	38.0
µg/g of creatinine*	1,948	1.64	< LOD	< LOD	2.12	9.57	32.0
Dimethyldithiophosphate							
µg/L of urine	1,949	NC	< LOD	< LOD	< LOD	2.30	12.0
µg/g of creatinine*	1,949	NC	< LOD	< LOD	< LOD	1.86	10.1
Diethylphosphate							
µg/L of urine	1,949	1.03	< LOD	< LOD	1.20	3.10	7.50
µg/g of creatinine*	1,949	0.92	< LOD	< LOD	0.93	2.73	7.94
Diethylthiophosphate							
µg/L of urine	1,949	NC	< LOD	< LOD	0.49	0.76	1.3
µg/g of creatinine*	1,949	NC	< LOD	< LOD	0.25	0.71	1.7
Diethyldithiophosphate							
µg/L of urine	1,949	NC	< LOD	< LOD	0.08	0.20	0.47
µg/g of creatinine*	1,949	NC	< LOD	< LOD	0.07	0.20	0.55

µg per gram of creatinine in urine.
<LOD= Less than the limit of detection for the analytical method.

NC=Not calculated - Proportion of results below limit of detection was too high to provide a valid result.

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-35, for more information.)

4.4.7 What is the level of exposure to persistent organic pollutants?

Persistent organic pollutants (POPs) are manmade organic chemicals that remain in the environment for long periods of time. Some POPs are toxic; others are not. Toxic POPs are of a special concern because they often remain toxic for decades or longer. The more persistent a toxic chemical is, the greater the probability for human exposure over time.

POPs have been linked to adverse health effects such as cancer, nervous system damage, reproductive disorders, and disruption of the immune system in both human and animals. POPs released in one part of the world can travel to regions far from their place of origin, because they circulate globally long after their release into the atmosphere, oceans, and other pathways (EPA, 2001b).

Under the United Nations Environment Program, the international community has identified 12 chemicals as primary POPs. These chemicals include certain insecticides such as dichlorodiphenyl-trichloroethane (DDT) and chlordane, which were once commonly used to control pests, and polychlorinated biphenyls (PCBs), which were used in hundreds of commercial applications for electrical, heat transfer, and hydraulic equipment, and in plasticizers in paints, plastics, and rubber products.

The 12 chemicals targeted by EPA as POPs are the pesticides aldrin, chlordane, DDT, mirex, toxaphene, dieldrin, endrin, and heptachlor; hexachlorobenzene, an industrial chemical; PCBs; polychlorinated dibenzo-p-dioxins (dioxins); and polychlorinated dibenzo-p-furans (furans) (EPA, 2001b).

The following discussion of human exposure to POPs is derived from the *Second National Report on Human Exposure to Environmental Chemicals,* published in January 2003 by the CDC National Center for Environmental Health (CDC, 2003). Four of the 12 POPs are not addressed by the CDC report and are therefore not addressed specifically in this chapter. These four chemicals are aldrin, toxaphene, dieldrin, and endrin. The remaining POPs were not evaluated for indicators at this time but EPA anticipates that these chemicals will become indicators in the future.

Chlordane and Heptachlor

In 1988, EPA banned the use and production of chlordane in the U.S. Chlordane is an organochlorine pesticide that was applied in and around buildings to eliminate termites and was also used as an agricultural and lawn pesticide. The technical grade of chlordane consists of a group of related chemicals, including heptachlor, *cis*-chlordane, *trans*-chlordane, and *trans*-nonachlor. Note that heptachlor was also used individually as a pesticide separate from chlordane. However, pesticide applications were mostly made with technical grade chlordane and therefore chlordane is the main form of heptachlor exposure.

Within the body, chlordane is metabolized to oxychlordane and heptachlor is metabolized to heptachlor epoxide. Human exposure to chlordane and heptachlor is determined by measuring the blood serum concentrations of oxychlordane, *trans*-nonachlor, and heptachlor epoxide. However, generally recognized guidelines for serum levels of these metabolites have not been established.

The NHANES 1999-2000 mean levels of oxychlordane and heptachlor epoxide in the overall population were below the lipid-adjusted level of detection, which averaged 7.4 ng/g of lipid. The NHANES II (1976-1980) 95th percentile level was about twice the NHANES 1999-2000 level for oxychlordane and *trans*-nonachlor.

DDT

DDT was initially used by the military during the 1940s to control mosquitoes that carried vector-borne diseases such as malaria. EPA banned the use of DDT in the U.S. in 1973. DDT, however, is still produced and used in other countries.

For the general population, food is the most common pathway of exposure. Diets that involve large amounts of Great Lakes fish will increase an individual's exposure to DDT. Food intake of DDT has decreased since the 1950s; however, food imported to the U.S. may have DDT contamination, especially food imported from tropical regions where DDT is used in the greatest quantities.

Dichlorodiphenyldichloroethylene (DDE) (more persistent than DDT) is a major DDT metabolite that can be produced in people or in the environment. DDT in the human body reflects either a relatively recent exposure or a cumulative past exposure over time. A high DDT-to-DDE ratio may indicate a recent exposure, and a low DDT-to-DDE ratio may indicate an exposure in the more distant past.

The NHANES 1999-2000 95th percentile levels (lipid-adjusted serum) for DDT and DDE in the overall population range from 5-fold to 15-fold lower than levels detected in a non-random subsample of NHANES II (1976-1980). These decreases in the U.S. levels are consistent with the decreased use and manufacture of these chemicals. Also, within NHANES 1999-2000, the group aged 12 to 19 years had DDE levels 2-fold lower than the group 20 years and older.

Hexachlorobenzene (HCB)

Hexachlorobenzene is a persistent, bioaccumulative, and toxic pollutant (EPA, 2003b). It was commonly used as a pesticide until 1965, as a fungicide to protect wheat seeds, and for a variety of industrial purposes, including rubber, aluminum, and dye production and wood preservation (EPA, 2003c). In 1984, EPA canceled its registered use. There currently are no commercial uses of HCB in the U.S. (EPA,

2003c); however, HCB is still formed as a by-product during the manufacture of other chemicals (mainly solvents) and pesticides.

