The Potential Impacts of Climate Variability and Change on Air Pollution-Related Health Effects in the United States

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Climate change may affect exposures to air pollutants by affecting weather, anthropogenic emissions, and biogenic emissions and by changing the distribution and types of airborne allergens. Local temperature, precipitation, clouds, atmospheric water vapor, wind speed, and wind direction influence atmospheric chemical processes, and interactions occur between local and global-scale environments. If the climate becomes warmer and more variable, air quality is likely to be affected. However, the specific types of change (i.e., local, regional, or global), the direction of change in a particular location (i.e., positive or negative), and the magnitude of change in air quality that may be attributable to climate change are a matter of speculation, based on extrapolating present understanding to future scenarios. There is already extensive evidence on the health effects of air pollution. Ground-level ozone can exacerbate chronic respiratory diseases and cause short-term reductions in lung function. Exposure to particulate matter can aggravate chronic respiratory and cardiovascular diseases, alter host defenses, damage lung tissue, lead to premature death, and possibly contribute to cancer. Health effects of exposures to carbon monoxide, sulfur dioxide, and nitrogen dioxide can include reduced work capacity, aggravation of existing cardiovascular diseases, effects on pulmonary function, respiratory illnesses, lung irritation, and alterations in the lung's defense systems. Adaptations to climate change should include ensuring responsiveness of air quality protection programs to changing pollution levels. Research needs include basic atmospheric science work on the association between weather and air pollutants; improving air pollution models and their linkage with climate change scenarios; and closing gaps in the understanding of exposure patterns and health effects. *Key words:* air pollution, climate change, criteria air pollutants, global warming, ozone, particulate matter. — *Environ Health Perspect* 109(suppl 2):199–209 (2001). *http://ehpnet1.niehs.nih.gov/docs/2001/suppl-2/199-209bernard/abstract.html*

The air is contaminated with pollutants that may adversely affect health. These pollutants have many sources: natural (e.g., volcanoes and decomposition of vegetation), agricultural (e.g., methane and pesticides), commercial (e.g., dry-cleaning operations and auto body shops), industrial (e.g., fossil fuel–fired electric power plants and manufacturing facilities), transportation (e.g., truck and automobile emissions), and residential (e.g., home gas and oil burners and wood stoves). People are constantly and ubiquitously exposed to air pollutants, whether indoors or outdoors. Through the Clean Air Act (*1*), however, the concentrations of key pollutants are regulated to protect the public's health. In this article we explore the potential linkages among climate change, air pollution exposures, and human health. Figure 1 sets out a conceptual framework for considering these linkages.

Climate change may affect exposures to air pollutants by *a*) affecting weather and thereby local and regional pollution concentrations (*2,3*); *b*) affecting anthropogenic emissions, including adaptive responses involving increased fuel combustion for fossil fuel-fired power generation; *c*) affecting natural sources of air pollutant emissions (*4,5*); and *d*) changing the distribution and types of airborne allergens (*6*). Local weather patterns—including temperature, precipitation, clouds, atmospheric water vapor, wind speed, and wind direction—influence atmospheric chemical reactions; they can also affect atmospheric transport processes and the rate of pollutant export from urban and regional environments to the global-scale environments (*2,3*). The chemical composition of the atmosphere may in turn have a feedback effect on the local climate. The multicomponent framework implied by Figure 1 involves hypotheses about climate change, its links to pollutant concentrations, and adaptive responses. The framework also incorporates the relationships between air pollution exposures and adverse health effects. Although the overall framework and its components are subject to numerous uncertainties, it offers an approach to addressing climate change, air pollution, and health effects.

Abundant evidence demonstrates that air pollution can have adverse health effects. Air pollution has been a public health concern in the United States for many decades, beginning with the well-chronicled air pollution disasters that caused acute and readily recognized excess morbidity and mortality. In Donora, Pennsylvania, an air pollution event over a 5-day period in October 1948 caused 19 deaths, well above the two deaths expected for this period. About 10% of the population of Donora was thought to be severely affected by the exposure, which resulted from thermal inversions that confined industrial combustion emissions at ground level (*7*). A few years later, when a slow-moving anticyclone came to a halt over London, England, in 1952, particulate and sulfur dioxide (SO_2) pollution built up over a 3-day period, causing 3,000–4,000 excess deaths (*7*). Prompted by these tragic episodes and mounting scientific evidence on the health effects of air pollution, the U.S. government began a series of measures to research, monitor, and regulate air pollution, culminating in the passage of the 1970 Clean Air Act.

Since 1970, implementation of the Clean Air Act (*1*) has resulted in controls over the ambient concentrations and emissions of the principal combustion-related pollutants and of several hazardous air pollutants (HAPS), primarily carcinogens and irritants. The combustion-related pollutants, which result from the combustion of coal and other fossil fuels, include particles, nitrogen oxides (NO_x), carbon monoxide (CO), and sulfur oxides (SO_x; depending on the sulfur content of the fuel). Another combustion-related pollutant, ozone (O_3), is a secondary pollutant formed

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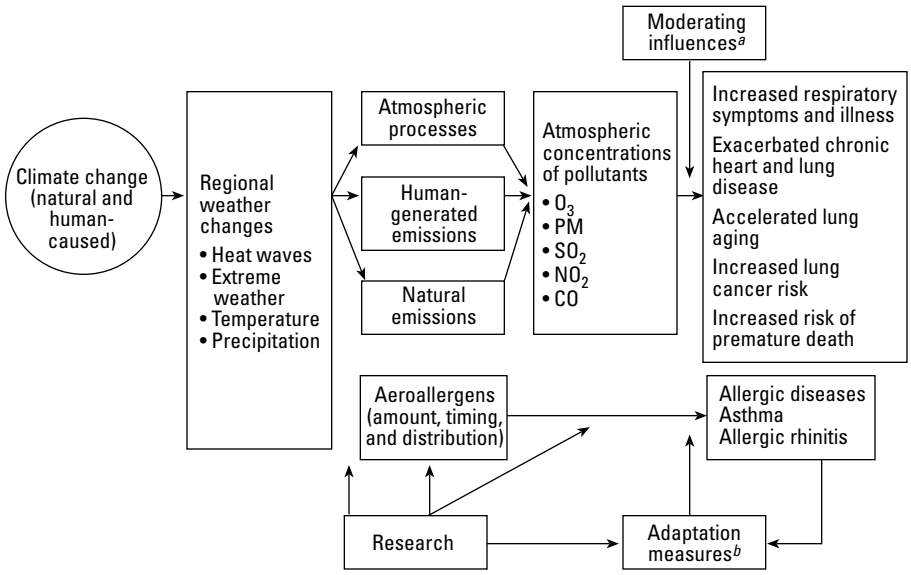

Figure 1. Potential air pollution-related health effects of climate change. [a]Moderating influences include nonclimate factors that affect climate-related health outcomes, such as population growth and demographic change, standards of living, access to health care, improvements in health care, and public health infrastructure. [b]Adaptation measures include actions to reduce risks of adverse health outcomes, such as emission control programs, use of weather forecasts to predict air quality levels, development of air quality advisory systems, and public education.

