CHAPTER 10

# Environmental Health, Pollution, and Toxicology

Houston,Texas, Ship Channel, where many oil refineries are located.

## LEARNING OBJECTIVES

Serious health problems may arise from toxic substances in water, air, soil, and even the rocks on which we build our homes. After reading this chapter, you should understand . . .

- How the terms *toxin, pollution, contamination, carcinogen, synergism*, and *biomagnification* are used in environmental health;
- What the classifications and characteristics are of major groups of pollutants in environmental toxicology;
- Why there is controversy and concern about synthetic organic compounds, such as dioxin;
- Whether we should be concerned about exposure to human-produced electromagnetic fields;
- What the dose-response concept is, and how it relates to LD-50, TD-50, ED-50, ecological gradients, and tolerance;
- How the process of biomagnification works, and why it is important in toxicology;
- Why the threshold effects of environmental toxins are important;
- What the process of risk assessment in toxicology is, and why such processes are often difficult and controversial.

CASE STUDY

# Toxic Air Pollution and Human Health: Story of a Southeast Houston Neighborhood

Manchester is a neighborhood in southeast Houston, Texas, that is nearly surrounded by oil refineries and petrochemical plants. Residents and others have long noted the peculiar and not so pleasant smells of the area, but only recently have health concerns been raised. The neighborhood is close to downtown Houston, and the houses are relatively inexpensive, the streets safe. It was a generally positive neighborhood except for the occasional complaints about nosebleeds, coughing, and acidic smoke smells. Over a period of years, the number of oil refineries, petrochemical plants, and waste-disposal sites grew along what is known as the Houston Ship Channel (see opening photograph).[1]

Cancer is the second leading cause of death of U.S. children who are not linked to a known health risk before being stricken by the disease. Investigations into childhood cancer from air pollution are few in number but now include exposure to benzene and 1,3-butadiene, commonly referred to simply as butadiene.[1]

Benzene is a colorless toxic liquid that evaporates into the air. Exposure to benzene has a whole spectrum of possible consequences for people, such as drowsiness, dizziness, and headaches; irritation to eyes, skin, and respiratory tract; and loss of consciousness at high levels of exposure. Long-term (chronic) exposure through inhalation can cause blood disorders, including reduced numbers of red blood cells (anemia), in industrial settings. Inhalation has reportedly resulted in reproductive problems for women and, in tests on animals, adverse effects on the developing fetus. In humans, occupational exposure to benzene is linked to increased incidence of leukemia (a cancer of the tissues that form white blood cells). The many potential sources of exposure to benzene include tobacco smoke and evaporating gasoline at service stations. Of particular concern are industrial sources; for example, the chemical is released when gasoline is refined from oil.

The chemical 1,3-butadiene is a colorless gas with a mild gasoline-like odor. One way it is produced is as a by-product of refining oil. Health effects from this toxin are fairly well known and include both acute and chronic problems. Some of the acute problems are irritation of the eyes, throat, nose, and lungs. Possible chronic health effects of exposure to 1,3-butadiene include cancer, disorders of the central nervous system, damage to kidneys and liver, birth defects, fatigue, lowered blood pressure, headache, nausea, and cancer.[1,2] While there is controversy as to whether exposure to 1,3-butadiene causes cancer in people, more definitive studies of animals (rats and mice) exposed to the toxin have prompted the Environmental Protection Agency to classify 1,3-butadiene as a known human carcinogen.[1,2]

Solving problems related to air toxins in the Houston area has not been easy. First of all, the petrochemical facilities along the Houston Ship Channel were first established decades ago, during World War II, when the area was nearly unpopulated; since then, communities such as Manchester have grown up near the facilities. Second, the chemical plants at present are not breaking state or federal pollution laws. Texas is one of the states that have not established air standards for toxins emitted by the petrochemical industry. Advocates of clean air argue that the chemical industry doesn't own the air and doesn't have the right to contaminate it. People in the petrochemical industry say they are voluntarily reducing emissions of some of the chemicals known to cause cancer. Butadiene emissions have in fact decreased significantly in the last several years, but this is not much comfort to parents who believe their child contracted leukemia as a result of exposure to air toxins. Some people examining the air toxins released along Houston's Ship Channel have concluded that although further reducing emissions would be expensive, we have the technology to do it. Petrochemical companies are taking steps to reduce the emissions and the potential health risks associated with them, but more may be necessary.

A recent study set out to study neighborhoods (census tracts near the Ship Channel) with the highest levels of benzene and 1,3-butadiene in the air and evaluate whether these neighborhoods had a higher incidence of childhood lymphohematopoietic cancer. After adjusting for sex, ethnicity, and socioeconomic status, the study found that census tracts with the highest exposure to benzene had higher rates of leukemia.[1] The study concluded that elevated exposure to benzene and 1,3-butadiene may contribute to increased rates of childhood leukemia, but the possible link between the air pollution and disease needs further exploration.

The case history of the Houston Ship Channel, oil refineries, and disease is a complex problem for several reasons:

1. Disease seldom has a one-cause/one-effect relationship.
2. Data on air-pollution exposure are difficult to collect and link to a population of people who are moving around and have different responses to exposure to chemicals.
3. It is difficult to definitively link health problems to toxic air pollutants.
4. There have been few other studies with which the Houston study can be compared.

In this chapter we will explore selected aspects of exposure to toxins in the environment and real and potential health consequences to people and ecosystems.

## 10.1 Some Basics

As members of Earth's biological community, humans have a place in the biosphere—dependent on complex interrelations among the biosphere, atmosphere, hydrosphere, and lithosphere. We are only beginning to inquire into and gain a basic understanding of the total range of environmental factors that affect our health and well-being. As we continue our exploration of minute quantities of elements in soil, rocks, water, and air in relation to regional and global patterns of climate and earth science, we are making important discoveries about how these factors influence death rates and the incidence of disease. Incidence of a particular disease varies significantly from one area to another,[3,4] and some of the variability is the result of geologic, hydrologic, biologic, and chemical factors linked to Earth's climate system.

**Disease**—impairment of an individual's well-being and ability to function—is often due to poor adjustment between the individual and the environment. Disease occurs on a continuum—between a state of health and a state of disease is a *gray zone* of suboptimal health, a state of imbalance. In the gray zone, a person may not be diagnosed with a specific disease but may not be healthy.[5] There are many gray zones in environmental health, such as the many possible states of suboptimal health resulting from exposure to man-made chemicals, including pesticides; food additives, such as coloring, preservatives, and artificial saturated fat, some of which alter the chemical structure of food; exposure to tobacco smoke; exposure to air pollutants, such as ozone; exposure to chemicals in gasoline and in many household cleaners; and exposure to heavy metals, such as mercury or lead. As a result of exposure to chemicals in the environment from human activity, we may be in the midst of an epidemic of chronic disease that is unprecedented in human history.[5]

As noted in the opening case study, disease seldom has a one-cause/one-effect relationship with the environment. Rather, the incidence of a disease depends on several factors, including the physical environment, biological environment, and lifestyle. Linkages between these factors are often related to other factors, such as local customs and the level of industrialization. More primitive societies that live directly off the local environment are usually plagued by different environmental health problems than those in an urban society. For example, industrial societies have nearly eliminated such diseases as cholera, dysentery, and typhoid.

People are often surprised to learn that the water we drink, the air we breathe, the soil in which we grow crops, and the rocks on which we build our homes and workplaces may affect our chances of experiencing serious health problems and diseases (although, as suggested, direct relationships between the environment and disease are difficult to establish). At the same time, the environmental factors that contribute to disease—soil, rocks, water, and air—can also influence our chances of living longer, more productive lives.

Many people believe that soil, water, and air in a so-called natural state must be good, and that if human activities have changed or modified them, they have become contaminated, polluted, and therefore bad.[6] This is by no means the entire story; many natural processes—including dust storms, floods, and volcanic processes—can introduce materials harmful to people and other living things into the soil, water, and air.

A tragic example occurred on the night of August 21, 1986, when there was a massive natural release of carbon dioxide ($CO_2$) gas from Lake Nyos in Cameroon, Africa. The carbon dioxide was probably initially released from volcanic vents at the bottom of the lake and accumulated there over time. Pressure of the overlying lake water normally kept the dissolved gas down at the bottom, but the water was evidently agitated by a slide or small earthquake, and the bottom water moved upward. When the $CO_2$ gas reached the surface of the lake, it was released quickly into the air. But because $CO_2$ gas is heavier than air, it flowed downhill from the lake and settled in nearby villages, killing many animals and more than 1,800 people by asphyxiation (Figure 10.1).

It was estimated that a similar event could recur within about 20 years, assuming that carbon dioxide continued to be released at the bottom of the lake.[7] Fortunately, a hazard-reduction project funded by the U.S. Office of Foreign Disaster Assistance (scheduled to be completed early in the 21st century) includes inserting pipes into the

(a)

(b)

**FIGURE 10.1** **(a)** In 1986, Lake Nyos in Cameroon, Africa, released carbon dioxide that moved down the slopes of the hills to settle in low places, asphyxiating animals and people. **(b).** Animals asphyxiated by carbon dioxide.

bottom of Lake Nyos, then pumping the gas-rich water to the surface, where the $CO_2$ gas is safely discharged into the atmosphere. In 2001, a warning system was installed, and one degassing pipe released a little more $CO_2$ than was seeping naturally into the lake. Recent data suggest that the single pipe now there barely keeps ahead of the $CO_2$ that continues to enter the bottom, so the lake's 500,000 tons of built-up gas have dropped only 6%. At this rate, it could take 30 to 50 years to make Lake Nyos safe. In the meantime, there could be another eruption.[8]

## Terminology

What do we mean when we use the terms *pollution, contamination, toxin,* and *carcinogen?* A polluted environment is one that is impure, dirty, or otherwise unclean. The term **pollution** refers to an unwanted change in the environment caused by the introduction of harmful materials or the production of harmful conditions (heat, cold, sound). **Contamination** has a meaning similar to that of *pollution* and implies making something unfit for a particular use through the introduction of undesirable materials—for example, the contamination of water by hazardous waste. The term **toxin** refers to substances (pollutants) that are poisonous to living things. **Toxicology** is the science that studies toxins or suspected toxins, and toxicologists are scientists in this field. A **carcinogen** is a toxin that increases the risk of cancer. Carcinogens are among the most feared and regulated toxins in our society.

An important concept in considering pollution problems is **synergism**, the interaction of different substances, resulting in a total effect that is greater than the sum of the effects of the separate substances. For example, both sulfur dioxide ($SO_2$) and coal dust particulates are air pollutants. Either one taken separately may cause adverse health effects, but when they combine, as when $SO_2$ adheres to the coal dust, the dust with $SO_2$ is inhaled deeper than $SO_2$ alone and causes greater damage to lungs. Another aspect of synergistic effects is that the body may be more sensitive to a toxin if it is simultaneously subjected to other toxins.

Pollutants are commonly introduced into the environment by way of **point sources**, such as smokestacks (see A Closer Look 10.1), pipes discharging into waterways, a small stream entering the ocean (Figure 10.2), or accidental spills. **Area sources**, also called *nonpoint sources*, are more diffused over the land and include urban runoff and **mobile sources**, such as automobile exhaust. Area sources are difficult to isolate and correct because the problem is often widely dispersed over a region, as in agricultural runoff that contains pesticides.

**FIGURE 10.2** This southern California urban stream flows into the Pacific Ocean at a coastal park. The stream water often carries high counts of fecal coliform bacteria. As a result, the stream is a point source of pollution for the beach, which is sometimes closed to swimming following runoff events.

## Measuring the Amount of Pollution

How the amount or concentration of a particular pollutant or toxin present in the environment is reported varies widely. The amount of treated wastewater entering Santa Monica Bay in the Los Angeles area is a big number, reported in millions of gallons per day. Emission of nitrogen and sulfur oxides into the air is also a big number, reported in millions of tons per year. Small amounts of pollutants or toxins in the environment, such as pesticides, are reported in units as parts per million (ppm) or parts per billion (ppb). It is important to keep in mind that the concentration in ppm or ppb may be by volume, mass, or weight. In some toxicology studies, the units used are milligrams of toxin per kilogram of body mass (1 mg/kg is equal to 1 ppm). Concentration may also be recorded as a percentage. For example, 100 ppm (100 mg/kg) is equal to 0.01%. (How many ppm are equal to 1%?)

