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# Human Population Health

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# EXECUTIVE SUMMARY

The sustained health of human populations requires the continued integrity of Earth's natural systems. The disturbance, by climate change, of physical systems (e.g., weather patterns, sea-level, water supplies) and of ecosystems (e.g., agroecosystems, disease-vector habitats) would therefore pose risks to human health. The scale of the anticipated health impacts is that of whole communities or populations (i.e., it is a public health, not a personal health, issue). These health impacts would occur in various ways, via pathways of varying directness and complexity, including disturbance of natural and managed ecosystems. With some exceptions, relatively little research has yet been done that enables quantitative description of these probable health impacts.

It is anticipated that most of the impacts would be adverse. Some would occur via relatively direct pathways (e.g., deaths from heat waves and from extreme weather events); others would occur via indirect pathways (e.g., changes in the range of vector-borne diseases). Some impacts would be deferred in time and would occur on a larger scale than most other environmental health impacts with which we are familiar. If long-term climate change ensues, indirect impacts probably would predominate.

Populations with different levels of natural, technical, and social resources would differ in their vulnerability to climate-induced health impacts. Such vulnerability, due to crowding, food insecurity, local environmental degradation, and perturbed ecosystems, already exists in many communities in developing countries. Hence, because of both the geography of climate change and these variations in population vulnerability, climate change would impinge differently on different populations.

- An increased frequency or severity of heat waves would cause an increase in (predominantly cardiorespiratory) mortality and illness (High Confidence). Studies in selected urban populations in North America, North Africa, and East Asia indicate that the number of heat-related deaths would increase severalfold in response to two general circulation model (GCM)–modeled climate change scenarios for 2050. In very large cities, this would represent several thousand extra deaths annually. Although this heat-related increase in deaths would be partially offset by fewer cold-related deaths, there are insufficient data to quantify this tradeoff; further, this balance would vary by location and according to adaptive responses (Medium Confidence).
- If extreme weather events (droughts, floods, storms, etc.) were to occur more often, increases in rates of death, injury, infectious diseases, and psychological disorders would result (High Confidence).
- Net climate change-related increases in the geographic distribution (altitude and latitude) of the vector organisms of infectious diseases (e.g., malarial mosquitoes, schistosome-spreading snails) and changes in the life-cycle dynamics of both vector and infective parasites would, in aggregate, increase the potential transmission of many vector-borne diseases (High Confidence). Malaria, of which there are currently around 350 million new cases per year (including two million deaths), provides a central example. Simulations with first-generation mathematical models (based on standard climate-change scenarios and incorporating information about the basic dynamics of climatic influences on malaria transmission) predict an increase in malaria incidence in Indonesia by 2070 and—with a highly aggregated model—an increase from around 45% to around 60% in the proportion of the world population living within the *potential* malaria transmission zone by the latter half of the next century. Although this predicted increase in *potential* transmission encroaches mostly into temperate regions, actual climate-related increases in malaria incidence (estimated by one model to be of the order of 50–80 million additional cases annually, relative to an assumed global background total of 500 million by 2100) would occur primarily in tropical, subtropical, and less well protected temperate-zone populations currently at the margins of endemically infected areas. Some localized decreases may also occur (Medium Confidence).
- Increases in non-vector-borne infectious diseases such as cholera, salmonellosis, and other food- and water-related infections also could occur, particularly in tropical and subtropical regions, because of climatic impacts on water distribution, temperature, and microorganism proliferation (Medium confidence).
- The effects of climate change on agricultural, animal, and fisheries productivity, while still uncertain, could increase the prevalence of malnutrition and hunger and their long-term health impairments, especially in children. This would most probably occur regionally, with some regions likely to experience gains, and others losses, in food production (Medium Confidence).
- There would also be many health impacts of the physical, social, and demographic disruptions caused by

rising sea levels and by climate-related shortages in natural resources (especially fresh water) (Medium Confidence).

- Because fossil-fuel combustion produces both carbon dioxide and various primary air pollutants, the climate change process would be associated with increased levels of urban air pollution. Not only is air pollution itself an important health hazard, but hotter temperatures, in urban environments, would enhance both the formation of secondary pollutants (e.g., ozone) and the health impact of certain air pollutants. There would be increases in the frequency of allergic disorders and of cardiorespiratory disorders and deaths caused by various air pollutants (e.g., ozone and particulates) (High Confidence).
- A potentially important category of health impact would result from the deterioration in social and economic circumstances that might arise from adverse impacts of climate change on patterns of employment, wealth distribution, and population mobility and settlement. Conflicts might arise over dwindling environmental resources (Medium Confidence).
- Stratospheric ozone is being depleted concurrently with greenhouse gas accumulation in the troposphere. Although there are some shared and interactive atmospheric processes between disturbances of the stratosphere and troposphere, both they and their health impacts arise via quite distinct pathways. A sustained 10–15% depletion of stratospheric ozone over several decades would cause increased exposure to ultraviolet radiation and an estimated 15–20% increase in the incidence of skin cancer in fair-skinned populations (High Confidence). Lesions of the eye (e.g., cataracts) also may increase in frequency, as might vulnerability to some infectious diseases via adverse effects on immune function (Medium Confidence).

Adaptive options to minimize health impacts include improved and extended medical care services; environmental management; disaster preparedness; protective technology (housing, air conditioning, water purification, vaccination, etc.); public education directed at personal behaviors; and appropriate professional and research training. It also will be important to assess in advance any risks to health from proposed technological adaptations (e.g., exposures that could result from using certain alternative energy sources and replacement chemicals for chlorofluorocarbons; effects of pesticide use on resistance of vector organisms and their predator populations).

There is immediate need for improved and internationalized monitoring of health-risk indicators in relation to climate change. Existing global monitoring activities should encompass health-related environmental and bioindicator-species measurements and, where appropriate, direct measures of human population health status. To assist the evolution of public understanding and social policy, the health sciences must develop improved methods, including integrated predictive models, to better assess how climate change (and other global environmental changes) would influence human health.

In conclusion, the impacts of global climate change, particularly if sustained in the longer term, could include a multitude of serious—but thus far underrecognized—impacts on human health. Human population health is an outcome that integrates many other inputs, and it depends substantially on the stability and productivity of many of Earth's natural systems. Therefore, human health is likely to be predominantly adversely affected by climate change and its effects upon those systems.

## 18.1. Introduction

### *18.1.1. Climate Change and Human Population Health: The Nature of the Relationship*

Global climate change over the coming decades would have various effects upon the health of human populations (WHO, 1990; Haines and Fuchs, 1991; McMichael, 1993; Last, 1993; *Lancet*, 1994). Because of the nature of the exposures involved, the scale of these climate-related changes would, in general, apply to whole populations or communities, rather than to small groups or individuals. The assessment of health impacts therefore focuses on changes in rates of death or disease in populations.

Many of the health impacts of climate change would occur via processes that are relatively unfamiliar to public-health science. They would not occur via the familiar toxicological mechanisms of localized exposure to environmental contaminants, nor via locally determined influences on the spread of infectious diseases. Instead, many of the impacts would arise via the indirect and often delayed effects of disturbances to natural systems and their associated ecological relationships. For example, changes in background climate may alter the abundance, distribution, and behavior of mosquitoes and the life cycle of the malarial parasite, such that patterns of malaria would change. Climate change also would have varied regional effects on agricultural productivity, so that some vulnerable populations may experience nutritional deprivation. There also would be some rather more readily predictable health impacts, arising, for example, from more frequent or severe heatwaves.

On a wider canvas, several of the world's ecosystems that are important in sustaining human health already have been weakened by damage, habitat loss, and species/genetic depletion. These include agricultural lands and ocean fisheries and the terrestrial ecosystems that influence the transmission of infectious diseases. Climate change may, via various processes, exacerbate those ecosystem disturbances. Because an ecosystem comprises a suite of interacting components, in which member organisms relate to the whole suite rather than to individual parts, the uncoupling of relationships by climate change could initiate a cascade of disturbances that might jeopardize human population health.

The range of potential major types of health impact is shown in Figure 18-1. For simplicity, they have been classified as "direct" and "indirect," according to whether they occur predominantly via the direct impact of a climate variable (temperature, weather variability, etc.) upon the human organism or are mediated by climate-induced changes in complex biological and geochemical systems or by climatic influences on other environmental health hazards.

### *18.1.2. Forecasting Health Impacts: The Challenge to Health Science*

Predictions of future trends in population health are readily made in relation to actual current exposures—for example, future lung cancer rates can be predicted as a function of a population's current cigarette smoking habits (Peto *et al.*, 1994). It is unusual to make predictions on the basis of some anticipated future profile of exposure (e.g., smoking habits in the year 2020), yet this is the nature of the present exercise: Potential health impacts are being assessed in relation to future scenarios of climate change. There are inevitable, multiple uncertainties in such an approach (McMichael and Martens, 1995).

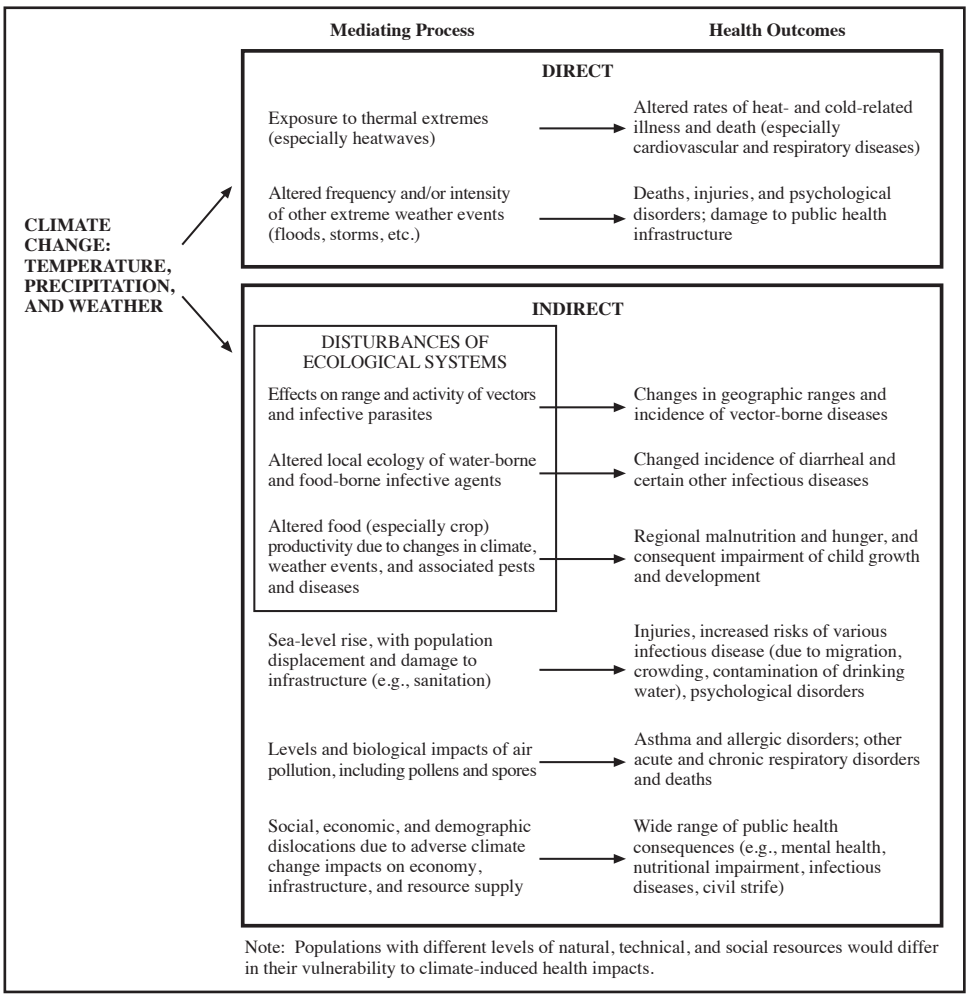


**Figure 18-1:** Ways in which climate change can affect human health.

Some aspects of climate change and its first-level impacts (on sea level, coastal ecosystems, forests, agriculture, fisheries, etc.), as projected by IPCC Working Group I, would lie outside the range of recorded human health experience. Hence, the forecasting of human health impacts must rely principally upon reference to historical analogy (where available) and reasonable extrapolation, judgment, and the use of integrated mathematical models (Niessen and Rotmans, 1993; McMichael and Martens, 1995). Most of the health impact modeling to date has been at a highly aggregated global or regional level, with no capacity to make finer-grained predictions. Ongoing developments in modeling techniques include the incorporation of complex nonlinear relationships and feedback processes, dealing with uncertainty, and richer use of local detail (Alcamo *et al*., 1994a, 1994b).

Some health impacts can be forecast by relatively simple extrapolation from empirical epidemiological dose-response data. For example, models to predict the mortality impacts of an increase in heatwaves can be based on existing empirical data. However, predicting the health impact of climate-induced shifts in ecological relationships and habitat boundaries (e.g., malarial mosquitoes, agricultural crops) poses a more complex challenge. Equally complex is the task of predicting the various indirect health impacts of such things as sea-level rise or of civil disruption and enforced migration because of deteriorating environments and dwindling resources.

A further difficulty arises with regional predictions. Not only is the ability to predict regional differences in climate change still limited, but human populations differ greatly in their environmental circumstances, social resources, and preexisting health status. They therefore differ in their vulnerability to climate-induced stresses. Although there is generally insufficient information available to make differentiated assessments of health impacts in different populations, the balance of the assessments published to date is that, because of the geography of the impacts of climate change (particularly in relation to infectious disease transmission and food production) and of population vulnerability, many of the anticipated adverse health impacts would be greater in the world's less-developed regions. Nevertheless, in developed countries, demographic trends including population aging and increasing levels of disability, chronic illness, and coastal retirement may increase the vulnerability of populations.

Although it is tempting to compile a list of discrete health impacts, it is important that the systemic quality of the impact of climate change be understood. There would be many cross-links, including interactions of climate change with other, coexisting environmental changes. Further, the attempts of society to mitigate the health effects of climate change may actually exacerbate some of them. For example, increased use of fertilizers to compensate for a decline in local agricultural production can increase algal blooms and hence the risk of cholera or shellfish poisoning; or the relocation of populations from drought-stricken areas might introduce them to unfamiliar pathogens (or introduce their unfamiliar pathogens). Neither the scope of this chapter nor available scientific knowledge allows comprehensive consideration of these issues.

### *18.1.3. Sensitivities and Thresholds*

Forecasting health impacts requires knowledge of, first, the sensitivity of change in population health status in response to climate change and, second, of any associated thresholds. "Sensitivity" refers to the rate of change in health outcome per unit change in climate (however defined), whereas "threshold" refers to a sudden change in slope or curvature in that dose-response graph (e.g., a certain amount of climate change may be tolerated before an impact on some particular health outcome occurs).

