# Chapter 80
# Air Pollution

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Air pollution is a heterogeneous mix of suspended solids, liquids, and gases. Even in the absence of human activity, aerosols are emitted by volcanic activity, windstorms, wildfires, and ocean waves. Traditionally, however, the term *air pollution* refers to the human impact of biomass and fossil fuel combustion, primarily from industry and vehicular traffic. The focus of this chapter is on the air pollutants with the most evidence of ongoing and widespread threat to public health, including both outdoor and indoor pollutants. Specific topics related to air pollution, including environmental tobacco smoke, plant antigens, and windblown agricultural dusts, are primarily addressed in other chapters.

## HISTORY

Air pollution has posed a threat to human health since the advent of fire, 500,000 years ago. Evidence of soot in prehistoric caves indicates that early humans were exposed to high levels of indoor air pollution. Outdoor pollution began to rise as populations grew and clustered in cities. During the Industrial Revolution, there was a dramatic increase in coal use by factories and households. The resulting smog caused significant morbidity and mortality, particularly when combined with stagnant atmospheric conditions. During the Great London Smog of 1952, heavy pollution for 5 days caused at least 4000 excess deaths (**Figure 80-1**). This episode highlighted the relationship between air pollution and human health and, together with other episodes, led to clean-air policies in Europe and in the United States. Over the next several decades, with improvements in the regulatory framework, air quality in the developed world steadily improved. Even as energy consumption and greenhouse gas emissions have increased, pollutants that are toxic to humans have been better controlled. Nevertheless, 186 million Americans, or 60% of the population, still live in counties with levels of air pollution higher than U.S. Environmental Protection Agency (EPA) standards (**Figure 80-2**). Air pollution also continues to be a growing problem in cities and households of the developing world.

## COMPOSITION OF AIR POLLUTION

Many different substances are considered "air pollutants," which typically exist in combination in the environment; these include *particulate matter* (PM), carbon monoxide (CO), ozone ($O_3$), nitrogen oxides (NOx), sulfur oxides (SOx), and *volatile organic compounds* (VOCs). The composition varies by season and regions, and the chemistry, state, and size of a pollutant influence the resulting health effects (**Figure 80-3**). Although CO has specific toxicity affecting oxygen transport, many pollutants appear to cause disease by triggering injury through inflammation and oxidative stress. Physiologic effects can be confirmed by experimental studies, but health impacts of air pollution on populations can be difficult to attribute to a single pollutant because they often share sources and vary together. A multipollutant approach to understanding health effects is becoming more common. Nevertheless, this section discusses some of the physiologic effects of individual pollutants before a description of the health outcomes seen with exposure to air pollution.

### Particulate Matter

Among air pollutants, PM is most consistently associated with increased mortality effects at ambient exposure concentrations regularly encountered today. Although PM can be formed by a wide variety of natural and man-made processes, one of the largest sources of PM pollution is through combustion of biomass and fossil fuels. PM is composed of organic and inorganic compounds that vary greatly in size (**Figure 80-4**). *Fine* PM (<2.5 μm) has the most well-recognized health effects, and the *ultrafine* PM fraction (<0.1 μm) may be especially important. Larger PM is filtered and trapped in the proximal respiratory tract, but fine particles can penetrate deep into the lung, where they are efficiently deposited, cause local effects, and may be systemically absorbed.

The composition of PM also influences its health effects. The mechanism of PM toxicity in the lung is still being elucidated but is likely multifactorial and can include impaired ciliary function, damage to epithelial cells, inflammation, and oxidative stress. Neutrophils and macrophages phagocytize the poorly soluble particles and initiate inflammatory cascades. PM also causes oxidative stress by generating reactive oxygen species (ROS) on the surface of particles that may then cause cellular effects. Inflammation and oxidative stress apparently provoke cardiovascular disease through atherosclerosis, thrombogenesis, vasoconstriction, and reduced heart rate variability (**Figure 80-5**).

Epidemiologic studies have associated changes in PM with respiratory symptoms, respiratory tract infections, impaired pulmonary function, asthma exacerbations, chronic obstructive pulmonary disease (COPD), cardiovascular events, cerebrovascular accident (CVA, stroke), lung cancer, and all-cause mortality.

### Ozone

The stratospheric ozone layer is a normal part in the Earth's atmosphere that absorbs high-frequency ultraviolet (UV) rays and protects us from their damaging effects. In contrast, ground-level ozone is an air pollutant, which has harmful respiratory and cardiovascular effects. Ground-level $O_3$ is largely the result