When dealing with water pollution, units of concentration for a pollutant may be milligrams per liter (mg/L) or micrograms per liter (μg/L). A milligram is one-thousandth of a gram, and a microgram is one-millionth of a gram. For water pollutants that do not cause significant change in the density of water (1 $g/cm^3$), a pollutant concentration of 1 mg/L is approximately equivalent to 1 ppm. Air pollutants are commonly measured in units such as micrograms of pollutant per cubic meter of air ($\mu g/m^3$).

Units such as ppm, ppb, or $\mu g/m^3$ reflect very small concentrations. For example, if you were to use 3 g (one-tenth of an ounce) of salt to season popcorn in order to have salt at a concentration of 1 ppm by weight of the popcorn, you would have to pop approximately 3 metric tons of kernels!

# 10.2 Categories of Pollutants

A partial classification of pollutants by arbitrary categories is presented below. We discuss examples of other pollutants in other parts of the book.

## Infectious Agents

Infectious diseases—spread by the interactions between individuals and by the food, water, air, soil, and animals we come in contact with—constitute some of the oldest health problems that people face. Today, infectious diseases have the potential to pose rapid threats, both local and global, by spreading in hours via airplane travelers. Terrorist activity may also spread diseases. Inhalation anthrax caused by a bacterium sent in powdered form in envelopes through the mail killed several people in 2001. New diseases are emerging, and previous ones may emerge again. Although we have cured many diseases, we have no known reliable vaccines for others, such as HIV, hantavirus, and dengue fever.

The H1N1 flu pandemic (widespread outbreak of a disease) that became apparent in 2009 started in Mexico and has spread around the world. The complete origin of H1N1 remains unknown, but it has genetic markers of two swine flues, a human flu, and an avian (bird) flu. As we live closer together, nearer large numbers of animals such as chickens and pigs in large industrial farms and tightly confined animals in smaller farms, the probability of a disease crossing from animals to humans increases. People working closely with pigs have an increased risk of contracting swine flu.

## A CLOSER LOOK 10.1

### Sudbury Smelters: A Point Source

A famous example of a point source of pollution is provided by the smelters that refine nickel and copper ores at Sudbury, Ontario. Sudbury contains one of the world's major nickel and copper ore deposits. A number of mines, smelters, and refineries lie within a small area. The smelter stacks used to release large amounts of particulates containing toxic metals—including arsenic, chromium, copper, nickel, and lead—into the atmosphere, much of which was then deposited locally in the soil. In addition, because the areas contained a high percentage of sulfur, the emissions included large amounts of sulfur dioxide ($SO_2$). During its peak output in the 1960s, this complex was the largest single source of $SO_2$ emissions in North America, emitting 2 million metric tons per year.

As a result of the pollution, nickel contaminated soils up to 50 km (about 31 mi) from the stacks. The forests that once surrounded Sudbury were devastated by decades of acid rain (produced from $SO_2$ emissions) and the deposition of particulates containing heavy metals. An area of approximately 250 $km^2$ (96 $mi^2$) was nearly devoid of vegetation, and damage to forests in the region has been visible over an area

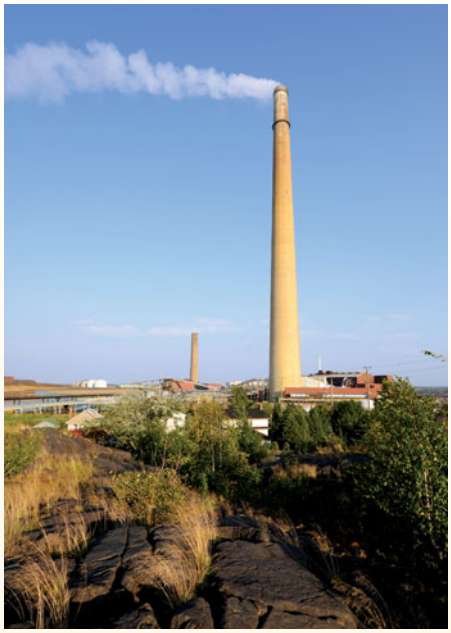
(a)   (b)

**FIGURE 10.3** **(a) Lake St. Charles, Sudbury, Ontario, prior to restoration**. Note high stacks (smelters) in the background and lack of vegetation in the foreground, resulting from air pollution (acid and heavy-metal deposition). **(b) Recent photo showing regrowth and restoration**.

of approximately 3,500 $km^2$ (1,350 $mi^2$); see Figure 10.3a. To control emissions from Sudbury, the Ontario government set standards to reduce emissions to less than 365,000 tons per year by 1994. The goal was achieved by reducing production from the smelters and by treating the emissions to reduce pollution.[9]

Reducing emissions from Sudbury has allowed surrounding areas to recover from the pollution (Figure 10.3b). Species of trees once eradicated from some areas have begun to grow again. Recent restoration efforts have included planting over 7 million trees and 75 species of herbs, moss, and lichens—all of which have contributed to the increase of biodiversity. Lakes damaged by acid precipitation in the area are rebounding and now support populations of plankton and fish.[9]

The case of the Sudbury smelters provides a positive example of emphasizing the key theme of thinking globally but acting locally to reduce air pollution. It also illustrates the theme of science and values: Scientists and engineers can design pollution-abatement equipment, but spending the money to purchase the equipment reflects what value we place on clean air.

## Environmentally Transmitted Infectious Disease

Diseases that can be controlled by manipulating the environment, such as by improving sanitation or treating water, are classified as environmental health concerns. Although there is great concern about the toxins and carcinogens produced in industrial society today, the greatest mortality in developing countries is caused by environmentally transmitted infectious disease. In the United States, thousands of cases of waterborne illness and food poisoning occur each year. These diseases can be spread by people; by mosquitoes and fleas; or by contact with contaminated food, water, or soil. They can also be transmitted through ventilation systems in buildings. The following are some examples of environmentally transmitted infectious diseases:

- Legionellosis, or Legionnaires' disease, which often occurs where air-conditioning systems have been contaminated by disease-causing organisms.
- Giardiasis, a protozoan infection of the small intestine, spread via food, water, or person-to-person contact.
- Salmonella, a food-poisoning bacterial infection that is spread via water or food.
- Malaria, a protozoan infection transmitted by mosquitoes.
- Lyme borreliosis (Lyme disease), transmitted by ticks.
- Cryptosporidiosis, a protozoan infection transmitted via water or person-to-person contact (see Chapter 19).[10]
- Anthrax, spread by terrorist activity.

We sometimes hear about epidemics in developing nations. An example is the highly contagious Ebola virus in Africa, which causes external and internal bleeding and kills 80% of those infected. We may tend to think of such epidemics as problems only for developing nations, but this may give us a false sense of security. True, monkeys and bats spread Ebola, but the origin of the virus in the tropical forest remains unknown. Developed countries, where outbreaks may occur in the future, must learn from the developing countries' experiences. To accomplish this and avoid potential global tragedies, more funds must be provided for the study of infectious diseases in developing countries.

## Toxic Heavy Metals

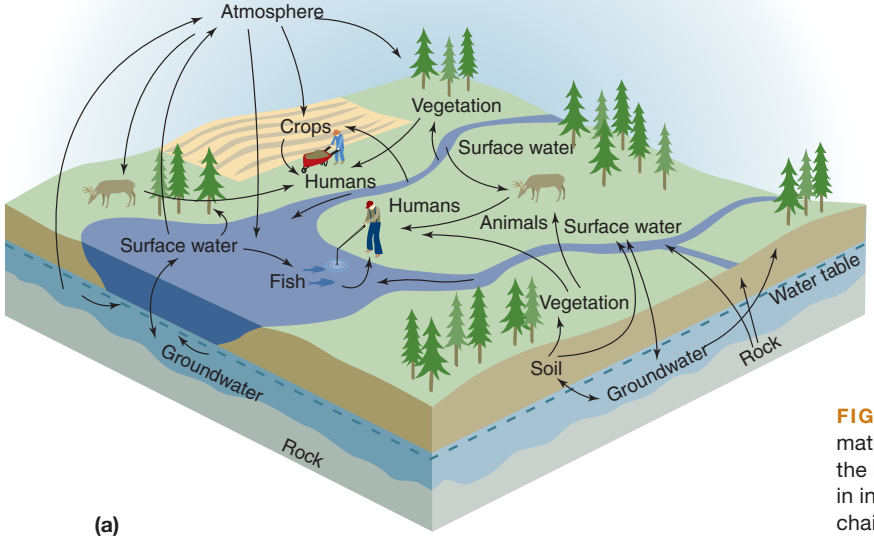
The major **heavy metals** (metals with relatively high atomic weight; see Chapter 6) that pose health hazards to people and ecosystems include mercury, lead, cadmium, nickel, gold, platinum, silver, bismuth, arsenic, selenium, vanadium, chromium, and thallium. Each of these elements may be found in soil or water not contaminated by people, each has uses in our modern industrial society, and each is also a by-product of the mining, refining, and use of other elements. Heavy metals often have direct physiological toxic effects. Some are stored or incorporated in living tissue, sometimes permanently. Heavy metals tend to be stored (accumulating with time) in fatty body tissue. A little arsenic each day may eventually result in a fatal dose—the subject of more than one murder mystery.

The quantity of heavy metals in our bodies is referred to as the *body burden*. The body burden of toxic heavy elements for an average human body (70 kg) is about 8 mg of antimony, 13 mg of mercury, 18 mg of arsenic, 30 mg of cadmium, and 150 mg of lead. The average body burden of lead (for which we apparently have no biological need) is about twice that of the others combined, reflecting our heavy use of this potentially toxic metal.

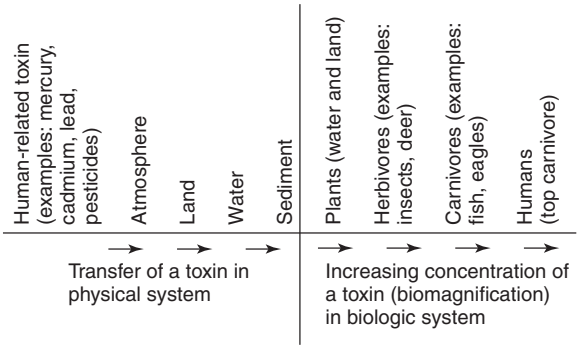
Mercury, thallium, and lead are very toxic to people. They have long been mined and used, and their toxic properties are well known. Mercury, for example, is the "Mad Hatter" element. At one time, mercury was used to stiffen felt hats, and because mercury damages the brain, hatters in Victorian England were known to act peculiarly. Thus, the Mad Hatter in Lewis Carroll's *Alice in Wonderland* had real antecedents in history.

## Toxic Pathways

Chemical elements released from rocks or human processes can become concentrated in people (see Chapter 6) through many pathways (Figure 10.4). These pathways may involve what is known as **biomagnification**—the accumulation or increasing concentration of a substance in living tissue as it moves through a food web (also known as *bioaccumulation*). For example, cadmium, which increases the risk of heart disease, may enter the environment via ash from burning coal. The cadmium in coal is in very low concentrations (less than 0.05 ppm). However, after coal is burned in a power plant, the ash is collected in a solid form and disposed of in a landfill. The landfill is covered with soil and revegetated. The low concentration of cadmium in the ash and soil is taken into the plants as they grow, but the concentration of cadmium in the plants is three to five times greater than the concentration in the ash. As the cadmium moves through the food chain, it becomes more and more concentrated. By the




(a)




(b)

**FIGURE 10.4** **(a)** Potential complex pathways for toxic materials through the living and nonliving environment. Note the many arrows into humans and other animals, sometimes in increasing concentrations as they move through the food chain **(b)**.

time it is incorporated into the tissue of people and other carnivores, the concentration is approximately 50 to 60 times the original concentration in the coal.

