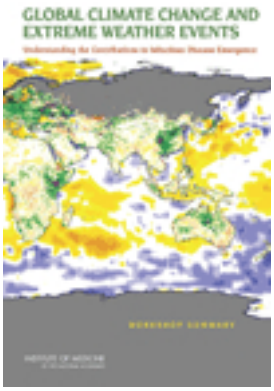

Global Climate Change and Extreme Weather Events: Understanding the Contributions to Infectious Disease Emergence: Workshop Summary

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Climate, Ecology, and Infectious Disease

OVERVIEW

As depicted in the convergence model of infectious disease emergence, illustrated in Figure SA-4, climate interacts with, and can alter, the complex ecological relationships underlying infectious disease transmission patterns. This chapter examines such interactions from several perspectives:

- Their consequences throughout the aquatic-marine food web, which defines ecological relationships for water-dwelling animals
- In patterns of distribution and transmission dynamics of individual infectious diseases (cholera, Rift Valley fever, chikungunya, and plague)
- Their effects on the dynamics of plant diseases, and their effects on agriculture and natural ecosystems
- As manifested in the public health challenges posed by climate change to human populations in the Arctic

Research on the effects of climate variation on infectious disease incidence and geographic range in these diverse contexts is providing the basis for developing climate-based early warning systems for disease risk. Such studies also represent a necessary first step toward anticipating how climate change may alter infectious disease dynamics in various ecological frameworks.

In her workshop presentation, Leslie Dierauf, director of the U.S. Geological Survey's National Wildlife Health Center in Madison, Wisconsin, described the apparent and predicted effects of climate on a broad cross-section of animal species that inhabit fresh- and saltwater ecosystems, as well as the intertidal

zones that unite aquatic and marine environments. Ecological connections among these environments are illustrated in Figure SA-8, which depicts the marine food web.

Dierauf also emphasized the physical connectedness of aquatic and marine environments, which makes it possible for infectious diseases of fish and wildlife to move from freshwater sources to intertidal zones to marine environments, affecting species that may not have encountered these disease agents before. Salmon, for example, hatch in small freshwater streams, travel hundreds of kilometers downstream to the ocean where they live for several years, only to return to the same streams where they hatched to spawn and die shortly thereafter. Thus, she observed, "if the temperature of the streams changes or the fish themselves pick up novel disease agents, because a vector, or an intermediate host, or a disease agent thrives in the new warmer environment, infectious disease may result."

Evidence-based studies of the effects of climate change on the health of aquatic and marine wildlife are few, Dierauf reported; therefore, current understanding of this topic derives from such sources as historical comparisons (of climatic conditions and of animal health and behaviors), long-term ecological research, correlation studies, and recognition of the physical, chemical, and biological processes governing climate change. Following the flow of water from inland streams to estuaries and into the open ocean, Dierauf considered the possible impacts of climate change in each of the three main elements of the aquatic continuum and how these changes may affect the health of their animal inhabitants.

In freshwater ecosystems, extreme weather events that produce flooding can trigger overwhelming influxes of nutrients into ecosystems. Storms can cause a range of environmental disturbances; Dierauf described the release of Nile tilapia into Mississippi streams from aquaculture facilities damaged by Hurricane Katrina. Several emerging diseases of inland aquatic animals, described and depicted in Box SA-2 in the Summary and Assessment, may also be influenced by climate change.

Intertidal areas, such as salt marshes and estuaries, are essential for maintaining a delicate balance among many complex and interactive variables (such as temperature, light, salinity, wave action, sea level rise, erosion, and sediment deposition) that characterize the transition from freshwater to saltwater environments, Dierauf explained. Storms, such as hurricanes, greatly affect intertidal zones. Heavy inland rainfall increases the speed and volume of the run-off that reaches estuaries, while marine storms drive saltwater and its contents past the intertidal buffer, affecting inland ecosystem health.

Climate change is expected to produce a range of important effects on oceans (as well as on large, deep-water lakes such as the Great Lakes), according to Dierauf. These include increased wave intensity, increased nutrient turnover, changes in nutrients, and changes in the food web. In addition, she noted, higher

concentrations of atmospheric carbon dioxide are dramatically increasing the acidity of ocean waters, which in turn is weakening the carbonate shells and skeletons of many marine species that comprise coral reef systems. She also noted the effects of harmful algal blooms (HABs), which are thought to result from nutrient influxes to the ocean (see Summary and Assessment). HABs appear to be increasing in both frequency and size as the climate warms, she said; this could result from increased upwelling of nutrients within the ocean or changes in ocean currents, as well as from the effects of extreme weather events inland. "What we do know is that HABs are affecting and often killing living things in the food web, like zooplankton, shellfish, fish, birds, and marine mammals, like manatees," she said.

Ocean warming, which is reducing the availability of food and sea ice for marine mammals, may also be compromising their resistance to infectious disease, Dierauf said. "Already, climate change and thinning of sea ice has reduced the time mother polar bears have to build the fat stores they need to sustain themselves over winter and to feed their young come spring when they emerge from their dens," she noted. Faced with shortages of food in their native waters, some marine mammals move to new territories where they both encounter and introduce novel disease agents (see Summary and Assessment).

"Climate change and climate variability will affect aquatic and marine species worldwide," Dierauf concluded. "We must act now at personal, professional, local, and global levels to protect vulnerable ecosystems and the aquatic and marine species that depend on these habitats for survival."

In contrast to the broad perspective on the effect of climate change on aquatic ecosystems offered by Dierauf, this chapter's first paper, by Rita Colwell of the University of Maryland, focuses on the specific and well-characterized effects of climate on cholera, a water-borne disease that affects an estimated 100,000 people per year, resulting in 10,000 deaths. The incidence and distribution of cholera are controlled by water temperature, precipitation patterns, and water salinity—all of which are influenced by global climate—and conducted through a complex web of ecological relationships. Sanitation and infrastructure also play a role in the incidence and distribution of cholera. Colwell noted, however, that "by simply educating women to filter drinking water through several layers of 'sari cloth,' we were able to reduce cholera incidence by 50 percent." Colwell described how, over the course of decades, she and coworkers deduced the circumstances under which the causal agent of cholera, the bacterium *Vibrio cholerae*, is transmitted to humans by the plankton species with which the bacterium associates. This knowledge led to the development of remote sensing systems capable of predicting the onset of cholera epidemics in the Ganges delta, known as the "home of cholera," because of its long history of epidemic disease.