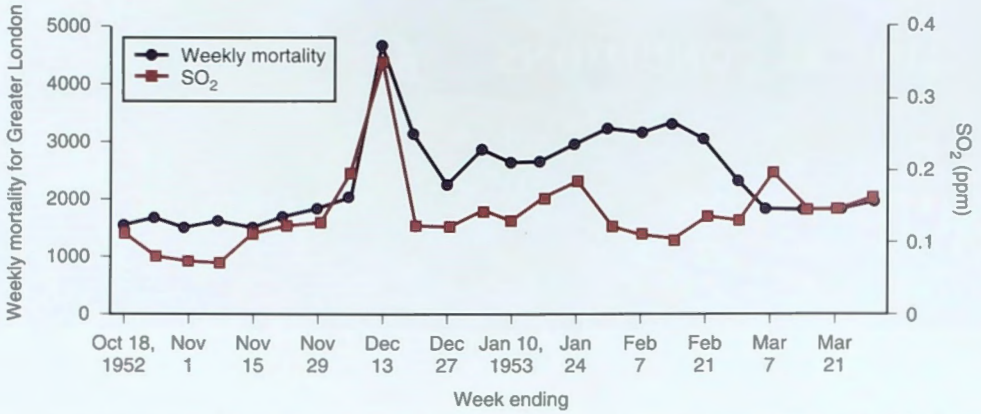
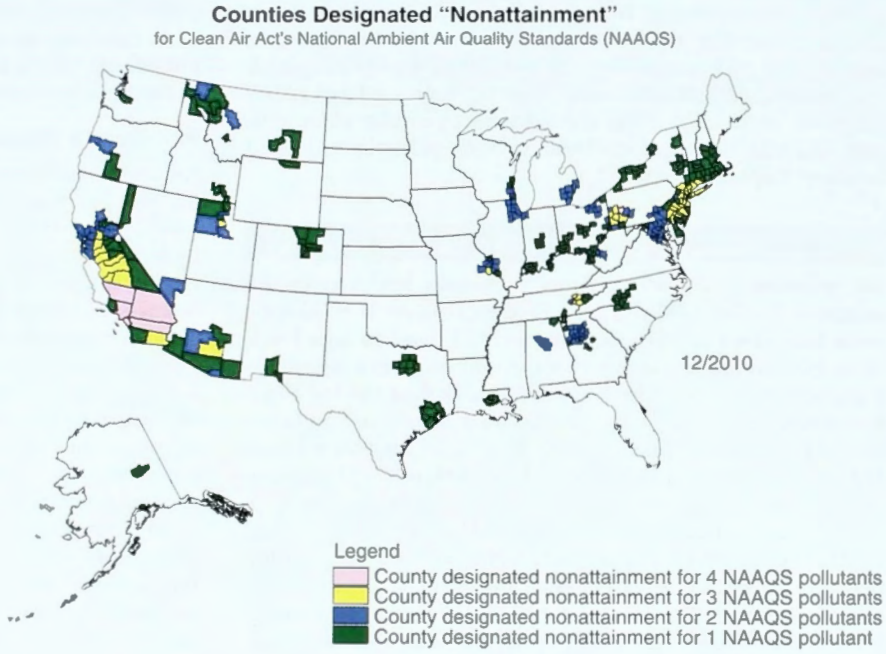



**Figure 80-1** Weekly mortality in greater London, October 1952 to March 1953; $So_2$, oxygen saturation. *(Modified from Bell ML, Davis DL: Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution,* Environ Health Perspect *109(suppl 3):389-394, 2001.)*

**Figure 80-2** Counties designated as "nonattainment areas" for EPA National Ambient Air Quality Standards (NAAQS) of various pollutants, including particulate matter, ozone, sulfur dioxide, carbon monoxide, and lead. *(From US Environmental Protection Agency Green Book, 2011. www.epa.gov/oaqps001/greenbk/mapnpoll.html.)*




of UV photolysis of NOx and VOCs (products of vehicular and industrial combustion). Inhaled $O_3$ reacts with biomolecules to form free radicals, which trigger proinflammatory and prooxidative mediators. Increased neutrophils, eosinophils, and inflammatory cytokines have all been noted in bronchoalveolar lavage (BAL) fluid in response to $O_3$ exposure. Exposure to even very low levels of $O_3$ leads to airway inflammation and bronchoconstriction. Clinically, exposure to $O_3$ causes exacerbation of asthma and COPD, impaired pulmonary function, and increased respiratory symptoms, and it has been associated with increased mortality. The effects of $O_3$ are further potentiated by exposure to PM.

## Sulfur Dioxide

Combustion of sulfur-containing fuels, including coal and petroleum, produces sulfur dioxide ($SO_2$), a highly water-soluble gas that is readily absorbed in the mucosa of the eyes and upper respiratory tract. $SO_2$ is a strong irritant that leads to local inflammation. $SO_2$ can also bind to organic PM and affect the distal airways. $SO_2$ increases vascular distention, mucosal edema, smooth muscle contraction, and intraairway secretions. Lung function in healthy individuals is relatively resistant to the effects of considerable doses of $SO_2$ (5 ppm), but even lower doses (e.g., <1 ppm) elicit acute and substantial bronchoconstriction in some asthmatic patients. Although $SO_2$ causes multiple respiratory symptoms, it does not seem to be independently responsible for air pollution–related mortality.

## Nitrogen Dioxide

Nitrogen dioxide ($NO_2$) is formed during the combustion of fossil fuels, predominantly at power plants and in vehicles. Being relatively insoluble, $NO_2$ is not significantly absorbed by proximal mucosa. Instead, $NO_2$ travels to the more distal airways, where it reacts with water in the respiratory tree to create nitric acid. $HNO_3$ causes both local and systemic inflammation. Acutely, this can lead to bronchospasm. $NO_2$ is