Human exposure to HCB can occur through work in or proximity to chemical manufacturing sites where it is formed as a by-product or to waste facilities where it is disposed. People also can be exposed by consuming foods tainted with hexachlorobenzene (EPA, 2003c). EPA has set the maximum contaminant level (MCL) for hexachlorobenzene in drinking water at 1 part per billion. If HCB levels exceed this level, the water supplier must notify the public (EPA, 2002g).

HCB has been found to potentially cause skin lesions and nerve and liver damage when people are exposed at levels above the MCL for relatively short periods (EPA, 2002g). Lifetime exposure at levels above the MCL can damage the liver and kidneys and cause reproductive effects, benign tumors of endocrine glands, and cancer (EPA, 2002g).

Epidemiologic studies of persons orally exposed to HCB have not shown an increased cancer incidence. However, EPA has classified HCB as a probable human carcinogen (Group B2) based on animal studies that have reported cancer of the liver, thyroid, and kidney from oral HCB exposure. Very few inhalation data are available (EPA, 2003c).

Generally recognized guidelines for HCB serum levels are not available. HCB was detected in 0.6 percent of people during the 1999-2000 NHANES study. Finding detectable amounts does not mean that those levels produce adverse health effects. HCB has a residence time of approximately 15 years in body fat.

PCBs

PCBs are chlorinated aromatic hydrocarbon chemicals that were once used as electrical insulating and heat exchange fluids. Within the U.S., peak production occurred in the early 1970s and production within the U.S. was banned in 1979. Concern over these chemicals remains high because they are still released into the environment.

Sources of exposure for the general population include release of PCBs from waste sites and from fires involving transformers and capacitors; ingestion of foods containing PCBs due to contamination of animal feeds; migration from packaging materials; and accumulation in the fatty tissues of livestock. PCBs are found at higher concentrations in fatty foods. In occupational settings, workers can be exposed to PCBs from remediation activities at hazardous waste sites and from the repair of transformers, capacitors, and hydraulic systems (CDC, 2003a).

The Food and Drug Administration and the Occupational Safety and Health Administration have developed criteria for allowable levels of PCBs in foods and the workplace. EPA has established criteria for water and for the storage and removal of PCB-contaminated wastes.

Overall, there are three categories of at least 25 different PCB compounds (termed congeners) as determined by molecular structure. Congeners are closely related chemical compounds. The three categories are coplanar PCBs, mono-ortho substituted PCBs, and other PCBs. The significance of these categories is that coplanar and mono-ortho substituted PCBs have health effects similar to dioxins. Overall, the human health effects of PCBs include liver disorders, elevated lipids, and gastrointestinal cancers (CDC, 2003a).

The detection of serum PCBs can reflect either recent or past exposures to PCBs. Those PCBs with higher degrees of chlorination persist in the human body from several months to years after exposure. In the NHANES 1999-2000 subsample, the frequency of detection of the eight mono-ortho substituted PCBs ranged from 2 percent to 47 percent. Finding detectable amounts does not mean that those levels result in adverse health effects. (For additional information on PCBs in the environment, see Chapter 2, Purer Water; Chapter 3, Better Protected Land; and Chapter 5, Ecological Condition.)

Polychlorinated Dibenzo-p-Dioxins (Dioxins) and Polychlorinated Dibenzo-p-Furans (Furans)

Dioxins and furans are similar classes of chlorinated aromatic chemicals usually generated as pollutants or by-products. They have no commercial or natural use. Processes that result in their generation include the incineration of waste, the production of pulp and paper, and the synthesis of various manmade chemicals. Releases from industrial sources have decreased by approximately 80 percent since the 1980s. The largest releases of dioxins and furans today are the open burning of household and municipal trash, landfill fires, and agricultural and forest fires. In the environment, dioxins and furans occur as a mixture of about 20 congeners (i.e., closely related chemical compounds).

Human exposure occurs primarily through foods that are contaminated with dioxins and furans. Food contamination occurs due to the accumulation of these chemicals in the food chain and in high-fat foods, such as dairy products, eggs, animal fats, and some types of fish. People have also been exposed through industrial accidents, the burning of PCBs, and through the spraying of contaminated herbicides such as Agent Orange. Workplace exposures are rare and generally recognized standards for external exposure have not been established.

Human health effects associated with dioxins and furans are wide-ranging. Given that the exposure of the general population occurs as exposure to a mixture of congeners, the effects of individual congeners are difficult to determine. Overall, associated dioxin and

furan health effects include liver disorders, fetal injury, porphyria, elevated lipid levels, chloracne, hormonal changes, neurologic damage, and immunogic changes. The dioxin cogener termed TCDD is the most toxic form of dioxin and it is classified as a known human carcinogen.

It is estimated that human serum lipid-based levels of overall dioxins and furans have decreased by 80 percent since the 1980s and the low NHANES 1999-2000 values support that estimation. The levels detected via NHANES 1999-2000 are far below those associated with occupational and unintentional exposures that resulted in human health effects.

Further, the NHANES 1999-2000 subsample reveals that the more highly chlorinated dioxin and furan cogeners are the main contributors to the human body burden. The higher concentrations in human tissues of these cogeners are due to their greater presence in the food chain, resistance to metabolic breakdown, and greater solubility in body fat. Half-lives for all the dioxin and furan cogeners range from 3 to 19 years and TCDD is estimated to be 7 years.

4.4.8 What are the trends in exposure to environmental pollutants for children?

Children may be affected by environmental pollutants quite differently than adults, both because children may be more highly exposed to pollutants and because they may be more vulnerable to the toxic effects of pollutants. Children generally eat more food, drink more water, and breathe more air relative to their size than do adults, and consequently may be exposed to relatively higher amounts of pollutants. Also, unlike adults, children's normal activities, such as putting their hands in their months or playing on the ground, create greater opportunities for exposures to pollutants. In addition, environmental pollutants may affect children disproportionately because their organ systems are still developing and therefore may be more susceptible (EPA, December 2000). This section presents three environmental pollutants that represent exposures of concern to children: lead, mercury, and cotinine.