Mercury in aquatic ecosystems offers another example of biomagnification. Mercury is a potentially serious pollutant of aquatic ecosystems such as ponds, lakes, rivers, and the ocean. Natural sources of mercury in the environment include volcanic eruptions and erosion of natural mercury deposits, but we are most concerned with human input of mercury into the environment by, for example, burning coal in power plants, incinerating waste, and processing metals such as gold. Rates of input of mercury into the environment through human processes are poorly understood. However, it is believed that human activities have doubled or tripled the amount of mercury in the atmosphere, and it is increasing at about 1.5% per year.[11]

A major source of mercury in many aquatic ecosystems is deposition from the atmosphere through precipitation. Most of the deposition is of inorganic mercury ($Hg^{++}$, ionic mercury). Once this mercury is in surface water, it enters into complex biogeochemical cycles and a process known as *methylation* may occur. Methylation changes inorganic mercury to methyl mercury $[CH_3Hg]^+$ through bacterial activity. Methyl mercury is much more toxic than inorganic mercury, and it is eliminated more slowly from animals' systems. As the methyl mercury works its way through food chains, biomagnification occurs, resulting in higher concentrations of methyl mercury farther up the food chain. In short, big fish that eat little fish contain higher concentrations of mercury than do smaller fish and the aquatic insects that the fish feed on.

Selected aspects of the mercury cycle in aquatic ecosystems are shown in Figure 10.5. The figure emphasizes the input side of the cycle, from deposition of inorganic mercury through formation of methyl mercury, biomagnification, and sedimentation of mercury at the bottom of a pond. On the output side of the cycle, the mercury that enters fish may be taken up by animals that eat the fish; and sediment may release mercury by a variety of processes, including resuspension in the water, where eventually the mercury enters the food chain or is released into the atmosphere through volatilization (conversion of liquid mercury to a vapor form).

Biomagnification also occurs in the ocean. Because large fish, such as tuna and swordfish, have elevated mercury levels, we are advised to limit our consumption of these fish, and pregnant women are advised not to eat them at all.

The threat of mercury poisoning is widespread. Millions of young children in Europe, the United States, and other industrial countries have mercury levels that exceed health standards.[12] Even children in remote areas of the far north are exposed to mercury through their food chain.

During the 20th century, several significant incidents of methyl mercury poisoning were recorded. One, in Minamata Bay, Japan, involved the industrial release of methyl mercury (see A Closer Look 10.2). Another, in Iran, involved a methyl mercury fungicide used to treat wheat seeds. In each of these cases, hundreds of people were killed and thousands were permanently damaged.[11]

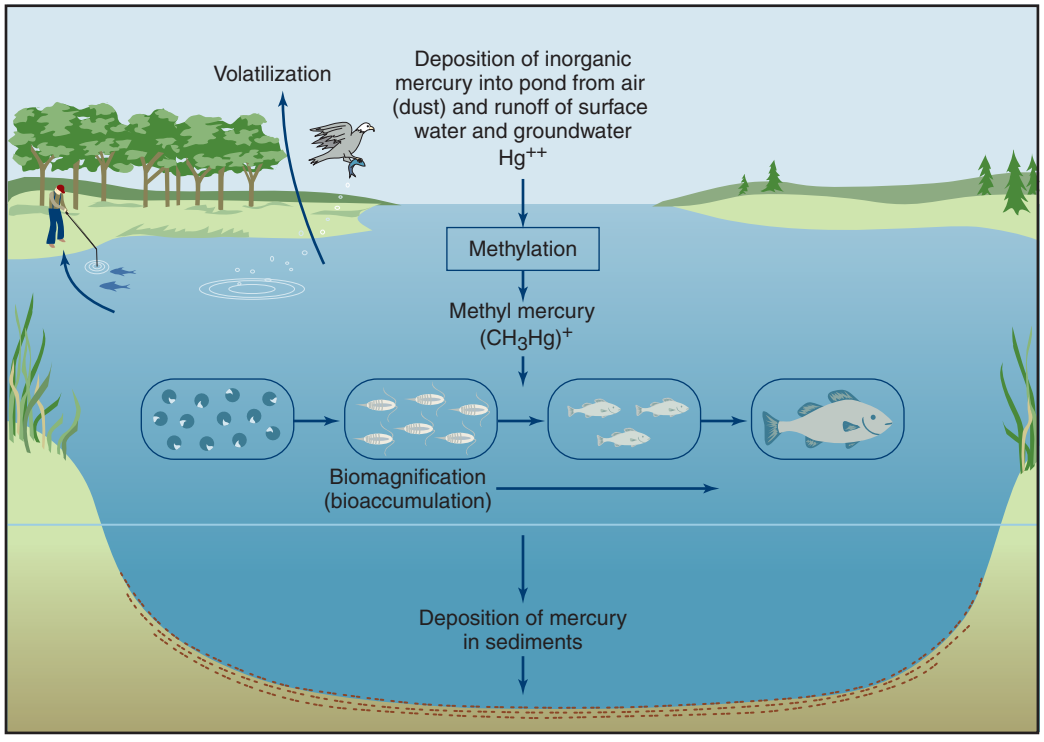



**FIGURE 10.5 Idealized diagram showing selected pathways for movement of mercury into and through an aquatic ecosystem.** (*Source*: Modified from G.L. Waldbott, *Health Effects of Environmental Pollutants*, 2nd ed. [Saint Louis, MO: C.V. Mosby, 1978].)

The cases in Minamata Bay and Iran involved local-exposure mercury. What is being reported in the Arctic, however, emphasizes mercury at the global level, in a region far from emission sources of the toxic metal. The Inuit people in Quanea, Greenland, live above the Arctic Circle, far from any roads and 45 minutes by helicopter from the nearest outpost of modern society. Nevertheless, they are some of the most chemically contaminated people on Earth, with as much as 12 times more mercury in their blood than is recommended in U.S. guidelines. The mercury gets to the Inuit from the industrialized world by way of what they eat. The whale, seal, and fish they eat contain mercury that is further concentrated in the tissue and blood of the people. The process of increasing concentrations of mercury farther up the food chain is an example of biomagnification.[12]

What needs to be done to stop mercury toxicity at the local to global level is straightforward. The answer is to reduce emissions of mercury by capturing it before emission or by using alternatives to mercury in industry. Success will require international cooperation and technology transfer to countries such as China and India, which, with their tremendous increases in manufacturing, are the world's largest users of mercury today.[12]

## Organic Compounds

**Organic compounds** are carbon compounds produced naturally by living organisms or synthetically by industrial processes. It is difficult to generalize about the environmental and health effects of artificially produced organic compounds because there are so many of them, they have so many uses, and they can produce so many different kinds of effects.

**Synthetic organic compounds** are used in industrial processes, pest control, pharmaceuticals, and food additives. We have produced over 20 million synthetic chemicals, and new ones are appearing at a rate of about 1 million per year! Most are not produced commercially, but up to 100,000 chemicals are now being used, or have been used in the past. Once used and dispersed in the environment, they may become a hazard for decades or even hundreds of years.

# A CLOSER LOOK 10.2

## Mercury and Minamata, Japan

In the Japanese coastal town of Minamata, on the island of Kyushu, a strange illness began to occur in the middle of the 20th century. It was first recognized in birds that lost their coordination and fell to the ground or flew into buildings, and in cats that went mad, running in circles and foaming at the mouth.[13] The affliction, known by local fishermen as the "disease of the dancing cats," subsequently affected people, particularly families of fishermen. The first symptoms were subtle: fatigue, irritability, headaches, numbness in arms and legs, and difficulty in swallowing. More severe symptoms involved the sensory organs; vision was blurred and the visual field was restricted. Afflicted people became hard of hearing and lost muscular coordination. Some complained of a metallic taste in their mouths; their gums became inflamed, and they suffered from diarrhea. Lawsuits were brought, and approximately 20,000 people claimed to be affected. In the end, according to the Japanese government, almost 3,000 people were affected and almost 1,800 died. Those affected lived in a small area, and much of the protein in their diet came from fish from Minamata Bay.

A vinyl chloride factory on the bay used mercury in an inorganic form in its production processes. The mercury was released in waste that was discharged into the bay. Mercury forms few organic compounds, and it was believed that the mercury, though poisonous, would not get into food chains. But the inorganic mercury released by the factory was converted by bacterial activity in the bay into methyl mercury, an organic compound that turned out to be much more harmful. Unlike inorganic mercury, methyl mercury readily passes through cell membranes. It is transported by the red blood cells throughout the body, and it enters and damages brain cells.[14] Fish absorb methyl mercury from water 100 times faster than they absorb inorganic mercury. (This was not known before the epidemic in Japan.) And once absorbed, methyl mercury is retained two to five times longer than is inorganic mercury.

In 1982, lawsuits were filed by plaintiffs affected by the mercury. Twenty-two years later, in 2004—almost 50 years after the initial poisonings—the government of Japan agreed to a settlement of $700,000.

Harmful effects of methyl mercury depend on a variety of factors, including the amount and route of intake, the duration of exposure, and the species affected. The effects of the mercury are delayed from three weeks to two months from the

time of ingestion. If mercury intake ceases, some symptoms may gradually disappear, but others are difficult to reverse.[14]

The mercury episode at Minamata illustrates four major factors that must be considered in evaluating and treating toxic environmental pollutants.

*Individuals vary in their response to exposure to the same dose, or amount, of a pollutant.* Not everyone in Minamata responded in the same way; there were variations even among those most heavily exposed. Because we cannot predict exactly how any single individual will respond, we need to find a way to state an expected response of a particular percentage of individuals in a population.

*Pollutants may have a threshold*—that is, a level below which the effects are not observable and above which the effects become apparent. Symptoms appeared in individuals with concentrations of 500 ppb of mercury in their bodies; no measurable symptoms appeared in individuals with significantly lower concentrations.

*Some effects are reversible.* Some people recovered when the mercury-filled seafood was eliminated from their diet.

*The chemical form of a pollutant, its activity, and its potential to cause health problems may be changed markedly by ecological and biological processes.* In the case of mercury, its chemical form and concentration changed as the mercury moved through the food webs.

*Sources*: Mary Kugler, R.N. Thousands poisoned, disabled, and killed. About.com. Created October 23, 2004. About.com Health's Disease and Condition content is reviewed by our Medical Review Board. Also, BBC News, "Japan remembers mercury victims." http://news.bbc.co.uk/go/pr/fr/-/2/hi/asia-pacific/4959562.stm Published 2006/05/01 15:03:11 GMT ©BBC MM VIII.

## Persistent Organic Pollutants

Some synthetic compounds are called **persistent organic pollutants**, or **POP**s. Many were first produced decades ago, when their harm to the environment was not known, and they are now banned or restricted (see Table 10.1 and A Closer Look 10.3). POPs have several properties that define them:[15]

- They have a carbon-based molecular structure, often containing highly reactive chlorine.
- Most are manufactured by people—that is, they are synthetic chemicals.
- They are persistent in the environment—they do not easily break down in the environment.
- They are polluting and toxic.
- They are soluble in fat and likely to accumulate in living tissue.
- They occur in forms that allow them to be transported by wind, water, and sediments for long distances.

For example, consider polychlorinated biphenyls (PCBs), which are heat-stable oils originally used as an insulator in electric transformers.[15] A factory in Alabama manufactured PCBs in the 1940s, shipping them to a General Electric factory in Massachusetts. They were put in insulators and mounted on poles in thousands of locations. The transformers deteriorated over time. Some were damaged by lightning, and others were damaged or destroyed during demolition. The PCBs leaked into the soil or were carried by surface runoff into streams and rivers. Others combined with dust, were transported by wind around the world, and were deposited in ponds, lakes, or rivers, where they entered the food chain. First the PCBs entered algae. Insects ate the algae and were in turn eaten by shrimp and fish. In each stage up the food web, the concentration of PCBs increased. Fish are caught and eaten, passing the PCBs on to people, where they are concentrated in fatty tissue and mother's milk.