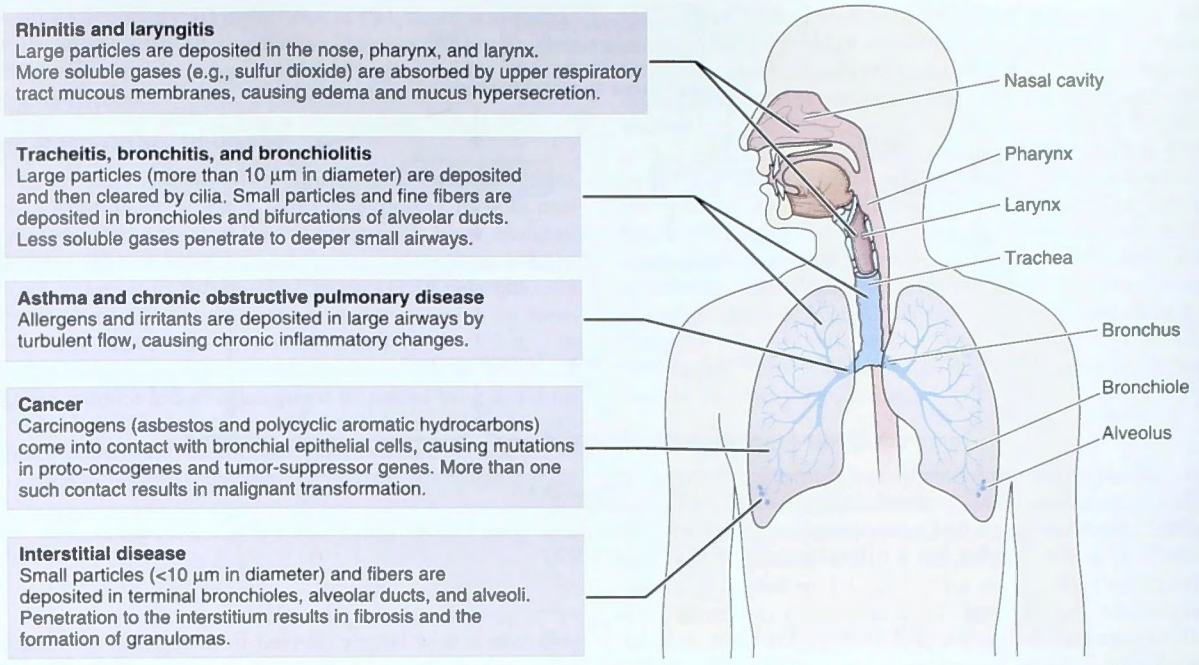



**Figure 80-3** Physiologic effects of air pollutants on the respiratory tract. *(Modified from Beckett WS: Occupational respiratory diseases,* N Engl J Med *342:406-413, 2000.)*

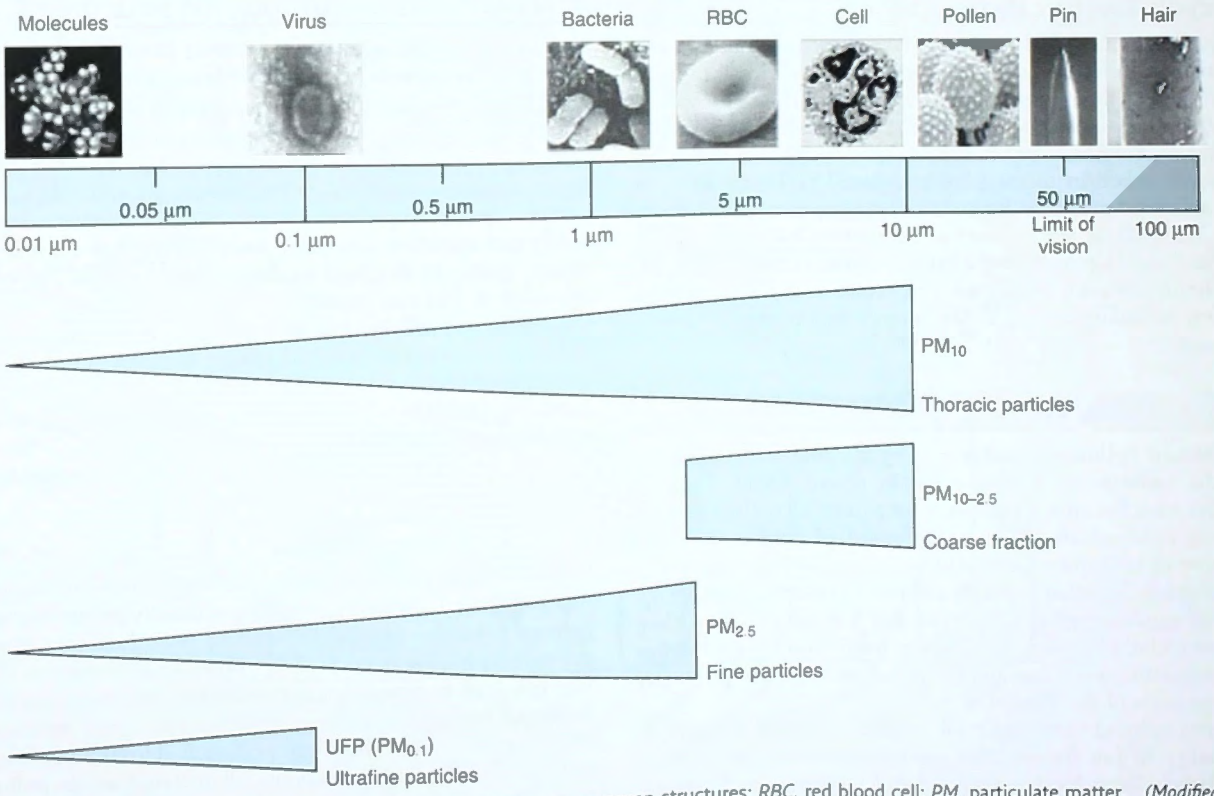



**Figure 80-4** Size classification of particulate matter with reference to common structures; *RBC*, red blood cell; *PM*, particulate matter. *(Modified from Brook RD, Franklin B, Cascio W, et al: Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association,* Circulation *109:2655-2671, 2004.)*

