RFF REPORT

Adapting to Climate Change

Public Health

Jonathan M. Samet

ADAPTATION | AN INITIATIVE OF THE CLIMATE POLICY PROGRAM AT RFF

JUNE 2009



Table of Contents

Summary	1
Introduction	2
1. Overview	2
2. What is Public Health?	2
3. Monitoring Public Health	4
Impacts	7
1. Heat Stress and Heat Waves	7
2. Aeroallergens and Allergic Diseases	11
3. Changes in Endemic and Epidemic Infectious Diseases	14
4. Ambient Air Pollution	18
Specific Adaptations	21
1. Heat	21
2. Aeroallergens and Allergic Diseases	23
3. Changes in Endemic and Epidemic Infectious Diseases	23
4. Ambient Air Pollution	24
Context	25
Conclusions	27
Deferences	20

Adapting to Climate Change: Public Health

Jonathan M. Samet*

Summary

The potential consequences of climate change extend to the health of the public, with warming of the planet projected to have both positive and negative consequences that will vary temporally and spatially. Climate change will not act to introduce new causes of morbidity and mortality, but to change the distributions of factors that affect the occurrence of morbidity and mortality. The time frames over which health consequences of climate change are anticipated to be manifest are both immediate and longer term and, consequently, adaptation measures could potentially reduce their impact. This paper addresses the projected health consequences of climate change, reviewing the projected adverse effects, the diverse strategies that might mitigate these effects, and the potential effectiveness of these strategies. It addresses temperature, aeroallergens and allergic diseases, air pollution, and infectious diseases.

The methods for addressing the health consequences of climate change, as evident in this review, are those of public health and disease control generally. The unique aspect of climate change is its upstream driver. The consequences of climate change for health range from being quite specific (e.g., heat waves) to general (e.g., increased exposure to air pollution) and from being acute in nature (e.g., infectious disease outbreaks) to longer term (e.g., changes in allergic diseases associated with shifts in aeroallergens). For some of the health consequences of climate change, such as emerging infections and heat waves, adaptation will take place through the routine functioning of effective public health systems, if in place. Some, such as allergic diseases, will be managed through routine medical care. And some, including increased emissions of air pollution, will be addressed through regulatory mechanisms.

Recognition and quantification of the health consequences of climate change will be difficult, given their lack of specificity. Risk assessment methods, including burden of disease estimation, will remain central as a tool for estimating the need for implementation of adaptive strategies and for quantifying their benefits.

 $\stackrel{\sim}{=}$

^{*} Jonathan M. Samet, M.D., M.S. Professor and Chair, Department of Preventive Medicine and Director, USC Institute for Global Health, Keck School of Medicine, USC/Norris Comprehensive Cancer Center. 1441 Eastlake Avenue, Room 4436, MS 44, Los Angeles, CA 90033. Telephone: 323-865-0803. Fax: 323-865-0127 jsamet@usc.edu. This report was prepared for the Resources for the Future project on adaptation to climate change. For more information, see www.rff.org/adaptation.

Introduction

1. Overview

The potential consequences of climate change extend to the health of the public, with warming of the planet projected to have both positive and negative consequences that will vary temporally and spatially. Climate change will not act to introduce new causes of morbidity and mortality, but to change the distributions of factors that affect the occurrence of morbidity and mortality. The time frames over which health consequences of climate change are anticipated to be manifest are sufficiently slow to allow adaptive measures to come into play that may modulate the occurrence of these effects.

This paper addresses the projected health consequences of climate change, reviewing the projected adverse effects, the diverse strategies that might mitigate these effects, and the potential effectiveness of these strategies. The review is qualitative and does not itself quantify the potential health burden of these effects; such assessments have been reported and are reviewed. The literature on climate change and health covered in this review was identified through searches of the biomedical literature using PubMed. Additionally, references included in the 2007 report of the Intergovernmental Panel on Climate Change (IPCC; Metz et al. 2007), the 2006 report of the Climate Change and Adaptation Strategies for Human Health (Menne and Ebi 2006), and recent review articles on climate change and health (Haines and Patz 2004; Patz et al. 2005; Haines et al. 2006; McMichael et al. 2006; Frumkin et al. 2008) are reviewed.

This review begins with a discussion of the concepts and methods of public health, covering the general strategies used to identify and manage threats to the health of the public and to prospectively implement programs to improve the public's health. Ongoing data collection to monitor health status and disease occurrence is fundamental to these strategies, as is evaluation of any interventions that are implemented. It draws on standard sources in public health (Teutsch and Churchill 2000; Detels et al. 2004; Wallace 2008) and covers major health consequences of climate change, including mortality associated with hot and cold temperatures, allergic diseases, air pollution, and infectious diseases. It does not address hurricanes and other climate-driven natural disasters. Models support the potential for climate change to increase the frequency and severity of such disasters, which may have dramatic public health consequences and necessitate responses at the national and global levels (Metz et al. 2007). However, the potential consequences of such disasters and the need for response mechanisms are already well documented.

2. What is Public Health?

Health, as defined by the World Health Organization (1948, 100), is "...a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity." The definition is notable for its emphasis on well-being. Public health refers to the approaches taken to protect and improve the health of *communities*, in contrast to clinical medicine, which addresses the health and disease of *individuals*. Prevention is fundamental to public health; primary prevention involves the control of the causes of disease, whereas secondary prevention involves detection of early cases of disease through screening and treatment at a stage at which cure is likely. Tertiary

prevention is the domain of clinical medicine—treating patients with clinically manifest disease. Frumkin and colleagues (2008, 435) have applied these disease control concepts to public health, proposing that mitigation is analogous to primary prevention and that adaptation is comparable to secondary and tertiary prevention, as it involves "...efforts to anticipate and prepare for the effects of climate change, and thereby to reduce the associated health burden."

The essential services of public health cover broad domains that are relevant to adaptation to climate change (Table 1; American Public Health Association 2008). Frumkin and colleagues (2008) match these functions to the anticipated consequences of climate change. These domains include monitoring the health of the population and investigating health problems that occur in communities. Communications and engagement are also essential, as is the maintenance of the infrastructure and capacity for sustaining the core public health functions. Public health, particularly in comparison to clinical care, has long been underfunded, and public health experts in the United States have repeatedly voiced concern as to the size and competence of the public health work force (Institute of Medicine and Committee on Assuring the Health of the Public in the 21st Century 2002; Institute of Medicine et al. 2003). Health problems that can be addressed through primary prevention remain as major, but remediable causes of morbidity and mortality and include, for example, tobacco use, obesity and physical inactivity, sexually transmitted diseases, and alcoholism and drug abuse (Mokdad et al. 2004). Globally, the status of public health is highly variable, ranging from completely lacking in some less developed countries to being highly effective in others (Beaglehole and Dal Poz 2003).

Table 1. Essential Services of Public Health

1.	Monitor	health status to identify community health problems
2.	Diagnose and investigate	health problems and health hazards in the community
3.	Inform, educate, and empower	people about health issues
4.	Mobilize	community partnerships to identify and solve health problems
5.	Develop policies and plans	that support individual and community health efforts
6.	Enforce	laws and regulations that protect health and ensure safety
7.	Link	people to needed personal health services and ensure the provision of health care when otherwise unavailable
8.	Ensure	a competent public health and personal healthcare workforce
9.	Evaluate	the effectiveness, accessibility, and quality of personal and population-based health services
10.	Research	for new insights and innovative solutions to health problems

Source: American Public Health Association n.d.

3. Monitoring Public Health

The health of a population can be gauged by a number of diverse indicators (Table 2; Etches et al. 2006). The most basic is the overall mortality rate and the complementary projection of life expectancy. In most of the more developed countries, cause-specific mortality is also tracked. Other key mortality indicators relate to pregnancy and the outcome of pregnancy. With regard to incidence—that is, new cases of disease—the occurrence of some infectious diseases is tracked through a variety of active and passive symptoms; of the chronic diseases (i.e., those with a lengthy course), incident cases of cancer are tracked in the United States and some other countries, but other major chronic diseases, such as coronary heart disease, are generally not.

Surveillance refers to the tracking of the health of a population, whether in general or for particular indicators. The concept of surveillance was formalized by Langmuir (1963, 182), who offered the following definition in a now-classic 1963 paper in the *New England Journal of Medicine*:

Surveillance, when applied to a disease, means the continued watchfulness over the distribution and trends of incidence through the systematic collection, consolidation and evaluation of morbidity and mortality reports and other relevant data.

Surveillance involves more than passive collection of data; it is grounded in process, such that the incoming data are analyzed and the findings reviewed and action is taken when needed (Figure 1; Teutsch and Churchill 2000). If intervention is undertaken, the continued monitoring provides a way to track its consequences. Although surveillance is central to tracking the occurrence of infectious illnesses, such as influenza and other respiratory pathogens, the same concepts are also applied to diseases that occur over far longer time frames, such as cancer and coronary heart disease.

Table 2. Selected Indicators of Population Health

Mortality	Total mortality rate
	Cause-specific mortality rates
	Maternal mortality rate
	Perinatal mortality rate
	Infant mortality rate
	Lifespan
Incidence	Infectious diseases
	Cancer
Prevalence	Chronic diseases
	Malnutrition
	Overweight and obesity
	Disease risk factors
	Vaccination
	Health care coverage

Source: Etches et al. 2006.

In the United States, the Centers for Disease Control and Prevention (CDC) has a broad set of surveillance activities in place, including many that are housed in the National Center for Health Statistics (CDC n.d. [a]; CDC n.d. [b]). The resulting extensive databases of spatial and temporal data provide a major resource for planning potential surveillance activities related to climate change in the United States. Additional databases are maintained at the regional, state, and local levels. Analytical tools have also been developed that facilitate the scanning of these data for patterns indicative of potential consequences of climate change or other factors.

One further type of tracking involves the periodic estimation of the burden of avoidable morbidity and mortality. This type of estimation has been carried out at the national level in some countries and at the global level through the Global Burden of Disease project, initially coordinated by the World Health Organization (n.d.). The estimation uses the concept of population attributable

risk to estimate disease burden and uses, as the comparison for a particular risk factor, the expected amount of disease absent the exposure (Levin 1953). The burden depends on the prevalence of exposure to the factor of interest and the risk associated with exposure; higher prevalence and greater risk increase the estimated burden of disease. For cigarette smoking, for example, the population attributable risk for lung cancer in the United States exceeds 80 percent, implying that, absent smoking, these cases would not have occurred (U.S. Department of Health and Human Services 2004). Burden estimates also address the combination of life lost and the extent of useful life lost through the calculation of disability-adjusted life years (DALYs) lost.

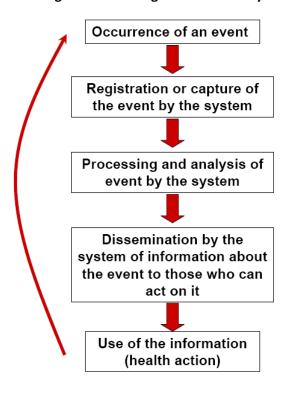


Figure 1. Modeling a Surveillance System

Source: Teutsch and Churchill 2000.

