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CENTENNIAL REVIEW

Climate change and health: global to local influences on disease risk

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The World Health Organization has concluded that the climatic changes that have occurred since the mid 1970s could already be causing annually over 150,000 deaths and five million disability-adjusted life-years (DALY), mainly in developing countries. The less developed countries are, ironically, those least responsible for causing global warming. Many health outcomes and diseases are sensitive to climate, including: heat-related mortality or morbidity; air pollution-related illnesses; infectious diseases, particularly those transmitted, indirectly, via water or by insect or rodent vectors; and refugee health issues linked to forced population migration. Yet, changing landscapes can significantly affect local weather more acutely than long-term climate change. Land-cover change can influence micro-climatic conditions, including temperature, evapo-transpiration and surface run-off, that are key determinants in the emergence of many infectious diseases. To improve risk assessment and risk management of these synergistic processes (climate and land-use change), more collaborative efforts in research, training and policy-decision support, across the fields of health, environment, sociology and economics, are required.

In the past half-century, global mean temperature has risen by 0.6°C, sea level has risen by a mean of 1–2 cm/decade, and ocean heat content has also measurably increased (Fig. 1; Anon., 2001). The rate of change in climate is faster now than in any period in the last 1000 years. Between 1990 and 2100, according to the United Nations Intergovernmental Panel on Climate Change (IPPC), mean global temperatures will increase by 1.4–5.8°C and sea level will rise by 9–88 cm, with mid-range estimates of 3°C and 45 cm, respectively (Anon., 2001). However, additional greenhouse-gas releases from warmer oceans (CO_2) and warmer soils (CO_2 and methane) will increase the estimated warming from human-induced emission another 2°C by the end of the century (Torn and Harte, 2006). Extremes of the hydrological cycle (e.g. floods and droughts) are expected to accompany the global warming.

NON-INFECTIOUS DISEASES

Heat Waves

Extremes in air temperature, both hot and cold, are associated with higher levels of human morbidity and mortality than seen within an intermediate or 'comfortable' range of temperatures. The relationship between temperature and mortality is typically 'J-shaped', indicating asymmetry, with a steeper slope at higher temperatures (Curriero *et al.*, 2002). In the U.S.A., heat waves are more deadly than hurricanes, floods and tornadoes combined.

The extreme heat wave that hit much of Europe in 2003 is estimated to have killed up to 45,000 people in just 2 weeks (Anon., 2004; Kosatsky, 2005). The summer of 2003 was probably Europe's hottest summer in >500 years, with mean temperatures 3.5°C

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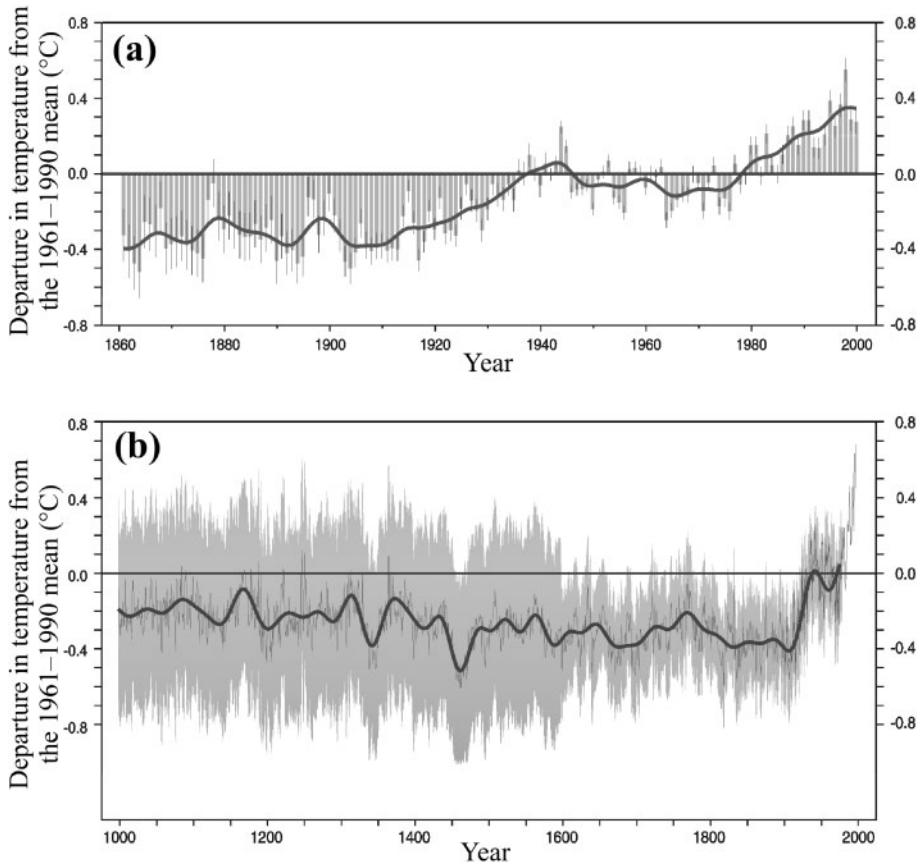

FIG. 1. Variations in the mean surface temperatures recorded (using thermometers) across the planet in the past 140 years (a) and (using a combination of tree-ring, coral and ice-core analysis and, for recent decades, thermometers) in the northern hemisphere over the past 10,000 years (b). These graphs are reprinted here with permission of the Intergovernmental Panel on Climate Change (Anon., 2001).

above normal (Beniston, 2004; Luterbacher *et al.*, 2004; Schar *et al.*, 2004). Although the level of temperature-related mortality seems to vary with geographical location, the temperature–mortality relationship found in European and North-American cities appears similar to that in São Paulo, a developing Brazilian city with sub-tropical conditions (Gouveia *et al.*, 2003). The results of the relevant studies conducted so far indicate a clear vulnerability to heat in the relatively cool, temperate regions, and tropical regions may show similar sensitivity as location-specific temperatures rise.

Built environments markedly modify the intensity of ambient temperatures, in a phenomenon known as the 'urban heat island effect'. Black asphalt and other dark surfaces (on roads, parking lots or roofs) reduce albedo (reflectivity) and consequently increase the heat retention of the surface. In addition, the loss of trees in urban areas diminishes the cooling effect of evapotranspiration. During heat waves, when stagnant atmospheric conditions may persist, air pollution often compounds the effects of the elevated air temperatures (Frumkin, 2002). Urban areas may therefore suffer from both global and localized warming.

