5

Health Effects

5.1 Air Pollution Episodes

Air pollution episodes are characterized by significant short-term increases in atmospheric pollutant concentrations above normal daily levels. Episodes vary from those that pose relatively limited public health concern to those more extreme. Extreme air pollution episodes were reported for the Meuse Valley, Belgium, in 1930; Donora, PA, and the Monongehela River Valley in 1948; and London in 1952. In each of these cases, a persistent (3–6 days) thermal inversion combined with significant industrial and, in the case of London, domestic pollutant emissions resulted in high ground-level concentrations that caused acute illness and, in some cases, death in exposed populations.

Sixty deaths were reported in the Meuse Valley disaster, 20 in Donora, and approximately 4,000 in London. In each case, death claimed individuals with existing respiratory or cardiovascular disease. Pneumonia was the primary cause of death in the 1952 London disaster. Based on retrospective studies, "excess mortality" was associated with a number of "London fogs," going back to as early as 1873. In each of these extreme episodes, many individuals reported becoming ill. Illness characterized by cough, shortness of breath, chest pain, and eye and nose irritation was reported by people of all ages. In the Donora area, more than half of the 14,000 residents reported experiencing acute illness symptoms.

Other somewhat less extreme episodes have been reported for London in 1956, 1957, 1959, 1962, 1975, and 1991; Dublin in 1982; the Ruhr Valley of Germany in 1962, 1979, 1982, and 1985; the Netherlands in 1985, 1987, and 1991; and New York City in 1953, 1962, and 1966.

These episodes are significant in that they provided solid scientific documentation that exposure to elevated ambient pollutant levels can cause acute illness and even death. The more disastrous episodes contributed to significant efforts to control ambient air pollution. This was particularly the case in London, where clean air legislation was enacted as early as 1956.

Despite significant control efforts, air pollution episodes continue to occur in developed countries. Although pollutant levels are now considerably

lower than in the disastrous episodes of the past, application of increasingly more sophisticated epidemiological techniques to what could be described as historically minor episodes shows significant associations between elevated pollutant levels, acute illness such as asthmatic attacks, and even increased death rates.

Air pollution episodes are not limited to developed countries. Increased industrialization, limited pollution control efforts, and population growth pose significant exposure risks on a day-to-day basis as well as during episode periods in many cities in China, India, Mexico, and other countries.

5.2 Pollutant Exposures and Cause–Effect Relationships

Ambient air pollution exposures that result in severe community-wide illness and death are, for the most part, relatively rare in the United States and other developed countries. Pollutant exposures in our cities, towns, and even countrysides vary considerably, both in types of pollutants present and prevailing concentrations. Exposures reflect the nature and extent of industrial activities, dispersion conditions of the surrounding environment, motor vehicle emissions, atmospheric chemistry, and, in some mountain valleys, wood space heating.

Pollutant exposures to the "normal" range of concentrations may result in a variety of health effects. These include acute but transient symptoms such as eye, nose, and throat irritation and, in some sensitive individuals, asthmatic attacks. Acute asthmatic responses to ambient pollutants are a major regulatory concern. The primary focus of most regulatory activity in North America and other developed countries has been to protect exposed or potentially exposed populations from the chronic effects of relatively low-level exposures. These may include respiratory and cardiovascular disease, neurotoxic effects, and cancer.

A good understanding of the observed or potential health effects of pollutants at levels typical of urban–suburban communities is requisite to all health-based regulatory programs. Ideally, such regulatory activities are supported by scientific data that adequately establish a cause-and-effect relationship between pollutant exposures and illness symptoms among those exposed so that an adequate margin of safety can be incorporated into air quality standards. Because humans experience a variety of exposures and other stresses, establishing definitive cause–effect relationships between adverse health effects and one or more atmospheric pollutants is a difficult undertaking.

A strong relationship between individual or combined pollutant exposures and health effects is indicated when there is a convergence of evidence from epidemiological, toxicological, and occupational exposure studies.

Information from such studies is of particular importance in evaluating the potential health effects of chronic exposures to ambient pollutants.

5.2.1 Epidemiological Studies

Epidemiological studies are conducted to determine potential relationships between a variety of environmental factors and human disease. They are characterized by the application of statistical analysis to data collected on the health status of populations of individuals, pollutant exposures, and potential confounding factors. Such studies often provide evidence of possible causal relationships between pollutant exposures and observed or reported health effects. In general, epidemiological studies become more important as the risk attributable to atmospheric pollutants becomes smaller and the duration of exposure required to produce effects becomes longer. They have been particularly useful in identifying the acute effects of elevated short-term pollutant exposures. These effects may include pulmonary function changes, asthmatic attacks, and increased mortality.

Epidemiological studies vary in design. They may be cross-sectional (assess the relationship between pollutant exposures and health effects over a cross section of a population), longitudinal (assess exposures and health effects over time), or case-control (exposed and non- or less-exposed populations are compared). They may be prospective (data to be collected) or retrospective (evaluation of existing data). Study designs differ in their power to identify potential causal relationships and statistical confidence in results obtained. Over the past decade, increasingly sophisticated statistical techniques have been applied to the study of short-term pollutant exposures. These have included time-series analyses of daily health data collected over a period of days or weeks. Such studies are longitudinal, and as such, each participant serves as his or her own control. Consequently, the effect of confounding variables (described below) is significantly reduced when data are statistically analyzed. Such studies are characterized by a strong study design, good exposure and health effects data, and large study populations.

5.2.1.1 Confounding Factors

Epidemiological assessment of potential relationships between pollutant exposures and observed parameters of human health has, in many cases, been confounded by a variety of coexisting factors. These have included individual sensitivity, age, existing disease, gender, race, socioeconomic status, tobacco smoking, lifestyle, occupation, and meteorological conditions. Assessment of potential effects may also be confounded by interaction between (1) two or more pollutants, (2) pollutants and meteorological variables such as temperature and relative humidity, (3) pollutants and other exposures (occupational, smoking, and indoor air pollution), and (4) pollutants

and infectious disease. Such interactions may, in part, explain the differences observed between epidemiological and toxicological studies.

5.2.1.2 Interaction Effects

5.2.1.2.1 Interactions between Pollutants

Because community air is commonly contaminated by complex mixtures of gases and particulate matter (PM), it is likely that some of these may interact to modify the physiological effects of others. This may occur in several different ways. For example, one pollutant may affect the site of deposition of another. This is apparently the case for gases such as sulfur dioxide (SO_2). Because of its solubility in watery fluids, SO_2 is normally removed in the upper respiratory system. Absorption and adsorption of SO_2 on particles provides a transport mechanism for SO_2 into the pulmonary system, where SO_2 toxicity occurs. Consequently, particles can potentiate the effect of SO_2. Harmful aerosols such as sulfuric acid (H_2SO_4) can be produced as a result of interaction of gaseous pollutants with the moist environment of the lungs.

Pollutant interactions may result in health and physiological effects that are additive, antagonistic, or synergistic. Exposure to pollutants with similar toxic effects may result in an additive response. The total toxic response is the sum of individual responses to each pollutant. Antagonism occurs when one pollutant interferes with the toxic effects of another, thus mitigating the effects of the more toxic pollutant. Reactions of ammonia (NH_3) and SO_2 in human breath are an example of such an effect. Synergism describes health responses that are significantly greater than the sum of the individual effects of toxic pollutants. This is particularly true for pollutants that inhibit pulmonary defense mechanisms (e.g., tobacco smoke). Inhibition of respiratory clearance increases the risk of disease (e.g., cancer) from pollutants such as asbestos and radon (Rn). In many cases, synergistic effects are multiplicative; that is, risk is increased by the product of each individual exposure risk.

Epidemiological and toxicological information on additive, antagonistic, and synergistic effects is limited. Because of the large variety of pollutants present in metropolitan atmospheres, it is probable that each of these interaction effects occurs. In day-to-day pollutant exposures, they cannot, for the most part, be evaluated using the scientific tools currently available. It is standard regulatory practice to set exposure limits on individual pollutants as if they were acting alone. Such exposure limits are subject to intense legal, political, and scientific challenges from those whose activities would be subject to regulation. Use of exposure limits based on multiple pollutant exposures would pose an enormous regulatory challenge.

5.2.1.2.2 Interactions between Pollutants and Meteorological Factors

Most major air pollution episodes were associated with meteorological extremes that confounded assessment of the causal role of ambient air

pollution in the genesis of mortality and morbidity. During the many "London fogs" and disasters in the Meuse Valley and Donora, weather was cold and damp. The independent effect of temperature extremes on mortality has been well established. Indeed, studies in the United Kingdom have indicated that mortality was more closely related to cold and humidity than to fog frequency. Studies in Los Angeles have noted a marked increase in mortality during periods of elevated photochemical oxidant levels and high temperature. In such cases, investigators commonly concluded that observed excess mortality was due to high temperatures rather than pollutant exposures. Heat waves, in general, are major stressors of the elderly and infirm and usually result in excess mortality. With the application of more powerful research designs and statistical techniques, epidemiologists are increasingly able to separate the independent effects of temperature extremes and pollutant exposures in evaluations of excess mortality.

5.2.1.2.3 Interaction between Ambient Air Pollutants and Other Pollutant Exposures

In addition to community air pollution exposures, many individuals are exposed to elevated pollutant levels from cigarette smoking and in their occupational and domestic environments. Such exposures may mask health risks associated with ambient pollutants. They may also increase health risks associated with ambient pollution.

Cigarette smokers may be at higher risk for adverse effects associated with exposures to ambient pollution. This increased risk may be due to one or more factors. First, cigarette smoking may initiate respiratory and cardiovascular disease. Individuals with such disease, no matter what the cause, are at increased risk of illness or death when exposed to elevated pollutant levels. Smoking may increase the effect of community air pollution exposures (Figure 5.1) by impairing respiratory clearance mechanisms.