Table 1. Selected health effects and biologic markers of response associated with air pollution.[a,b]

Premature cardiorespiratory mortality
Deaths from heart or lung disease in excess of number expected
Increased health care use
Increased hospitalizations, physician visits, emergency department visits
Asthma exacerbations
Increased physician and emergency department visits, medication use, symptom reporting
Decreased peak flow measurements
Increased respiratory illness
Increased respiratory infections, physician visits, episodic symptoms
Increased respiratory symptoms
Decreased lung function
Spirometry, peak flow rates, airways resistance
Increased airways reactivity
Altered response to challenge with methacholine, carbachol, histamine, cold air
Lung inflammation
Influx of inflammatory cells, mediators, proteins
Altered host defense
Altered mucociliary clearance, macrophage function, immune response

[a]Clinical or public health significance of some effects is unknown. [b]Adapted with permission from the American Thoracic Society (*7*).

through complicated atmospheric reactions involving NO_x and hydrocarbons as the substrates and driven by sunlight. These pollutants [sulfur dioxide (SO_2), nitrogen dioxide (NO_2), particles, CO, and O_3] along with lead are regulated under the Clean Air Act as "criteria pollutants," referring to the process for developing the pollutant standards (*8*).

Although pollution control measures have reduced concentrations of the regulated pollutants, adverse effects of air pollution are still found at current concentrations using epidemiologic approaches. Monitoring data from the U.S. Environmental Protection Agency (U.S. EPA) show declining trends in pollutant concentrations over recent decades (*4*). However, epidemiologic studies of morbidity and mortality associated with air pollution exposure continue to show associations (*7,9–11*), and experimental data continue to elucidate the underlying mechanisms. At present, there is substantial concern about the public health consequences of particulate matter (PM), and a major national research program is now in progress to address key uncertainties in the evidence available for setting public policy (*12*).

The health effects of air pollution are diverse, extending from dramatic episodes of increased mortality at high concentrations to more subtle but detectable effects on respiratory health, particularly for persons made susceptible by underlying chronic heart or lung disease. Tables 1 and 2, taken from a recent comprehensive review by the American Thoracic Society, summarize health effects for the six criteria pollutants. This summary was based on extensive evidence drawn from animal and *in vitro* toxicology, human clinical exposure studies, field exposure studies, and epidemiologic studies. Since these tables were prepared for 1996 publication, further evidence of the effects of particles at contemporary concentrations has increased concern about the public health consequences of PM, particularly for persons with asthma, chronic obstructive lung disease, and coronary heart disease.

We do not attempt to review comprehensively the evidence of the health effects of air pollution. This literature is extensive and has been well reviewed elsewhere (*7,9–11*). For the criteria pollutants, the U.S. EPA's criteria documents offer periodic, complete summaries of the evidence (*13,14*). We do use our present understanding of air pollution and health to explore and illustrate how climate change may affect health through changing patterns of air pollution exposure.

Although in this section we focus primarily on the health effects of exposure to criteria air pollutants in particular, we also discuss briefly the issue of airborne allergens. Production of these allergens depends on the time of year and may increase with temperature increase from climate change. Climate change may affect the timing or duration of seasonal allergies such as hay fever.

Current Air Pollution Levels

A series of federal legislative efforts to control air pollution began in the 1950s. Major amendments of the Clean Air Act, in 1970 and subsequently, established the current federal air pollution program (*15–17*). The Clean Air Act has been amended several times, most recently in 1990. An important element of the Clean Air Act is the setting of National Ambient Air Quality Standards (NAAQS). Sections 108 and 109 of the Act require the U.S. EPA to identify pollutants that "may reasonably be anticipated to endanger public health or welfare" and to issue air quality criteria for them (*8*). The criteria, which must "accurately reflect the latest scientific knowledge" (*8*), are set with the participation of an independent scientific review panel, the Clean Air Scientific Advisory Committee. The U.S. EPA prepares Criteria Documents that extensively review and consolidate the current scientific literature for these pollutants. Besides health effects data, other scientific data are evaluated to provide a better understanding of the nature, sources, distribution, measurement, and atmospheric concentration of these pollutants. The U.S. EPA is required to review the adequacy of the NAAQS every 5 years. Most recently, the NAAQS for O_3 and PM were revised in a July 1997 rulemaking. However, the new NAAQS are not currently in effect because of legal challenge (*18*). The data described below concern the NAAQS in effect before 18 July 1997.

The U.S. EPA must propose and promulgate primary and secondary NAAQS for contaminants; these standards are based on the air quality criteria. Primary standards are set to protect the public health with an adequate margin of safety, including the health of sensitive populations such as asthmatics, children, and the elderly, whereas secondary standards protect against welfare effects such as decreased visibility and damage to animals,

Table 2. Health effects of air pollutants and populations at greatest risk.[a,b]

Agent	Numbers exposed at levels above NAAQS[c]	Groups at risk	Clinical consequences	Comments
O_3	69.7×10^6	Healthy adults and children	Decreased lung function Increased airway reactivity Lung inflammation Increased respiratory symptoms	Effects found at or below current NAAQS; effects increased with exercise
		Athletes, outdoor workers	Decreased exercise capacity	
		Asthmatics (and others with respiratory illnesses)	(Increased hospitalizations)	Effects seen in combination with acid aerosols and particles
NO_2	8.9×10^6	Healthy adults	Increased airway reactivity	Effects occur at levels found indoors with unvented sources of combustion
		Asthmatics Children	Decreased lung function Increased respiratory symptoms (Increased respiratory infections)	
SO_2	5.2×10^6	Healthy adults and COPD patients	Increased respiratory symptoms	Highly soluble gas with little penetration to distal airways
			Increased respiratory mortality and increased hospital visits for respiratory disease	
		Asthmatics	Decreased lung function	Observations related to short-term exposures
Acid aerosols	?	Healthy adults	Altered mucociliary clearance	Currently not a criteria pollutant: no NAAQS established
		Children	Increased respiratory illness	Effects seen in combination with O_3 and particles
		Asthmatics and others	Decreased lung function (Increased hospitalizations)	
Particles (PM_{10})	21.5×10^6	Children	Increased respiratory symptoms Increased respiratory illness	Effects seen alone or in combination with SO_2
		Patients with chronic lung/heart disease	Decreased lung function Premature mortality	
		Asthmatics	Increased asthma exacerbations	
CO	19.9×10^6	Healthy adults Children	Decreased exercise capacity	Effects increased with anemia or chronic lung disease
		Patients with ischemic heart disease	Decreased exercise capacity Angina pectoris (Premature mortality)	
Lead	14.7×10^6	Children	Altered neurobehavioral function	Elimination of leaded gasoline has resulted in marked reduction in atmospheric levels
		Adults	Increased blood pressure	

COPD, chronic obstructive pulmonary disease. [a]Effects shown at exposure levels occurring in the United States. Items in parentheses are associations that have been shown in some studies; additional confirmation is suggested. See Table 1 for definitions of health effects. [b]Adapted with permission from the American Thoracic Society (*7*). [c]Number of people residing in U.S. counties where exposures exceed NAAQS. Data are based on 1990 population data and 1991 air quality data. These do not reflect exposures indoors or to brief peak levels of pollutants outdoors.

crops, vegetation, and buildings. Standards can be set for both long-term (annual average) and short-term (24 hr or less) averaging times. They are enforced primarily by the states.

There are currently six criteria air pollutants—CO, lead, NO_2, O_3, PM, and SO_2. Both emissions and ambient air concentration data about these pollutants have been collected nationwide for almost three decades.

The Clean Air Act also provides for regulation of HAPs (also called air toxics). In 1990, the hazardous air pollutant program was substantially revised. Technology-based standards are set for HAPs, with further "residual risk" standards to be set if necessary where, even after implementation of the control technology, a source category is emitting HAPs at levels presenting an unacceptable risk to the public or the environment. Much less information is available concerning exposure to and health effects of air toxics than on criteria air pollutants. Another important federal air pollution control program is the effort to reduce acid rain by controlling emissions of precursor pollutants (SO_2 and NO_x) from fossil fuel-fired power plants. These and other programs are not discussed in detail in this report.