Severe Storms and Rise in Sea Level

Floods, droughts and extreme storms have claimed millions of lives during the recent

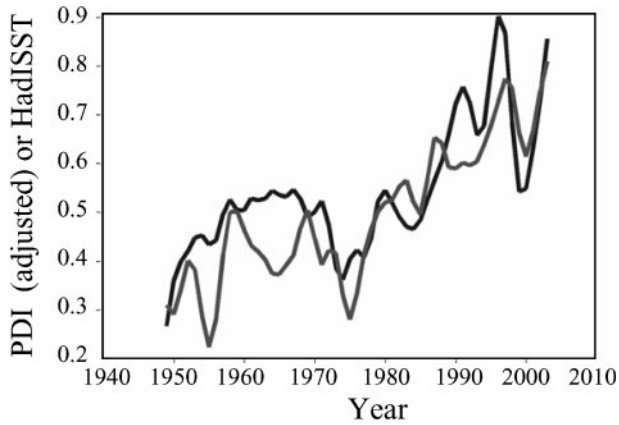

FIG. 2. The increasing trend in strong tropical storms seen over the last 50 years. For the plot, the power dissipation indices (PDI) for the Atlantic Ocean and Western Pacific (■) were adjusted (by multiplying them by a factor of 5.8×10^{-13}) so that they could be plotted on the same *y*-axis as the HadISST. The annual HadISST indices (■) — combined measures of sea-surface temperatures and sea-ice concentrations — were averaged between 30°S and 30°N, and the lines for both variables were smoothed (Emanuel, 2005). The PDI has nearly doubled over the past 30 years. Reproduced from a figure created by Emanuel (2005), with the permission of Nature Publishing.

past, and have adversely affected the lives of many more people. On average, disasters killed 123,000 people world-wide each year between 1972 and 1996. Africa suffers the highest rate of disaster-related deaths, even though 80% of the people affected by natural disasters are in Asia (Loretti and Tegegn, 1996). Disaster-related mental disorders, such as post-traumatic-stress disorder (PTSD), may substantially affect population well-being, depending upon the unexpectedness of the impact, the intensity of the experience, the degree of personal and community disruption, and the long-term exposure to the visual signs of the disaster.

Hurricanes only form in regions where sea surface temperatures exceed 26°C, and sea-surface warming by slightly more than 2°C intensifies hurricane wind speeds by 3–7 m/s (or 5%–12%) (Knutson *et al.*, 1998). Records indicate that sea-surface temperatures have steadily increased over the last 100 years, and more sharply over the last 35 years. The highest mean sea-surface temperatures ever recorded occurred between 1995 and 2004 (Trenberth, 2005). During the first half of this period, there was a doubling in the overall hurricane activity in the North Atlantic and a five-fold increase in such activity in the Caribbean (Goldenberg *et al.*, 2001). The North Atlantic Oscillation (NAO) was in its warm phase at this time, making it difficult to attribute the extra hurricanes to the long-term trends in warming. Sea-surface temperature is, however, correlated with hurricane intensity, and the frequency of higher-category storms has increased in many other parts of the world (Fig. 2; Emanuel, 2005).

RISE IN SEA LEVEL

Warmer oceans also cause sea levels to increase, primarily as the result of the thermal expansion of salt water. Even if the mid-range predictions of climate change are correct and sea levels in the 2080s are, on average, 'only' 40 cm higher than the current values, the coastal regions at risk of storm surges will become much greater and the population at risk will increase from the current 75 million to 200 million (McCarthy *et al.*, 2001). Rising sea levels will result in the salination of coastal freshwater aquifers and the disruption of storm-water drainage and sewage disposal. A case study for Bangladesh (Nicholls and Leatherman, 1995) indicates that >15% of

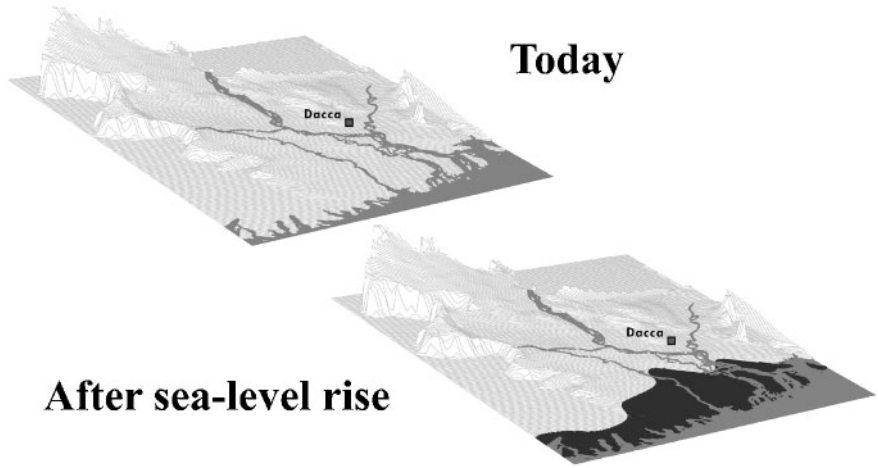

FIG. 3. The potential impact of sea-level rise on Bangladesh. If sea levels rose by 1.5 m, 17 million people (15% of the population) and an area of 22,000 km^2 would be affected. This figure is adapted from one produced by the United Nations Environment Programme (Patz, 2005) and is reprinted here with the permission of John Wiley and Sons.

the total population would be adversely affected by a 1.5-m rise in sea level (Fig. 3).

DROUGHTS

That droughts cause famines is well recognized. Malnutrition remains one of the largest health crises world-wide, with approximately 800 million people — close to half residing in Africa — currently undernourished (WHO, 2002). Droughts and other climate extremes not only have direct impacts on food crops but can also indirectly influence food supply by altering the ecology of plant pathogens. While projections of the effect of climate change on global food-crop production appear to be broadly neutral, such change will probably exacerbate regional inequalities in the food supply (Parry *et al.*, 2004). As there is a breakdown in sanitation as water resources become depleted, droughts can also increase the incidence of diarrhoea and diseases, such as scabies, conjunctivitis and trachoma, associated with poor hygiene (Patz and Kovats, 2002).

Air Quality and Climate

Air temperature affects the problems posed by air pollutants. Ground-level ozone smog tends to become worse with increasing air temperature but the relationship is non-linear, with a strong correlation only seen at temperatures above 32°C. In their recent study, Bell *et al.* (2006) predicted that, because of global warming, the mean number of days exceeding the health-based '8-h ozone standard' will increase by 60% in the eastern U.S.A. — from 12 to almost 20 days per summer — by the 2050s.

Pollen levels in the air may also increase with global warming, as higher levels of CO_2 promote growth and reproduction by many plants. When, for example, ragweed (*Ambrosia artemisiifolia*) plants were experimentally exposed to high levels of CO_2 they increased their pollen production several-fold; this response is perhaps part of the reason for rising levels of ragweed pollen observed in recent decades (Ziska and Caulfield, 2000; Wayne *et al.*, 2002). Ziska *et al.* (2003) found that ragweed grew faster, flowered earlier and produced more pollen in urban locations than in rural locations, presumably because of the relatively high air temperatures and CO_2 levels in the urban areas.

Finally, if the frequency of flooding increases, significant exposure to moulds may also pose respiratory health risks during the post-flood clean-ups (Patz *et al.*, 2001).

INFECTIOUS DISEASES

Water- and Food-borne Diseases

Water shortages, as mentioned above, contribute to diarrhoeal disease through poor hygiene, especially in poor countries. On the other hand, flooding can contaminate drinking water with run-off from sewage lines, containment lagoons (such as at animal-feeding operations), or conventional (non-point-source) pollution from across watersheds.

