The Potential Health Impacts of Climate Variability and Change for the United States: Executive Summary of the Report of the Health Sector of the U.S. National Assessment

Jonathan A. Patz,[1,] Michael A. McGeehin,[2,*] Susan M. Bernard,[1] Kristie L. Ebi,[3] Paul R. Epstein,[4] Anne Grambsch,[5] Duane J. Gubler,[6] Paul Reiter,[7] Isabelle Romieu,[2] Joan B. Rose,[8] Jonathan M. Samet,[9] and Juli Trtanj[10]*

[1]Department of Environmental Health Sciences, Johns Hopkins University School of Hygiene and Public Health, Baltimore, Maryland, USA; [2]National Center for Environmental Health, U.S. Centers for Disease Control and Prevention, Atlanta, Georgia, USA; [3]EPRI, Palo Alto, California, USA; [4]Center for Health and the Global Environment, Harvard Medical School, Boston, Massachusetts, USA; [5]Office of Research and Development, U.S. Environmental Protection Agency, Washington, D.C., USA; [6]Division of Vector-Borne Diseases, U.S. Centers for Disease Control and Prevention, Fort Collins, Colorado, USA; [7]Division of Vector-Borne Diseases, U.S. Centers for Disease Control and Prevention, San Juan, Puerto Rico; [8]Department of Marine Sciences, University of South Florida, St. Petersburg, Florida, USA; [9]Department of Epidemiology, Johns Hopkins University School of Hygiene and Public Health, Baltimore, Maryland, USA; [10]Office of Global Programs, National Oceanic and Atmospheric Administration, Silver Spring, Maryland, USA

We examined the potential impacts of climate variability and change on human health as part of a congressionally mandated study of climate change in the United States. Our author team, comprising experts from academia, government, and the private sector, was selected by the federal interagency U.S. Global Change Research Program, and this report stems from our first 18 months of work. For this assessment we used a set of assumptions and/or projections of future climates developed for all participants in the National Assessment of the Potential Consequences of Climate Variability and Change. We identified five categories of health outcomes that are most likely to be affected by climate change because they are associated with weather and/or climate variables: temperature-related morbidity and mortality; health effects of extreme weather events (storms, tornadoes, hurricanes, and precipitation extremes); air-pollution-related health effects; water- and foodborne diseases; and vector- and rodentborne diseases. We concluded that the levels of uncertainty preclude any definitive statement on the direction of potential future change for each of these health outcomes, although we developed some hypotheses. Although we mainly addressed adverse health outcomes, we identified some positive health outcomes, notably reduced cold-weather mortality, which has not been extensively examined. We found that at present most of the U.S. population is protected against adverse health outcomes associated with weather and/or climate, although certain demographic and geographic populations are at increased risk. We concluded that vigilance in the maintenance and improvement of public health systems and their responsiveness to changing climate conditions and to identified vulnerable subpopulations should help to protect the U.S. population from any adverse health outcomes of projected climate change. ***Key words*: air pollution, climate change, flooding, global warming, heat waves, vectorborne diseases, waterborne diseases.** ***Environ Health Perspect* 108:367–376 (2000). [Online 15 March 2000]**
http://ehpnet1.niehs.nih.gov/docs/2000/108p367-376patz/abstract.html

As part of a congressionally mandated national study of the impacts of climate variability and change in the United States, we assessed the potential impacts that projected changes in climate (based on modeled data developed for the national study) might have on a limited number of health outcomes that are associated with weather and/or climate.

In 1990, the U.S. Congress established the U. S. Global Change Research Program and required that it conduct a national assessment of the potential impacts of climate variability and change. The U.S. National Assessment of the Potential Consequences of Climate Variability and Change, which began in 1997, involves an assessment of the potential impacts of climate change over two time frames (to 2030 and to 2100) for geographic regions of the United States and for national sectors and/or interests, including health.

We conducted a literature review on, and consulted with a number of experts concerning, each of the health outcomes of interest: *a*) temperature-related morbidity and mortality; *b*) health effects of extreme weather events (i.e., storms, tornadoes, hurricanes, and precipitation extremes); *c*) air-pollution-related health effects; *d*) water- and foodborne diseases; and *e*) vector- and rodentborne diseases.

Some of these outcomes are relatively direct (e.g., the effects of exposure to extreme heat or extreme events); others involve intermediate and multiple pathways, making assessments more challenging (Figure 1). We used climate change projections developed for the national assessment as an underlying set of assumptions in our assessment. However, our analysis was for the most part not quantitative because of many layers of uncertainties in the data.

The climate change projections for the national assessment were the responsibility of a number of government and private climate scientists from the National Center for Atmospheric Research, the National Climatic Data Center of the National Oceanic and Atmospheric Administration, the Canadian Climate Center, and the Hadley Centre for Climate Prediction & Research (Bracknell, UK).

The scope of our inquiry was defined by the national assessment process, under which we were required to investigate the potential impacts of projected climate change on human health within a given framework of questions:

- What is the current status of the nation's health and what are current stresses on our health?
- How might climate variability and change affect the country's health and existing or predicted stresses on health?

Address correspondence to J.A. Patz, Program on Health Effects of Global Environmental Change, Department of Environmental Health Sciences, Johns Hopkins University School of Hygiene and Public Health, 615 N. Wolfe Street, Baltimore, MD 21205 USA. Telephone: (410) 955-4195. Fax: (410) 955-1811. E-mail: jpatz@jhsph.edu

*Co-chairs.

Special thanks to the U.S. EPA Global Change Research Program Director, J.D. Scheraga. We thank J.C. Beier, B. Boutin, R. Calderon, W.R. Daley, D. Dockery, D. Driscoll, D. Easterling, D. Engelberg, D.A. Focks, G. Greenough, M. Habib, W. Jakubowski, L. Kalkstein, T. Karl, E. Lipp, M. Lipsett, M. Mirabelli, R. Nasci, E. Noji, D. Paxman, W. Reisen, J. Riad, J. Schwartz, J. Selanikio, B.H. Sherman, R. Shope, A. Spielman, M. Wilson, and W. Yap. We also thank H. Curriero for report preparation and A. Redmon-Norwood for editing assistance.

This health sector assessment was sponsored by the U.S. Environmental Protection Agency Global Change Research Program as part of the overall U.S. National Assessment of the Potential Consequences of Climate Variability and Change (cooperative agreement CR 827040).

Received 15 November 1999; accepted 31 January 2000.

- What is the country's capacity to adapt to climate change; for example, through modifications to the health infrastructure or by adopting specific adaptive measures?
- What essential knowledge gaps must be filled to fully understand the possible impacts of climate variability and change on human health?

In our paper, we first describe information concerning climate variability and change generated by the climatology component of the national assessment process and provided to us as foundation climate assumptions to be used in our assessment. We then discuss uncertainties in vulnerabilities and adaptive capacities and describe the current and projected future background health status for context. We next discuss the potential impacts of climate change on each of the health outcomes analyzed and identify other potential health outcomes that may be assessed in the future. Finally, we discuss adaptation and prevention strategies.

Projections About Climate Variability and Climate Change

The national assessment climate models project that over the relevant time period the U.S. climate will be characterized by increased temperatures, altered hydrologic cycle, and increased variability.

Climatologists distinguish between the concepts of climate variability and climate change. Climate variability generally refers to short- to medium-term fluctuations around some mean climate state on time scales ranging from less than annual to multidecadal (e.g., 30 years). (*1,2*). For example, El Niño or La Niña events fall into this category. Climate change, on the other hand, refers to a fundamental shift in the mean state of the climate that generally pertains to longer term trends (*3*). Although future projections of climate change often are given as average values, climatologists caution that such change cannot be assumed to occur as a gradual true linear rise (*4,5*). Shorter term climate variability and the frequency of extreme climate events are projected to be altered as part of the physical consequences of long-term climate change (*6*).

These projections are based in part on historical data; however, a detailed systematic record of weather parameters is only available for some places for approximately the last 100 years, although indirect measurements from ice cores, tree rings, other paleodata, and written history extend further (*7*). In the past 100 years, the global surface temperature has warmed 0.7–1.4°F (*3,8,9*). In the contiguous United States, temperatures have increased by approximately 1°F (*10*), and precipitation in the United States has been increasing; much of this change is due to increases in heavy precipitation events (> 5 cm/day) and decreases in light-precipitation events (*4,10,11*). These historical data are consistent with climate change theory, which suggests that an altered hydrologic cycle accompanies the warming of the earth's surface (*12–14*).