Sources and Trends of Air Pollution

Overview

Since the advent of the 1970 Clean Air Act, there have been substantial reductions in criteria air pollutant emissions and ambient pollutant concentrations, despite the fact that the population, the economy, and the nation's use of on-road vehicles have grown. Between 1970 and 1997 aggregate criteria air pollutant emissions decreased 31%, although the total U.S. population increased 31%, the number of vehicle miles traveled increased 127%, and the gross domestic product increased 114% (*5*). Changes in emissions of individual criteria pollutants over this period ranged from a 98% decrease in lead emissions to an 11% increase in NO_2 emissions (*5*). Table 3 summarizes the percent changes in national air quality concentrations and emissions over the last 10 years.

Overall, the declining trends in the emissions and monitored concentrations of several criteria pollutants provide strong evidence that air quality in the United States has improved significantly since the mid-1970s. The air quality in many areas, however, falls short of the NAAQS health-based standards. In 1997, approximately 52.6 million people in the United States resided in counties that did not meet the air quality standards for at least one criteria pollutant (*5*).

Emissions and air quality trends are not uniform among the various criteria pollutants. In general, O_3 has proven the most resistant to efforts to reduce its presence in the environment, whereas the presence of lead has been dramatically reduced since 1970 (after leaded gasoline was phased out).

As Figure 2 shows, fossil fuel combustion accounts for most of the carbon dioxide (CO_2) emissions in the United States. In addition,

most criteria air pollutants are also derived from fossil fuel combustion (*4,5*). Increased energy and fuel use would likely increase greenhouse gas emissions and, without pollution controls, increase emissions of criteria air pollutants. Sources and trends for each of the pollutants are discussed separately below.

Ozone

Ground-level O_3 is formed in the atmosphere by the reaction of volatile organic compounds (VOCs) and NO_x in the presence of sunlight. VOCs are emitted from a variety of manmade and natural sources. Anthropogenic sources include motor vehicles, chemical plants, refineries, factories, consumer and commercial products, and other industrial sources. Plant species responsible for biogenic VOC emissions of isoprene and monoterpenes vary by region of the country and include oak, citrus, eucalyptus, and pine in the Southwestern United States and oak, spruce, maple, hickory, pine, fir, and cottonwood in the Northeastern United States. NO_x is emitted from motor vehicles, fossil fuel-fired power plants, other sources of combustion, and natural sources including lightning and biologic processes in soil. Between 1988 and 1997, anthropogenic emissions of VOCs decreased 20% while those of NO_x increased by 1%.

Estimates of biogenic emissions consider variations in climate and land use, which have a strong impact on emissions rates. For example, roughly 60% of all biogenic VOCs are estimated to occur in the summer, when temperatures are higher than at other times of the year (*4*). Land use differences affect spatial variation in the density of biogenic VOC emissions; for example, higher VOC densities in the Southern United States are strongly linked to large areas of high-emitting oak trees.

The overall relative contributions of anthropogenic versus biogenic sources of VOCs have not been clearly established, and there is geographic variation in the relative contributions of these broad source groups. In the most recent estimates (1997), manmade VOC emissions (19.2 million tons) were exceeded by biogenic emissions (28.2 million tons), but there are large uncertainties in both biogenic and anthropogenic VOC emission inventories (*5*).

Changing weather patterns can contribute to yearly differences in O_3 concentrations (*5*). For example, the hot, dry, stagnant meteorologic conditions in 1995 in the Central and Eastern United States were highly conducive to O_3 formation. O_3 and precursor pollutants that cause O_3 can be transported into an area from pollution sources hundreds of miles upwind. In addition, ambient O_3 trends are influenced by VOC to NO_x ratios in the atmosphere and by changes in emissions from ongoing control measures (*5*). Between 1988 and 1997 ambient O_3 concentrations (1-hr average) decreased 19%.

O_3 patterns vary across the country. In 1997, about 48 million people lived in the 77 counties where O_3 levels exceeded the NAAQS. The highest O_3 concentrations that year were found in southern California, the Gulf Coast, and the ortheastern and North Central states, but for the first time the highest O_3 levels were recorded not in Los Angeles, California, but in Houston, Texas (*5*).

Sulfur Oxides, Particulates, and Acid Aerosols

The sources of and exposure to sulfur oxides, PM, and acid aerosols are presented together (here and frequently in other sources) because they often have common sources, primarily combustion processes (*19*). Acid aerosols (such as sulfuric acid) are generated by chemical reactions involving combustion emissions and sulfur and nitrogen oxides, which form sulfates and nitrates, respectively. Although SO_2 is one of the listed criteria pollutants under the Clean Air Act, it is likely that many of the health effects of concern for SO_2 reflect the combined action of the diverse components of the mixture of pollutants created by fossil fuel combustion (*19*).

Fuel combustion accounted for 85% of all SO_2 emissions in 1997 (*5*). Other sources included industrial processes and transportation.

Between 1988 and 1997, national annual mean SO_2 concentrations decreased 39%. The largest single-year reduction of 19% occurred between 1994 and 1995, mainly caused by Phase I implementation of the Acid Rain Program, which aimed to reduce SO_2 and NO_x emissions (*5*). In a 1995 study, the U.S. Geological Survey reported decreases in rainfall acidity in the eastern United States, particularly along the Ohio River Valley and in the mid-Atlantic states, indicating that reductions in SO_2 emissions have resulted in less acidic rainfall in these areas (*20*). Only one county, with a population of more than 80,000, failed to meet the ambient SO_2 NAAQS in 1997.

PM consists of solid or liquid particles found in the air, including dust, pollens, and soot and aerosols from combustion activities (*19*). Particles originate from a variety of mobile, stationary, and natural (e.g., wind erosion) sources, and their chemical and physical compositions vary widely. PM can be emitted directly or can be formed in the atmosphere when gaseous pollutants such as SO_2 and NO_x undergo transformation to form fine particles. The chemical and physical composition of PM varies depending on location, source, time of year, and meteorology. Typically, suspended particles in the atmosphere are categorized into two sizes: Those that are 3–30 μm in aerodynamic diameter (coarse mode) tend to be of natural origin and to be alkaline in total pH; those that are < 3 μm in aerodynamic diameter (fine mode) derive largely from manmade sources and include acid concentrates of combustion processes (*19*). The size of the particle is significant because particles < 2–3 μm in aerodynamic diameter tend to deposit deep in the lungs, in the terminal bronchioles and alveoli; larger particles tend to deposit in the upper airways (*19*). Early air monitoring for particulates measured total suspended particulates (TSPs), which included all suspended particles up to a vaguely defined size of 30–40 μm (*19*). Particulate air quality standards were changed in 1987 to control inhalable particles, defined as those < 10 μm aerodynamic diameter (PM_{10}). PM_{10} samples replaced the earlier TSP monitors, and the first complete year of PM_{10} data for most monitors was 1988. Between 1988 and 1997 average PM_{10} concentrations decreased 26%.