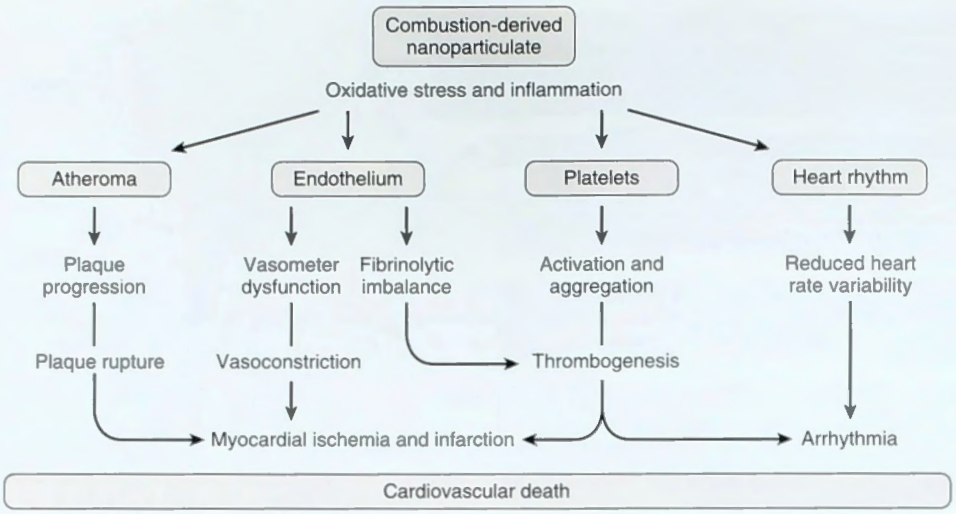



**Figure 80-5** Mechanism of cardiovascular disease from exposure to air pollution. *(From Mills NL, Donaldson K, PW Hadoke, et al: Adverse cardiovascular effects of air pollution,* Nat Clin Pract Cardiovasc Med *6:36-44, 2009.)*

generally less potent than other pollutants and is likely to have its most harmful effects by acting in synergy with these other compounds. Although high levels of indoor $NO_2$ can increase susceptibility to viral infection, this has not been observed with outdoor levels of $NO_2$ pollution. In epidemiologic research, $NO_2$ concentrations are often considered a surrogate for exposure to traffic-related air pollution; thus the relationship of $NO_2$ with disease in these studies is probably caused by other pollutants that share exposure profiles.

### Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) include a number of organic molecules that are found in biomass and fossil fuels or are formed in their combustion. Use of these fuels aerosolizes PAHs along with other VOCs. Although there are many different types of PAH, small and lipophilic PAHs are the most readily absorbed in the respiratory tract. The effects of each PAH species depends on its underlying composition and structure. Many PAHs are irritants to respiratory mucosa. Others, such as benzo[a]pyrene, one of the carcinogens first described, and the dioxins, are mutagenic and carcinogenic, with special concern regarding cancer of the mouth, nasopharynx, larynx, and lung.

## OUTDOOR AIR POLLUTION

Outdoor air pollution is influenced by the source of emissions and the surrounding environment. In recent decades, motor vehicles have become a predominant source of pollution, particularly in urban areas. Coal and fossil fuel combustion also continue to be a major source of emissions from industry and power plants. Together, vehicles and power generation make up 90% of outdoor air pollution in the United States. Other sources include biomass combustion from wood fires. During winter months, wood burning is a principal source of pollution in some parts of the United States.

Topography, climate, and local legislation further impact the air quality. In Los Angeles the nearby mountains trap pollutants, largely from local mobile vehicular sources, and intense UV rays act to create high $O_3$ levels. In Pittsburgh, a major city with some of the highest PM levels in the United States, most pollution is now largely derived from regional sources, carried by southeasterly winds bringing emissions from more distant power plants and factories. Outside the developed world, there are fewer environmental controls, resulting in higher levels of pollution, especially in areas of rapid industrialization and urbanization. China has many of the most polluted cities in the world, a factor of its heavy coal use and limited environmental protection.

### *HEALTH EFFECTS OF OUTDOOR AIR POLLUTION*

Outdoor air pollution has many diverse health effects, ranging from mild respiratory irritation to hospitalization and death. The World Health Organization estimates that an excess 800,000 deaths occur annually because of exposure to outdoor air pollution. Daily fluctuation in levels of air pollution causes an acute increase in mortality, mostly from cardiovascular causes. Chronic air pollution exposure also contributes to morbidity and mortality through a variety of cardiopulmonary illnesses. Some of the first evidence for mortality of chronic exposure to PM was demonstrated by the Six Cities Study, in which citizens of six U.S. cities (representing a range of air pollution severity) were studied as a prospective cohort. Cities with higher levels of fine PM had an increased risk of all-cause mortality (risk ratio, 1.26 for most polluted vs. least polluted city; **Figure 80-6**). Many other cohort studies confirm this relationship, with improved determination of exposures and health outcomes.

### Acute Respiratory Illnesses

Exposure to air pollution produces acute changes in lung function in healthy adults. These changes usually go unnoticed by patients who are not predisposed to bronchospasm. Among patients with asthma and COPD, however, exposure to pollution can lead to serious effects. Asthmatic patients experience increased cough, wheezing, breathlessness, and medication needs on days with high air pollution. Hospitalizations for asthma, COPD, and pneumonia all increase as air pollution rises. There are also more respiratory symptoms and upper respiratory infections in the general population. Even small changes in PM concentration can lead to these effects

(Figure 80-7). For every 10 μg/m³ rise in PM, there is approximately 1% increase in all respiratory diseases. Although this effect size seems small, it has a notable impact when applied to large populations.