Indicator: Blood lead level in children – Category 1

Infants, children, and fetuses are more vulnerable to the effects of lead because the blood-brain barrier is not fully developed (Nadakavukaren, 2000). Thus, a smaller amount of lead will have a greater effect in children than in adults. In addition, ingested lead is more readily absorbed into a child's bloodstream. Children absorb 40 percent of ingested lead into the bloodstream, while adults absorb only 10 percent. Because of lead's adverse effects on cognitive development, CDC has defined an elevated blood lead level as equal to or greater than 10 µg/dL for children under 6 years of age (CDC, 2001c).

What the Data Show

In NHANES III (1988-1994), the mean blood lead levels for children ages 1 to 5 declined from 3.6 µg/dL in Phase 1 (1988 to 1991) to 2.7 µg/dL in Phase 2 (1991 to 1994). Over the same time interval, the percentage of children aged 1 to 5 years with elevated blood lead levels decreased from 8.6 percent to 4.4 percent (Pirkle, 1998). In NHANES 1999-2000, the geometric median blood lead level for children 1 to 5 years old is 2.2 µg/dL. The median blood lead level for children 6 to 11 years old is 1.5 µg/dL (see exhibit 4-8 in this chapter).

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-35, for more information.)

Indicator Blood mercury level in children - Category 1

Children may be more highly exposed to mercury and may be more vulnerable to its toxic effects. The health effects of mercury are diverse and can include developmental and neurological effects in children.

What the Data Show

Extremely limited information has been available about children's exposure to mercury and how it relates to levels in adults. Exhibit 4-38 shows that the geometric mean of blood mercury levels among U.S. children measured in NHANES 1999-2000 was 0.34 µg/L. The geometric mean of blood mercury levels of women of childbearing age was 1.02 µg/L. Levels among women of childbearing age are particularly important because they reflect levels of mercury to which the fetus is exposed (NRC, 2000). During a toxicological review of mercury levels, the National Research Council estimated a benchmark dose, which was an estimate of a methylmercury exposure to the fetus, associated with an increase in abnormal scores on cognitive function tests among children. The lower 95 percent confidence bound on the benchmark dose was 58 µg/L (NRC, 2000). To account for uncertainties in exposure measures and variability in individual response to toxic effects of mercury, the NRC recommended an uncertainty factor of 10 to calculate a reference dose. EPA published its final reference dose of 5.8 µg/L, agreeing with NRC. Ninety percent of children 1 to 5 years old and women of childbearing age are below this level (CDC, 2001c).

Exhibit 4-38: Geometric mean and selected percentiles of total blood mercury concentrations (in µg/L) for United States children aged 1-5 years, and women aged 16-49 years, National Health and Nutrition Examination Survey (NHANES), 1999-2000

			Selected Percentiles				
	Sample Size	Geometric Mean	10th	25th	50th	75th	90th
Children, aged 1-5 years, males and females	705	0.34	<LOD	<LOD	0.30	0.50	1.40
Females, 16-49 years	1,709	1.02	0.20	0.40	0.90	2.00	4.90

<LOD = below the limit of detection of the analytical method of 0.14 µg/dL blood.
Source: CDC. *Second National Report on Human Exposure to Environmental Chemicals.* 2003.

Data Source

NHANES III (1999), National Center for Health Statistics. (See Appendix B, page B-35, for more information.)

Indicator Blood cotinine level in children - Category 1

Children are at particular risk from ETS, which may exacerbate asthma among susceptible children and also greatly increase the risk for lower respiratory-tract illness, such as bronchitis and pneumonia, among young children (CDC, 2001c). NHANES 1999-2000 data show that people younger than 20 years have higher cotinine levels than people 20 years and older (CDC, 2003). (See Exhibit 4-35 located in Section 4.4.4.) Blood cotinine level is an indicator of exposure to ETS. During NHANES 1999-2000, the average blood cotinine level for children aged 3 to 11 years was 0.11 ng/mL. This level was the same for children in the 12 to 19 years subgroup (CDC, 2003).

For the general population, as part of NHANES III (1988-1991), CDC determined that the median serum level (50th percentile) of cotinine among non-smokers in the U.S. was 0.20 ng/mL. As determined during NHANES 1999-2000, the median cotinine level decreased to 0.059 ng/mL, a 70 percent decrease. This reduction suggest a marketed decrease in exposure of the general U.S. population to ETS since the 1988-1991 period. Further, compared with the results for the 1988-1991 period, NHANES 1999-2000 reveals that cotinine levels declined in each of the population groups defined by age, sex, and race/ethnicity (CDC, 2003).

Data Source

NHANES 1999-2000, National Center for Health Statistics. (See Appendix B, page B-36, for more information.)

4.4.9 Pollutants for Which Biomonitoring Data Are Not Available

As mentioned above, biomonitoring is an emerging field. More biomonitoring indicators are available now than a few years ago. Still, there are many environmental pollutants for which biomonitoring techniques are not available or feasible. These include radiation, air pollutants (except for lead), biological pollutants, and disinfection by-products. Biomonitoring efforts have begun recently for disinfection by-products; however, at this time data are not sufficient to develop indicators for these pollutants. All these pollutants are of concern because exposure is widespread. For these pollutants, exposure assessments currently rely primarily on ambient data.

What is the level of exposure to radiation?

Radiation is energy given off by atoms in the form of particles or electromagnetic rays. There are actually many different types of electromagnetic radiation that have a range of energy levels. They form the electromagnetic spectrum and include radio and micro waves, heat, light, and x-rays (EPA, 2002w).