Many individuals are exposed to relatively high levels of gaseous and particulate-phase pollutants in their place of employment. These exposures are usually much greater than those associated with breathing polluted community air. Because they are occupationally exposed, some individuals may be at higher risk from ambient pollutant exposures. This increased risk may be due to occupationally induced respiratory or cardiovascular disease or impaired defense mechanisms.

Assessment of health effects of community air pollutants is confounded, to some degree, by the fact that individuals spend more time indoors than in the ambient environment. On average, Americans only spend 2 h/day outdoors. Therefore, their ambient pollution exposure may be quite different than a police officer's or construction worker's. An expanded discussion of indoor air pollution and its health implications can be found in Chapter 11.

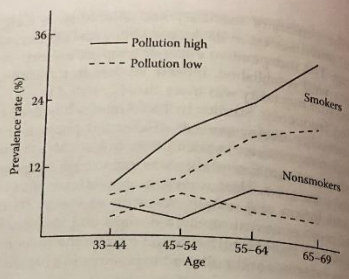

FIGURE 5.1
Prevalence of chronic bronchitis associated with tobacco smoking and exposure to ambient air pollution. (From Lambert, P.M. and Reid, D.D., *Lancet*, 5, 853, 1970. With permission.)

5.2.1.3 Exposure Assessment

Most early epidemiological studies of air pollution and related health effects were limited by inadequate information on pollutants present and data on population exposures. As a result, many of these studies used the place of residence as an index of exposure. The place of residence often introduces bias into statistical analyses because it may be associated with unique ethnic or cultural traits, living standards, occupations, and exposure to infectious agents. Exposure assessment in many recent longitudinal studies is carried out using personal exposure monitors, time activity diaries, and exposure models based on fixed-site monitors and dispersion modeling of various emission sources. Exposure assessment, like other aspects of epidemiological studies, has become more sophisticated and better serves the needs of pollutant effects studies.

5.2.1.4 Population Susceptibility

Epidemiological assessment of air pollution–related health effects has historically been complicated by variations in population susceptibility. Such assessments are now simplified by identifying the populations at risk, for example, the aged, the very young, those with existing respiratory or cardiovascular disease, those occupationally exposed, and cigarette smokers. These populations/individuals may be more sensitive to normally encountered pollutant levels than the average healthy individual. Epidemiological studies of sensitive populations are more powerful in identifying air pollution–health effects associations when they indeed exist. Present air quality

standards (Chapter 8) promulgated in the United States are intended to protect those at special risk as well as the general population.

Despite the confounding factors and historical limitations described, epidemiological studies have been able to provide valuable insight into defining the probability that air pollution exposures contribute to the incidence or prevalence of specific community health problems. A strong association or high probability of disease causation can be inferred when (1) there are a number of different populations in which a similar association is observed, including different population groups, locations, climate, and times of year; (2) the incidence or severity of the health effect increases with increasing exposure and, conversely, decreases with decreasing exposure; and (3) a plausible biological mechanism can be hypothesized for the observed association.

The value of epidemiological studies is increased when they are used in conjunction with controlled biological (toxicological) studies on animals and humans. They are important in identifying possible associations that can be tested under controlled laboratory conditions. In addition, they can be used to evaluate potential human health risks identified in laboratory exposures.

5.2.2 Toxicological Studies

Toxicological, or controlled biological studies are conducted on animals and, less commonly, humans to determine the functional, structural, and biochemical effects of toxic substances. Historically, toxicological studies have been conducted to determine the toxicity of a substance administered in varying doses, with dose being a function of the concentration of the substance and duration of exposure. Significant differences in toxic response occur when the same dose of toxic material is administered over different exposure periods. Acute exposures result when an organism is subjected to a high dose rate, that is, a high concentration of a substance over a relatively short period. Response to the exposure is sudden, severe, and usually lasts for a brief period. If the dose rate is sufficiently high, death may result. Indeed, the term *toxic* implies that a substance has the potential to cause death. Lower doses, that is, lower concentrations over longer periods, generally do not directly cause mortality. As dose rate decreases, the response is less severe, may take longer to develop, and may be more persistent. In chronic exposures, adverse effects may take years, if not decades, to develop.

In traditional toxicology, animals are exposed to varied doses of toxic substances to determine their ability to cause physiological and pathological changes and to characterize dose–response relationships. In addition to evaluating such physiological and pathological changes, the scope of toxicology in recent years has been expanded to include studies of carcinogenesis (cancer induction), teratogenesis (induction of birth defects), mutagenesis (production of mutations), gametotoxicity (damage to sex cells), and endocrine disruption.

One of the major advantages of toxicological studies is that the investigator can control many variables that may confound epidemiological studies. Controllable variables in animal studies include age, sex, environmental conditions such as temperature and relative humidity, genetic constitution, nutrition, and pollutant dose. By controlling dose as a function of exposure duration, dose–response relationships can be developed. Human exposures are also controllable, but to a more limited degree. In addition to those variables described for animals, factors peculiar to humans such as smoking, occupation, and health status are, to some degree, under the experimenter's control.

5.2.2.1 Human Studies

Ideally, toxicological studies of humans should provide the strongest scientific evidence available for establishing a cause–effect relationship between air pollutant exposures and adverse health effects. In such studies, it is possible to expose humans under controlled conditions and monitor them for signs indicating the onset of disease. Most human studies are, however, limited to short-term exposures. Because of the long exposure periods required to induce chronic responses and the irreversibility of some of these responses, most human studies are not conducted at realistic concentrations of pollutants that occur in ambient atmospheres. In addition, ethical considerations, which are imperative when designing human exposure studies, limit the kind of information that can be acquired.

In theory, a true cause–effect relationship can only be developed by human experimentation. Ethical and other limitations make it virtually impossible to conduct such studies.

5.2.2.2 Animal Studies

Animal exposures can provide valuable information on the effects of pollutants when no other acceptable means of obtaining such information are available. Because animals can be sacrificed for pathological and biochemical analyses, they are ideally suited for toxicological studies. Additionally, chronic exposures can easily be conducted.

Use of animals as models for humans does have limitations; animals are not humans. Although they may have the same general organ systems, they may be structurally and physiologically different. In addition, species differ in their sensitivity to pollutants. Dogs, for example, are 10 times more resistant to nitrogen dioxide (NO_2) than rodents. Also, most animals are not as long-lived as humans. Consequently, results obtained from animal studies can only be extrapolated to humans with some degree of uncertainty. Uncertainty is reduced when similar results can be demonstrated in more than one mammalian species, especially primates, and the organ systems under study are closely analogous to those of humans.

To provide observable toxicological responses, exposures used in controlled studies are usually much higher than those occurring in ambient environments. In general, controlled exposures are limited to one toxic pollutant.

5.2.3 Occupational Exposure Studies

Although ethical and legal constraints limit the use of human volunteers for experimental evaluation of the effects of toxic substances, such constraints have not limited the voluntary or involuntary exposure of industrial workers. For many air pollutants of interest, exposures in the work environment occur at much higher levels than in ambient air. Because of potential health risks and legal concerns, some industries maintain detailed exposure and health data on employees. As a consequence, occupational exposures may be relatively well defined and often provide an important source of information on illness development and expression, job-related disease, and dose–response relationships. In the absence of human toxicological studies, occupational exposures often provide the only available human dose–response data.

Studies of occupationally exposed workers have limitations. Although one or more pollutants may be common to both occupational and ambient environments, the overall pollutant mix varies considerably. In addition, occupational exposures are limited to workday and workweek cycles. Community exposures are not only more heterogeneous and variable; they are further complicated by differences in indoor and ambient air quality and the amount of time individuals spend in such environments.

A major limitation of occupational studies is that the population of workers does not reflect the general population. In many industries, workers are nominally healthy males between the ages of 18 and 65. Studies of such workers may provide important information on disease syndromes induced by specific pollutants or pollutant combinations. They cannot, however, provide adequate information on those who are not industrially exposed and who may be at special risk. In such instances, information on health effects on healthy individuals may not be as important as responses of individuals who are more sensitive. Such individuals may be excluded from the work force or, because of existing health problems, be excluded from specific occupations.

5.3 Effect of Pollutants on the Human Body

Pollutant effects are normally manifested in specific target organs. These may be direct; that is, pollutants come in intimate contact with the organ affected. Such is the case for eye and respiratory irritation. Effects may be

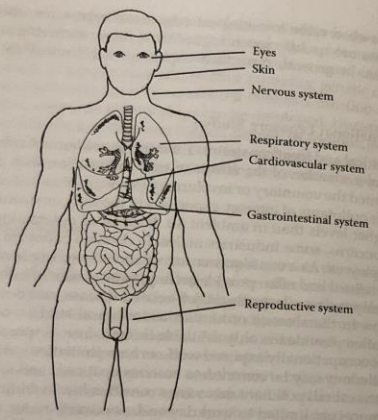

FIGURE 5.2
Target organs for air pollutant exposures.

indirect. For example, pollutants may enter the bloodstream from the lungs or gastrointestinal system by respiratory clearance. Effects may then be distant from the immediate organ of contact. Indeed, a target organ may not have immediate intimate contact with air contaminants. The principal target organs or organ systems are eyes and the respiratory and cardiovascular systems (Figure 5.2).

5.3.1 Eye Irritation

Eye irritation is one of the more prevalent manifestations of pollutant effects on the human body. It is most often associated with exposure to aldehydes and photochemical oxidants. The threshold for eye irritation by oxidants is approximately 0.10 to 0.15 ppmv (reported as ozone [O_3]). Eye irritation increases with increasing oxidant concentration, although O_3 and NO_2, the two principal oxidants, do not cause eye irritation. Apparently, oxidant levels are indicators of the eye irritation potential of photochemical pollutants such as peroxyacyl nitrate (PAN), acrolein, formaldehyde (HCHO), and other photochemically produced compounds. There is some question whether eye irritation should be categorized as a significant health effect because no other

physiological changes are detectable and eye irritation resolves quickly after exposure ceases.