**Table 10.1 SELECTED COMMON PERSISTENT ORGANIC POLLUTANTS (POPs)**

| CHEMICAL | EXAMPLE OF USE |
|---|---|
| Aldrin[a] | Insecticide |
| Atrazine[b] | Herbicide |
| DDT[a] | Insecticide |
| Dieldrin[a] | Insecticide |
| Endrin[c] | Insecticide |
| PCBs[a] | Liquid insulators in electric transformers |
| Dioxins | By-product of herbicide production |

[a] Banned in the United States and many other countries.

[b] Degrades in the environment. It is persistent when reapplied often.

[c] Restricted or banned in many countries.

*Source*: Data in part from Anne Platt McGinn, "Phasing Out Persistent Organic Pollutants," in Lester R. Brown et al., *State of the World 2000* (New York: Norton, 2000).

# A CLOSER LOOK 10.3

## Dioxin: How Dangerous Is It?

Dioxin, a persistent organic pollutant, or POP, may be one of the most toxic man-made chemicals in the environment. The history of the scientific study of dioxin and its regulation illustrates the interplay of science and values.

Dioxin is a colorless crystal made up of oxygen, hydrogen, carbon, and chlorine. It is classified as an organic compound because it contains carbon. About 75 types of dioxin and dioxinlike compounds are known; they are distinguished from one another by the arrangement and number of chlorine atoms in the molecule.

Dioxin is not normally manufactured intentionally but is a by-product of chemical reactions, including the combustion of compounds containing chlorine in the production of herbicides.[16] In the United States, there are a variety of sources for dioxinlike compounds (specifically, chlorinated dibenzo-*p*-dioxin, or CDD, and chlorinated dibenzofurans, or CDF). These compounds are emitted into the air through such processes as incineration of municipal waste (the major source), incineration of medical waste, burning of gasoline and diesel fuels in vehicles, burning of wood as a fuel, and refining of metals such as copper.

The good news is that releases of CDDs and CDFs decreased about 75% from 1987 to 1995. However, we are only beginning to understand the many sources of dioxin emissions into the air, water, and land and the linkages and rates of transfer from dominant airborne transport to deposition in water, soil, and the biosphere. In too many cases, the amounts of dioxins emitted are based more on expert opinion than on high-quality data, or even on limited data.[17]

Studies of animals exposed to dioxin suggest that some fish, birds, and other animals are sensitive to even small amounts. As a result, it can cause widespread damage to wildlife, including birth defects and death. However, the concentration at which it poses a hazard to human health is still controversial. Studies suggest that workers exposed to high concentrations of dioxin for longer than a year have an increased risk of dying of cancer.[18]

The Environmental Protection Agency (EPA) has classified dioxin as a known human carcinogen, but the decision is controversial. For most of the exposed people, such as those eating a diet high in animal fat, the EPA puts the risk of developing cancer between 1 in 1,000 and 1 in 100. This estimate represents the highest possible risk for individuals who have had the greatest exposure. For most people, the risk will likely be much lower.[19] The EPA has set an acceptable intake of dioxin at 0.006 pg per kilogram of body weight per day (1 pg = $10^{-12}$ g; see Appendix for prefixes and multiplication factors). This level is deemed too low by some scientists, who argue that the acceptable intake ought to be 100 to 1,000 times higher, or approximately 1 to 10 pg per day.[18] The EPA believes that setting the level this much higher could result in health effects.

The dioxin problem became well known in 1983 when Times Beach, Missouri, a river town just west of Saint Louis with a population of 2,400, was evacuated and purchased for $36 million by the government. The evacuation and purchase occurred after the discovery that oil sprayed on the town's roads to control dust contained dioxin, and that the entire area had been contaminated. Times Beach was labeled a dioxin ghost town (Figure 10.6). The buildings were bulldozed, and all that was left was a grassy and woody area enclosed by a barbed-wire-topped chain-link fence. The evacuation has since been viewed by some scientists (including the person who ordered the evacuation) as a government overreaction to a perceived dioxin hazard. Following clean up, trees were planted and today Times Beach is part of Route 66 State Park and a bird refuge.

The controversy about the toxicity of dioxin is not over.[20-23] Some environmental scientists argue that the regulation of dioxin must be tougher, whereas the industries producing the chemical argue that the dangers of exposure are exaggerated.

**FIGURE 10.6** Soil samples from Times Beach, Missouri, thought to be contaminated by dioxin.

## Hormonally Active Agents (HAAs)

HAAs are also POPs. An increasing body of scientific evidence indicates that certain chemicals in the environment, known as **hormonally active agents (HAAs)**, may cause developmental and reproductive abnormalities in animals, including humans (see A Closer Look 10.4). HAAs include a wide variety of chemicals, such as some herbicides, pesticides, phthalates (compounds found in many chlorine-based plastics), and PCBs. Evidence in support of the hypothesis that HAAs are interfering with the growth and development of organisms comes from studies of wildlife in the field and laboratory studies of human diseases, such as breast, prostate, and ovarian cancer, as well as abnormal testicular development and thyroid-related abnormalities.[24]

Studies of wildlife include evidence that alligator populations in Florida that were exposed to pesticides, such as DDT, have genital abnormalities and low egg production. Pesticides have also been linked to reproductive problems in several species of birds, including gulls, cormorants, brown pelicans, falcons, and eagles. Studies are ongoing on Florida panthers; they apparently have abnormal ratios of sex hormones, and this may be affecting their reproductive capability. In sum, the studies of major disorders in wildlife have centered on abnormalities, including thinning of birds' eggshells, decline in populations of various animals and birds, reduced viability of offspring, and changes in sexual behavior.[25]

With respect to human diseases, much research has been done on linkages between HAAs and breast cancer by exploring relationships between environmental estrogens and cancer. Other studies are ongoing to understand relationships between PCBs and neurological behavior that results in poor performance on standard intelligence tests. Finally, there is concern that exposure of people to phthalates that are found in plastics containing chlorine is also causing problems. Consumption of phthalates in the United States is considerable, with the highest exposure in women of childbearing age. The products being tested as the source of contamination include perfumes and other cosmetics, such as nail polish and hairspray.[25]

In sum, there is good scientific evidence that some chemical agents, in sufficient concentrations, will affect human reproduction through endocrine and hormonal disruption. The human endocrine system is of primary importance because it is one of the two main systems (the other is the nervous system) that regulate and control growth, development, and reproduction. The human endocrine system consists of a group of hormone-secreting glands, including the thyroid, pancreas, pituitary, ovaries (in women), and testes (in men). The bloodstream transports the hormones to virtually all parts of the body, where they act as chemical messengers to control growth and development of the body.[24]

The National Academy of Sciences completed a review of the available scientific evidence concerning HAAs and recommends continued monitoring of wildlife and human populations for abnormal development and reproduction. Furthermore, where wildlife species are known to be experiencing declines in population associated with abnormalities, experiments should be designed to study the phenomena with respect to chemical contamination. For people, the recommendation is for additional studies to document the presence or absence of associations between HAAs and human cancers. When associations are discovered, the causality is investigated in the relationship between exposure and disease, and indicators of susceptibility to disease of certain groups of people by age and sex.[25]

# A CLOSER LOOK 10.4

## Demasculinization and Feminization of Frogs

The story of wild leopard frogs (Figure 10.7) from a variety of areas in the midwestern United States sounds something like a science-fiction horror story. In affected areas, between 10 and 92% of male frogs exhibit gonadal abnormalities, including retarded development and hermaphroditism, meaning they have both male and female reproductive organs. Other frogs have vocal sacs with retarded growth. Since their vocal sacs are used to attract female frogs, these frogs are less likely to mate.

What is apparently causing some of the changes in male frogs is exposure to atrazine, the most widely used herbicide in the United States today. The chemical is a weed killer, used primarily in agricultural areas. The region of the United States with the highest frequency (92%) of sex reversal of male frogs is in Wyoming, along the North Platte River. Although the region is not near any large agricultural activity, and the use of atrazine there is not particularly significant, hermaphrodite frogs are common there because the North Platte River flows from areas in Colorado where atrazine is commonly used.

The amount of atrazine released into the environment of the United States is estimated at approximately 7.3 million kg (16 million lbs) per year. The chemical degrades in the environment, but the degradation process is longer than the application cycle. Because of its continual application every year, the waters of the Mississippi River basin, which drains about 40% of the lower

**FIGURE 10.7** Wild leopard frogs in America have been affected by man-made chemicals (the herbicide atrazine) in the environment.

United States, discharge approximately 0.5 million kg (1.2 million lbs) of atrazine per year to the Gulf of Mexico. Atrazine easily attaches to dust particles and has been found in rain, fog, and snow. As a result, it has contaminated groundwater and surface water in regions where it isn't used. The EPA states that up to 3 parts per billion (ppb) of atrazine in drinking water is acceptable, but at this concentration it definitely affects frogs that swim in the water. Other studies around the world have confirmed this. For example, in Switzerland, where atrazine is banned, it commonly occurs with a concentration of about 1 ppb, and that is sufficient to change some male frogs into females. In fact, atrazine can apparently cause sex change in frogs at concentrations as low as one-thirteenth of the level set by the EPA for drinking water.

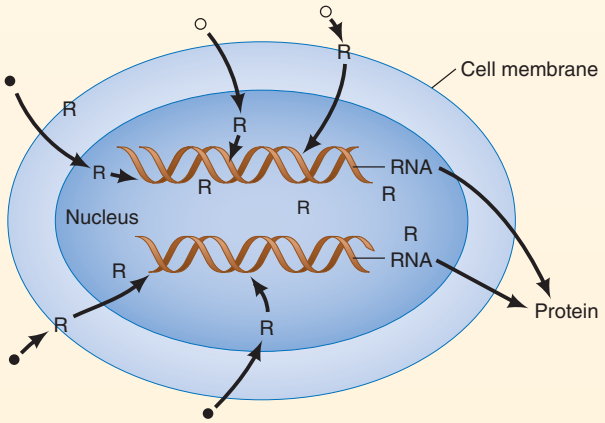
Of particular interest and importance is the process that causes the changes in leopard frogs. We begin the discussion with the endocrine system, composed of glands that secrete hormones such as testosterone and estrogen directly into the bloodstream, which carries them to parts of the body where they regulate and control growth and sexual development. Testosterone in male frogs is partly responsible for development of male characteristics. The atrazine is believed to switch on a gene that turns testosterone into estrogen, a female sex hormone. It's the hormones, not the genes, that actually regulate the development and structure of reproductive organs.

Frogs are particularly vulnerable during their early development, before and as they metamorphose from tadpoles into adult frogs. This change occurs in the spring, when atrazine levels are often at a maximum in surface water. Apparently, a single exposure to the chemical may affect the frog's development. Thus, the herbicide is known as a hormone disrupter.

In a more general sense, substances that interact with the hormone systems of an organism, whether or not they are linked to disease or abnormalities, are known as hormonally active agents (HAAs). These HAAs are able to trick the organism's body (in this case, the frog's) into believing that the chemicals have a role to play in its functional development. An analogy you might be more familiar with is a computer virus that fools the computer into accepting it as part of the system by which the computer works. Similar to computer viruses, the HAAs interact with an organism and the mechanisms for regulating growth and development, thus disrupting normal growth functions.

What happens when HAAs—in particular, hormone disrupters (such as pesticides and herbicides)—are introduced into the system is shown in Figure 10.8. Natural hormones produced by the body send chemical messages to cells, where receptors for the hormone molecules are found on the outside and inside of cells. These natural hormones then transmit instructions to the cells' DNA, eventually directing development and growth. We now know that chemicals, such as some pesticides and herbicides, can also bind to the receptor molecules and either mimic or obstruct the role of the natural hormones. Thus, hormonal disrupters may also be known as HAAs.[24–28]

The story of wild leopard frogs in America dramatizes the importance of carefully evaluating the role of man-made chemicals in the environment. Populations of frogs and other amphibians are declining globally, and much research has been directed toward understanding why. Studies to evaluate past or impending extinctions of organisms often center on global processes such as climate change, but the story of leopard frogs leads us down another path, one associated with our use of the natural environment. It also raises a number of more disturbing questions: Are we participating in an unplanned experiment on how man-made chemicals, such as herbicides and pesticides, might transform the bodies of living beings, perhaps even people? Are these changes in organisms limited to only certain plants and animals, or are they a forerunner of what we might expect in the future on a much broader scale? Perhaps we will look back on this moment of understanding as a new beginning in meaningful studies that will answer some of these important questions.