Uncertainties of Vulnerability and Adaptive Capabilities

Projections of the extent and direction of some potential health impacts of climate variability and change can be made, but there are many layers of uncertainty (Table 1). First, methods to project changes in climate over time continue to improve, but climate models are unable to accurately project regional-scale impacts. Second, basic scientific information on the sensitivity of human health to aspects of weather and climate is limited. In addition, the vulnerability of a population to any health risk varies considerably depending on moderating factors such as population density, level of economic and technological development, local environmental conditions, preexisting health status, the quality and availability of health care, and the public health infrastructure.

It is also difficult to anticipate what adaptive measures might be taken in the future to mitigate risks of adverse health outcomes, such as vaccines, disease surveillance, protective technologies (e.g., air conditioning or water filtration/treatment), the use of weather forecasts and warning systems, emergency management and disaster preparedness programs, and public education (Figure 1).

The need for and the success of adaptation measures can be expected to vary in different parts of the country—for example, Chicago, Illinois, must plan for heat waves, and communities along the southeast coast must be prepared for hurricanes. For the most part, government organizations fund public health systems within the United States. Continued investments in advancing the public health infrastructure are crucial for adapting to the potential impacts of climate variability and change.

Climate/Health Impacts in the Context of Current Health Issues

To establish a baseline for projections of the potential impacts of climate on health, we reviewed the current status and context of health in the United States, as reflected in indicators such as life expectancy and the leading causes of death. We identified possible strains on public health and health care systems such as cost and population growth.

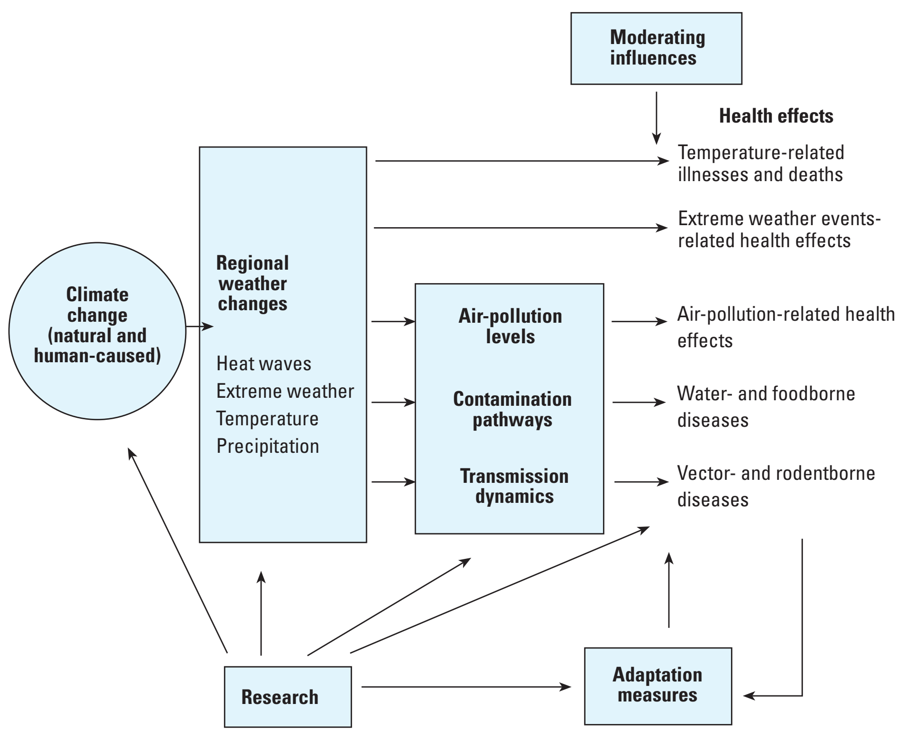

Figure 1. Potential health effects of climate variability and change. Moderating influences include nonclimate factors that affect climate-related health outcomes, such as population growth and demographic change, standards of living, access to health care, improvements in health care, and public health infrastructure. Adaptation measures include actions to reduce risks of adverse health outcomes, such as vaccination programs, disease surveillance, monitoring, use of protective technologies (e.g., air conditioning, pesticides, water filtration/treatment), use of climate forecasts and development of weather warning systems, emergency management and disaster preparedness programs, and public education.

Table 1. Summary of the health sector assessment.

Potential health impacts	Weather factors of interest[a]	Direction of possible change in health impact	Examples of some specific adaptation strategies	Priority research areas
Heat-related illnesses and deaths	Extreme heat and stagnant air masses	↑	Air conditioning Early warning	Improved prediction, warning, and response Urban design and energy systems Exposure assessment
Winter deaths	Extreme cold Snow and ice	↓		Weather relationship to influenza and other causes of winter mortality
Extreme weather events-related health effects	Precipitation variability (heavy rainfall events)[b]	↑	Early warning Engineering Zoning and building codes	Improved prediction, warning, and response Improved surveillance Investigation of past impacts and effectiveness of warnings
Air-pollution-related health effects	Temperature Stagnant air masses	↑	Early warning Mass transit Urban planning Pollution control	Relationships between weather and air pollution concentrations Combined effects of temperature/humidity on air pollution Effect of weather on vegetative emissions and allergens (e.g., pollen)
Water- and foodborne diseases	Precipitation Estuary water temperatures	↑	Surveillance Improved water systems engineering	Improved monitoring of weather/environment on marine-related diseases Land use impacts on water quality (watershed protection) Enhanced monitoring/mapping of fate and transport of contaminants
Vector- and rodentborne diseases	Temperature Precipitation variability Relative humidity	↑ or ↓	Surveillance Vector control studies	Rapid diagnostic tests Improved surveillance Climate-related disease transmission dynamics

[a]Based on projections provided by the National Assessment Synthesis Team (*15*). Other scenarios might yield different changes. [b]Projected change in frequency of hurricanes and tornadoes is unknown.

Urbanization, funding for public health infrastructure (e.g., sanitation systems and medical research), and scientific developments contributed to advances in health status in the past and are expected to do so in the future. Environmental conditions, such as air and water quality, are important determinants of health.

Chronic diseases—heart disease, cancer, stroke, and chronic obstructive pulmonary disease are the leading four—accounted for almost 75% of all deaths in 1996 for the 25- to 64-year-old age group (*16*). Injuries and infectious diseases remain significant causes of morbidity and mortality in the United States; infectious diseases caused one-third of the deaths in the United States in 1992, primarily because of respiratory tract infections, human immunodeficiency virus (HIV), and septicemia (*17*). Patterns of illness and death vary substantially by socioeconomic status, geographic region, race, age, and sex (*16*).

Populations at risk. Certain populations within the United States—the poor, the elderly, children, and immunocompromised individuals—may be more vulnerable to many of the health risks that might be initially exacerbated by climate change. For example, poverty is a risk factor for heat-related illnesses and deaths because the poor are more likely to live in urban areas and are less likely to be able to afford air-conditioning. Thus, making air-conditioned environments readily available to the poor is an adaptive response strategy to reduce illnesses and deaths in heat waves. Understanding what groups may be the most affected by climate change is critical to effective targeting of prevention or adaptation strategies. For example, air pollution and heat advisory warnings should specifically target children and the elderly, respectively.

It is important to recognize that the proportion of elderly (65 years of age and older) and very elderly (85 years of age and older) residents is expected to rise in the coming decades. The proportion of the senior population in the very elderly category is growing fast: their numbers rose 274% between 1960 and 1994, while the entire U.S. population grew only 45% (*18*). Age can be expected to be accompanied by multiple chronic illnesses that may result in increased vulnerability to infectious disease or external/environmental stresses such as extreme heat (*18*). Poverty, which increases with age in the elderly, may add to this vulnerability (*19*).

Similarly, although the proportion of children younger than 5 years of age is not expected to grow as significantly as the proportion of the elderly, their number will increase even if immigration levels are kept constant. The variables that may affect children's special vulnerability to the possible impacts of climate change include poverty (currently, approximately 20% of children in the United States are poor) (*16*), access to medical care, and children's susceptibility to environmental hazards because of their size, their behavior, and the fact that they are growing and developing (*20*).

Finally, it is anticipated that the proportion of immunocompromised people in the United States may increase with the aging of the population and the success of medical treatments (e.g., cancer therapy and HIV medications), but data are difficult to obtain. For example, survival has improved for acquired immunodeficiency syndrome (AIDS) patients, resulting in a 12% increase in 1996–1997 in the number of people living with AIDS (*21*). AIDS patients and other immunocompromised individuals may be more susceptible to waterborne and vectorborne pathogens, to the adverse impacts of exposure to elevated levels of certain air pollutants, and to debilitation due to physical stresses, such as those experienced during heat waves or in adverse emergency weather conditions, unless they can be adequately protected from those stresses with access to air conditioning, sanitation, safe water, and sufficient food.