The parasites in the genus *Cryptosporidium* are usually associated with domestic livestock but can contaminate water intended for human consumption, especially during periods of heavy precipitation. In 1993 a cryptosporidiosis outbreak in Milwaukee, which killed more than 50 people and potentially exposed over 400,000 more to *Cryptosporidium*, coincided with unusually heavy spring rains and run-off from melting snow (Mac Kenzie *et al.*, 1994). A review of outbreaks of any water-borne disease in the U.S.A. over a 50-year period demonstrated a distinct seasonality, a spatial clustering in the key watersheds, and a strong association with heavy precipitation (Curriero *et al.*, 2001).

Certain food-borne diseases are also affected by fluctuations in temperature. Across much of continental Europe, for example, an estimated 30% of reported cases of salmonellosis occur when air temperatures are 6°C above the mean (Kovats *et al.*, 2004). In the U.K., the monthly incidence of food poisoning is strongly correlated with air temperatures in the previous 2–5 weeks (Bentham and Langford, 1995).

COASTAL WATERS

One type of phytoplankton, the dinoflagellates, thrive in warm waters with adequate nitrogen, and they are the primary component of toxic 'red tides'. They can cause acute paralytic, diarrhoeic, and amnesiac poisoning in humans, as well as extensive die-offs of fish and shellfish and the marine mammals and birds that depend on the marine food-web. The frequency and global distribution of toxic algal incidents and the incidence of human intoxication from algal sources appear to be increasing (Van Dolah, 2000).

Vibrio species also proliferate in warm marine waters. Zooplankton that feed on algae can serve as reservoirs for *Vibrio cholerae* and other enteric pathogens of humans. In Bangladesh, cholera follows the seasonal increase in sea-surface temperatures that can enhance plankton blooms (Colwell, 1996). During the El Niño event in 1997–1998, winter temperatures in Lima increased to $>5^{\circ}$C above normal, and the number of daily admissions for diarrhoea rose to levels that were twice as high as recorded, over the same months, in the previous 5 years (Checkley *et al.*, 2000). Although long-term studies of the El Niño Southern Oscillation (ENSO) have shown a consistent association with cholera and other diarrhoeal diseases, the oscillation appears to have played an increasing role in cholera outbreaks in recent years, perhaps because of concurrent climate change (Rodo *et al.*, 2002). A detailed understanding of the inter-annual cycles of cholera and other infectious diseases, however, requires the combined analyses of both environmental exposure and the host's intrinsic immunity to a disease. When they considered these factors together, Koelle *et al.* (2005) found that the inter-annual variability seen in cholera in Bangladesh was strongly correlated, across periods of <7 years, with sea-surface temperatures in the Bay of Bengal, ENSO and the extent of flooding in Bangladesh, and, across longer periods, with the monsoon rains and the discharge of the Brahmaputra river.

Vector-borne Diseases

As the human pathogens transmitted indirectly by insect or rodent vectors spend considerable time outside of their vertebrate

hosts, they may easily be affected by environmental conditions. The range of suitable climatic conditions within which each vector-borne pathogen and its vector can survive and reproduce is limited. The incubation time of a vector-borne infective agent within its vector is typically very sensitive to changes in temperature and humidity (Gubler *et al.*, 2001). The Table shows some examples of temperature thresholds.

MALARIA

Between 700,000 and 2.7 million people — mostly children in sub-Saharan Africa — die each year of malaria (www.cdc.gov/malaria), and, thanks to climate and land-use change, drug resistance, ineffective control efforts, and various socio–demographic factors, there is no evidence that malaria-attributable mortality is falling. Malaria is an extremely climate-sensitive tropical disease, making the assessment of the potential change in malarial risk, caused by past or projected global warming, one of the most important topics in the field of climate change and health (Patz *et al.*, 2005). The incidence of malaria varies seasonally in highly endemic areas, and malaria transmission has been associated with temperature anomalies in some African highlands (Zhou *et al.*, 2005). In the Punjab region of India, excessive monsoon rainfall and the resultant high humidity have been recognized for years as major factors in the occurrence of malaria epidemics. More recently in the region, the frequency of malaria epidemics was observed to increase approximately five-fold during the year following an El Niño event (Bouma and van der Kaay, 1996). In Botswana, Thomson *et al.* (2006) recently showed that indices of El Niño-related climate variability can serve as the basis of malaria-risk prediction and early warning.

HIGHLAND MALARIA. Air temperatures decrease by a mean of 6°C for every

TABLE. *Temperature thresholds of some human pathogens and their vectors**

	Pathogen			Vector	
		Threshold (°C)			
Disease	Name	Minimum for transmission	Maximum for survival	Name	Lower threshold (°C)
Malaria	*Plasmodium falciparum*	16–19	33–39	*Anopheles* mosquitoes	8–10 for biological activity
	P. vivax	14.5–15	33–39	*Anopheles* mosquitoes	8–10 for biological activity
Chagas disease	*Trypanosoma cruzi*	18	38	Triatomine bugs	2–6 for survival, 20 for biological activity
Schistosomiasis	*Schistosoma* spp.	14.2	>37	Snails (*Bulinus* and others)	5 for biological activity, 25 ± 2 as optimum
Dengue fever	Dengue virus	11.9	NYD	*Aedes* mosquitoes	6–10 for biological activity
Lyme disease	*Borrelia burgdorferi*	NYD	NYD	*Ixodes* ticks	5–8 for biological activity

*The thresholds shown assume optimum humidity (vector survival tends to decrease rapidly as dryness increases) and differ considerably within and between species. This table is based on one drawn up by the Intergovernmental Panel on Climate Change (Anon., 2001), using data from various studies (Purnell, 1966; Pfluger, 1980; Curto de Casas and Carcavallo, 1984; Molineaux, 1988; Rueda *et al.*, 1990), and is published here with permission of the Cambridge University Press.
NYD, Not yet determined.

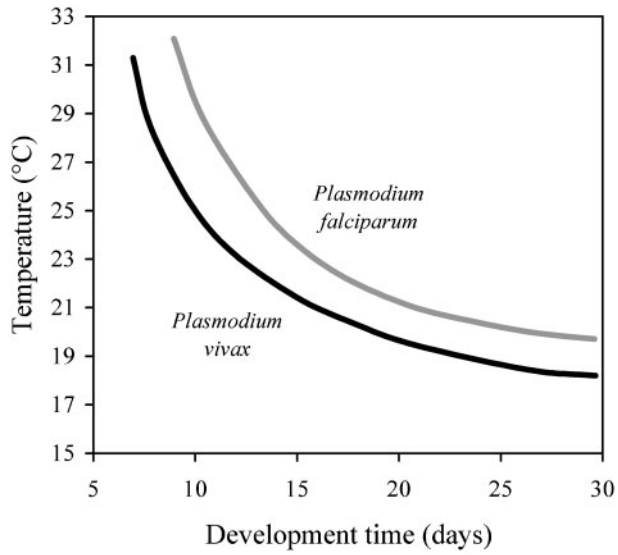

FIG. 4. As this graph produced by McDonald (1957) illustrates, air temperature has a marked effect on the extrinsic incubation periods (EIP — the times taken by the parasites to produce sporozoites in their mosquito vectors) of *Plasmodium falciparum* and *P. vivax*. As air temperatures rise, EIP shortens and so infected mosquitoes become infectious sooner. At or below minimum temperature thresholds — of about 18°C for *P. falciparum* and 15°C for *P. vivax* — no sporogony occurs and the infected mosquitoes never become capable of transmitting the parasites.