5.3.2 Effects on the Cardiovascular System

Pollutants such as carbon monoxide (CO) and lead (Pb) are absorbed into the bloodstream and may have both direct and indirect effects on the cardiovascular system. These effects will be discussed in subsequent sections. Cardiovascular disease may also result from the indirect effects of other air pollution–incited disease. For example, some individuals die of cor pulmonale, heart failure resulting from the stress of severe chronic respiratory disease. Recent epidemiological studies have shown that premature cardiovascular system–related mortality is strongly associated with exposures to small (≤2.5 μm) particles.

5.3.3 Effects on the Respiratory System

The respiratory system is responsible for gas exchange and, therefore, receives direct exposure to airborne contaminants. Effects on the respiratory system are determined in great measure by its structural and functional anatomy as well as respiratory defense mechanisms.

The principal function of the respiratory system is to supply the body with oxygen (O_2) for metabolism. In addition, it removes waste carbon dioxide (CO_2) from the bloodstream and tissues. These functions are facilitated by the three major units of the respiratory tract: the nasopharyngeal, tracheobronchial, and pulmonary regions (Figure 5.3).

5.3.3.1 Units of the Respiratory System

5.3.3.1.1 The Nasopharyngeal Region

The nasopharyngeal region, or upper airway, consists of nasal passages, nasopharynx, oropharynx, and glottis. It begins at the terminus of the bony turbinates and extends to the region of the vocal cords. The nasal area is subdivided into two cavities by a septum. These cavities, just beyond the nostrils, are ringed with coarse hairs. Projecting from the wall of the cavities are bony turbinates that force inspired air to flow along a winding course where, on contact with the large surface area of the nasal passages, it is warmed and moistened. Nasal surfaces are covered by a mucus layer and hairlike projections called cilia that serve to remove the larger particles in the thoracic range (≤10 μm).

The oropharynx extends from the soft palate to the glottis, located at the junction of the trachea and esophagus, where eating and breathing functions are separated. The glottis is the space between the vocal cords that are found in the larynx, or voice box.

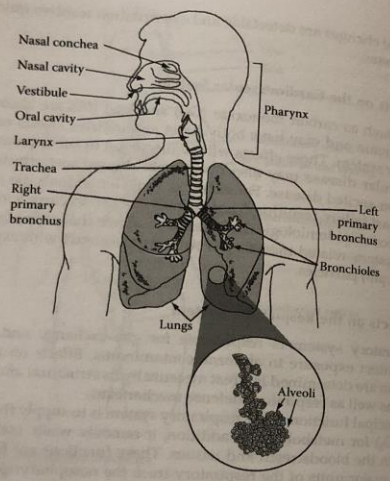

FIGURE 5.3
General anatomy of the human respiratory system.

5.3.3.1.2 *The Tracheobronchial Region*

The tracheobronchial region of the respiratory system is a series of tubes or ducts that transport inspired air to lung tissue, where O_2 and CO_2 are exchanged. This region consists of a large tube, the trachea, which subdivides to form smaller-diameter tubes, bronchi. Bronchi in turn branch to form smaller and smaller airways. From the trachea to the site of gas exchange, there may be as many as 23 to 32 generations of branching, with each generation being smaller in diameter and length. Bronchi comprise the first 12 to 22 branching generations. Their walls have cartilaginous plates and a muscular layer that constricts when foreign substances enter bronchi. Air flows from bronchi into the smaller bronchioles (which do not have cartilaginous plates and are usually <1 mm in diameter). The interior surfaces of the trachea, bronchi, and bronchioles are lined by ciliated cells interspersed with mucus-secreting cells. The mucus layer produced by these cells is moved toward the oropharynx by the rhythmically beating cilia. Terminal bronchioles are the smallest bronchioles to have cilia.

5.3.3.1.3 *The Pulmonary Region*

The pulmonary region of the respiratory system consists of respiratory bronchioles, alveolar ducts, and alveoli (Figure 5.3). Alveoli are membranous air sacs at the terminus of the alveolar ducts. A fully developed lung is estimated to have approximately 300 million alveoli. The large alveolar surface area facilitates efficient exchange of O_2 and CO_2 between the lungs and the blood capillaries that cover the alveoli.

Movement of air into (inspiration) and out of (expiration) the lungs is accomplished by muscles of the chest and diaphragm, which alternately compress and expand the lungs. During inspiration, air pressure in the alveoli decreases, becoming slightly negative. Because of this negative pressure, air flows into the lungs. During expiration, alveolar air pressure rises, causing air to flow out of the lungs.

5.3.3.2 *Defense Mechanisms of the Respiratory System*

The respiratory system is protected from airborne contaminants by a variety of defense mechanisms. In the nasal region, the stiff nasal hairs or impaction on the mucus layer of the winding passages of the turbinates may remove large particles. Cilia sweep the mucus layer and entrapped particles toward the back of the throat where they are swallowed or expectorated. Contaminants may also be removed from upper airways by the sneeze reflex.

Other defense mechanisms serve to remove or prevent contaminants (mainly particles) from entering the tracheobronchial and pulmonary regions. When, for example, particles enter the upper portion of the bronchial tree, muscle layers constrict the bronchi, thus narrowing bronchial diameter and reducing the amount of particles entering the lungs. The cough reflex, similar to the sneeze reflex, assists this process by eliminating particles trapped in mucus in the tracheobronchial region. Cilia that line the tracheobronchial region clean the airways by propelling mucus and particles upward where they are removed by coughing and expectoration or swallowing. In the latter way, some particulate-phase pollutants may enter the gastrointestinal system.

Despite these defense mechanisms, many fine particles and some relatively insoluble gases enter pulmonary tissue. Within the alveoli and bronchioles of the pulmonary region, specialized cells called phagocytes and macrophages ingest deposited matter (phagocytes and macrophages, derived from white blood cells, consume bacteria, viruses, and other particles and, as a result, serve as a pulmonary defense mechanism). These phagocytes and the matter they contain are normally transported out of the lungs in mucus by the cilia. However, they may also move through the alveolar membrane and enter either the lymphatic or circulatory system. Additionally, soluble components deposited in the alveoli may be absorbed by adjacent tissue and enter the lymphatic or circulatory system without being phagocytized.

Although these mechanisms affect clearance from the respiratory system, they may expose other body systems to toxic materials. They may also result in tissue damage when phagocytes are destroyed and spill their contents when attempting to ingest toxic particles.

5.3.3.3 Air Pollution and Respiratory Disease

Community air pollution has been implicated as a causal or aggravating agent in diseases of the respiratory system such as chronic bronchitis, pulmonary emphysema, lung cancer, bronchial asthma, and infections.

5.3.3.3.1 Chronic Bronchitis

Bronchitis is a respiratory disease characterized by inflammation of the membrane lining the bronchial airways. Bronchitis may be caused by pathogenic infections or respiratory irritants, for example, those that occur in cigarette smoke, industrial exposures, and ambient and indoor air pollution.

When bronchial inflammation persists for 3 months or longer, it is classified as chronic bronchitis. Extended irritation of the bronchial membrane is the primary cause of chronic bronchitis. It is characterized by a persistent cough and excessive mucus or sputum production, often accompanied by destruction of cilia and thickening of bronchial epithelium. Chronic bronchitis significantly affects respiratory function, as airway resistance is increased by occlusion of the bronchi, swelling of the inflamed membrane, and concomitant excess mucus production. Consequently, persons afflicted with the disease have difficulty breathing.

Severe cases of chronic bronchitis are often followed by the development of pulmonary emphysema. Combined chronic bronchitis and pulmonary emphysema are usually diagnosed and described as chronic obstructive lung disease (COLD) or chronic obstructive pulmonary disease (COPD).

Some epidemiological studies suggest that ambient pollutants may initiate or aggravate chronic bronchitis. The problem of identifying ambient air pollutants as causal agents in the etiology of chronic bronchitis is confounded by the significant causal role of cigarette smoking in initiating this disease. It is further obscured by the effects of occupational exposures. However, available epidemiological and toxicological evidence suggest that long-term community exposures to a combination of ambient pollutants such as PM and SO_2 may contribute to the initiation of chronic bronchitis.

5.3.3.3.2 Pulmonary Emphysema

Whereas chronic bronchitis is a disease that affects the upper portion of the respiratory airway system, pulmonary emphysema is a disease of lung tissue that normally facilitates gas exchange with blood.

Emphysema is often a disease of older individuals. It is characterized by the destruction or degeneration of walls of the alveolar sacs. As a consequence, the total surface area of pulmonary tissue is reduced, diminishing aeration of blood. Individuals afflicted with emphysema usually develop pulmonary hypertension. In addition to elevation of arterial pressure, pulmonary resistance is increased. Pulmonary emphysema is consequently accompanied by shortness of breath and other breathing difficulties.

Patients with emphysema usually have problems exhaling because air remains trapped in the lungs and overinflates damaged lung tissue. This overinflation contributes to the "barrel chest" characteristic of most patients with emphysema. Exhalation difficulties are due to the compression and collapse of some of the smaller airways and the overall decrease in lung elasticity common to those afflicted by emphysema.

Little epidemiological evidence exists to implicate ambient air pollutants as a contributing factor to the initiation and development of pulmonary emphysema. Animal studies suggest, however, that chronic exposures to NO_2 can initiate preemphysematous lung changes.

5.3.3.3.3 Lung Cancer

Cancer is one of the leading causes of death in the United States, accounting for approximately 370,000, or one in five, deaths each year. Of these, approximately 150,000 are due to lung cancer, which is the leading cause of cancer deaths. Common to all cancers is the characteristic unrestrained cell growth that produces malignant tumors, which have a higher rate of growth than normal tissue that surrounds them. Most cancers metastasize, that is, spread to other parts of the body. The tendency for lung cancer cells to metastasize is particularly great; as a consequence, the prognosis for patients with lung cancer is poor, with an approximate 90% mortality.

