Climate change and the distribution and intensity of infectious diseases

RICHARD S. OSTFELD[1]

Cary Institute of Ecosystem Studies, P.O. Box AB, Millbrook, New York 12545 USA

Many infectious diseases of humans, including malaria, dengue, cholera, and schistosomiasis, are restricted to, or more prevalent in, tropical and subtropical zones. Within the tropics and subtropics, they are more prevalent at lower than at higher altitudes. Warmer temperatures characteristic of lower latitudes and altitudes generally increase rates of survival, development, and replication of parasites and of blood-feeding vectors such as mosquitoes. Warmer conditions also increase activity (including biting) rates of vectors, resulting in higher rates of parasite transmission (reviewed by Harvell et al. 2002). A series of papers in the 1990s (e.g., Shope 1991, Martens et al. 1995, Colwell 1996, McMichael et al. 1996, Patz et al. 1996) contended that recent and future trends in climate warming were likely to increase the incidence and geographic distribution of infectious diseases, particularly those caused by vector-borne and water-borne parasites and pathogens. Owing to media attention and popular concern, the spread of infectious diseases was featured in the growing list of negative outcomes known or anticipated to arise from anthropogenic climate change (e.g., Intergovernmental Panel on Climate Change 2001; the 2006 documentary film, *An Inconvenient Truth*). However, unequivocal demonstrations of a causal link between climate change and human infectious diseases are rare (albeit increasing). Some diseases are likely to decrease in incidence and range with climate warming (Harvell et al. 2002), and others are likely to respond to precipitation or humidity more than to temperature, leading to poor predictive power under warming scenarios. Many diseases are strongly influenced by other ecological, sociological, economic, and evolutionary factors besides climate change. These latter observations have stimulated the emergence of critics of a climate-change–infectious-disease linkage. Lafferty (2009) provides an overview of recent criticisms, emphasizing three major categories: (1) In many cases, we should expect diseases to shift geographically without net expansion under climate change; (2) non-climatic factors are more important than climate; and (3) models that predict increasing disease transmission with climate warming are flawed if transmission rates fail to exceed a specific

threshold ($R_0 > 1$) that allows disease persistence. Here, I discuss these criticisms in turn.

Diseases might shift without expanding.—The geographic range of diseases might fail to expand under climate warming scenarios due to complex relationships between temperature and vital rates of parasites or vectors. Given an optimal temperature range for parasite or vector fitness, temperature increases beyond the optimum will reduce disease transmission. The critical issue for the climate-change–disease debate is whether local temperatures will exceed the optimal range for specific parasites or vectors. The Intergovernmental Panel on Climate Change (2007) Synthesis Report synthesizes the evidence that local maximum temperatures will increase only modestly, while minimum temperatures will increase dramatically under climate change scenarios. The stability of temperature maxima compared to minima extends to both daily and seasonal variation. Consequently, when temperature optima exist, we can reasonably expect that climate warming will affect parasite and vector performance asymmetrically, with temperatures more likely to move from below optimum to optimum, as compared to moving from optimum to above optimum. Therefore, experiments in which temperatures raised above the range of natural variation cause declines in parasite performance (e.g., Dawson et al. 2005) do not necessarily mean that real climate warming will reduce parasitism. Of course, these expectations should be addressed empirically.

An influential study by Rogers and Randolph (2000) created a statistical model to predict future changes in the distribution of falciparum malaria under climate warming scenarios. Rogers and Randolph mapped the recorded, present-day geographic distribution of *falciparum* malaria cases. They then ascertained the boundary conditions, based on the mean, maximum, and minimum of temperature, precipitation, and saturation vapor pressure, within which *falciparum* malaria is currently reported, and outside of which it is not. Using general circulation model scenarios of climate change, they projected where those boundary conditions are likely to occur in 2050. One model, the HadCM2 "medium–high" scenario, predicted 23 million additional people living in malarious areas, whereas another, the HadCM2 "high" scenario predicted 25 million fewer people living in malarious areas (Rogers and Randolph 2000). In essence, they predicted that *falciparum* malaria will shift without expanding. However, these models are

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predicated on the assumption that climate, and only climate, determines the present-day distribution of *falciparum* malaria, which is known to be false (Reiter 2001). Historically, malaria (from several *Plasmodium* species including *falciparum*) occurred throughout temperate Eurasia and North America. A combination of habitat management and insecticide use is largely responsible for its large-scale eradication by the middle of the 20th century. Clearly, the actual range of climatic conditions permissive to malaria is much broader than represented by the model of Rogers and Randolph (2000). Projections from a model based on only a subset of favorable climatic conditions are likely to include only a subset of the geographical areas into which malaria can expand in the future. Thus, model conservatism might be extreme.