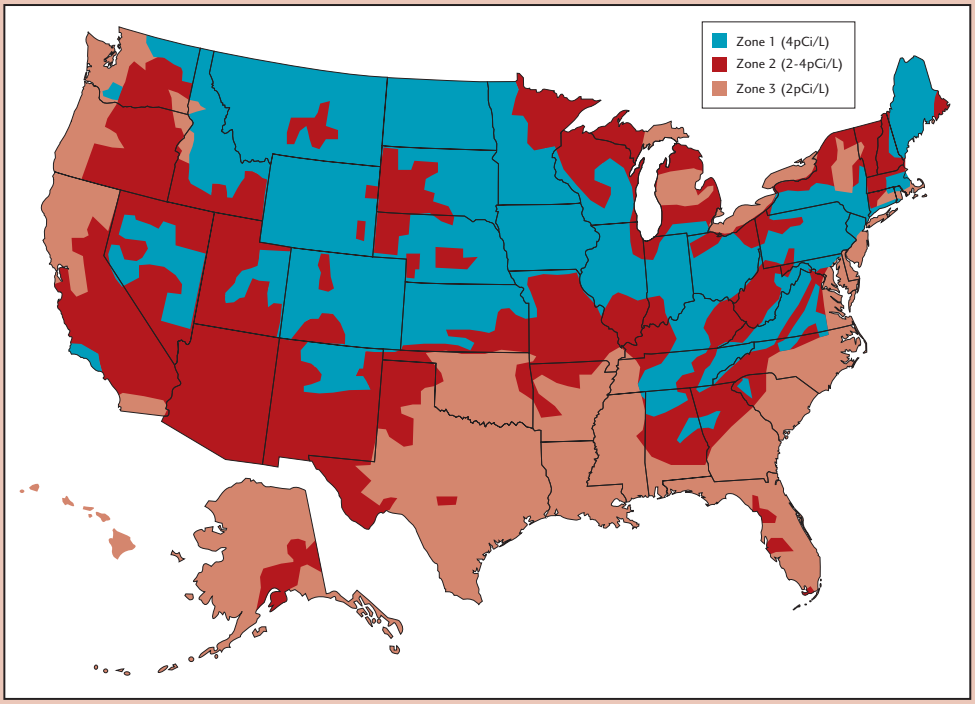
Radiation that has enough energy to move atoms in a molecule around or cause them to vibrate, but not enough to change them chemically, is referred to as "non-ionizing radiation." Examples of this kind of radiation are sound waves, visible light, and microwaves (EPA, 2002y). Non-ionizing radiation can be used for some common tasks, such as using microwave radiation for telecommunications and heating food, infrared radiation for producing warmth, and radio waves for broadcasting (EPA, 2002y). Non-ionizing radiation has relatively long wavelengths and low frequencies, in the range of 1 million to 10 billion Hertz (EPA, 2002y).

Radiation that has enough energy to actually break chemical bonds or strip electrons away from atoms is called "ionizing radiation (EPA, 2002x)." Radioactive materials that decay spontaneously produce ionizing radiation. Any living tissue in the human body can be damaged by ionizing radiation. The body attempts to repair the damage, but sometimes the damage is too severe or widespread, or mistakes are made in the natural repair process. The most common forms of ionizing radiation are alpha and beta particles, or gamma and X-rays (EPA, 2002x). Ionizing radiation has very short wavelengths, and very high frequencies, in the range of 100 billion billion Hertz (EPA, 2002y). This is the type of radiation that people usually think of as 'radiation.' Ionizing radiation can be used to generate electric power, to kill cancer cells, and in many manufacturing processes (EPA, 2002y).

Radiation can affect the body in a number of ways, and the adverse health consequences of exposure may not be seen for many years. These adverse health effects can range from mild effects, such as skin reddening, to serious effects such as cancer and death, depending on the amount of radiation absorbed by the body (the dose), the type of radiation, the route of exposure, and the length of time a person is exposed. Exposure to very large doses of radiation may cause death within a few days or months. Exposure to lower doses of radiation may lead to an increased risk of developing cancer or other adverse health effects (CDC, 2003).

There are three basic pathways for radiation exposure. These are inhalation, ingestion, and direct exposure. Each of the different routes, or pathways, by which people can be exposed to radiation result in exposure to different parts of the body (EPA, 2002z). Exposure by the inhalation

Exhibit 4-39: EPA map of radon zones in the United States



Source: Modified from EPA, Office of Air and Radiation. EPA Map of Radon Zones. April 2003. (May 2, 2003; *http://www.epa.gov/iaq/radon/zonemap.html*).

pathway occurs when people breathe radioactive materials into the lungs. The chief concerns are radioactively contaminated dust, smoke, or gaseous radionuclides such as radon (EPA, 2002z). Radon is a colorless, tasteless, and odorless gas that comes from the decay of uranium found in nearly all soils. Levels of radon vary throughout the country. Radon usually moves upward from the ground and migrates into homes and other buildings through cracks and other holes in their foundations. The buildings trap radon inside, where it accumulates and may become a health hazard if the building is not properly ventilated (EPA, June 2000; EPA, 2002b).

No biomonitoring data are feasible for national estimates of exposure to radon. Data for average national indoor and outdoor radon levels are available, but unlike biomonitoring data, these data do not represent the amount of radon found in human tissue. Rather, they are the levels of radon measured in the air. Radon levels vary throughout the U.S. Exhibit 4-39 shows the distribution of radon levels throughout the country (EPA, 2003d). Based on a national residential radon survey completed in 1991, the average indoor radon level is 1.3 picocuries per liter in the U.S. The average outdoor level is about 0.4 picocuries per liter (EPA, 2002b).

Radiation exposure by the ingestion pathway occurs when someone swallows radioactive materials. For example, exposure by ingestion can occur when drinking water becomes radioactively contaminated, or when food is grown in contaminated soil. Alpha and beta emitting radionuclides are of most concern for ingested radioactive materials. They release large amounts of energy directly to tissue, causing DNA and other cell damage (EPA, 2002z).