Cancer is usually a disease of the elderly and individuals in late middle age. The pattern is due in part to the fact that cancers are commonly latent diseases; that is, there is a delay of several decades or more between the initiation of exposures to a carcinogenic agent and development of the disease syndrome. For lung cancer, the latency period may be as long as 30 years or more, with peak incidence at age 50. The long interval between initial exposure to the carcinogenic (i.e., cancer producing) agent and disease onset makes it especially difficult to conclusively identify a specific causal agent. During this latency period, an individual may undergo changes in occupation, socioeconomic status, place of residency, nutrition, and personal habits such as smoking. These changes tend to confound the epidemiological evaluation of urban air pollution in the etiology of lung cancer. The latency period not only confounds the identification of causal agents in diseases such as lung cancer but also tends to give individuals a false sense of security relative to the safety of a variety of chemical or physical exposures.

The most common form of lung cancer is bronchogenic; that is, it originates in the bronchial lining and invades tissues of the bronchial tree. The malignancy may spread to the rest of the lung and eventually to other parts of the body. Available epidemiological evidence indicates that nearly 90% or more of the approximately 150,000 lung cancer deaths in the United States

each year are caused by chronic exposure to tobacco smoke. Lung cancer has also been linked to passive tobacco smoke exposures and occupational exposures to asbestos, arsenic, and radioactive gases (Rn) and dusts.

A causal role for ambient air pollution in the development of some lung cancers is suggested from several types of epidemiological investigations, including (1) comparisons of urban versus rural populations and migrants from high- to low-air pollution countries, and (2) statistical regression analyses of the relationships between lung cancer deaths and various indices of air pollution.

Comparisons of rural and urban nonsmoking populations indicate that urban lung cancer death rates are approximately twice those found in rural areas. These studies suggest that there is an apparent urban factor that contributes to the overall incidence of lung cancer. However, they do not identify the characteristics of the urban environment that are primarily responsible.

Studies of individuals migrating from countries that had high ambient air pollution levels to those where air pollution levels were much lower indicate that lung cancer rates in such migrants tend to be intermediate between those of the country of origin and their new homeland. Even when differences in smoking habits are considered, lung cancer rates of migrants from historically high-air pollution countries such as the United Kingdom were greater than those from countries such as New Zealand. These data suggest that ambient air pollution exposures may produce effects that persist long after exposures have ceased.

The strongest evidence for a link between ambient air pollution and lung cancer comes from an ongoing prospective mortality study of 1.2 million adults started in 1982. Based on an epidemiological risk factor analysis of 500,000 of these adults, lung cancer mortality was shown to be significantly linked with exposures to fine particles ($PM_{2.5}$), with an 8% increase in lung cancer mortality for each 10 $\mu g/m^3$ increase in $PM_{2.5}$.

5.3.3.3.4 Bronchial Asthma

Asthma is an acute respiratory ailment characterized by the constriction of muscles and swelling of the lining of respiratory airways, excessive mucus production, and increased resistance to airflow. Such reactions are episodic, being described as asthmatic attacks. They may occur suddenly and without warning. Overt symptoms include severe shortness of breath, wheezing, chest tightness, and coughing. Attacks may occur for a few minutes to several hours.

Asthma is a relatively common respiratory ailment with a prevalence rate of approximately 4.3% in the United States, or approximately 10 to 12 million individuals. Asthma prevalence in children is relatively high, with a rate of 8.3% in midwestern children and 11.8% in southern children ages 3 to 11 years. Highest asthma rates are reported for African-American children (13.4%). The overall prevalence rate of asthma has increased by 50% since 1980, whereas the annual death rate has doubled to 5,000 per year.

Asthma has been strongly linked with exposure and sensitization to inhalant allergens. There is limited evidence that exposure to air pollutants may potentiate allergenic responses that cause asthma.

Asthmatic attacks may be caused by exposure to inhalant allergens and nonspecific irritant gas-phase and particulate-phase substances. In controlled exposures, SO_2 has been observed to cause asthmatic attacks, and a number of epidemiological studies have linked admissions to hospital emergency rooms for treatment of asthmatic symptoms with high-O_3 pollution days. Increased asthmatic symptoms are reported for children in "asthma camps" on such days. Recent studies indicate that O_3 exposures not only initiate asthmatic attacks but also contribute to asthma development.

5.3.3.3.5 Respiratory System Infections

A number of animal studies over the past three decades have shown that exposure to pollutants such as O_3, NO_2, SO_2, and PM increases the risk of respiratory system infections. Such infections include pneumonia and bronchitis and are primarily caused by bacteria. The effect that pollutants have on viral infections such as influenza is less clear.

The primary factors involved in increased infection risks may include modulation of pulmonary immune cells (e.g., macrophages), lung surfactant, and physical factors such as ciliary beating and particle clearance. Because of differences in exposure conditions and sensitivity, it is difficult to extrapolate infection risks from animals to humans. Nevertheless, results from human exposure studies and retrospective epidemiological studies of air pollution episodes strongly suggest that young and aged members of an exposed population are at greatest risk of infection, particularly of the upper respiratory tract.

5.4 Health Effects of Regulated Air Pollutants

Although polluted atmospheres may contain hundreds of different substances, only a relatively small number have been identified as having the potential to cause adverse health effects under community-wide exposure conditions encountered in ambient environments. Most notable are those for which National Ambient Air Quality Standards (NAAQSs) have been promulgated. These are CO, sulfur oxides (SO_x), O_3, PM with aerodynamic diameters of ≤10 and ≤2.5 μm (PM_{10} and $PM_{2.5}$, respectively), NO_2, and Pb. Although nonmethane hydrocarbons (NMHCs) are regulated under NAAQSs, their regulation is not based on any human health risks. Rather, they are regulated to achieve the O_3 standard. A large number of other substances, commonly referred to as air toxics, are regulated under National Emissions Standards for Hazardous Air Pollutants (NESHAP) and Air Toxics provisions of clean

air legislation. Most pollutants regulated under NESHAP and Air Toxics provisions are associated with sources that result in localized, rather than community-wide, exposures. Because of their ubiquitous presence, population exposure potential, and potential to cause adverse health effects, pollutants regulated under NAAQS provisions are discussed here in detail.

5.4.1 Carbon Monoxide

Potentially harmful exposures to CO occur from a variety of sources and environments. At very high concentrations (>1,000 ppmv), CO exposures may be lethal, with death resulting from asphyxia. At lower concentrations (several hundred ppmv), CO may cause neurological-type symptoms, including headache, fatigue, nausea, and, in some cases, vomiting. Asphyxiation and sublethal symptoms are usually caused by exposures associated with poorly vented combustion appliances, idling motor vehicles in closed environments, excessive CO production and inadequate ventilation associated with occupational industrial activities, and smoke inhalation from structural fires. Carbon monoxide poisoning, although not uncommon, represents exposures that are considerably higher than those that occur in the ambient air of our cities and towns, the kind of exposure conditions addressed by the NAAQS for CO. Such community exposures rarely exceed the 35-ppmv, 1-h standard.

The principal mechanism of CO toxicity is tissue hypoxia associated with preferential bonding of CO with hemoglobin (Hb) to form carboxyhemoglobin (COHb). Carbon monoxide has an Hb affinity 240 times that of O_2. In addition, CO binds more tightly with Hb than does O_2. As a consequence, the blood's capacity to carry O_2 is decreased. Carbon monoxide also binds to several intracellular proteins, which may result in extravascular effects.

The quantity of COHb formed depends on a variety of factors, for example, the concentration and duration of CO exposure, exercise, temperature, health status, and metabolism of the individual exposed. At low exposure concentrations, the quantity (%) of Hb tied up by CO is a linear function of exposure concentration and duration (Figure 5.4). Note the effect of exercise on lung ventilation rate and COHb formation in blood.

The formation of COHb is reversible. Depending on initial COHb levels, the elimination half-time is approximately 2 to 6.5 h. Because elimination is slow, COHb levels in blood may increase on continuous exposure. Approximately 10% to 50% of the body store of CO is external to the vascular system.

The primary health concerns associated with low-level ambient (<50 ppmv) exposures to CO are effects on the cardiovascular system (particularly in sensitive individuals) and neurobehavior. There are more than 500,000 deaths in the United States each year from heart attacks, and 5 to 7 million people are estimated to have a history of heart attack, angina (heart-related chest pain), or both. Scientific support for the current CO NAAQS comes from studies of

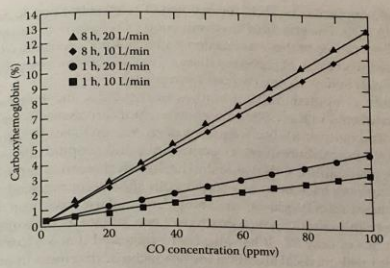

FIGURE 5.4
COHb levels in the blood as a function of exposure concentration, duration, and ventilation rate. (From U.S. EPA, EPA/600/8-90/045f, EPA, Washington, DC, 1991.)

patients with stable angina associated with cardiovascular disease. The lowest observed physiological effect level in patients with exercise-induced ischemia (tissue oxygen deficiency) is somewhere between 3% and 4% COHb. Baseline values in nonexposed nonsmokers are approximately 0.5% to 0.7%. Because nonsmokers may be exposed from a variety of sources, average COHb levels are approximately 1%; COHb levels in smokers vary from 3% to 8%, with an average of approximately 4%.

The literature on the effects of CO on the cardiovascular system is extensive. Many studies have been confounded by the effects of cigarette smoking. Cigarette smoking significantly increases the risk of atherosclerosis and myocardial infarction (heart attack). It is not certain whether chronically elevated COHb levels in smokers are major causal factors for these effects. It has been suggested that chronic exposure to CO may cause atherosclerosis, whereas acute exposures may trigger myocardial arrythmias and infarction. Evidence that CO exposure from sources other than smoking can produce chronic effects is limited.