Table 3. National air pollutant emissions and concentrations.[a,b]

Pollutant	Emissions 1988	Emissions 1997	Emissions % change	Air quality concentration 1988–1997 (% change)
O_3[c]	24,027	19,214	–20	–19 (1 hr)
NO_2[d]	23,718	23,582	–1	–14
SO_2	23,154	20,369	–12	–39?
PM_{10}[e]	3,528	3,112	–12	–26
CO	116,081	87,451	–25	–38
Lead	7,053	3,915	–44	–67

[a]Lead emissions in short tons; other emissions in thousands of short tons. [b]Data from U.S. EPA (*5,13*). [c]Emissions of volatile organic compounds, many of which are ozone precursors. [d]Emissions of nitrogen oxides. [e]Includes only directly emitted particles. Does not include PM_{10} emissions estimates from agriculture and forestry, other combustion, cooling towers, fugitive dust, and natural sources. These sources totaled 30,469 thousand short tons in 1997. In addition, secondary PM formed from SO_2, NO_x, and other gases comprises a significant fraction of ambient PM.

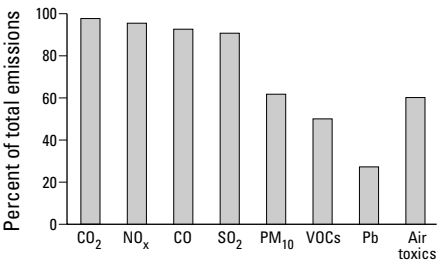

Figure 2. Emissions of air pollutants associated with fossil fuel combustion. Data from U.S. EPA (*5*).

Directly emitted PM generally has origins in two separate source groups, neither of which includes PM secondarily formed in the atmosphere from gaseous pollutants (e.g., SO_2 and NO_x). The first group comprises source categories similar to those in traditional emission inventories and includes fuel combustion, industrial processes, and transportation. Altogether, this group accounted for roughly 9% of total direct PM_{10} emissions nationwide in 1997. The remaining 91% of direct PM_{10} emissions in 1997 were accounted for by the second group, made up of miscellaneous anthropogenic and natural sources. These include fugitive dust, agriculture and forestry, wind erosion, wildfires, and managed burning. However, this second group can be difficult to quantify relative to the more traditionally inventoried sources, and its contribution tends to fluctuate a great deal from year to year (*45*).

Considering both the annual and 24-hr standards, there were 8 million people living in 13 counties with PM_{10} concentrations above the NAAQS in 1997.

Nitrogen Dioxide

High-temperature combustion processes (e.g., in motor vehicles, fossil fuel-fired power plants, and industrial boilers) generate nitric oxide (NO) and, to a lesser extent, NO_2; NO converts to NO_2 through oxidation reactions (*19*). NO_2 is a strong oxidizing agent that reacts in the air to form nitric acid, as well as toxic organic nitrates. It also plays a central role in the atmospheric reactions that produce ground-level O_3.

The two primary sources of NO_x emissions in 1997 were fuel combustion for energy (e.g., coal-fired electric utilities) and transportation (e.g., motor vehicles). Nationally, annual mean NO_2 concentrations decreased by 14% from 1988 to 1997 (*5*). All monitoring locations across the nation met the federal NO_2 NAAQS in 1997, for the fifth year in a row.

There are biogenic sources of NO, which oxidizes in ambient air to form NO_2. Biogenic NO emissions were estimated to be 1.5 million tons in 1997. As with biogenic emissions of VOCs, estimates of natural NO emissions are strongly affected by differences in climatology and land use. For example, relatively high densities of biogenic NO in the Midwestern United States are associated with areas of fertilized crop land (*4*).

Carbon Monoxide

National total CO emissions decreased 25% from 1988 to 1997. Transportation accounted for about three-fourths of CO emissions nationwide in 1997, with highway vehicle exhaust contributing more than half of all CO emissions. In cities, car exhaust contributes as much as 95% of all CO emissions, producing high concentrations of CO in local areas with heavy traffic congestion. Other sources of CO emissions in 1997 included industrial processes and nontransportation fuel combustion. Miscellaneous emissions (e.g., agriculture fires, forest wildfires) represented more than 10% of CO emissions in 1997 (*5*).

Nationally, average CO levels have decreased 38% from 1988 to 1997, and ambient levels in 1997 were the lowest recorded during the past 20 years of monitoring. Improvements in ambient CO levels have occurred despite a 25% increase in vehicle miles traveled. In 1997, only three counties with a combined population of 9 million people failed to meet the CO NAAQS.

Lead

Overall lead emissions decreased by 98% between 1970 and 1997 (*5*). Historically, the major source of lead emissions was on-road vehicles. However, the use of lead in gasoline for on-road fuels has been phased out entirely (on-road vehicles contributed only about 0.05% to total 1997 lead emissions). Leaded gasoline is still used in off-road vehicles such as farm equipment and nonroad transportation. Metals processing is now the major source of lead emissions, accounting for about half of lead emissions in 1997 (*5*).

Between 1988 and 1997, average lead concentrations at population-oriented monitors decreased 67% (*5*). Currently, the highest concentrations of lead are found around nonferrous smelters and other stationary sources of lead emissions. In 1997, four lead point sources had one or more source-oriented monitors that exceeded the NAAQS. The four counties containing these point sources, with a population of 2.4 million, did not meet the lead NAAQS in 1997 (*5*).

Health Effects of Air Pollution Exposure

Since the 1950s, with the initial impetus coming from the air pollution disasters, the health effects of air pollution have been extensively investigated. Despite substantial research, many questions remain unanswered concerning the risks of outdoor air pollution. Some of the key health effects ascribed to exposure to criteria air pollutants are listed in Table 1. Some of the outcomes listed in Table 1 are plainly adverse, e.g., premature death and increased hospitalizations or exacerbation of asthma. Others are biologic indications of responses that have uncertain clinical significance (*7,21*). The information linking these effects to air pollution exposure comes from a variety of scientific approaches, including animal toxicology, human clinical exposure studies, field exposure assessment studies, and epidemiologic investigations. The resulting data encompass molecular mechanisms to population-level impacts. For some observed effects, the mechanisms and specific pollutants responsible have not been definitively established (*7*). Furthermore, these pollutants and their health impacts have been studied (and are regulated and controlled) separately, although mixtures of these individual pollutants commonly occur (*7*). Nonetheless, evidence of health effects from these exposures has been the basis for setting policies to control air pollution throughout the world. Comprehensive reviews of this evidence have been reported elsewhere (*7–11,19,22*).

We do not attempt to review comprehensively the evidence on the health effects of the various air pollutants. Instead, we emphasize O_3 and particulate air pollution, the two pollutants for which concentrations are anticipated to be affected by climate change in the framework proposed in Figure 1. Although research is in progress on both of these pollutants, the evidence has identified a number of adverse health effects and has been the basis for standard-setting for both. With regard to climate change, we consider less relevant the combustion-related gaseous pollutants NO_x, sulfur oxides, and CO, although both nitrogen and sulfur oxides contribute to the formation of secondary particles.

Ozone

As noted above, photochemical pollution, for which O_3 is the index pollutant, is a continuing air pollution problem in many parts of the United States. In warm weather, ambient O_3 concentrations can exceed 0.12 parts per million (ppm) (the current 1-hr regulatory average) for several hours daily for 1 or more days (*7*). For many people in the United States, outdoor exposure to O_3 is limited because only a small part of the day is spent outside and people are encouraged to stay indoors, where levels are generally low, during episodes of particularly high O_3 levels. Some occupations and activities by adults and children lead to higher exposures and lung doses, caused by the time spent outside and the increased breathing rates involved in these activities, such as outdoor work in landscape and construction, strenuous outdoor exercise (adults and children), and outdoor play (*19*).