The third pathway of concern is direct or external exposure from radioactive material. The concern about exposure to different kinds of radiation varies by the particular type of particle or wave that is being emitted. Alpha particles cannot penetrate the outer layer of skin, but open wounds may pose a risk. Beta particles can burn the skin in some cases, or damage eyes. Greatest concern is about gamma radiation. Different radionuclides emit gamma rays of different strength, but gamma rays can travel long distances and penetrate entirely through the body. Gamma rays can be slowed by dense material (shielding), such as lead, and can be stopped if the material is thick enough. Examples of shielding are containers; protective clothing, such as a lead apron; and soil covering buried radioactive materials (EPA, 2002z).

Radiation can occur from man-made sources such as x-ray machines; or from natural sources such as the sun and outer space, and from some radioactive materials such as uranium in soil (CDC, 2003). About 80 percent of human exposure to radiation is from naturally occurring forms of radiation. The remaining 20 percent of exposure is to manmade radiation sources, primarily medical x-rays (CDC, 2003).

Radiation doses that people receive are measured in units called "rem (CDC, 2003)." Most people receive about 300 mrem every year from natural background sources of radiation, primarily radon. Health physicists generally agree on limiting a person's exposure beyond background radiation to about 100 millirem (mrem) per year from all sources. Exceptions are occupational, medical or accidental exposures. (Medical X-rays generally deliver less than 10 mrem). EPA and other regulatory agencies generally limit exposures from specific sources to the public to levels well under 100 mrem. This is far below the exposure levels that cause acute health effects (EPA, 2002x).

For additional information on radiation in the environment, see Chapter 1, Cleaner Air.

What is the level of exposure to air pollutants?

Criteria air pollutants are common air pollutants comprised of ozone, nitrogen dioxide, carbon monoxide, sulfur dioxide, lead, and particulate matter. The health effects associated with criteria air pollutants are discussed in Chapter 1, Cleaner Air, Section 1.1.3. Ozone is the result of a chemical reaction in the atmosphere between VOCs and nitrogen oxides. Nitrogen dioxide comes from the burning of gasoline, natural gas, coal, and oil. Cars are an important source of nitrogen dioxide.

Carbon monoxide comes from the burning of gasoline, natural gas, coal, and oil. Carbon monoxide reduces the ability of blood to bring oxygen to body cells and tissues. Carbon monoxide may be particularly hazardous to people who have heart or circulatory problems.

Particulate matter (PM) can be emitted directly into the atmosphere, such as dust from roads or elemental carbon (soot) from wood combustion. PM can also be formed in the atmosphere from primary gaseous emissions such as sulfur dioxide and nitrogen oxides, which come from power plants, industrial facilities, automobiles, and other types of combustion sources.

The primary source of sulfur dioxide is the burning of coal and oil, especially high-sulfur coal from the eastern U.S., and industrial processes (paper, metals). The primary source of lead in ambient air was leaded gasoline, which has been phased out in the U.S. Other sources of lead include paint, smelters, and the manufacture of lead storage batteries. Major health effects associated with lead are discussed in Section 4.1.

Except for lead, biomonitoring methods are not available or feasible for the remaining criteria air pollutants. Data for average national ambient air pollutant levels are available (see Chapter 1, Cleaner Air). Research on actual intake measures of air pollutants and their relationship to ambient levels as measured by monitoring networks is under way. Many other studies have found links between air

pollutants and disease, as noted in the discussion of diseases and their relationships to environmental pollutants (see Section 4.1).

What is the level of exposure to biological pollutants?

Biological pollutants are or were living organisms. In addition to arthropod-borne, foodborne, or waterborne disease discussed previously, other biological agents can promote poor indoor air quality and may be a major cause of days lost from work or school and of doctor and hospital visits. Some can even damage surfaces inside and outside the residence. Some common indoor biological pollutants include: animal dander (minute scales from hair, feathers, or skin); dust mite and cockroach parts, fungi (molds); infectious agents (bacteria or viruses); and pollen.

Everyone is exposed to biological pollutants. The effects on one's health, however, depend upon the type and amount of biological pollution and the individual person. Some people do not experience health reactions from certain biological pollutants, while others may experience one or more of the following reactions: allergic, infectious, or toxic.

Except for the spread of infections indoors, allergic reactions may be the most common health problem with indoor air quality in homes. They are often connected with animal dander (mostly from cats and dogs), with house dust mites (microscopic animals living in household dust), and with pollen. Allergic reactions can range from mildly uncomfortable to life-threatening, as in a severe asthma attack. Health experts are especially concerned about people with asthma, who have very sensitive airways that can react to various irritants, making breathing difficult. Infectious diseases caused by bacteria and viruses, such as flu, measles, chicken pox, and tuberculosis, may be spread indoors. Most infectious diseases pass from person to person through physical contact. Crowded conditions with poor air circulation can promote this spread. Some bacteria and viruses thrive in buildings and circulate through indoor ventilation systems. (For additional information on indoor air pollution, see Chapter 1, Cleaner Air.)

As with air pollutants and radiation, biomonitoring methods are not available or feasible for many of the biological pollutants discussed in this section.

What is the level of exposure to disinfection by-products?

Disinfection by-products (DBPs) are chemicals that form in drinking water when disinfectants are added during the drinking water treatment process. Disinfectants are added to drinking water to kill bacteria and other microbes that cause disease. DBPs are formed when the disinfectants react with organic matter (primarily from leaf and vegetation decay) that is found naturally in drinking water sources such as rivers and lakes (EPA, 2002c). The most common drinking water disinfectant is chlorine. Other lesser-used disinfectants include chloramines, chlorine dioxide, ozone, and ultraviolet light. More than 200 million people within the U.S. drink disinfected water (EPA, June 2001a).

Hundreds of different DBPs—most of which result from chlorine—have been identified in drinking water, and occurrence data have been reasonably established for over 30 DBPs (EPA, ORD, November 1997). The two types of DBPs that are typically measured by drinking water utilities are trihalomethanes (THMs) and haloacetic acids (HAs).

DBP levels vary throughout the country because the levels are dependent on several factors, including amount of organic matter in the drinking water source, amount of rainfall in the area, season of the year, water temperature, type of disinfectant used, water treatment plant configuration, and size of the water system distribution system (EPA, 1999).