This approach of burden estimation has been extended to climate change (McMichael et al. 2004). Although they are inherently subject to great uncertainty, burden of disease estimates provide an indication of the magnitude of anticipated impact and a way to compare the future burden under various scenarios of mitigation and adaptation. The McMichael et al. (2004) report on the burden of mortality and morbidity related to climate change includes estimates of the attributable burden (in the past) and of the projected burden (for the future). The particular difficulties of burden estimation in regard to climate change have been a topic of several commentaries (Kovats et al. 2005; Campbell-Lendrum and Woodruff 2006).

Impacts

1. Heat Stress and Heat Waves

Heat Waves and Their Consequences

Temperature has long been associated with adverse effects on health and mortality. At the extremes of temperature exposure, the well-known clinical entities of hypothermia and hyperthermia are well-documented causes of death (Basu and Samet 2002). Hypothermia typically affects persons at risk for unprotected exposure to cold because of socioeconomic status and limited resources for space heating. Hyperthermia occurs among persons carrying out physical activities when temperatures are high that lead to thermal stress as well as those who are susceptible to heat because of limited adaptive capacity, such as the elderly and persons taking certain medications that impair responses to thermal stress. Even in more developed countries, deaths occur that are attributable to hypothermia and hyperthermia. In the United States, for example, approximately 600 deaths from hypothermia (CDC 2004) and slightly fewer than 700 deaths from hyperthermia (CDC 2006) occur each year.

The phenomenon of excess mortality during heat waves has been extensively documented and is well recognized as a potential consequence of global warming arising from climate change (IPCC 2007). In recent decades, the dramatic epidemics of death associated with heat waves in Chicago in 1995 (Semenza et al. 1996) and in Europe in 2003 (Vandentorren et al. 2004; Kovats et al. 2006) have alerted the public to the dangers of heat waves and led to protective actions by governments and public health agencies. Moreover, warmer temperatures are associated with mortality even at times when heat waves are not in progress (Basu and Samet 2002; Kovats et al. 2006). The relationship between temperature and mortality has been characterized as "J-shaped" (Figure 2), such that mortality increases with both colder and warmer temperatures from some temperate optimum at which it is lowest (Curriero et al. 2002; McMichael et al. 2008). The value of this optimum temperature varies with average temperature, and hence latitude, as well as the extent to which adaptive measures are available for acclimating to warmer or colder temperatures (The Eurowinter Group 1997; Curriero et al. 2002; Kovats et al. 2006).

This J-shaped relationship has implications for the potential overall effect of global warming consequent to climate change on heat-associated mortality. Warming would reduce the cold-associated mortality while increasing heat-associated mortality, absent new adaptive measures (Figure 2; Curriero et al. 2002). However, beyond the rise in average temperature, climate change is also projected to increase the variability of temperature and the frequency of heat waves (IPCC 2007).

Heat is already associated with ongoing mortality. Temperatures above the optimum value can be assumed to contribute to mortality. Estimates for Germany, for example, ranged from 5 to 10 percent in excess beyond the optimum for temperatures that were below the extreme (Kovats et al. 2006). Clear excesses of thousands of deaths during heat waves have been well documented (Basu and Samet 2002; Kovats et al. 2006; IPCC 2007).

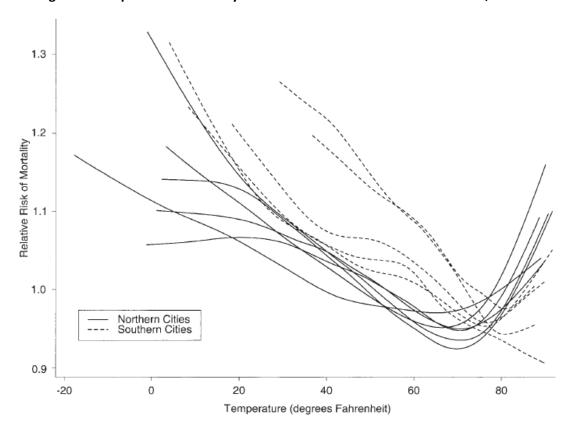


Figure 2. Temperature-Mortality Relative Risk Functions for 11 U.S. Cities, 1973-1994

Notes: Northern cities: Boston, Massachusetts; Chicago, Illinois; New York, New York; Philadelphia, Pennsylvania; Baltimore, Maryland; and Washington, DC. Southern cities: Charlotte, North Carolina; Atlanta, Georgia; Jacksonville, Florida; Tampa, Florida; and Miami, Florida. $^{\circ}$ C = 5/9 x ($^{\circ}$ F – 32). *Source:* Curriero et al. 2002.

The recent heat waves in Chicago and Europe are particularly informative for indicating the vulnerabilities that contributed to remarkably high mortality during the episodes and for identifying the subgroups within the population that are particularly susceptible. In Chicago, 465 deaths were certified as heat-related over the period July 11–27, 1995 (CDC 1995). A case–control study found strong associations between increased mortality and the presence of medical problems and social isolation, whereas having an air conditioner and transportation were associated with reduced mortality (Semenza et al. 1996). Mortality was also higher in neighborhoods that were less socially cohesive (Klinenberg 2002).

The 2003 heat wave in Europe was dramatic for its scope and for the failure to recognize the resulting mortality and to take action in a sufficiently timely way. Numerous analyses have been reported on the mortality caused by the heat wave (see Kovats et al. 2006; IPCC 2007). France experienced approximately 15,000 excess deaths, and the total for Europe was estimated at 35,000 (IPCC 2007). As was the case in Chicago, the at-risk groups included the elderly, those living alone and without social support, and the less advantaged (Poumadere et al. 2005). Inquiries in France

identified failures that led to the tragic excess: inadequate surveillance, limited public health capacity, and insufficient communication (Poumadere et al. 2005; IPCC 2007).

Evidence also suggests that the risk of heat-related excess mortality has declined on longer time frames. Carson and colleagues (2006) examined weekly mortality in London during the 20th century and assessed temperature-associated mortality over a period during which a major shift occurred in the underlying causes of death and a progressive increase in the age of the population and the proportion affected by chronic diseases. They found declines in susceptibility to death from both cold and heat. They attributed this finding to a variety of factors related to social and environmental conditions, behavior, and health care. Davis et al. (2003) examined heat-related mortality over the period 1964–1998 in the United States; they also found declining heat mortality, which they attributed to a variety of adaptations, including the increased availability of air conditioning. Barnett (2007) found that the association of warm temperature with cardiovascular mortality during the summer declined substantially over the period 1987–2004.

Because the elderly and people with underlying chronic diseases are particularly susceptible, the hypothesis has been advanced that the excess mortality associated with heat waves represents only a brief advancement of the time of dying, a phenomenon sometimes referred to as *mortality displacement* or *harvesting*. This same hypothesis has been advanced in interpreting the associations found between daily mortality counts and air pollution concentrations on the same or recent days. If such mortality displacement is prominent, a reduction in mortality would be anticipated following the excess associated with the heat wave; analytical approaches have been developed for assessing mortality displacement (Zeger et al. 1999; Zanobetti et al. 2000). For the 2003 heat wave in France, the extent of mortality displacement was found to be modest (Toulemon and Barbieri 2008). A parallel analysis of heat-related deaths in Delhi, São Paulo, and London, using distributed lag models, found evidence of mortality displacement in London and a lesser indication of this phenomenon in Delhi (Hajat et al. 2005); the pattern was intermediate for São Paulo.

Evidence is noticeably lacking on temperature-associated mortality in the developing countries. The ISOTHURM project examined the temperature–mortality relationship in 12 urban areas, including several in low- and middle-income countries (McMichael et al. 2008). The data from most of the cities showed a J-shaped relationship with temperature. In Delhi and Salvador, mortality did not increase at colder temperatures, nor did an increase occur at hotter temperatures in Chiang Mai and Cape Town.

Determinants of Severity

The impact of a heat wave varies with the magnitude of the thermal stress, the duration of the episode, and the characteristics of the population affected. In general, models of the relationship between temperature (or other indicators) and mortality show increasing mortality with increasing temperature (for example, Curriero et al. 2002). Based on analyses of data from London, Budapest, and Milan, Hajat et al. (2006) found a "heat wave effect," such that mortality from a sustained temperature elevation exceeds that predicted by the rise in temperature alone.

Population characteristics also determine the impact of heat waves. The elderly and persons with underlying chronic diseases, such as coronary artery disease and congestive heart failure, are

particularly at risk. Additionally, persons taking diuretics, certain agents used for blood pressure control, and other drugs may have impaired cardiovascular responses to thermal stress, as may obese persons. In the aging populations of the more developed countries, these at-risk groups are increasing in size and likely to continue to do so. Epidemiological analyses, described above, have identified additional risk factors for mortality during heat waves, including lack of social support, socioeconomic status, and housing characteristics. In urban areas, the "urban heat island" phenomenon tends to increase the risk of mortality associated with heat waves (U.S. Environmental Protection Agency, n.d. [b]; Buechley et al. 1972). Determinants of risk in less developed countries have received little research attention.

Spatial and Temporal Distribution

Analyses included in the IPCC (2007) report make clear that increases in mean temperature will be widespread and that variability will also increase, leading to the potential for more frequent and severe heat waves. Most regions of the world will probably be affected. Although time trends of heat wave–associated mortality are not clearly apparent, recent dramatic episodes, including Chicago in 1995 and Europe in 2003, document that heat waves continue to have unabated impact.

Synthesis and Summary

Excess mortality during heat waves has long been documented. The present potential for heat waves to cause substantial morbidity and mortality in cities, even in developed countries, has been established by several dramatic events. The IPCC's (2007) projections of rising temperatures and increasing variability support the conclusion that there is a high probability of future climate change–caused heat waves with excess mortality. The burden of climate change–attributable cardiovascular disease mortality has been estimated for the various regions of the World Health Organization and summarized for the world (Tables 3 and 4; McMichael et al. 2004). For temperature variation (both hotter and colder temperatures) associated with climate change, an estimated 12,000 cardiovascular disease deaths were advanced by climate change for the year 2000. This number, a global estimate, is much smaller than the actual numbers of excess deaths during well-documented heat waves; the estimate, however, refers to the burden of temperature-associated mortality from the effect of climate change on temperature and not to the consequences of temperature itself.

Table 3. Estimated Mortality (000s) Attributable to Climate Change in the Year 2000, by Cause and Subregion

Subregion	Malnutrition	Diarrhea	Malaria	Floods	CVD	All causes	Total
							deaths/million
							population
AFR-D	8	5	5	0	1	19	66.83
AFR-E	9	8	18	0	1	36	109.40
AMR-A	0	0	0	0	0	0	0.15
AMR-B	0	0	0	1	1	2	3.74
AMR-D	0	1	0	0	0	1	10.28
EMR-B	0	0	0	0	0	1	5.65
EMR-D	9	8	3	1	1	21	61.30
EUR-A	0	0	0	0	0	0	0.07
EUR-B	0	0	0	0	0	0	1.04
EUR-C	0	0	0	0	0	0	0.29
SEAR-B	0	1	0	0	1	2	7.91
SEAR-D	52	22	0	0	7	80	65.79
WPR-A	0	0	0	0	0	0	0.09
WPR-B	0	2	1	0	0	3	2.16
World	77	47	27	2	12	166	27.82

Notes: CVD, cardiovascular disease; AFR, African region; AMR, Region of the Americas; EMR, Eastern Mediterranean region; EUR, European region; SEAR, South-East Asian region; WPR, Western Pacific region. Source: McMichael et al. 2004.