A number of clinical studies of volunteers have been conducted over the past several decades. Of note are results of the Health Effects Institute's (HEI) multicenter study. Carbon monoxide exposures sufficient to elevate COHb to 2% produced significant angina-related effects in exercising patients with coronary artery disease. A dose–response relationship was observed, with no obvious threshold. The significance of the HEI study is that exposure at the 8-h CO NAAQS of 9 ppmv is sufficient to produce a COHb level of 2.1%. If one assumes that 10% of the US population may reach a COHb concentration of 2.1% (independent of tobacco smoking), and couples this with the widespread prevalence of coronary disease, it is apparent that a significant

number of people may be at risk even when CO concentrations do not exceed the 8-h NAAQS. The effects of environmental CO exposures on smokers are likely to be additive to those associated with smoking. Smokers, who are, as a group, at greater risk of coronary disease, would therefore be subject to an increased risk of developing coronary symptoms.

A number of epidemiological studies in the 1970s and better-designed studies conducted in the 1990s have shown significant associations between CO levels and daily mortality, frequency of admission to hospital emergency departments for cardiorespiratory complaints, and hospital admissions for congestive heart failure. Other potential health effects such as chronic flu-like illness symptoms have been associated with short-term repeated exposure to CO. Complaints of headache, irritability, and malaise have been described for children and adults. Symptoms have been shown at measured COHb concentrations of only 5%. It has also been suggested that 3% to 5% of individuals who seek medical attention for headache or dizziness in urban hospital emergency rooms may be suffering from effects of CO exposure. Such effects have been described as "chronic occult CO poisoning syndrome." The nature of exposures responsible for this syndrome is unknown. It is probable that they may be associated with malfunction or improper operation of gas furnaces, stoves, or ovens, augmented by ambient exposures.

The brain is the body organ most sensitive to reduced tissue O_2 levels associated with CO exposures. Although it can, under normal circumstances, initially compensate for CO-induced hypoxia by increased blood flow and tissue O_2 extraction, some neurological or neurobehavioral effects can be anticipated. Behaviors that require sustained attention or performance seem to be very sensitive to COHb-induced effects. Human studies of hand–eye coordination, vigilance, and continuous performance have reported consistent relationships with COHb as low as 5%. The effects at lower COHb values seem to be minimal.

Neurological and cardiovascular effects clearly demonstrated to be associated with different COHb blood concentrations are summarized in Table 5.1.

TABLE 5.1

Neurological and Cardiovascular Responses of Humans at Various COHb Saturation Levels

Blood COHb (%)	Effect
0–1	None known
2.0	Quicker onset of angina pain in exercising patients; pain duration lengthened
2.5	Impairment of time interval discrimination
3.0	Change in relative brightness thresholds
4.5	Increased reaction time to visual stimuli
10	Performance changes in driving simulation
10–20	Headache, fatigue, dizziness, coordination loss

Source: NACPA, USDHEW, Publication AP-62, Washington, DC, 1970.

The levels of COHb required to produce these changes are likely to occur only in worst-case atmospheric conditions in the United States, that is, in downtown areas with heavy traffic and very poor atmospheric mixing conditions. They are also likely to occur in underregulated environments of large cities in developing countries. High-population, high-altitude Mexico City would be particularly at risk.

The NAAQS for CO, like other NAAQSs, is designed to protect the most sensitive populations; in this case, individuals with existing cardiovascular disease. Based on both theoretical and experimental research, other populations may be at risk. These would include the fetus, infants, the elderly, individuals with preexisting disease that decreases the availability of O_2, and individuals using certain medications and recreational drugs. Exposure to other pollutants and residence at high altitude may also increase CO exposure health risks.

5.4.2 Sulfur Oxides

The sulfur oxides include both gas-phase and particulate-phase chemical species. Of these, only SO_2 is present in sufficient ambient concentrations to be a public health concern. Particulate-phase SO_x includes strongly to weakly acidic substances such as H_2SO_4 and its neutralization products, such as acid ammonium sulfate (NH_4HSO_4) and ammonium sulfate ($[NH_4]_2SO_4$); most acid sulfate studies have focused on H_2SO_4.

5.4.2.1 Sulfur Dioxide

Much of the available information on acute and chronic effects of SO_2 is based on laboratory animal studies conducted at exposure concentrations considerably higher than those found in polluted atmospheres. Because of its solubility in watery fluids, SO_2 is efficiently removed in the upper respiratory tract. It forms sulfurous acid (H_2SO_3), which readily dissociates into bisulfite and sulfite ions that move into the systemic circulation. Less than 1% of inspired SO_2 reaches the alveoli. Exposure of the lower airways and alveoli increases during exercise, mouth breathing, and under low ambient SO_2 levels. The major physiological effects of SO_2 are changes in mechanical function of the upper airways, including an increase in nasal flow resistance and a decrease in nasal mucus flow rate. Animal studies indicate that SO_2 exposures can adversely affect pulmonary defense mechanisms such as mucociliary transport, alveolar clearance of particles, and macrophage function. There is some evidence that exposures may cause chronic bronchitis.

When the primary air quality standard for SO_2 (0.03 ppmv [80 μg/m^3] annual mean, 0.14 ppmv [365 μg/m^3] maximum 24-h concentration) was promulgated in 1971, no health effects had been reported for short-term exposures (<1 h) at levels observed in the ambient environment (≤1 ppmv, 2.67 mg/m^3). In 2010, the U.S. Environmental Protection Agency (U.S. EPA) revised the

primary SO_2 standard by establishing a new 1-h standard at a level of 75 ppb. Since 1980, numerous challenge studies conducted on asthmatic individuals have shown that when asthmatics and others with hyperactive airways are briefly exposed to SO_2 at concentrations of 0.25 to 0.50 ppmv (0.66–1.3 mg/m³), they exhibit acute responses characterized by bronchoconstriction (airway narrowing) with increased airway resistance and decreased forced expiratory flow rate, and clinical symptoms of shortness of breath and wheezing. Bronchoconstriction occurs within 5 to 10 min of exposure and is brief in duration, with lung function returning to normal for most subjects within an hour of exposure. Such responses occur even at lower SO_2 levels during moderate exercise. This results from increases in breathing rate and oral breathing and thus exposure. Oral breathing increases SO_2 penetration to the lower respiratory tract and results in airway drying.

Mild and moderately asthmatic children and adults are at greatest risk for short-term SO_2-induced respiratory effects. Individuals with more severe asthma are at lower risk because their low exercise tolerance deters them from engaging in sufficiently intense exercise that could cause any effects.

A substantial percentage (20%–25%) of mild and moderately asthmatic individuals exposed for 5 to 10 min to concentrations of 0.6 to 1.0 ppmv (1.60–2.67 mg/m³) of SO_2 during moderate exercise or activity would be expected to have changes in respiratory function and symptom induction that clearly would exceed those associated with daily variation in lung function or in response to other stimuli. At SO_2 exposures of less than 0.5 ppmv, the severity of effects may be sufficient to cause disruption of ongoing activities, increase use of bronchodilator medication, and possibly increase the need for medical attention. Less than 20% of mild and moderately asthmatic individuals exposed to 0.2 to 0.5 ppmv of SO_2 during moderate exercise are likely to experience lung function changes greater than they experience daily. Such responses are less likely to be perceptible and of immediate public health concern.

Available epidemiological data do not show any evidence of excess asthma mortality associated with SO_2 exposures in urban areas. Viewed from a national perspective, it seems that the probability of asthmatic individuals being exposed to peak levels of SO_2 greater than 0.25 ppmv for 5 to 10 min is low. However, some monitoring data indicate that peak SO_2 concentrations higher than 0.25 ppmv do occasionally occur, suggesting that mild and moderately asthmatic individuals residing in the vicinity of major sources may be at increased risk.

5.4.2.2 Acid Sulfate

Sulfuric acid is a strong irritant. However, consistent effects on pulmonary function or respiratory symptoms have not been shown in acute exposure studies of healthy adults. There is some evidence that asthmatic individuals are more sensitive to the effects of acid sulfate on lung mechanical function.

They may experience moderate bronchoconstriction after exposure to H_2SO_4 at concentrations of less than 1 mg/m³. These effects are not as consistent or dramatic as those reported for SO_2 exposures. Children with allergic asthma seem to be more sensitive to H_2SO_4 compared with older asthmatics.

Sulfuric acid, like SO_2, can affect pulmonary defense mechanisms. Acute exposures as low as 0.1 mg/m³ have been shown to alter mucociliary transport in normal humans. Clearance of deposited particles seems to be stimulated at low concentrations and retarded at higher concentrations. Sulfate exposure can also adversely affect macrophage functioning. There is also some evidence that acid sulfates can enhance sensitization to potentially allergenic substances and modulate the activity of cells involved in allergic responses.

Biological responses to acid sulfates are likely due to the deposition of hydrogen ions (H^+) on the surface tissues of respiratory airways. The relative potency of acid sulfates has been shown in toxicological studies to be directly related to its degree of acidity (increased potency with increased H^+ concentration). Because H^+ may be responsible for acid sulfate toxicity, the biological response is likely to be affected by how it is inhaled. Acid sulfates are more likely to be neutralized by ammonia (NH_3) as a result of mouth, as compared with nasal, breathing.

There is insufficient evidence to definitively establish a causal relationship between acid aerosol exposure and adverse health effects. This may reflect, in part, the fact that few epidemiological studies have included measurements of H^+ and that acid aerosols vary colinearly with PM_{10} and $PM_{2.5}$ concentrations. Long-term and population-based annual assessments of mortality (comparing different communities) indicate only a weak effect of acid aerosol. Recent studies with direct measurements of acid aerosols and other major pollutants have shown consistent relationships between acid aerosol levels and hospital admissions for respiratory disease. Acid aerosol has also been related to a significantly higher prevalence of bronchitis in children and reduced lung growth (determined from lung function measurements) as a result of chronic exposures.