### Chronic Respiratory Illnesses

Just as acute exposure to air pollution can cause transient changes in pulmonary function, chronic exposure to pollution is associated with structural and functional lung changes. Studies show that children living in more polluted areas have lower forced expiratory volume in 1 second ($FEV_1$). In patients with cystic fibrosis, exacerbations are more common in communities with higher $PM_{2.5}$ concentrations (PM <2.5 μm in diameter). One study observed that exposure to air pollution in pregnant women led to subsequent impaired lung development in children. There is also evidence that exposure to pollution may contribute to the pathogenesis and incidence of asthma among children. Children living close to motor vehicle traffic have been found to have higher prevalence of asthma and smaller $FEV_1$ after controlling for potential confounders. European and U.S. cohorts also show that children exposed to higher amounts of air pollution have a higher incidence of asthma.

The association between outdoor air pollution and the development of COPD is less clear, with studies showing inconsistent and inconclusive results. The greatest risk factor for COPD remains tobacco smoke. However, air pollution causes recurrent respiratory symptoms and infections, both of which may contribute to COPD. Similarly, data are limited on the association of outdoor air pollution and lung cancer. Lung cancer is a rare disease with a long latency period, although a few studies found a modest increase in lung cancer with exposure to outdoor air pollution.

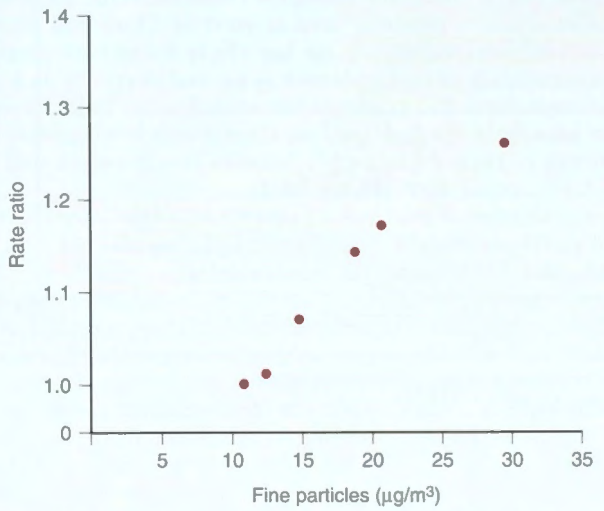



**Figure 80-6** Association of fine particulate concentration and mortality from Harvard Six Cities Study. *(Modified from Dockery DW et al:* N Engl J Med *329:1753-1759, 1993.)*

### Nonrespiratory Illnesses

Some of the most important and well-studied diseases associated with air pollution are cardiovascular illnesses, including ischemic heart disease, arrhythmias, congestive heart failure, and sudden cardiac death. A metaanalysis on air pollution and hospital admissions found that for every 10 μg/m³ increase in PM, there was 0.8% increase in heart failure admissions and 0.7% increase in ischemic heart disease admissions. Driving or otherwise being in traffic has been associated with an acute increase in risk of myocardial infarction (odds ratio, 2.92), although pollution is only one potential culprit. Air pollution has further been associated with ventricular arrhythmias, implantable defibrillator shocks, and atrial fibrillation. The majority of excess deaths associated with air pollution are from cardiovascular causes. There is less conclusive data on the association of air pollution with other nonrespiratory diseases, including stroke, peripheral vascular disease, deep venous thrombosis, and adverse perinatal outcomes.

## GENETICS AND AIR POLLUTION

There has been substantial investigation of respiratory gene-environment interactions with special attention to the ambient air pollution. This research may ultimately inform identification of not only especially susceptible populations but also

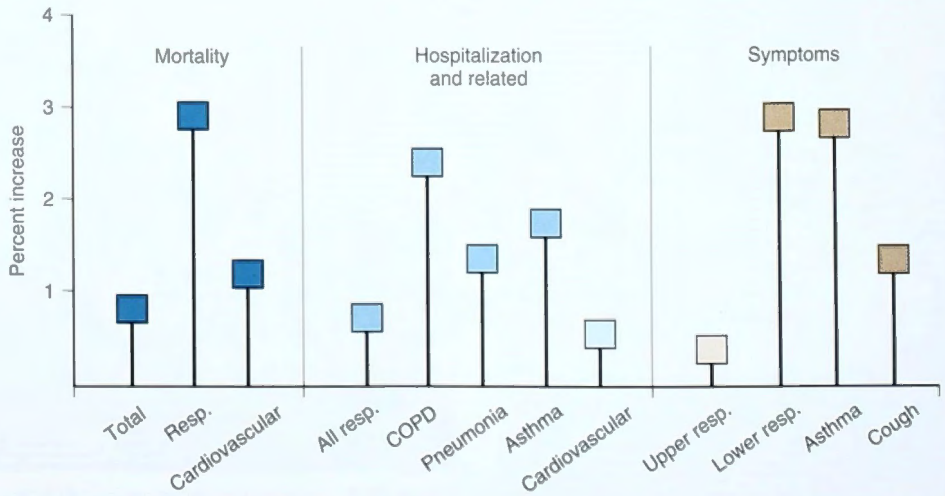



**Figure 80-7** Meta-analysis results demonstrating the increase in mortality, morbidity, and symptoms associated with acute, 10 μg/m³ increase in $PM_{10}$. *COPD*, chronic obstructive pulmonary disease; *resp.*, respiratory. *(Modified from Donaldson K, Mills N, MacNee W, et al: Role of inflammation in cardiopulmonary health effects of PM,* Toxicol Appl Pharmacol *207(2 suppl):483-488, 2005.)*