1000 m gained in elevation. In areas where human malaria is endemic, this effect usually precludes the transmission of malarial parasites at high altitudes, partly because the parasites cannot produce sporozoites in mosquitoes living at low temperatures. The minimum temperatures for the sporogony of *Plasmodium falciparum* and *P. vivax*, for example, are approximately 18°C and 15°C, respectively (Fig. 4). As seen in the African highlands (Bodker *et al.*, 2003), mosquito abundance tends to decrease with increasing altitude. Global warming is likely to result in an increase in the altitudes at which no malaria transmission occurs. In Africa, Tanser *et al.* (2003) estimated that the risk of exposure to malaria, measured in person-months, will be 16%–28% higher in 2100 than at present. Having compared climate suitability maps for malaria in the topographically diverse country of Zimbabwe, Ebi *et al.* (2005) concluded that the warming predicted from global-climate models could make the country's entire highland area climatologically suitable for malarial transmission by 2050. The highland areas of Africa that are not currently endemic for malaria but are, as the result of global warming, at high risk of becoming areas where transmission occurs are shown in Figure 5. Pascual *et al.* (2006) recently reported that the East African highlands had generally become warmer since 1950, over a period in which malaria incidence had also increased. There are well-recognized non-linear and threshold responses of malaria to the effect of regional temperature changes. In a form of biological 'amplification', the response of mosquito populations to warming can be more than an order of magnitude larger than the measured change in temperature, an increase of just 0.5°C translating into a 30%–100% increase in mosquito abundance (Pascual *et al.*, 2006). In the African highlands, where mosquito populations are relatively small compared with those in lowland areas (Minakawa *et al.*, 2002), such biological responses may be especially significant in determining the risk of malaria.

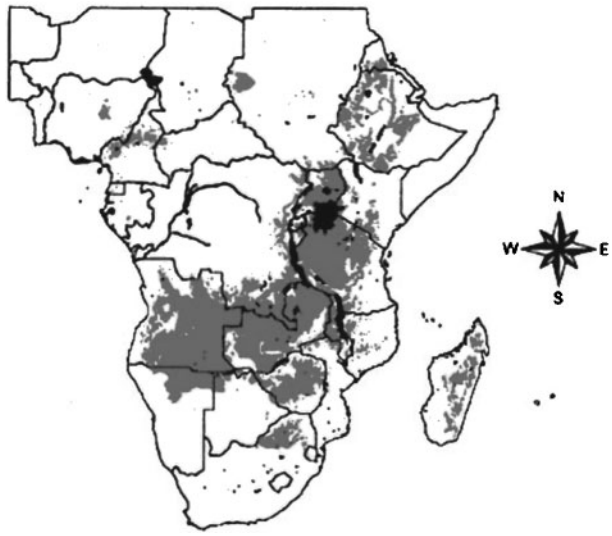

FIG. 5. Areas of the African highlands that, though currently non-endemic, are probably vulnerable to malaria as the result of climate warming (■). These areas, which are at altitudes of >1000 m, have ratios of precipitation to potential evapo-transpiration that exceed 0.5 during the five wettest consecutive months of the year, and have minimum temperatures in excess of 15°C, are considered to be on the threshold of malaria transmission. Reproduced, from a figure in an article by Patz and Lindsay (1999), with the permission of Elsevier.

MALARIA AND LOCAL EFFECTS ON CLIMATE FROM LAND-USE CHANGE. Changing landscapes can significantly affect local climate more acutely than long-term global warming. Land-cover change, for example, can influence the micro-climatic conditions, including temperature, evapo-transpiration and surface run-off (Foley *et al.*, 2005), that are key to determining mosquito abundance and survivorship. In Kenya, Afrane *et al.* (2005) observed that open treeless habitats had warmer mean midday temperatures than forested habitats, and that deforestation also affected indoor hut temperatures (Fig. 6). As a result, the gonotrophic cycle of female *Anopheles gambiae* s.l. during the dry and rainy seasons was found to be 2.6 days (52%) and 2.9 days (21%) shorter, respectively, in the deforested sites than in the forested. Similar findings have been documented in Uganda, where temperatures in communities bordering cultivated fields have been found higher than those in communities adjacent to natural wetlands, and the number of *An. gambiae* s.l./house has been found to increase with increasing minimum temperature, after adjustment for potentially confounding variables (Lindblade *et al.*, 2000). In Kenya, mosquito breeding sites in farmland have been found to be relatively warm and this warmth speeds up the development of the immature insects (Munga *et al.*, 2006). Increased canopy cover in western Kenya is negatively associated with the presence of larval *An. gambiae* s.l. and *An. funestus* in natural aquatic habitats (Minakawa *et al.*, 2002). In artificial pools, survivorship of the larvae of *An. gambiae* s.s. in sunlit open areas was 50-fold higher than that in forested areas, and also related to assemblages of predatory species (Tuno *et al.*, 2005). In short, deforestation and cultivation of natural swamps in the African highlands creates conditions favourable for the survival of *An. gambiae* larvae, making an analysis of the effects of land-use change on local climate, habitat, and biodiversity key to any malaria-risk assessments.

Deforestation has also affected malaria in other regions, such as the Amazon basin (Guerra *et al.*, 2006). Vittor *et al.* (2006)

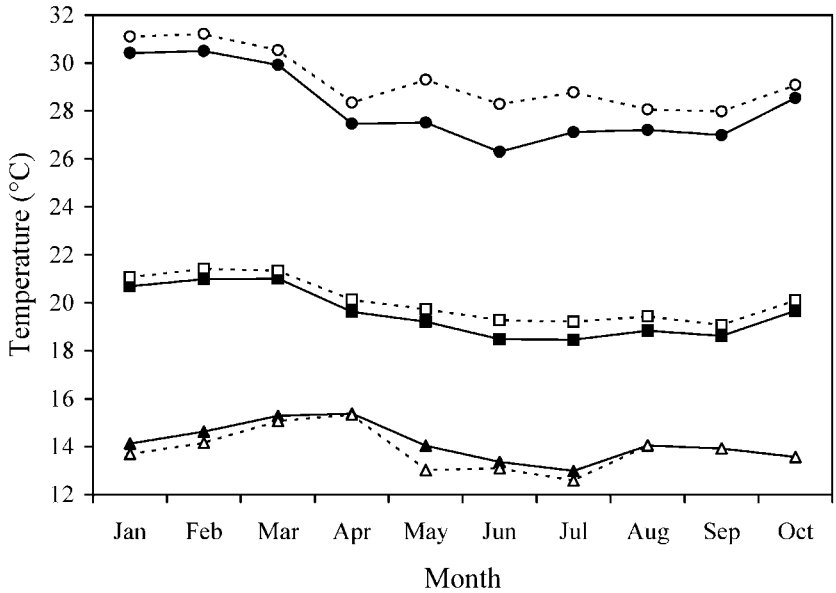

FIG. 6. Comparison of the maximum (○), mean (□) and minimum (Δ) temperatures recorded within huts in deforested agricultural lands with the corresponding maximum (●), mean (■) and minimum (▲) temperatures recorded within huts in forests. This graph was produced by Afrane *et al.* (2005), using data collected in western Kenya, and is reprinted here with the permission of the Entomological Society of America.

found a strong association between the biting rates of *An. darlingi* and the extent of deforestation in the Amazon; after controlling for the variation in human population densities, the biting rates of *An. darlingi* were still >200-fold higher in sites experiencing >80% deforestation than in sites with <30% deforestation.