O_3 and other photochemical oxidants injure the epithelial surfaces onto which they are adsorbed. Experimental animal and *in vitro* studies have shown increased permeability and inflammation of airways; morphologic, biochemical, and functional changes; and decreased host defense functioning as a result of acute O_3 exposure (*23*). *In vitro* studies using very high concentrations of O_3 (> 10 mg/m^3) suggest that O_3 has a low potential to cause mutagenic, cytogenic, or

cellular transformation effects (*24*). Thus, the health effects of concern relate primarily to lung inflammation, with clinical manifestations arising from direct effects on the lung and possibly indirect effects arising from systemic consequences of lung inflammation and mediator release.

Exposure of healthy individuals (including children) to relatively low O_3 concentrations can cause lung inflammation, acutely decreased lung function, and respiratory impairment (*19*). The evidence of short-term effects on lung function comes from both experimental exposure and observational studies (*7*). The experimental studies have involved exposures ranging from less than 1 hr to up to 8 hr with intermittent periods of exercise (*7*). These studies have consistently shown transient decline of the forced vital capacity and symptoms of irritation, such as cough and chest pain, with exposure to O_3 at concentrations frequently measured in urban areas of the United States. The effects of O_3 on airways and alveoli have been evaluated in studies in which human subjects have been exposed while at rest and while exercising in a chamber on a stationary cycle or treadmill (*25–27*). Decrements in measures of lung function and physical performance, aggravation of respiratory tract symptoms, increased airway reactivity and evidence of acute inflammation have been demonstrated at exposure levels as low as 0.08 ppm (*25–27*). In healthy young adults, intense and sustained exercise, which increases the effective dose to the lungs, is required to provoke changes in lung function (*25–27*). Significant losses in lung function and symptoms of cough and pain with deep breathing have been observed after prolonged exposure to concentrations at 0.12 ppm O_3 (*28*). Similar acute, reversible changes in lung function have been observed in exercising children exposed to 0.12 ppm of O_3 (*29,30*). These studies all show wide but reproducible variability among individuals' sensitivity to O_3; the factors that contribute to this large intersubject variability remain undefined. These results suggest that children and adults who engage in prolonged exercise or labor outdoors may be at risk for adverse health effects at O_3 concentrations near the ambient standard and typical of summertime levels in some cities (*19*). Complementary evidence has been obtained from epidemiologic studies conducted at summer camps (*31*). In these studies, children showed transient reductions in lung function associated with ambient O_3 concentrations that were comparable to those measured in the experimental studies. There appears to be some degree of adaptation to the early, reversible effects.

Long-term exposure is suspected to contribute to the development and exacerbation of chronic lung diseases by causing permanent changes in the airways and alveoli and accelerating lung function decline. Animal models of long-term exposure to O_3 show alveolar changes consistent with the earliest stages of emphysema and also subtle changes in the small airways; these abnormalities would be expected to have the physiologic consequence of airflow obstruction (*31*). These experimental observations of airways and alveolar injury suggest that O_3 exposure may reduce the rate of lung growth during childhood and accelerate the decline of lung function during adulthood. Several long-term epidemiologic studies have been carried out to test these hypotheses. One prospective cohort study of children and adults in southern California showed increased decline for persons living in the communities with the higher levels of O_3 compared with those living in the lower-O_3 communities (*32*). In this study physiologic measurements were taken over 5-year intervals; there was a statistically significant decrease in respiratory function and nitrogen washout—an indicator of damage to small airways—in persons in communities exposed to higher levels of O_3, sulfates, and PM than those in less exposed communities. Reductions in nitrogen washout values in the range of 2–5% were observed in children. These findings suggest that aging of the lungs may be accelerated by long-term exposure to a mix of photochemical oxidants and other air contaminants (*32,33*). Cross-sectional evidence from college students in California also suggests effects of O_3 exposure during childhood on the level of lung function attained (*34*). A recent study of children in Los Angeles, California, also found lower lung function associated with peak O_3 exposure, particularly among children reported to spend more time outdoors (*35,36*).

The health consequences of short-term O_3 exposure have also been investigated in persons considered potentially susceptible, particularly those with asthma and chronic obstructive lung disease. Both epidemiologic and experimental approaches have been used. The results from clinical studies on subgroups (other than children) of the population that may be at particular risk from O_3 exposure, such as persons with asthma or other lung disorders, have been mixed. Generally, the lung function and symptom responses of asthmatics and patients with chronic obstructive pulmonary disease (COPD) do not appear different from those of healthy subjects experimentally exposed to O_3 (*37–39*), although the available evidence is limited. However, epidemiologic studies provide evidence that O_3 exposure may increase morbidity from asthma. Recent studies of asthmatics, particularly asthmatic children, have shown worsening of clinical symptoms and decreases in lung function associated with exposure to O_3 (*13*). In clinical studies O_3 exposure potentiates the effect of allergen exposure in sensitive asthmatics, perhaps as a consequence of increased penetrability of the respiratory epithelium from O_3 exposure (*40*). Time-series studies provide complementary evidence linking daily O_3 concentrations to indicators of asthma morbidity. For example, daily hospital asthma admissions are consistently associated with ambient O_3 levels in various locations in the Northeastern United States (*13*).

Time-series studies also indicate that O_3 may be a more general cause of morbidity and mortality. Numerous time-series studies (*31*) have reported that increased O_3 (and other pollutants) are associated with increased daily mortality counts. Total mortality counts are associated with O_3 levels, as are some cause-specific categories, including cardiac causes (*31*). Studies of hospitalization (*31*) also show associations of O_3 concentrations with cardiac and respiratory admissions, even after taking other pollutants into consideration. These findings have added to the concern that the health effects of O_3 exposure may still constitute a substantial public health problem, even at concentrations around current standards.

Particulate Matter

Particles are another pollutant of primary interest in relation to climate change. Particles are classified as primary or secondary in their origins. As noted, the primary particles are directly emitted from sources, and the secondary particles are formed from gaseous precursors, including the sulfur oxides and NO_x. During the 1990s there was resurgent interest in the health effects of PM, following evidence of short-term and long-term associations between levels of PM and mortality and morbidity (*41*). The health effects of particles and the mechanisms contributing to these health effects are presently under intensive investigation, and we do not attempt to cover either the full literature or the many areas of uncertainty currently under investigation (*12*). With regard to climate change, combustion-related particles are considered particularly relevant. Climate change may also lead to changes in particulate allergen exposures.

Both historically documented episodes and more recent time-series studies link PM to adverse effects on morbidity and mortality. The sudden increase in illness and death seen in the London air pollution episode of 1952 and the Donora incident of 1948, described above, was associated with high concentrations of PM and SO_2. There is little doubt that the pollution exposure caused those adverse effects (*19*). During the London episode, measurements of particles and SO_2 reached levels at least 10-fold greater than

peaks found in the most polluted cities in the United States at present. As noted, 3,000–4,000 excess deaths were attributed to this 1952 episode. An air pollution episode a decade later, which had similar SO_2 concentrations but much lower particle concentrations, was associated with about 800 excess deaths (*19*). This suggests that the excess deaths from the 1952 event may have been more closely associated with PM than with SO_2 pollution (*19*). Over the last decade, numerous reports of time-series analyses have assessed the association of daily mortality counts with levels of PM and other pollutants on the same or previous days (*14,41,42*). Overall, these studies show associations with measures of PM concentration that are robust with respect to control for levels of other pollutants and to control for weather (*10,11, 14,43*). Interpretation of these associations for public health purposes is clouded by uncertainty about the degree of life-shortening resulting from these short-term effects. However, the findings of several long-term cohort studies, including the Harvard Six Cities Study (*44*) and the Cancer Prevention Study of the American Cancer Society I (*45,46*), suggest that there may be a long-term effect of PM on mortality.