Table 4. Estimated Disease Burden (000s of DALYs) Attributable to Climate Change in the Year 2000, by Cause and Subregion

Subregion						Total
	Malnutrition	Diarrhea	Malaria	Floods	All causes	DALYs/million
						population
AFR-D	293	154	178	1	626	2185.78
AFR-E	323	260	682	3	1267	3839.58
AMR-A	0	0	0	4	4	11.85
AMR-B	0	0	3	67	71	166.62
AMR-D	0	17	0	5	23	324.15
EMR-B	0	14	0	6	20	147.57
EMR-D	313	277	112	46	748	2145.91
EUR-A	0	0	0	3	3	6.66
EUR-B	0	6	0	4	10	48.13
EUR-C	0	3	0	1	4	14.93
SEAR-B	0	28	0	6	34	117.19
SEAR-D	1918	612	0	8	2538	2080.84
WPR-A	0	0	0	1	1	8.69
WPR-B	0	89	43	37	169	111.36
World	2846	1459	1018	193	5517	925.35

Notes: DALY, disability-adjusted life year; CVD, cardiovascular disease; AFR, African region; AMR, Region of the Americas; EMR, Eastern Mediterranean region; EUR, European region; SEAR, South-East Asian region; WPR, Western Pacific region.

Source: McMichael et al. 2004.

2. Aeroallergens and Allergic Diseases

Aeroallergens, Allergic Diseases, and Their Consequences

Aeroallergens—biological agents associated with allergic responses—are ubiquitous in indoor and outdoor environments. Contact of these agents with the mucosal surfaces of the eyes and nose causes allergic responses, as does inhalation into the lung. The two principal diseases associated with aeroallergens are allergic rhinitis, also referred to as hay fever, and asthma. These are prevalent diseases, affecting substantial proportions of children and adults (Avila-Tang et al. 2008). Both diseases are presumed to have a genetic basis as familial aggregation is well documented. In spite of several decades of investigation, however, only modest progress has been made in identifying the genes associated with allergic rhinitis, asthma, and allergy.

The frequency of allergic rhinitis and asthma is tracked with periodic surveys using questionnaires and other approaches. Such surveys have documented a remarkable and unexplained rise of asthma and other allergic disorders in children. Prevalence estimates range up to 20 percent for asthma, which tends to be more frequent in developed countries (Avila-Tang et al. 2008). Multiple hypotheses have been offered with regard to the rise in childhood asthma, but uncertainty remains as to the basis for the increase.

The development of asthma is broadly considered to be a consequence of gene-by-environment interaction; that is, environmental exposures trigger the onset of disease in persons who are genetically at risk. Aeroallergens may have a role in this triggering; exposure to the house dust mite, for example, has been associated with earlier onset of wheezing in young children (Sporik et al. 1990). Aeroallergens are not considered to be a sufficient cause of asthma onset, absent underlying genetic susceptibility.

Aeroallergens are well established as an exposure that can exacerbate asthma and trigger attacks of allergic rhinitis. Numerous aeroallergens are found in outdoor air, particularly pollens that can trigger allergic diseases. In the United States, pollen counts are routinely monitored outdoors by the National Allergy Bureau (http://www.aaaai.org/nab), and the monitoring data are communicated to the public. The levels of pollen in the air display strong seasonal patterns, with peaks in the spring and fall. Indoor sources of aeroallergens include dogs and cats, rodents, and house dust mites.

Determinants of Severity

Fortunately, allergic rhinitis and asthma are diseases that can be effectively managed in most affected persons. A variety of medical management approaches are directed at controlling symptoms and reducing the likelihood of exacerbations (Pearce et al. 1998; National Heart Lung and Blood Institute and National Asthma Education and Prevention Program 2007; Avila-Tang et al. 2008). The phenotypic severity of these diseases, particularly of asthma, is highly variable, and this variation probably has both environmental and genetic bases. In addition to medications, asthma severity may be lessened through environmental management strategies that reduce exposure to indoor aeroallergens, tobacco smoke, and other types of indoor air pollution, and also by avoiding pollutants in outdoor air by staying indoors.

For persons with allergic rhinitis and asthma, climate change might increase the risk of exacerbation through altered local and regional pollen production. Warming has already caused an earlier onset of the spring pollen season in the Northern Hemisphere (IPCC 2007). It may also increase the duration of the pollen season, change the spatial distribution of vegetation, and possibly alter pollen production (Beggs 2004; Beggs and Bambrick 2005; IPCC 2007). More prolonged and intense exposure to aeroallergens could result in more severe disease and possibly greater morbidity, and even mortality, from asthma. Beggs and Bambrick (2005) have proposed that climate change could be contributing to the global rise in asthma as a consequence of greater pollen exposure.

Spatial and Temporal Distribution

Evidence suggests that climate change has already affected exposures of populations to aeroallergens (IPCC 2007; Shea et al. 2008). Vegetation patterns have changed, and pollination is occurring earlier for some species in some places. New species could potentially become successful in additional areas, leading to exposures of populations to new antigens.

Synthesis and Summary

Evidence already suggests that patterns of exposure to aeroallergens have been altered by climate change. Many people throughout the world have allergic rhinitis and asthma, diseases that make them sensitive to aeroallergens. Lengthened periods of exposure and higher concentrations are very likely to increase the frequency and severity of exacerbations. An increase in the incidence of allergic diseases as a result of increased aeroallergen exposure is less likely.

3. Changes in Endemic and Epidemic Infectious Diseases

Epidemiological Aspects of Infectious Diseases

Worldwide, in both developed and developing countries, infectious agents remain a leading cause of disease and death (Nelson et al. 2007). The numerous known infectious diseases differ in their causative organisms, pathways of transmission, clinical manifestations, responses to therapy, and outcomes. Vector-borne diseases are of greatest concern with regard to potential adverse consequences of climate change. The transmission of these diseases is conceptually described by the "epidemiological triangle" (Figure 3), which captures the interplay between the agent, the environment, and the vector. Environmental conditions that promote or extend the geographic range of the vector increase the potential for infection by the agent. In addition to potentially affecting vector-borne diseases, climate change may also extend or change the geographic regions in which an infectious agent is present. Climate change may affect both endemic disease (i.e., disease occurring in a population), and epidemic disease (i.e., disease occurring in excess of the usual background). Epidemiological aspects of major infectious diseases were recently summarized by Nelson et al. (2007).

Mathematical models of infectious disease transmission provide quantitative insight into the potential for climate change to increase rates of vector-borne diseases. The transmission of infectious diseases has been characterized by the basic reproductive rate (R_0) which describes the number of new cases of infection arising from one case in a population of susceptible persons (Rogers and Randolph 2006). Values above unity imply the possibility of epidemic disease; a value of unity means that endemic disease will be maintained, and a value below unity means that the disease will decline. Warming can increase R_0 through its effect on vector numbers, transmission probabilities, and biting rates (Rogers and Randolph 2006). The geographic spread of vectors may also be affected by the extension of their ranges resulting from warmer conditions. A number of vector-borne diseases are considered to be potentially sensitive to climate change (Table 5; Haines et al. 2006).

Host Vector Agent **Environment**

Figure 3. The Epidemiological Triangle

Table 5. Examples of Vector-Borne Diseases Likely To Be Sensitive to Climate Change

Vector	Major diseases
Mosquitoes	Malaria, filariasis, dengue fever, yellow fever, West Nile fever
Sandflies	Leishmaniasis
Triatomines	Chagas disease
beodes ticks	Lyme disease, tick-borne encephalitis
Tsetse flies	African trypanosomiasis
Blackflies	Onchocerciasts
Snails (intermediate host)	Schistosomiasis

Source: Haines et al. 2006.

Waterborne and airborne diseases may also be affected by climate change. For diseases transmitted by water, warming may enlarge the geographic area in which conditions are suitable for the survival of disease-causing organisms and for propagation of infection (Colwell 1996; Lipp et al. 2002). Colwell and colleagues have set out a schema by which global climate change alters patterns of cholera infections (Colwell 1996). The occurrence of waterborne infections has also been linked to extreme weather events (Charron et al. 2004). To date, little emphasis has been given to the possible impact of climate change on airborne infections. Increased air conditioning and more time spent indoors, because of warming, might affect patterns for diseases that are transmitted in indoor environments by droplets or by contact.

The potential impact of climate change on infectious diseases has been addressed through modeling approaches as well as through the investigation of specific shifts in infectious disease occurrence that could be attributed to climate change. Case studies of particular outbreaks and changes in infectious disease occurrence in relation to climate indicators provide further evidence of the role of climate change in altering patterns of infectious disease occurrence. For some agents, such as malaria, there is substantial controversy as to whether warming will increase occurrence. The case studies below exemplify the evidence used to link climate change to infectious diseases.

Cholera illustrates the complexity of understanding how climate change can alter the occurrence of infectious diseases (Figure 4; Lipp et al. 2002). Multiple global cholera pandemics have been documented; the seventh, which began in 1961, is still ongoing. The disease-causing organism, *Vibrio cholera*, is endemic and widely found in water. The present epidemic began with the emergence of a new biotype, the El Tor biotype of *V. cholerae* 01, in Indonesia. In 1991, the pandemic moved to South America with outbreaks along the Pacific coast. The occurrence of the epidemic was linked to a plankton bloom that was driven by the El Niño Southern Oscillation (ENSO). The planktonic copepod organism harbors the *V. cholera* organisms on its surface; consequently, a higher concentration of plankton increases the dose of the infectious agent received from water. A time-series analysis of cholera in Bangladesh found a link with the ENSO phenomenon (Rodo et al. 2002). Lipp and colleagues (2002) propose that climate change could affect each step in their model for cholera transmission.

Checkley and colleagues (2000) carried out a time-series analysis of temperature changes associated with the ENSO and all hospital admissions for diarrhea in children in Peru. Over the period 1993–1998 they found that the numbers of admissions were positively associated with temperature and also with the ENSO, which had an effect on the admissions rate beyond that expected from the temperature increase alone.

The IPCC (2007) report identified increased transmission of malaria as a potential consequence of climate change, coming from the effect of warming on vector numbers and on geographic spread. The potential for climate change to increase malaria is, however, still controversial in spite of empirical and modeling-based research. Loevinsohn (1994) published one of the initial time-series analyses based on data for Rwanda. Subsequently, there have been conflicting analyses of the potential for climate change to increase the spread of malaria and to cause it to become endemic in areas where it currently no longer occurs. The approaches in these conflicting publications are

conceptually comparable, involving time-series analyses to characterize the temperature–malaria relationship and the use of biological transmission models to incorporate the effect of temperature (Loevinsohn 1994; Sharp 1996; Epstein 1998; Haines 1998; Reiter 1998; Hales and Woodward 2003; Reiter et al. 2004). The sensitivity of findings to model assumptions indicates a need for more robust data. The IPCC (2007) report acknowledged the complexity of interpreting the empirical, time-series analyses and called for more research.