Human activities have the capacity to shift the biodiversity of local ecosystems rapidly, intentionally and unintentionally increasing or decreasing malarial risk factors by altering the environment and mosquito habitat. The direction of the trend depends heavily on the *Anopheles* species present and on local conditions (Guerra *et al.*, 2006). In north–eastern India, expansive deforestation has caused the numbers of *An. dirus* and *An. culicifacies* to decline (Dev *et al.*, 2003). The effects of changing land-use patterns on the regulation of malaria (or other infectious disease) across a large area are species- and site-specific, and therefore cannot be generalised.

ARBOVIRUSES

Although *Aedes aegypti* is known to be strongly affected by ecological and human 'drivers' in urban settings, this species is also influenced by climate, including variability in temperature, moisture and solar radiation. Similar to the extrinsic incubation periods of malarial parasites (Fig. 4), the rate of replication of dengue virus in *Ae. aegypti* increases directly with air temperature, at least in the laboratory. Biological models have been developed to explore the influence of projected temperature change on the incidence of dengue fever. These models indicate that, given viral introduction into a susceptible human population, relatively small increases in temperature could significantly increase the potential for epidemics of dengue (Patz *et al.*, 1998). In addition, for relatively small countries with presumably **some** climate uniformity, a climate-based dengue model has been developed that strongly correlates with the inter-annual variability seen in the incidence

of dengue reported at the national level (Fig. 7; Hopp and Foley, 2003).

Certain other arboviruses, such as Saint Louis encephalitis virus (SLEV), are also associated with climatic factors. In Florida, the appearance of SLEV in sentinel chicken flocks is preceded by a wet period followed by drought (Shaman *et al.*, 2002). It has been suggested that spring drought forces the mosquito vector, *Culex nigripalpus*, to converge with immature and adult wild birds in restrictive, densely vegetated, hammock habitats. This forced interaction of mosquito vectors and avian hosts then creates an ideal setting for rapid transmission and amplification of SLEV. Once the drought ends and water sources are restored, the infected vectors and hosts disperse and transmit SLEV to a much broader geographical area (Shaman *et al.*, 2002).

Climate variability may also have an effect on West Nile virus (WNV), a pathogen only recently introduced into the New World. Reisen *et al.* (2006) found that the strain of WNV that entered New York, during the record hot July of 1999, differed from the South African strain in that it required warmer temperatures for efficient transmission. It seems likely that, during the epidemic summers of 2002–2004 in the U.S.A., epicentres of WNV were linked to above-average temperatures.

Rodent-borne Diseases

Hantavirus is transmitted to humans largely by exposure to infectious rodent excreta, and may then cause serious disease, with a high level of mortality. In the emergence of hantavirus pulmonary syndrome in the south–western U.S.A., in 1993, it was the weather conditions, especially El Niño-driven heavy rainfall, that appear to have led to a growth in rodent populations and subsequent viral transmission (Glass *et al.*, 2000).

Extreme flooding or hurricanes can lead to outbreaks of leptospirosis. In 1995, an epidemic of this disease occurred in Nicaragua after heavy flooding, and a major risk factor for the disease was found to be walking through the flood waters (Trevejo *et al.*, 1998).

ATTRIBUTION OF DISEASE BURDEN RESULTING FROM CLIMATE CHANGE

The World Health Organization (WHO) has examined the global burden of disease already attributable to anthropogenic climate change up to the year 2000 and made model-based forecasts of the health risks from global climate change up to the year 2030 (McMichael *et al.*, 2004). Conservative assumptions were made about climate–health relationships (e.g. that socio–economic conditions would prevent a climate-driven spread of vector-borne disease from endemic tropical regions to temperate regions) and many plausible health impacts were excluded for lack of quantitative models. The results indicate that the current burden from climate-sensitive diseases such as diarrhoea, malaria and malnutrition is so large that even the subtle climatic changes that have occurred since the mid-1970s could already be causing >150,000 deaths and approximately 5 million disability-adjusted life-years (DALY) each year. Although climate change is a global threat to public health, the WHO's assessment also revealed that the poorer regions of the world may be the most vulnerable (Fig. 8). When the WHO's estimates of morbidity and mortality caused by human-induced climate change were extrapolated to 2030, it was found that the climate-change-induced excess risk of the various health outcomes considered could more than double by that year (McMichael *et al.*, 2004).

CONCLUSIONS

The health outcomes from climate change are diverse and occur via multiple pathways