Potential Impacts of Projected Climate Change on Health

Temperature-related illnesses and deaths. Heat and heat waves are projected to increase in severity and frequency with increasing global mean temperatures. Studies of heat waves in urban areas have shown an association between increases in mortality and increases in heat, measured by maximum or minimum temperature, heat index (a measure of temperature and humidity), or air-mass conditions (*22,23*). For example, after a 5-day heat wave in 1995 in which maximum temperatures in Chicago ranged from 93 to 104°F, the number of deaths increased 85% over the number recorded during the same period of the preceding year (*24*). At least 700 excess deaths (deaths beyond those expected for that period in that population) were recorded, most of which were directly attributed to heat (*22,24,25*).

Exposure to extreme and prolonged heat is associated with heat cramps, heat syncope (fainting), heat exhaustion, and heat stroke. These health effects appear to be related to environmental temperatures above those to which the population is accustomed. Models of weather–mortality relationships indicate that populations in northeastern and midwestern U.S. cities may experience the greatest number of heat-related illnesses and deaths in response to changes in summer temperature, and that the most sensitive regions are those where extremely high temperatures occur infrequently or irregularly (*26*). For example, Chicago, Philadelphia, Pennsylvania, and Cincinnati, Ohio, have recently experienced a heat wave that resulted in an increased number of heat-related deaths. Physiologic and behavioral adaptations among vulnerable populations may reduce morbidity and mortality due to heat. Although long-term physiologic adaptation to heat events has not been documented, adaptation appears to occur as the summer season progresses; heat waves early in the summer often result in more deaths than subsequent heat waves or than those occurring later in the summer (*26*). Heat waves are episodic, and although populations may adapt to gradual temperature increases, physiologic adaptation for extreme heat events is unlikely.

Within heat-sensitive regions, populations in urban areas are the most vulnerable to adverse heat-related health outcomes. The heat index and heat-related mortality rates are higher in the urban core than in surrounding areas (*27*). Urban areas retain heat throughout the nighttime more efficiently than do outlying suburban and rural areas (*28,29*). The absence of nighttime relief from heat for urban inhabitants may be a factor in excessive heat-related deaths.

The size of U.S. cities and the proportion of U.S. residents living in them is projected to increase; therefore, the population at risk for heat-related illnesses and death may also increase. High-risk subpopulations include people who live in the top floors of apartment buildings in cities and who lack access to air-conditioned environments (either at home or elsewhere). The elderly (*30–33*), young children (*30*), the poor (*34,35*), and people who are bedridden or on medications that affect the body's thermoregulatory ability are particularly vulnerable (*36–38*).

There is evidence that heat-related illnesses and deaths are largely preventable through behavioral adaptations including the use of air-conditioning and increased fluid intake (*36*), although the magnitude of mortality reduction cannot be predicted. The proportion of housing units with central and/or room unit air-conditioning ranges from below 30% in the Northeast to almost 90% in the South (*39*). The use of air-conditioning in homes, workplaces, and vehicles has increased steadily over the past 30 years and is projected to become nearly universally available in the United States by the year 2050 (*39,40*).

Death rates are higher in the winter than in the summer and it is expected that milder winters could reduce the number of deaths in winter months (*23*). However, the relationship between winter weather and mortality has been difficult to interpret. For example, many winter deaths are due to respiratory infections such as influenza, and it is unclear how influenza transmission would be affected by warmer winter temperatures. In addition, studies indicate an association between snowfall and fatal heart attacks (from winter precipitation rather than cold temperatures) (*41,42*). The net effect on winter mortality from climatic changes is uncertain and the overall balance between changes in summer and winter weather-related deaths is unknown.

Beyond individual behavioral changes, adaptation measures include the development of communitywide heat emergency plans, improved heat warning systems, and better heat-related illness management plans. Research can refine each of these measures, including which weather parameters are the most important in the weather–health relationship, the associations between heat and nonfatal illnesses, the evaluation of implemented heat response plans, and the effectiveness of urban design in reducing heat retention.

Health effects related to storms, tornadoes, hurricanes, and precipitation extremes. Climate change may alter the frequency, timing, intensity, and duration of extreme weather events (*4,12,13*), i.e., meteorologic events that have a significant impact on local communities. There is evidence that increases in heavy precipitation occurred over the last 20 years and may occur in the future as temperature increases (*4*). Climate models currently are unable to accurately project changes in extreme events such as floods, hurricanes, and tornadoes, making it difficult to assess future potential health impacts of such events.

Injury and death are the direct health impacts most often associated with natural disasters. Secondary health effects may also occur. These impacts are mediated by changes in ecologic systems and public health infrastructures, such as bacterial proliferation and the availability of safe drinking water. The health impacts of extreme weather events such as floods and storms hinge on the vulnerabilities of the natural environment and the local population, as well as on their capacity to recover. A community's level of preparedness greatly affects the severity of the health impacts of an extreme event.

From 1945 to 1989, 145 natural disasters caused 14,536 deaths in the United States, an average of 323 deaths/year (*43*). According to the National Weather Service (*44*), severe storms caused 600 deaths and 3,799 reported injuries in 1997. Floods are the most frequent natural disaster and the leading cause of death from natural disasters in the United States; the average annual loss of life is estimated to be as high as 146 deaths/year (*45*). Hurricanes also pose an ongoing threat; an average of two each year make landfall on the U.S. coastline (*46*). The impacts of hurricanes may include injuries and deaths resulting from strong winds and heavy rains.

Depending on the severity and nature of the weather event, people may experience disabling fear or aversion (*47*). There is controversy about the incidence and continuation of significant mental problems, such as posttraumatic stress disorder (PTSD), after disasters (*48*). However, an increase in the number of mental disorders has been observed after several natural disasters in the United States. Increased psychologic problems were reported during a 5-year period after Hurricane Agnes caused widespread flooding in Pennsylvania in 1972 (*49*). More recently, a longitudinal study of local residents who lived through Hurricane Andrew showed that 20–30% of the adults in the area met the criteria for PTSD at 6 months and 2 years after the event (*50*).

A population's ability to minimize the potential health effects associated with extreme weather events is based on a number of diverse and interrelated factors, including building code regulations, warning systems, and disaster policies; evacuation plans; adequate relief efforts; and recovery (*51*). There are many federal, state, and local government agencies and nongovernment organizations involved in planning for and responding to natural disasters in the United States. For example, the Federal Emergency Management Agency recently launched its

National Mitigation Strategy (*52*), which is designed to increase public awareness of natural hazard risk and to reduce the risk of death, injury, community disruption, and economic loss. This strategy represents a comprehensive effort to address severe events with a series of initiatives and public–private partnerships.

Future research on extreme weather events and associated health effects should focus on improving climate models to project trends, if any, in regional extreme events. This type of improved prediction capability will assist in public health mitigation and preparedness. In addition, epidemiologic studies of health effects beyond the direct impacts of disaster will provide a more accurate measure of the full health impacts and will assist in planning and resource allocation.

Air-pollution-related health effects. Air pollutants have many sources: natural (e.g., vegetation and volcanoes), agricultural (e.g., methane and pesticides), commercial (e.g., dry cleaning operations and auto body shops), industrial (electric power plants and manufacturing facilities), transportation (truck and automobile emissions), and residential (home gas, oil burners, and wood stoves). Ambient levels of regulated air pollutants (which include particulate matter, ozone, carbon monoxide, and sulfur and nitrogen oxides) have generally dropped since the mid-1970s, but air quality in many parts of the country falls short of health-based air quality standards. In 1997, approximately 107 million people in the United States lived in counties that did not meet the air quality standards for at least one regulated pollutant.

Air pollution is related to weather both directly and indirectly. Climate change may affect exposures to air pollutants by *a*) affecting weather and thereby local and regional pollution concentrations (*53,54*); *b*) affecting anthropogenic emissions, including adaptive responses involving increased fuel combustion for power generation; *c*) affecting natural sources of air pollutant emissions (*55,56*); and *d*) changing the distribution and types of airborne allergens (*57*). 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 exports from urban and regional environments into the global scale environments (*53,54*). In addition, the chemical composition of the atmosphere may in turn have a feedback effect on the local climate.