Non-climatic factors are more important than climate.—Clearly, many factors besides climate can be crucial in determining the incidence and distribution of infectious diseases (examples in Lafferty 2008). For instance, the removal of mosquito breeding sites and use of insecticides has been critical not only in elimination of malaria from historically impacted areas, but also in preventing range expansions of diseases like dengue and yellow fever, which are transmitted by *Aedes* mosquitoes. If these interventions are sustained, they can counteract incipient range expansions driven by warming climatic conditions. However, anti-mosquito interventions such as wetland and storm runoff management, elimination of container breeding sites (e.g. discarded tires), and window screens or bed nets, are often prohibitive in economically depressed regions. Similarly, appliances that reduce human encounter rates with mosquitoes such as air conditioners, television, and other electronic devices, require economic development and infrastructure less available in many countries at risk. The argument that interventions can prevent the advance of infectious diseases as the climate warms, therefore, applies much less effectively to developing than to developed countries.

Incidence of malaria at high-elevation sites in East Africa has increased dramatically since the 1970s. Responding to the assertion that increases in highland malaria are being caused by anthropogenic climate warming, Hay et al. (2002) assessed trends in monthly mean values of surface climatic variables at four high-altitude East African sites during most of the 20th century. Their analysis indicated that conditions warm and wet enough to facilitate *falciparum* malaria transmission had not changed significantly during this time, and they claimed that factors other than climate change must be responsible for the resurgence of highland malaria (Hay et al. 2002). However, a rigorous reanalysis of the East African climate data updated through 2002 clearly shows a significant warming trend at all four sites (Pascual et al. 2006). Furthermore, using the direct and detrended temperature data to drive a dynamic population model for mosquito vectors,

Pascual et al. (2006) found that even small changes in the climate signal can dramatically amplify mosquito abundance. In highland areas with relatively low mosquito numbers, even small increases in mosquito abundance strongly increase malaria transmission. An appropriate alternative to rejecting climate effects in favor of unidentified factors would be to test a series of potential causal variables including climate.

Failure to exceed a transmission threshold.—As described by Lafferty (2009), early models that compared the transmission potential of malaria before and after projected changes in climate predicted a 60% increase in malaria incidence by 2020 (Martens 1999). These models have been criticized for relying on climate-caused increases in suitability for malaria transmission *relative to prior conditions* rather than on estimated *absolute values* for malaria transmission (Rogers and Randolph 2000). According to this argument, any climate-caused increases in transmission potential are epidemiologically irrelevant if climate warming fails to cause R_0 (the average number of infected individuals resulting from a single infected host arriving in a population that is entirely susceptible) to exceed 1.0. This argument overlooks at least two critical observations. The first is that historical records of the distribution of malaria (e.g., Reiter 2001) include large geographical areas (e.g., Europe, North America) within which R_0 is projected to be <1.0. This observation suggests that values of R_0 are sometimes underestimated. Second, $R_0 < 1.0$ does not negate the possibility of disease invasion, particularly when an initial outbreak is caused by simultaneous arrival of many infected hosts. For diseases with average values of $R_0 < 1.0$, the size of an outbreak depends largely on the number of primary cases (Hudson et al. 2008). Dispersal of large numbers of *Plasmodium*-infected people into areas where residents are entirely susceptible seems plausible, especially given increasing impacts of environmental degradation, economic crises, and armed conflicts on human movement patterns. Therefore, it seems reasonable to model climate-driven increases in suitability for disease transmission, irrespective of the absolute (estimated) value of R_0, when projecting changes in the distribution or incidence of disease.

Concluding thoughts.—Clear effects of climate change have now been established for several human infectious diseases, including malaria (Pascual et al. 2006), cutaneous leishmaniasis (Chaves and Pascual 2006), cholera (Koelle et al. 2005), plague (Stenseth et al. 2006, Snäll et al. 2008), and dengue (Cazelles et al. 2005), as well as for livestock (Gubbins et al. 2008), wildlife (Harvell et al. 2002), and coral diseases (Harvell et al. 2002, Bruno et al. 2007). The complexities of these systems pose enormous challenges for the detection of climate effects, and for the isolation and integration of climatic and non-climatic effects. Most of the studies cited above were able to detect a climate signal because they (1) obtained high-quality data over long time

periods; (2) explicitly accounted for nonstationarity in the time series data so that climate forcing could be isolated from other trends; (3) incorporated both extrinsic (e.g., climatic) effects and intrinsic ones (e.g., build-up of immunity in host populations) in models of disease dynamics; (4) confronted the data with models including specific, biologically interpretable climatic variables (e.g., season-specific temperature or precipitation, ENSO index); and (4) incorporated potential effects of climate on reservoir hosts, as well as on vectors and pathogens. The increasing quality of data and sophistication of analytical methods give hope that progress will continue. Even with these advances, the debate about the magnitude of climate warming effects on the future distribution and incidence of infectious diseases is not likely to end soon. Unbiased critiques of claims and counter-claims are an essential component of this debate. Hay et al. (2002) close their critique of climate drivers of highland malaria by asserting, "The more certain climatologists become that humans are affecting global climates, the more critical epidemiologists should be of the evidence indicating that these changes affect malaria" (Hay et al. 2002:909). Good science demands that skepticism be applied equally to evidence for and against climatic effects on disease.

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