Although there is only limited information on the sensitivities and thresholds of human health response to climate change, some illustrative comments can be made:

- The impact of thermal stress depends on physiological tolerance thresholds being exceeded (see Section 18.2.1). For example, most studies of heat-related mortality show that increased mortality occurs only after a critical temperature has been exceeded for a certain duration. This critical temperature varies geographically, reflecting socioeconomic differences, physiological acclimatization, and cultural-technical adaptation.
- Infectious disease pathogens and insect pests are normally constrained by bioclimatic thresholds. That is, there is a range of climatic conditions with upper and lower thresholds within which a population of organisms is viable (Dobson and Carper, 1993). These thresholds account for seasonal and longer-term fluctuations in the distribution and abundance of most organisms. The uncoupling, by climate change, of previously stable ecological relationships between species would reveal other thresholds as population imbalances pass critical points.
- Changes in the distribution of organisms (e.g., mosquitoes) that spread vector-borne diseases (e.g., malaria) would occur if climate change causes their geographic range to shift. This shift would reflect the critical thresholds of temperature, precipitation, and humidity (i.e., the bioclimatograph) for vector maturation and persistence. For example, the range of *Plasmodium vivax* malaria is limited because the parasite cannot develop inside its mosquito host at temperatures below 14–16°C (Gilles, 1993). Further, blood-feeding arthropods feed and reproduce only above certain temperatures and need less time to complete their life cycle as temperatures increase above that threshold (Curto de Casas and Carcavallo, 1984; Burgos *et al*., 1994). Threshold effects also apply to the life cycle of the infecting parasite.
- Some animal and plant populations would be unable to migrate or adapt behaviorally to climate change, in part because of constraints imposed by non-climate

variables such as day-length and soil type. Other species with either migratory or dispersive activity (particularly arthropod and weed pests in agriculture and disease vectors that affect human health) would cope with the shifts in climate zones. Humans generally would be less sensitive to changes in background climate because of their capacity to adapt via culture, technology, migration, and behavior.

- The sensitivity of health-outcome response depends on population susceptibility. For example, the impact of a climate-related increase in exposure to infectious agents would depend on prior contact (i.e., herd immunity), on general biological resilience (especially nutritional and immune status), and on population density and patterns of interpersonal contact. Social infrastructure and health-care resources also would condition the impact. In general, the most vulnerable populations or communities would be those living in poverty, with a high prevalence of undernutrition, chronic exposure to infectious disease agents, and inadequate access to social and physical infrastructure.

Table 18-1 lists illustrative examples of the relative sensitivity of selected human health responses to key aspects of climate change. It also summarizes what is known about thresholds in those responses. Further, the "conditionality" of response (i.e., the extent to which it is modulated by other influences) is indicated. This latter criterion is important: Many factors (including the intrinsic vulnerability of the local population) influence the determination of health status, and many of these would condition the health impact of climate change.

Further, many of the impacts of climate change would depend on parameters other than the central changes in mean values. For example, weather variability would be important for extreme events; rates of change would influence the production in ecosystems (e.g., agriculture and fisheries); and the ecology of infectious disease vectors is sensitive to many aspects of climate, including changes in the day-night differential in temperature. These aspects of climate change would vary in their relative importance for different health impacts, as illustrated in Table 18-2. However, information on such details is still rather incomplete.

### *18.1.4. Major Trends in World Health: Backdrop to Climate Change Impacts*

Information about levels and time trends in specific health outcomes facilitates both the prediction and the appraisal of the health impacts of climate change. For example, patterns of malaria and malnutrition are changing around the world in response to various other changes in social, biological, and ecological circumstances. Any predicted impact of climate change upon such health outcomes should be assessed either by differentiating that impact from those of other independent background trends or, if appropriate, by assessing its interactive impact with those other, coexisting influences.

The main contemporary features of world health (World Bank, 1993; Murray and Lopez, 1994) are as follows: (1) near-worldwide increases in life expectancy [with the ex-Eastern Bloc countries standing in sharp recent contrast (Feachem, 1994)], (2) a decline in infant and child mortality in most developing countries, (3) persistent gradients in health status between rich and poor (within and between populations), (4) reductions in certain vaccine-preventable diseases (e.g., polio and measles), (5) increases in the chronic noninfectious diseases of adult life

***Table 18-1:*** *Examples of important aspects of the relationship of selected health outcomes to climate change.*

| Health Outcome | Sensitivity[a] | Conditionality[b] | Thresholds |
|---|---|---|---|
| Climate Stress/Mortality | +++ | ++ | Temperature 33°C[c] |
| Climate Stress/Morbidity | + | | |
| Allergy | ++ | ++ | Not applicable |
| Asthma | + | + | Not applicable |
| Vector-Borne Diseases (malaria, yellow fever, dengue, onchocerciasis, encephalitis)[d] | ++(+) | ++++ | Temperature isotherms and humidity levels (bioclimatic functions) e.g., 10°C: *Ae*. mosquito, 14–16°C: *P. vivax* parasite |
| Other Infectious Diseases (e.g., cholera) | +(++) | | Temperature thresholds for algal and bacterial proliferation in sea- and freshwater—optimal ranges |

++++ = great effect, + = small effect, (+) = possible additional effect

[a] Extent of change in health outcome per unit change in climate (equivalent to "slope" of regression line).

[b] Extent to which sensitivity depends on preceding and coexistent circumstances (i.e., notions of vulnerability/susceptibility and interactive effects).

[c] Based principally on northeastern U.S. data. Critical temperature depends on local climate and population acclimatization.

[d] See also Table 18-3.

***Table 18-2:*** *Probable relative impact on health outcomes of the aspects of climate change.*

| Health Outcome | Aspects of Climate Change: Change in Mean Temperature | Extreme Events | Rate of Change of Climate Variable | Day-Night Difference |
|---|---|---|---|---|
| Heat-Related Deaths and Illness | | +++ | | + |
| Physical and Psychological Trauma due to Disasters | | ++++ | | |
| Vector-Borne Diseases | +++ | ++ | + | ++ |
| Other Infectious Diseases | + | + | | |
| Food Availability and Hunger | ++ | + | ++ | |
| Consequences of Sea-Level Rise | ++ | ++ | + | |
| Respiratory Effects | | | | |
| – Air Pollutants | + | ++ | | + |
| – Pollens, Humidity | ++ | | | |
| Demographic Disruption | ++ | + | + | |

Notes: ++++ = great effect, + = small effect; empty cells indicate no known relationship.

(especially heart disease, diabetes, and certain cancers) in urban middle classes in rapidly developing countries, and (6) widespread increases in HIV infection. Rates of disease and death from cigarette smoking are likely to escalate markedly in many countries over the coming decades, as the tobacco industry takes advantage of freer trade and market-based economies (Peto *et al*., 1994). In many urban populations, drug abuse and violence are increasing.

There appears to be a widespread increase in the tempo of new and resurgent infectious diseases (Levins *et al*., 1994). This primarily reflects the combination of environmental and demographic changes in the world, plus increases in antibiotic and drug resistance, pesticide resistance, and decreased surveillance (Morse, 1991; CDC, 1994a). The interaction of local climate change with other disruptions of ecosystems may have facilitated various infectious disease outbreaks: the emergence of rodent-borne hantavirus pulmonary syndrome in the United States during 1992–3; various rodent-borne arenaviruses in Africa and South America; the spread of harmful algal blooms—and its association with cholera (which is now affecting more nations worldwide than at any earlier time this century); the rapid resurgence of dengue in the Americas since 1981; and the occurrence of dengue and malaria at higher altitudes than previously recorded (Levins *et al*., 1994).

Many other important influences on population health are changing over time. For example, new vaccines are being developed, and existing ones are being used more widely; contraception is becoming more widely used, with benefits to maternal and child health; and safe drinking water is becoming available, albeit slowly, to an increasing proportion of householders in poorer countries. Other adverse effects (from cigarettes, drugs, urban traffic, social breakdown, violence, etc.) are increasing widely, and persistent widespread poverty remains a major structural impediment to improved health (WHO, 1995a). Against this complex balance sheet, it is inevitably difficult to estimate the likely net impact on population health status after the additional inclusion of climate change.

## 18.2. Potential Direct Health Impacts of Climate Change

The direct effects of climate change upon health result from changes in climate characteristics or short-term weather extremes that impinge directly on human biology. The following subsections deal with the health impacts of thermal stress and extreme weather events.

### *18.2.1. Health Impacts of Altered Patterns of Thermal Stress*

Many studies, particularly in temperate countries, have observed a J-shaped relationship (often more generally referred to as a U-shaped relationship) between daily outdoor temperature and daily death rate: Mortality is lowest within an intermediate comfortable temperature range. The graph is not symmetrical; the death rate increases much more steeply with rising temperatures, above this comfort zone, than it does with falling temperatures below that zone (Longstreth, 1989; Kalkstein, 1993; Kunst *et al*., 1993; Touloumi *et al*., 1994). Relatedly, death rates in temperate countries appear to be affected across a wider band of decreasing, cold, temperatures than that for increasing, hot, temperatures (Kilbourne, 1992; Kunst *et al*., 1993). Because death rates in temperate and subtropical zones are higher in winter than in summer (Kilbourne, 1992), it is a reasonable expectation that milder winters in such countries would entail a reduction in cold-related deaths and illnesses. However, since summer-related deaths appear to be more related to temperature extremes than are winter-related deaths, this reduction may not fully offset the heat-related increases. Further, this balance of gains and losses would vary among geographic regions and different populations. The issue of balance is examined further later in this section.

Quantitative interpretation of the impacts of altered daily-temperature distribution also is hampered by the fact that, for both heat-related and cold-related deaths, many of the apparent excess deaths occur in already-vulnerable persons (especially the elderly and the sick). Some analyses indicate that, in the absence of extreme temperatures, many of those persons would

have died in the near future. This "mortality displacement" issue also will be considered further.

Global warming is predicted to increase the frequency of very hot days (see Chapter 6, *Climate Models–Projections of Future Climate*, of the IPCC Working Group I volume). The frequency of such days in temperate climates (e.g., USA, UK, Australia) would approximately double for an increase of 2–3°C in the average summer temperature (e.g., CDC, 1989; Climate Change Impacts Review Group UK, 1991). Extensive research has shown that heat waves cause excess deaths (Weihe, 1986; Kilbourne, 1992). Recent analyses of concurrent meteorological and mortality data in cities in the United States, Canada, the Netherlands, China, and the Middle East provide confirmatory evidence that overall death rates rise during heat waves (Kalkstein and Smoyer, 1993; Kunst *et al*., 1993), particularly when the temperature rises above the local population's threshold value. Therefore, it can be predicted confidently that climate change would, via increased exposure to heat waves, cause additional heat-related deaths and illnesses (Kalkstein, 1993; Haines *et al*., 1993).

The effect of extreme heat on mortality is exacerbated by low wind, high humidity, and intense solar radiation (Kilbourne, 1992). Indeed, these meteorological elements can be treated synoptically, to evaluate the net impact of weather on human health. For example, recent studies in the United States have described "oppressive" air masses (analogous to the meteorologist's "stagnating" air masses), which represent synoptic meteorological situations that exceed physiological tolerance levels. This approach recognizes that humans principally respond to the umbrella of air that surrounds them rather than to individual meteorological elements (Kalkstein, 1993). It also should be noted that concurrent hot weather and air pollution have interactive impacts on health (Katsouyanni *et al.,* 1993; see also Section 18.3.5).

Healthy persons have efficient heat regulatory mechanisms that cope with increases in temperature up to a particular threshold. The body can increase radiant, convective, and evaporative heat loss by vasodilation (enlargement of blood vessels in the skin) and perspiration (Horowitz and Samueloff, 1987; Diamond, 1991). Further, some acclimatization to persistent oppressive weather conditions can occur within several days (Kilbourne, 1992). Nevertheless, the risk of death increases substantially when thermal stress persists for several consecutive days (Kalkstein and Smoyer, 1993). The elderly and very young are disproportionately affected because of their limited physiological capacity to adapt. Although some individuals die from heat exhaustion or heatstroke, the deaths associated with very hot weather are predominantly associated with preexisting cardiovascular and respiratory disorders, as well as accidents (Larsen, 1990a, 1990b). Although there is less evidence on nonfatal illness episodes, it is a reasonable general assumption that thermal stress also increases rates of morbidity.

Socioeconomic factors may have important modulating effects on thermal stress-related mortality. From studies in the United States, the extent of protection from air conditioning remains unclear (Ellis and Nelson, 1978; Larsen, 1990a; Rogot *et al*., 1992; Kalkstein, 1993). Of more general importance, people living in poverty, including segments of many urban populations in developing countries, are particularly vulnerable to heat stress. Poor housing, the urban heat island effect, and lack of air conditioning are among the primary causes (Kilbourne, 1989). Complete acclimatization may take up to several years (Babeyev, 1986), rendering immigrants (e.g., rural-to-urban) vulnerable to weather extremes for a considerable time. Ongoing rapid increases in urbanization (see Chapter 12) will increase the number of vulnerable persons.

The question in relation to the abovementioned notion of "mortality displacement" is: Would some of those who die during heat waves have succumbed soon afterward from preexisting frailty or disease (Kalkstein, 1993; U.S. EPA, in press)? Various time-series analyses indicate a "deficit" in daily deaths for up to a month after heat waves (e.g., Figure 18-2), and U.S.-based research suggests that 20–40% of the deaths occurring during heat waves would have occurred within the next few weeks (Kalkstein, 1993). A related uncertainty is whether, as the frequency of heat waves increases, the mortality excess remains constant or whether, as some research suggests, successive heat waves entail a progressive lessening of the associated mortality peak.

Kalkstein (1993) made predictions of heat-related mortality for selected urban populations in North America, North Africa, and East Asia in relation to IPCC-specified climate-change scenarios. This entailed, first, identifying for each population

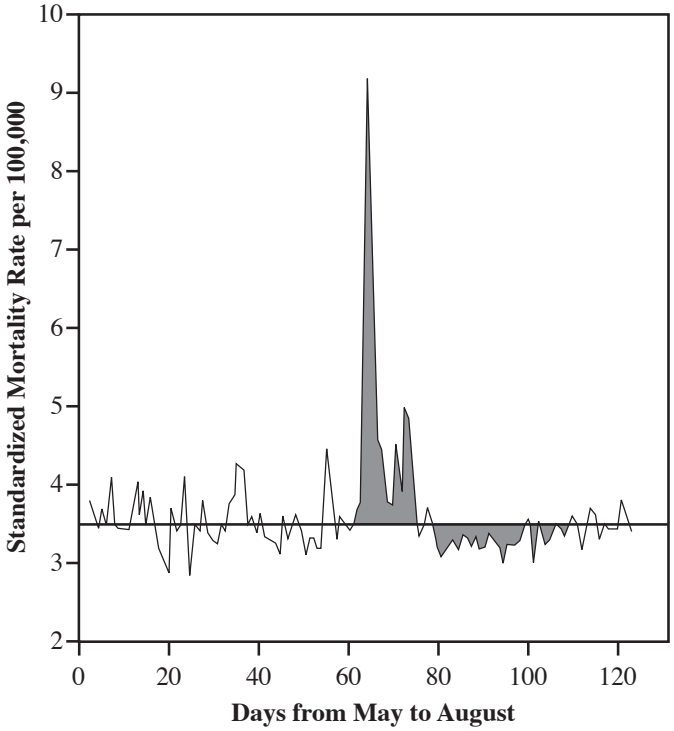


**Figure 18-2:** Daily summer mortality during a New York heatwave, 1966 (Kalkstein, 1993).

setting the synoptic weather situations that are "oppressive" (i.e., exceed physiological tolerance levels). The annual number of oppressive synoptic situation days was then predicted for the climate-change scenarios, and the annual total of associated excess deaths was estimated. Model-based predictions of the numbers of additional deaths attributable to heat during a typical summer in a future warmer world were thus obtained. Two sets of predicted mortality increases were made: one assuming that the population does not acclimatize and the other assuming partial acclimatization (i.e., complete physiological acclimatization but without improved socioeconomic conditions such as the development of more protective housing).