This chapter's second paper also describes the use of remote sensing to monitor the effects of climate variation on specific infectious diseases. Speaker Jean-Paul Chretien, of the Department of Defense Global Emerging Infections

Surveillance and Response System (DOD-GEIS), and coauthors describe the use of satellite and epidemiological data to examine connections between the El Niño/Southern Oscillation (ENSO) and recent epidemics of two mosquito-borne viral diseases: Rift Valley fever (RVF) and chikungunya fever. In the first case, the association of RVF outbreaks in East Africa with periods of heavy rainfall, which occur during the El Niño phase of ENSO, led researchers to develop a model to forecast RVF risk in that region based on vegetation density (a marker for rainfall), as measured by satellite (Linthicum et al., 1999). The authors describe the operation of this model in the El Niño season of 2006-2007, when its prediction of elevated risk of disease prompted intensified surveillance for RVF in Kenya and, ultimately, to an international effort to stem a pending epidemic.

Chikungunya fever caused a series of outbreaks along the Kenyan coast in 2004, from which it apparently spread to several western Indian Ocean islands and India, resulting in the largest chikungunya fever epidemic on record (Chretien et al., 2007). At the time of the initial outbreaks in Kenya, a regional drought—corresponding to the La Niña phase of ENSO—had gripped the region. Chretien and coauthors discuss several possible, nonexclusive mechanisms connecting the epidemic with the drought, some of which may have also have influenced the first appearance of chikungunya fever in Europe in 2007.

In the chapter's third paper, speaker Nils Stenseth of the University of Oslo provides a much longer view of climate variation and its effects on infectious disease dynamics. Throughout recorded history, the various forms of plague, caused by the bacterium *Yersinia pestis* and transmitted by fleas among a wide range of hosts, are known to have caused both endemic and epidemic disease. Stenseth examines the dynamic ecology and epidemiology of plague in its ancient reservoir in Central Asia, and compares these patterns with local climate variation over the course of decades (as recorded in regular measurements of temperature and rainfall) and centuries (as reflected in tree-ring data for the past 1,000 years).

Using data collected twice annually between 1949 and 1995 in Kazakhstan, a focal region for plague where human cases are regularly reported, Stenseth and colleagues determined that *Y. pestis* prevalence increases dramatically in its primary host, the great gerbil (*Rhombomys opimus*), during warmer springs and wetter summers (Stenseth et al., 2006). Rodent populations also tend to increase under these conditions and, along with them, the possibility that plague will be transmitted to humans. Analyses of historical climate variation, as reflected in tree-ring patterns, suggest that similar warm, wet conditions existed in Central Asia during the onset of the Black Death in the fourteenth century, as well as in the years preceding a mid-nineteenth-century plague pandemic. As Earth's climate warms, warmer springs and wetter summers are expected to become more common in Central Asia (as well as in North America) therefore raising the possibility that plague activity—and therefore the potential for epidemic disease—will increase.

"Although the number of human cases of plague is relatively low, it would

be a mistake to overlook its threat to humanity, because of the disease's inherent communicability, rapid spread, rapid clinical course, and high mortality if left untreated," Stenseth notes. Moreover, he adds, even a minor plague outbreak can result in panic, with severe economic repercussions; a 1994 plague outbreak in India that caused 50 deaths also led to a nationwide collapse in tourism and trade, costing the nation an estimated $600 million (Fritz et al., 1996). "Plague remains a fairly poorly understood threat that we cannot afford to ignore," Stenseth concludes. "Only by knowing more about how the eco-epidemiological plague systems in the different parts of the world will respond to given climate scenarios can we take the necessary precautionary measures to reduce the risks of human infections."

While climate-based early warning systems for human disease are in an early stage of development, plant disease forecasting systems based on variables such as temperature and precipitation have been used for many years, according to speaker Karen Garrett of Kansas State University. However, she adds, these well-established models will need to be adapted (based on sound science) to account for climate change, as will plant disease management policies that flow from climate-based forecasts. In her contribution to this chapter, Garrett establishes a framework for this critical effort. She describes standard methods for managing plant disease, reviews observed effects of climate variation on plant diseases and their implications given projected future climatic conditions, and discusses research and policy needs for plant disease management in response to climate change. In considering the consequences of climate change for plant health, Garrett emphasizes threshold effects: environmental perturbations that produce disproportionate ecological upheaval. Examples of such thresholds include longer growing seasons; pathogen introductions and range shifts; pathogen overwintering; and the removal of constraints on pathogen reproduction at a critical population size.

Much as it has been argued that the most effective available protective measures against the adverse human health effects of climate change are basic public health interventions (see Campbell-Lendrum in Chapter 4), Garrett observes that "the good news for formulation of strategies for plant disease management under changing climate conditions is that much of what needs to be done is the same with or without climate change." Thus, she advocates research to advance our understanding of plants' adaptive capacities and mechanisms, and policies to encourage the development of "diverse, flexible, and resilient agricultural systems that can adapt more readily to new climatic conditions."

The chapter's final paper, by Alan Parkinson of the Centers for Disease Control and Prevention's (CDC's) Arctic Investigations Program in Anchorage, Alaska, presents a panoramic view of the public health challenges faced by people living in the Arctic, where the physical effects of climate change are dramatically apparent. Temperatures in this region have increased at nearly twice the global average over the past century, causing widespread melting of land and

sea ice (see Figure SA-13; Borgerson, 2008; IPCC, 2007). These conditions are exposing the Arctic's human inhabitants, many of whom have limited access to public health and/or sanitation services, to an increasingly broad range of infectious disease threats (among other health challenges). Parkinson describes the observed and projected effects of climate change in the Arctic environment, discusses the direct effects of higher ambient temperatures on the health of Arctic inhabitants, and catalogs the many ways in which climate change may increase the risk of infectious disease for Arctic residents.