If the climate becomes warmer and more variable, air quality is likely to be affected. For example, if warmer temperatures lead to more air-conditioning use, power plant emissions could increase without additional air pollution controls. Increased temperatures may enhance the formation of ground-level ozone, particularly in urban areas (*56,58–61*). Changing weather patterns contribute to yearly differences in ozone concentrations (*56*); for example, the hot, dry, stagnant meteorologic conditions in 1995 in the central and eastern United States were highly conducive to ozone formation. However, the specific type of change (local, regional, or global), the direction of change in a particular location (positive or negative), and the magnitude of change in air quality that may be attributable to climate change are not known.

Because the effect of climate change on all of the air pollutants of concern, especially particulate matter, is unknown, it is difficult to determine the overall effect of climate variability and change on respiratory health. Health effects associated with climate impacts on air pollution will depend on future air pollution levels. Since 1970, emissions and ambient air pollutants have declined overall (*61*). However, the majority of regulated air pollutants are from fossil fuel combustion (*55,56*) and, as a result, increased energy and fuel use would increase emissions of air pollutants without additional air pollutant controls. Integrated air quality modeling studies will be necessary to assess more quantitatively the potential health impacts of air quality changes associated with global climate change. These models would need to incorporate variables such as future anthropogenic emissions (driven by economic growth, air pollution controls, vehicle usage, and possible changes in the use of fuel for heating and cooling); future biogenic emissions (factoring in possible responses to changing climate); and changes in local meteorology due to global climate change.

Exposures to air pollutants have serious public health consequences. Ground-level ozone can exacerbate respiratory diseases by damaging lung tissue, reducing lung function, and sensitizing the lungs to other irritants (*62*). Short-term drops in lung function caused by ozone are often accompanied by chest pain, coughing, and pulmonary congestion (*63*). Epidemiologic studies have found that exposure to particulate matter can aggravate existing respiratory and cardiovascular diseases, alter the body's defense systems against foreign materials, damage lung tissue, and may cause cancer and premature death (*63,64*). Health effects of exposures to carbon monoxide, sulfur dioxide, and nitrogen dioxide can include visual impairment, reduced work capacity, aggravation of existing cardiovascular diseases, effects on breathing, respiratory illnesses, lung irritation, and alterations in the lung's defense systems (*63,64*).

In addition to affecting exposure to air pollutants (whether man-made or naturally emitted), climate change may also play a role in human exposure to airborne allergens. Plant species are sensitive to weather, and warmer temperatures may enhance pollen production or alter the geographic distribution of plant species (*57*). Consequently, climate change may adversely impact the occurrence and severity of asthma, the most common chronic disease of childhood, and affect the timing or duration of seasonal allergies such as hay fever.

Climate change may affect the amount of time individuals spend indoors (e.g., individuals may spend more time in air-conditioned environments to avoid extreme heat, or may spend more time outdoors if winter temperatures are milder), resulting in changed exposure to indoor air pollutants and allergens. In some cases, these indoor environments may be more dangerous than the ambient conditions.

Adaptation measures include ensuring the responsiveness of federal and state air quality protection programs to changing pollution levels. These standards are designed to protect the public health by limiting emissions of key air pollutants and thus reducing ambient concentrations. The Pollutants Standards Index (*65*), a U.S. Environmental Protection Agency coordinated health advisory system that provides warnings for both the general population and susceptible individuals, could be further strengthened for specific pollutants.

Future research in the area of health effects associated with air pollution should include basic atmospheric science elucidating the association between weather, ozone, particulates, and other air pollutants and aeroallergens; improving existing models (e.g., expanding the spatial domain and lengthening the duration of modeled events) and their linkage with climate change scenarios; and closing the gaps in our understanding of common pollutants, such as particulate matter and ozone, and of individual exposures to these pollutants.

Water- and foodborne diseases. More than 200 million people in the United States have direct access to treated public water supply systems, yet as many as 9 million annual cases of waterborne disease have been estimated (*66*), although high uncertainty accompanies this estimate, and reporting is variable by state (*67*). Although most of these cases of waterborne disease involve mild gastrointestinal illnesses, other severe outcomes such as myocarditis are now recognized. These infections and illnesses can be chronic and even fatal in infants, the elderly, pregnant women, and people with weakened immune systems (*68,69*).

In the United States, foodborne diseases are estimated to cause 76 million cases of illness, with 325,000 hospitalizations and 5,000 deaths/year (*70*). Microbiologic agents in water (e.g., viruses, bacteria, and protozoa) can contaminate food (e.g., shellfish and fish). In addition, there have been instances of contamination of fresh fruits and vegetables by waterborne pathogens (*71*).

The routes of exposure to water- and foodborne diseases include ingestion, inhalation, and dermal absorption of microbial organisms or algal toxins. For example, people can ingest waterborne microbiologic agents by drinking contaminated water, by eating seafood from contaminated waters, or by eating fresh produce irrigated or processed with contaminated water (*71*). They also may be exposed by contact with contaminated water through commerce (e.g., fishing) or recreation (e.g., swimming) (*72*). The waterborne pathogens of current concern include viruses, bacteria, and protozoa. Examples include *Vibrio vulnificus*, a naturally occurring estuarine bacterium responsible for a high percentage of the deaths associated with shellfish consumption (*73,74*); *Cryptosporidium parvum* and *Giardia lamblia*, associated with gastrointestinal illnesses (*75*); and biologic toxins associated with harmful algal blooms (*76*). Many of these were discovered only recently and are the subject of ongoing research.

Between 1980 and 1996, 401 disease outbreaks associated with drinking water were reported, with more than 750,000 associated cases of disease (*75*). More than 400,000 of those cases (including 54 deaths) occurred in a 1993 *Cryptosporidium* outbreak that resulted from the contamination of the Milwaukee, Wisconsin, water supply (*77*). A contributing factor in the contamination, in addition to treatment system malfunctions, was heavy rainfall and runoff that resulted in a decline in the quality of raw surface water arriving at the Milwaukee drinking water plant (*78*). Studies from other locations in the United States found positive correlations between rainfall and *Cryptosporidium* oocyst and *Giardia* cyst concentrations in river water (*79*) and human disease outbreaks (*80*). Many water treatment facilities still have difficulty removing these pathogens.

Changes in precipitation, temperature, humidity, salinity, and wind have a measurable effect on the quality of water used for drinking, recreational, and commercial use, and as a source of fish and shellfish. Direct weather associations have been documented for waterborne disease agents such as *Vibrio* bacteria (*81*), viruses (*82*), and harmful algal blooms (*83*). In Florida during the strong El Niño winter of 1997–1998, high precipitation and runoff greatly elevated the counts of fecal bacteria and infectious viruses in local coastal waters (*83*). In Gulf Coast waters, *Vibrio vulnificus* bacteria are especially sensitive to water temperature, which dictates its seasonality and geographic distribution (*81,84*). In addition, toxic red tides proliferate as seawater temperatures increase (*85*). Over the past 25 years along the East Coast, reports of marine-related illnesses increased in correlation with El Niño events (*83*).

For many waterborne diseases, the management and disposal of sewage, biosolids, and other animal wastes and the protection of watersheds and fresh water flows are critical variables that impact water quality and the risk of waterborne disease (*68*). In September 1999, the largest reported waterborne associated outbreak of *Escherichia coli* O157:H7 occurred at a fairground in the state of New York and was linked to contaminated well water (*86*). The likelihood of this type of problem occurring increases under conditions of high soil saturation, which enhances the rapid transport of microbiologic organisms (*87*). Finally, many communities in the United States continue to use combined sewer and storm water drainage systems; these may pose a health risk should the frequency or intensity of storms increase, because raw sewage bypasses treatment and is discharged into receiving surface waters during storms (*88*).

Current adaptations for assessing and preventing waterborne diseases include legal and administrative measures such as water safety criteria, monitoring requirements, and health outcome surveillance, as mandated under the Safe Drinking Water Act, with amendments in 1996 (*89*). Recent legislative and regulatory attention has focused on improved treatment of surface water to address microbial contaminants and on ground water and watershed protection (*68,90*).

With respect to marine-related human disease outbreaks, protection is provided by measures such as adequate sewage/sanitation systems and safe food storage infrastructures, and beach and recreational water monitoring (*91*). However, these measures are inadequate for microbial contaminants. With increasing trends in food importation, improved surveillance and preventive measures are required (*71*), as well as a better understanding of how climate and weather might affect food and water safety outside the United States.

Important knowledge gaps must be addressed to improve the assessment of the association of climate with waterborne disease issues. Determinants of transport and the fate of microbial pollutants associated with rainfall and melting snow are not well quantified. Further studies should address the influence of varying land use on the water quality in watersheds. For urban watersheds, much of the current annual load of contaminants is transported into fresh and marine bodies of water during storm events. For these reasons, regional and even localized projections of changes in the intensity and frequency of storms and changes in land use are required for improving climate variability/health assessments.