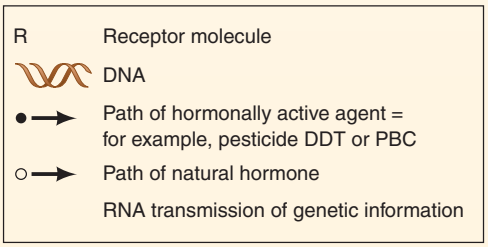




**FIGURE 10.8** Idealized diagram of hormonally active agents (HAAs) binding to receptors on the surface of and inside a cell. When HAAs, along with natural hormones, transmit information to the cells' DNA, the HAAs may obstruct the role of the natural hormones that produce proteins that in turn regulate the growth and development of an organism.

## Nuclear Radiation

Nuclear radiation is introduced here as a category of pollution. We discuss it in detail in Chapter 17, in conjunction with nuclear energy. We are concerned about nuclear radiation because excessive exposure is linked to serious health problems, including cancer. (See Chapter 21 for a discussion of radon gas as an indoor air pollutant.)

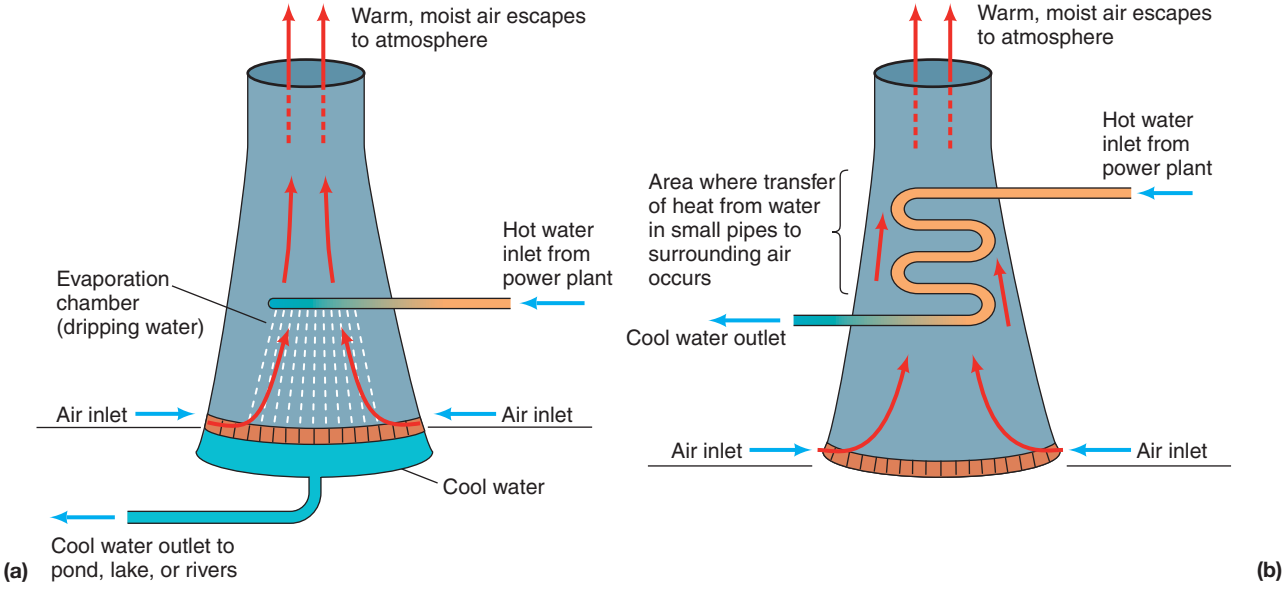
## Thermal Pollution

**Thermal pollution**, also called *heat pollution*, occurs when heat released into water or air produces undesirable effects. Heat pollution can occur as a sudden, acute event or as a long-term, chronic release. Sudden heat releases may result from natural events, such as brush or forest fires and volcanic eruptions, or from human activities, such as agricultural burning.

The major sources of chronic heat pollution are electric power plants that produce electricity in steam generators and release large amounts of heated water into rivers. This changes the average water temperature and the concentration of dissolved oxygen (warm water holds less oxygen than cooler water), thereby changing a river's species composition (see the discussion of eutrophication in Chapter 19). Every species has a temperature range within which it can survive and an optimal temperature for living. For some species of fish, the range is small, and even a small change in water temperature is a problem. Lake fish move away when the water temperature rises more than about 1.5°C above normal; river fish can withstand a rise of about 3°C.

Heating river water can change its natural conditions and disturb the ecosystem in several ways. Fish spawning cycles may be disrupted, and the fish may have a heightened susceptibility to disease. Warmer water also causes physical stress in some fish, making them easier for predators to catch, and warmer water may change the type and abundance of food available for fish at various times of the year.

There are several solutions to chronic thermal discharge into bodies of water. The heat can be released into the air by cooling towers (Figure 10.9), or the heated water can be temporarily stored in artificial lagoons until it





(c)

**FIGURE 10.9 Two types of cooling towers. (a) Wet cooling tower.** Air circulates through the tower; hot water drips down and evaporates, cooling the water. **(b) Dry cooling tower.** Heat from the water is transferred directly to the air, which rises and escapes the tower. **(c) Cooling towers emitting steam at Didcot power plant**, Oxfordshire, England. Red and white lines are vehicle lights resulting from long exposure time (photograph taken at dusk).

cools down to normal temperatures. Some attempts have been made to use the heated water to grow organisms of commercial value that require warmer water. Waste heat from a power plant can also be captured and used for a variety of purposes, such as warming buildings (see Chapter 14 for a discussion of cogeneration).

## Particulates

**Particulates** here refer to small particles of dust (including soot and asbestos fibers) released into the atmosphere by many natural processes and human activities. Modern farming and the burning of oil and coal add considerable amounts of particulates to the atmosphere, as do dust storms, fires (Figure 10.10), and volcanic eruptions. The 1991 eruptions of Mount Pinatubo in the Philippines were the largest volcanic eruptions of the 20th century, explosively hurling huge amounts of volcanic ash, sulfur dioxide, and other volcanic material and gases as high as 30 km (18.6 mi) into the atmosphere. Eruptions can have a significant impact on the global environment and are linked to global climate change and stratospheric ozone depletion (see Chapters 21 and 22). In addition, many chemical toxins, such as heavy metals, enter the biosphere as particulates. Sometimes, nontoxic particulates link with toxic substances, creating a synergetic threat. (See discussion of particulates in Chapter 21.)

## Asbestos

**Asbestos** is a term for several minerals that take the form of small, elongated particles, or fibers. Industrial use of asbestos has contributed to fire prevention and has provided protection from the overheating of materials. Asbestos is also used as insulation for a variety of other purposes. Unfortunately, however, excessive contact with asbestos has led to asbestosis (a lung disease caused by inhaling asbestos) and to cancer in some industrial workers. Experiments with animals have demonstrated that asbestos can cause tumors if the fibers are embedded in lung tissue.[29] The hazard related to certain types of asbestos under certain conditions is considered so serious that extraordinary steps have been taken to reduce the use of asbestos or ban it outright. The expensive process of asbestos removal from old buildings (particularly schools) in the United States is one of those steps.

**FIGURE 10.10** Fires in Indonesia in 1997 caused serious air pollution. The person here is wearing a surgical mask in an attempt to breathe cleaner air.

There are several types of asbestos, and they are not equally hazardous. Most commonly used in the United States is white asbestos, which comes from the mineral chrysolite. It has been used to insulate pipes, floor and ceiling tiles, and brake linings of automobiles and other vehicles. Approximately 95% of the asbestos that is now in place in the United States is of the chrysolite type. Most of this asbestos was mined in Canada, and environmental health studies of Canadian miners show that exposure to chrysolite asbestos is not particularly harmful. However, studies involving another type of asbestos, known as crocidolite asbestos (blue asbestos), suggest that exposure to this mineral can be very hazardous and evidently does cause lung disease. Several other types of asbestos have also been shown to be harmful.[29]

A great deal of fear has been associated with nonoccupational exposure to chrysolite asbestos in the United States. Tremendous amounts of money have been spent to remove it from homes, schools, public buildings, and other sites, even though no asbestos-related disease has been recorded among those exposed to chrysolite in nonoccupational circumstances. It is now thought that much of the removal was unnecessary and that chrysolite asbestos doesn't pose a significant health hazard. Additional research into health risks from other varieties of asbestos is necessary to better understand the potential problem and to outline strategies to eliminate potential health problems.

For example, from 1979 to 1998 a strip mine near Libby, Montana, produced vermiculite (a natural mineral) that was contaminated (commingled) with a fibrous form of the mineral tremolite, classified as an asbestos. People in Libby were exposed to asbestos by workers in the mines (occupational exposure) who brought it home on clothes. Libby is in a valley with very poor ventilation, allowing the asbestos particles to settle out over everything. The EPA has documented hundreds of asbestos-related cases of disease, including many deaths. Asbestos mortality in Libby was much higher than expected, compared to the United States as a whole and to other parts of Montana. In 2009 the EPA declared Libby a public-health emergency. Medical care is being provided, and plans for cleanup of the now closed mine and Libby are under way.[30]

## Electromagnetic Fields

**Electromagnetic fields (EMFs)** are part of everyday urban life. Cell phones, electric motors, electric transmission lines for utilities, and our electrical appliances—toasters, electric blankets, computers, and so forth—all produce magnetic fields. There is currently a controversy over whether these fields produce a health risk.

Early on, investigators did not believe that magnetic fields were harmful, because fields drop off quickly with distance from the source, and the strengths of the fields that most people come into contact with are relatively weak. For example, the magnetic fields generated by power transmission lines or by a computer terminal are normally only about 1% of Earth's magnetic field; directly below power lines, the electric field induced in the body is about what the body naturally produces within cells.[31]

Several early studies, however, concluded that children exposed to EMFs from power lines have an increased risk of contracting leukemia, lymphomas, and nervous-system cancers.[32] Investigators concluded that children so exposed are about one and a half to three times more likely to develop cancer than children with very low exposure to EMFs, but the results were questioned because of perceived problems with the research design (problems of sampling, tracking children, and estimating exposure to EMFs).

A later study analyzed more than 1,200 children, approximately half of them suffering from acute leukemia. It was necessary to estimate residential exposure to magnetic fields generated by power lines near the children's present and former homes. That study, the largest such investigation to date, found no association between childhood leukemia and measured exposure to magnetic fields.[31, 32]

In other studies, electric utility workers' exposure to magnetic fields has been compared with the incidence of brain cancer and leukemia. One study concluded that the association between exposure to magnetic fields and both brain cancer and leukemia is not strong and not statistically significant.[33]

Saying that data are not statistically significant is another way of stating that the relationship between exposure and disease cannot be reasonably established given the database that was analyzed. It does not mean that additional data in a future study will not find a statistically significant relationship. Statistics can predict the strength of the relationship between variables, such as exposure to a toxin and the incidence of a disease, but statistics cannot prove a cause-and-effect relationship between them.