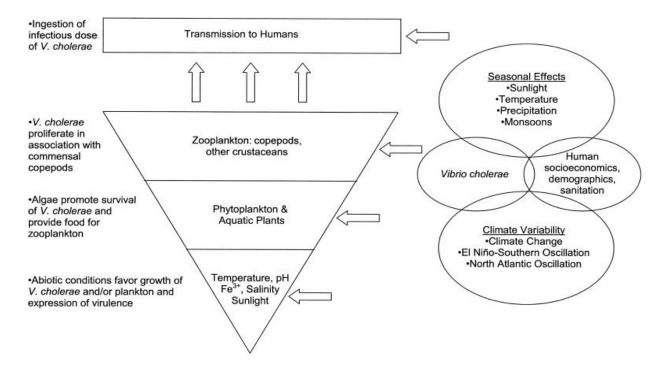


Figure 4. Hierarchical Model for Environmental Cholera Transmission

Source: Lipp et al. 2002.

Changes in the epidemiological characteristics of a variety of other infectious diseases have been examined in relation to climate change. Dengue transmission was addressed in the IPCC (2007) report and in specific studies (see, for example, Cazelles et al. 2005). In Western Australia, Woodruff et al. (2006) found that climate data predicted epidemics of Ross River virus disease, which is spread by mosquitoes, with reasonable predictive value, particularly if data on mosquitoes were incorporated into the model. Studies have also addressed tick-borne disease (Lindgren and Gustafson 2001; Ogden et al. 2008) and food-borne disease (D'Souza et al. 2008).

Determinants of Severity

Many factors determine susceptibility to infection and the severity of the resulting illness, including the risk of dying. In developed countries, key factors include age, immunocompetence, presence of comorbid chronic diseases (e.g., coronary heart disease, chronic obstructive pulmonary

disease, and diabetes), access to vaccines and medical care, and the quality of medical care available. In developing countries, additional determinants of severity include general nutritional status and specific micronutrient deficiencies, the level of sanitation, the availability of preventive measures (such as bed nets and vector control), and the availability of health care and vaccinations. In both developed and developing countries, large populations are at risk for more severe infections and mortality.

Spatial and Temporal Distribution

Warming from climate change has the potential to affect the transmission of infectious diseases across the globe. Although concern has been focused on countries with tropical, subtropical, and temperate climates, one outbreak of *Vibrio parahaemolyticus* gastroenteritis on a cruise ship in Alaska illustrates how warming can alter the spread of infectious organisms in colder climates (McLaughlin et al. 2005). In this outbreak, passengers developed gastroenteritis from eating contaminated oysters grown on a farm 1,000 kilometers north of the most northern point at which the organism had previously been identified.

The diseases of potential concern with regard to global warming are ubiquitous throughout the world. The time frames over which climate change could affect infectious diseases extend from relatively short, as even the temperature rise to date has had apparent impact, to relatively long, as continued temperature increases would be predicted to continue to alter vector distributions and densities.

Synthesis and Summary

Infectious diseases remain a leading cause of death throughout the world. Climate change could affect their frequency and distribution through multiple modes of transmission and diverse pathways. Several case studies document that small increases in temperature can affect the geographic distribution of infectious organisms and the occurrence of vector-borne diseases. The burden of premature mortality attributable to climate change for 2000 was estimated at 47,000 for diarrheal disease and 27,000 for malaria; however, these estimates are highly uncertain and only address two types of infection.

4. Ambient Air Pollution

Health Risks of Ambient Air Pollution

Climate change could potentially worsen air pollution, either directly, through increased tropospheric (ground-level) ozone production, or indirectly, through greater power plant emissions as power generation increases to meet the demand for greater air conditioning capacity. Ozone pollution is projected to increase because warmer temperatures increase ozone production (IPCC 2007). Ozone is a secondary pollutant, formed via sunlight driven photochemical reactions involving precursor hydrocarbons and oxides of nitrogen. Warmer temperatures enhance the chemical reactions that generate ozone. Under various scenarios, increases of several parts per billion are projected over the next two decades (Dentener et al. 2006) up to a range of 10 to 30

 parts per billion by the end of this century (Wilson et al. 2007). Fossil fuel combustion contaminates the atmosphere with the primary particles generated by combustion and with the secondary particles formed from gaseous components of power plant emissions through complex chemical and physical processes. Increased fossil fuel burning could also worsen particulate air pollution, beyond predictions from climate change scenarios (Davis and Working Group on Public Health and Fossil-Fuel Combustion 1997; IPCC 2007).

The health risks of both ozone and airborne particles have been characterized with reasonable certainty in epidemiological studies, with supporting evidence coming from in vivo and in vitro toxicological research (Pope and Dockery 2006; World Health Organization 2006). Both ozone and particulate matter (PM) air pollution have been associated with an increased risk of mortality in time-series studies of daily mortality (Samet et al. 2000; Bell et al. 2004; Pope and Dockery 2006), and airborne particles have also been associated with an increased risk of dying on longer time frames (Pope and Dockery 2006). Both types of pollution are also associated with morbidity, including an increased risk for hospitalization (Dominici et al. 2006; Pope and Dockery 2006) and other adverse outcomes (World Health Organization 2006). A quickly expanding body of evidence links particulate air pollution to adverse cardiovascular effects (Brook et al. 2004).

Two analyses that link climate change model outputs to ozone concentrations have been carried out for cities in the United States. Knowlton et al. (2004) projected the future increase in ozone concentration for 31 counties of the New York metropolitan region. Considering the impact of climate change alone on ozone concentration, they estimated a median 4.5 percent increase in summertime ozone-related, acute, all-cause mortality for the 31 counties. Bell et al. (Bell 2007) performed similar modeling for 50 cities in the eastern United States. They estimated the average increase in the daily one-hour maximum concentration as 4.8 parts per billion. Depending on the concentration–response relationship used, the increase in ozone concentration corresponded to an increase in daily, all-cause mortality of 0.11 to 0.27 percent.

Several analyses have addressed particulate air pollution, considering the potential risks of increased fossil fuel combustion and the benefits for health of reducing air pollution through greenhouse gas mitigation (Davis and Working Group on Public Health and Fossil-Fuel Combustion 1997; Cifuentes et al. 2001). Enhanced energy consumption based on fossil fuel combustion is predicted to lead to a substantial increase in premature mortality (Davis and Working Group on Public Health and Fossil-Fuel Combustion 1997); in one scenario of business as usual leading to increased exposure to PM air pollution, an additional 700,000 premature deaths were projected. Correspondingly, in an analysis of four of the world's major cities (Mexico City, New York City, Santiago, and São Paulo), Cifuentes et al. (2001) predicted that reductions of ozone and particles from mitigation would substantially reduce premature mortality.

Determinants of Severity

The risks of premature mortality and morbidity associated with exposure to ozone and PM air pollution increase with concentration; the most recent studies do not provide a clear indication of a threshold below which effects do not occur (U.S. Environmental Protection Agency 2006; World Health Organization 2006).

The factors determining responses to ambient air pollution have been studied extensively. As for heat stress, a wide range of groups are considered to be at risk: infants and the elderly, persons with chronic heart and lung disease, and the socially disadvantaged. Persons with greater potential to receive high doses of inhaled pollutants are also considered at risk; these large populations include those doing physical work or exercising outdoors when air pollution concentrations are elevated (U.S. Environmental Protection Agency 2008).

In addition, air pollution and thermal stress may act synergistically: both affect largely the same susceptible populations, and hot temperatures increase ozone production. Only a few studies have explored synergism between these two environmental stressors. In Athens, the short-term effect of sulfur dioxide on daily mortality was independent of temperature over the period 1975-1982 (Hatzakis et al. 1986). An analysis of mortality in Athens during a 1987 heat wave suggested synergism of air pollution with higher temperature (Katsouyanni et al. 1993). Filleul and colleagues (2006) examined the contributions of ozone and temperature to the excess mortality observed in nine French cities during the 2003 heat wave. They estimated that both ozone and heat contributed to the excess and that the relative contributions varied from city to city. Fischer et al. (2004) published similar findings for the Netherlands during 2003 as well. Neither analysis tested for synergism between ozone and temperature.

Spatial and Temporal Distribution

Air pollution is largely a problem in urban areas, where dominant sources include motor vehicles, industry, and power generation. Additional pollution may occur from fuel combustion, particularly biomass fuels, used for heating or cooking. In rural areas, emissions from these sources may also lead to locally significant ambient air pollution (Smith 2006). With regard to the effects of global climate change on ozone pollution, urban areas are of greatest concern; typically, ozone pollution extends well beyond urban centers across surrounding areas.

Synthesis and Summary

The risks of ambient air pollution to health have been studied extensively. For the two pollutants of concern with regard to global climate change—ozone and airborne PM—exposures have been strongly and consistently associated with increased risks for excess mortality and for morbidity. Effects are documented at levels that are prevalent throughout the major cities of the world in both developed and developing countries.

Warming will increase summertime ozone production, leading to greater exposures unless emissions of precursors are reduced. Particle levels may also increase, particularly if power generation from coal-fired power plants increases to support more air conditioning. Tools are available to estimate the potential burden of disease associated with worsening air pollution, but disentangling the contributions of climate change from those of other factors will not be practicable.

Specific Adaptations

1. Heat

Surveillance and Warning Systems

The needed tools for protecting people from heat stress are available. Temperature is readily and widely measured, and the weather conditions that lead to dangerous heat stress can be forecasted. The many epidemics of heat-caused deaths have identified those who need to be protected during heat waves, and there is a single stressor, heat, to be avoided. In fact, model heat watch systems have been implemented and their impact evaluated (Ebi and Schmier 2005).

Kalkstein and colleagues (1995) established one model for such systems based on the identification of weather conditions historically associated with increased mortality in a particular location and then the prospective issuance of a warning when such conditions arise. The approach uses exploratory and clustering statistical methods to identify synoptic conditions, *oppressive air masses*, that have been linked to increased mortality. The anticipated occurrence of such conditions triggers a protective response from public health and municipal authorities. In a 1996 paper describing this approach for the city of Philadelphia, Kalkstein et al. (1996) suggested that the implementation of this type of system may have reduced the impact of a heat wave in Philadelphia during the summer of 1995.

A decade later, Sheridan and Kalkstein (2004) reported on the widespread application of this approach in multiple cities in North America, Europe, and Asia. The underlying algorithm is set out in Figure 5 (Sheridan and Kalkstein 2004). Its implementation requires certain data, the capability to implement the synoptic classification system, and the capacity to implement a system of warning and response. Measures that might be taken to protect the public include media announcements, the activation of support networks, the implementation of a "heatline," taking steps to protect susceptible groups, and providing air-conditioned shelters. Although evaluation is difficult, studies in Philadelphia, Rome, and Shanghai indicate that this approach can reduce the mortality associated with heat waves (CDC et al. 2004; Ebi et al. 2004; Tan et al. 2004).