Advances in monitoring are necessary to improve our knowledge base and enhance early warning and prevention capabilities. Application of existing technologies could be expanded, such as molecular fingerprinting to track contaminant sources (*92*), improvement of monitoring systems (*93*), and the use of satellite remote sensing to detect coastal algal blooms (*94*). Coordination and integration of monitoring across the varying agencies responsible for waterborne, foodborne, and coastal surveillance systems could greatly enhance our knowledge and adaptive potential.

Vector- and rodentborne diseases. Diseases transmitted between humans by blood-feeding arthropods (insects, ticks, and mites), such as plague, typhus, malaria, yellow fever, and dengue fever were once common in the United States and in Europe (*95–97*). The ecology and transmission dynamics of these vectorborne infections are complex and the factors that influence transmission are unique to each disease. It is not possible, therefore, to make broad generalizations on the effect of climate on vectorborne diseases (*97,98*). Many of these diseases are no longer present in the United States, mainly because of changes in land use, agricultural methods, residential patterns, human behavior, and vector control. However, diseases that may be transmitted to humans from wild animals (zoonoses) continue to circulate in nature in many parts of the country. Humans may become infected with the pathogens that cause these diseases through transmission by insects or ticks. For example, Lyme disease, which is tickborne, circulates among white-footed mice in woodland areas of the Mid-Atlantic, Northeast, upper Midwest, and West Coast of the United States, and humans acquire the pathogen when they are bitten by infected ticks (*99*). Fleaborne plague incidence increased in conjunction with increasing rodent populations after unseasonal winter–spring precipitation in New Mexico (*100*).

Humans may also become infected with pathogens that cause zoonotic diseases by direct contact with the host animals or their body fluids, as occurs with Hantavirus

Pulmonary Syndrome (HPS). Hantaviruses are carried by numerous rodent species and are transmitted to humans through contact with rodent urine, droppings, and saliva, or by inhaling aerosols of these products. In 1993, a previously undocumented hantavirus, Sin Nombre, emerged in the Four Corners region of the rural southwestern United States, causing HPS (*101*). As of 1999, 231 cases had been confirmed in the United States and > 650 in the Americas, with a mortality of 42% in otherwise healthy individuals (*102*).

The impact of weather on rodent populations may affect disease transmission. The Four Corners outbreak was attributed to an explosion in the mouse population caused by an increase in their food supply resulting from unusually prolonged rainfall associated with the 1991–1992 El Niño event (*103*).

Flooding has also been associated with rodentborne leptospirosis, as occurred in the 1995 epidemic in Nicaragua. A case–control study showed a 15-fold risk of disease associated with walking through flood waters (*104*). In Salvador, Brazil, a large epidemic of leptospirosis peaked two weeks after severe flooding in 1996 (*105*). Although leptospirosis cases are rare in the United States, the disease is underdiagnosed (*106*), and the bacteria has been found in samples from both rats and children from surveys conducted in urban areas (*106,107*).

Changes in ecosystems and sociologic factors play a critical role in the occurrence of these diseases. For instance, the increasing numbers of cases and spread of Lyme disease in the United States and Europe stemmed from the reversion of large tracts of agricultural land to woodland and the subsequent increase in mouse, deer, and tick populations combined with the spread of residential areas into undeveloped areas and farmland (*108*).

Most vectorborne diseases exhibit a distinct seasonal pattern which clearly suggests that they are weather sensitive. Rainfall, temperature, and other weather variables affect in many ways both the vectors and the pathogens they transmit. Rainfall may increase the abundance of some mosquitoes by increasing the number of their breeding sites (*109*), but excessive rainfall can flush these habitats and thus destroy the mosquitoes in their aquatic larval stages. Increased humidity can extend vector survival times (*109*). Dry conditions may eliminate the smaller breeding sites, such as ponds and puddles, but create productive new habitats as river flow is diminished. Thus, epidemics of malaria are associated with rainy periods in some parts of the world but with drought in others. High temperatures can increase the rate at which mosquitoes develop into adults, the rate of development of the pathogens in the mosquitoes (*110*), and feeding and egg-laying frequency. The key factor in transmission is the survival rate of the vector (*111*). Higher temperatures may increase or reduce survival rate, depending on the vector, its behavior, ecology, and many other factors. Thus, the probability of transmission may or may not be increased by higher temperatures.

In some cases, specific weather patterns over several seasons appear to be associated with increased transmission rates. For example, in the midwestern United States, outbreaks of St. Louis encephalitis (SLE), a viral infection of birds that can also infect and cause disease in humans, appear to be associated with the sequence of warm wet winters, cold springs, and hot dry summers (*112*). The factors underlying this association remain a matter for speculation (*113,114*).

In the western United States, one study (*115*) predicted that a 3–5°C increase in average temperature may cause a northern shift in the distribution of both Western equine encephalitis (WEE) and SLE outbreaks and a decreased range of WEE in southern California based on temperature sensitivity of both virus and mosquito carrier.

Many other factors are important in transmission dynamics. For example, dengue fever—a viral disease mainly transmitted by *Aedes aegypti*, a mosquito that is closely associated with human habitation—is greatly influenced by house structure, human behavior, and general socioeconomic conditions. There is a marked difference in the incidence of the disease above and below the United States–Mexico border: in the period 1980–1996, 43 cases were recorded in Texas as compared to 50,333 in the three contiguous border states in Mexico (*116*).

The tremendous growth in international travel increases the risk of importation of vectorborne diseases, some of which can be transmitted locally under suitable circumstances at the right time of the year (*99*). Key preventive measures must be directed both at protecting the increasing number of U.S. travelers going to disease-endemic areas, as well as preventing importation of disease by U.S. and non-U.S. citizens. The recent importation of West Nile virus encephalitis into New York illustrates the continued need for vigilance for zoonotic diseases potentially brought in by imported animals or international travelers (*117*). An active survey in Florida (*118*) recently documented underreporting for some diseases, such as dengue fever, further demonstrating the need for improved surveillance to better estimate risk.

Preventive measures from these types of risks include vaccinations and drug prophylaxis for travelers, information for travelers, and the use of repellants and other protective measures. In the United States, medical personnel should be made aware of this increased risk to travelers and of the need to improve surveillance of imported vectorborne diseases.

A high standard of living and well-developed public health infrastructure are central to the current capacity to adapt to changing risks of vector- and rodentborne diseases in the United States. Maintaining and improving this infrastructure—including surveillance, early warning, prevention, and control—remain a priority. Integration of climate, environmental, health, and socioeconomic data may facilitate implementing public health prevention measures. For example, climate forecasts may assist in disease prevention by predicting short-term events such as El Niño, and early warning from improved vector and disease surveillance can help prevent local transmission of imported vectorborne diseases (*119*).

Potential health outcomes not addressed. Other health outcomes identified in the literature and by stakeholders as potentially affected by climate variability and change may warrant future study but are beyond the scope of this current assessment. For example, we did not address the potential impacts on health of economic losses or gains due to climate variability or attempt to assign a monetary value to the health outcomes of climate change. We did not address the potential impact that changes in the hydrologic cycle might have on crop production and food storage in the United States (*120*). Finally, we did not address stratospheric ozone depletion (*121*), although climate change may contribute to the delayed recovery of the stratospheric ozone hole (*122*) and possibly lead to adverse health impacts from increased ultraviolet exposure.

Adaptation/Prevention Strategies

If climate change occurs as projected, it may have significant impacts on virtually all systems on which human life depends—biologic, hydrologic, and ecologic. The extent of the impact that climate change may have on human health is uncertain because it is dependent on multiple interrelated variables as well as on the condition of our public health infrastructure. Climate variability and change will likely have both positive and negative consequences for the health of the U.S. population (Table 1).

The future vulnerability of the U.S. population to the health impacts of climate change depends on our capacity to adapt to any adverse changes through legislative, administrative, institutional, technological, educational, and research-related measures. Examples include building codes and zoning

Table 2. Summary of research needs and knowledge gaps.