Indeed, Parkinson observes, infectious disease risks are already increasing in the Arctic through the indirect influence of climate change on the populations and ranges of disease vector species (e.g., mosquitoes, ticks) and the population density and range of reservoir hosts that can transmit disease (e.g., rodents, foxes). Flooding and the loss of permafrost are also damaging the sanitation infrastructure of Arctic communities, thereby increasing the risk of water-borne infectious diseases, respiratory diseases, and skin infections. Meanwhile, increasing mean ambient temperatures raise the risk of food-borne diseases, particularly for Arctic residents who rely on traditional methods of subsistence and food preservation (e.g., fermentation, air-drying, burying).

In the face of these public health challenges, Parkinson recommends a range of public health responses, including monitoring of high-risk, climate-sensitive infectious diseases with potentially large public health impacts (e.g., water-borne diseases such as giardiasis), prompt investigation of infectious disease outbreaks that may be related to climate change, and research on the relationship between climate and infectious disease emergence to guide early detection and public health interventions. He also encourages the creation of infectious disease monitoring networks to connect typically small, isolated Arctic communities and link them to regional, national, and international health organizations. Such networks would encourage the standardization of monitoring methods, the sharing of data, and the detection of infectious disease trends over a larger geographic area.

THE MARINE ENVIRONMENT AND HUMAN HEALTH: THE CHOLERA MODEL

Rita Colwell, Ph.D.[1]
University of Maryland

Cholera, a disease I have studied for more than 30 years, is a model of the complex interactions between climate, ecology, environment, and weather related to epidemics of infectious diseases. Revealing cholera's secrets has required inter-

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disciplinary research examining all of these influences, as well as a point of view that I call biocomplexity: recognizing that infectious diseases operate on a wide range of time and space scales. Thus, we employ gene probes, environmental measurements (ground truth), and other precise techniques for pathogen detection, but at the same time, we take a holistic approach that integrates information from the atomic to the atmospheric—and perhaps, in some cases, even the cosmic—in order to build a predictive model for cholera outbreaks.

Cholera is a significant, global public health problem, as shown in Table 2-1. Annually, it results in approximately 100,000 hospitalizations and approximately 10,000 deaths, varying from year to year. A few cases of cholera appear each year in the United States, usually associated with seafood harvested from closed beds near sewage outfalls in the Gulf of Mexico.

Most of my group's research on cholera has focused on the Ganges delta, which feeds into the Bay of Bengal. This area is known as the home of cholera due to spring and fall epidemics, of varying but predictable intensity, that have recurred there for hundreds of years (see Figure 2-1). During the monsoon season, flooding rains wash nutrients down from the Himalayas, while winds drive water from the Bay of Bengal up into the Ganges and its tributaries, creating ideal conditions (discussed later) for cholera outbreaks. The fall 2007 epidemic, which followed massive flooding, was catastrophic. The Center for Diarrheal Disease

TABLE 2-1 Cholera Cases Officially Reported to WHO, 2004—Selected Countries

Country	Number of Cases	Imported	Deaths	Mortality Rate (%)
Benin	642		9	1.40
Burundi	819		14	1.71
Cameroon	8,005		137	1.71
Comoros	1		0	0.00
Côte d'Ivoire	105		9	8.57
DROC (Congo)	7,665		228	2.97
Niger	2,178		57	2.62
Nigeria	3,186		185	5.81
Somalia	4,490		26	0.58
Uganda	3,380		91	2.69
Tanzania	10,319		272	2.64
Zambia	12,149		373	3.07
Zimbabwe	119		9	7.56
India	4,695		7	0.15
Japan	66	55	0	0.00
Singapore	11	1	1	9.09
Total	57,830	56	1,418	2.45

SOURCE: WHO (2005).

FIGURE 2-1 Bangladesh border, barrier islands, and location of Dacca, Matlab, Mathbaria, and Bakerganj.
SOURCE: Printed with permission from Google.

Research in Dacca admitted about a thousand new cases per day for almost 30 days and had to use temporary space to house cholera victims. We are working to create predictive models to provide advance warning of conditions that produce severe epidemics in this region of the world.

However, *V. cholerae,* the bacterium, is a natural inhabitant of rivers, estuaries, and coastal waters throughout the world. Currently, we are sequencing approximately 50 different strains of *Vibrio cholerae*, the causative agent of cholera collected from many geographic locations to examine their genetic relationships. Preliminary sequencing studies of *V. cholerae* collected at a depth of 2,000 m at a site located off the coast of Oregon indicate that those isolates may represent ancestral strains; interestingly, one strain studied in detail has genes in common with other *Vibrio* pathogens, as well, including *Vibrio vulnificus* and *Vibrio parahaemolyticus*, the latter being the most common food-borne pathogen in Asian countries, where raw seafood is consumed.

The Ecology of Cholera

My laboratory accomplished the first isolation of *Vibrio cholerae* from the Chesapeake Bay more than two decades ago, and we now know that this bacterium is found in estuaries of similar salinity, (*ca.* 15 parts per thousand), where the water temperature rises seasonally to 15ºC or higher and where an influx of nutrients encourages plankton blooms (Colwell, 1996). Other species of *Vibrio*, including *V. parahaemolyticus* and *V. vulnificus*, also thrive under these conditions. One of my current graduate students, Brad Haley, has just returned from Iceland, where he was able to isolate *V. cholerae* at locations where geothermal effluent flows into bays. Clearly, water temperature is critical to the growth of this pathogen.

Vibrio cholerae also has a dormant state, which it assumes between epidemics and during which it cannot be cultured but can be detected with probes (fluorescent antibodies and gene signature sequences). Only during the peak of the zooplankton bloom, in the spring and the fall, is *V. cholerae* easily culturable. We were able to show that by adding nalidixic acid and nutrient (yeast extract) to water containing the quiescent bacterium, we can stimulate cell elongation and metabolism.