molecular mechanisms involved in the environmental effect. Genes that regulate inflammation and oxidative stress play a significant role in modulating these individual differences. Several studies have identified polymorphisms in oxidative stress genes (*NQO1*, *GSTM1*, and *GSTP1*) and inflammatory genes (TNF) that are associated with respiratory symptoms, decreased pulmonary function, and risk of asthma. For example, *NQO1* (NADPH:quinone oxidoreductase 1) is an ROS scavenger. Mutations in this gene have been associated with decreased $FEV_1$ in healthy individuals exposed to ozone. Polymorphisms of *NQO1* are also correlated with higher rates of asthma among children in Mexico City. Identifying genetic susceptibilities to air pollution not only helps characterize individuals who are at the greatest risk, but also helps target interventions. A clinical trial of children with a mutation in the antioxidant gene *GSTM1* found a protective effect of antioxidants, including vitamins C and E, on pulmonary function. Further studies are needed to characterize these genetic susceptibilities further and focus interventions.

### *AIR QUALITY INDEX*

Governmental agencies worldwide measure and report on the air quality in major cities and suburban areas. The U.S. EPA produces an Air Quality Index (AQI) for more than 100 U.S. cities and regions. The AQI is reported on a scale from 0 to 500, with higher values being more hazardous (**Table 80-1**). An AQI score more than 100 indicates unhealthy air quality, especially when compounded by high-risk weather conditions, such as hot and sunny days. Many newspapers, weather reporters, and radio stations report this score. On days with high AQI scores, it is recommended that people stay indoors as much as possible. Susceptible individuals should also limit physical activity, which increases ventilation and ultimately leads to five times the amount of PM deposition in lungs.

**Table 80-1 Definitions of Air Quality Index (AQI) Levels by U.S. Environmental Protection Agency**

| AQI Levels of Health Concern | Numeric Value | Description |
|---|---|---|
| Good | 0-50 | Air quality is considered satisfactory, and air pollution poses little or no risk. |
| Moderate | 51-100 | Air quality is acceptable; for some pollutants, however, there may be a moderate health concern for a small number of people unusually sensitive to air pollution. |
| Unhealthy for sensitive groups | 101-150 | Members of sensitive groups may experience health effects; the general public is not likely to be affected. |
| Unhealthy | 151-200 | Everyone may begin to experience health effects; members of sensitive groups may experience more serious health effects. |
| Very unhealthy | 201-300 | Health alert; everyone may experience more serious health effects. |
| Hazardous | 301-500 | Health warnings of emergency conditions. The entire population is more likely to be affected. |

Data from www.airnow.gov. Accessed March 2011.

## INDOOR AIR POLLUTION

The vast majority of adverse health effects related to air pollution likely occur in developing countries, where there are higher levels of both outdoor and indoor air pollution. More than half of the world's population depends on solid fuel as the primary source of domestic energy for cooking and heat. Most of this is in rural regions of the developing world where cooking is done over open fires in poorly ventilated homes (**Figure 80-8**). This creates intense exposure to *indoor air pollution* (IAP) with devastating health consequences.

Among households that rely on solid fuels for energy, approximately 95% use *biomass* fuels (wood, charcoal, crop residues, dung) in open indoor fires. Household coal use is less common and is primarily used in parts of China and South Africa. Overall, solid fuels are less efficient and have greater emissions than costlier fuels such as gas and electricity. In fact, although developed countries use several times more energy per household, the fuels used are cleaner with less exposure to pollutants. These differences in domestic energy are referred to as the "energy ladder" (**Figure 80-9**).

Combustion of solid fuels can emit many of the same chemical pollutants as outdoor pollution, including PM, CO, $NO_2$, $SO_2$, and PAHs, although the concentrations of these substances tend to be much higher. For example, concentrations of PM in households that rely on solid fuels often reach levels more than 100 times greater than outdoor concentrations. Women and children are disproportionately exposed to this pollution. On average, women in the developing world spend 3 to 7 hours per day near the fire, preparing food. They frequently do so with large children by their side and small children on their backs. As the population with the greatest exposure to IAP, women and children also suffer the most damaging health effects.

**Figure 80-8** Ecuadorian child sitting next to indoor cooking fire. *(From Rinne ST, Rodas EJ, Rinne ML, et al: Use of biomass fuel is associated with infant mortality and child health in trend analysis,* Am J Trop Med Hyg *76:585-591, 2007.)*

## HEALTH EFFECTS OF INDOOR AIR POLLUTION

The World Health Organization estimates that exposure to solid fuel smoke causes 1.6 million deaths annually, primarily in the developing world (**Figure 80-10**). This includes deaths caused by pneumonia, COPD, and lung cancer (**Table 80-2**). Increasing evidence also is revealing the role of IAP in other infectious and noninfectious diseases. Most diseases associated with outdoor pollution are also linked to IAP exposure. Overall, IAP is responsible for 38.5 million disability-adjusted life years (DALYs), making it the 8th leading cause of DALYs and 11th cause of death worldwide.