Current information on DBP exposures draws on monitoring results from drinking water systems. Data for average national levels of THMs in treated drinking water are available. Water monitoring for DBPs is of limited value in classifying or identifying individual exposures to DBPs. Individual exposures are influenced by route of exposure (ingestion, inhalation, dermal absorption), individual habits relating to water use or consumption, time and spatial distribution of DBPs in the water system, and seasonal variables that affect the precursors to DBPs (e.g., rainfall, temperature). The complex nature of exposure to DBPs will require a better understanding of the chain of events as illustrated in Exhibit 4-1.

4.4.10 Endocrine Disruptors—An Emerging Issue

An endocrine disruptor is defined as an exogenous agent that alters the function of the endocrine system and consequently causes adverse health effects in an intact organism, or its progeny or (sub)populations (IPCS, 2002). A number of pharmaceuticals, pesticides, commercial chemicals and environmental contaminants are known to disrupt the endocrine system across a wide range of species—invertebrates, fish, birds, reptiles, and mammals.

There is little information on the magnitude and pattern of human exposures to endocrine disruptors. The limited exposure data that exist are primarily for various environmental media, such as chemical concentrations in air, food, and water. Often these data are limited by geographical regions and cannot be extrapolated to national trends. More relevant measures of human exposure, such as chemical concentrations in human blood, breast milk, and human tissue, are rare. Often these data are available only for high exposure areas and populations. As chemicals suspected of contributing to endocrine disruption in humans are identified, it will be necessary to obtain high-quality exposure data to perform human risk assessments. Each major state of the science report on endocrine disruptors has acknowledged the critical need for research to increase our understanding of human exposures and related health outcomes.

The human health issue regarding exposure to endocrine disruptors primarily relates to: (1) adverse effects observed in fish and wildlife, (2) the increased incidence of specific endocrine-related adverse human health outcomes/diseases, and (3) observations of endocrine disruption in well-conducted experiments involving laboratory animals. These chemicals can affect the endocrine system in several ways including interfering with hormone synthesis and release from the endocrine gland, competing with the hormone for the binding sites on transport proteins in the blood, binding to the receptor to either block hormone action or mimic it, and producing changes in hormone metabolism and elimination (IPCS, 2002).

There are a few clear examples of adverse human health effects following high exposures to environmental chemicals (e.g., accidental releases or poisoning incidents). Analysis of the human data by itself has not provided firm evidence of direct causal associations between low level exposure to endocrine-disrupting chemicals and adverse human health outcomes.

Of particular interest is exposure during very early development, both *in utero* and postnatally. Sexual differentiation, growth, and development are under hormonal control. Many of these early processes are unique to this time period and disruptions of carefully timed processes may lead to irreversible adverse human health outcomes. Interest has focused on: (1) adverse effects on reproductive and sexual development and function, (2) altered immune system, nervous system, and thyroid development and function, and (3) cancers of various endocrine-sensitive tissues including the testes, breast, and prostate. Additional research is needed to determine whether linkages exist between these adverse human health outcomes/diseases and exposure to suspected endocrine disruptors. However, this research is challenging as the manifestation of the condition is frequently not observed until years after exposure has occurred and the measured concentration of the chemicals in the affected adult may be very different from in utero, neonatal, or pre-pubertal exposures/concentrations that may have given rise to the adverse outcome.

4.5 Assessing the Environmental Burden of Disease

Many factors may cause or influence disease in humans. These factors include heredity, social factors, dietary factors, and environmental factors (e.g., chemical pollutants, infectious microorganisms, and radiation). The extent to which environmental factors influence overall disease is not entirely understood. Disease burden, global burden of disease, and environmental burden of disease are concepts used to express the burden of disease on society:

- **Disease burden** is the effect on society of both disease-related mortality and disease-related morbidity (Kay, 2000; WHO, 2002). It is assessed by several health measures, including mortality rates, morbidity rates, and the number of days in the hospital. Historically, disease burden has been investigated by analyzing disease outcomes, such as cancer, rather than analyzing risk factors that may cause cancer or disease in general. For example, it is easier to compare cancer incidence between two countries than to compare risk factors of cancer; ionizing radiation may be the major risk factor for cancer in country A, while dioxin may be the major risk factor in country B.
- **Global burden of disease (GBD)** assesses the disease burden on a worldwide basis and then apportions that burden to various causes, such as genetic, behavioral, and environmental.
- **Environmental burden of disease (EBD)** measures that portion of the GBD which is due solely to environmental risk factors.

EBD provides a method for summarizing the environmental health of populations. The summary health data collected from EBD measurements help identify environmental risk factors with significant public health implications. EBD data can also be used to help prioritize funding allocations for health and environmental research, assist in environmental policy development, justify environmental advocacy, assess the cost-effectiveness of interventions, and monitor the progress of a population's health (Prüss, et al., 2001). More important, EBD provides a way to normalize risk factors, allowing comparable health evaluations between populations. Two approaches are commonly used to determine the degree of disease burden that stems from environmental risk factors:

- The **outcome-based approach** determines the degree to which specific environmental risk factors cause a disease relative to other environmental risk factors.

- The **exposure-based approach** assesses the adverse health outcomes resulting from dose-response relationships between risk factors and associated disease outcomes (Prüss, et al., 2001).

This section summarizes estimates, in different studies, of the environmental burden of disease.

World Health Organization Evaluation

In 1998, WHO estimated that 23 percent of GBD is due to environmental hazards, including occupational exposures (WRI, et al., 1998). In 1999, WHO researchers and researchers from the University of California reported that an estimated 25 to 30 percent of the GBD was attributable to the environment (Smith, et al., 1999).

In 2000, WHO introduced a new methodology for evaluating changes to EBD, termed comparative risk assessment (CRA). CRA measures the GBD due to risk factors. WHO is currently developing CRA guidelines to help countries and smaller population groups, such as villages and towns, measure their respective EBD (Kay, 2000). CRA does not have one standard unit, however, and it incorporates other methodologies used to assess EBD. Because of this variability in assessment methodologies, comparing EBD for different countries can be difficult. Further, because EBD has not been quantified extensively in the U.S., this country's level of EBD cannot be easily compared with that of the rest of the world.