Another important discovery was that cholera is transmitted by plankton. Thus, it is not enough to say that its growth correlates with sea surface temperature and salinity; it is important to recognize the ecological interactions that produce these correlations. There is a commensal relationship—which may prove to be symbiosis—between *Vibrio* bacteria and zooplankton. Vibrios are chitinolytic (i.e., capable of breaking down chitin, the material that forms the carapaces of zooplankton and crustaceans (e.g., crabs, shrimp). *V. cholerae* also produces a powerful proteolytic enzyme that the bacterium apparently uses to perform an additional function for zooplankton: breaking down its egg sac, enabling the eggs to disperse into the water column. We are discovering that interactions between *V. cholerae* and various zooplankton species are quite intricate; for example, certain strains of the bacterium attach preferentially to certain species of zooplankton (Rawlings et al., 2007). All of this leads to the conclusion that *V. cholerae* is integral to marine ecosystems, and therefore cannot be eradicated.

The Epidemiology of Cholera

We have determined in earlier studies that between 10,000 and 50,000 *Vibrio cholerae* bacteria may be attached to an individual copepod (the zooplankton favored by *V. cholerae*). A liter of water drawn by a villager from a pond in Bangladesh between epidemics may contain 10 copepods. However, during a zooplankton bloom, that concentration can increase a hundredfold or more per liter, carrying a dose of cholera bacteria sufficient to cause cholera. The severity of the disease is dose dependent: a low concentration of bacterial cells will pro-

duce mild diarrhea; hospitalized cases—which represent about 25 percent of all infections—require more since one million bacteria per milliliter has been shown to be required to produce the disease. Thus, it has been estimated that only 25 percent of those with cholera end up in hospitals and many more may have been infected (Colwell and Huq, 2004).

Cholera is a disease with rapid onset. Within 24 to 48 hours, the typical patient can lose up to 18 liters of fluid. If that fluid can be replenished quickly, either intravenously or through oral rehydration (using a simple mixture of bicarbonate of soda, table salt, and sugar), recovery is fairly rapid.

From years of study in Bangladesh, we have determined several factors that interact and are associated with the massive annual biennial (spring and fall) cholera epidemics, so that we can predict the onset and severity of epidemics. Our recent research focuses on the communities of Mathbaria and Bakerganj, which are located in the barrier islands region of the Ganges delta (see Figure 2-1). Mangrove-based ecosystems are abundant in copepods. Thus, the *Vibrio* population is also abundant, and during the zooplankton/*Vibrio* bloom, cholera results from drinking untreated water.

In Bakerganj and Mathbaria, copepods comprise the majority of zooplankton species. We now have evidence that the severity of a given local cholera epidemic can be determined by copepod population dynamics, with intense epidemics occurring during times of abundance of those copepod species to which epidemic strains of *V. cholera* preferentially attach. We are currently conducting a seasonal study of zooplankton species in an attempt to determine which species carry *V. cholera* and to identify factors that influence population size; we will use this information, with other environmental data, to build a predictive capacity for cholera epidemics.

We are also using our knowledge of cholera epidemiology to help the people of Bangladesh to avoid contracting cholera. In one study, for example, we found that by simply educating women to filter drinking water through several layers of sari cloth, we were able to reduce cholera incidence by 50 percent. This result supported our hypothesis that plankton and particulates—to which the bacteria are attracted—transmit cholera and when removed by simple filtration, the incidence of the disease is significantly reduced.

Predictive Models of Cholera

Currently, the spring bloom of phytoplankton in the Bay of Bengal can be measured by satellite sensors that measure chlorophyll intensity and, therefore, the phytoplankton population. Phytoplankton blooms are followed by zooplankton blooms, but the latter cannot yet be measured directly by satellite sensors. However, the zooplankton peak can be inferred using a series of calculations from measurements of the phytoplankton populations that precede the zooplankton

population peak. This information taken together with salinity, temperature, and other environmental factors, provides a more complete picture.

We have also gathered ground truth data over the past 10 years in the Bakerganj area, including conductivity of the water, presence of inorganic nutrients, temperature, and salinity. With these data, we are able to improve our prediction of the timing and, possibly, the severity of cholera epidemics.

In our original work, we were able to use satellite imagery to measure sea surface temperature and sea surface height in the Bay of Bengal. As shown in Figure 2-2, the correlation of chlorophyll and temperature data, measured by satellite sensors, provides a predictive capacity for conditions conducive to cholera outbreaks. We are currently working on a predictive model that takes into account ocean currents to monitor the movement of plankton into the Bay of Bengal estuaries from the southern tip of India. This could provide as much as a 3-month warning prior to an impending cholera outbreak.

In Latin America, the 1991-1992 El Niño event corresponded with a cholera epidemic that was initially attributed to the dumping of ballast water by a ship in the harbor of Lima, Peru (Gil et al., 2004). We were able to disprove this hypothesis by demonstrating that cholera outbreaks had occurred in three different cities along the coast of Peru, starting before the peak of the 1991-1992 El Niño event. The epidemic more likely resulted from the effect of increased sea surface temperatures on existing plankton and *V. cholerae* populations.

Our most sophisticated predictive model for cholera incorporates chlorophyll, sea surface height, temperature, and extensive ground truth data. Within a few years, the National Oceanic and Atmospheric Administration (NOAA) will launch a satellite that may provide salinity data. We are also refining our model, based on the 40 years of data accumulated on cholera in Bangladesh and in India, which we are presently analyzing. Nevertheless, with the analyses we have performed to date—sea surface temperature and sea surface height from satellite sensors; measurements of chlorophyll intensity (corrected for the time lag from chlorophyll-phytoplankton bloom to the zooplankton bloom that feeds on the phytoplankton); and measurements of vibrio dispersion in the water—we are able to determine significant correlations and, thus, a foundation from which to predict cholera epidemics.