In sum, despite the many studies that have evaluated relationships between cancer (brain, leukemia, and breast) and exposure to magnetic fields in our modern urban environment, the jury is still out. [34, 35] There seems to be some indication that magnetic fields cause health problems for children,[36, 37] but the risks to adults (with the exception of utility workers) appear relatively small and difficult to quantify.[38-41]

## Noise Pollution

**Noise pollution** is unwanted sound. Sound is a form of energy that travels as waves. We hear sound because our ears respond to sound waves through vibrations of the eardrum. The sensation of loudness is related to the intensity of the energy carried by the sound waves and is measured in decibels (dB). The threshold for human hearing is 0 dB; the average sound level in the interior of a home is

**Table 10.2 EXAMPLES OF SOUND LEVELS**

| SOUND SOURCE | INTENSITY OF SOUND (dB) | HUMAN PERCEPTION |
|---|---|---|
| Threshold of hearing | 0 | |
| Rustling of leaf | 10 | Very quiet |
| Faint whisper | 20 | Very quiet |
| Average home | 45 | Quiet |
| Light traffic (30 m away) | 55 | Quiet |
| Normal conversation | 65 | Quiet |
| Chain saw (15 m away) | 80 | Moderately loud |
| Jet aircraft flyover at 300 m | 100 | Very loud |
| Rock music concert | 110 | Very loud |
| Thunderclap (close) | 120 | Uncomfortably loud |
| Jet aircraft takeoff at 100 m | 125 | Uncomfortably loud |
| | 140 | Threshold of pain |
| Rocket engine (close) | 180 | Traumatic injury |

about 45 dB; the sound of an automobile, about 70 dB; and the sound of a jet aircraft taking off, about 120 dB (see Table 10.2). A tenfold increase in the strength of a particular sound adds 10 dB units on the scale. An increase of 100 times adds 20 units.[13] The decibel scale is logarithmic—it increases exponentially as a power of 10. For example, 50 dB is 10 times louder than 40 dB and 100 times louder than 30 dB.

Environmental effects of noise depend not only on the total energy but also on the sound's pitch, frequency, and time pattern and length of exposure to the sound. Very loud noises (more than 140 dB) cause pain, and high levels can cause permanent hearing loss. Human ears can take sound up to about 60 dB without damage or hearing loss. Any sound above 80 dB is potentially dangerous. The noise of a lawn mower or motorcycle will begin to damage hearing after about eight hours of exposure. In recent years, there has been concern about teenagers (and older people, for that matter) who have suffered some permanent loss of hearing following extended exposure to amplified rock music (110 dB). At a noise level of 110 dB, damage to hearing can occur after only half an hour. Loud sounds at the workplace are another hazard. Even noise levels below the hearing-loss level may still interfere with human communication and may cause irritability. Noise in the range of 50–60 dB is sufficient to interfere with sleep, producing a feeling of fatigue upon awakening.

### Voluntary Exposure

Voluntary exposure to toxins and potentially harmful chemicals is sometimes referred to as exposure to personal pollutants. The most common of these are tobacco, alcohol, and other drugs. Use and abuse of these substances have led to a variety of human ills, including death and chronic disease; criminal activity, such as reckless driving and manslaughter; loss of careers; street crime; and the straining of human relations at all levels.

## 10.3 General Effects of Pollutants

Almost every part of the human body is affected by one pollutant or another, as shown in Figure 10.11a. For example, lead and mercury (remember the Mad Hatter) affect the brain; arsenic, the skin; carbon monoxide, the heart; and fluoride, the bones. Wildlife is affected as well. Locations in the body where pollutants may affect humans and wildlife are shown in Figure 10.11b; effects of pollutants on wildlife populations are listed in Table 10.3.

The lists of potential toxins and affected body sites for humans and other animals in Figure 10.11 may be somewhat misleading. For example, chlorinated hydrocarbons, such as dioxin, are stored in the fat cells of animals, but they cause damage not only to fat cells but to the entire organism through disease, damaged skin, and birth defects. Similarly, a toxin that affects the brain, such as mercury, causes a wide variety of problems and symptoms, as illustrated in the Minamata, Japan, example (discussed in A Closer Look 10.2). The value of Figure 10.11 is in helping us to understand in general the adverse effects of excess exposure to chemicals.

### Concept of Dose and Response

Five centuries ago, the physician and alchemist Paracelsus wrote that "everything is poisonous, yet nothing is poisonous." By this he meant, essentially, that too much of any substance can be dangerous, yet in an extremely

**Table 10.3 EFFECTS OF POLLUTANTS ON WILDLIFE**

| EFFECT ON POPULATION | EXAMPLES OF POLLUTANTS |
|---|---|
| Changes in abundance | Arsenic, asbestos, cadmium, fluoride, hydrogen sulfide, nitrogen oxides, particulates, sulfur oxides, vanadium, POPs[a] |
| Changes in distribution | Fluoride, particulates, sulfur oxides, POPs |
| Changes in birth rates | Arsenic, lead, POPs |
| Changes in death rates | Arsenic, asbestos, beryllium, boron, cadmium, fluoride, hydrogen sulfide, lead, particulates, selenium, sulfur oxides, POPs |
| Changes in growth rates | Boron, fluoride, hydrochloric acid, lead, nitrogen oxides, sulfur oxides, POPs |

[a] Pesticides, PCBs, hormonally active agents, dioxin, and DDT are examples (see Table 10.1).

*Source*: J.R. Newman, *Effects of Air Emissions on Wildlife*, U.S. Fish and Wildlife Service, 1980. Biological Services Program, National Power Plant Team, FWS/OBS-80/40, U.S. Fish and Wildlife Service, Washington, DC.

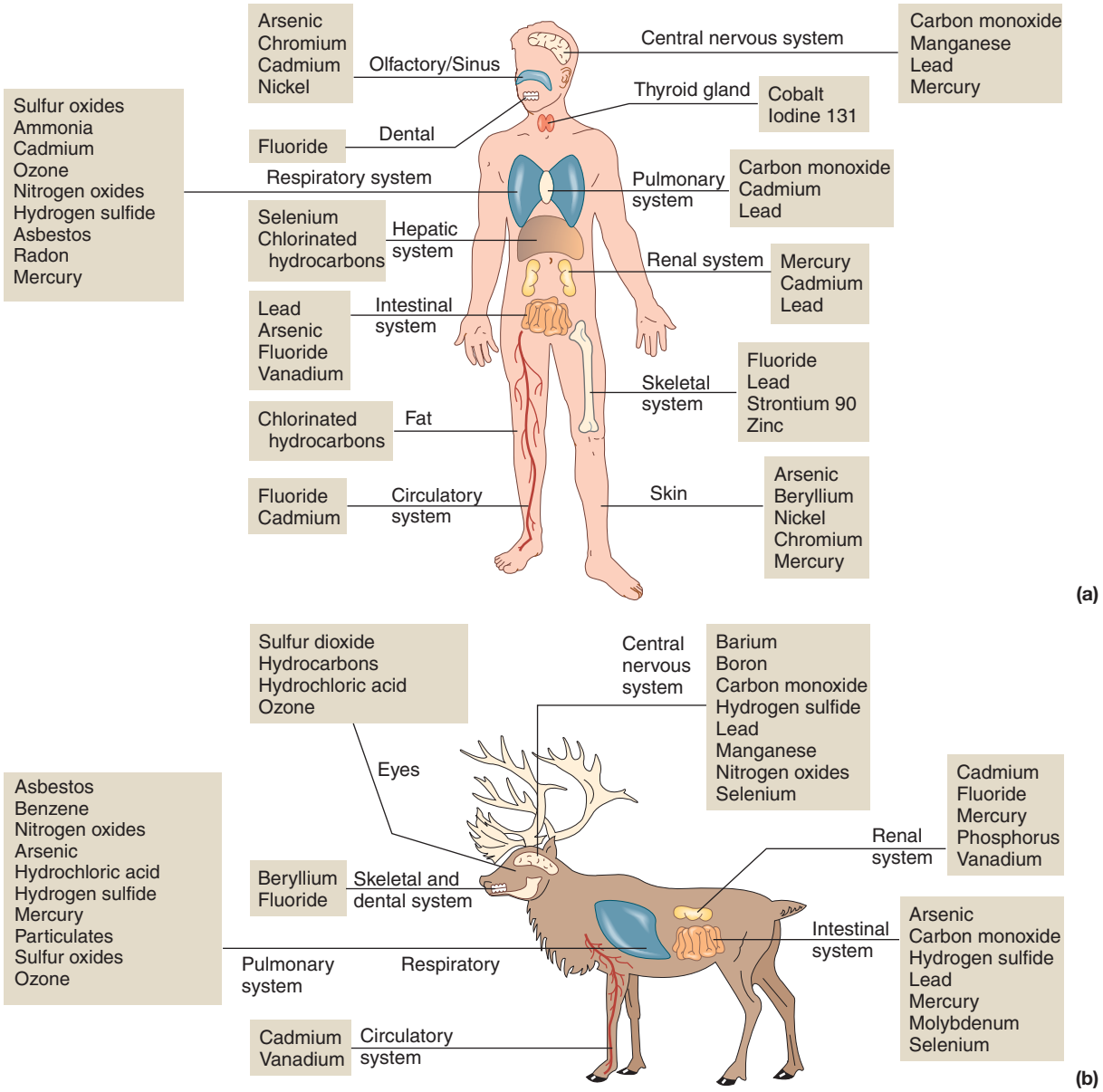



**FIGURE 10.11** **(a)** Effects of some major pollutants in human beings. **(b)** Known sites of effects of some major pollutants in wildlife.

small amount can be relatively harmless. Every chemical element has a spectrum of possible effects on a particular organism. For example, selenium is required in small amounts by living things but may be toxic or increase the probability of cancer in cattle and wildlife when it is present in high concentrations in the soil. Copper, chromium, and manganese are other chemical elements required in small amounts by animals but toxic in higher amounts.

It was recognized many years ago that the effect of a certain chemical on an individual depends on the dose. This concept, termed **dose response**, can be represented by a generalized dose-response curve, such as that shown in Figure 10.12. When various concentrations of a chemical present in a biological system are plotted against the effects on the organism, two things are apparent: Relatively large concentrations are toxic and even lethal (points *D*, *E*, and *F* in Figure 10.12), but trace concentrations may actually be beneficial for life (between points *A* and *D*). The dose-response curve forms a plateau of optimal concentration and maximum benefit between two points (*B* and *C*). Points *A*, *B*, *C*, *D*, *E*, and *F* in Figure 10.12 are important thresholds in the dose-response curve. Unfortunately, the amounts at which points *E* and *F* occur are known only for a few substances for a few organisms, including people, and the very important point *D* is all but unknown. Doses that are beneficial, harmful, or lethal may differ widely for different organisms and are difficult to characterize.

Fluorine provides a good example of the general dose-response concept. Fluorine forms fluoride compounds that prevent tooth decay and promote development of a healthy bone structure. Relationships between the concentration of fluoride (in a compound of fluorine, such

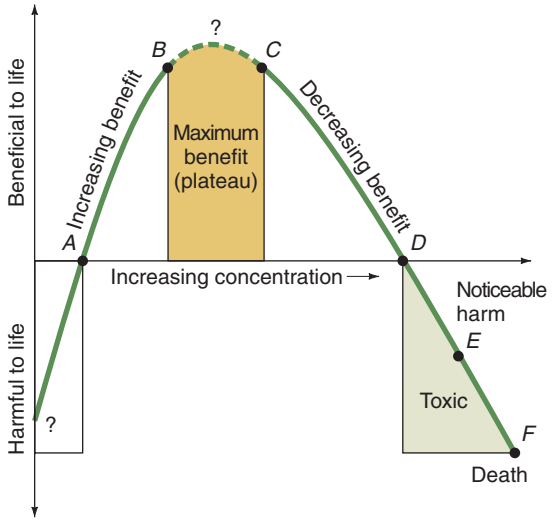



**FIGURE 10.12** Generalized dose-response curve. Low concentrations of a chemical may be harmful to life (below point *A*). As the concentration of the chemical increases from *A* to *B*, the benefit to life increases. The maximum concentration that is beneficial to life lies within the benefit plateau (*B*–*C*). Concentrations greater than this plateau provide less and less benefit (*C*–*D*) and will harm life (*D*–*F*) as toxic concentrations are reached. Increased concentrations above the toxic level may result.

as sodium fluoride, NaF) and health show a specific dose-response curve (Figure 10.13). The plateau for an optimal concentration of fluoride (point *B* to point *C*) to reduce dental caries (cavities) is from about 1 ppm to just less than 5 ppm. Levels greater than 1.5 ppm do not significantly decrease tooth decay but do increase the occurrence of tooth discoloration. Concentrations of 4–6 ppm reduce the prevalence of osteoporosis, a disease characterized by loss of bone mass; and toxic effects are noticed between 6 and 7 ppm (point *D* in Figure 10.13).