Doll and Peto Estimates

Richard Doll and Richard Peto quantified the environmental contribution to disease in their 1981 landmark study *The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today.* In that study, they concluded that pollutants in air, water, and food contributed from 2 to 5 percent to cancer mortality (Doll and Peto, 1981). They quantified the portion of cancer deaths that were attributable to various environmental causes, excluding tobacco smoke (Exhibit 4-40). Thirty percent of cancer was ascribed to tobacco use.

Other Estimates

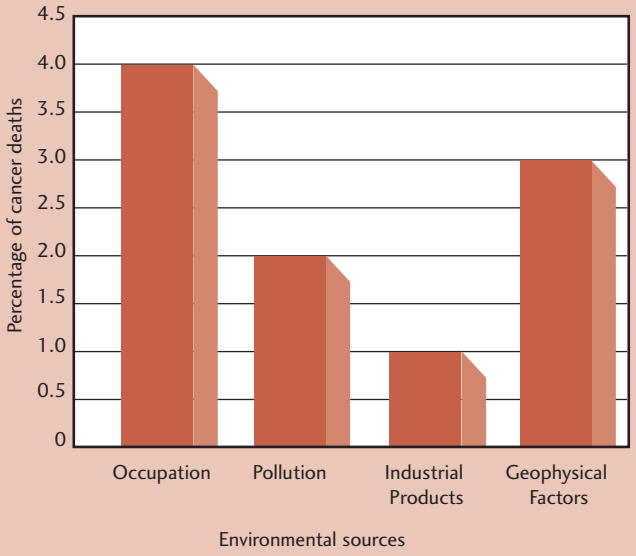
Other studies of EBD have investigated specific environmental risk factors and disease outcomes. For example, Wynder and Gori concluded in 1972 that environmental factors caused 12 percent of all cancer cases for men and 14 percent for women in the U.S. (Doll and Peto, 1981).

Why EBD Estimates Differ

EBD estimates are affected by the definition of "environment" that is used in making the determination (Smith, et al., 1999), as well as the measurement unit used, such as reporting mortality as a percentage of the population. For example, some researchers include factors such as stress or injury as environmental causes of disease, while others include stress and injury as social causes of disease.

The quantity of disease burden (such as disease outcome or risk factors) measured in EBD studies also produces variation in EBD estimates. These differences can be attributed to the different ways that risk factors are categorized, or to differences in the amount of disease burden attributed to a particular source.

Exhibit 4-40: Estimated proportions of cancer deaths in the United States attributed to selected environmental factors



Note: Tobacco is not included in this table. There was not a distinction between environmental tobacco smoke and mainstream smoke.

Source: Doll, Richard and Richard Peto. *The Causes of Cancer: Quanitative Estimates of Avoidable Risks of Cancer in the United States Today.* 1981.

4.6 Challenges and Data Gaps

This chapter described key indicators for health and exposure. Many exposure indicators presented were measured by biomonitoring. Where biomonitoring data are not available, ambient exposure measures serve to describe human exposure to key environmental pollutants. Areas where strong associations have been demonstrated between environmental exposures and health outcomes were highlighted. However, in many areas those associations have not yet been demonstrated.

The success of environmental decisions in improving public health can be measured on a variety of levels:

- **National level** (e.g., U.S. Department of Health and Human Services' Healthy People 2010 initiative).
- **Geographic/regional level** (e.g., East Coast versus West Coast, CDC's state health reports).
- **Community level** (e.g., air and water quality monitoring).
- **Individual level** (e.g., screening programs for blood lead in children).

Many indicators may be used at a number or all of these levels. This report has focused on describing indicators and impacts at a national level. Future versions of this report may utilize indicators to evaluate success in reducing environmental health exposure and outcomes at some of the other levels as well.

Use of Health Outcome Measures to Evaluate Environmental Policy Decisions or Interventions

Mortality data were chosen as one of the major disease indicators because these are collected nationwide in every state, county, and community. These mortality data constitute a comprehensive database, since every death is presumed to be reported. This information has been collected for more than the past 50 years and has been used to document the success of major public health programs. For example, treatment of drinking water through filtration or chlorination eliminated diarrhea diseases as a major cause of death in the 20th century. More recently, anti-smoking campaigns aimed at men are believed to be responsible for the sudden downward trend in deaths due to lung cancer. In fact, an analysis of the key indicators of health for the country confirm that the health of the U.S. population is improving. The U.S. population is living longer (life expectancy) and death rates for major causes of death (cancer, cardiovascular disease) are declining. Except for those rare diseases that have a short survival period and 100 percent death rate, death represents only a small fraction of the true number of cases for a disease in the population (see Section 4.2).

Better information and insight into the health of the U.S. population can be obtained from evaluating incidence data (new cases of illness) or prevalence data (all existing cases of illness). At this time, no comprehensive nationwide systems for collecting incidence or prevalence data on disease are in place. The majority of morbidity data reported in this chapter are available either from national surveys that sample the U.S. and are assumed to be representative of the nation, or from data (e.g., birth defects and cancer registries) collected by the state-based centers around the country. The actual picture of health may differ from that suggested by the data, as in the case of childhood asthma prevalence that has been rising (as described in Section 4.3.4). CDC has launched an initiative to improve the nation's health tracking system. CDC recently awarded grants to state and local health departments to begin developing a national environmental health tracking network and to develop capacity in monitoring environmental health at the state and local levels (<http://www.cdc.gov/nceh/tracking/EPHTracking/EPHTracking.htm>).

Several emerging areas of health concern (e.g., Parkinson's disease, diabetes) and emerging areas of environmental exposure (e.g., endocrine disrupters) were recognized in this chapter. In many of these areas, either the link between environmental exposures and the disease has not been established or no systematic surveillance or established indicators currently exist. Future reports may well include many of the diseases and exposures identified as emerging issues and may establish associated indicators. Major efforts to address diabetes, asthma, and obesity also present a very promising opportunity to incorporate research on the role of environmental exposures into such plans.