5.4.3 Particulate Matter

Before atmospheric particles can cause adverse health effects, they must enter and be deposited in the human respiratory system. Respiratory penetration and deposition depend on the aerodynamic size of particles, respiratory defense mechanisms, and breathing patterns. Particles can be described as inhalable (IPM), thoracic (TPM), or respirable (RPM), based on their penetration and potential for deposition. Size ranges and fractional penetration of IPM, TPM, and RPM are illustrated in Figure 5.5. Inhalable PM can enter the upper airways of the head; TPM, the airways and gas exchange regions of the lung. Respirable particles are a subset of TPM with a high probability of being deposited in the pulmonary region.

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Slow particle clearance is considered to be detrimental because harmful substances are in contact with sensitive tissue for longer periods.

Until the late 1970s and early 1980s, it was widely held that exposures to high concentrations of PM that were prevalent in the United States and Europe before 1970 were a significant health hazard and that evidence for adverse effects at the much lower concentrations present in the atmosphere after 1970 was relatively weak.

By the mid-1980s and 1990s, investigators began to report significant associations between ambient PM levels and various adverse health effects. These reports included (1) the Harvard Six Cities Study, which showed that long-term PM exposure was associated with increased risk of respiratory illness in children and cardiopulmonary mortality in adults; (2) the Utah Valley studies, which showed that PM levels were significantly associated with various illness indices, including respiratory hospitalizations, lung function changes, respiratory symptoms, school absenteeism, and premature mortality; and (3) a series of studies that showed a relationship between daily changes in ambient PM levels and mortality.

Most recent epidemiological investigations have focused on short-term, acute exposures. They show consistent effects across a range of related health outcomes. A summary of acute effect estimates as a percentage change with a 10 $\mu g/m^3$ increase in PM_{10} is presented in Figure 5.7. These indicate that a 10 $\mu g/m^3$ increase in average 24-h PM_{10} concentration is associated with an increase of approximately 0.8% in daily mortality, a somewhat larger association with cardiovascular mortality, and a much larger association with respiratory mortality. Increased hospitalizations and related health care visits are significant for various respiratory diseases and, to a lesser extent,

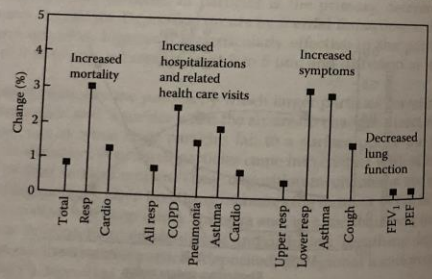


FIGURE 5.7
Estimated percent change in health endpoints associated with a 10 $\mu g/m^3$ increase in PM_{10} levels in acute exposure epidemiological studies. (From Pope, C.A., in *Air Pollution and Health*, Holgate, S.T., Samet, J.M., Koren, H.S., and Maynard, R.L., Eds., Academic Press, San Diego, 1999, p. 688. With permission.)

cardiovascular disease. Increased symptom prevalence is associated with PM_{10} levels; these include lower respiratory system symptoms, asthma, and cough.

Chronic studies indicate that long-term exposure to PM, particularly in the small particle size range ($PM_{2.5}$), is associated with various cardiac and pulmonary health effects (Figure 5.8). Total mortality seems to increase by 2% to 4% for every 5 $\mu g/m^3$ increase in $PM_{2.5}$, with somewhat larger associations for cardiopulmonary mortality. Chronic exposure studies have also reported significant increases in respiratory disease (bronchitis) and small declines in lung function.

Although epidemiological evidence linking both acute and chronic PM exposures to significant health effects is strong, toxicological mechanisms for these effects are less clear. Considerable research has focused on the biological mechanisms that may be responsible for the cardiopulmonary and respiratory effects reported in epidemiological studies.

Associations of mortality and morbidity (illness) with ambient PM levels suggest that a variety of tissues may be affected by particles in the PM_{10} range in ways that cause lung disease and systemic effects. Several biologically plausible mechanisms have been proposed to explain these results. They consistently include some form of inflammatory response. Because the production of clots in coronary vessels (coronary thrombosis) is the cause of myocardial infarction (heart attack), the apparent causal relationship between coronary thrombosis and particle deposition in the airways is somewhat problematic. It has been proposed that inflammatory responses in the lungs produce procoagulant factors locally or as a result of mediators

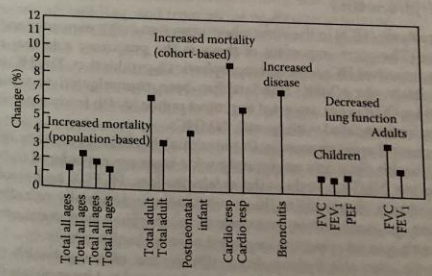


FIGURE 5.8
Estimated percent change in health endpoints associated with a 5 $\mu g/m^3$ increase in $PM_{2.5}$ levels in chronic exposure studies. (From Pope, C.A., in *Air Pollution and Health*, Holgate, S.T., Samet, J.M., Koren, H.S., and Maynard, R.L., Eds., Academic Press, San Diego, 1999, p. 694. With permission.)

that, when released, act on the liver to increase the levels of procoagulant factors. There is limited epidemiological evidence to support this, as increased blood viscosity has been observed at elevated pollutant levels.

Ultrafine particle (UFP; ≤0.01 μm in diameter) research provides a biologically plausible explanation for the toxicity of ambient particles. Such particles induce significant toxic effects in lung tissue even when they are formed from materials that are not toxic as larger respirable particles. Factors that affect or are associated with UFP toxicity include surface area and chemistry, particle number, oxidative stress, and particle interstitialization (i.e., movement of particles from lung spaces into surrounding tissues).

The large surface area of UFPs seems to facilitate the adsorption and absorption of substances from the environment or from the fluid of lung epithelia onto particle surfaces, which then increases particle reactivity. Iron (Fe), one such substance, produces reactive oxygen species. Macrophages attempting to phagocytize UFPs seems to be stimulated to release inflammatory mediators such as tumor necrosis factor, and on sustained stimulation, epithelial cells release chemokines that contribute to inflammation. UFPs present in large numbers commonly undergo interstitialization and therefore cannot be removed by normal pulmonary clearance mechanisms.

Particles may cause lung tissue injury as a result of oxidative stress. The production of free radicals (including hydroxyl radical, OH) may occur in response to exposures to small particles. Oxidative stress seems to result from the localized release of high concentrations of transition metals that stimulate the release of reactive oxygen species.

5.4.4 Hydrocarbons

Hydrocarbons (HCs) in the atmosphere pose two major exposure and health concerns. The more important of these is the precursor role that reactive (and less reactive) HCs have on tropospheric O_3 production. The HC or, more appropriately, the NMHC standard has been promulgated to achieve the NAAQS for O_3; it is not designed to protect public health from exposure risks that may be associated with specific NMHCs.

As a general rule, exposures to individual NMHC species are relatively low and health risks on a community-wide basis are considered to be small to negligible. NMHCs such as the aldehydes (e.g., HCHO) and acrolein are potent irritants. Exposure to these substances, individually or collectively, may cause eye, nose, throat, and sinus irritation. Such symptoms are transitory, resulting in no apparent long-term adverse health effects.

Polycyclic aromatic hydrocarbons (PAHs) are of some health concern. The PAHs include a group of substances characterized by multiple benzene rings. They are commonly produced as a result of incomplete combustion of coal and wood and, to a lesser extent, other fuels. The PAHs are carcinogenic, with benzo-α-pyrene being one of the most potent carcinogens and common PAHs found in aerosol samples.

As a result of fuel use changes, PAH levels in urban areas in the United States have declined to about one-third of the levels present in the mid-1950s. Similar declines have been reported for northern Europe. Although PAHs have been suspected of contributing to increased lung cancer rates in urban areas, epidemiological studies have not shown such a definitive relationship.

Many NMHCs are regulated as hazardous or toxic air pollutants. One of these is benzene, one of the seven pollutants initially regulated under the hazardous pollutant provision of the 1970 Clean Air Act (CAA) Amendments. They also include most of the 182 other substances regulated under the Air Toxics provision of the 1990 CAA Amendments.

Pollutants regulated under Hazardous/Toxics provisions include substances that vary in their toxicological properties and potential risks to humans and the environment. Many are suspected human carcinogens, mutagens, teratogens, and the like; some are irritants, and some neurotoxic.

5.4.5 Nitrogen Oxides

Nitric oxide is a relatively nonirritating gas believed to pose little or no health risk at ambient exposure levels. Its importance lies in the fact that it is rapidly oxidized to NO_2, which has a much higher toxicity.

Unlike SO_2, which is rapidly absorbed in fluids of the upper tracheobronchial region, NO_2 is less soluble and thus passes into the pulmonary region where tissue damage may occur. In individuals occupationally exposed to high NO_2 levels, adverse effects such as pulmonary edema only manifest themselves hours after exposure has ended. Elevated NO_2 exposures have been known to occur in the manufacture of nitric acid (HNO_3), in farm silos, from electric arc welding, and from use of explosives in mining.

In toxicological studies of animals, abnormal pathological and physiological changes have only been observed at concentrations higher than those found in ambient environments. At the lowest NO_2 exposure levels at which adverse effects have been detected (0.5 ppmv, 1,000 μg/m^3), pathological changes have included destruction of cilia, alveolar tissue disruption, and obstruction of respiratory bronchioles. The exposure of rats and rabbits at higher levels have caused severe tissue damage resembling emphysema.

Results of animal toxicological studies support the hypothesis that NO_2 may be a causal or aggravating agent in respiratory infections. There is evidence that NO_2 can damage respiratory defense mechanisms, allowing bacteria to multiply and invade lung tissues. In mice, hamsters, and monkeys infected with pneumonia-causing bacteria, exposure to elevated NO_2 levels resulted in reduced ability to clear bacteria from the lungs, reduced life span, and increased mortality.