Consider the population of Atlanta, for example. Presently, Atlanta experiences an average of 78 heat-related deaths each summer. Under the climate projections of the Geophysical Fluid Dynamics Laboratory (GFDL) 1989 (transient) climate-change model, and assuming no change in population size or age profile, this number would increase to 191 in the year 2020 and to 293 in the year 2050. If population acclimatization occurs, the annual total would increase less, to 96 and 147 in those two years. Under the UKTR (transient) model run, the corresponding four projections of heat-related mortality are 20–40% higher than for the GFDL model run: 247 and 436 deaths (unacclimatized, 2020 and 2050) and 124 and 218 deaths (acclimatized, 2020 and 2050). These and other results for selected North American cities, Shanghai, and Cairo indicate that the annual number of heat-related deaths would, very approximately, double by 2020 and would increase several-fold by 2050. Thus, in very large cities with populations displaying this type of sensitivity to heat stress, climate change would cause several thousand extra heat-related deaths annually.

As mentioned earlier, the seasonal death rates in developed countries are highest in winter (Kilbourne, 1992; Tan, 1994). In temperate-zone countries, death rates increase particularly during periods of severe winter weather. However, no single study has yet been published that allows a direct comparison of the anticipated winter gains and summer losses that would accompany global warming. A substantial proportion of winter-related deaths are from cardiovascular disease (Kunst *et al*., 1993; Langford and Bentham, 1995). It is likely that this increased risk of cardiovascular disease reflects an increased cold-induced tendency for blood to clot (Keatinge *et al*., 1989), perhaps exacerbated by the fibrinogen-enhancing effect of winter respiratory infections (Woodhouse *et al*., 1994). The relative importance of respiratory infections and cardiovascular diseases to anticipated reductions in mortality, and how these would vary geographically, remains uncertain. One recent British study has forecast that approximately 9,000 fewer winter-related deaths (estimated to represent a reduction of 2–3%) would occur annually by the year 2050 in England and Wales, under typical climate-change scenarios that entail 2–2.5°C wintertime increases (Langford and Bentham, 1995). Just over half the avoided deaths would be from ischaemic heart disease and stroke, with chronic bronchitis and pneumonia each contributing 5–10%. Other researchers have concluded that a significant portion of the overall winter-related mortality is due to respiratory infections such as influenza (Curwen, 1991). Since these respiratory infections depend upon aerosol transmission—usually in confined, poorly ventilated places—a small rise in winter temperatures should reduce this risk if it encouraged outdoor activities and improved ventilation. However, annual influenza outbreaks do not appear to correlate with mean winter or monthly temperature (CDC, 1994b; Langford and Bentham, 1995).

Overall, the sensitivity of death rates to hotter summers is likely to be greater than to the accompanying increase in average winter temperature. The overall balance is difficult to quantify, and also would depend on the population's capacity for adaptive responses. However, research to date suggests that global warming would, via an increased frequency of heat waves, cause a net increase in mortality and associated morbidity. This conclusion must be qualified by noting that there is an imbalance in the published research—most of which refers to developed, non-tropical, countries.

### *18.2.2. Health Impacts of Weather Variability and Extreme Events*

Global warming may affect ocean currents, air currents, and atmospheric humidity (see Chapter 6, *Climate Models–Projections of Future Climate*, of the IPCC Working Group I volume). Any consequent changes in weather variability may alter the frequency and severity of extreme weather events: bushfires, droughts, floods, storms, and landslides (Gordon *et al*., 1992; Meehl *et al*., 1992). However, some of these relationships remain uncertain (e.g., for tropical cyclones; see Lighthill *et al*., 1994) and have proven difficult to model with GCMs. Such events increase deaths, injuries, infectious diseases, stress-related disorders, and the many adverse health effects associated with social disruption, environmentally enforced migration (Myers, 1993), and settlement in urban slums. Health impacts would be greatest on those communities that are most exposed and have the fewest technical and social resources.

Low-lying, poorly resourced populations would be particularly vulnerable to an increase in frequency of storms and storm surges. In the 1970 Bangladesh cyclone, mortality varied from around 5% inland to almost 50% in coastal communities. Widespread destruction of food supplies may also occur; in 1970, two-thirds of fishing activities along the coasts and plains in Bangladesh were destroyed, along with 125,000 animals (Alexander, 1993). In Andhra Pradhesh, India, in 1970, many victims died when wind and rain caused the collapse of houses (Sommer and Mosely, 1972).

Flash floods and landslides (see Chapter 12) would increase in frequency in regions experiencing increased torrential rainfall. Heavy rains can erode soil, thereby impairing agricultural productivity. Flooding also can affect the incidence of vector-borne diseases. On the one hand, flood waters may wash away mosquito eggs/larvae; on the other hand, residual water may increase mosquito populations and consequent infectious diseases. In southeastern Australia, epidemics of Ross River virus

infection follow heavy rains in the Murray-Darling basin (Nicholls, 1993). Flooding also may affect the transmission of diarrheal diseases: In Bolivia, for example, flooding associated with El Niño in 1983 led to a 70% increase in salmonella infections, particularly in children (Telleria, 1986).

Flash flooding is a leading cause of weather-related mortality in the United States (French and Holt, 1989). Damage to homes and displacement of residents may facilitate the spread of infectious diseases because of crowded living conditions. Flooding can contaminate water sources with fecal material or toxic chemicals. Flooding also increases runoff from agricultural lands and urban stormwater systems (Thurman *et al*., 1991, 1992). Water resources thus are contaminated by toxic chemical wastes, agricultural chemicals, and pathogens (e.g., *Cryptosporidium*). Hazardous exposure may arise from contaminated drinking water and from edible fish that bioaccumulate contaminants.

The many psychosocial effects of natural disasters have been studied in survivors of storms, floods, earthquakes, and fires (e.g., Gregg, 1989). Severe disturbance (including "post-traumatic stress disorder") usually affects only a minority, many of whom recover (de Girolamo and McFarlane, 1994)—although children in affected families may show developmental problems over the ensuing years (Titchener and Frederic, 1976). The level of psychological impact may depend upon the suddenness and unexpectedness of the impact, the intensity of the experience, the degree of personal and community disruption, and long-term exposure to the visual signs of the disaster (Green, 1982; Green *et al*., 1991). Data from disasters in developing countries are sparse, but a follow-up study after the disastrous 1988 floods in Bangladesh showed increased behavioral disorders in young children (Durkin *et al*., 1993).

Two final comments on extreme events are appropriate here. First, a major effect of extreme climatic events on human health has been malnutrition and starvation due to severe drought and its consequences (Escudero, 1985). This, in turn, causes increased susceptibility to infection (Tomkins, 1986). Second, there is evidence that environmental degradation, unequal access to resources, and population growth may be potent factors in provoking conflict (Homer-Dixon *et al*., 1993). By affecting water and food supply, climate change could increase the possibility of violent conflicts in a number of regions.

## 18.3. Potential Indirect Health Impacts of Climate Change

The indirect effects of climate change upon health are those that do not entail a direct causal connection between a climatic factor (such as heat, humidity, or extreme weather event) and human biology.

### *18.3.1. Vector-Borne Diseases*

The transmission of many infectious diseases is affected by climatic factors. Infective agents and their vector organisms are sensitive to factors such as temperature, surface water, humidity, wind, soil moisture, and changes in forest distribution (Bradley, 1993). This applies particularly to vector-borne diseases (VBDs) like malaria, which require an intermediate organism such as the mosquito to transmit the infective agent. It is therefore projected that climate change and altered weather patterns would affect the range (both latitude and altitude), intensity, and seasonality of many vector-borne and other infectious diseases. In general, increased warmth and moisture would enhance transmission of these diseases. However, any such climate-related redistribution of disease may also entail—perhaps in conjunction with other environmental stresses—some localized reductions in rates of infection.

The sustained (or endemic) transmission of VBDs requires favorable climatic-environmental conditions for the vector, the parasite, and, if applicable, the intermediate host species. For vectors with long life spans (e.g., tsetse flies, bugs, ticks), vector abundance is a major determinant of disease distribution; arthropods that transmit VBDs generally thrive in warmth and moisture. For vectors with short life spans (e.g., mosquitoes, sandflies, blackflies), the temperature-sensitive extrinsic maturation period of the parasite is of critical importance. The geographic distributions of many of the parasites, both unicellular (protozoa) and multicellular (e.g., flukes and worms), correlate closely with temperature (Gillet, 1974; Shope, 1991). The geographic distributions of vector-borne viral infections, such as dengue and yellow fever, are affected by temperature and surface water distribution. For many VBDs, such as plague and hantavirus, rodents act as intermediate infected hosts or as hosts for the arthropod vector (Wenzel, 1994); rodent activity would also tend to increase in a warmer world (Shope, 1991).

In tropical countries, VBDs are a major cause of illness and death. For the major VBDs, estimates of numbers of people at risk and infected, and of VBD sensitivity to climate change, are shown in Table 18-3. While the potential transmission of many such diseases would increase (geographically or from seasonal to year-round) in response to climate change, the capacity to control these diseases also will change. New or improved vaccines can be expected; some vector species can be constrained by use of pesticides. Nevertheless, there are uncertainties and risks here, too: for example, long-term pesticide use breeds resistant strains and kills many predators of pests.

#### *18.3.1.1. Malaria*

Malaria remains a huge global public-health problem, currently causing around 350 million new infections annually, predominantly in tropical countries. Malaria is caused by an infective parasite (plasmodium) transmitted between humans by a mosquito vector. Its incidence is affected by temperature, surface water, and humidity (Gill, 1920a, 1920b; Sutherst, 1983). Although anopheline mosquito species that transmit malaria do not usually survive where the mean winter temperature drops below 16–18°C, some higher-latitude species are able to hibernate in sheltered sites. Sporogonic development (i.e., the extrinsic incubation phase of

***Table 18-3:*** *Major tropical vector-borne diseases and the likelihood of change of their distribution with climate change.*

| Disease | Vector | Population at Risk (million)[a] | Number of People Currently Infected or New Cases per Year | Present Distribution | Likelihood of Altered Distribution with Climate Change |
|---|---|---|---|---|---|
| Malaria | Mosquito | 2,400[b] | 300–500 million | Tropics/Subtropics | +++ |
| Schistosomiasis | Water Snail | 600 | 200 million | Tropics/Subtropics | ++ |
| Lymphatic Filariasis | Mosquito | 1,094[c] | 117 million | Tropics/Subtropics | + |
| African Trypanosomiasis (Sleeping Sickness) | Tsetse Fly | 55[d] | 250,000–300,000 cases per year | Tropical Africa | + |
| Dracunculiasis (Guinea Worm) | Crustacean (Copepod) | 100[e] | 100,000 per year | South Asia/ Arabian Peninsula/ Central-West Africa | ? |
| Leishmaniasis | Phlebotomine Sand Fly | 350 | 12 million infected, 500,000 new cases per year[f] | Asia/Southern Europe/Africa/ Americas | + |
| Onchocerciasis (River Blindness) | Black Fly | 123 | 17.5 million | Africa/Latin America | ++ |
| American Trypanosomiasis (Chagas' disease) | Triatomine Bug | 100[g] | 18 million | Central and South America | + |
| Dengue | Mosquito | 1,800 | 10–30 million per year | All Tropical Countries | ++ |
| Yellow Fever | Mosquito | 450 | <5,000 cases per year | Tropical South America and Africa | ++ |

+ = likely, ++ = very likely, +++ = highly likely, ? = unknown.
[a]Top three entries are population-prorated projections, based on 1989 estimates.
[b]WHO, 1995b.
[c]Michael and Bundy, 1995.
[d]WHO, 1994a.
[e]Ranque, personal communication.
[f]Annual incidence of visceral leishmaniasis; annual incidence of cutaneous leishmaniasis is 1-1.5 million cases/yr (PAHO, 1994).
[g]WHO, 1995c.

the plasmodium within the mosquito) ceases below around 18°C for *Plasmodium falciparum* and below 14°C for *P. vivax*. Above those temperatures, a small increase in average temperature accelerates the parasite's extrinsic incubation (Miller and Warrell, 1990). Temperatures of 20–30°C and humidity above 60% are optimal for the anopheline mosquito to survive long enough to incubate and transmit the parasite.

Until recent decades, parts of today's developed world were malarious. These included the United States, southern Europe, and northern Australia. In the last century, outbreaks of *P. vivax* malaria occurred in Scandinavia and North America. Although climate change would increase the potential transmission of malaria in some temperate areas, the existing public-health resources in those countries—disease surveillance, surface-water management, and treatment of cases—would make reemergent malaria unlikely. Indeed, malaria is most likely to extend its spread (both latitude and altitude) and undergo changes in seasonality in tropical countries, particularly in populations currently at the fringe of established endemic areas (Martens *et al*., 1994). Newly affected populations would initially experience high case fatality rates because of their lack of natural acquired immunity.

**Box 18-1. Examples of Modeling the Future Impact of Climate Change upon Malaria**

Mathematical models have been recently developed for the quantitative prediction of climate-related changes in the potential transmission of malaria (e.g., Sutherst, 1993; Matsuoka and Kai, 1994; Martin and Lefebvre, 1995). Note the use of the important word "potential" here; the models are primarily predicting where malaria could occur as a function of climate and associated environment, irrespective of the influence of local demographic, socioeconomic, and technical circumstances.

A simple model has been used for Indonesia, based on empirical historical data from selected provinces on the relationship of annual average temperature and total rainfall to the incidence of malaria (and dengue and diarrhea) (Asian Development Bank, 1994). The model forecasts that annual malaria incidence (currently 2,705 per 10,000 persons) would, in response to the median climate-change scenario, increase marginally by 2010 and by approximately 25% by 2070. Because of the limited technical information, it is not easy to appraise these particular forecasts.