Use of Exposure Measures to Evaluate Environmental Policy Decisions or Interventions

Most exposure indicators described in this chapter were biomonitoring indicators. Ambient exposure measures were described for a number of areas where, at present, biomonitoring data are not available (e.g., for certain air pollutants where there are no markers in blood or urine).

The NHANES data provide examples where biomonitoring data have reflected a public health benefit from EPA actions. For example, the decline in blood lead levels confirms that the removal of lead from gasoline, water, and paint has successfully reduced exposures. Similarly, the decline in urinary cotinine levels demonstrates that efforts to reduce smoking have led to public health improvements. However, interpretation of many of the other exposure indicators is difficult at this time. Either not enough is known about the exposure levels in the population, or data gathering at a national level has just begun. It will take time for a stable reference base to emerge.

Efforts to establish a national reference base are under way through the work of CDC's National Center for Environmental Health, which is developing the National Human Exposure to Environmental Chemicals Report. The first report was released in 2001 (<http://www.cdc.gov/nceh/dls/report/PDF/CompleteReport.pdf>) and a second one was released in January 2003 with data on 116 chemicals (<http://www.cdc.gov/humanexposure>). CDC is committed to expanding this database, and its recent *Federal Register* notice called for nominations of chemicals to consider for inclusion in the third report, to be published in 2005. The report will fill a critical need to describe exposure. Use of the report indicators for explanatory or predictive functions will require an understanding of pathways and sources that may have contributed to the exposure and the exposure's relationships to health effects. With this additional understanding the report ultimately could be used to guide exposure reduction programs.

Monitoring Environmental Health Status at the Community Level

Except for mortality data, many communities must look to their own local public health officials to monitor the health status of their community. This is true for a number of reasons, including:

- Current health surveys have limited application at the community level and often require extrapolation from a larger population (state or national).
- Current disease reporting systems, whether national sample or reporting systems (e.g., National Notifiable Diseases Reporting System), can rarely provide an answer for a specific community.
- Biomonitoring surveys that apply to specific communities are extremely rare. For example, blood lead screening programs, while common across the country, do not report in a systematic fashion to a centralized location for compilation and analysis of the data.

Until such systems are developed, communities will continue to rely on environmental monitoring programs to tell them about their exposure to air or water pollution. EPA is pursuing a number of activities to increase the capacity of information providers (e.g., states) and users (e.g., communities) to share information. This effort includes working closely with other federal agencies, such as CDC, to build compatible systems for linking health and environmental data bases. One potential outcome of such partnerships is an opportunity to revisit and refine current sampling designs such that future data collection efforts would provide better information for smaller units (community level) and would ensure better temporal and spatial congruence between environmental, biomonitoring, and surveillance programs.

Future Challenges

For EPA to make better use of more direct indicators of public health outcomes, the science underlying the Agency's key public health functions (describe, explain, predict, evaluate) will need to be strengthened. EPA will continue to work on providing a better understanding of the components of the source-dose-health continuum (Exhibit 4-1). Key among them will be establishing the necessary degree of predictive validity between indicators of each component (e.g., exposure versus dose). Such an understanding is critical to defining the degree to which one indicator can be successfully used as a surrogate for another. However, this may not be conducive to widespread use in surveys or may be difficult to ascertain in smaller populations (e.g., at a community level).

EPA also will continue to build collaborations with CDC and other federal agencies responsible for collecting health surveillance and human exposure data. Such partnerships are essential to any effort to describe the status and trends of exposure and disease in the U.S. with the eventual goal of every U.S. citizen understanding what the status is for his or her family and community. An important initiative along these lines is the interagency effort to develop the National Children's Study, in which EPA is a collaborator. The Children's Health Act of 2000 authorized the National Institutes of Child Health and Disease and a consortium of federal agencies "to conduct a national longitudinal study of environmental influences on children's health and development." The study will investigate the interaction of biologic, genetic, social, and environmental factors to better understand their role(s) in children's health.

EPA will also seek to develop and evaluate methodologies for understanding the contribution of other risk factors to a given health condition in comparison to the environmental exposure (i.e., partitioning out the risk attributable to the environmental exposure[s] of concern). Such measures will assist in prioritizing intervention/prevention programs and will allow the benefits and cost of environmental management to be placed in the context of the larger public health picture.

Other issues of emerging, or emerged, concern include:

- **Susceptible populations.** This chapter identified children as a susceptible population and described indicators relating specifically to them. EPA also recently announced an initiative to define the environmental risks associated with the ever-increasing aging population (<http://www.epa.gov/epahome/headline_103002.htm>) to be undertaken in partnership with other federal agencies and the many alliances for the aging. Many of the indicators in this report are particularly relevant to the elderly (e.g., cardiovascular disease, chronic obstructive pulmonary disease), and they are, or can be, reported by age group. As other susceptible populations are identified, EPA will need to continue working with its federal partners to see that the data are collected and analyzed to track those populations.

- **Aggregate and cumulative risks.** Individuals are not exposed to single chemicals, but rather to multiple pollutants and other stressors through multiple pathways and routes over the course of a day. The reality of aggregate and cumulative exposures further complicates attempts to attribute risk to a single environmental agent. EPA has begun to look at this issue, stimulated in part by mandates under the Food Quality Protection Act. The recently released Cumulative Risk Guidance report (EPA, 2003e) lays the groundwork for taking on this challenge and will help target the research to better understand the nature and impact of such "composite" exposures, especially as related to targeting regulatory and health prevention strategies.

Finally, the health and exposure indicators described in this chapter are only a portion of the story on the state of the environment. These indicators should be viewed in conjunction with the other indicators identified in the companion chapters on ecological condition, land, air, and water. As presented in Exhibit 4-1, that integration is vital to fully developing the understanding envisioned by the cascade of events from source to effects.