Epidemiological studies have been conducted to determine whether NO_2 exposures at ambient levels are associated with respiratory symptoms and disease or other adverse health effects. Most have focused on the potential effects of either outdoor or indoor NO_2 exposures on children's health. A few

have attempted to evaluate the potential adverse effects of combined indoor and outdoor exposures.

Results from a variety of outdoor exposure studies on children's health have been reported. The earliest of these (known as the Chattanooga School Children Study) investigated potential health effects associated with emissions of NO_2 from a trinitrotoluene (TNT)-manufacturing facility. Lung function (ventilation rate) of second-grade children in the high-NO_2 area was significantly lower than that of children in the control area. Respiratory illness rates of families in the exposed area were also higher. Later studies indicated that respiratory symptom duration increased with increasing annual mean NO_2 concentrations, more frequent hospitalizations with increasing 2-year means, and more treatments in hospitals for lower respiratory illness with higher lifelong NO_2 exposures in female children. Similar results have also been reported for Swedish school children in 10 communities where chronic cough and respiratory infections (bronchitis or pneumonia) were associated with increased annual mean NO_2 exposures.

Studies of adults have yielded mixed results. Positive associations between NO_2 and respiratory illness have been reported in some investigations but not in others. In the European Study on Air Pollution and Health (APHEA), significant positive associations were observed between daily death rates and NO_2 concentrations. An increase of 25 ppbv (50 μg/m³) 1-h maximum concentration was associated with a 1.3% increase in the daily number of deaths. Other less definitive studies indicate that increased death rates are associated with increased NO_2 exposures.

Significant public health concern was expressed relative to potential health effects of NO_2 exposures on children's health after the publication of a 1970s epidemiological study in the United Kingdom that observed increased respiratory symptoms among children living in homes using gas cooking stoves (compared with those using electric stoves).

In 1992, the U.S. EPA conducted a review and meta-analysis of 11 epidemiological studies that evaluated potential effects of indoor NO_2 exposures on respiratory illness among children younger than 12 years of age. Relatively good agreement was observed among these studies, with an approximate 20% increase in respiratory illness associated with the presence of gas cooking stoves in households. This increase in respiratory symptoms was described as being equivalent to a 15 ppbv (30 μg/m³) increase in NO_2.

In a recent residential Australian study, the presence of a gas stove and exposure to NO_2 were observed to be significant risk factors for asthma and respiratory symptoms. The presence of a gas cooking stove in a household increased the likelihood of asthma threefold and respiratory illness twofold.

Several studies indicate that NO_2 exposures associated with kerosene heater usage increase the risk of respiratory illness in children younger than 7 years of age. Nitrogen dioxide exposures of more than 16 ppbv (32 μg/m³) were reported to more than double the risk of lower respiratory symptoms

and illness (fever, chest pain, productive cough, wheeze, chest cold, bronchitis, pneumonia, or asthma) compared with children at lower levels of exposure in nonkerosene stove–unused households. Increased risk of upper respiratory symptoms (sore throat, nasal congestion, dry cough, croup, or head cold) was also reported.

The annual average NAAQS for NO_2 in the United States is 53 ppbv (106 μg/m³). The World Health Organization (WHO), however, recommends a much lower annual average guideline (21 ppbv, 42 μg/m³), as well as a 1-h guideline of 100 ppbv (200 μg/m³). These recommendations indicated that the US NAAQS for NO_2 may not have been sufficiently protective of public health and that a short-term standard was needed to protect individuals (particularly children) from peak exposures. In 2010, the U.S. EPA adopted a 1-h NAAQS of 100 ppbv.

5.4.6 Ozone

Ozone is one of the most ubiquitous and toxic pollutants found in ambient atmospheres. Depending on the year, approximately 20% or more of the US population lived in areas where the 1-h NAAQS (regulatory requirement prior to 2004) of 120 ppbv (235 μg/m³) was exceeded.

Pathological and physiological effects of O_3 exposures (at the high end, or higher, of those reported in urban areas) have been evaluated in animal (usually rodent) exposure studies. Ciliated cells in respiratory airways as well as squamous epithelial cells in alveoli were injured or killed. This was followed by cell proliferation and tissue repair, with a loss of damaged epithelial cells and formation of exudates (fluid) and inflammatory cells. Longer-term exposures are characterized by continued hyperplasia (excess epithelial cell proliferation), low-grade inflammation with exudate, and synthesis of collagen (scar-forming substance).

Ozone may injure tissue membranes by oxidizing amino acids, sulfhydryl (SH) groups on enzymes and other proteins, and polyunsaturated fatty acids (lipids). This lipid peroxidation and resultant production of free radicals increases membrane permeability, with a concomitant leakage of essential electrolytes and enzymes. It also results in swelling and disintegration of cell organelles such as lysosomes and mitochondria, and inhibition of metabolic pathways. Significant increases in activity of various antioxidant enzymes and enzyme systems have been reported at continuous exposure concentrations in the range of 0.4 to 0.8 ppmv (783–1,567 μg/m³).

At the physiological level, O_3 exposures result in significant lung function changes. These include increased respiratory rates and pulmonary resistance, decreased tidal volume, and changes in respiratory mechanics. Changes seem to be transient, returning to normal after exposure ceases or after several days of adaptation to continuous exposures. Such changes are dose-dependent and increase with exercise-associated lung ventilation.

12

Environmental Noise

Environmental noise associated with human activities is a phenomenon that characterizes the acoustical environment and our perceptions of it in the communities in which we live. Noise is sound energy that is objectionable because of its physiological and psychological effects on humans.

Humans vary in their reception and perception of sound. As such, the concept of noise can and does vary from one individual to another. What is an acceptable or unacceptable sound, or level of it, depends on the individual exposed. Nevertheless, there are sound types and exposure levels that most individuals agree are not acceptable and, therefore, noise. Major noise sources include transportation, industry, construction, buildings and households, and humans and pets. In most cases, noise is produced as a result of human activities.

Noise in industrial, construction, and military environments poses unique exposure concerns because it has the potential to cause hearing loss. Noise exposure in industrial and construction environments is regulated by governmental agencies whose mission is to protect worker health (in the United States, the federal Occupational Safety and Health Administration [OSHA] and a number of state agencies). These occupational safety and health concerns differ from noise exposures in the ambient environment, that is, environmental or community noise.

Noise is treated in this book as a form of ambient or atmospheric pollution because the sound energy that is the cause of noise is primarily transmitted through the air environment.

12.1 Sound and Its Measurement

Sound is a form of energy produced by vibrating objects or aerodynamic disturbances. The former includes our vocal cords and operating motors, automobiles, and airplanes; the latter, thunder, sonic booms, and air movement.

Disturbance of air molecules by sound energy produces variations in normal atmospheric pressure. As these pressure variations reach our ears, they cause the eardrums to vibrate. Transmission of vibrations to the inner ear, and their interpretation by the brain, results in our sensory perception of sound. Sound can only be transmitted through a medium that contains molecules; that is, it cannot move through a vacuum. The speed of sound

depends on the density of the transmission medium, with increased speed associated with increased density. The speed of sound in air, water, and steel is 343, 1,482, and 5,030 m/s (1,125, 4,860, and 16,500 ft/s), respectively.

12.1.1 Sound Energy

When objects vibrate, they radiate acoustical energy. The energy produced by a source is described as sound power. In conventional use, sound power (Sp) values are expressed in dimensionless units called decibels (dB), calculated from the following equation:

$$\text{Sp} = 10 \log_{10}(W_m/W_r)\ \text{dB} \tag{12.1}$$

where W_m is the measured sound energy in watts and W_r is the reference sound energy, 10^{-12} W. Sound power is the logarithm of the ratio of the measured and reference sound energy multiplied by 10.

When sound energy passes through a given area, its flow is described as sound intensity (SI), expressed as watts per square meter (W/m^2). Sound intensity values are also expressed in decibels and are calculated similarly to Sp:

$$\text{SI} = 10 \log_{10}(I_m/I_r)\ \text{dB} \tag{12.2}$$

where I_m is the measured sound energy and I_r is the reference sound energy, 10^{-12} W/m^2.

When sound energy radiating from a source strikes a surface, it induces a pressure. This pressure can be measured by instruments known as sound pressure level meters. Sound pressure values are also expressed in decibels. Calculation of sound pressure differs slightly from those for Sp and SI. Because pressure in sound waves varies sinusoidally, the mean square values of the pressure changes are used to calculate sound pressure levels (Spl):

$$\begin{aligned}\text{Spl} &= 10 \log_{10}\left(P_m^2/P_r^2\right)\text{dB} \\ &= 20 \log_{10}(P_m/P_r)\ \text{dB}\end{aligned} \tag{12.3}$$

In calculations of Spl, the reference pressure (P_r) is the threshold of human hearing, 2×10^{-4} μbars or 2×10^{-5} N/m^2, where N is newtons. P_m is the measured sound pressure. For purposes of illustration, let us calculate the Spl when $P_m = 2.0$ μbars.

$$\begin{aligned}\text{Spl} &= 20 \log_{10}(2.0/0.0002)\ \text{dB} \\ &= 20 \log_{10}(10,000)\ \text{dB} \\ &= 20\,(4)\ \text{dB}\end{aligned}$$

As an object vibrates, sound waves radiate outward in an ever-expanding sphere. At increasing distances, Sp remains the same, but SI declines as the area through which sound energy passes increases. In a free field (space where sound is not reflected), SI and Spl are reduced by 6 dB (factor of 4) each time the distance from the source is doubled. This relationship is illustrated in Figure 12.1. At a distance of two radii ($2r$), the area of the sound sphere is four times as large as the area at a distance of one radius ($1r$). The change in sound intensity and pressure follows the inverse square law:

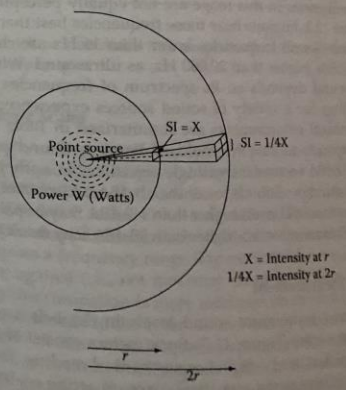
$$\text{SI} = W/4\pi r^2 \tag{12.4}$$

where W is watts and $4\pi r^2$ is the area of the sound sphere.