Following the 2003 European heat wave, a heat watch and warning system, including a national action plan, was implemented in France (Pascal et al. 2006). The system was based on an analysis of data from 14 cities in France and used temperature alone, rather than the synoptic approach advanced by Kalkstein and others. A heat wave in 2006 afforded the opportunity to assess the effectiveness of the system (Fouillet et al. 2008). This event, the second most severe since 1950 after the 2003 heat wave, led to more than 2,000 excess deaths in France—this was 4,400 fewer deaths than predicted based on the 2003 event. The evaluation documents that an effective warning system can be rapidly implemented.

Housing and Air Conditioning

Housing style and the use of air conditioning can lessen the impact of heat waves. In a number of studies, the availability of air conditioning has been shown to reduce the risk of mortality during a heat wave. As a longer-run strategy, increased use of air conditioning in homes would be expected

to protect against the heat-associated mortality, although the strategy has associated costs with regard to its implementation and the electric power to support the air conditioning.

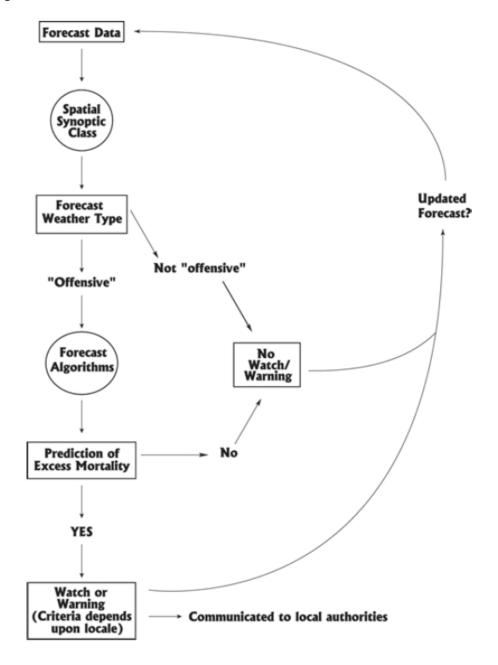


Figure 5. Flow Chart for the Determination of Whether to Call a Heat-Watch Warning

Source: Sheridan and Kalkstein 2004.

2. Aeroallergens and Allergic Diseases

Surveillance and Warning Systems

In more developed countries, such as the United States, tracking is in place both for aeroallergens and for the prevalence of allergic diseases, particularly asthma (American Academy of Allergy Asthma & Immunology n.d.). The aeroallergen monitoring is sensitive to changes in pollen and mold concentrations and to changes in their sources. Asthma surveillance remains difficult, in part because of the variability of the phenotype of asthma, the changing classification over time, and the potential for misclassifying other, minor conditions as asthma (Moorman et al. 2007). On the other hand, routine surveillance in the United States and in other countries has identified variation in asthma mortality rates with several epidemic rises over the past 50 years as well as the still unexplained rise in childhood asthma over the last several decades (Avila-Tang et al. 2008).

Most cases of asthma and other allergic diseases can be treated, and symptoms limited, if adequate medical care and treatment are available (National Heart Lung and Blood Institute and National Asthma Education and Prevention Program 2007). On the relatively slow time frame over which aeroallergen exposures may change, the medical care systems of many countries should be able to accommodate increasing numbers of persons with these diseases; by contrast, in many countries these disorders are untreated and are a substantial source of morbidity and even mortality (Braman 2006; Pearce et al. 2007; Shea et al. 2008).

3. Changes in Endemic and Epidemic Infectious Diseases

Surveillance

Surveillance is the fundamental tool for identifying changes in the patterns of infectious disease occurrence. In the United States, CDC maintains a variety of surveillance systems for specific infectious diseases. Some are passive, relying on proactive reporting by health care providers and facilities, whereas others are active, involving the collection of data through established systems and networks. The World Health Organization tracks the occurrence of key infectious diseases on a global basis. Absent effective surveillance—to detect outbreaks as well as more subtle, longer–time frame changes—adaptation cannot be successful.

Examples of established and ongoing surveillance at local, national, and global levels include those for HIV/AIDS and tuberculosis. For tuberculosis, the World Health Organization monitors not only the occurrence of disease, but also the operational success with which therapy is delivered (World Health Organization 2008). Notable, sentinel outbreaks are also likely to be detected; examples include the outbreak of *V. parahaemolyticus* aboard the cruise ship in Alaska (McLaughlin et al. 2005), the 2003 outbreak of severe acute respiratory syndrome (SARS) in countries of Asia and elsewhere (Naylor et al. 2004), and the very recent outbreak of Chikungunya in Italy (Charrel et al. 2007). In the future, such outbreaks are likely to occur more often in developed countries.

However, there may be barriers to establishing surveillance systems that extend across national boundaries, even if they are needed to protect global public health. In the initial phase of the SARS

epidemic in China, the scope of the epidemic was initially minimized and not revealed with sufficient warning (Naylor et al. 2004). The alerting function that should be an element of an effective surveillance system also failed. A case study of Hong Kong and Toronto also identified difficulty in linkages of clinical and reference laboratories into data systems (Naylor et al. 2004). In China, the failures in surveillance during the SARS outbreak were followed by a strengthening of capacity of the China Center for Disease Control, documenting that surveillance capacity can be addressed on a short-term basis.

Nonetheless, access to surveillance data persisted as a barrier in avian influenza surveillance in 2006, three years after the SARS epidemic (*Nature* 2006; Normile 2006). An approach to solving this problem was made with the Global Initiative on Sharing Avian Influenza Data (GISAID), a set of principles for sharing samples and data (GISAID n.d.). The need for this type of approach was evident, given the potential gravity of an avian influenza pandemic, and the key actors were moved to take action.

Can current surveillance methods identify the potential consequences of climate change for the occurrence of infectious diseases? Some reports of epidemics support this potential, at least for sentinel outbreaks (e.g., McLaughlin et al. 2005). Unless implemented with sufficient sensitivity and in vulnerable locations, changes in the zones of vector-borne and waterborne diseases may not be readily detected. Models of climate change and infectious diseases should be useful for guiding the design of surveillance systems.

Public Health Responses

Public health responses can be effective in controlling specific disease outbreaks; recently, they have proven most effective for controlling acute epidemics of disease, particularly those associated with emerging infections, such as SARS. On longer time frames, the eradication of smallpox was possible through a global initiative, and the delivery of curative therapy for tuberculosis has been enhanced. Slow changes in endemic diseases are less likely to be addressed in a timely and ongoing fashion.

Clinical Responses

Clinicians hold key roles, both in treating infectious diseases and in recognizing the occurrence of sentinel cases that signal a possible outbreak. The first cases of AIDS, for example, were recognized in the United States in 1981 because of the occurrence of a cluster of cases of Pneumocystis carinii pneumonia in gay men with immunocompromise (CDC 1981). Clinicians are an integral element of surveillance, a role they can better fill if they are alerted to the potential consequences of climate change and the possibility of emerging infections.

4. Ambient Air Pollution

In many countries, air quality regulations or guidelines are in place, along with extensive air quality management programs to control air pollution levels. The World Health Organization provides guidelines for the major ambient pollutants; the 2006 revisions (World Health Organization 2006) were more stringent than earlier versions as mounting evidence showed that

contemporary levels of air pollution are associated with continued risk, particularly for the elderly and for persons with chronic heart and lung diseases. A substantial proportion of the world's population is exposed to outdoor air pollutants at concentrations exceeding the World Health Organization's guideline values. In the most recent global burden of disease estimates, urban air pollution, using PM_{10} (coarse PM) as the surrogate, was estimated to cause about 3 percent of mortality attributable to cardiopulmonary disease in adults, 5 percent of lung cancers, and 1 percent of childhood mortality from acute respiratory illnesses (Cohen et al. 2004). In this analysis, many cities of the world were estimated to have PM_{10} concentrations well above the current standards of the U.S. Environmental Protection Agency (n.d. [a]) as well as the target values proposed in the 2006 World Health Organization guidelines.

The anticipated changes in ozone, and possibly in PM, air pollution will happen on a relatively long time frame, approximately over decades. During this same time period, increasing numbers of motor vehicles are anticipated to be used in the major cities of the developing world, adding to the potential for ozone production to increase. On the other hand, as petroleum supplies lessen and fuel prices increase, the growth of motor vehicle use may be slowed, and efforts at conservation may reduce emissions as well.

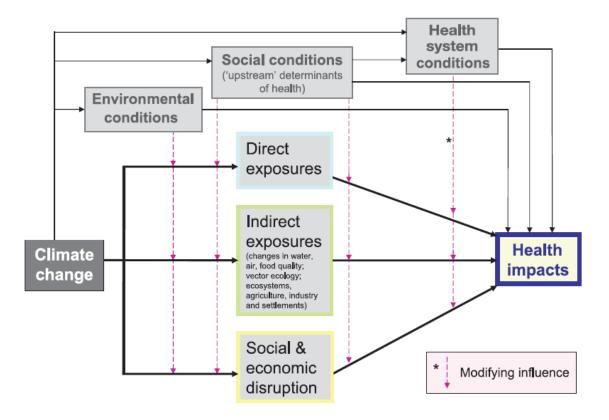
Ozone and PM are monitored routinely in an increasing number of cities; consequently, data should be available on trends of these pollutants in the world's major cities. Time–trend analysis will probably be insufficiently sensitive for the identification of the specific contribution of climate change, given the numerous determinants of concentrations of urban air pollution. On the other hand, air quality management strategies will be directed at limiting the emissions of ozone precursors and controlling primary particle emissions and contributors to secondary particle formation. Strategies directed at the control of greenhouse gas emissions will also reduce ambient air pollution (Davis and Working Group on Public Health and Fossil-Fuel Combustion 1997). To date in the United States and many countries of Europe, levels of the major urban and regional air pollutants have dropped, showing that air quality management strategies can be effective (U.S. Environmental Protection Agency n.d. [a]; World Health Organization 2006). Over the short term, these same strategies should be effective in controlling air pollution concentrations. Longer-run predictions are made difficult by uncertainty around possibly rising power plant emissions—if warming leads to the need for greater capacity for electricity for cooling—and because of potential changes in the powering of motor vehicles.

Context

Numerous systems are in place to protect the public against threats to health, including those threats predicted to take place consequent to climate change. The systems vary in levels of organization—from local to global—and in their capacity, competence, and effectiveness (White and Nanan 2008). These systems will represent the starting point in any effort to track and control the health consequences of climate change. The occurrence of health outcomes potentially affected by climate change reflects the intersection of multiple factors (Figure 6; IPCC 2007). These factors are operative across multiple levels of societal organization, indicating that adaptation to the health

consequences of climate change will necessarily engage agencies and institutions that extend from the local to the global level.

Figure 6. Schematic Diagram of Pathways by Which Climate Change Affects Health, and Concurrent Direct-Acting and Modifying (Conditioning) Influences of Environmental, Social, and **Health System Factors**



Source: IPCC 2007.