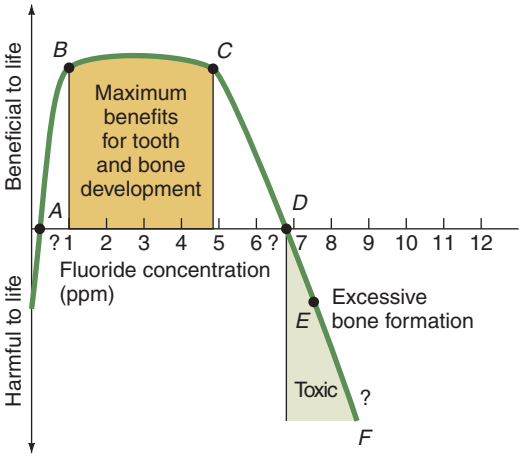



**FIGURE 10.13** General dose-response curve for fluoride, showing the relationship between fluoride concentration and physiological benefit.

## Dose-Response Curve (LD-50, ED-50, and TD-50)

Individuals differ in their response to chemicals, so it is difficult to predict the dose that will cause a response in a particular individual. It is more practical to predict instead what percentage of a population will respond to a specific dose of a chemical.

For example, the dose at which 50% of the population dies is called the lethal dose 50, or LD-50. The **LD-50** is a crude approximation of a chemical's toxicity. It is a gruesome index that does not adequately convey the sophistication of modern toxicology and is of little use in setting a standard for toxicity. However, the LD-50 determination is required for new synthetic chemicals as a way of estimating their toxic potential. Table 10.4 lists, as examples, LD-50 values in rodents for selected chemicals.

**Table 10.4 APPROXIMATE LD–50 VALUES (FOR RODENTS) FOR SELECTED AGENTS**

| AGENT | LD-50(mg/kg)[a] |
|---|---|
| Sodium chloride (table salt) | 4,000 |
| Ferrous sulfate (to treat anemia) | 1,520 |
| 2,4-D (a weed killer) | 368 |
| DDT (an insecticide) | 135 |
| Caffeine (in coffee) | 127 |
| Nicotine (in tobacco) | 24 |
| Strychnine sulfate (used to kill certain pests) | 3 |
| Botulinum toxin (in spoiled food) | 0.00001 |

[a] Milligrams per kilogram of body mass (termed mass weight, although it really isn't a weight) administered by mouth to rodents. Rodents are commonly used in such evaluations, in part because they are mammals (as we are), are small, have a short life expectancy, and their biology is well known.

*Source:* H.B. Schiefer, D.C. Irvine, and S.C. Buzik, *Understanding Toxicology* (New York: CRC Press, 1997).

The **ED-50** (effective dose 50%) is the dose that causes an effect in 50% of the observed subjects. For example, the ED-50 of aspirin would be the dose that relieves headaches in 50% of the people observed.[42]

The **TD-50** (toxic dose 50%) is defined as the dose that is toxic to 50% of the observed subjects. TD-50 is often used to indicate responses such as reduced enzyme activity, decreased reproductive success, or the onset of specific symptoms, such as hearing loss, nausea, or slurred speech.

For a particular chemical, there may be a whole family of dose-response curves, as illustrated in Figure 10.14. Which dose is of interest depends on what is being evaluated. For example, for insecticides we may wish to know the dose that will kill 100% of the insects exposed; therefore, LD-95 (the dose that kills 95% of the insects) may be the minimum acceptable level. However, when considering human health and exposure to a particular toxin, we often want to know the LD-0—the maximum dose that does not cause any deaths.[42] For potentially toxic compounds, such as insecticides that may form a residue on food or food additives, we want to ensure that the expected levels of human exposure will have no known toxic effects. From an environmental perspective, this is important because of concerns about increased risk of cancer associated with exposure to toxic agents.[42]

For drugs used to treat a particular disease, the efficiency of the drug as a treatment is of paramount importance. In addition to knowing what the effective dose (ED-50) is, it is important to know the drug's relative safety. For example, there may be an overlap between the effective dose (ED) and the toxic dose (TD). That is, the dose that causes a positive therapeutic response in some individuals might be toxic to others. A quantitative measure of the relative safety of a particular drug is the *therapeutic index*, defined as the ratio of the LD-50 to the ED-50. The greater the therapeutic index, the safer the drug is believed to be.[43] In other words, a drug with a large difference between the lethal and therapeutic dose is safer than one with a smaller difference.

## Threshold Effects

Recall from A Closer Look 10.2 that a **threshold** is a level below which no effect occurs and above which effects begin to occur. If a threshold dose of a chemical exists, then a concentration of that chemical in the environment below the threshold is safe. If there is no threshold dose, then even the smallest amount of the chemical has some negative effect (Figure 10.15).

Whether or not there is a threshold for environmental toxins is an important environmental issue. For example, the U.S. Federal Clean Water Act originally stated a goal to reduce to zero the discharge of pollutants into water. The goal implies there is no such thing as a threshold—that no level of toxin will be legally permitted. However, it is unrealistic to believe that zero discharge of a water pollutant can be achieved or that we can reduce to zero the concentration of chemicals shown to be carcinogenic.

A problem in evaluating thresholds for toxic pollutants is that it is difficult to account for synergistic effects. Little

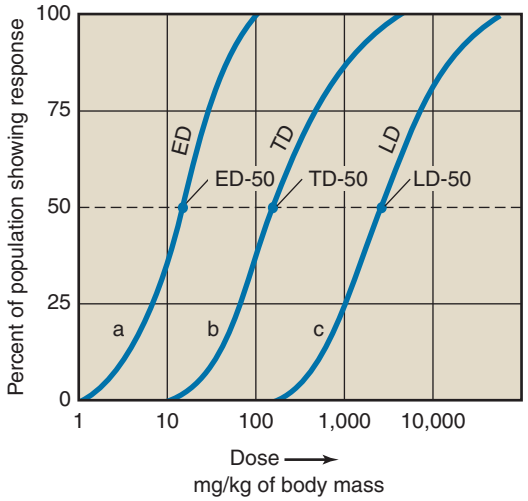



**FIGURE 10.14 Idealized diagram illustrating a family of dose-response curves for a specific drug**: ED (effective dose), TD (toxic dose), and LD (lethal dose). Notice the overlap for some parts of the curves. For example, at ED-50, a small percentage of the people exposed to that dose will suffer a toxic response, but none will die. At TD-50, about 1% of the people exposed to that dose will die.

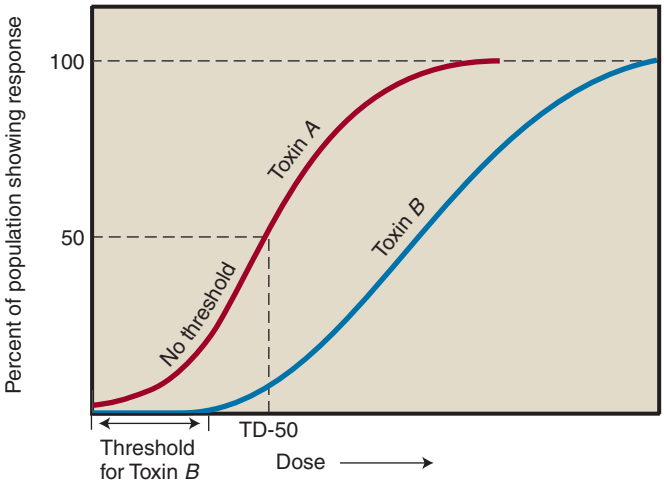



**FIGURE 10.15** In this hypothetical toxic dose-response curve, toxin *A* has no threshold; even the smallest amount has some measurable effect on the population. The TD-50 for toxin *A* is the dose required to produce a response in 50% of the population. Toxin *B* has a threshold (flat part of curve) where the response is constant as the dose increases. After the threshold dose is exceeded, the response increases.

is known about whether or how thresholds might change if an organism is exposed to more than one toxin at the same time or to a combination of toxins and other chemicals, some of which are beneficial. Exposures of people to chemicals in the environment are complex, and we are only beginning to understand and conduct research on the possible interactions and consequences of multiple exposures.

### Ecological Gradients

Dose response differs among species. For example, the kinds of vegetation that can live nearest to a toxic source are often small plants with relatively short lifetimes (grasses, sedges, and weedy species usually regarded as pests) that are adapted to harsh and highly variable environments. Farther from the toxic source, trees may be able to survive. Changes in vegetation with distance from a toxic source define the **ecological gradient**.

Ecological gradients may be found around smelters and other industrial plants that discharge pollutants into the atmosphere from smokestacks. For example, ecological gradient patterns can be observed in the area around the smelters of Sudbury, Ontario, discussed earlier in this chapter (see A Closer Look 10.1). Near the smelters, an area that was once forest was a patchwork of bare rock and soil occupied by small plants.

### Tolerance

The ability to resist or withstand stress from exposure to a pollutant or harmful condition is referred to as **tolerance**. Tolerance can develop for some pollutants in some populations, but not for all pollutants in all populations. Tolerance may result from behavioral, physiological, or genetic adaptation.

*Behavioral tolerance* results from changes in behavior. For example, mice learn to avoid traps.

*Physiological tolerance* results when the body of an individual adjusts to tolerate a higher level of pollutant. For example, in studies at the University of California Environmental Stress Laboratory, students were exposed to ozone ($O_3$), an air pollutant often present in large cities (Chapter 21). The students at first experienced symptoms that included irritation of eyes and throat and shortness of breath. However, after a few days, their bodies adapted to the ozone, and they reported that they believed they were no longer breathing ozone-contaminated air, even though the concentration of $O_3$ stayed the same. This phenomenon explains why some people who regularly breathe polluted air say they do not notice the pollution. Of course, it does not mean that the ozone is doing no damage; it is, especially to people with existing respiratory problems. There are many mechanisms for physiological tolerance, including *detoxification*, in which the toxic chemical is converted to a nontoxic form, and internal transport of the toxin to a part of the body where it is not harmful, such as fat cells.

*Genetic tolerance*, or adaptation, results when some individuals in a population are naturally more resistant to a toxin than others. They are less damaged by exposure and more successful in breeding. Resistant individuals pass on the resistance to future generations, who are also more successful at breeding. Adaptation has been observed among some insect pests following exposure to some chemical pesticides. For example, certain strains of malaria-causing mosquitoes are now resistant to DDT, and some organisms that cause deadly infectious diseases have become resistant to common antibiotic drugs, such as penicillin.

### Acute and Chronic Effects

Pollutants can have acute and chronic effects. *An acute effect* is one that occurs soon after exposure, usually to large amounts of a pollutant. A *chronic effect* occurs over a long period, often from exposure to low levels of a pollutant. For example, a person exposed all at once to a high dose of radiation may be killed by radiation sickness soon after exposure (an acute effect). However, that same total dose received slowly in small amounts over an entire lifetime may instead cause mutations and lead to disease or affect the person's DNA and offspring (a chronic effect).

## 10.4 Risk Assessment

**Risk assessment** in this context can be defined as the process of determining potential adverse health effects of exposure to pollutants and potentially toxic materials (recall the discussion of measurements and methods of science in Chapter 2). Such an assessment generally includes four steps:[44]

1. *Identification of the hazard.* This consists of testing materials to determine whether exposure is likely to cause health problems. One method used is to investigate populations of people who have been previously exposed. For example, to understand the toxicity of radiation produced from radon gas, researchers studied workers in uranium mines. Another method is to perform experiments to test effects on animals, such as mice, rats, or monkeys. This method has drawn increasing criticism from groups who believe such experiments are unethical. Another approach is to try to understand how a particular chemical works at the molecular level on cells. For example, research has been

done to determine how dioxin interacts with living cells to produce an adverse response. After quantifying the response, scientists can develop mathematical models to assess dioxin's risk.[18, 19] This relatively new approach might also be applicable to other potential toxins that work at the cellular level.