Conclusion

Climate change is likely to increase the burden of cholera in Bangladesh, but even greater suffering will occur if sea levels rise to predicted levels, displacing millions of people. However, our interdisciplinary, international (as demonstrated by our large number of collaborators from many countries), and biocomplexity approach to studying cholera extends well beyond Bangladesh and even beyond the disease itself. By gaining an understanding of the complex interactions between infectious disease, ecology, and the physical environment, we can

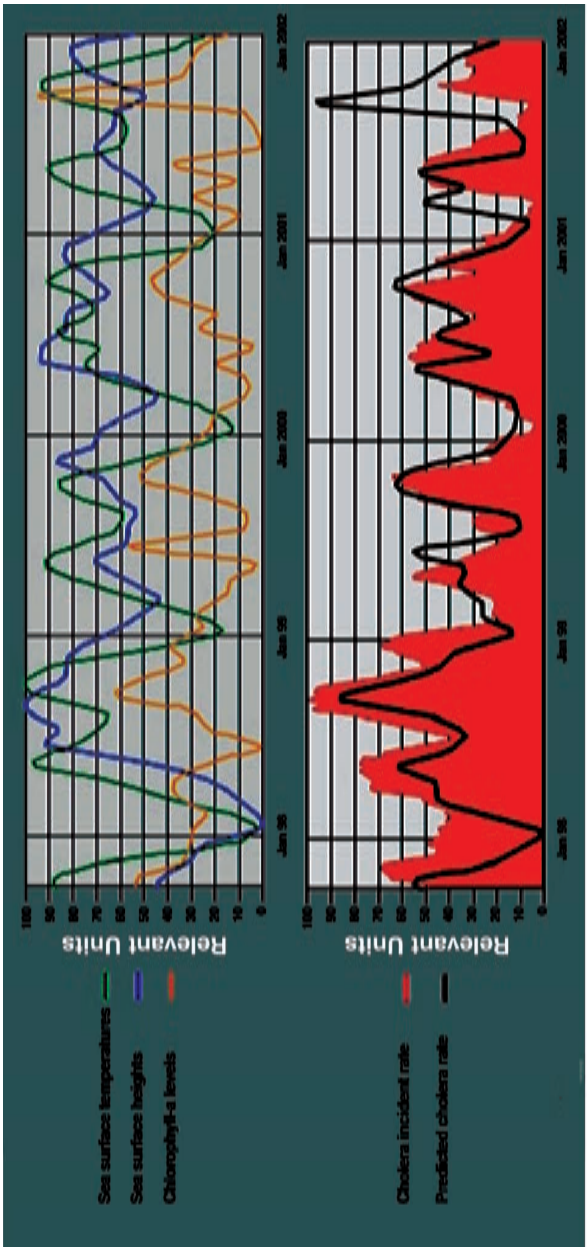

FIGURE 2-2 Environmental parameters (top) and predicted versus actual cholera incidence rate (bottom).
SOURCE: Printed with permission from John Calkins, ESRI User Conference (2004).

develop predictive models of infectious diseases that in turn will allow us to develop a preemptive medicine: that is, to mitigate the impact of infectious disease, if not to prevent it by having an early warning system to initiate appropriate and responsive public health measures.

EXTREME WEATHER AND EPIDEMICS: RIFT VALLEY FEVER AND CHIKUNGUNYA FEVER[2]

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As Earth's climate changes, the frequency and intensity of heat waves, droughts, floods, and other extreme weather events are expected to increase over large regions (IPCC, 2007b). Trends already are apparent, with regions affected by drought and the frequency of heavy precipitation that leads to flooding increasing since the 1950s (IPCC, 2007a). Besides obvious, direct effects on human health, extreme events can facilitate infectious disease epidemics—for example, through effects on disease vector ecology, infrastructure, and human behavior.

Satellite observations and modeling allow prediction of some extreme weather events and consequent infectious disease activity. In this paper, we use

[2]The views expressed in this paper are the private views of the authors and are not to be construed as official or representing the true views of the Department of Defense.

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satellite and epidemiological data to examine connections between the El Niño/Southern Oscillation (ENSO) phenomenon and two recent mosquito-borne epidemics in Africa: Rift Valley fever (RVF) and chikungunya fever, which followed heavy rains and drought, respectively. These case studies suggest considerations in developing early warning systems for extreme weather-associated epidemics.

El Niño/Southern Oscillation and Rift Valley Fever Prediction

The ENSO is an irregular, but natural, feature of the global climate system. It results from interactions between the oceans and the atmosphere across the Indo-Pacific region and affects the weather around the world. In the warm, or El Niño, phase of the cycle, sea surface temperatures are warmer than usual in the eastern-central equatorial Pacific Ocean. El Niño sometimes is followed by a cool, or La Niña, phase with colder-than-usual temperatures in the eastern-central equatorial Pacific. The warm and cool phases cycle over irregular intervals of several years but have characteristic effects on precipitation and temperature throughout much of the tropics.

In areas where it influences climate, El Niño is associated with increased risk of some infectious diseases (Kovats et al., 2003). For example, in East Africa, El Niño is associated with flooding and RVF activity (Linthicum et al., 1999)—epizootics among economically important livestock, with humans infected incidentally by the mosquito vectors or by handling or consuming infected animal products. Outbreaks begin near natural depressions ("dambos") that harbor *Aedes* mosquito eggs infected directly by the parent during development. The eggs hatch with dambo flooding, producing an initial wave of RVF vectors; other species that transmit the virus emerge over subsequent weeks (Linthicum et al., 1984) and propagate the outbreak. The largest recorded RVF outbreak, in 1997-1998, coincided with a strong El Niño. There were an estimated 89,000 human infections and hundreds of deaths in northeastern Kenya and southern Somalia (CDC, 1998).

Following the 1997-1998 outbreak, scientists at the U.S. National Aeronautics and Space Administration Goddard Space Flight Center (NASA-GSFC) and the Department of Defense Global Emerging Infections Surveillance and Response System (DOD-GEIS) initiated a partnership to forecast conditions favorable for RVF activity in Africa by monitoring ENSO and other climatic phenomena. The program uses satellite data from ongoing NASA and NOAA climate and environmental observation programs to provide predictions of areas at elevated RVF risk. The primary data sets are sea surface temperature (SST), rainfall, outgoing longwave radiation (OLR; which is correlated with cloud cover and rainfall), and Normalized Difference Vegetation Index (NDVI; a key measure for identifying risk areas). NDVI is correlated with rainfall but integrates effects of other climatic parameters, responds most to sustained rather than intermittent rains, and is available globally since 1981, while ground-based rain gauge coverage is limited in Africa.