A more complex integrated global model has been developed by Martens *et al*. (1994). This model takes account of how climate change would affect the mosquito population directly—i.e., mosquito development, feeding frequency, and longevity—and the incubation period of the parasite inside the mosquito (see Figure 18-3). As a highly aggregated model, it does not take account of local environmental-ecological factors, and it therefore cannot be regarded as a source of precise projections. The model's output refers to geographic changes in *potential* transmission (i.e., the range within which both the mosquito and parasite could survive with sufficient abundance for sustained transmission of malaria). This is not, therefore, a projection of actual disease incidence, and it must be interpreted in relation to local control measures, health services, parasite reservoir, and mosquito densities. Further, until such models have been validated against historical data sets, their predictions must be viewed cautiously.

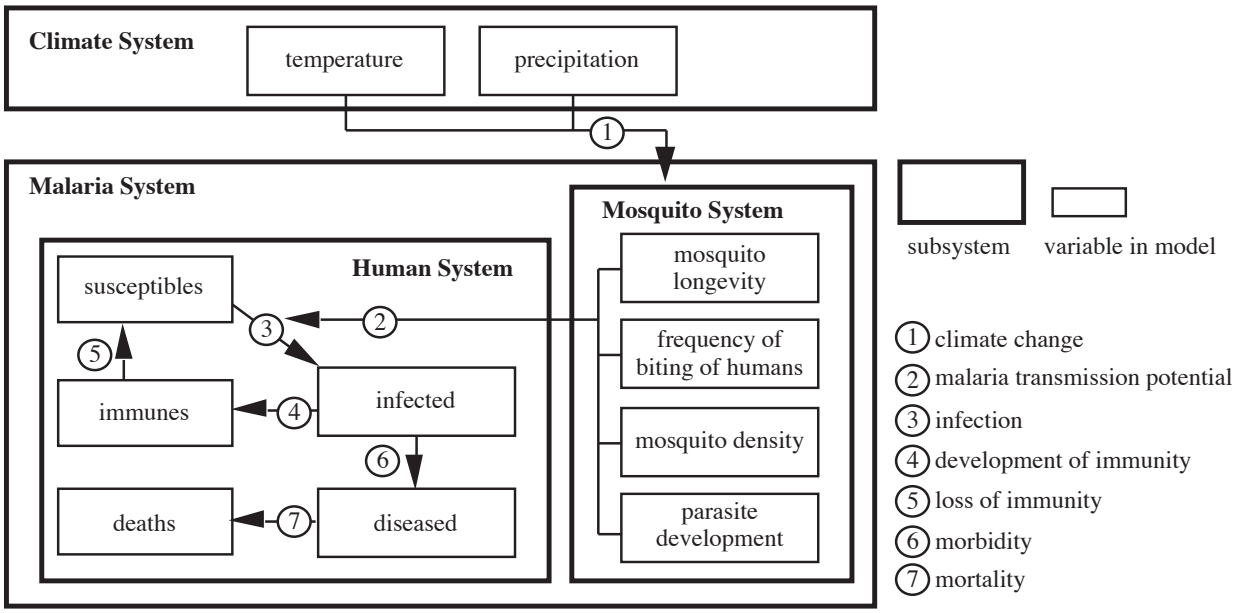


**Figure 18-3:** Systems diagram of a model designed to assess the impact of climate change on the potential transmission of malaria (adapted from Martens *et al*., 1994).

This particular model predicts widespread increases in the potential habitat range and the "vectorial capacity" of the mosquito, and therefore potential malaria transmission, in response to climate change. For example, using GCM predictions of 3–5°C increases in global mean temperature by the year 2100, the malaria epidemic potential of the mosquito population is estimated to increase twofold in tropical regions and substantially more than tenfold in temperate climates (Martens *et al*., 1995a). Overall, there is an increase from around 45% to around 60% of the world population living within the potential malaria transmission zone by the latter half of the next century. However, most of the developed countries have effective control and surveillance measures that should preclude reintroduction of endemic malaria. In the already endemic areas, especially in the subtropics, malaria may increase (although in some hot climates, further temperature increases may shorten the life span of mosquitoes, and local malaria transmission would then decrease). In particular, in adjoining areas of lower endemicity or unstable malaria, the occurrence of infection is far more sensitive

**Box 18-1 (continued)**

to climate variation, so climate change may have a marked effect on its incidence and stability. Simulations with this model, using several different GCMs, predict a climate-induced increase in the incidence of annual malaria cases of approximately 50–80 million in response to a temperature rise of around 3°C by the year 2100, relative to an assumed approximate base of 500 million annual cases in a 2100 world without climate change (Martens *et al*., 1995b).

Another recent attempt at aggregated global modeling (Martin and Lefebvre, 1995) has predicted that potential malaria transmission would spread to higher latitudes, while some currently stable endemic areas near the equator would become unstable, leading to reductions in population immunity levels.

Such models, despite their highly aggregated predictions and simplifying assumptions, provide indicative information about the likely impact of climate change on the potential transmission of vector-borne diseases (assuming that other relevant factors remain constant). There is a clear need for validation of these models and for incorporating more extensive detail into them. Meanwhile, this line of research has begun to elucidate the interdependent relationships among climate change, vector population dynamics, and human disease dynamics.

Recent evidence of the responsiveness of malaria incidence to local climate change comes from observations of marked increases in malaria incidence in Rwanda in 1987, when atypically hot and wet weather occurred (Loevinsohn, 1994), and annual fluctuations in falciparum malaria intensity in northeast Pakistan that correlated with annual temperature variations during the 1980s (Bouma *et al*., 1994). Hence, it is a reasonable prediction that, in eastern Africa, a relatively small increase in winter temperature could extend the mosquito habitat and thus enable falciparum malaria to reach beyond the usual altitude limit of around 2,500 m to the large, malaria-free, urban highland populations, e.g., Nairobi in Kenya and Harare in Zimbabwe. Indeed, the monitoring of such populations around the world, currently just beyond the boundaries of stable endemic malaria, could provide early evidence of climate-related shifts in malaria distribution (Haines *et al*., 1993).

#### *18.3.1.2. African Trypanosomiasis*

African trypanosomiasis, or "sleeping sickness," is transmitted by tsetse flies. The disease is a serious health problem in tropical Africa, being generally fatal if untreated. Research in Kenya and Tanzania shows only a very small difference in mean temperature between areas where the vector, *Glossina morsitans,* does and does not occur. This indicates that a small change in temperature may significantly affect the limits of the vector's distribution (Rogers and Packer, 1993).

#### *18.3.1.3. American Trypanosomiasis (Chagas' Disease)*

American trypanosomiasis is transmitted by insects of the subfamily *Triatominae*. It is a major problem in Latin America, with 100 million people at risk and 18 million infected (WHO, 1995c). An estimated 15–20% of infected people develop clinical Chagas' disease. Most of the triatomine vector species need a minimum temperature of 20°C for feeding and reproduction (Curto de Casas and Carcavallo, 1984), but at higher temperatures (28–30°C) they feed more frequently, have a shortened life cycle, and an increased population density (Carcavallo and Martinez, 1972, 1985). At even higher temperatures, the most important vector species, *Triatoma infestans*, doubles its reproductive rate (Hack, 1955).

#### *18.3.1.4. Schistosomiasis*

Schistosomiasis is a water-based disease caused by five species of schistosomal flukes. Water snails act as the intermediate host (and, strictly speaking, are not active "vectors"). The infection has increased in worldwide prevalence since mid-century, perhaps largely because of the expansion of irrigation systems in hot climates, where viable snail host populations interact with infected humans (White *et al.,* 1972; Grosse, 1993; Hunter *et al.,* 1993).

Data from both the field and the laboratory indicate that temperature influences snail reproduction and growth, schistosome mortality, infectivity and development in the snail, and human-water contact (Martens *et al.,* 1995b). In Egypt, for example, water snails tend to lose their schistosome infections during winter, but if temperatures increase, snails may mediate schistosomiasis transmission throughout the year (Gillet, 1974; WHO, 1990). Predictive modeling indicates that a change in background temperatures may cause the infection to extend to currently unaffected regions. Fluctuations in temperature may also play an important role in optimizing conditions for the several life-cycle stages of schistosomiasis (Hairston, 1973).

#### *18.3.1.5. Onchocerciasis (River Blindness)*

Onchocerciasis, or "river blindness," is a VBD affecting approximately 17.5 million people—some in Latin America, most in West Africa. The vector is a small blackfly of the genus *Simulium,* and the infectious agent is the larva of the *Onchocerca volvulus* parasite. This threadlike worm damages the skin, the

lymphatic system, and, in the most extreme cases, the eye. Climate affects the occurrence of onchocerciasis because the vector requires fast-flowing water for successful reproduction (WHO, 1985), and the adult vector can be spread by wind.

A recent simulation study on the potential impact of climate change on blackfly populations in West Africa showed that if temperature and precipitation were to change across parts of the sub-Sahel, as predicted by the Goddard Institute for Space Studies (GISS) GCM (Hansen *et al.,* 1988), blackfly populations may increase by as much as 25% at current breeding sites (Mills, 1995). Since these vectors can travel hundreds of kilometers on wind currents, new habitats in previously unsuitable areas could be quickly colonized by blackflies, introducing onchocerciasis into new areas (Garms *et al*., 1979; Walsh *et al*., 1981; WHO, 1985).

#### *18.3.1.6. Trematode Infections*

Some trematode infections, such as fascioliasis (a liver fluke that currently affects around 2.4 million persons), would be affected by climate changes because the life cycle and population size of the snail host are very sensitive to temperature. Similarly, the incidence of cercarial dermatitis (skin inflammation) would be increased at higher temperatures. This infection is currently found in Europe (Beer and German, 1994) and the United States (CDC, 1992), where it causes "swimmer's itch"; its incidence has recently increased in Russia (Beer and German, 1993) and in very poor rural communities in developing countries.

#### *18.3.1.7. Vector-Borne Viral Infections*

Many vector-borne infective agents are viruses. The human-infecting arboviruses (i.e., arthropod-borne viruses) generally have a mosquito vector. Arboviral infections span a wide clinical spectrum, from those that cause mild feverish illness or subclinical infections to those causing severe and often fatal encephalitis (brain inflammation) or hemorrhagic fever. Under favorable environmental conditions, an arboviral disease can become epidemic (population-wide), from a local endemic base or by its introduction to a previously unaffected area. The distribution and abundance of vectors are influenced by various physical factors (temperature, rainfall, humidity, and wind) and biological factors (vegetation, host species, predators, parasites, and human interventions) (WHO, 1990). Temperature also affects the rapidity of the virus' life cycle—e.g., the extrinsic incubation period for the mosquito-hosted stage of the yellow fever virus varies from several weeks to 8–10 days, depending on temperature.

Increased temperature and rainfall in Australia would influence the range and intensity of various vector-borne viral infections. For example, certain arthropod vectors and natural vertebrate hosts would spread southward and proliferate in response to warming and increased rain, resulting in increased incidence of arboviral infections such as Murray Valley encephalitis (which can cause serious brain damage), Ross River virus (which causes multiple, often long-lasting joint inflammation), and dengue (e.g., Sutherst, 1993; Nicholls, 1993).

Dengue is a severe influenza-like disease, which in some cases may take the form of a hemorrhagic fever, which can cause an average of 15% mortality without proper medical attention. Dengue is transmitted by the *Aedes aegypti* mosquito, as is urban yellow fever. In parts of Asia, dengue also is transmitted by *Ae. albopictus,* which now is colonizing North and South America. Research in Mexico has shown that an increase of 3–4°C in average temperature doubles the rate of transmission of the dengue virus (Koopman *et al*., 1991). Although there is no clear evidence of regional climatic influence, annual epidemics of dengue have returned to Central America over the past decade (as they did about twenty years ago in Asia), and, in Mexico, dengue has recently spread to previously unaffected higher altitudes (Herrera-Basto *et al*., 1992). *Ae. aegypti* mosquitoes, once limited to 1,000 meters altitude by temperature in Colombia, have been recently reported above 2,200 meters. The habitat of the mosquito is restricted to areas with a mean midwinter temperature of more than 10°C. Epidemic transmission of dengue is seldom sustained at temperatures below 20°C (Halstead, 1990).

#### *18.3.1.8. Other Vector-Borne Diseases*

VBDs are now relatively rare in most developed countries; however, it has been predicted that various VBDs might enter or increase in incidence in the United States because of higher temperatures (Longstreth, 1989; Freier, 1993; Martens *et al*., 1994); Venezuelan equine encephalitis, dengue, and leishmaniasis could extend into the southern United States; Western equine encephalitis might move further north within the United States (Reeves *et al.,* 1994). The dengue-transmitting *Ae. albopictus* mosquito, which is more cold-hardy than *Ae. aegypti*, is now well established in the United States and may extend toward Canada if temperatures increase.

Climate change would influence the global pattern of VBDs via other disturbances of ecological relationships. For example, it would bring together vertebrate animals of different species and would thereby expose animals to new arthropod vectors. Warming (and rising sea levels) would displace some human populations, perhaps resulting in migration into wilderness areas where zoonotic infectious agents are being transmitted in silent wildlife cycles. Migratory humans would thus be at risk of infection with enzootic (i.e., locally prevalent animal-infecting) agents. Climate-induced changes in ecology also could force the rapid evolution of infectious agents, with newly emergent strains of altered virulence or pathogenicity. Additionally, changes in climate means and variability can disrupt predator/prey ratios, thus loosening natural controls on pests and pathogens.

Two other general points should be noted. First, because vector control methods exist for many of these diseases, developed

countries should be able to minimize their impact. Second, however, the quicker "turnover" of the life cycle of parasites at higher temperatures will increase their likelihood of evolving greater resistance to drugs and other control methods. This would pose a particular problem to those tropical countries with high infection rates and limited socioeconomic resources.

### *18.3.2. Water-Borne and Food-Borne Infectious Diseases*

Climatic effects on the distribution and quality of surface water—including increases in flooding and water shortages that concentrate organisms, impede personal hygiene, and impair local sewerage—would influence the risks of diarrheal (including cholera) and dysentery epidemics, particularly in developing countries. Diarrheal diseases can be caused by a large variety of bacteria (e.g., *Salmonella*, *Shigella*, and *Campylobacter*), viruses (e.g., *Rotavirus*), and protozoa (e.g., *Giardia lamblia*, amoebas, and *Cryptosporidium*). Many of these organisms can survive in water for months, especially at warmer temperatures, and increased rainfall therefore could enhance their transport between groups of people. An increased frequency of diarrheal disease is most likely to occur within impoverished communities with poor sanitation. There have been outbreaks of diarrheal disease after flooding in many such settings. If flooding increased, there also would be risks of outbreaks of infection in developed countries within temporary settlements of displaced communities.