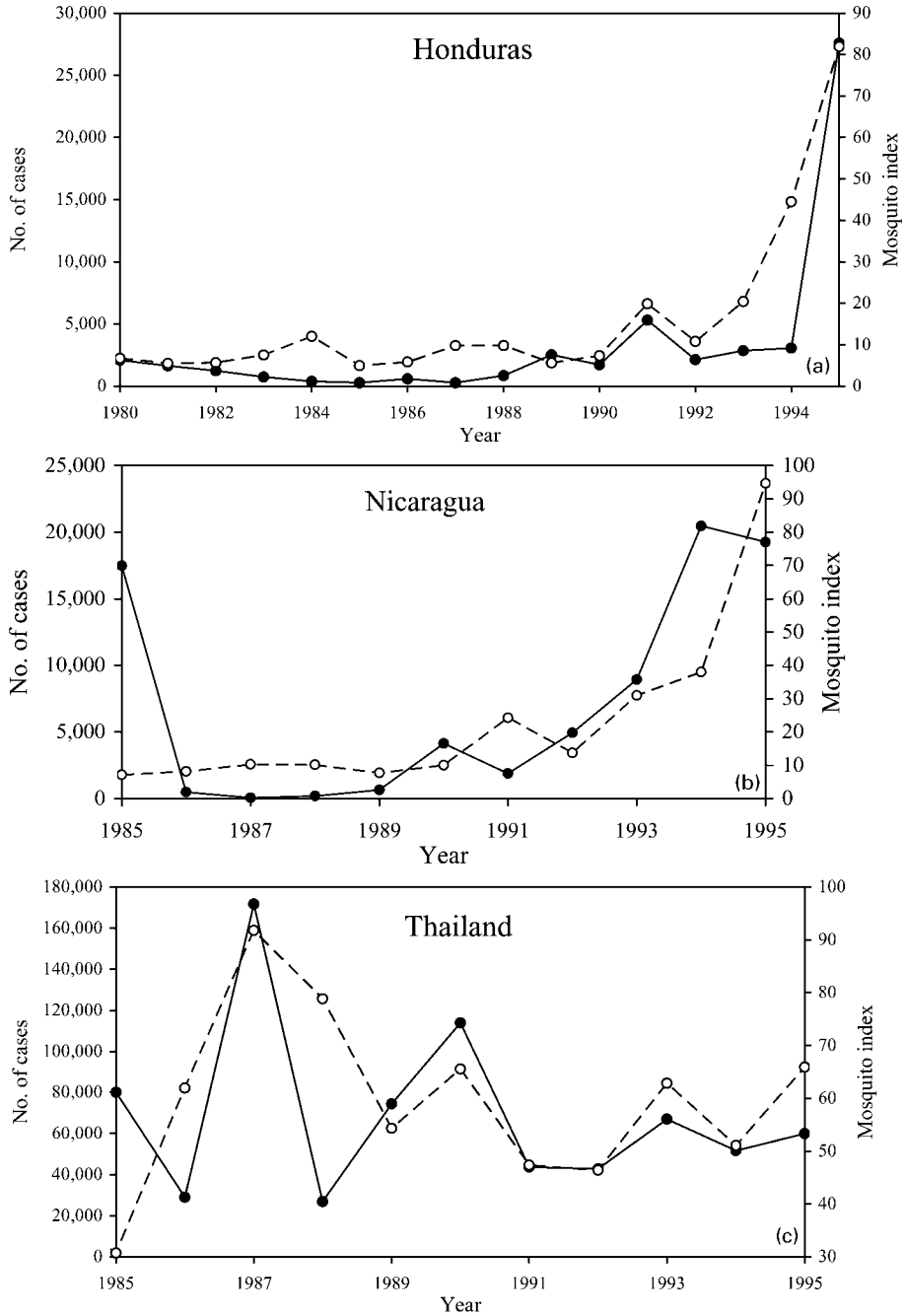

FIG. 7. Correlation between simulated, climate-driven variations in *Aedes aegypti* mosquito density (○) and observed variations in the annual numbers of cases (●) of dengue, including dengue haemorrhagic fever, in three countries. Using a computer model of mosquito physiology and development, estimated changes in the relative abundance of *Ae. aegypti* that were driven only by month-to-month and year-to-year variations in temperature, humidity, solar radiation and rainfall were analysed. In Honduras, Nicaragua and Thailand (and many other 'small-area' countries of Central America and South–east Asia), disease incidence and the modelled mosquito densities were found to be significantly correlated ($P<0.05$ for each). These graphs are adapted from those of Hopp and Foley (2003) and are produced here with the permission of Nature Publishing.

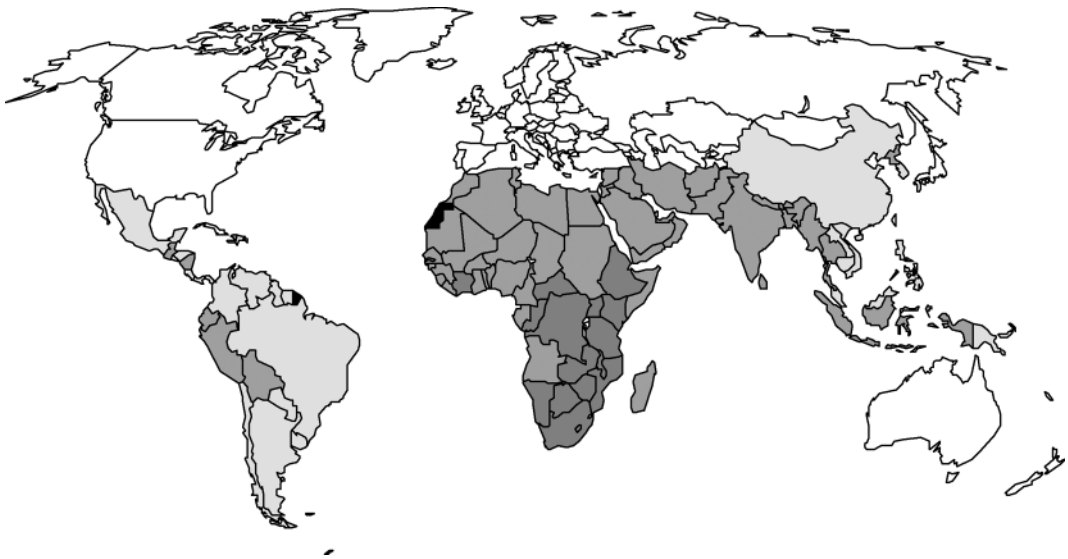

FIG. 8. The World Health Organization's estimates of mortality attributable to climate change by the year 2000: 0–2 (□), >2–4 (▫), >4–70 (▪) or >70–120 (■) deaths/million people (no estimate could be made for Western Sahara or French Guiana because of a lack of data). The Intergovernmental Panel on Climate Change's 'business-as-usual' scenario for greenhouse-gas (GHG) emissions (IS92a) and the HadCM2 general-circulation model of the Hadley Centre for Climate Prediction and Research (Exeter, U.K.) were used to estimate climate changes relative to the 1961–1990 levels of GHG and associated climate conditions used as a 'baseline'. The results of earlier quantitative studies on climate–health relationships were used to estimate relative changes in a range of climate-sensitive health outcomes for the years from 2000 to 2030. The outcomes considered (cardiovascular diseases, diarrhoea, malaria, inland and coastal flooding, and malnutrition) form only a partial list of the potential health outcomes, and there are significant uncertainties in all of the underlying models. The results should therefore be considered as conservative and approximate estimates of the health burden of climate change. Even so, the total mortality due to anthropogenic climate change by 2000 is estimated to be at least 150,000 people/year. Reproduced from a figure created, using data from McMichael *et al.* (2004), by Patz *et al.* (2005), and reprinted here with the permission of Nature Publishing.

of exposure. Whereas some disease resurgence has been attributed to recent warming trends, some of the long-term and complex problems posed by climate change may not be readily discernible from other causal factors. Accordingly, expanded efforts are required in both classical and future-scenario-based risk assessment, to anticipate these problems. In addition, the many health impacts of climate change must be examined in the context of many other environmental and behavioral determinants of disease. Increased disease surveillance, integrated modelling, and the use of geographically-based data systems will enable more anticipatory measures by the public-health and medical communities.

There are clear ethical challenges. The regions with the greatest burden of climate-sensitive diseases are often the regions with the lowest capacity to adapt to the new risks. Many of the regions most vulnerable to climate change are also those least responsible for causing the problem. Africa, for example, is thought to harbour about 70% of all malaria cases but has the lowest per-capita emissions of the 'greenhouse' gases that cause global warming. In today's globalized world, with its international trade and travel, an increase in disease anywhere on the globe can affect every country.

Health is just one of the many sectors expected to be affected by climate change. It represents just a part of the interconnected

context in which decision makers must implement strategies to prevent or reduce the adverse effects of such change. To achieve the greatest disease prevention, 'upstream' environmental approaches, rather than assaults on single agents of disease, must form part of any intervention. If the truly global public-health challenge of climate change is to be adequately addressed, an unprecedented co-operation between natural and social/health scientists, as well as between rich and poor countries, must occur.

REFERENCES

Afrane, Y. A., Lawson, B. W., Githeko, A. K. & Yan, G. (2005). Effects of microclimatic changes caused by land use and land cover on duration of gonotrophic cycles of *Anopheles gambiae* (Diptera: Culicidae) in western Kenya highlands. *Journal of Medical Entomology*, **42**, 974–980.