### Acute Respiratory Illnesses

Worldwide, acute lower respiratory tract infections are the most important cause of mortality in children younger than 5 years. Exposure to IAP more than doubles the risk of developing acute lower respiratory infections. Annually, this leads to 910,000 of the 2 million deaths caused by pneumonia. Several studies also show an association between indoor air pollution and upper respiratory infections, including otitis media and mastoiditis. In addition to infectious diseases, exposure to indoor smoke leads to increased airway reactivity, which can exacerbate asthma and COPD.

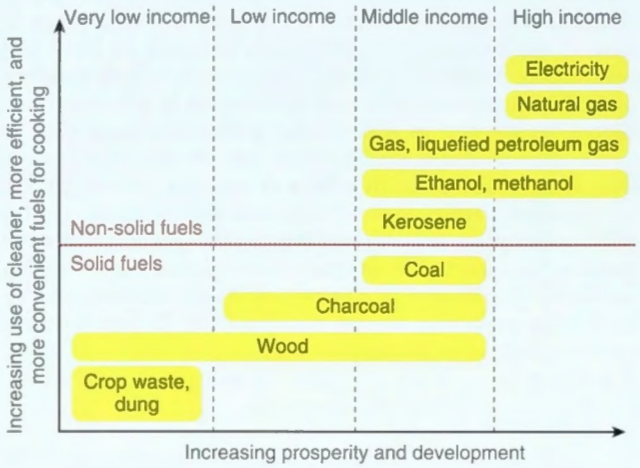



**Figure 80-9** "Energy ladder" emphasizing the association of development with household energy. *(Modified from World Health Organization:* Fuel for life: household energy and health, *Geneva, 2006, WHO.)*

**Table 80-2 Results from Metaanalyses on Association of Indoor Air Pollution with Various Health Outcomes**

| Health Outcome | Population | Relative Risk | 95% CI | Evidence |
|---|---|---|---|---|
| Pneumonia | Children 0–4 yr | 2.3 | 1.9–2.7 | Sufficient |
| COPD | Women ≥30 yr | 3.2 | 2.3–4.8 | Sufficient |
| | Men ≥30 yr | 1.8 | 1.0–3.2 | Sufficient |
| Lung cancer (coal) | Women ≥30 yr | 1.9 | 1.1–3.5 | Sufficient |
| | Men ≥30 yr | 1.5 | 1.0–2.5 | Sufficient |
| Lung cancer (biomass) | Women ≥30 yr | 1.5 | 1.0–2.1 | Insufficient |
| Asthma | Children 5–14 yr | 1.6 | 1.0–2.5 | Insufficient |
| | Adults ≥15 yr | 1.2 | 1.0–1.5 | Insufficient |
| Cataracts | Adults ≥15 yr | 1.3 | 1.0–1.7 | Insufficient |

Modified from World Health Organization: *Fuel for life: household energy and health*, Geneva, 2006, WHO.
*CI*, confidence interval; *COPD*, chronic obstructive pulmonary disease.

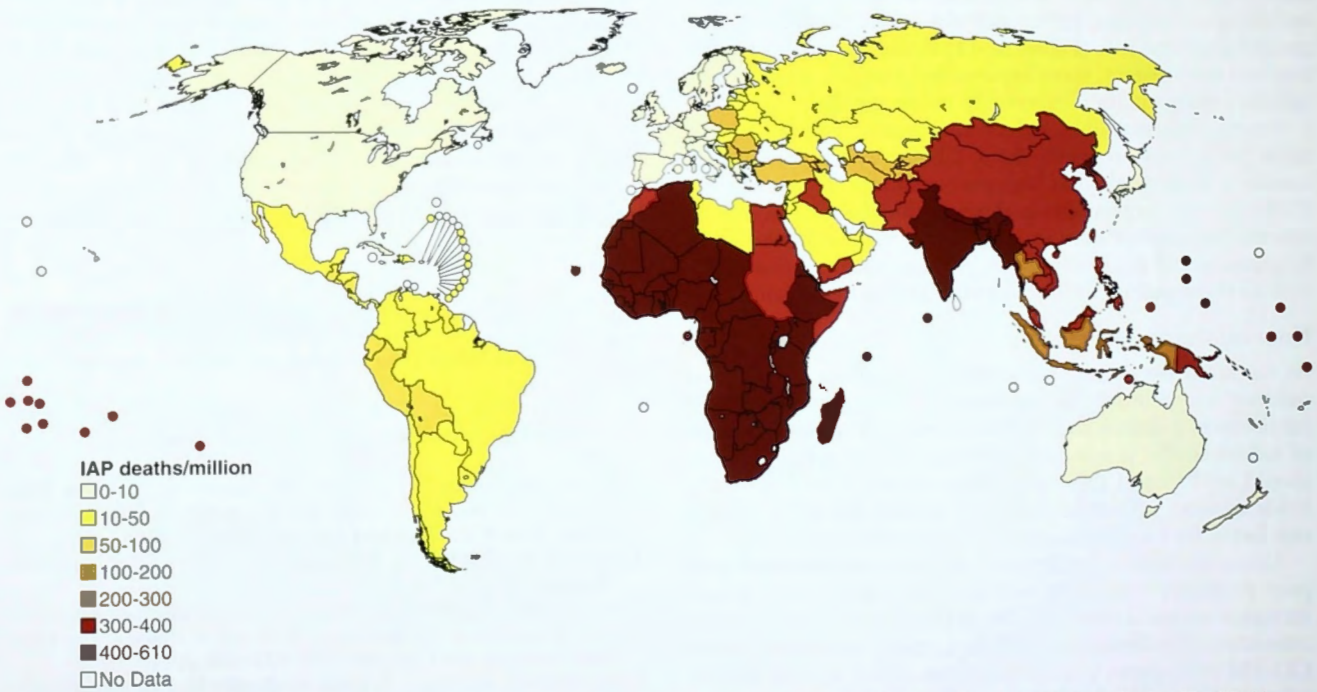



**Figure 80-10** Distribution of mortality from indoor air pollution *(IAP)* throughout the world. *(Modified from World Health Organization:* The World Health Report 2002: reducing risks, promoting healthy life, *Geneva, 2002, WHO.)*