The cholera organism, *Vibrio cholerae*, can survive in the environment by sheltering beneath the mucous outer coat of various algae and zooplankton—which are themselves responsive to climatic conditions and to nutrients from wastewater and fertilizers (Epstein, 1992; Smayda, 1990; Anderson, 1992). Increases in coastal algal blooms may therefore amplify *V. cholerae* proliferation and transmission. This might also assist the emergence of new genetic strains of vibrios. Algal blooms also are associated with biotoxin contamination of fish and shellfish (Epstein *et al*., 1993). With ocean warming, toxins produced by phytoplankton, which are temperature-sensitive, could cause contamination of seafood more often (see also Chapter 16), resulting in increased frequencies of amnesic, diarrheic and paralytic shellfish poisoning and ciguatera poisoning from reef fish. Thus, climate-induced changes in the production of both aquatic pathogens and biotoxins may jeopardize seafood safety for humans, sea mammals, seabirds, and finfish.

Climate change also could create a problem via the warming of aboveground piped-water supplies. In parts of Australia, for example, there has been a seasonal problem of meningoencephalitis caused by the *Naegleria fowleri* amoeba, which proliferates in overland water pipes in summer (NHMRC, 1991). Soil-based pathogens (e.g., the tetanus bacterium and various fungi) would tend to proliferate more rapidly with higher temperature and humidity, depending on the effectiveness of microclimatic homeostatic mechanisms. Higher temperatures would also increase the problem of food poisoning by enhancing the survival and proliferation of bacteria, flies, cockroaches, and so forth in foodstuffs.

### *18.3.3. Agricultural Productivity and Food Supplies: Effects upon Nutrition and Health*

Food, as energy and nutrients, is fundamentally important to health. Malnutrition is a major cause of infant mortality, physical and intellectual stunting in childhood, and immune impairment (thus increasing susceptibility to infections). Currently, around one-tenth of the world's population may be hungry (Parry and Rosenzweig, 1993) and a larger proportion malnourished—although estimates differ according to definition.

Human societies have evolved farming methods to counter various local climatic and environmental constraints on agriculture, especially via irrigation, fertilization, mechanization, and the breeding of better-adapted varieties. Today, as gains in per capita agricultural productivity appear to be diminishing, widespread land degradation accrues, and access to new arable land is declining, the further possibility exists of adverse effects of climate change upon aspects of world food production (Houghton *et al*., 1990; Kendall and Pimentel, 1994). The impacts of climate change upon crop and livestock yield would be realized within a complex setting that encompasses climate change scenarios, crop yield response, pest population response, demographic trends, patterns of land use and management, and social and economic responses.

#### *18.3.3.1. Modes of Climatic Impact upon Agricultural Productivity*

Global warming would alter regional temperature and rainfall. Changes in these two major influences on agriculture, and consequent reductions in soil moisture, could impair the growth of many crops. Increases in the intensity of rainfall in some regions would exacerbate soil erosion. The net global impact of these climate-related changes upon food production is highly uncertain (Reilly, 1994). Although the IPCC assessment is uncertain about the overall impact, it foresees productivity gains and losses in different regions of the world (see Chapter 13). While productivity may increase initially, longer-term adaptations to sustained climate change would be less likely because of the limitations of plant physiology (Woodward, 1987).

Climate change also could affect agriculture by long-term changes in agroecosystems, by an increased frequency and severity of extreme events, and by altered patterns of plant diseases and pest infestations (e.g., Farrow, 1991; Sutherst, 1991). Debate persists over whether enrichment of the atmosphere with carbon dioxide will have a "fertilization effect" (Idso, 1990b; Bazzar and Fajer, 1992; Körner, 1993). Experiments consistently indicate that $C_3$ plants (e.g., wheat, soya beans, rice, and potatoes) would respond more positively than $C_4$ plants (e.g., millet, sorghum, and maize), which would be unaffected (see Chapter 13). This effect may be temperature-dependent (Vloedbeld and

Leemans, 1993). Such influences on the climatically optimal mix of crop species would disturb patterns of traditional agriculture in some regions.

#### *18.3.3.2. Impacts upon Food Supplies, Costs, and the Risk of Hunger*

Since climate change may threaten food security in poorer countries within the semi-arid and humid tropics (Rosenzweig *et al*., 1993; see also Chapter 13), poorer countries, already struggling with large and growing populations and marginal climatic conditions, would be particularly vulnerable to food shortages, malnutrition, and demographic disruption. In such countries, there is minimal capacity for adaptive change (Leemans, 1992). Already in Africa, more than 100 million people are "food insecure," many of them in the arid Sahel region. The cost of food on world markets would increase if crop production declined in the world's mid-latitude breadbasket regions. The large minority of the world population that already suffers from malnutrition would then face an increased threat to health from agricultural failure and rising food costs. A recent analysis predicts an extra 40–300 million people at risk of hunger in the year 2060 because of the impact of climate change, on top of a predicted 640 million people at risk of hunger by that date in the absence of climate change (Rosenzweig *et al*., 1993).

#### *18.3.3.3. Impacts of Climate Change on Non-Cereal Food Production*

Climate change may influence the production of noncrop food supplies, including animal productivity. For example, the U.S. Environmental Protection Agency has identified several infectious diseases—such as the horn fly in beef and dairy cattle and insect-borne anaplasmosis infection in sheep and cattle—that could increase in prevalence in response to climate changes (Rosenzweig and Daniel, 1989). An increase in temperature and temperature extremes also could affect the growth and health of farm animals (Furquay, 1989); young animals are much less tolerant of temperature variation than are adult animals (Bianca, 1976).

Changes in ocean temperatures and currents could affect the base of the marine food web and alter the distribution, migration, and productivity of fish species, a major source of protein for many human populations (Glantz, 1992). Increased soil erosion from intensified rainfall raises the turbidity of lakes and rivers, reducing photosynthesis and therefore fish nutrition. As in agriculture, climate change may contribute to the decline of some fisheries and the expansion of others (see Chapter 16).

### ***18.3.4. Health Impacts of Sea-Level Rise***

Each of the vast changes in sea level that have occurred during the past million years, before and after ice ages, typically took many thousands of years. The predicted rise of around half a meter over the next century (see Chapter 7, *Changes in Sea Level*, of the IPCC Working Group I volume) would be much faster than anything experienced by human populations since settled agrarian living began. Such a rise would inundate much of the world's lowlands, damage coastal cropland, and displace millions of persons from coastal and small island communities (see Chapter 12).

Much of coastal Bangladesh and Egypt's heavily populated Nile Delta would be flooded. Some low-lying, small island states such as the Maldives and Vanuatu would be at risk of partial immersion, and many other low-lying coastal regions (for example, eastern England, parts of Indonesia, the Florida Everglades, parts of the northeast coast of Latin America) would be vulnerable. The displacement of inundated communities—particularly those with limited economic, technical, and social resources—would greatly increase the risks of various infectious, psychological, and other adverse health consequences.

Sea-level rise could have a number of other effects, of varying directness, upon public health. In some locations, it could disrupt stormwater drainage and sewage disposal. Poverty and the absence of social infrastructure would compound the health consequences of storm damage, disruption of sanitation, and displacement of coastal dwellers. In many places, industrial and agricultural depletion of groundwater already are causing land subsidence, thus decreasing the threshold for impact. Meanwhile, widespread damage to coral reefs is reducing their capacity to buffer shorelines. Rising seas also would cause saltwater to encroach upon freshwater supplies from estuarine and tidal areas. Some changes in the distribution of infectious disease vectors could occur (e.g., *Anopheles sundaicus*, a saltwater vector of malaria).

### ***18.3.5. Climate and Air Pollution: Impacts on Respiratory and Other Health Disorders***

The incidence of respiratory disorders—many of which are caused primarily by dusts, noxious gases, allergic reactions, or infections—may be modulated by climate change. Some of these modulatory effects may occur via extreme temperatures or amplification of pollutant levels. Rapid changes in air masses associated with frontal passages may alter the intensity of respiratory illnesses (Ayres, 1990). People with chronic obstructive pulmonary disease (bronchitis and emphysema) often experience exacerbation during winter.

Seasonal allergic disorders would be affected by changes in the production of pollen and other biotic allergens; plant aeroallergens are very sensitive to climate (Emberlin, 1994). Changes in pollen production would principally reflect changes in the natural and agriculturally managed distribution of many plant species—for example, birch trees, grasses, various crops (e.g., oilseed rape, sunflowers), and ragweed species. Hay fever (allergic rhinitis) increases seasonally and may reflect the impact of pollen release. The seasonal distribution and the causation/exacerbation of asthma is more complex. It peaks in the pollen

season and increases again later in the year in temperate climates; in the tropics, asthma occurs more frequently in the wet season (LAIA, 1993; *Lancet*, 1985). In many asthmatic individuals, aspects of weather can exacerbate bronchial hyperresponsiveness. For example, the passage of a cold front followed by strong high pressure was found to be associated with unusually high number of asthma admission days in two U.S. cities (Goldstein, 1980). Sandstorms in Kansas (USA) and the Sudan have been accompanied by increases in bronchitis and asthma (Ayres, 1990).

It is well established that exposure to air pollutants, individually and in combinations, has serious public health consequences. For example, exposure to ozone has been shown to exacerbate asthma and impair lung function in children and the elderly (Beckett, 1991; Schwartz, 1994), and both chronic and acute exposures to fine particles are a cause of excess deaths (Dockery *et al.,* 1993; Pope *et al.,* 1995; Schwartz, 1994) even at exposures below prevailing air-quality standards. Since the combustion of fossil fuels is a major source of both carbon dioxide (a major greenhouse gas) and various air pollutants, climate change can be expected to entail more frequent occasions that combine very hot weather with increases in air-pollutant concentrations. In urban environments, the weather conditions that characterize oppressive air masses (see Section 18.2.1) also enhance the concentrations of air pollutants (Seinfeld, 1986); conditions of low wind speed and high humidity occur periodically in which neither heat nor air pollutants are rapidly dispersed. Further, increases in temperature or in ultraviolet irradiation of the lower atmosphere enhance the chemical reactions that produce secondary photochemical oxidant pollutants such as tropospheric ozone (Akimoto *et al*., 1993; de Leeuw and Leyssius, 1991; Chamiedes *et al.,* 1994).

In many urban settings, studies have shown that daily mortality from cardiovascular and respiratory diseases is a combined function of temperature and air pollutant concentrations. This combination of exposures is also likely to have interactive impacts on health. Indeed, some epidemiological evidence indicates a synergy (a positive interaction) between stressful weather and various air pollutants, especially particulates, upon mortality (e.g., Shumway *et al*., 1988; Katsouyanni *et al*., 1993; Shumway and Azari, 1992). The net effect on morbidity/mortality therefore would be greater than anticipated from prior estimates of the separate effects of weather and pollutants.

## 18.4. Stratospheric Ozone Depletion and Ultraviolet Radiation: Impacts on Health

Stratospheric ozone depletion is a quite distinct process from accumulation of greenhouse gases in the lower atmosphere (troposphere). Depletion of stratospheric ozone has recently occurred in both hemispheres, from polar regions to mid-latitudes (Kerr and McElroy, 1993; see also IPCC Working Group I volume). The major cause of this ongoing depletion is human-made gases, especially the halocarbons (UNEP, 1994).

The problem can be considered alongside climate change for three reasons: (1) several of the greenhouse gases (especially the chlorofluorocarbons) also damage stratospheric ozone; (2) altered temperature in the troposphere may influence stratospheric temperature and chemistry (Rind and Lacis, 1993); and (3) absorption of solar radiation by stratospheric ozone influences the heat budget in the lower atmosphere (see also IPCC Working Group I volume).

Stratospheric ozone absorbs part of the sun's incoming ultraviolet radiation (UVR), including much of the UV-B and all of the highest-energy UV-C. Sustained exposure to UV-B radiation is harmful to humans and many other organisms (UNEP, 1994). It can damage the genetic (DNA) material of living cells and can induce skin cancers in experimental animals. UV-B is implicated in the causation of human skin cancer and lesions of the conjunctiva, cornea, and lens; it may also impair the body's immune system (Jeevan and Kripke, 1993; Armstrong, 1994; UNEP, 1994).

### *18.4.1. Skin Cancers*

Solar radiation has been consistently implicated in the causation of nonmelanocytic and melanocytic skin cancers in fair-skinned humans (IARC, 1992; WHO, 1994b).

Nonmelanocytic skin cancers (NMSCs) comprise basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). The incidence rates, especially of squamous cell carcinoma, correlate with cumulative lifetime exposure to solar radiation (IARC, 1992; Kricker *et al.,* 1995). Studies of the action spectrum (i.e., the relative biological effect of different wavelengths) for skin carcinogenesis in mice indicate that the UV-B band is primarily responsible for NMSC (Tyrrell, 1994). Malignant melanoma arises from the pigment-producing cells (melanocytes) of the skin. Although solar radiation is substantially involved in melanoma causation (IARC, 1992; Armstrong and Kricker, 1993), the relationship is less straightforward than for NMSC; exposure in early life appears to be a major source of increased risk. The marked increases in incidence of melanoma in Western populations over the past two decades (Coleman *et al*., 1993) probably reflect increases in personal exposure to solar radiation due to changes in patterns of recreation, clothing, and occupation (Armstrong and Kricker, 1994).

The UN Environment Programme predicts that an average 10% loss of ozone (such as occurred at middle-to-high latitudes over the past decade), if sustained globally over several decades, would cause approximately 250,000 additional cases of NMSC worldwide each year (UNEP, 1994). This prediction assumes that a 1% depletion of stratospheric ozone results in a 2.0% (±0.5%) increase in NMSC incidence (80% of which are BCC). Another estimation of this "amplification factor" gives a figure of 2.25% (Slaper *et al*., 1992; den Elzen, 1994). At higher geographic resolution, Madronich and de Gruijl (1993) predict that persistence of the ozone losses of the 1979–92

period for several decades would cause the incidence of BCC to increase by 1–2% at low latitude (5°), 3–5% at 15–25°, 8–12% at 35–45°, and, at 55–65°, by 13–15% in the northern hemisphere and 20–30% in the south. They estimate that the percentage increases for SCC would be approximately double those for BCC.

### *18.4.2. Cataracts and Other Damage to the Eye*

The external epithelial (keratotic) layer of the eye, comprising cornea and conjunctiva, absorbs virtually all UVR of less than 290 nm wavelength. Corneal photokeratitis, pterygium (a growth of the conjunctival epithelium), and climatic droplet keratopathy are thought to be UVR-related (Taylor *et al*., 1989; Gray *et al*., 1992; WHO, 1994b). Inside the eye, the lens absorbs much of the residual UVR, and this absorbed radiation may cause cataracts (Taylor *et al*., 1988; Dahlback *et al*., 1989; West *et al*., 1989; WHO, 1994b).

Cataracts (lens opacities) are independent of skin pigmentation (unlike skin cancer). They occur predominantly in old age and cause more than half of the world's estimated 25–35 million cases of blindness (Harding, 1991). In Western countries, 5–10% of people aged over 65 have cataracts (Klein *et al*., 1992). The prevalence often is much higher among elderly, malnourished persons in poor countries, where micronutrient deficiencies and the metabolic consequences of severe diarrheal episodes may contribute to cataract formation (Harding, 1992). Scientific debate persists over the extent of the influence of UV-B upon cataract formation (Dolin, 1994; WHO, 1994b); some epidemiological studies have found clear-cut positive results, but others have not. The relationship is most evident for cortical and posterior subcapsular cataracts but less so for the more commonly occurring nuclear cataracts.