Anon. (2001). *Climate Change 2001: the Scientific Basis: Contribution of Working Group I to the Third Assessment Report*. Cambridge, U.K.: Cambridge University Press.

Anon. (2004). *World Disasters Report 2004*. Geneva: International Federation of Red Cross and Red Crescent Societies.

Bell, M. L., Goldberg, R., Hogrefe, C., Kinney, P., Knowlton, K., Lynn, B., Rosenthal, J., Rosenzweig, C. & Patz, J. A. (2006). Climate change, ambient ozone, and health in 50 U.S. cities. *Climatic Change*, in press.

Beniston, M. (2004). The 2003 heat wave in Europe: a shape of things to come? An analysis based on Swiss climatological data and model simulations. *Geophysical Research Letters*, **31**, L02202.

Bentham, G. & Langford, I. H. (1995). Climate change and the incidence of food poisoning in England and Wales. *International Journal of Biometeorology*, **39**, 81–86.

Bodker, R., Akida, J., Shayo, D., Kisinza, W., Msangeni, H. A., Pedersen, E. M. & Lindsay, S. W. (2003). Relationship between altitude and intensity of malaria transmission in the Usambara Mountains, Tanzania. *Journal of Medical Entomology*, **40**, 706–717.

Bouma, M. J. & van der Kaay, H. J. (1996). The El Nino Southern Oscillation and the historic malaria epidemics on the Indian subcontinent and Sri Lanka: an early warning system for future epidemics? *Tropical Medicine and International Health*, **1**, 86–96.

Checkley, W., Epstein, L. D., Gilman, R. H., Figueroa, D., Cama, R. I., Patz, J. A. & Black, R. E. (2000). Effect of El Nino and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children. *Lancet*, **355**, 442–450.

Colwell, R. R. (1996). Global climate and infectious disease: the cholera paradigm. *Science*, **274**, 2025–2031.

Curriero, F., Patz, J., Rose, J. & Lele, S. (2001). The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948–1994. *American Journal of Public Health*, **91**, 1194–1199.

Curriero, F. C., Heiner, K. S., Samet, J. M., Zeger, S. L., Strug, L. & Patz, J. A. (2002). Temperature and mortality in 11 cities of the eastern United States. *American Journal of Epidemiology*, **155**, 80–87.

Dev, V., Bhattacharyya, P. C. & Talukdar, R. (2003). Transmission of malaria and its control in the northeastern region of India. *Journal of the Association of Physicians of India*, **51**, 1073–1076.

Ebi, K. L., Hartman, J., Chan, N., McConnell, K. J., Schlesinger, M. & Weyant, J. (2005). Climate suitability for stable malaria transmission in Zimbabwe under different climate change scenarios. *Climatic Change*, **73**, 375–393.

Emanuel, K. (2005). Increasing destructiveness of tropical cyclones over the past 30 years. *Nature*, **436**, 686–688.

Foley, J. A., Defries, R., Asner, G. P., Barford, C., Bonan, G., Carpenter, S. R., Chapin, F. S., Coe, M. T., Daily, G. C., Gibbs, H. K., Helkowski, J. H., Holloway, T., Howard, E. A., Kucharik, C. J., Monfreda, C., Patz, J. A., Prentice, I. C., Ramankutty, N. & Snyder, P. K. (2005). Global consequences of land use. *Science*, **309**, 570–574.

Frumkin, H. (2002). Urban sprawl and public health. *Public Health Reports*, **117**, 201–217.

Glass, G. E., Cheek, J. E., Patz, J. A., Shields, T. M., Doyle, T. J., Thoroughman, D. A., Hunt, D. K., Enscore, R. E., Gage, K. L., Irland, C., Peters, C. J. & Bryan, R. (2000). Using remotely sensed data to identify areas at risk for hantavirus pulmonary syndrome. *Emerging Infectious Diseases*, **6**, 238–247.

Goldenberg, S. B., Landsea, C. W., Mestas-Nuñez, A. & Gray, W. M. (2001). The recent increase in Atlantic hurricane activity: causes and implications. *Science*, **293**, 474–479.

Gouveia, N., Hajat, S. & Armstrong, B. (2003). Socioeconomic differentials in the temperature–mortality relationship in Sao Paulo, Brazil. *International Journal of Epidemiology*, **32**, 390–397.

Gubler, D. J., Reiter, P., Ebi, K. L., Yap, W., Nasci, R. & Patz, J. A. (2001). Climate variability and change in the United States: potential impacts on vector- and rodent-borne diseases. *Environmental Health Perspectives*, **109**, 223–233.

Guerra, C. A., Snow, R. W. & Hay, S. I. (2006). A global assessment of closed forests, deforestation and malaria risk. *Annals of Tropical Medicine and Parasitology*, **100**, 189–204.

Hopp, M. J. & Foley, J. A. (2003). Worldwide fluctuations in dengue fever cases related to climate variability. *Climate Research*, **25**, 85–94.

Knutson, T. R., Tuleya, R. E. & Kurihara, Y. (1998). Simulated increase of hurricane intensities in a CO_2-warmed climate. *Science*, **276**, 1018–1020.

Koelle, K., Rodo, X., Pascual, M., Yunus, M. & Mostafa, G. (2005). Refractory periods and climate forcing in cholera dynamics. *Nature*, **436**, 696–700.

Kosatsky, T. (2005). The 2003 European heat waves. *Euro Surveillance*, **10**, 148–149.

Kovats, R. S., Edwards, S. J., Hajat, S., Armstrong, B. G., Ebi, K. L. & Menne, B. (2004). The effect of temperature on food poisoning: a time-series analysis of salmonellosis in ten European countries. *Epidemiology and Infection*, **132**, 443–453.

Lindblade, K. A., Walker, E. D., Onapa, A. W., Katungu, J. & Wilson, M. L. (2000). Land use change alters malaria transmission parameters by modifying temperature in a highland area of Uganda. *Tropical Medicine and International Health*, **5**, 263–274.

Loretti, A. & Tegegn, Y. (1996). Disasters in Africa: old and new hazards and growing vulnerability. *World Health Statistics Quarterly*, **49**, 179–184.

Luterbacher, J., Dietrich, D., Xoplaki, E., Grosjean, M. & Wanner, H. (2004). European seasonal and annual temperature variability, trends, and extremes since 1500. *Science*, **303**, 1499–1503.

Mac Kenzie, W. R., Hoxie, N. J., Proctor, M. E., Gradus, M. S., Blair, K. A., Peterson, D. E., Kazmierczak, J. J., Addiss, D. G., Fox, K. R., Rose, J. B. & David, J. P. (1994). A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. *New England Journal of Medicine*, **331**, 161–167.

McCarthy, J. J., Canziani, O., Leary, N., Kokken, D. & White, K. (2001). *Climate Change 2001: Impacts, Adaptation, and Vulnerability*. New York, NY: Cambridge University Press.

McDonald, G. (1957). *The Epidemiology and Control of Malaria*. Oxford, U.K.: Oxford University Press.