Updated forecasts are available monthly, or more frequently if conditions warrant, on the DOD-GEIS public website.[6] Forecasts and alerts also are communicated to public health agencies that can act on them to enhance surveillance or community preparedness in at-risk areas. Important partners in responding to forecasts and alerts include the World Health Organization (WHO), Food and Agriculture Organization of the United Nations (FAO), the U.S. Centers for Disease Control and Prevention's (CDC's) International Emerging Infections Program in Kenya, and two members of the DOD-GEIS network: the U.S. Army Medical Research Unit-Kenya (USAMRU-K) in Nairobi and the U.S. Naval Medical Research Unit-3 (NAMRU-3) in Cairo.

Rift Valley Fever Outbreaks in East Africa, 2006-2007

In September 2006, the NASA-GSFC/DOD-GEIS monitoring program identified indications of an impending El Niño episode, with SSTs anomalously elevated in the central-eastern Pacific Ocean (+2ºC) and the western Indian Ocean (+1ºC) (see Figure 2-3). These conditions enhanced precipitation over these areas and the Horn of Africa through November (see Figure 2-4). Rainfall increased through December, with vegetation response (see Figure 2-5A) and conditions favorable for RVF activity in large areas of northeastern Kenya and nearby areas in Somalia and Ethiopia, as well as in southern Kenya and northern Tanzania (see Figure 2-5B).

The NASA-GSFC/DOD-GEIS program released a series of epidemic warnings based on these observations. In September 2006, it issued a global, regional-scale forecast covering late 2006-early 2007 for possible El Niño-linked outbreaks, including RVF in East Africa, to the DOD-GEIS network (these forecasts were published online in the *International Journal of Health Geographics,* an open access journal, in December; Anyamba et al., 2006). As rainfall increased in the Horn of Africa, the FAO Emergency Prevention System for Transboundary Animal Diseases issued an RVF alert for the Horn in November, identifying areas flagged as conducive to RVF activity (FAO, 2006). NASA-GSFC/DOD-GEIS also communicated with the WHO, which transmitted alerts to the countries at risk for RVF activity and called for enhanced surveillance and community awareness.

USAMRU-K, in coordination with Kenya Medical Research Institute (KEMRI) and CDC's International Emerging Infections Program (IEIP), deployed a field team in early December to assess high-risk areas in the Garissa district of northeastern Kenya (which was experiencing severe flooding). USAMRU-K tested mosquitoes collected by the team in Garissa and from established collection sites in other areas (see Figure 2-6), identifying RVF virus-infected mosquitoes from Garissa. The field team also investigated local reports of possible

[6]See http://www.geis.fhp.osd.mil.

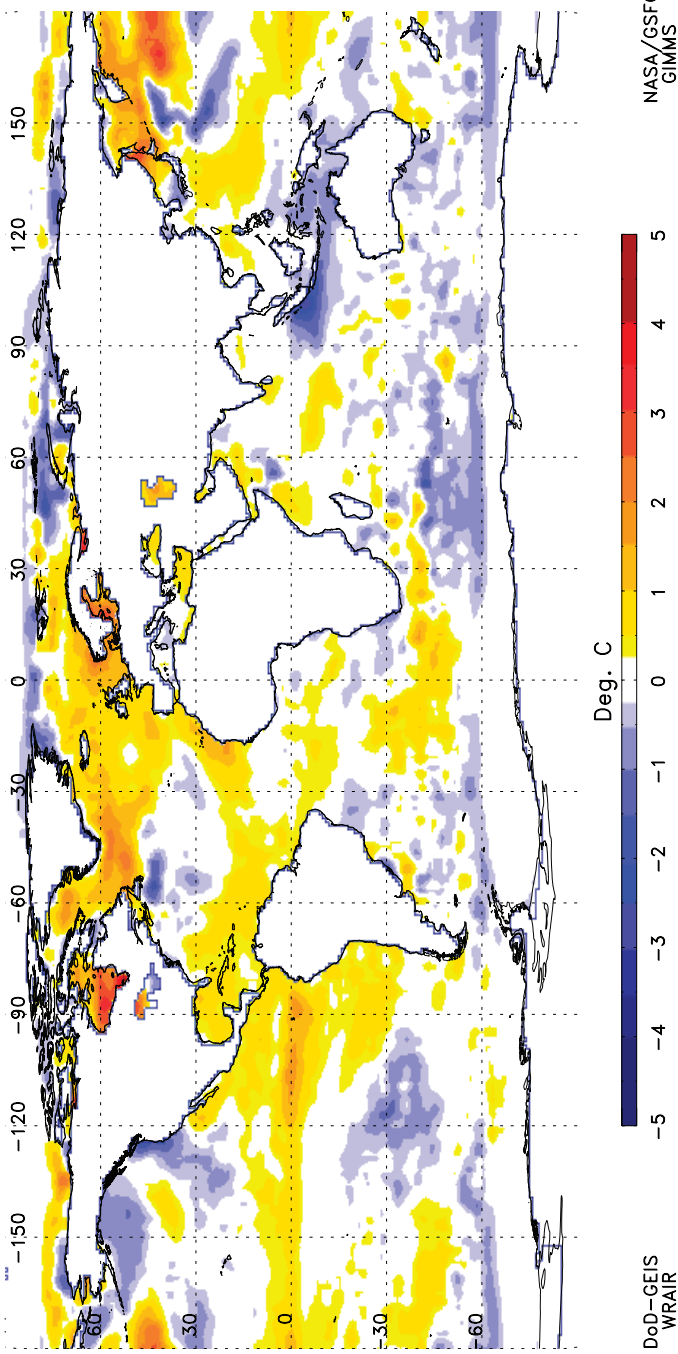

FIGURE 2-3 Global SST anomalies, September 2006.