Ocular photodamage by UVR is enhanced by certain clinical drugs used in photochemical therapy that can cause photosensitizing reactions (Lerman, 1988). Various other photosensitizing medications would render individuals generally more susceptible to adverse health effects from increased exposure to UVR; these medications include psoralens, thiazides, phenothiazines, barbiturates, allopurinol, and retinoic acid compounds (Lerman, 1986).

### *18.4.3. Alteration of Immune Function*

Human and animal evidence indicates that UV-B irradiation of skin at quite modest levels causes local and, probably, systemic suppression of immunity (Morison, 1989; Noonan and DeFabo, 1990; Jeevan and Kripke, 1993). Most of the evidence is for local immunosuppression, in which the skin's contact hypersensitivity response is impaired (Giannini, 1986; Yoshikawa *et al*., 1990; UNEP, 1994). UV-B exposure disturbs the function of the skin's Langerhan cells and stimulates the release of certain cytokines (messenger chemicals) that promote the activity of suppressor T lymphocytes, thus dampening the local immune system (UNEP, 1994).

Evidence for more generalized (i.e., systemic) suppression of immunity comes from studies in humans, which show that sunlight exposure increases the suppressor T cells in blood (Hersey *et al*., 1983). Although there is evidence in humans of UV-induced changes in the profile of circulating immunologically active lymphocytes for several days to weeks, the extent of systemic immune suppression involved remains uncertain (de Gruijl and van der Leun, 1993). Systemic suppression also occurs in UV-irradiated mice (Kripke, 1981; Jeevan and Kripke, 1990).

Immune suppression would alter susceptibility to infectious diseases (Armstrong, 1994). Exposure to UV-B modifies various immunological reactions in mice that influence the pathogenesis of infectious diseases, such as those due to *Herpes simplex* viruses (Otani and Mori, 1987; Yasumoto *et al*., 1987), leishmania (Gianinni, 1986; Giannini and DeFabo, 1989), candida (Denkins *et al*., 1989), and mycobacteria (Jeevan and Kripke, 1989). The relevance of these findings for naturally occurring infectious diseases, and for vaccination efficacy, in humans remains unknown. UNEP (1994) concluded that: "It will be very difficult to assess the role of UV-B radiation on natural infections in human populations. Based on current knowledge, we would predict that an effect of UV-B radiation would manifest as an increase in the severity or duration of disease and not necessarily as an increase in disease incidence."

### *18.4.4. Indirect Effects of Ozone Depletion upon Human Health*

An increase in UV-B irradiance is predicted to impair photosynthesis on land and sea (UNEP, 1994). Although the magnitude is uncertain, and may well not be large, there would be at least a marginal reduction in crop yields (Worrest and Grant, 1989) and in the photosynthetic production of biomass by marine phytoplankton, the basis of the aquatic food chain (Smith and Baker, 1989; Smith *et al*., 1992). Thus, adverse effects of UV-B upon photosynthesis would, to some extent, reduce global food production.

## 18.5. Options for Adaptation

Various adaptation strategies are possible to reduce the impacts of climate change on human health. Such adaptation could be developed at the population or individual level. The feasibility of adaptation would be constrained for many of the world's populations by a lack of local resources.

At the population level, environmental management of ecosystems (e.g., freshwater resources, wetlands, and agricultural areas sensitive to invasion by vectors), public health surveillance and control programs (especially for infectious diseases), and introduction of protective technologies (e.g., insulated buildings, air conditioning, strengthened sea defences, disaster warning systems) would be important. Improved primary health care for vulnerable populations could play a significant

role in reducing a range of health impacts, including some vector-borne and other communicable diseases, and the effects of extreme events. One example is extension of vaccination coverage, although no suitable vaccines exist for some of the diseases most sensitive to climate change (e.g., dengue and schistosomiasis) or for many of the newly emerging infections.

At the individual level, people should be encouraged to refrain from or to limit dangerous exposures (e.g., by use of domestic cooling, protective clothing, mosquito nets). Such behavioral responses could complement any physiological adaptation that might occur spontaneously through acclimatization (to heat stress) or acquired immunity (to infectious diseases).

In view of limitations to the forecasting of health impacts at this stage of our knowledge, an important and practical form of adaptation would be to improve large-scale monitoring and surveillance systems, especially for vulnerable populations and areas. Recently initiated efforts to observe and monitor aspects of the Earth's environment and ecosystems in relation to climate change now should incorporate health-related monitoring (Haines *et al*., 1993). Advances in climate forecasting and in the regional integration of ecological and health monitoring (including local vulnerability factors) will facilitate development of early-warning systems.

Finally, if health impacts of climate change are probable and serious, then the only effective long-term basis for mitigation lies in primary prevention at the societal level. This would require acceptance of the Precautionary Principle as the foundation of policy response. This, in turn, would suggest some fundamental, and therefore difficult, reorientations of social, economic, and political priorities. Meanwhile, care must be taken that alternative technologies do not introduce new health hazards.

## 18.6. Research Needs

- Development and validation of integrated mathematical models for the prediction of health impacts. Such models must draw on multiple scientific disciplines and should take maximal account of regional and local influences on the effects being modeled and on their interaction with other environmental stresses.
- Identification and analysis of current or recent settings in which the health impacts of local or regional climate changes (occurring for whatever reason) can be studied. The apparent recent changeable patterns of infectious diseases around the world may afford good opportunities for clarifying and quantifying the influences of climatic factors.
- Incorporation of health-related measurements in global, regional, and local monitoring activities. This would enhance the early detection of shifts in health risks, the evaluation of alternative indices for monitoring health (including the use of sensitive species as bioindicators), and the opportunity to detect and/or examine previously unsuspected or undocumented environment–health relationships.
- Some specific research needs include:
  - Comparison of impacts of heat waves in urban and rural populations, to clarify the relative importance of thermal stress and air pollutants
  - Examination of the interplay between climatic impacts on forests and other terrestrial ecosystems on the range and dynamics of vector-borne disease
  - Study of factors influencing population vulnerability to climate change.

## 18.7. Concluding Remarks

Forecasting the health impacts of global climate change entails unavoidable uncertainty and complexity. Human populations vary greatly in their vulnerability to climate changes and in their resources for protection and mitigation. Likewise, the responses of infectious disease vectors to changes in climate depend greatly on other concomitant environmental stresses and the adequacy of control measures and health care systems. Meanwhile, population health status continues to be influenced by a rich mix of cultural and socioeconomic factors. Hence, assessing the health impact of climate change requires a systems-based modeling approach that integrates information about climatic factors, other environmental stresses, ecological processes, and social-economic-political inputs and responses.

Alongside the need for improved health impact assessment capability is a precautionary need to develop global, regional, and local monitoring systems for the early detection of climate-induced changes in human health. There have, indeed, been various recent events that, plausibly, might be early signals of such change. The increased heat-related deaths in India in 1995; the changes in geographic range of some vector-borne diseases; the coastal spread of cholera: Could these be early indications of shifts in population health risk in response to aspects of climate change? Of course, it is not possible to attribute particular, isolated events to a change in climate or weather pattern; other plausible explanations exist for each of them, and a number of different factors may combine to produce each event. However, it is important that we begin to assess patterns of change in the various indices of human health that will provide early insight and will assist further the development of predictive modeling.

There is thus a clear need for enhanced research and monitoring activities. This need reflects the assessment that the potential health impacts of climate change, particularly if sustained in the longer term and if generally adverse, could be a serious consequence of the ongoing anthropogenic changes in the composition of Earth's atmosphere.

## References


**Akimoto**, H., H. Nakane, and Y. Matsumoto, 1993: The chemistry of oxidant generation. In: *Chemistry of the Atmosphere: The Impact on Global Change* [Calvert, J.G. (ed.)]. Blackwell Scientific Publications, London, UK, pp. 261-273.

**Alcamo**, J., G.J.J. Kreilman, M. Krol, and G. Zuidema, 1994a: Modelling the global society-biosphere-climate system, part 1: model description and testing. *Water, Air and Soil Pollution*, **76**, 1-35.

**Alcamo**, J., G.J. van den Born, A.F. Bouwman, B.J. de Haan, H. Klein-Goldewijk, O. Klepper, R. Leemans, J. Krabec, R. Leemans, J.G.J. Olivier, A.M.C. Toet, H.J.M. de Vries, and H.J. van der Woerd, 1994b: Modelling the global society-biosphere-climate system, part 2: computed scenarios. *Water, Air and Soil Pollution*, **76**, 37-78.

**Alexander**, D., 1993: *Natural Disasters*. UCL Press, London, UK, 632 pp.

**Anderson**, D.M., 1992: The Fifth International Conference on Toxic Marine Phytoplankton: a personal perspective. *Harmful Algae News. Supplement to International Marine Science*, **62**, 6-7.

**Armstrong**, B.K., 1994: Stratospheric ozone and health. *International Journal of Epidemiology*, **23(5)**, 873-885.

**Armstrong**, B.K. and A. Kricker, 1993: How much melanoma is caused by sun exposure? *Melanoma Research*, **3**, 395-401.

**Armstrong**, B.K. and A. Kricker, 1994: Cutaneous melanoma. In: *Cancer Surveys,* vol. 19/20 [Doll, R., J.F. Fraumeni, and C.S. Muir (eds.)]. Cold Spring Harbor Laboratory Press, New York, NY, pp. 219-240.

**Asian Development Bank**, 1994: *Climate Change in Asia: Indonesia Country Report*. Asian Development Bank, Manila, Philippines, pp. 37-42.

**Ayres**, J.G., 1990: Meteorology and respiratory disease. *Update 1990,* **40**, 596-605.

**Babayev**, A.B., 1986: Some aspects of man's acclimatization to hot climates. In: *Climate and Human Health, WHO/UNEP/WMO International Symposium,* vol. 2. WMO, Leningrad, pp. 125-126.

**Bazzar**, F.A. and E.D. Fajer, 1992: Plant life in a $CO_2$-rich world. *Scientific American,* **266(1)**, 18-24.

**Beckett**, W.S., 1991: Ozone, air pollution, and respiratory health. *Yale Journal of Biology and Medicine*, **64**, 167-75.

**Beer**, S.A. and S.M. German, 1993: The ecological prerequisites for a worsening of cerciasis situation in the cities of Russia. *Parazitologia,* **27(6)**, 441-449 [in Russian].

**Beer**, S.A. and S.M. German, 1994: Ekologicheskie predposylki rasprostraneniia shistosomatidenykh dermatoitov tserkariozov v Moskve i Podomoskov'e. *Medicinschke Parazitologie—Moskau Jan-Mar*, **1**, 16-19.

**Bianca**, W., 1976: The significance of meteorology in animal production. *International Journal of Biometeorology*, **20**, 139-56.

**Bouma**, M.J., H.E. Sondorp, and H.J. van der Kaay, 1994: Health and climate change. *The Lancet*, **343**, 302.

**Bradley**, D.J., 1993: Human tropical diseases in a changing environment. In: *Environmental Change and Human Health* [Lake J., G. Bock, and K. Ackrill (eds.)]. Ciba Foundation Symposium, CIBA Foundation, London, UK, pp. 146-162.

**Burgos**, J.J., S.I. Curto de Casas, R.U. Carcavallo, and I. Galíndez Girón, 1994: Global climate change influence in the distribution of some pathogenic complexes (malaria and Chagas disease) in Argentina. *Entomologia y Vectores,* **1(2)**, 69-78.

**Carcavallo**, R.U. and A. Martinez, 1972: Life cycles of some species of Triatoma (Hemipt. Reduviidae). *Canadian Entomology*, **104**, 699-704.

**Carcavallo**, R.U. and A. Martinez, 1985: Biologica, ecologia y distribucion geografica de los Triatominos Americanos. *Chagas (Special Issue),* **1**, 149-208.

**CDC**, 1989: Heat-related deaths—Missouri, 1979–1988. *Morbidity and Mortality Weekly Report,* **38**, 437–439.

**CDC**, 1992: Cercarial dermatitis outbreak in a state park—Delaware 1991. *Journal of the American Medical Association*, **267(19)**, 2581-2586.

**CDC**, 1994a: Hantavirus Pulmonary Syndrome—United States, 1993. *Morbidity and Mortality Weekly Report*, **43(3)**, 45-48.

**CDC**, 1994b: Update: influenza activity—United States and worldwide, 1993-93 season and composition of the 1994-95 influenza vaccine. *Morbidity and Mortality Weekly Report,* **43(10)**, 179-183.

**Chameides**, W.L., P.S. Kasibhatla, J. Yienger, and H. Levy, 1994: Growth of continental-scale metro-agro-plexes, regional ozone pollution, and world food production. *Science*, **264**, 74-77.

**Climate Change Impacts Review Group UK**, 1991: *The Potential Effects of Climate Change in the United Kingdom*. HMSO, London, UK, pp. 3-13.

**Coleman**, M., J. Esteve, P. Damiecki, A. Arslan, and H. Renard, 1993: *Trends in Cancer Incidence and Mortality*. International Agency for Research on Cancer, IARC Scientific Publications No. 121, Lyon, France, 806 pp.

**Curto de Casas**, S.I. and R.U. Carcavallo, 1984: Limites del triatomismo en la Argentina. I: Patagonia. *Chagas,* **1(4)**, 35-40.

**Curwen**, M., 1991: Excess winter mortality: a British phenomenon? *Health Trends,* **22**, 169–175.

**Dahlback**, A., T. Henriksen, S.H.H. Larsen, and K. Stamnes, 1989: Biological UV doses and the effect of ozone layer depletion. *Photochemistry and Photobiology,* **49(5)**, 621-625.

**de Girolamo**, G. and A. McFarlane, 1994: *Epidemiology of Posttraumatic Stress Disorders: A Comprehensive Review of the Literature*. Unpublished paper.

**de Gruijl**, F.R. and J.C. van der Leun, 1993: Influence of ozone depletion on incidence of skin cancer: quantitative prediction. In: *Environmental UV Photobiology* [Young, A.R. *et al*. (eds.)]. Plenum Press, New York, NY, pp. 89-112.

**de Leeuw**, F.A.A. and H.J.v.R. Leyssius, 1991: Sensitivity of oxidant concentrations on changes in UV radiation and temperature. *Atmospheric Environment*, **25A**, 1024-1033.

**den Elzen**, M., 1994: *Global Environmental Change. An Integrated Modelling Approach*. International Books, Utrecht, Netherlands, 253 pp.

**Denkins**, Y.D., I.J. Fidler, and M.L. Kripke, 1989: Exposure of mice to UV-B radiation suppresses delayed hypersensitivity to *Candida albicans*. *Photochemistry and Photobiology*, **49**, 615-619.