Particle concentration in outdoor air has also been associated with morbidity, particularly in the elderly. A series of reports based on Medicare data have assessed associations between hospital admissions in the elderly and levels of PM and other pollutants. These studies indicate an association between PM levels and increased risk for admissions for respiratory causes, including COPD and pneumonia, and for cardiovascular causes, including ischemic heart disease (*10,11, 47–50*). The potential mechanisms linking inhaled particles to acute cardiac consequences are still uncertain, although hypotheses have been offered concerning lung inflammation and cytokine release with effects on the heart (*51*). Exposure to particulates has been associated with increases in respiratory symptoms in a diary study of adults with COPD (*52*). A series of analyses on adults participating in the Health Interview Study have shown associations between exposure to particles and respiratory symptoms severe enough to restrict activity (*53*). These studies suggest that ambient particulate pollution, even at relatively low concentrations, exacerbates chronic respiratory conditions in adults (*19*).

Particle concentrations have also been associated with respiratory morbidity in children, as assessed by rates of hospital admissions and emergency room visits and by direct assessment of symptoms and lung function level (*41*). For example, Schwartz et al. reported an association between PM_{10} concentrations and daily rates of lower respiratory tract symptoms in a diary study of school children in the Harvard Six Cities study (*54*). Dockery et al. (*55*) have also reported associations of the rates of chronic cough, bronchitis, and chest illness in school children in the Harvard study with various measures of particulate pollution, including TSP, PM_{15}, $PM_{2.5}$, and sulfate. Associations with SO_2 were also positive, though weaker.

Carbon Monoxide

High exposures to CO, another criteria air pollutant, now occur primarily with certain occupations (e.g., firefighting) and unintended poisoning and suicide (e.g., defective or improperly used combustion devices). High exposure can cause acute poisoning, resulting in coma and death. Although exposures to CO in urban settings are generally several orders of magnitude lower than those associated with intoxication and poisoning, some exposures during urban activities may adversely affect the heart and the brain, the most oxygen-sensitive organs. Prolonged exposure to low-level outdoor CO may lead to development of carboxyhemoglobin levels at which adverse health effects have been clinically demonstrated for susceptible persons. Conditions such as cardiovascular disease, chronic respiratory disease, and pregnancy may put significant fractions of the population at elevated risk. Recent time-series studies continue to show evidence of associations of CO with mortality and hospitalization (*19*), but the effect of CO cannot be readily separated from those of other pollutants.

Studies of low-level exposures to date have focused primarily on subpopulations whose cardiovascular or respiratory health is compromised (*19*). Susceptible groups include people with ischemic heart disease, peripheral vascular disease, and COPD. In patients with coronary heart disease, controlled exposure studies suggest that exposure to elevated levels of CO impairs the response of the myocardium to increased metabolic demands and that ventricular arrhythmias associated with ischemia induced by exercise can be aggravated by exposure to CO (*19*). Although controlled clinical studies have also been conducted on patients with COPD, the evidence for effects of CO on exercise performance in this potentially susceptible subgroup is limited (*19*). There are also only inconsistent data addressing the possible impairment of central nervous system function after exposure to low concentrations of CO. Varying effects on visual perception (*56*), auditory perception (*57*), manual dexterity (*58*), and vigilance (*59*) have been reported, but in normal subjects, clinically important neurobehavioral deficits have not been observed below 10% carboxyhemoglobin (*19*).

Nitrogen Oxides

In outdoor air, NO_2 does not generally occur by itself, but as part of a complex mixture of primary and secondary pollutants. Consequently, characterizing the effects of NO_2 in outdoor air has proved difficult. The contribution of NO_2 to secondary particles and its role in the formation of O_3 may be more relevant to public health than any direct effect of the gas.

However, NO_2 does have the potential to compromise respiratory health. This oxidant gas combines with water in the lungs to form nitric and nitrous acids, which are believed to damage the lung epithelium via oxidation mechanisms (*60*). Dosimetric modeling suggests that NO_2 is absorbed principally in the large and small airways and that little is deposited in the alveoli (*61,62*). At extremely high concentrations (> 200 ppm), NO_2 causes extensive lung injury, including fatal pulmonary edema and bronchopneumonia (*63*).

Animal experiments show that exposure to NO_2 concentrations an order of magnitude higher than those generally found in ambient urban air can impair the defense mechanisms of the lung (*64,65*). Few similar studies have been done on human subjects. However, one study by Goings et al. (*66*) on the effects of acute NO_2 exposures of 1–3 ppm on 152 young, nonsmoking adults found that during the first 2 years of the investigation there were no statistically significant differences in immune response to controlled influenza infections between subjects and controls (similar adults not exposed to NO_2). However, during the third year 90% of the subjects developed an antibody response to exposure, compared with 70% of those exposed to placebo (*66*). Although these results suggest an effect, they are limited by the small sample size and the possibility that susceptibility to infection may have varied across the 3 years because of immunity acquired from natural influenza infections (*19*).

From a public health perspective, the relatively high concentrations and lengthy exposures used in animal and clinical studies are not representative of exposure in the community, although daily average personal exposure levels as high as 0.065 ppm may occur (*67*). Few data exist on the frequency and level of transient elevated exposures during daily activities. The relation between respiratory illness and symptoms and NO_2 exposure has been studied more frequently in the indoor setting. Much of the population's exposure to NO_2 takes place indoors, where sources include cooking stoves and space heaters (*19*). Brief exposure to concentrations as high as 0.500 ppm may be experienced while cooking with a gas stove or driving in traffic (*68*). Elevated levels are also expected when an unvented gas space heater is operated, but

such elevated levels generally are not sustained (*19*). Several epidemiologic studies have examined the relation between respiratory tract illness and symptoms and ambient levels of NO_x, with mixed findings (*19*).

Lead

As noted above, lead emissions (especially from leaded fuels) have declined dramatically over the past 30 years, as have ambient lead levels. The reduction of lead in air has greatly reduced exposure to lead through inhaled air and swallowed dust, food, water, and beverages contaminated with lead deposited from air (*19*). Some lead exposure will continue to occur through background exposure resulting from natural sources of lead and from past deposition of lead onto soil and into other media from which it can become airborne or enter the human food chain. Although lead contamination is generally a diminishing air pollution concern in the United States, lead will continue to be an important public health concern in this country because of its well-established neurologic impacts on children [see, e.g., U.S. EPA Lead Criteria Document (*69*)].

The Role of Climate

Climate change may affect exposures to air pollutants by *a*) affecting weather and thereby local and regional pollution concentrations; *b*) affecting anthropogenic emissions, including adaptive responses involving increased fuel combustion for power generation; *c*) affecting natural sources of air pollutant emissions; and *d*) changing the distribution and types of aeroallergens.

Local weather patterns—including temperature, precipitation, clouds, atmospheric water vapor, wind speed, and wind direction—influence atmospheric chemical reactions. They can also affect atmospheric transport processes and the rate of export of pollutants from urban and regional environments to global environments (*2,3*). In addition, the chemical composition of the atmosphere may in turn have a feedback effect on the local climate.

Weather is also associated with energy demands (e.g., for space heating and cooling) that could alter patterns of fossil fuel combustion. In particular, individual responses to extremely hot weather can result in large increases in air conditioner use. In addition, high temperatures cause increased VOC evaporative emissions when people fuel and run motor vehicles.