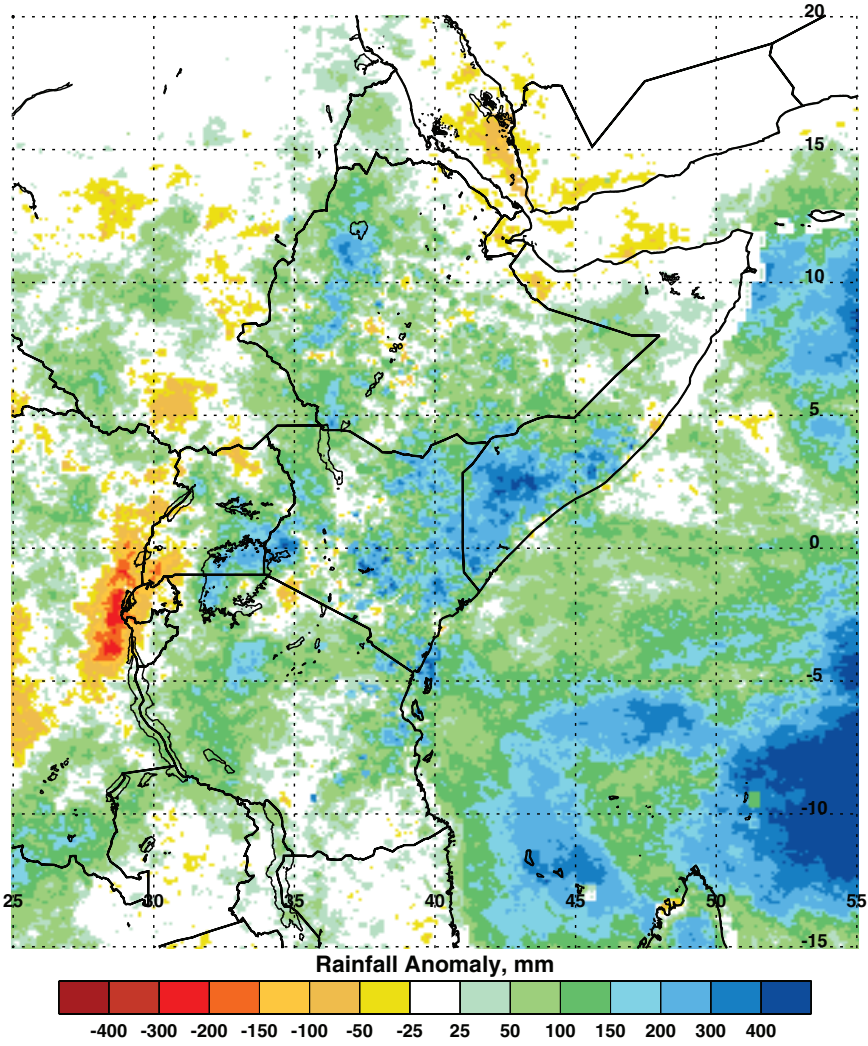

FIGURE 2-4 Seasonal rainfall anomalies in the Horn of Africa, September-November 2006.

animal RVF cases and traveled with Ministry of Health staff to hospitals that recently had admitted patients with suspected RVF, obtaining specimens for testing at KEMRI.

On December 21, KEMRI confirmed RVF virus infection in specimens taken from several patients in the Garissa district (WHO, 2007a). The Kenya Ministry

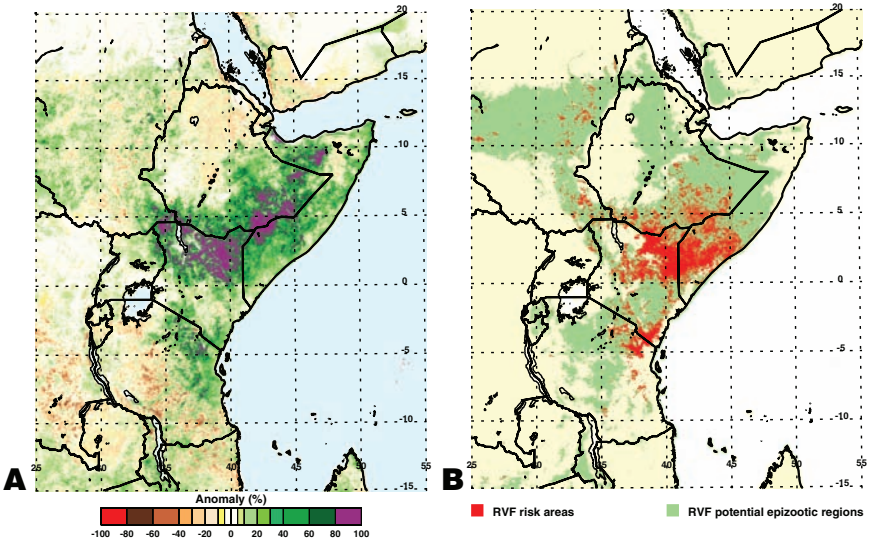

FIGURE 2-5 NDVI anomalies (A) and RVF calculated risk (B) in the Horn of Africa, December 2006. In (B), green identifies areas included in the NDVI-based RVF risk assessment (based on permissive permanent environmental features) and red indicates areas currently at elevated risk, based on persistence of positive NDVI anomalies over at least 3 months.
SOURCE: Figure based on data in Anyamba et al. (2002).

of Health initiated a response with international partners, including WHO, CDC, USAMRU-K, NAMRU-3, and the U.S. Department of Agriculture. An intensive social mobilization campaign began in northeastern Kenya in late December, along with a locally enforced ban on animal slaughtering over most of Eastern and North Eastern Provinces (animal vaccination began in January, but by then the epidemic was waning). NASA-GSFC/DOD-GEIS provided frequent, high-spatial-resolution risk assessment updates to facilitate targeted surveillance during the epidemic response.

Between November 30, 2006, retrospectively identified as the date of onset for the index case, and March 9, 2007, when the last case was identified, 684 cases with 155 deaths were reported in Kenya. North Eastern province, which includes the Garissa district, reported the most cases of affected provinces (N = 333). Smaller RVF epidemics in Somalia and Tanzania followed the Kenya outbreaks: in Somalia, 114 cases with 51 deaths were reported between late December 2006 and February 2007; in Tanzania, 264 cases with 109 deaths were reported between mid-January and early May.