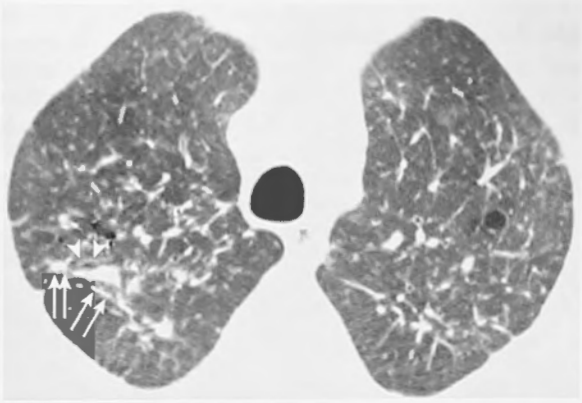


**Figure 80-11** Axial high-resolution CT scan of chest demonstrating characteristic findings in domestic acquired particulate lung disease, including nodular interlobular septal thickening *(arrowheads)* and bronchovascular thickening with nodular appearance *(double arrows)*. *(Modified from Diaz JV, Koff J, Gotway MB, et al: Case report: a case of wood-smoke-related pulmonary disease,* Environ Health Perspect *114:759–762, 2006.)*

### Chronic Respiratory Illnesses

Chronic exposure to indoor smoke causes a broad range of chronic disorders referred to as *domestic acquired particulate lung disease* (DAPLD) or "hut lung." DAPLD includes noninfectious, nonmalignant diseases such as COPD and pulmonary fibrosis. Women who cook with solid fuels are three times more likely to develop COPD than women who cook with cleaner fuels. Globally, IAP causes 22% of COPD cases. Although most people exposed to chronic indoor smoke develop obstructive pulmonary function, some also have *restrictive* patterns. Bronchoscopy typically reveals anthracotic plaques. Computed tomography (CT) may demonstrate a variety of patterns, including reticulation, peribronchial thickening, nodules, and ground-glass opacities (**Figure 80-11**). Although studies are still few and inconsistent, some reports find a higher incidence of asthma among children exposed to indoor smoke.

Strong evidence links indoor coal smoke to lung cancer, again with most research from China, where coal use is common. Coal smoke has high concentrations of carcinogenic PAHs. Chronic exposure to coal smoke doubles the risk of lung cancer. Few studies have examined the relationship between lung cancer and other solid fuels; but the concern is that exposure to these pollutants also increases risk of malignancy.

### Nonrespiratory Illnesses

As discussed previously, exposure to particulate matter from outdoor air pollution has consistently been associated with cardiovascular disease and mortality. Research on similar effects of indoor smoke is minimal, although the same relationships should hold true. If these risks were consistent among households cooking with solid fuels, IAP would represent a major risk factor for morbidity and mortality worldwide.

Unvented cooking with solid fuels has been associated with *poor pregnancy outcomes*, including low birth weight and increased perinatal mortality. This is likely caused by a complex interaction of pollutants in solid-fuel smoke, including PM and CO. PM predisposes to maternal illness affecting fetal growth, whereas CO avidly binds hemoglobin and decreases oxygen delivery to the placenta. On average, babies born to households that cook with biomass fuels weigh 63 g less than those born in households that use cleaner fuels.

*Cataracts* are common in the developing world and frequently lead to blindness. Large epidemiologic studies in India have shown a 30% increase in cataracts among people living in households dependent on solid fuels after adjusting for confounding factors. The mechanism for this effect remains a matter of speculation.

## CONTROVERSIES AND PITFALLS

Environmental influences on respiratory health cannot be interpreted in isolation. Not only are these environmental factors inherently interacting with the genetic predisposition of the individual, but various environmental processes are closely linked. Changes in air pollution are associated with changes in climate, and both are currently driven primarily by fossil fuel combustion. As global warming progresses, it is likely to affect air pollution patterns, although the impact is still uncertain. Global climate change is driven by greenhouse gases, such as carbon dioxide and methane, which are not generally considered to have direct health effects at ambient concentrations. Nevertheless, warmer days are associated with worse air quality, and most scientists believe that climate change will significantly increase the number of summer days that exceed air quality standards. This change in climate will likely increase ground-level ozone as warmer temperatures promote $O_3$ formation. In contrast, particulate matter levels could conceivably decrease, if global warming leads to increased precipitation or a concerted effort at reducing fossil fuel use.

## CONCLUSION

Additional research is needed to define the effects of outdoor air pollution in a multipollutant context. Most published research examines the health effects of individual pollutants, while the interaction between multiple pollutants and the role of particular sources of pollution is likely just as important. Ongoing research should also focus on identifying safe levels of air pollution and effective ways of decreasing exposure to pollution. In the developed world, research into clean energy may provide valuable answers to improving human health as well as the environment. In the developing world, solutions relating to improving indoor air quality and decreasing solid-fuel dependence would also have a widespread and dramatic impact.

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# CLINICAL RESPIRATORY MEDICINE

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