International health agencies, key to global approaches, have been classified as multilateral, bilateral, nongovernmental, and other. The lead, multilateral organization for health is the World Health Organization. Its reach is global, coordinated from its headquarters in Geneva to regional and national offices; it maintains a variety of surveillance systems and responds to major acute and chronic threats to health. There are many instances of collaboration between nations on matters of health, often through a pairing of more developed and less developed countries. Nongovernmental organizations are playing an increasing role in global public health, both by providing substantial resources for disease control and through program implementation. In the specific example of climate change, they may also motivate action, toward both mitigation and adaptation.

Examples already cited have challenged this global system to respond to far-reaching public health threats. There have been successes, and the example of smallpox eradication remains a

RFF I 26 **SAMET**

model. SARS has been contained, and surveillance in China should identify further cases. Problems with surveillance for avian influenza were identified and steps were taken to address them.

For public health problems on a longer time frame, reflecting global causes, responses have been slower and less organized at the global level. In the example of cigarette smoking, an extraordinarily potent cause of chronic diseases and death, unified action was not taken at the global level until 50 years after firm evidence on smoking and lung cancer was published. Researchers and tobacco control professionals had established networks, but the needed institutional response at the global level was delayed, even though the root cause of the epidemic was the multinational tobacco companies. The World Health Organization's Tobacco Free Initiative was established at the end of the 20th century, 100 years after smoking became widespread among men in many countries. Now a global treaty, the Framework Convention on Tobacco Control, addresses the root cause of the epidemic. Global tobacco control initiatives have been launched, and surveillance systems are coming into place to address smoking among children and adults.

Conclusions

Much has been written on adaptation to climate change; books and reviews address not only the means of adaptation but the policy context (Fussel 2007). The European Union funded the Climate Change Adaptation Strategies for Human Health project to systematically assess adaptation strategies for Europe (Menne and Ebi 2006). This extensive project charted the potential threats of climate change to human health in Europe and addressed policy implications and the potential for adaptation to mitigate these consequences. It is comprehensive in its coverage of the topic, but evidence for the utility of this effort will be forthcoming only as the extent of its use by decisionmakers plays out. The term *climate change adaptation science* has been used, implying a formalism and the emergence of evidence-based approaches (Fussel et al. 2006).

The methods for addressing the health consequences of climate change, as evident in this review, are those of public health and disease control generally. A unique aspect of the health consequences of climate change is that climate change is an extremely "upstream" driver (Figure 6). The consequences of climate change for health range from being quite specific (e.g., heat waves), to general (e.g., increased exposure to air pollution), and from being acute in nature (e.g., infectious disease outbreaks), to longer-term (e.g., changes in allergic diseases associated with shifts in aeroallergens). For some of the health consequences of climate change—such as emerging infections and heat waves—adaptation will take place through the routine functioning of effective public health systems, if in place. Some, such as allergic diseases, will be managed through routine medical care, and others, including increased emissions of air pollution, will be addressed through regulatory mechanisms.

Across the public health community, views vary on the urgency of addressing the public health consequences of climate change. All concur with the need for primary prevention—that is, slowing climate change as quickly as possible. Some propose that the health sector needs to become more proactively engaged in pushing for solutions and advancing strategies for adaptation (Haines and Patz 2004; Menne and Bertollini 2005; Frumkin and McMichael 2008; McMichael et al. 2008). A

recent issue of the *American Journal of Preventive Medicine* covers the medical dimensions of climate change and sets out strategies for attempting to mitigate them. In introducing the issue, Frumkin and McMichael (2008) comment on the need for a reorientation of public health approaches to reflect the long time frame for action and the need for "systems thinking," along with "effective framing and communication" and proactive leadership.

Although the time frame over which climate change is anticipated to affect health is long, in fact far longer than the time domains on which public health planning usually takes place, some steps should be taken immediately. One such immediate step is to assess capacity and begin to address gaps (Ebi 2009). A variety of stakeholders are involved, as described by Ebi (2009), and these should be surveyed. Other actions should also be taken without delay. For example, places at risk for heat events should have warning systems in place, along with programs to reduce the consequences of thermal stress.

Will the health consequences of climate change be a useful lever for enhancing public health data systems and capacity and for engaging health professionals in mitigating the health consequences? At the national level, the projected risks of climate change may motivate the enhancement of data systems and improved preparedness for addressing possibly more frequent and more severe disastrous weather events. On the other hand, at more local levels, the threat of climate change may appear remote, viewed in the context of pressing, local issues.

The recognition and quantification of the health consequences of climate change will be difficult, given their lack of specificity. Risk assessment methods, including burden of disease estimation, will remain central as a tool for estimating the need for the implementation of adaptive strategies and for quantifying their benefits. Tracking the benefits of adaptation for the purpose of accountability will probably prove difficult, given the multiplicity of factors affecting the health outcomes of concern (Health Effects Institute 2003). At the national level, the government should ensure the clear designation of the locus within the federal government that will track the health consequences of climate change and assess the extent to which adaptation strategies are in place as well as their effectiveness. Absent this monitoring function, there will inevitably be uncertainty as to whether the right steps have been taken and whether they have worked.

References

- American Academy of Allergy Asthma & Immunology. No date. American Academy of Allergy, Asthma & Immunology (AAAAI). http://www.aaaai.org/ (accessed April 9, 2009).
- American Public Health Association. No date. 10 Essential Public Health Services. http://www.apha.org/programs/standards/performancestandardsprogram/resexxentials ervices.htm (accessed May 8, 2009).
- Avila-Tang, E., E. Matsui, D.G. Wiesch, J.M. Samet. 2008. Epidemiology of Asthma and Allergic Diseases. In *Allergy: Principles and Practices*, 7th ed., edited by N.F. Adkinson, W.W. Busse, B.S. Bochner, S.T. Holgate, F.E. Simons, and R.F. Lemanske Jr. Edinburgh: Mosby-Elsevier, 42-1-42-53.
- Barnett, A.G. 2007. Temperature and Cardiovascular Deaths in the U.S. Elderly: Changes over Time. *Epidemiology* 18(3): 369–372.
- Basu, R., and J.M. Samet. 2002. Relation between Elevated Ambient Temperature and Mortality: A Review of the Epidemiologic Evidence. *Epidemiological Reviews* 24(2): 190–202.
- Beaglehole, R., and M.R. Dal Poz. 2003. Public Health Workforce: Challenges and Policy Issues. *Human Resources for Health* 1(1): 4.
- Beggs, P.J. 2004. Impacts of Climate Change on Aeroallergens: Past and Future. *Clinical & Experimental Allergy* 34(10): 1507–1513.
- Beggs, P.J., and H.J. Bambrick. 2005. Is the Global Rise of Asthma an Early Impact of Anthropogenic Climate Change? *Environmental Health Perspectives* 113(8): 915–919.
- Bell, M.L., R. Goldberg, C. Hogrefe, P.L. Kinney, K. Knowlton, B. Lynn, J. Rosenthal, C. Rosenzweig, and J.A. Patz. 2007. Climate Change, Ambient Ozone, and Health in 50 US Cities. *Climatic Change* 82: 61-76.
- Bell, M.L., J.M. Samet, and F. Dominici. 2004. Time-Series Studies of Particulate Matter. *Annual Review of Public Health* 25: 247–280.
- Braman, S.S. 2006. The Global Burden of Asthma. *Chest* 130(1 Suppl): 4S–12S.
- Brook, R.D., B. Franklin, W. Cascio, Y. Hong, G. Howard, M. Lipsett, R. Luepker, M. Mittleman, J. Samet, S.C. Smith Jr., and I. Tager. 2004. Air Pollution and Cardiovascular Disease: A Statement for Healthcare Professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation* 109(21): 2655–2671.
- Buechley, R.W., B.J. Van, and L.E. Truppi. 1972. Heat Island Equals Death Island? *Environmenal Research* 5(1): 85–92.

- Campbell-Lendrum, D., and R. Woodruff. 2006. Comparative Risk Assessment of the Burden of Disease from Climate Change. *Environmental Health Perspectives* 114(12): 1935–1941.
- Carson, C., S. Hajat, B. Armstrong, and P. Wilkinson. 2006. Declining Vulnerability to Temperature-Related Mortality in London over the 20th Century. *American Journal of Epidemiology* 164(1): 77–84.
- Cazelles, B., Chavez, M., McMichael, A.J., and S. Hales. 2005. Nonstationary Influence of El Nino on the Synchronous Dengue Epidemics in Thailand. *PLoS Medicine* 2(4): e106.
- (CDC) Centers for Disease Control and Prevention. 1981. Pneumocystis pneumonia—Los Angeles. *Morbidity and Mortality Weekly Report* 30(21): 250–252.
- ——. 1995. Heat-Related Mortality—Chicago, July 1995. *Morbidity and Mortality Weekly Report* 44(31): 577–579.
- ——. 2004. Hypothermia-Related Deaths—United States, 2003. *Morbidity and Mortality Weekly Report* 53(8): 172–173.
- ———. 2006. Heat-Related Deaths—United States, 1999–2003. *Morbidity and Mortality Weekly Report* 55(29): 796–798.
- ——. No date (a). National Center for Health Statistics. http://www.cdc.gov/nchs/ (accessed April 9, 2009).
- ——. No date (b). Nationally Notifiable Infectious Diseases. http://www.cdc.gov/ncphi/disss/nndss/phs/infdis.htm (accessed April 9, 2009).
- CDC, P. Michelozzi, F. de' Denato, G. Accetta, F. Forastiere, M. D'Ovideo, C. Perucci, and L. Kalkstein. 2004. Impact of Heat Waves on Mortality—Rome, Italy, June–August 2003. *Morbidity and Mortality Weekly Reports* 53(17): 369–371.
- Charrel, R.N., L.X. de, and D. Raoult. 2007. Chikungunya Outbreaks—the Globalization of Vectorborne Diseases. *New England Journal of Medicine* 356(8): 769–771.
- Charron, D., M. Thomas, D. Waltner-Toews, J. Aramini, T. Edge, R. Kent, A. Maarouf, and J. Wilson. 2004. Vulnerability of Waterborne Diseases to Climate Change in Canada: A Review. *Journal of Toxicology and Environmental Health, Part A* 67(20–22): 1667–1677.
- Checkley, W., L.D. Epstein, R.H. Gilman, D. Figueroa, R.I. Cama, J.A. Patz, and R.E. Black. 2000. Effect of El Nino and Ambient Temperature on Hospital Admissions for Diarrhoeal Diseases in Peruvian Children. *Lancet* 355(9202): 442–450.
- Cifuentes, L., V.H. Borja-Aburto, N. Gouveia, G. Thurston, and D.L. Davis. 2001. Assessing the Health Benefits of Urban Air Pollution Reductions Associated with Climate Change Mitigation (2000–2020): Santiago, Sao Paulo, Mexico City, and New York City. *Environmental Health Perspectives* 109(Suppl 3): 419–425.