McMichael, A. J., Campbell-Lendrum, D. H., Kovats, S., Edwards, S., Wilkinson, P. W., Wilson, T., Nicholls, R., Hales, S., Tanser, F. C., Le Sueur, D., Schlesinger, M. & Andronova, N. (2004). Global climate change. In *Comparative Quantification of Health Risks: Global and Regional Burden of Disease due to Selected Major Risk Factors*, eds Ezzati, M., Lopez, A. D., Rodgers, A. & Murray, C. J. L. pp. 1543–1649. Geneva: World Health Organization.

Minakawa, N., Sonye, G., Mogi, M., Githeko, A. & Yan, G. (2002). The effects of climatic factors on the distribution and abundance of malaria vectors in Kenya. *Journal of Medical Entomology*, **39**, 833–841.

Munga, S., Minakawa, N., Zhou, G., Mushinzimana, E., Barrack, O. O., Githeko, A. K. & Yan, G. (2006). Association between land cover and habitat productivity of malaria vectors in Western kenyan highlands. *American Journal of Tropical Medicine and Hygiene*, **74**, 69–75.

Nicholls, R. & Leatherman, S. (1995). Global sea-level rise. In *As Climate Changes: International Impacts and Implications*, eds Strzepek, K. & Smith, J. pp. 92–123. New York, NY: Cambridge University Press.

Parry, M. L., Rosenzweig, C., Iglesias, A., Livermore, M. & Fischer, G. (2004). Effects of climate change on global food production under SRES emissions and socio–economic scenarios. *Global Environmental Change — Human and Policy Dimensions*, **14**, 53–67.

Pascual, M., Ahumada, J. A., Chaves, L. F., Rodo, X. & Bouma, M. J. (2006). Malaria resurgence in East African highlands: temperature trends revisited. *Proceedings of the National Academy of Sciences of the United States of America*, **103**, 5829–5834.

Patz, J. A. (2005). Climate change. In *Environmental Health: from Global to Local*, ed. Frumkin, H. pp. 000–000. San Francisco, CA: John Wiley & Sons.

Patz, J. A. & Kovats, R. S. (2002). Hotspots in climate change and human health. *British Medical Journal*, **325**, 1094–1098.

Patz, J. A. & Lindsay, S. W. (1999). New challenges, new tools: the impact of climate change on infectious diseases. *Current Opinion in Microbiology*, **2**, 445–451.

Patz, J. A., Martens, W. J. M., Focks, D. A. & Jetten, T. H. (1998). Dengue fever epidemic potential as projected by general circulation models of global climate change. *Environmental Health Perspectives*, **106**, 147–153.

Patz, J. A., McGeehin, M. A., Bernard, S. M., Ebi, K. L., Epstein, P. R., Grambsch, A., Gubler, D. J., Reiter, P., Romieu, I., Rose, J. B., Samet, J. M. & Trtanj, J. (2001). The potential health impacts of climate variability and change for the United States. Executive summary of the report of the health sector of the U.S. National Assessment. *Journal of Environmental Health*, **64**, 20–28.

Patz, J. A., Campbell-Lendrum, D., Holloway, T. & Foley, J. A. (2005). Impact of regional climate change on human health. *Nature*, **438**, 310–317.

Reisen, W. K., Fang, Y. & Martinez, V. (2006). Effects of temperature on the transmission of West Nile virus by *Culex tarsalis* (Diptera: Culicidae). *Journal of Medical Entomology*, **43**, in press.

Rodo, X., Pascual, M., Fuchs, G. & Faruque, A. S. (2002). ENSO and cholera: a non-stationary link related to climate change? *Proceedings of the National*

Academy of Sciences of the United States of America, **99**, 12,901–12,906.

Schar, C., Vidale, P. L., Luthi, D., Frei, C., Haberli, C., Liniger, M. A. & Appenzeller, C. (2004). The role of increasing temperature variability in European summer heatwaves. *Nature*, **427**, 332–336.

Shaman, J., Stieglitz, M., Stark, C., Le Blancq, S. & Cane, M. (2002). Using a dynamic hydrology model to predict mosquito abundances in flood and swamp water. *Emerging Infectious Diseases*, **8**, 6–13.

Tanser, F. C., Sharp, B. & Le Sueur, D. (2003). Potential effect of climate change on malaria transmission in Africa. *Lancet*, **362**, 1792–1798.

Thomson, M. C., Doblas-Reyes, F. J., Mason, S. J., Hagedorn, R., Connor, S. J., Phindela, T., Morse, A. P. & Palmer, T. N. (2006). Malaria early warnings based on seasonal climate forecasts from multi-model ensembles. *Nature*, **439**, 576–579.

Torn, M. S. & Harte, J. (2006). Missing feedbacks, asymmetrie uncertainties, and the underestimation of future warming. *Geophysical Research Letters*, **33**, L10703.

Trenberth, K. (2005). Uncertainty in hurricanes and global warming. *Science*, **308**, 1753–1754.

Trevejo, R. T., Rigau-Perez, J. G., Ashford, D. A., McClure, E. M., Jarquan-Gonzalez, C., Amador, J. J., de los Reyes, J. O., Gonzalez, A., Zaki, S. R., Shieh, W. J., McLean, R. G., Nasci, R. S., Weyant, R. S., Bolin, C. A., Bragg, S. L., Perkins, B. A. & Spiegel, R. A. (1998). Epidemic leptospirosis associated with pulmonary hemorrhage — Nicaragua, 1995. *Journal of Infectious Diseases*, **178**, 1457–1463.

Tuno, N., Okeka, W., Minakawa, N., Takagi, M. & Yan, G. (2005). Survivorship of *Anopheles gambiae sensu stricto* (Diptera: Culicidae) larvae in western Kenya highland forest. *Journal of Medical Entomology*, **42**, 270–277.

Van Dolah, F. M. (2000). Marine algal toxins: origins, health effects, and their increased occurrence. *Environmental Health Perspectives*, **108**, 133–141.

Vittor, A. Y., Gilman, R. H., Tielsch, J., Glass, G., Shields, T., Lozano, W. S., Pinedo-Cancino, V. & Patz, J. A. (2006). The effect of deforestation on the human-biting rate of *Anopheles darlingi*, the primary vector of falciparum malaria in the Peruvian Amazon. *American Journal of Tropical Medicine and Hygiene*, **74**, 3–11.

Wayne, P., Foster, S., Connolly, J., Bazzaz, F. & Epstein, P. (2002). Production of allergenic pollen by ragweed (*Ambrosia artemisiifolia* L.) is increased in CO_2-enriched atmospheres. *Annals of Allergy, Asthma and Immunology*, **88**, 279–282.

World Health Organization (2002). *The World Health Report 2002*. Geneva: WHO.

Zhou, G., Minakawa, N., Githeko, A. K. & Yan, G. (2005). Climate variability and malaria epidemics in the highlands of East Africa. *Trends in Parasitology*, **21**, 54–56.

Ziska, L. H. & Caulfield, F. A. (2000). Rising carbon dioxide and pollen production of common ragweed, a known allergy-inducing species: implications for public health. *Australian Journal of Plant Physiology*, **27**, 893–898.

Ziska, L. H., Gebhard, D. E., Frenz, D. A., Faulkner, S., Singer, B. D. & Straka, J. G. (2003). Cities as harbingers of climate change: common ragweed, urbanization, and public health. *Journal of Allergy and Clinical Immunology*, **111**, 290–295.