**Diamond**, J., 1991: *The Rise and Fall of the Third Chimpanzee*. Radius, London, UK, 360 pp.

**Dobson**, A. and R. Carper, 1993: Biodiversity. *The Lancet,* **342**, 1096-1099.

**Dockery**, D.W., C.A. Pope, X. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer, 1993: An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine*, **329**, 1753-1759.

**Dolin**, P.J., 1994: Ultraviolet radiation and cataract: a review of the epidemiological evidence. *British Journal of Ophthalmology,* **798**, 478-482.

**Durkin**, M.S., N. Khan, and L.L. Davidson, 1993: The effects of a natural disaster on child behaviour: evidence for post-traumatic stress. *American Journal of Public Health*, **83**, 1549-1553..

**Ellis**, F.P. and F. Nelson, 1978: Mortality in the elderly in a heat wave in New York City, August, 1974. *Environmental Research*, **5**, 1-58.

**Emberlin**, J., 1994: The effects of patterns in climate and pollen abundance on allergy. *Allergy*, **49**, 15-20.

**Epstein**, P.R., 1992: Cholera and the environment. *The Lancet,* **339**, 1167–1168.

**Epstein**, P.R., T.E. Ford, and R.R. Colwell, 1993: Marine ecosystems. *The Lancet,* **342**, 1216-1219.

**Escudero**, J.C., 1985: Health, nutrition and human development. In: *Climatic Impact Assessment* [Kates, R.W., J.H. Ausubel, and M. Berberian (eds.)]. Scope 27, John Wiley and Sons, Chichester, UK, pp. 251–272.

**Farrow**, R.A., 1991: Implications of potential global warming on agricultural pests in Australia. *EPPO Bulletin*, **21**, 683-696.

**Feachem**, R., 1994: Health decline in Eastern Europe. *Nature*, **367**, 313-314.

**Freier**, J.E., 1993: Eastern equine encephalomyelitis. *The Lancet*, **342**, 1281-1282.

**French**, J.G. and K.W. Holt, 1989: Floods. In: *The Public Health Consequences of Disasters* [Gregg, M.B. (ed.)]. U.S. Department of Health and Human Services, CDC, Atlanta, GA, pp. 69-78.

**Furquay**, J.W., 1989: Heat stress as it affects animal production. *Journal of Animal Science*, **52**, 164-174.

**Garms**, R., J.F. Walsh, and J.B. Davis, 1979: Studies on the reinvasion of the Onchocerciasis Control Programme in the Volta River Basin by *Sumulium damnosum s.l.* with emphasis on the Southwestern areas. *Tropical Medicine and Parasitology,* **30**, 345-362.

**Giannini**, M.S.H., 1986: Suppression of pathogenesis in cutaneous leishmaniasis by UV-irradiation. *Infection and Immunity,* **51**, 838-843.

**Giannini**, M.S.H. and E.C. DeFabo, 1989: Abrogation of skin lesions in cutaneous leishmaniasis by ultraviolet-B radiation. In: *Leishmaniasis: The Current Status and New Strategies for Control* [Hart, D.T. (ed.)]. NATO ASI Series A: Life Sciences, Plenum Press, London, UK, pp. 677-684.

**Gill**, C.A., 1920a: The relationship between malaria and rainfall. *Indian Journal of Medical Research,* **37(3)**, 618-632.

**Gill**, C.A., 1920b: The role of meteorology and malaria. *Indian Journal of Medical Research*, **8(4)**, 633-693.

**Gilles**, H.M., 1993: Epidemiology of malaria. In: *Bruce-Chwatt's Essential Malariology* [Gilles, H.M. and D.A. Warrell (eds.)]. Edward Arnold, London, UK, pp. 124-163.

**Gillett**, J.D., 1974: Direct and indirect influences of temperature on the transmission of parasites from insects to man. In: *The Effects of Meteorological Factors Upon Parasites* [Taylor, A.E.R. and R. Muller (eds.)]. Blackwell Scientific Publications, Oxford, UK, pp. 79–95.

**Glantz**, M.H. (ed.), 1992: *Climate Variability, Climate Change and Fisheries*. Cambridge University Press, Cambridge, UK, 450 pp.

**Goldstein**, I.F., 1980: Weather patterns and asthma epidemics in New York City and New Orleans, USA. *International Journal of Biometeorology,* **24**, 329-339.

**Gordon**, H.B., P.H. Whetton, A.B. Pittock, A.M. Fowler, and M.R. Haylock, 1992: Simulated changes in daily rainfall intensity due to the enhanced greenhouse effect: implications for extreme rainfall events. *Climate Dynamics*, **8**, 83-102.

**Gray**, R.H., G.J. Johnson, and A. Freedman, 1992: Climatic droplet keratopathy. *Surveys of Ophthalmology*, **36**, 241-253.

**Green**, B.L., 1982: Assessing levels of psychological impairment following disaster: consideration of actual and methodological dimensions. *Journal of Nervous Disease*, **170**, 544-548

**Green**, B.L., M. Korol, M.C. Grace, M.G. Vary, A.C. Leonard, G.C. Gleser, and S. Smitson-Cohen, 1991: Children and disaster: age, gender, and parental effects of PTSD symptoms. *Journal of the Academy of Child and Adolescent Psychiatry*, **30**, 945-951.

**Gregg**, M.B. (ed.), 1989: *The Public Health Consequences of Disasters*. U.S. Department of Health and Human Services, CDC, Atlanta, GA, 137 pp.

**Grosse**, S., 1993: *Schistosomiasis and Water Resources Development: A Reevaluation of an Important Environment-Health Linkage*. The Environment and Natural Resources Policy and Training Project, Working Paper EPAT/MUCIA, Technical Series No. 2., May 1993. University of Michigan Press, Ann Arbor, MI, 32 pp.

**Hack**, W.H., 1955: Estudio sobre la biologia del Triatoma infestans. *Anales Instituto de Medicina Regional Tucuman,* **4**, 125-147.

**Haines**, A. and C. Fuchs, 1991: Potential impacts on health of atmospheric change. *Journal of Public Health Medicine*, **13**, 69–80.

**Haines**, A., P.R. Epstein, and A.J. McMichael, 1993: Global Health Watch: monitoring the impacts of environmental change. *The Lancet,* **342**, 1464-1469.

**Hairston**, N.G., 1973: The dynamics of transmission. In: *Epidemiology and Control of Schistosomiasis* [Ansari, N. (ed.)]. Karger, Basel, Switzerland, pp. 250-336.

**Halstead**, S.B., 1990: Dengue. In: *Tropical and Geographical Medicine* [Warren, K. and A.A.F. Mahmoud (eds.)]. McGraw-Hill, New York, NY, 2nd ed., pp. 675-685.

**Hansen**, J., I. Fung, A. Lacis, S. Lebedoff, D. Rind, R. Ruedy, and G. Russell, 1988: Global climate changes as forecast by the GISS 3-D model. *Journal of Geophysical Research*, **93**, 9341-9364.

**Harding**, J., 1991: *Cataract: Biochemistry, Epidemiology and Pharmacology*. Chapman Hall, London, UK, 382 pp.

**Harding**, J., 1992: Physiology, biochemistry, pathogenesis, and epidemiology of cataract. *Current Opinion in Ophthalmology*, **3**, 3-12.

**Herrera-Basto**, E., D.R. Prevots, M.L. Zarate, J.L. Silva, and J.S. Amor, 1992: First reported outbreak of classical dengue fever at 1,700 meters above sea level in Guerrero State, Mexico, June 1988. *American Journal of Tropical Medicine,* **46(6)**, 649-653.

**Hersey**, P., G. Haran, E. Hasic, and A. Edwards, 1983: Alteration of T-cell subsets and induction of suppressor T-cell activity in normal subjects after exposure to sunlight. *Journal of Immunology,* **131**, 171-174.

**Homer-Dixon**, T., J.H. Boutwell, and G.W. Rathjens, 1993: Environmental change and violent conflict. *Scientific American*, **268**, 38-45.

**Horowitz**, M. and S. Samueloff, 1987: Circulation under extreme heat load. In: *Comparative Physiology of Environmental Adaptations,* vol. 2 [Dejours, P. (ed.)]. Karger, Basel, Switzerand, pp. 94-106.

**Houghton**, J.T., G.J. Jenkins, and J.J. Ephraums (eds.), 1990: *Climate Change. The IPCC Scientific Assessment*. Cambridge University Press, Cambridge, UK, 365 pp.

**Hunter**, J.M.L., L. Rey, K.Y. Chu, E.O. Adekolu-John, and K.E. Mott, 1993: *Parasitic Diseases in Water Resources Development: The Need for Intersectoral Negotiation*. WHO, Geneva, Switzerland, 152 pp.

**IARC**, 1992: *Solar and Ultraviolet Radiation. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, vol. 55. IARC, Lyon, France, 316 pp.

**Idso**, S.B., 1990a: *The Carbon Dioxide/Trace Gas Greenhouse Effect: Greatly Overestimated?* American Society of Agronomy, Madison, WI, pp. 19-26.

**Idso**, S.B., 1990b: *Interactive Effects of Carbon Dioxide and Climate Variables on Plant Growth*. American Society of Agronomy, Madison, WI, pp. 61-69.

**Jeevan**, A. and M.L. Kripke, 1989: Effect of a single exposure to UVB radiation on *Mycobacterium bovis* bacillus Calmette-Guerin infection in mice. *Journal of Immunology*, **143**, 2837-2843.

**Jeevan**, A. and M.L. Kripke, 1990: Alteration of the immune response to Mycobacterium bovis BCG in mice exposed chronically to low doses of UV radiation. *Cellular Immunology*, **130**, 32-41.

**Jeevan**, A. and M.L. Kripke, 1993: Ozone depletion and the immune system. *The Lancet*, **342**, 1159-1160.

**Kalkstein**, L.S., 1993: Health and climate change: direct impacts in cities. *The Lancet*, **342**, 1397-1399.

**Kalkstein**, L.S. and K.E. Smoyer, 1993: The impact of climate change on human health: some international implications. *Experientia*, **49**, 969-979.

**Katsouyanni**, K., A. Pantazopoulou, G. Touloumi, *et al*., 1993: Evidence for interaction between air pollution and high temperature in the causation of excess mortality. *Archives of Environmental Health*, **48**, 235-242.

**Keatinge**, W.R, S.R.K. Coleshaw, and J. Holmes, 1989: Changes in seasonal mortalities with improvement in home heating in England and Wales from 1964 to 1984. *International Journal of Biometeorology*, **33**, 71-76.

**Kendall**, H.W. and D. Pimentel, 1994: Constraints on the expansion of the global food supply. *Ambio*, **23(3)**, 198-205.

**Kerr**, R.A. and M.B. McElroy, 1993: Evidence for large upward trends of ultraviolet-B radiation linked to ozone depletion. *Science*, **262**, 1032-1034.

**Kilbourne**, E.M., 1989: Heatwaves. In: *The Public Health Consequences of Disasters* [Gregg, M.B. (ed.)]. U.S. Department of Health and Human Services, Public Health Service, CDC, Atlanta, GA, pp. 51-61.

**Kilbourne**, E.M., 1992: Illness due to thermal extremes. In: *Public Health and Preventive Medicine* [Last, J.M. and R.B. Wallace (eds.)]. Maxcy-Rosenau-Last, Norwalk, Appleton Lange, 13th ed., pp. 491–501.

**Klein**, B.E., R. Klein, and K.L.P. Linton, 1992: Prevalence of age-related lens opacities in a population. The Beaver Dam Eye Study. *Ophthalmology*, **99**, 546-552.

**Koopman**, J.S., D.R. Prevots, M.A.V. Marin, H.G. Dantes, M.L.Z. Aqino, I.M. Longini, and J.S. Amor, 1991: Determinants and predictors of dengue infection in Mexico. *American Journal of Epidemiology*, **133**, 1168–1178.

**Körner**, C., 1993: $CO_2$ fertilization: the great uncertainty in future vegetation development. In: *Vegetation Dynamics and Global Change* [Solomon, A.M. and H.H. Shugart (eds.)]. Chapman and Hall, New York, NY, pp. 9953-9970.

**Kricker**, A., B.K. Armstrong, D. English, and P.J. Heenan, 1995: A dose response curve for sun exposure and basal cell carcinoma. *International Journal of Cancer*, **60**, 482-488.

**Kripke**, M.L., 1981: Immunologic mechanisms in UV radiation carcinogenesis. *Advances in Cancer Research*, **34**, 69-81.

**Kunst**, A.E., C.W.N. Looman, and J.P. Mackenbach, 1993: Outdoor air temperature and mortality in the Netherlands: a time-series analysis. *American Journal of Epidemiology,* **137**, 331-341.

**LAIA**, 1993*: Seasonal Variations in Asthma*. Lung and Asthma Information Agency Factsheet 93/4, St. George's Medical School, London, UK.

***Lancet***, 1985: Asthma and the weather. *The Lancet*, **i**, 1079-1080.

***Lancet***, 1994: *Health and Climate Change*. The Devonshire Press, London, UK.

**Langford**, I.H. and G. Bentham, 1995: The potential effects of climate change on winter mortality in England and Wales. *International Journal of Biometeorology*, **38**, 141-147.

**Larsen**, U., 1990a: The effects of monthly temperature fluctuations on mortality in the United States from 1921 to 1985. *International Journal of Biometeorology*, **34**, 136-145.

**Larsen**, U., 1990b: Short-term fluctuations in death by cause, temperature, and income in the United States 1930 to 1985. *Social Biology,* **37(3-4)**, 172-187.

**Last**, J.M., 1993: Global change: ozone depletion, greenhouse warming and public health. *Annual Review of Public Health,* **14**, 115-136.

**Leemans**, R., 1992: Modelling ecological and agricultural impacts of global change on a global scale. *Journal of Scientific and Industrial Research*, **51**, 709-724.

**Lerman**, S., 1986: Photosensitizing drugs and their possible role in enhancing ocular toxicity. *Ophthalmology*, **93**, 304-318.

**Lerman**, S., 1988: Ocular phototoxicity. *New England Journal of Medicine,* **319**, 1475-1477.

**Levins**, R., T. Awerbuch, U. Brinkman, I. Eckhardt, P.R. Epstein, N. Makhoul, C. Albuquerque de Possas, C. Puccia, A. Spielman, and M.E. Wilson, 1994: The emergence of new diseases. *American Scientist,* **82**, 52-60.

**Lighthill**, J., G. Holland, W. Gray, C. Landsea, J. Evans, Y. Kurihara, and C. Guard, 1994: Global climate change and tropical cyclones. *Bulletin of the American Meteorological Society,* **75(11)**, 2147-2157.

**Loevinsohn**, M., 1994: Climatic warming and increased malaria incidence in Rwanda. *The Lancet,* **343**, 714-718.

**Longstreth**, J.A., 1989: Human health. In: *The Potential Effects of Global Climate Change on the United States* [Smith, J.B. and D. Tirpak (eds.)]. EPA-230-05-89-050, U.S. EPA, Washington, DC, pp. 525-556.