- Cohen, A.J., H.R. Anderson, B. Ostro, K.D. Pandey, M. Krzyzanowski, N. Kunzli, K. Gutschmidt, C.A. Pope III, I. Romieu, J.M. Samet, K.R. Smith. 2004. Urban Air Pollution. In *Comparative Quantification of Health Risks. Global and Regional Burden of Disease Attributable to Selected Major Risk Factors*, edited by M. Ezzati, A.D. Lopez, A. Rogers, and C.J.L. Murray. Geneva: World Health Organization, 1353–1434.
- Colwell, R.R. 1996. Global Climate and Infectious Disease: The Cholera Paradigm. *Science* 274(5295): 2025–2031.
- Curriero, F.C., K.S. Heiner, J.M. Samet, S.L. Zeger, L. Strug, and J.A. Patz. 2002. Temperature and Mortality in Eleven Cities of the Eastern United States. *American Journal of Epidemiology* 155(1): 80–87.
- Davis, D.L., and Working Group on Public Health and Fossil-Fuel Combustion. 1997. Short-Term Improvements in Public Health from Global-Climate Policies on Fossil-Fuel Combustion: An Interim Report. Working Group on Public Health and Fossil-Fuel Combustion. *Lancet* 350(9088): 1341–1349.
- Davis, R.E., P.C. Knappenberger, P.J. Michaels, and W.M. Novicoff. 2003. Changing Heat-Related Mortality in the United States. *Environmental Health Perspectives* 111(14): 1712–1718.
- Dentener, F., D. Stevenson, K. Ellingsen, N.T. Van, M. Schultz, M. Amann, C. Atherton, N. Bell, D. Bergmann, I. Bey, L. Bouwman, T. Butler, J. Cofala, B. Collins, J. Drevet, R. Doherty, B. Eickhout, H. Eskes, A. Fiore, M. Gauss, D. Hauglustaine, L. Horowitz, I.S. Isaksen, B. Josse, M. Lawrence, M. Krol, J.F. Lamarque, V. Montanaro, J.F. Muller, V.H. Peuch, G. Pitari, J. Pyle, S. Rast, I. Rodriguez, M. Sanderson, N.H. Savage, D. Shindell, S. Strahan, S. Szopa, K. Sudo, D.R. Van, O. Wild, and G. Zeng. 2006. The Global Atmospheric Environment for the Next Generation. *Environmental Science and Technology* 40(11): 3586–3594.
- Detels, R., J. McEwen, R. Beaglehole, and H. Tanaka. 2004. *Oxford Textbook of Public Health*. New York: Oxford University Press.
- Dominici, F., R.D. Peng, M.L. Bell, L. Pham, A. McDermott, S.L. Zeger, and J.M. Samet. 2006. Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases. *Journal of the American Medical Association* 295(10): 1127–1134.
- D'Souza, R.M., G. Hall, and N.G. Becker. 2008. Climatic Factors Associated with Hospitalizations for Rotavirus Diarrhoea in Children under 5 Years of Age. *Epidemiology and Infection* 136(1): 56–64.
- Ebi, K.L. 2009. Public Health Responses to the Risks of Climate Variability and Change in the United States. *Journal of Occupational and Environmental Medicine* 51(1): 4–12.
- Ebi, K.L., and J.K. Schmier. 2005. A Stitch in Time: Improving Public Health Early Warning Systems for Extreme Weather Events. *Epidemiologic Reviews* 27: 115–121.
- Ebi, K.L., T.J. Teisberg, L.S. Kalkstein, L. Robinson, and R.F. Weiher. 2004. Heat Watch/Warning Sytems Save Lives. Estimated Costs and Benefits for Philadelphia 1995–98. *Bulletin of the American Meteorological Society* 85(8): 1067–1073.

- Epstein, P.R. 1998. Global Warming and Vector-borne Disease. Lancet 351(9117): 1737.
- Etches, V., J. Frank, R.E. Di, and D. Manuel. 2006. Measuring Population Health: A Review of Indicators. *Annual Review of Public Health* 27: 29–55.
- Filleul, L., S. Cassadou, S. Medina, P. Fabres, A. Lefranc, D. Eilstein, T.A. Le, L. Pascal, B. Chardon, M. Blanchard, C. Declercq, J.F. Jusot, H. Prouvost, and M. Ledrans. 2006. The Relation between Temperature, Ozone, and Mortality in Nine French Cities during the Heat Wave of 2003. *Environmental Health Perspectives* 114(9): 1344–1347.
- Fischer, P.H., B. Brunekreef, and E. Lebret. 2004. Air Pollution Related Deaths during the 2003 Heat Wave in the Netherlands. *Atmospheric Environment* 38(8): 1083–1085.
- Fouillet, A., G. Rey, V. Wagner, K. Laaidi, P. Empereur-Bissonnet, T.A. Le, P. Frayssinet, P. Bessemoulin, F. Laurent, P. De Crouy-Chanel, E. Jougla, and D. Hemon. 2008. Has the Impact of Heat Waves on Mortality Changed in France Since the European Heat Wave of Summer 2003? A Study of the 2006 Heat Wave. *International Journal of Epidemiology* 37(2): 309–317.
- Frumkin, H., J. Hess, G. Luber, J. Malilay, and M. McGeehin. 2008. Climate Change: The Public Health Response. *American Journal of Public Health* 98(3): 435–445.
- Frumkin, H., and A.J. McMichael. 2008. Climate Change and Public Health: Thinking, Communicating, Acting. *American Journal of Preventive Medicine* 35(5): 403–410.
- Fussel, H.M. 2007. Adaptation Planning for Climate Change: Concepts, Assessment Approaches and Key Lessons. *Sustainability Science* 2(2): 265–275.
- Fussel, H.M., R.J.T. Klein, and K.L. Ebi. 2006. Adaptation Assessment for Public Health. In *Climate Change and Adaptation Strategies for Human Health*, edited by B. Menne and K.L. Ebi. Darmstadt, Germany: Steinkopff Verlag, 41–62.
- GISAID (The Global Initiative on Sharing Avian Influenza Data). No date. The Global Initiative on Sharing Avian Influenza Data (GISAID). http://platform.gisaid.org/ (accessed April 9, 2009).
- Haines, A. 1998. Global Warming and Vector-borne Disease. Lancet 351(9117): 1737–1738.
- Haines, A., R.S. Kovats, D. Campbell-Lendrum, and C. Corvalan. 2006. Climate Change and Human Health: Impacts, Vulnerability, and Mitigation. *Lancet* 367(9528): 2101–2109.
- Haines, A., and J.A. Patz. 2004. Health Effects of Climate Change. *Journal of the American Medical Association* 291(1): 99–103.
- Hajat, S., B. Armstrong, M. Baccini, A. Biggeri, L. Bisanti, A. Russo, A. Paldy, B. Menne, and T. Kosatsky. 2006. Impact of High Temperatures on Mortality: Is There an Added Heat Wave Effect? *Epidemiology* 17(6): 632–638.

- Hajat, S., B.G. Armstrong, N. Gouveia, and P. Wilkinson. 2005. Mortality Displacement of Heat-Related Deaths: A Comparison of Delhi, Sao Paulo, and London. *Epidemiology* 16(5): 613–620.
- Hales, S., and A. Woodward. 2003. Climate Change Will Increase Demands on Malaria Control in Africa. *Lancet* 362(9398): 1775.
- Hatzakis, A., K. Katsouyanni, A. Kalandidi, N. Day, and D. Trichopoulos. 1986. Short-term Effects of Air Pollution on Mortality in Athens. *International Journal of Epidemiology* 15: 73-81.
- Health Effects Institute. 2003. Assessing Health Impact of Air Quality Regulations: Concepts and Methods for Accountability Research. HEI Communication 11. Boston, MA: Health Effects Institute Accountability Working Group.
- Institute of Medicine and Committee on Assuring the Health of the Public in the 21st Century. 2002. The Future of the Public's Health in the 21st Century. Washington, DC: National Academies Press.
- Institute of Medicine, K. Gebbie, L. Rosenstock, and L.M. Hernandez. 2003. *Who Will Keep the Public Healthy? Educating Public Health Professionals for the 21st Century*. Washington, DC: National Academies Press.
- IPCC (Intergovernmental Panel on Climate Change). 2007. *Climate Change 2007: Impacts, Adaptation and Vulnerability*. Geneva: IPCC.
- Kalkstein, L.S., C.D. Barthel, J.S. Greene, and M.C. Nichols. 1995. A New Spatial Synoptic Classification: Application to Air Mass Analysis. *International Journal of Climatology* 26: 23–31.
- Kalkstein, L.S., P.F. Jamason, J.S. Greene, J. Libby, and L. Robinson. 1996. The Philadelphia Hot Weather-Health Watch/Warning System: Development and Application, Summer 1995. *Bulletin of the American Meteorological Society* 77(7): 1519–1528.
- Katsouyanni, K., A. Pantazopoulou, G. Touloumi, I. Tselepidaki, K. Moustris, D. Asimakopoulos, G. Poulopoulou, and D. Trichopoulos. 1993. Evidence for Interaction between Air Pollution and High Temperature in the Causation of Excess Mortality. *Archives of Environmental Health* 48(4): 235–242.
- Klinenberg, E. 2002. *Heat Wave. A Social Autopsy of Disaster in Chicago*. Chicago: University of Chicago Press.
- Knowlton, K., J.E. Rosenthal, C. Hogrefe, B. Lynn, S. Gaffin, R. Goldberg, C. Rosenzweig, K. Civerolo, J.Y. Ku, and P.L. Kinney. 2004. Assessing Ozone-Related Health Impacts under a Changing Climate. *Environmental Health Perspectives* 112(15): 1557–1563.
- Kovats, R.S., D. Campbell-Lendrum, and F. Matthies. 2005. Climate Change and Human Health: Estimating Avoidable Deaths and Disease. *Risk Analysis* 25(6): 1409–1418.