Each time the radius doubles, the area of the sound sphere increases by $4\pi r^2$ of its previous value and SI decreases inversely with this increase in area.

Sound produced by a source is emitted in the form of waves. These waves are characterized by alternating compressions (wave peaks) and rarefactions (wave troughs) and vary in length, frequency, and amplitude. The shorter the wavelength, the more frequent the waves are per unit of time. The higher the wave (increased amplitude), the more energy it has.

Frequency is a major characteristic of sound, and its discrimination by the human auditory organ constitutes the phenomenon of hearing. Frequency



FIGURE

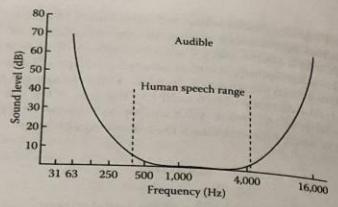


FIGURE 12.2
Frequency range of human hearing.

is expressed in cycles (waves) per second or hertz. If 1,000 complete sound waves pass a point per second, that sound has a frequency of 1,000 Hz.

A sound source does not usually produce discrete frequencies. Most sounds include a range of frequencies characterized by dominant frequencies. Male human speech, for example, is characterized by dominant frequencies lower than 2,000 Hz; for female speech, the range of dominant frequencies is somewhat higher.

Humans can hear sounds in the frequency range of 20 to 20,000 Hz. However, frequencies in this range are not equally perceptible. As can be seen in Figure 12.2, humans hear those frequencies best that correspond to human speech. Sound frequencies lower than 16 Hz are characterized as infrasound; those higher than 20,000 Hz, as ultrasound. Whether humans can hear a sound depends on its spectrum of frequencies and intensity. Decibel readings for a variety of sound sources experienced in the home, work, and ambient environments are summarized in Table 12.1. Note that 0 dBA corresponds to the threshold of hearing. Sound pressure levels higher than 70 dBA become increasingly objectionable, as they interfere with speech communication. Sounds lower than 0 dBA occur but cannot be heard. Brief exposures to sound levels higher than 140 dBA may be painful, and long-term exposures to sound levels higher than 90 dBA may result in hearing loss.

12.1.2 Sound Measurement

Instruments used to measure sound levels in decibels are described as sound pressure meters (Figure 12.3). Such meters consist of a microphone, attenuator, amplifiers, and a digital or mechanical reading. Sound generates a voltage proportional to the acoustical pressure acting on the microphone. This voltage produces a deflection of the meter needle or is converted to a digitally displayed value. An integral part of sound-measuring instruments

TABLE 12.1
Sound Pressure Levels, Sources, and Human Responses

Sources	dB	Human Responses
	150	
	140	
Jet aircraft on ground, 20 ft	130	Painful
Rock and roll band	120	
Loud motorcycle, 20 ft	115	
	110	
	100	
Heavy truck, 50 ft	90	Permanent hearing loss
Pneumatic drill, 50 ft	85	
	80	
	75	Annoying
Motor vehicle, 50 ft	70	Interfere with speech communication
	60	
Living room	55	
	50	Quiet
Bedroom	45	
Library	40	
Soft whisper	30	
	20	Very quiet
	10	Just audible
	0	Threshold of hearing

is weighting scales or networks that emphasize or deemphasize sound in selected frequency ranges.

12.1.2.1 *Weighting Scales*

Sound level meters are generally equipped with three circuits that correspond to weighting scales. Three weighting scales have been established by the American National Standards Institute (ANSI) for general-purpose use. These are the A, B, and C scales. Other scales (D and E) have been developed and used for special applications. Each scale differs in its ability to measure sound levels across a frequency range (Figure 12.4). The A scale is designed to discriminate against (i.e., not measure) frequencies lower than 600 Hz. Low-frequency discrimination is more moderate on the B scale. The C scale corresponds more closely to the actual sound energy produced; its response is relatively uniform across the frequency spectrum. Readings on all three scales provide an indication of the frequency distribution of a recorded sound. If the level is greater on the C scale, some of the sound is in frequencies lower than 600 or higher than 10,000 Hz. If no differences are observed among the three scales, most of the measured sound is in the middle frequencies.

hearing impairments are limited to the frequency range of 500 to 2,000 Hz. The A scale approximates noise exposure risk to human hearing relatively well. Because humans show limited response to frequencies lower than 500 Hz, there is little value in measuring such frequencies when controlling worker noise exposure. Similarly, it is the audible middle frequencies that are important in community noise exposures.

Despite its almost universal use for measurements of Spl in industrial and community environments, the A scale has some important limitations. It apparently does not simulate the spectral selectivity of human hearing or its nonlinear relationship to sound energy. If different spectral characteristics are compared, dBA values may not be an accurate indicator of human subjective response. Laboratory and field studies have shown that the A scale predicts perceptions of loudness and annoyance relatively poorly. It is strongly dependent on the exposure pattern with time and underestimates the effect of low-frequency components of sound. It is also unrepresentative of loud sounds that contain a mixture of distinctive tonal components. As such, it is unsuitable for predicting loudness or annoyance.

12.1.2.2 Meter Response and Instrument Accuracy

In addition to weighting scales, most sound pressure level instruments have slow and fast measuring options. The averaging time for the fast response is 0.125 s; whereas for the slow response, it is 1 s. Although the fast response corresponds to the response time of the human auditory system, the slow response averaging time is usually used because it is easier to read.

Commonly available sound pressure level meters are rated as Type I, II, or III. These designations reflect their accuracy. Type I meters have an accuracy of ±1 dB; they are primarily research instruments. Type II meters have an accuracy of ±2 dB; they are general-purpose instruments recommended for use in measuring both industrial and community sound levels. Type III meters have an accuracy of ±3 dB and are not recommended for regulatory or technical use.

12.1.2.3 Measurement of Impulse Sound

General-purpose sound pressure level meters cannot be used to measure impulse noises, that is, those produced by a variety of industrial processes such as punch presses and pile drivers. These sounds consist of rapidly rising and falling sound pressures. Instruments must be specially designed to measure both peak and average impulse sound levels. An averaging time of 0.035 s is typically used.

12.1.2.4 Spectrum Analysis

The audible frequency range is covered by 10 octave bands. An octave is a range of frequencies in which the upper frequency is, in most cases, twice

TABLE 12.2
Octave Band Frequency Ranges and Center Frequencies

Frequency Range (Hz)	Center Frequency (Hz)
18–45	31.5
45–90	63
90–180	125
180–355	250
355–710	500
710–1,400	1,000
1,400–2,800	2,000
2,800–5,600	4,000
5,600–11,200	8,000

the lower frequency (Table 12.2). Each octave band is characterized by a center frequency that is the geometric mean of the upper and lower limits. The octave band sound level is the amount of acoustical energy (measured as sound pressure) associated with sound frequencies in the octave band. Noise assessments are often conducted using one-third octave band filters. Each octave is subdivided into thirds to better characterize sound spectra.

12.2 Sound Exposure Descriptors

The U.S. Environmental Protection Agency (U.S. EPA) uses a system of four sound descriptors to summarize how people hear sound and how environmental noise affects public health and welfare. These are the A-weighted sound level, A-weighted sound exposure level, equivalent sound level, and day–night sound level.

12.2.1 A-Weighted Sound Level

A weighting is recommended because of its convenience of use, relative accuracy for most purposes, and widespread acceptance around the world. It is used in the other descriptors described below. When used by itself, an A-weighted decibel value describes a sound value at a given instant, a maximum value, or a steady-state value.

12.2.2 A-Weighted Sound Exposure Level

Sound exposure levels take into account the moment-to-moment variations that occur when measuring environmental noise. The changing magnitude of sound levels can be observed on a strip-chart recording. Changes in sound

TABLE 12.4

Outdoor L_{dn} Levels by Location

Location	L_{dn} (dBA)
Airport, near freeway	88
0.75 mi. from commercial aircraft landing	86
Urban center construction activity	78.5
Urban high-density apartment	78
Urban row housing—major street	68
Old urban residential area	59
Residential—wooded neighborhood	51
Residential—rural	39
Wilderness	35

Source: U.S. EPA, *Protective Noise Levels*, http://www.nonoise.org/library/levels/levels/htm, 2000 (condensed version of EPA Levels document).

daytime L_{eq} were 60 dBA and the nighttime L_{eq} were 50 dBA, the weighted nighttime sound level would be 50 + 10 dBA = 60 dBA. The day–night sound level (L_{dn}) would therefore be 60 dBA. Table 12.4 indicates L_{dn} values for different outdoor locations. The L_{dn} is commonly used in noise ordinances.

12.3 Loudness

The physical magnitude of sound energy can be described as sound intensity or pressure. The subjective, or perceived, magnitude is called loudness. Perception of loudness depends on sound energy, frequency, and duration. Humans do not hear sound frequencies equally well across the frequency spectrum (Figure 12.2). Additionally, physiological processes in the human auditory system protect it from excessive sound stimulation. Consequently, the relationship between sound energy received and our perception of loudness is not linear; it is closer to being logarithmic.

The sone is the basic unit of loudness. It is defined as the loudness of a pure 1,000-Hz tone at a sound pressure of 40 dBA under specified listening conditions. A 2-sone sound would be perceived as twice as loud as a 1-sone sound.

Loudness is proportional to sound energy over a significant fraction of audible frequencies. This psychophysical "power law" is referred to as Steven's law. In the middle frequencies, the exponent of the power function is such that a twofold increase in perceived loudness corresponds to a 10-dB increase in sound pressure.

The relationship between loudness and sound pressure can be seen from the suite of equal loudness contours in Figure 12.6. Loudness levels are