**Madronich**, S. and F.R. de Gruijl, 1993: Skin cancer and UV radiation. *Nature*, **366**, 23.

**Martens**, W.J.M., J. Rotmans, and L.W. Niessen, 1994: *Climate Change and Malaria Risk: An Integrated Modelling Approach*. GLOBO Report Series No. 3, Report No. 461502003, Global Dynamics & Sustainable Development Programme, Bilthoven, RIVM, 37 pp.

**Martens**, W.J.M., L.W. Niessen, J. Rotmans, T.H. Jetten, and A.J. McMichael, 1995a: Potential impact of global climate change on malaria risk. *Environmental Health Perspectives,* **103(5)**, 458-464.

**Martens**, W.J.M., T.H. Jetten, J. Rotmans, and L.W. Niessen, 1995b: Climate change and vector-borne diseases: a global modelling perspective. *Global Environmental Change*, **5(3)**, 195-209.

**Martin**, P. and M. Lefebvre, 1995: Malaria and climate: sensitivity of malaria potential transmission to climate. *Ambio,* **24(4)**, 200-207.

**Matsuoka**, Y. and K. Kai, 1994: An estimation of climatic change effects on malaria. *Journal of Global Environment Engineering*, **1**, 1-15.

**McMichael**, A.J., 1993: Global environmental change and human population health: a conceptual and scientific challenge for epidemiology. *International Journal of Epidemiology*, **22**, 1-8.

**McMichael**, A.J. and W.J.M. Martens, 1995: The health impacts of global climate change: grappling with scenarios, predictive models, and multiple uncertainties. *Ecosystem Health*, **1(1)**, 23-33.

**Meehl**, G.A., G.W. Branstator, and W.M. Washington, 1993: Tropical Pacific interannual variability and $CO_2$ climate change. *Journal of Climate*, **6**, 42-63.

**Michael**, E. and D.A.P. Bundy, 1995: The global burden of lymphatic filariasis. In: *World Burden of Diseases* [Murray, C.J.L. and A.D. Lopez (eds.)]. WHO, Geneva, Switzerland (in press).

**Miller**, L.H. and D.A. Warrell, 1990: Malaria. In: *Tropical and Geographical Medicine* [Warren, K.S. and A.A.F. Mahmoud (eds.)]. McGraw-Hill, New York, NY, 2nd ed., pp. 245-264.

**Mills**, D.M., 1995: A climatic water budget approach to blackfly population dynamics. *Publications in Climatology*, **48**, 1-84.

**Morison**, W.L., 1989: Effects of ultraviolet radiation on the immune system in humans. *Photochemistry and Photobiology*, **50**, 515-524.

**Morse**, S.S., 1991: Emerging viruses: defining the rules for viral traffic. *Perspectives in Biology and Medicine*, **34(3)**, 387-409.

**Murray**, C.J.L. and A.D. Lopez (eds.) 1994: *Global Comparative Assessments in the Health Sector: Disease Burden, Expenditures and Intervention Packages*. WHO, Geneva, Switzerland, 196 pp.

**Myers**, N., 1993: Environmental refugees in globally warmed world. *BioScience,* **43(11)**, 752-761.

**NHMRC**, 1991: *Health Implications of Long Term Climatic Change*. Australian Government Printing Service, Canberra, Australia, 83 pp.

**Nicholls**, N., 1993: El Niño-Southern Oscillation and vector-borne disease. *The Lancet,* **342**, 1284-1285.

**Niessen**, L.W. and J. Rotmans, 1993: *Sustaining Health: Towards an Integrated Global Health Model*. RIVM Report No. 461502001, Bilthoven, RIVM, 32 pp.

**Noonan**, F.P. and E.C. DeFabo, 1990: Ultraviolet-B dose-response curves for local and systemic immunosuppression are identical. *Photochemistry and Photobiology,* **52**, 801-810.

**Otani**, T. and R. Mori, 1987: The effects of ultraviolet irradiation of the skin on herpes simplex virus infection: alteration in immune function mediated by epidermal cells in the course of infection. *Archives of Virology*, **96**, 1-15.

**PAHO**, 1994: Leishmaniasis in the Americas. *Epidemiological Bulletin*, **15(3)**, 8-13.

**Parry**, M.L. and C. Rosenzweig, 1993: Food supply and the risk of hunger. *The Lancet*, **342**, 1345-1347.

**Peto**, R., A.D. Lopez, J. Boreham, M. Thun, and C. Heath, 1994: *Mortality from Smoking in Developed Countries 1950-2000*. Oxford University Press, New York, NY, 553 pp.

**Pope**, C.A., D.V. Bates, M.E. Raizenne, 1995: Health effects of particulate air pollution: time for reassessment? *Environmental Health Perspectives*, **103**, 472-480.

**Reeves**,W.C., J.L. Hardy, W.K. Reisen, M.M. Milby, 1994: Potential effect of global warming on mosquito-borne arboviruses. *Journal of Medical Entomology*, **31(3)**, 323-332.

**Reilly**, J., 1994: Crops and climate change. *Nature*, **367**, 118-119.

**Rind**, D. and A. Lacis, 1993: The role of the stratosphere in climate change. *Surveys in Geophysics*, **14**, 133-165.

**Rogers**, D.J. and M.J. Packer, 1993: Vector-borne diseases, models and global change. *The Lancet*, **342**, 1282-1284.

**Rogot**, E., P.D. Sorlie, and E. Backland, 1992: Air-conditioning and mortality in hot weather. *American Journal of Epidemiology,* **136**, 106-116.

**Rosenzweig**, C. and M.M. Daniel, 1989: Agriculture. In: *The Potential Effects of Global Climate Change in the United States* [Smith, J.B. and D. Tirpak (eds.)]. EPA-230-05-89-050, U.S. EPA, Washington, DC, pp. 93-122.

**Rosenzweig**, C., M.L. Parry, G. Fischer, and K. Frohberg, 1993: *Climate Change and World Food Supply*. Research Report No. 3, Environmental Change Unit, Oxford University, Oxford, UK, 28 pp.

**Seinfeld**, J.H., 1986: *Air Pollution: Physical and Chemical Fundamentals*. MacGraw-Hill, New York, NY, 523 pp.

**Schwartz**, J., 1994: Air pollution and daily mortality: a review and meta-analysis. *Environmental Research*, **64**, 36-52.

**Shope**, R., 1991: Global climate change and infectious diseases. *Environmental Health Perspectives*, **96**, 171–174.

**Shumway**, R.H. and A.S. Azari, 1992: *Structural Modeling of Eepidemiological Time Series*. Final Report ARB A 833-136, Air Resources Board, Sacramento, CA, 67 pp.

**Shumway**, R.H., A.S. Azari, and Y. Pawitan, 1988: Modelling mortality fluctuations in Los Angeles as functions of pollution and weather effects. *Environmental Research*, **45**, 224-241.

**Slaper**, H., M.G.H. den Elzen, H.J. van den Woerd, and J. Greef, 1992: *Ozone Depletion and Skin Cancer Incidence, Integrated Modelling Approach*. Report No. 749202001, Bilthoven, RIVM, 45 pp.

**Smayda**, T.J., 1990: Novel and nuisance phytoplankton blooms in the sea: evidence for a global epidemic. In: *Toxic Marine Phytoplankton* [Graneli, E. *et al*. (eds.)]. Elsevier Science Publishers, New York, NY, pp. 29-40.

**Smith**, R.C. and K.S. Baker, 1989: Stratospheric ozone, middle ultraviolet radiation and phytoplankton productivity. *Oceanography,* **2**, 4-10.

**Smith**, R.C., B.B. Prezelin, K.S. Baker, R.R. Bidigare, N.P. Boucher, T. Coley, D. Karentz, S. MacIntyre, H.A. Matlock, D. Menzies, M. Ondrusek, Z. Wan, and K.J. Waters, 1992: Ultraviolet radiation and phytoplankton biology in Antarctic water. *Science*, **255**, 952-959.

**Sommer**, A. and W.H. Mosely, 1972: East Bengal cyclone of November 1970: epidemiological approach to disaster assessment. *The Lancet*, **I**, 1029-1036.

**Sutherst**, R.W., 1991: Pest risk analysis and the greenhouse effect. *Review of Agricultural Entomology*, **79(11/12)**, 1177-1187.

**Sutherst**, R.W., 1993: Arthropods as disease vectors in a changing environment. In: *Environmental Change and Human Health* [Lake, J.V. (ed.)]. Ciba Foundation Symposium, John Wiley and Sons, New York, NY, pp. 124-145.

**Tan**, G., 1994: The potential impacts of global warming on human mortality in Shanghai and Guangzhou, China. *Acta Scientiae Cicumstantiae*, **14**, 368-373.

**Taylor**, H.R., S.K. West, F.S. Rosenthal, B. Munoz, H.S. Newland, and E.A. Emmett, 1989: Corneal changes associated with chronic UV irradiation. *Archives of Ophthalmology*, **107**, 1481-1484.

**Taylor**, H.R., S.K. West, F.S. Rosenthal, M. Beatriz, H.S. Newland, H. Abbey, and E.A. Emmett, 1988: The effect of ultraviolet radiation on cataract formation. *New England Journal of Medicine,* **319**, 1411-1415.

**Telleria**, A.V., 1986: Health consequences of floods in Bolivia in 1982. *Disasters*, **10**, 297-307.

**Thurman**, E.M., D.A. Goolsby, M.T. Meyer, D.W. Kolpin, 1991: Herbicides in surface waters of the United States: the effect of spring flush. *Environmental Science and Technology*, **25**, 1794-1796.

**Thurman**, E.M., D.A. Goolsby, M.T. Meyer, M.S. Mills, M.L. Pomes, D.W. Kolpin, 1992: A reconnaissance study of herbicides and their metabolites in surface water of the mid-western United States using immunoassay and gas chromatography/mass spectrometry. *Environmental Science and Technology*, **26**, 2440-2447.

**Titchener**, J.L. and T.K. Frederic, 1976: Family and character change at Buffalo Creek. *American Journal of Psychiatry*, **133**, 295-299.

**Tomkins**, A.M., 1986: Protein-energy malnutrition and risk of infection. *Proceedings of the Nutrition Society*, **45**, 289-304.

**Touloumi**, G., S.J. Pocock, K. Katsouyanni, D. Trichopoulos, 1994: Short-term effects of air pollution on daily mortality in Athens: a time-series analysis. *International Journal of Epidemiology*, **23(5)**, 957-967.

**Tyrell**, R.M., 1994: The molecular and cellular pathology of solar ultraviolet radiation. *Molecular Aspects of Medicine*, **15(3)**, 1-77.

**UNEP**, 1994: *Environmental Effects of Ozone Depletion: 1994 Assessment*. UNEP, Nairobi, Kenya, 110 pp.

**U.S. EPA**, in press: *Preliminary Assessment of the Benefits to the US of Avoiding, or Adapting to Climate Change*. EPA Climate Change Division, Washington, DC.

**Vleodbeld**, M. and R. Leemans, 1993: Quantifying feedback processes in the response of the terrestrial carbon cycle to global change—modelling approach of image-2. *Water, Air and Soil Pollution,* **70**, 615-628.

**Walsh**, J.F., J.B. Davis, and R. Garms, 1981: Further studies on the reinvasion of the Onchocerciasis Control Programme by *Simulium damnosum s.l. Tropical Medicine and Parasitology*, **32(4)**, 269-273.

**Weihe**, W.H., 1986: *Life Expectancy in Tropical Climates and Urbanization*. Proc. WMO Technical Conference Urban Climate, Mexico City, 26-30 November 1984, WMO-No. 652, WMO, Geneva, Switzerland, 15 pp.

**Wenzel**, R.P., 1994: A new hantavirus infection in North America. *New England Journal of Medicine*, **330(14)**, 1004-1005.

**West**, S.K., F.S. Rosenthal, N.M. Bressler, S.B. Bressler, B. Munoz, S.L. Fine, and H.R. Taylor, 1989: Exposure to sunlight and other risk factors for age related macular degeneration. *Archives of Ophthalmology,* **107**, 875-879.

**White**, G.F., D.J. Bradley, A.U. White, 1972: *Drawers of Water: Domestic Water Use in East Africa*. University of Chicago Press, London, UK, 306 pp.

**WHO**, 1985: *Ten Years of Onchocerciasis Control in West Africa: Review of Work of the OCP in the Volta River Basin Area from 1974 to 1984*. OCP/GVA/85.1B. WHO, Geneva, Switzerland, 113 pp.

**WHO**, 1990: *Potential Health Effects of Climate Change: Report of a WHO Task Group*. WHO/PEP/90.10, WHO, Geneva, Switzerland, 58 pp.

**WHO**, 1994a: *Progress Report Control of Tropical Diseases*. CTD/MIP/94.4, unpublished document.

**WHO**, 1994b: *The Effects of Solar UV Radiation on the Eye. Report of an Informal Consultation, Geneva, 1993*. WHO, Geneva, Switzerland, 50 pp.

**WHO**, 1995a: *The World Health Report 1995: Bridging the Gaps*. WHO, Geneva, Switzerland, 118 pp.

**WHO**, 1995b: *Action Plan for Malaria Control 1995-2000*. Unpublished document.

**WHO**, 1995c: *Chagas Disease: Important Advances in Elimination of Transmission in Four Countries in Latin America*. WHO Press Office Feature No. 183, WHO, Geneva, Switzerland.

**Woodhouse**, P.R., K.T. Khaw, M. Plummer, A. Foley, T.W. Meade, 1994: Seasonal variation of plasma fibrinogen and factor VII activity in the elderly: winter infections and death from cardiovascular disease. *The Lancet*, **343**, 435-439.

**Woodward**, F.I., 1987: Stomatal numbers are sensitive to increases in $CO_2$ from pre-industrial levels. *Nature*, **327**, 617-618.

**World Bank**, 1993: *World Development Report 1993*. Oxford University Press, New York, NY, 329 pp.

**Worrest**, R.C. and L.D. Grant, 1989: Effects of ultraviolet-B radiation on terrestrial plants and marine organisms. In: *Ozone Depletion: Health and Environmental Consequences* [Jones, R. and T. Wigley (eds.)]. John Wiley and Sons, Chichester, UK, pp. 197–206.

**Yasumoto**, S., Y. Hayashi, and L. Aurelian, 1987: Immunity to herpes simplex virus type 2: suppression of virus-induced immune response in ultraviolet B-irradiated mice. *Journal of Immunology*, **139**, 2788-2793.

**Yoshikawa**, T., V. Rae, W. Bruins-Slot, J.-W. van den Berg, J.R. Taylor, and J.W. Streilein, 1990: Susceptibility to effects of UVB radiation on induction of contact hypersensitivity as a risk factor for skin cancer in man. *Journal of Investigative Dermatology*, **95**, 530-536.