Research need	Knowledge gap
Temperature-related morbidity and mortality	Improvement of the early prediction of these events by determining the key weather parameters associated with health Improvement of urban design to facilitate trees, shade, wind and other heat-reducing conditions to limit the urban heat island effect Better personal exposure assessment Heat mortality modeling Understanding of weather relationship to causes of winter mortality
Extreme weather events-related health effects	Improvement of warning systems to provide early, easily understood messages to the populations most likely to be affected Evaluation of the effectiveness of educational materials and early warning systems Long-term health effects from severe events, such as nutritional deficiency and mental health effects Standardization of information collection after disasters to better measure morbidity and mortality Effects of altered land use on vulnerability to extreme weather
Air-pollution-related health effects	Association between weather and pollutants Health impacts of chronic exposure to high levels of ozone Health effects of exposure to ozone in people with asthma and other lung diseases Interaction of ozone with other air pollutants Mechanisms responsible for the adverse effects of ozone and other air pollutants in the general population and within susceptible subgroups Measures that can modulate the impact of air pollution on health, such as nutrition and other lifestyle characteristics Urban weather modeling for inversions, etc.
Water- and foodborne diseases	Links between land use and water quality, through better assessment at the watershed level of the transport and fate of microbial pollutants associated with rain and snowmelt Methods to improve surveillance and prevention of waterborne disease outbreaks Epidemiologic studies Molecular tracing of waterborne pathogens Links between drinking water, recreational exposure, and foodborne disease monitoring Links between marine ecology and toxic algae Vulnerability assessment to improve water and waste water treatment systems
Vector- and rodentborne diseases	Improvement of rapid diagnostic tests for pathogens Vaccines Improvement of active laboratory-based disease surveillance and prevention systems at the state and local level Transmission dynamics (including reservoir host and vector ecology) studies Improvement of surveillance systems for the arthropod vector and vertebrate hosts involved in the pathogen maintenance/transmission cycles to allow for more accurate predictive capability for epidemic/epizootic transmission More effective and rapid electronic exchange of surveillance data

to prevent storm or flood damage, weather-watch/warning systems, improved disease surveillance and prevention programs, fortified sanitation systems, education of health professionals and the public, and research addressing key knowledge gaps in climate/health relationships (Table 2).

Many of these adaptive responses are desirable from a public health perspective irrespective of climate change. For example, reducing air pollution obviously has both short- and long-term benefits to the health of the population. Improving warning systems for extreme weather events and eliminating existing combined sewer and storm water drainage systems are other measures that can ameliorate some of the potential adverse impacts of current climate extremes and of the possible impacts of climate change. Improved disease surveillance and prevention systems at the state and local levels are desperately needed. Of course, adverse effects of adaptive measures are possible (e.g., children playing indoors avoid ozone exposure but may not get sufficient exercise); analysis of the pros and cons of adaptation measures is an important area for future research.

In sum, we found that most of the U.S. population is presently protected against adverse health outcomes associated with weather and/or climate, although certain demographic and geographic populations are at increased risk. Vigilance in the maintenance and improvement of public health systems and their responsiveness to changing climate conditions and to identified vulnerable subpopulations should help to protect the U.S. population from any adverse health outcomes of projected climate change.

References and Notes

1. Diaz HF, Pulwarty RS. An analysis of the time scales of variability in centuries-long ENSO-sensitive records. Clim Change 26:317–342 (1994).
2. Mearns LO. Implications of global warming for climate variability and the occurrence of extreme weather events. In: Drought Assessment Management and Planning: Theory and Case Studies (Wilhite DA, ed). Boston:Kluwer, 1993;109–130.
3. Jones PD, New DM, Parker DE, Martins, Rigor IG. Surface air temperature and its changes over the past 150 years. Rev Geophys 37:173–200 (1999).
4. Karl TR, Knight RW, Plummer N. Trends in high-frequency climate variability in the twentieth century. Nature 377:217–220 (1995).
5. Overpeck JT. Warm climate surprises. Science 271:1820–1821 (1996).
6. Meehl GA, Zwiers F, Evans J, Knutson T, Mearns LO, Whetton P. Trends in extreme weather and climate events: issues related to modeling extremes in projections of future climate change. Bull Am Meteorol Soc (in press).
7. Houghton J. Global Warming: The Complete Briefing. Cambridge, UK:Cambridge University Press, 1997.
8. Easterling DR, Horton B, Jones PD, Peterson TC, Karl TR, Parker DE, Salinger MJ, Razuvaev V, Plummer N, Jamason P, et al. Maximum and minimum temperature trends for the globe. Science 277:364–367 (1997).
9. NRC. Reconciling Observations of Global Temperature Change. Washington, DC:National Academy Press, 2000.
10. Karl TR, Knight RW, Easterling DR, Quayle RG. Indices of climate change for the United States. B Am Meteorol Soc 77:279–303 (1996).
11. Karl TR, Knight RW. Secular trends of precipitation amount, frequency, and intensity in the USA. Bull Am Meteorol Soc 79:231–241 (1998).
12. Fowler AM, Hennessey KJ. Potential impacts of global warming on the frequency and magnitude of heavy precipitation. Nat Hazards 11:283–303 (1995).
13. Mearns LO, Giorgi F, McDaniel L, Shields C. Analysis of daily variability of precipitation in a nested regional climate model: comparison with observations and doubled CO_2 results. Global Planet Change 10:55–78 (1995).
14. Trenberth KE. Conceptual framework for changes of extremes of the hydrologic cycle with climate change. Climatic Change 42:327–339 (1999).
15. National Assessment Synthesis Team. U.S. National Assessment Scenarios and Data. Available: http://www.nace.usgcrp.gov/scenarios [cited 17 February 2000].
16. National Center for Health Statistics. Health, United States, 1998, With Socioeconomic Status and Health Chartbook. PHS 98-1232. Hyattsville, MD:U.S. Department of Health and Human Services, 1998.
17. Pinner RW, Teutsch SM, Simonsen L, Klug LA, Graber JM, Clarke MJ, Berkelman RL. Trends in infectious diseases mortality in the United States. JAMA 275:189–193 (1996).
18. Hobbs FB, Damon BL. 65+ in the United States. P23-190. Washington, DC:U.S. Government Printing Office, 1998.
19. Day JC. Population Projections of the United States by Age, Sex, Race and Hispanic Origin: 1995–2050. U.S. Census Bureau Current Population Reports P25-1130. Washington, DC:U.S. Government Printing Office, 1996.