When assessing health impacts, we must consider the potential for interactions among climate change, lifestyle, and pollution exposures. For example, greater use of air conditioning to avoid heat stress would produce greater emissions of air pollutants. However, the closing of windows could reduce outdoor–indoor penetration of pollutants such as particles and O_3. As a result, total personal exposures might drop under this scenario. The net effect of these factors on health risks—increased pollutant levels and reduced exposures—has not been evaluated.

The seasonal variation in natural emissions of VOCs and NO suggests that warmer temperatures are associated with increased natural emissions. For example, an increase of 10°C can cause over a 2-fold increase in both VOC and NO biogenic emissions (*4*). Natural particulate emissions (e.g., from wildfires and soil erosion) can also be affected by weather patterns such as droughts.

Thus, changes in weather that may accompany climate change may affect atmospheric concentrations of air pollutants (*70,71*). Of particular concern are potential changes in O_3 and particulate concentrations. Nonetheless, the type (i.e., local, regional, or global), the direction (i.e., positive or negative), and the magnitude of changes in air quality that may be attributable to climate change are presently unknown.

Weather and climate could also affect health through exposures to biologic agents, including aeroallergens and microbiologic agents. For example, many allergies exhibit a seasonal pattern, reflecting pollen releases and levels in the air. This seasonality suggests that climate variability and weather may play a role in the amount and timing of such releases and consequent health outcomes. Over the longer term, climate influences the geographic distribution of plant species associated with allergens, although the precise impact of climatic changes on allergens is unknown.

The Role of Climate Change

Of the few studies that have attempted to quantify the potential effects of climate change on air quality, most have examined the impact of increased temperature on O_3 formation (*72–74*). In general, these studies find that O_3 concentrations increase as temperatures rise, although the estimated magnitude of the effect varies considerably. However, the ability of atmospheric models to simulate complex photochemical reactions in the atmosphere is limited for several reasons, including uncertainties in emission inventories. Further, several of these studies relied on assumptions for key variables such as emission levels, mixing heights, and cloudiness (*70,72–74*).

Because many aspects of weather affect air quality, and most of these have been held constant in modeling studies, the results of these studies should not be considered predictions of future air quality levels associated with climate change. Rather, they demonstrate the sensitivity of atmospheric air pollutants to changes in specific meteorologic variables. For example, the air quality simulations driven by increases in temperature and ultraviolet illumination have not addressed such issues as the frequency of occurrence of the kinds of stagnant weather episodes associated with the highest observed O_3 concentrations over broad areas.

Climate Change and O_3

As noted previously, ground-level O_3 is not emitted directly but rather is formed through interactions between the O_3 precursors (NO_x and VOCs) in the presence of sunlight. Meteorologic factors that, in theory, could influence surface O_3 levels include ultraviolet radiation, temperature, wind speed, precipitation, atmospheric mixing and transport, and surface scavenging.

There is a direct correlation between temperature and O_3 levels (Figure 3) (*75–77*). In general, an increase in atmospheric temperature accelerates photochemical reaction rates in the atmosphere and increases the rate at which tropospheric O_3 and other oxidants (e.g., hydroxyl radicals) are produced (*73,78*). However, O_3 levels do not always increase with an increase in temperature (e.g., when the ratio of VOCs to NO_x is low).

Increases in water vapor increase the potential for O_3 formation (*2*), as do frequent or intense high-pressure systems. Furthermore, forests, shrubs, grasslands, and other sources of natural hydrocarbons (VOCs) emit greater quantities at higher temperatures. Soil microbial activity may also increase with warmer temperatures, leading to an increase in NO_x emissions (*5*). Higher natural emissions of VOCs and NO_x could lead to an increase in tropospheric O_3 (*71*).

Climate change could reduce O_3 concentrations, however, by modifying factors that govern O_3-producing reactions (*70,71*); for example, a more vigorous hydrologic cycle could lead to an increase in cloudy days. More cloud cover, especially in the morning hours, could diminish reaction rates and thus lower O_3 formation.

In a preliminary study, Gery et al. (*76*) examined the effects of increased temperature and decreased stratospheric O_3 on tropospheric O_3 formation in 15 separate combinations of city and meteorologic episodes. The episodes covered varying levels of O_3 concentrations in Los Angeles; New York City; Philadelphia, Pennsylvania; Washington, DC; Phoenix, Arizona; Tulsa, Oklahoma; Nashville, Tennessee; and Seattle, Washington. The city-episode pairs (cases) were grouped into four classes according to the general level of photochemical reactivity in each case. Combinations of higher temperatures (+2°C and +5°C) and

decreased O_3 column (losses of about one-sixth to about one-third of the stratospheric O_3) were modeled in each case using the OZIPM3 photochemical trajectory box model (*76*).

The results of the study are summarized for the four groups of cases for both separate and combined atmospheric perturbations. The effects of stratospheric O_3 depletion (in the range considered, which corresponds to very large depletions) were larger than those of the temperature increases, as measured by changes in ground-level O_3 concentrations. The temperature effect (assuming base case stratospheric O_3) was found to increase ground-level O_3 by about 2–4% for a 2°C increase and by about 5–10% for a 5°C increase (*76*). These are averages over all of the groups. The more reactive groups showed greater increases and the less reactive groups showed smaller increases, which would imply that temperature impacts would be worst in those places where ground-level O_3 is already highest.

Morris et al. (*72*) examined the effects of future climate change on air quality, using an improved air quality simulation mode—the RTM-III, a three-dimensional regional oxidant model. The study examined the effects on daily tropospheric O_3 concentrations of a 4°C uniform temperature increase and an attendant increase in water vapor concentration, assuming a constant relative humidity. The model results indicated that changes in the highest daily O_3 concentrations could range from –2.4 to +20%. The number of exceedences of NAAQS for O_3 concentrations was estimated to increase by 1 to 2 times over the number of exceedences in the base case (i.e., no future temperature change scenario).

A further study (*73*) used an improved version of the OZIPM3 box model and a different set of cities. This study examined a broader range of reactivity conditions and used locally specific, model-predicted temperature changes corresponding to a doubled CO_2 experiment. The impact of temperature on biogenic hydrocarbon emissions was included. The study estimated the changes in VOC emission controls required to attain the O_3 NAAQS in each city (Memphis, Tennessee; Dallas, Texas; Philadelphia; Baton Rouge, Louisiana; and Atlanta, Georgia). In all cases the required controls increased approximately in proportion to the local increases in temperature. The stringency of the required controls increased further when the combined temperature and stratospheric O_3 perturbations were applied, confirming the results of the earlier study (*72*).

In the most recent of these studies (*74*), an updated regional photochemical grid model was applied in a simulation of impacts of climate change without the stratospheric O_3 perturbation. This study applied a 4°C temperature perturbation uniformly on a region encompassing the Northeastern United States. In addition, the amount of upward penetration of emission plumes and the mass of emitted hydrocarbons from biogenic sources were allowed to respond to the temperature increase. [Lamb et al. (*79*) estimated that natural VOC emissions from deciduous forests would increase by a factor of three with a temperature change from 20 to 30°C.] The response of evaporative hydrocarbon emissions from motor vehicles to the temperature increase was also included in the analysis (*74*). The model simulations showed that under global warming conditions the concentrations of O_3 increased throughout the region. Incremental increases in O_3 concentration associated with the 4°C increase ranged from about 28 parts per billion (ppb) at the maximum (on a simulated base level of 145 ppb) to 8 ppb at the minimum (on a base of about 27 ppb).