<u>≧ rff i Samet</u>

- Kovats, R.S., G. Jendritzky, B. Menne, and K.L. Ebi. 2006. Heatwaves and Human Health. *Climate Change and Adaptation Strategies for Human Health*, edited by B. Menne and K.L. Ebi. Darmstadt, Germany: Steinkopff-Verlag Darmstadt, 63–97.
- Langmuir, A.D. 1963. The Surveillance of Communicable Diseases of National Importance. *New England Journal of Medicine* 268: 182–192.
- Levin, M.L. 1953. The Occurrence of Lung Cancer in Man. Acta Un Intern Cancer 9: 531–541.
- Lindgren, E., and R. Gustafson. 2001. Tick-borne Encephalitis in Sweden and Climate Change. *Lancet* 358(9275): 16–18.
- Lipp, E.K., A. Huq, and R.R. Colwell. 2002. Effects of Global Climate on Infectious Disease: The Cholera Model. *Clinical Microbiology Reviews* 15(4): 757–770.
- Loevinsohn, M.E. 1994. Climatic Warming and Increased Malaria Incidence in Rwanda. *Lancet* 343(8899): 714–718.
- McLaughlin, J.B., A. DePaola, C.A. Bopp, K.A. Martinek, N.P. Napolilli, C.G. Allison, S.L. Murray, E.C. Thompson, M.M. Bird, and J.P. Middaugh. 2005. Outbreak of Vibrio parahaemolyticus Gastroenteritis Associated with Alaskan Oysters. *New England Journal of Medicine* 353(14): 1463–1470.
- McMichael, A.J., D. Campbell-Lendrum, S. Kovats, S. Edwards, P. Wilkinson, T. Wilson, R. Nicholls, S. Hales, F. Tanser, D. Le Sueur, M. Schlesinger, N. Andronova, M. Ezzati, A.D. Lopez, A. Rodgers, and C.J.L. Murray. 2004. Global Climate Change. In *Comparative Quantification of Health Risks*, vol. 2, edited by M. Ezzati, A.D. Lopez, A. Rogers, and C.J.L. Murray. Geneva: World Health Organization, 1543–1649.
- McMichael, A.J., P. Wilkinson, R.S. Kovats, S. Pattenden, S. Hajat, B. Armstrong, N. Vajanapoom, E.M. Niciu, H. Mahomed, C. Kingkeow, M. Kosnik, M.S. O'Neill, I. Romieu, M. Ramirez-Aguilar, M.L. Barreto, N. Gouveia, and B. Nikiforov. 2008. International Study of Temperature, Heat and Urban Mortality: The "ISOTHURM" Project. *International Journal of Epidemiology* 37: 1121–1131.
- McMichael, A.J., R.E. Woodruff, and S. Hales. 2006. Climate Change and Human Health: Present and Future Risks. *Lancet* 367(9513): 859–869.
- Menne, B., and R. Bertollini. 2005. Health and Climate Change: A Call for Action. *British Medical Journal* 331(7528): 1283–1284.
- Menne, B., and K.L. Ebi. 2006. *Climate Change and Adaptation Strategies for Human Health*. Darmstadt, Germany: Steinkopff-Verlag Darmstadt.
- Metz, B., O.R. Davidson, P.R. Bosch, R. Dave, and L.A. Meyer (eds). 2007. *Climate Change 2007: Mitigation of Climate Change. Contribution of Working Group III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change.* Cambridge: Cambridge University Press.

- Mokdad, A.H., J.S. Marks, D.F. Stroup, and J.L. Gerberding. 2004. Actual Causes of Death in the United States, 2000. *Journal of the American Medical Association* 291(10): 1238–1245.
- Moorman, J.E., R.A. Rudd, C.A. Johnson, M. King, P. Minor, C. Bailey, M.R. Scalia, and L.J. Akinbami. 2007. National Surveillance for Asthma—United States, 1980–2004. *MMWR: Surveillance Summaries* 56(8): 1–54.
- National Heart Lung and Blood Institute and National Asthma Education and Prevention Program. 2007. Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health.
- Nature. 2006. Boosting Access to Disease Data. 442(7106): 957.
- Naylor, C.D., C. Chantler, and S. Griffiths. 2004. Learning from SARS in Hong Kong and Toronto. *Journal of the American Medical Association* 291(20): 2483–2487.
- Nelson, K.E., C.F. Masters Williams, and N.M.H. Graham. 2007. *Infectious Disease Epidemiology: Theory and Practice*. Sudbury, MA: Jones and Bartlett Publishers.
- Normile, D. 2006. Avian influenza. Is China Coming Clean on Bird Flu? Science 314(5801): 905.
- Ogden, N.H., L. St.Onge, I.K. Barker, S. Brazeau, M. Bigras-Poulin, D.F. Charron, C.M. Francis, A. Heagy, L.R. Lindsay, A. Maarouf, P. Michel, F. Milord, C.J. O'Callaghan, L. Trudel, and R.A. Thompson. 2008. Risk Maps for Range Expansion of the Lyme Disease Vector, Ixodes scapularis, in Canada Now and with Climate Change. *International Journal of Health Geographics* 7: 24.
- Pascal, M., K. Laaidi, M. Ledrans, E. Baffert, C. Caserio-Schonemann, T.A. Le, J. Manach, S. Medina, J. Rudant, and P. Empereur-Bissonnet. 2006. France's Heat Health Watch Warning System. *International Journal of Biometeorology* 50(3): 144–153.
- Patz, J.A., D. Campbell-Lendrum, T. Holloway, and J.A. Foley. 2005. Impact of Regional Climate Change on Human Health. *Nature* 438(7066): 310–317.
- Pearce, N., R. Beasley, C. Burgess, and J. Crane. 1998. *Asthma Epidemiology. Principles and Methods.* New York: Oxford University Press.
- Pearce, N., N. it-Khaled, R. Beasley, J. Mallol, U. Keil, E. Mitchell, and C. Robertson. 2007. Worldwide Trends in the Prevalence of Asthma Symptoms: Phase III of the International Study of Asthma and Allergies in Childhood (ISAAC). *Thorax* 62(9): 758–766.
- Pope, C.A. III, and D.W. Dockery. 2006. Health Effects of Fine Particulate Air Pollution: Lines That Connect. *Journal of the Air and Waste Management Association* 56(6): 709–742.
- Poumadere, M., C. Mays, M.S. Le, and R. Blong. 2005. The 2003 Heat Wave in France: Dangerous Climate Change Here and Now. *Risk Analysis* 25(6): 1483–1494.
- Reiter, P. 1998. Global-Warming and Vector-borne Disease in Temperate Regions and at High Altitude. *Lancet* 351(9105): 839–840.

- Reiter, P., C.J. Thomas, P.M. Atkinson, S.I. Hay, S.E. Randolph, D.J. Rogers, G.D. Shanks, R.W. Snow, and A. Spielman. 2004. Global Warming and Malaria: A Call for Accuracy. *Lancet Infectious Diseases* 4(June): 323–324.
- Rodo, X., M. Pascual, G. Fuchs, and A.S. Faruque. 2002. ENSO and Cholera: A Nonstationary Link Related to Climate Change? *Proceedings of the National Academy of Sciences of the United States of America* 99(20): 12901–12906.
- Rogers, D.J., and S.E. Randolph. 2006. Climate Change and Vector-borne Diseases. *Advances in Parasitology* 62: 345–381.
- Samet, J.M., F. Dominici, F.C. Curriero, I. Coursac, and S.L. Zeger. 2000. Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987–1994. *New England Journal of Medicine* 343(24): 1742–1749.
- Semenza, J.C., C.H. Rubin, K. Falter, J.D. Selanikio, W.D. Flanders, H.L. Howe, and J.L. Wilhelm. 1996. Heat-Related Deaths during the July 1995 Heatwave in Chicago. *New England Journal of Medicine* 335(2): 84–90.
- Sharp, D. 1996. Malarial Range Set to Spread in a Warmer World. *Lancet* 347(9015): 1612.
- Shea, K.M., R.T. Truckner, R.W. Weber, and D.B. Peden. 2008. Climate Change and Allergic Disease. *The Journal of Allergy and Clinical Immunology* 122(3): 443–453.
- Sheridan, S.C., and L.S. Kalkstein. 2004. Progress in Heat Watch-Warning System Technology. *Bulletin of the American Meterological Society* 65: 1931–1941.
- Smith, K.R. 2006. *Rural Air Pollution: A Major but Often Ignored Development Concern*. New York: United Nations, Commission on Sustainable Development Thematic Session on Integrated Approaches to Addressing Air Pollution and Atmospheric Problems.
- Sporik, R., S.T. Holgate, T.A. Platts-Mills, and J.J. Cogswell. 1990. Exposure to House-Dust Mite Allergen (Der p I) and the Development of Asthma in Childhood. A Prospective Study. *New England Journal of Medicine* 323(8): 502–507.
- Tan J., L.S. Kalkstein, J. Huang, S. Lin, H. Yin, and D. Shao. 2004. An Operational Heat/Health Warning System in Shanghai. *International Journal of Biometeorology* 48(3): 157–162.
- Teutsch, S.M., and R.E. Churchill. 2000. *Principles and Practice of Public Health Surveillance*. New York: Oxford University Press.
- The Eurowinter Group. 1997. Cold Exposure and Winter Mortality from Ischaemic Heart Disease, Cerebrovascular Disease, Respiratory Disease, and All Causes in Warm and Cold Regions of Europe. The Eurowinter Group. *Lancet.* 349(9062): 1341–1346.
- Toulemon, L., and M. Barbieri. 2008. The Mortality Impact of the August 2003 Heat Wave in France: Investigating the "Harvesting" Effect and Other Long-Term Consequences. *Population Studies (Cambridge)* 62(1): 39–53.

- U.S. Department of Health and Human Services. 2004. *The Health Consequences of Smoking. A Report of the Surgeon General.* Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.
- U.S. Environmental Protection Agency. 2006. *Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final)*. Washington, DC: U.S. Environmental Protection Agency.
- ——. 2008. Clean Air Scientific Advisory Committee's (CASAC) Consultation on EPA's Draft Scope and Methods Plan for Risk/Exposure Assessment: Secondary NAAQS Review for NO_x and SO_x. Washington, DC: U.S. Environmental Protection Agency.
- ———. No date (a). Air Quality Index (AQI)—A Guide to Air Quality and Your Health. http://airnow.gov/ (accessed April 9, 2009).
- ——. No date (b). Heat Island Effect. http://www.epa.gov/heatisland/research/index.htm (accessed April 9, 2009).
- Vandentorren, S., F. Suzan, S. Medina, M. Pascal, A. Maulpoix, J.C. Cohen, and M. Ledrans. 2004. Mortality in 13 French Cities during the August 2003 Heat Wave. *American Journal of Public Health* 94(9): 1518–1520.
- Wallace, R. (ed.) 2008. *Maxcy-Rosenau-Last Public Health and Preventive Medicine*, 15th ed. New York: McGraw-Hill Medical.
- White, F.M.M., and D.J. Nanan. 2008. International and Global Health. In *Maxcy-Rosenau-Last Public Health and Preventive Medicine*, 15th ed., edited by R.B. Wallace. New York: McGraw-Hill Medical, 1251–1258.
- Wilson, S.R., K.R. Solomon, and X. Tang. 2007. Changes in Tropospheric Composition and Air Quality Due to Stratospheric Ozone Depletion and Climate Change. *Photochemical and Photobiological Sciences* 6(3): 301–310.
- Woodruff, R.E., C.S. Guest, M.G. Garner, N. Becker, and M. Lindsay. 2006. Early Warning of Ross River Virus Epidemics: Combining Surveillance Data on Climate and Mosquitoes. *Epidemiology* 17(5): 569–575.
- World Health Organization. 1948. *Constitution of the World Health Organization*. Geneva: World Health Organization.
- ———. 2006. Air Quality Guidelines: Global Update 2005—Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide. Copenhagen: World Health Organization.
- ———. 2008. *Global Tuberculosis Control 2008. Surveillance, Planning, Financing*. Geneva: World Health Organization.
- ——. No date. Global Burden of Disease Project.

 http://www.who.int/healthinfo/global_burden_disease/en/index.html (accessed April 9, 2009).

- Zanobetti, A., M. Wand, and J. Schwartz. 2000. Generalized Additive Distributed Lag Models: Quantifying Mortality Displacement. *Biostatistics* 1: 279–292.
- Zeger, S.L., F. Dominici, and J. Samet. 1999. Harvesting-Resistant Estimates of Air Pollution Effects on Mortality. *Epidemiology* 10(2): 171–175.

THE REF I SAMET