20. Landrigan PJ, Suk W, Amler RW. Chemical wastes, children's health, and the Superfund basic research program. Environ Health Perspect 107:423–427 (1999).
21. CDC. HIV/AIDS Surveillance Report, December 1998. Atlanta, GA:Centers for Disease Control and Prevention, 1998.
22. Semenza JC, Rubin CH, Falter KH. Heat-related deaths during the July 1995 heat wave in Chicago. N Engl J Med 335:84–90 (1996).
23. Kalkstein LS, Greene JS. An evaluation of climate/mortality relationships in large U.S. cities and the possible impacts of climate change. Environ Health Perspect 105:84–93 (1997).
24. CDC. Heat-related mortality—Chicago, July 1995. MMWR 44:577–579 (1995).
25. Semenza JC, McCullough J, Flanders DW, McGeehin MA, Lumpkin JR. Excess hospital admissions during the 1995 heat wave in Chicago. Am J Prev Med 16:269–277 (1999).
26. Kalkstein LS, Smoyer KE. The impact of climate change on human health: some international implications. Experientia 49:969–979 (1993).
27. Landsberg HE. The Urban Climate. International Geophysics Series, Vol. 28. New York:Academic Press, 1981.
28. Buechley RW, Bruggen JV, Truppi LE. Heat island = death island? Environ Res 5:85–92 (1972).
29. Clarke JF. Some effects of the urban structure on heat mortality. Environ Res 5:93–104 (1972).
30. CDC. Heat-related deaths—United States, 1993. MMWR 42:558–560 (1993).
31. Ramlow JM, Kuller LH. Effects of the summer heat wave of 1988 on daily mortality in Allegheny County, PA. Public Health Rep 105:283–289 (1990).
32. Whitman S, Good G, Donoghue ER, Benbow N, Shou W, Mou S. Mortality in Chicago attributed to the July 1995 heat wave. Am J Public Health 87:1515–1518 (1997).
33. Semenza JC. Are electronic emergency department data predictive of heat-related mortality? J Med Syst 23:419–424 (1999).
34. Applegate WB, Rynyan JW, Brasfield L, Williams ML, Konigsberg C, Fouche C. Analysis of the 1980 heat wave in Memphis. J Am Geriatr Soc 29:337–420 (1981).
35. Schuman SH. Patterns of urban heat-wave deaths and implications for prevention: data from New York and St. Louis during 1966. Environ Res 5:59–75 (1972).
36. Kilbourne EM, Choi K, Jones TS, Thacker SB, Team FI. Risk factors for heat stroke: a case-control study. JAMA 247:3332–3336 (1982).
37. Di Maio DJ, Di Maio VJM. The effects of heat and cold: hyperthermia and hypothermia. In: Forensic Pathology. Boca Raton, FL:CRC Press, 1993;377–388.
38. Marzuk PM, Tardiff K, Leon AC, Hirsch CS, Portera L, Iqbal MI, Nock MK, Hartwell N. Ambient temperature and mortality from unintentional cocaine overdose. JAMA 279:1795–1800 (1998).
39. U.S. Census Bureau. American Housing Survey for the United States in 1995. H-150-95RV. Washington, DC:U.S. Census Bureau, 1997.
40. U.S. Census Bureau. Statistical Abstracts of the United States: 1997. 117th ed. Washington, DC:U.S. Census Bureau, 1997.
41. Spitalnic SJ, Jagminas L, Cox J. An association between snowfall and ED presentation of cardiac arrest. Am J Emerg Med 14:572–573 (1996).
42. Gorjanc ML, Flanders WD, VanDerslice J, Hersh J, Malilay J. Effects of temperature and snowfall on mortality in Pennsylvania. Am J Epidemiol 149:1152–1160 (1999).
43. Glickman TS, Silverman ED. Acts of God and Acts of Man. Washington, DC:Center for Risk Management, Resources for the Future, 1992.
44. NWS. Summary of Natural Hazard Statistics. Available: http://www.nws.noaa.gov/om/hazstats.htm [cited 17 February 2000].
45. NWS. Summary of Natural Hazard Deaths for 1991 in the U.S. Rockville, MD:National Weather Service, 1992.
46. NWS. Hurricane Andrew: South Florida and Louisiana, August 23–26, 1992. Natural Disaster Survey Report. Silver Spring, MD:National Weather Service, 1993.
47. Drabek TE. The Social Dimensions of Disaster. Emmitsburg, MD:Federal Emergency Management Agency, Emergency Management Institute, 1996.
48. Quarantelli EL. An assessment of conflicting views on mental health: the consequences of traumatic events. In: Trauma and Its Wake: The Study and Treatment of Post-Traumatic Stress Disorder, Vol. 4 (Stress BMP, ed). New York:Brunner/Mazel, 1985;173–215.
49. Logue JN, Hansen H, Struening E. Emotional and physical distress following Hurricane Agnes in Wyoming Valley of Pennsylvania. Public Health Rep 94:495–502 (1979).
50. Norris FH, Perilla JL, Riad JK, Kaniasty K, Lavizzo E. Stability and change in stress, resources, and psychological distress following natural disaster: findings from a longitudinal study of Hurricane Andrew. Anxiety Stress Coping 12:363–396 (1999).
51. Noji EK. The nature of disaster: general characteristics and public health effects. In: The Public Health Consequences of Disasters (Noji EK, ed). New York, NY:Oxford University Press, 1997;3–20.
52. Federal Emergency Management Agency. National Mitigation Strategy: Partnerships for Building Safer Communities. Available: http://www.fema.gov/MIT/ntmstrat.htm [cited 17 February 2000].
53. Penner JE, Connell PS, Wuebbles DJ, Covey CC. Climate change and its interactions with air chemistry: perspective and research needs. In: The Potential Effects of Global Climate Change on the United States (Smith JB, Tirpak DA, eds). Washington, DC:U.S. EPA, Office of Policy, Planning and Evaluation, 1989;1-1 to 1-78.
54. Robinson P. The effects of climate change. In: Global Climate Change Linkages: Acid Rain, Air Quality, and Stratospheric Ozone (White JC, ed). New York, NY:Elsevier, 1989.
55. U.S. EPA. National Air Pollutant Emission Trends Update: 1970–1996. EPA 454/R-97-011. Washington, DC:U.S. Environmental Protection Agency, 1997.
56. U.S. EPA. National Air Quality and Emissions Trends Report, 1997. EPA Trends 1998. EPA/454/F-98-016. Washington, DC:U.S. EPA, Office of Air Quality Planning and Standards, 1998.
57. Ahlholm JU, Helander ML, Savolainen J. Genetic and environmental factors affecting the allergenicity of birch (*Betula pubescens* ssp. *czerepanovii* [Orl.] Hamet-ahti) pollen. Clin Exp Allergy 28:1384–1388 (1998).
58. Morris RE, Gery MS, Liu MK, Moore GE, Daly C, Greenfield SM. Sensitivity of a regional oxidant model to variations in climate parameters. In: The Potential Effects of Global Climate Change on the United States (Smith JB, Tirpak DA, eds). Washington, DC:U.S. Environmental Protection Agency Office of Policy, Planning and Evaluation, 1989.
59. NRC. Rethinking the Ozone Problem in Urban and Regional Air Pollution. Washington, DC:National Academy Press, 1991.
60. Sillman S, Samson PJ. Impact of temperature on oxidant photochemistry in urban, polluted rural, and remote environments. J Geophys Res 100:11497–11508 (1995).
61. U.S. EPA. Air Quality Criteria for Ozone and Related Photochemical Oxidant: Volume I of III. EPA/600/P-93/004AF. Washington, DC:U.S. Environmental Protection Agency Office of Research and Development, 1996.
62. Romieu I. Epidemiological studies of the health effects arising from motor vehicle air pollution. In: Urban Traffic Pollution (Schwela D, Zali O, eds). New York:World Health Organization, 1999;10–69.
63. American Thoracic Society. Health effects of outdoor air pollution. Part 2. Am J Res Crit Care Med 153:477–498 (1996).
64. Lambert WE, Samet JM, Dockery DW. Community air pollution. In: Environmental and Occupational Medicine (Rom WN, ed). Philadelphia:Lippincott-Raven, 1998;1501–1522.
65. Davies CJ, Mazurek J. Pollution Control in the United States: Evaluating the System. Washington, DC:Resources for the Future, 1998.
66. Bennett JV, Homberg SD, Rogers MF, Soloman SL. Infectious and parasitic diseases. Am J Prev Med 55:102–114 (1987).
67. Frost FJ, Craun GF, Calderon RL. Waterborne disease surveillance. J Am Water Works Assoc 88:66–75 (1996).
68. ASM. Microbial Pollutants in Our Nation's Water: Environmental and Public Health Issues. Washington, DC:American Society for Microbiology Office of Public Affairs, 1998.
69. Gerba CP, Rose JB, Haas CN. Sensitive populations: who is at the greatest risk? Int J Food Microbiol 30:113–123 (1996).
70. Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C, Griffen PM, Tauxe RV. Food-related illness and death in the United States. Emerg Infect Dis 5:607–625 (1999).
71. Tauxe RV. Emerging foodborne diseases: an evolving public health challenge. Emerg Infect Dis 3:425–434 (1997).
72. Coye MJ, Goldoft M. Microbiological contamination of the ocean, and human health. NJ Med 86:533–538 (1989).
73. Johnston JM, Becker SF, McFarland LM. *Vibrio vulnificus*. Man and the sea. JAMA 253:2850–2853 (1985).
74. Shapiro RL, Altekruse S, Griffin PM. The role of Gulf Coast oysters harvested in warmer months in *Vibrio vulnificus* infections in the United States, 1988–1996. J Infectious Dis 178:752 (1998).
75. Craun GF. Waterborne Disease in the United States. Boca Raton, FL:CRC Press, 1998.
76. Baden DG, Glemming LE, Bean JA. Marine toxins. In: Handbook of Clinical Neurology, Intoxications of the Nervous System: Part II (de Wolf F, ed). Amsterdam:Elsevier Science, 1996;141–75.
77. Hoxie NJ, Davis JP, Vergeront JM, Nashold RD, Blair KA. *Cryptosporidiosis*-associated mortality following a massive waterborne outbreak in Milwaukee, Wisconsin. Am J Public Health 87:2032–2035 (1997).
78. MacKenzie WR, Hoxie NJ, Proctor ME, Gradus S, Blair KA, Peterson DE, Kazmierczak JJ, Addiss DG, Fox KR, Rose JB, et al. A massive outbreak in Milwaukee of *cryptosporidium* infection transmitted through the public water supply. N Engl J Med 331:161–167 (1994).
79. Alterholt TB, LeChevallier MW, Norton WD, Rosen JS. Effect of rainfall on giardia and crypto. J Am Water Works Ass 90:66–80 (1998).
80. Weniger BG, Blaser MJ, Gedrose J, Lippy EC, Juranek DD. An outbreak of waterborne giardiasis associated with heavy water runoff due to warm weather and volcanic ashfall. Am J Public Health 73:868–872 (1983).
81. Motes ML, DePaola A, Cook DW, Veazey JE, Hunsucker JC, Garthright WE, Blodgett RJ, Chirtel SJ. Influence of water temperature and salinity on *Vibrio vulnificus* in Northern Gulf and Atlantic Coast Oysters (*Crassostrea virginica*). Appl Environ Microbiol 64:1459–1465 (1998).
82. Lipp EK, Rose JB, Vincent R, Kurz R, Rodriguez-Palacios C. Assessment of the Microbiological Water Quality in Charlotte Harbor, FL. Technical Report to the Southwest Florida Water Management District, Surface Water Improvement and Management Plan. Tampa, FL:Southwest Florida Water Management District, 1999.
83. Harvell CD, Kim K, Burkholder JM, Colwell RR, Epstein PR, Grimes DJ, Hofmann EE, Lipp EK, Osterhaus AD, Overstreet RM, et al. Emerging marine diseases—climate links and anthropogenic factors. Science 285:1505–1510 (1999).
84. Lipp EK, Rose JB. The role of seafood in foodborne diseases in the United States of America. Rev Sci Tech (Off Int Epizoot) 16:620–640 (1997).
85. Valiela I. Marine Ecological Processes. New York:Springer Verlag, 1984.
86. CDC. Outbreak of *Escherichia coli* O157:H7 and Campylobacter among attendees of the Washington County Fair—New York, 1999. MMWR 48:803 (1999).
87. Yates ML, Yates SR. Modeling microbial fate in the subsurface environment. Crit Rev Environ Control 17:307–344 (1988).
88. Rose JB, Simonds J. King County Water Quality Assessment: Assessment of Public Health Impacts Associated with Pathogens and Combined Sewer Overflows. Seattle, WA:King County Department of Natural Resources, 1998.
89. U.S. EPA. Water on Tap: A Consumer's Guide to the Nation's Drinking Water. EPA-815-K-97-002. Washington, DC:U.S. Environmental Protection Agency Office of Water, 1997
90. U.S. EPA. National primary drinking water regulations: interim enhanced surface water treatment; final rule. Fed Reg 63:241 (1998).
91. U.S. EPA. Action Plan for Beaches and Recreational Waters. EPA/600/R-98/079. Washington, DC:U.S. Environmental Protection Agency, Office of Research and Development, Office of Water, 1999.
92. CDC. PulseNet: The National Molecular Subtyping Network for Foodborne Disease Surveillance. Available: http://www.cdc.gov/ncidod/dbmd/pulsenet/pulsenet.htm [cited 18 February 1999].
93. CDC. FoodNet: CDC/FSIS/FDA Foodborne Diseases Active Surveillance Network, CDC's Emerging Infections Program. 1998 Surveillance Results, Preliminary Report.