Climate Change and SO_2 and Nitrogen Oxides

SO_2 and NO_x oxidize in the atmosphere to form sulfuric acid and nitric acid, respectively. These acids may be deposited to the earth's surface in dry form as gases or aerosols or in wet form as acid rain. Wet deposition is determined by the amount, duration, and location of precipitation and changes in the total acid levels, which are in turn determined by atmospheric chemistry and precipitation patterns (*5,80*). Although regional patterns of acid deposition are uncertain, many of the factors that affect O_3 formation also influence acid deposition (*2,5*). Higher temperatures accelerate the oxidation rates of SO_2 and NO_x to sulfuric and nitric acids, increasing the potential for acid deposition.

Conversely, an increase in cloud cover may reduce the rates of transformation from SO_2 to acidic materials, thus reducing the potential for acid deposition. In contrast, a decline in the stratospheric O_3 concentration may increase acid deposition, because more ultraviolet radiation will be available to accelerate chemical reactions (*2,5*).

Changes in circulation and precipitation patterns will affect transport of acidic materials, which in turn will determine the geographic location of acid deposition (*2,80*). Local, regional, and national air quality levels, therefore, will be partially determined by changes in circulation and precipitation patterns (*2,70,80*).

Hales (*81*) used a storm-cloud model (PLUVIUS-2) to examine the impacts of a temperature increase on the production of acidic materials. The model results indicated that sulfate production increased by about 2.5 times for a 10°C increase in temperature.

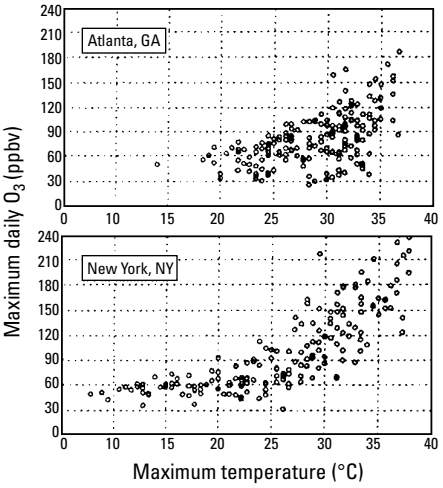

Figure 3. Maximum daily O_3 concentrations in Atlanta, Georgia, and New York, New York, versus maximum daily temperature, May–October, 1988–1990. ppbv, parts per billion volume. Data from U.S. EPA (*13*).

Greenhouse Gas Mitigation Policies and Air Quality

As noted above, fossil fuel combustion processes that produce CO_2 and other greenhouse gases also produce criteria pollutant emissions. Consequently, policies that strive to reduce greenhouse gas emissions (e.g., by reducing energy demand) can be expected to yield an ancillary benefit of reduced criteria pollutant emissions. One study found that such ancillary benefits can be substantial (*82*). For the United States the impact of reduced exposure to PM_{10} caused by climate mitigation via reduction of fossil fuel emissions that produce both PM and CO_2 emissions (energy-related CO_2 emissions 15% below 1990 levels in developed countries and 10% below projected 2010 levels in developing countries) was estimated to be about 30,000 fewer premature deaths per year by 2020. Because this study relied on numerous critical assumptions, the results should be viewed as illustrating the potential magnitude of the health benefits of a mitigation policy.

Natural Allergens and Fungal Growth

It has been suggested that climate change will increase exposure to natural allergens. The levels of aeroallergens, e.g., pollens, change with the seasons and affect a variety of allergy-related conditions. Pollen counts from birch trees (the main cause of seasonal allergies in northern Europe) increase with increasing temperature (*6*). However, the relationships among airborne fungal spores, pollen, peak expiratory flow rates, and temperature in asthmatic subjects could not be clearly discerned in one study (*83*). In another study, it was found that the incidence of seasonal allergic rhinitis was linked more strongly to land use change and farming practices than to climate (*84*).

Fungi have adapted to virtually all environments, but fungal growth is often enhanced at increased temperature and/or humidity. Climate change may lead to increases and changes in clinical behavior in fungal infections that penetrate into subcutaneous tissue, such as sporotrichosis (*85*). Debility due to persistent dermatophyte infection was a problem during the Vietnam War when troops spent time in tropical jungle conditions.

In addition, increased aridity and eventual desertification from increasing temperatures may increase particulate-carried fungal spores, multiplying the potential for endemic and epidemic pulmonary and systemic fungal infection with species indigenous to North America. This is documented most extensively for coccidioidomycosis, which is spread by dust, often preceded by increased rain (*86,87*). A well-documented outbreak of coccidioidomycosis followed the 1994 Northridge, California, earthquake (*88*). Much dust was disseminated following that earthquake, and dust exposure increased the risk for coccidioidomycosis. Global warming and population growth in arid areas such as the U.S. Southwest are likely to increase the risk of such hazards.

Research Needs and Data Gaps

Assessing air pollution-related health effects will require the information shown in Figure 4. Future emission inventories should take into account factors such as economic growth and vehicle miles traveled, air pollution control programs, and (for estimating cobenefits) greenhouse gas mitigation policies. These emission estimates can then be added to appropriate air quality models (e.g., Models3, Regional Acid Deposition Model, Urban Airshed Model). Frequently these models include weather variables, such as temperature and wind speed, that can be adjusted based upon future climate scenarios. The resulting air quality scenarios can then be combined with demographic and dose–response information to estimate possible health effects.

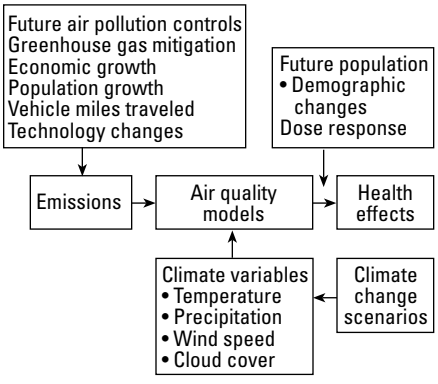

Figure 4. Assessing air pollution-related health effects of climate change.

Consideration of climate change, air pollution, and health effects inevitably takes place in a framework of uncertainty. Despite decades of intensive investigation, gaps remain in our understanding of the health effects of the most common combustion-related pollutants: PM, O_3, and the primary gases NO_x and sulfur oxides. Our understanding of the complex mixtures found in urban environments is particularly limited. Nonetheless, there is clear evidence of adverse health effects of air pollution, even at levels now present in many cities in the United States. An intensive research agenda on PM should accelerate our understanding of this key pollutant.

In the future, the pollutants of concern are likely to remain, as now, PM—both from its primary sources and from the secondarily formed particles—and photochemical pollution—O_3. Emissions of sulfur oxides and NO_x due to power generation might increase, but control technologies are available and can control such emissions. Both vehicles and stationary sources can emit toxic air pollutants such as butadiene. Whether such HAPs will change in the future is uncertain.

Despite the many uncertainties and the evidence concerning air pollution and its health effects and the potential impact of climate change on air pollution levels, reasonable models could be developed to assess the potential impact of climate change on the adverse effects of air pollution on populations. We already have available a variety of models relating pollutant concentrations to population-level damages. Such models are available, for example, for fossil fuel-fired power plants. The U.S. EPA performs risk assessments as an element of standard setting for the NAAQS. These damage functions would need to be joined to scenarios of pollution concentrations under various climate change alternatives. An important intermediate step involves downscaling global circulation model results to the geographic scale needed by air quality models.

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