2. *Dose-response assessment*. This next step involves identifying relationships between the dose of a chemical (therapeutic drug, pollutant, or toxin) and the health effects on people. Some studies involve administering fairly high doses of a chemical to animals. The effects, such as illness, or symptoms, such as rashes or tumor development, are recorded for varying doses, and the results are used to predict the response in people. This is difficult, and the results are controversial for several reasons:
   - The dose that produces a particular response may be very small and subject to measurement errors.
   - There may be arguments over whether thresholds are present or absent.
   - Experiments on animals such as rats, mice, or monkeys may not be directly applicable to humans.
   - The assessment may rely on probability and statistical analysis. Although statistically significant results from experiments or observations are accepted as evidence to support an argument, statistics cannot establish that the substance tested *caused* the observed response.
3. *Exposure assessment.* This step evaluates the intensity, duration, and frequency of human exposure to a particular chemical pollutant or toxin. The hazard to society is directly proportional to the total population exposed. The hazard to an individual is generally greater closer to the source of exposure. Like dose-response assessment, exposure assessment is difficult, and the results are often controversial, in part because of difficulties in measuring the concentration of a toxin in doses as small as parts per million, billion, or even trillion. Some questions that exposure assessment attempts to answer follow:
   - How many people were exposed to concentrations of a toxin thought to be dangerous?
   - How large an area was contaminated by the toxin?
   - What are the ecological gradients for exposure to the toxin?
   - How long were people exposed to a particular toxin?
4. *Risk characterization:* The goal of this final step is to delineate health risk in terms of the magnitude of the health problem that might result from exposure to a particular pollutant or toxin. To do this, it is necessary to identify the hazard, complete the dose-response assessment, and evaluate the exposure assessment, as has been outlined. This step involves all the uncertainties of the prior steps, and results are again likely to be controversial.

In sum, *risk assessment* is difficult, costly, and controversial. Each chemical is different, and there is no one method of determining responses of humans to specific EDs or TDs. Toxicologists use the scientific method of hypothesis-testing with experiments (see Chapter 2) to predict how specific doses of a chemical may affect humans. Warning labels listing potential side effects of a specific medication are required by law, and these warnings result from toxicology studies to determine a drug's safety. Finally, risk assessment requires making scientific judgments and formulating actions to help minimize health problems related to human exposure to environmental pollutants and toxins.

The process of *risk management* integrates the assessment of risk with technical, legal, political, social, and economic issues.[18, 19] The toxicity of a particular material is often open to debate. For example, there is debate as to whether the risk from dioxin is linear. That is, do effects start at minimum levels of exposure and gradually increase, or is there a threshold exposure beyond which health problems occur? (See A Closer Look 10.3.)[18, 19, 29] It is the task of people in appropriate government agencies assigned to manage risk to make judgments and decisions based on the risk assessment and then to take appropriate actions to minimize the hazard resulting from exposure to toxins. This might involve invoking the precautionary principle discussed in Chapter 1.

# CRITICAL THINKING ISSUE

## Is Lead in the Urban Environment Contributing to Antisocial Behavior?

Lead is one of the most common toxic metals in our inner-city environments, and it may be linked to delinquent behavior in children. Lead is found in all parts of the urban environment (air, soil, older pipes, and some paint, for example) and in biological systems, including people (Figure 10.16). There is no apparent biological need for lead, but it is sufficiently concentrated in the blood and bones of children living in inner cities to cause health and behavior problems. In some populations, over 20% of the children have blood concentrations of lead that are higher than those believed safe.[45]

Lead affects nearly every system of the body. Thus, acute lead toxicity may cause a variety of symptoms, including anemia, mental retardation, palsy, coma, seizures, apathy, uncoordination, subtle loss of recently acquired skills, and bizarre behavior.[46,47] Lead toxicity is particularly a problem for young children, who are more apt than adults to put things in their mouths and apparently are also more susceptible to lead poisoning. In some children the response to lead poisoning is aggressive, difficult-to-manage behavior.[45-48]

The occurrence of lead toxicity or lead poisoning has cultural, political, and sociological implications. Over 2,000 years ago, the Roman Empire produced and used tremendous amounts of lead for a period of several hundred years. Production rates were as high as 55,000 metric tons per year. Romans had a wide variety of uses for lead. Lead was used in pots in which grapes were crushed and processed into a syrup for making wine, in cups and goblets from which wine was drunk, and as a base for cosmetics and medicines. In the homes of Romans wealthy enough to have running water, lead was used to make the pipes that carried the water. It has been argued that lead poisoning among the upper class in Rome was partly responsible for Rome's decline. Lead poisoning probably resulted in widespread stillbirths, deformities, and brain damage. Studies analyzing the lead content of bones of ancient Romans tend to support this hypothesis.[49]

**FIGURE 10.16** The lead in urban soils (a legacy of our past use of lead in gasoline) is still concentrated where children are likely to play. Lead-based paint in older buildings, such as these in New York, also remains a hazard to young children, who sometimes ingest flakes of paint.

The occurrence of lead in glacial ice cores from Greenland has also been studied. Glaciers have an annual growth layer of ice. Older layers are buried by younger layers, allowing us to identify the age of each layer. Researchers drill glaciers, taking continuous samples of the layers. The samples look like long, solid rods of glacial ice and are called *cores*. Measurements of lead in these cores show that lead concentrations during the Roman period, from approximately 500 B.C. to A.D. 300, are about four times higher than before and after this period. This suggests that the mining and smelting of lead in the Roman Empire added small particles of lead to the atmosphere that eventually settled out in the glaciers of Greenland.[49]

Lead toxicity, then, seems to have been a problem for a long time. Now, an emerging, interesting, and potentially significant hypothesis is that, in children, even lead concentrations below the levels known to cause physical damage may be associated with an increased potential for antisocial, delinquent behavior. This is a testable hypothesis. (See Chapter 2 for a discussion of hypotheses.) If the hypothesis is correct, then some of our urban crime may be traced to environmental pollution!

A recent study in children aged 7 to 11 measured the amount of lead in bones and compared it with data concerning behavior over a four-year period. Even taking into account such factors as maternal intelligence, socioeconomic status, and quality of child rearing, the study concluded that an above-average concentration of lead in children's bones was associated with an increased risk of attention-deficit disorder, aggressive behavior, and delinquency.[45]

### Critical Thinking Questions

1. What is the main point of the discussion about lead in the bones of children and children's behavior?
2. What are the main assumptions of the argument? Are they reasonable?
3. What other hypotheses might be proposed to explain the behavior?

## SUMMARY

- Disease is an imbalance between an organism and the environment. Disease seldom has a one-cause/one-effect relationship, and there is often a gray zone between the state of health and the state of disease.
- Pollution produces an impure, dirty, or otherwise unclean state. Contamination means making something unfit for a particular use through the introduction of undesirable materials.
- Toxic materials are poisonous to people and other living things; toxicology is the study of toxic materials.
- A concept important in studying pollution problems is synergism, whereby actions of different substances produce a combined effect greater than the sum of the effects of the individual substances.
- How we measure the amount of a particular pollutant introduced into the environment or the concentration of that pollutant varies widely, depending on the substance. Common units for expressing the concentration of pollutants are parts per million (ppm) and parts per billion (ppb). Air pollutants are commonly measured in units such as micrograms of pollutant per cubic meter of air ($\mu g/m^3$).
- Categories of environmental pollutants include toxic chemical elements (particularly heavy metals), organic compounds, nuclear radiation, heat, particulates, electromagnetic fields, and noise.
- Organic compounds of carbon are produced by living organisms or synthetically by people. Artificially produced organic compounds may have physiological, genetic, or ecological effects when introduced into the environment. The potential hazards of organic compounds vary: Some are more readily degraded in the environment than others; some are more likely to undergo biomagnification; and some are extremely toxic, even at very low concentrations. Organic compounds of serious concern include persistent organic pollutants, such as pesticides, dioxin, PCBs, and hormonally active agents.
- The effect of a chemical or toxic material on an individual depends on the dose. It is also important to determine tolerances of individuals, as well as acute and chronic effects of pollutants and toxins.
- Risk assessment involves identifying the hazard, assessing the exposure and the dose response, and characterizing the possible results.

## REEXAMINING THEMES AND ISSUES

**Human Population**

As the total population and population density increase, the probability that more people will be exposed to hazardous materials increases as well. Finding acceptable ways to dispose of hazardous substances also becomes more difficult as populations increase and people live closer to industrial areas and waste-disposal sites.

**Sustainability**

Ensuring that future generations inherit a relatively unpolluted, healthy environment remains a challenging problem. Sustainable development requires that our use of chemicals and other materials not damage the environment.

**Global Perspective**

Releasing toxins into the environment can cause global patterns of contamination or pollution, particularly when a toxin or contaminant enters the atmosphere, surface water, or oceans and becomes widely dispersed. For example, pesticides, herbicides, and heavy metals emitted into the atmosphere in the midwestern United States may be transported by winds and deposited on glaciers in polar regions.

## Urban World

Industrial processes in urban areas concentrate potentially toxic materials that may be inadvertently, accidentally, or deliberately released into the environment. Human exposure to a variety of pollutants—including lead, asbestos, particulates, organic chemicals, radiation, and noise—is often greater in urban areas.

## People and Nature

Feminization of frogs and other animals from exposure to human-produced, hormonally active agents (HAAs) is an early warning or red flag that we are disrupting some basic aspects of nature. We are performing unplanned experiments on nature, and the consequences to us and other living organisms with which we share the environment are poorly understood. Control of HAAs seems an obvious candidate for application of the precautionary principle, discussed in Chapter 1.

## Science and Values

Because we value both human and nonhuman life, we are interested in learning all we can about the risks of exposing living things to chemicals, pollutants, and toxins. Unfortunately, our knowledge of risk assessment is often incomplete, and the dose response for many chemicals is poorly understood. What we decide to do about exposure to toxic chemicals reflects our values. Increased control of toxic materials in homes and the work environment is expensive. To reduce environmental hazards at worksites in other countries, are we willing to pay more for the goods those workers manufacture?

## KEY TERMS

area sources **188**
asbestos **199**
biomagnification **191**
carcinogen **188**
contamination **188**
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## STUDY QUESTIONS

1. Do you think the hypothesis that some crime is caused in part by environmental pollution is valid? Why? Why not? How might the hypothesis be further tested? What are the social ramifications of the tests?
2. What kinds of life-forms would most likely survive in a highly polluted world? What would be their general ecological characteristics?
3. Some environmentalists argue that there is no such thing as a threshold for pollution effects. What do they mean? How would you determine whether it was true for a specific chemical and a specific species?
4. What is biomagnification, and why is it important in toxicology?

5. You are lost in Transylvania while trying to locate Dracula's castle. Your only clue is that the soil around the castle is known to have an unusually high concentration of the heavy metal arsenic. You wander in a dense fog, able to see only the ground a few meters in front of you. What changes in vegetation warn you that you are nearing the castle?
6. Distinguish between acute effects and chronic effects of pollutants.
7. Design an experiment to test whether tomatoes or cucumbers are more sensitive to lead pollution.
8. Why is it difficult to establish standards for acceptable levels of pollution? In giving your answer, consider physical, climatological, biological, social, and ethical reasons.
9. A new highway is built through a pine forest. Driving along the highway, you notice that the pines nearest the road have turned brown and are dying. You stop at a rest area and walk into the woods. One hundred meters away from the highway, the trees seem undamaged. How could you make a crude dose–response curve from direct observations of the pine forest? What else would be necessary to devise a dose–response curve from direct observation of the forest? What else would be necessary to devise a dose–response curve that could be used in planning the route of another highway?
10. Do you think your personal behavior is placing you in the gray zone of suboptimal health? If so, what can you do to avoid chronic disease in the future?

## FURTHER READING

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**Schiefer, H.B., D.G. Irvine, and S.C. Buzik,** ***Understanding Toxicology: Chemicals, Their Benefits and Risks*** (Boca Raton, FL: CRC Press, 1997). A concise introduction to toxicology as it pertains to everyday life, including information about pesticides, industrial chemicals, hazardous waste, and air pollution.

**Travis, C.C., and H.A. Hattemer-Frey.** "Human Exposure to Dioxin," *The Science of the Total Environment* 104: 97–127, 1991. An extensive technical review of dioxin accumulation and exposure.