Available: http://www.cdc.gov/ncidod/dbmd/foodnet/98surv.htm [cited 18 August 1999].

94. Gower JFR. Detection and mapping of bright plankton blooms and river plumes using AVHRR imagery. In: Third Thematic Conference on Remote Sensing for Marine and Coastal Environments, 18–20 September 1995, Seattle, Washington. Ann Arbor, MI:Environmental Research Institute, 1995.
95. Philip CB, Rozeboom LE. Medico-veterinary entomology: a generation of progress. In: History of Entomology (Smith RF, Mittler TE, Smith CN, eds). Palo Alto, CA:Annual Reviews, Inc., 1973.
96. Beneson AS. Control of Communicable Diseases Manual, 16th ed. Washington, DC:American Public Health Association, 1995.
97. Reiter P. Global warming and mosquito-borne disease in USA. Lancet 348:622 (1996).
98. Reiter P. From Shakespeare to Defoe: malaria in England in the Little Ice Age. Emerg Infect Dis 6:1–11 (2000).
99. Gubler DJ. Resurgent vector-borne diseases as a global health problem. Emerg Infect Dis 4:442–450 (1998).
100. Parmenter RR, Yadav EP, Parmenter CA, Ettestad P, Gage KL. Incidence of plague associated with increased winter-spring precipitation in New Mexico. Am J Trop Med Hyg 62:814–821 (2000).
101. Schmaljohn C, Hjelle B. Hantaviruses: a global disease problem. Emerg Infect Dis 3:95–104 (1997).
102. Centers for Disease Control and Prevention. Unpublished data.
103. Engelthaler DM, Mosley DG, Cheek JE, Levy CE, Komatsu KK, Ettestad P, Davis T, Tanda DT, Miller L, Frampton JW, et al. Climatic and environmental patterns associated with hantavirus pulmonary syndrome, Four Corners region, United States. Emerg Infect Dis 5:87–94 (1999).
104. Trevejo RT, Rigau PRJG, Ashford DA, McClure EM, Jarquan-Gonzalez C, Amador JJ, de los Reyes JO, Gonzalez A, Zaki SR, Shieh WJ, et al. Epidemic leptospirosis associated with pulmonary hemorrhage—Nicaragua, 1995. J Infect Dis 178:1457–1463 (1998).
105. Ko AI, Galvao Reis M, Ribeiro Dourado CM, Johnson WD, Jr, Riley LW. Urban epidemic of severe leptospirosis in Brazil. Salvador Leptospirosis Study Group. Lancet 354:820–825 (1999).
106. Demers RY, Thiermann A, Demers P, Frank R. Exposure to *Leptospira icterohaemorrhagiae* in inner-city and suburban children: a serologic comparison. J Fam Pract 17:1007–1011 (1983).
107. Childs JE, Schwartz BS, Ksiazek TG, Graham RR, LeDuc JW, Glass GE. Risk factors associated with antibodies to leptospires in inner-city residents of Baltimore: a protective role for cats. Am J Public Health 82:597–599 (1992).
108. IOM. Emerging infections: microbial threats to health in the United States. 92-26480. Washington, DC:Institute of Medicine, 1992.
109. Reisen WK, Lothrop HD, Hardy JL. Bionomics of *Culex tarsalis* (Diptera: Culicidae) in relation to arbovirus transmission in southeastern California. J Med Entomol 32:316–327 (1995).
110. Watts DM, Burke DS, Harrison BA, Whitmire RE, Nisalak A. Effect of temperature on the vector efficiency of *Aedes aegypti* for Dengue 2 virus. Am J Trop Med Hyg 36:143–152 (1987).
111. Gilles HM. Epidemiology of Malaria. In: Bruce-Chwatt's Essential Malariology (Gilles HM, Warrell DA, eds). London:Edward Arnold Division of Hodder & Stoughton, 1993;124–163.
112. Monath TP. Epidemiology. In: St. Louis Encephalitis (Monath TP, ed). Washington, DC:American Public Health Association, 1980;239–312.
113. Reeves WC, Hammon WM. Epidemiology of the arthropod-borne viral encephalitides in Kern County, California, 1943–1952. Univ Calif Berk Public Health 4:1–257 (1962).
114. Reiter P. Weather, vector biology, and arboviral recrudescence. In: The Arboviruses: Epidemiology and Ecology (Monath TP, ed). Boca Raton, FL:CRC Press, 1988;245–255.
115. Reeves WC, Hardy JL, Reisen WK, Milby MM. Potential effect of global warming on mosquito-borne arboviruses. J Med Entomol 31:323–332 (1994).
116. Reiter P. Global climate change and mosquito-borne disease. In: Encyclopedia of Human Ecology (Watt K, ed). London, UK:Academic Press, 1999.
117. Lanciotti RS, Roehrig JT, Deubel V, Smith J, Parker M, Steele K, Crise B, Volpe KE, Crabtree MB, Scherret JH, et al. Origin of the West Nile virus responsible for an outbreak of encephalitis in the northeastern United States. Science 286:2333–2337 (1999).
118. Gill J, Stark LM, Clark GC. Dengue surveillance in Florida, 1997–98. Emerg Infect Dis 6:30–35 (2000).
119. Colwell RR, Patz JA. Climate, Infectious Disease and Health: An Interdisciplinary Perspective. Washington, DC:American Academy of Microbiology, 1998.
120. McMichael AJ, Haines A, Sloof R, Kovats S. Climate Change and Human Health. Geneva:World Health Organization, 1996.
121. McKenzie R, Connor B, Bodeker G. Increased summertime UV radiation in New Zealand in response to ozone loss. Science 285:1709–1711 (1999).
122. Shindell DT, Rind D, Lonergan P. Increased polar stratospheric ozone losses and delayed eventual recovery owing to increasing greenhouse-gas concentrations. Nature 392:589–592 (1998).