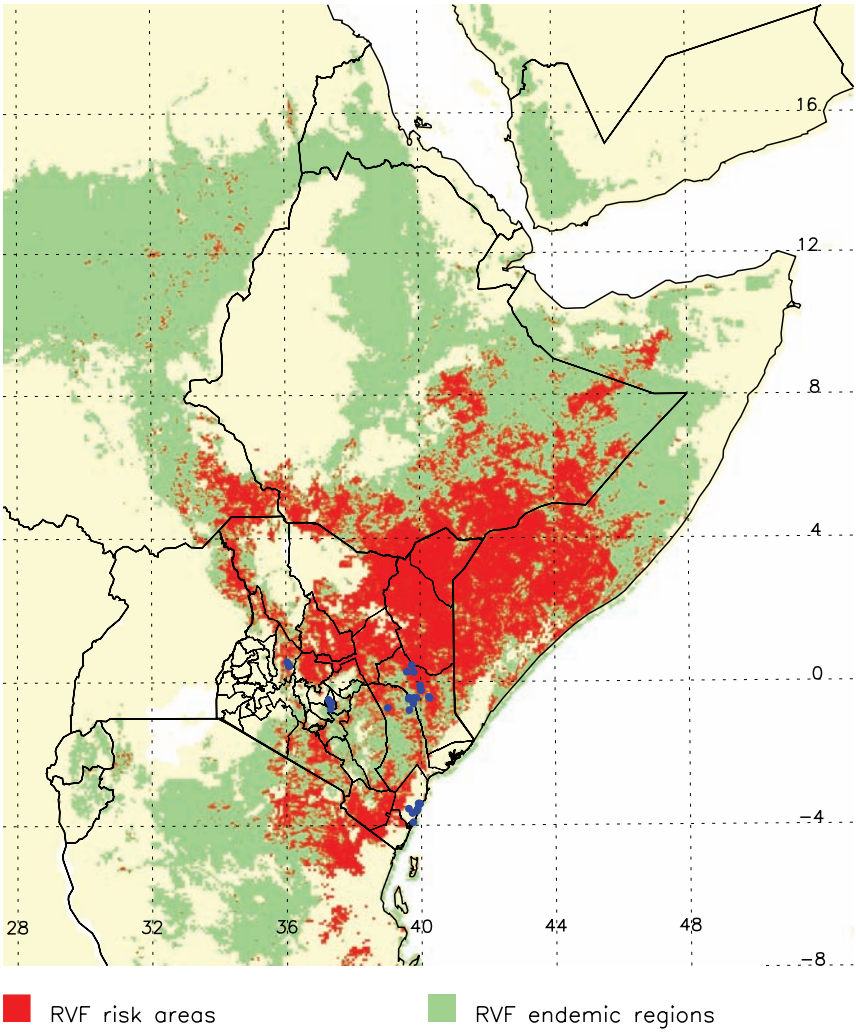

FIGURE 2-6 USAMRU-K mosquito collection sites (blue dots) and RVF risk assessment, December 2006.

Chikungunya Fever Outbreaks in Kenya and Other Regions, 2004-2008

In July 2004, while East Africa experienced a severe drought, a public hospital in Lamu, a coastal island city of Kenya, noted a sharp increase in cases of acute febrile illness. Many patients reported joint pain and had negative malaria blood smears (Bedno et al., 2006). The Ministry of Health launched an outbreak

investigation, which was supported by USAMRU-K and the CDC's IEIP. Laboratory testing of outbreak specimens identified chikungunya virus as the cause. After the outbreak, a population-based serological study led by the Kenya Field Epidemiology Training Program estimated that 13,500 people, or 75 percent of the Lamu population, were infected (Sergon et al., 2008). In November, a chikungunya outbreak was reported in Mombasa, around 200 miles south of Lamu on the Kenya coast.

Though rarely fatal, chikungunya virus infection may cause prolonged and debilitating joint pain. The disease is endemic throughout much of tropical Africa, maintained by transmission cycles involving forest-dwelling *Aedes* mosquitoes and wild primates in which humans are infected incidentally. Urban *Aedes aegypti* and *Aedes albopictus* cause epidemics in tropical Asia without nonhuman hosts. The vectors in urban Lamu and Mombasa were thought to be peridomestic *Aedes aegypti*, which were found in unprotected domestic water sources that were not changed frequently because of water shortages during the drought. The outbreaks marked the first confirmation of chikungunya fever transmission in coastal Kenya.

Retrospective analysis of climate data preceding the Lamu outbreak (assumed to have begun in June 2004) showed anomalously warm, dry conditions over much of East Africa, but especially coastal Kenya, during May 2004 (Chretien et al., 2007). NDVI anomalies in Lamu were the most negative in the available record (1998-2003), reflecting substantially reduced rainfall. When the outbreaks occurred in Lamu and Mombasa, each city had experienced a cumulative rainfall deficit of approximately 100 mm compared to the average (see Figure 2-7).

The warm, dry conditions may have enabled the epidemic in two ways: unsafe domestic water storage practices, along with infrequent changes of water stores because of the drought, may have increased peridomestic *Aedes* vector abundance; and the warm, dry conditions may have enhanced *Aedes* vectorial capacity by decreasing the extrinsic incubation period (Watts et al., 1987).

Following the Kenya chikungunya fever outbreaks, the epidemics spread to other areas with susceptible human populations and competent vectors: to western Indian Ocean islands, including Reunion, where viral mutation may have facilitated adaptation to the highly efficient *Aedes albopictus* vector (Tsetsarkin et al., 2007) and more than 200,000 people likely were infected (WHO, 2006), and to India, which reported well over 1 million cases (Mavalankar et al., 2007).

Also, for the first time ever, chikungunya fever reached Europe. In a northeastern Italian province, public health authorities identified 205 cases during July-September 2007 (Rezza et al., 2007). The presumed index case developed symptoms after visiting relatives in an affected area of India. Local *Aedes albopictus* mosquitoes, an invasive species introduced into Italy around 1990 (tire importation is suspected as the mechanism), then propagated the epidemic. While the role of climatic conditions in the Italian outbreak is unclear, much of southern Europe had experienced an anomalously warm, dry summer (see Figure 2-8)

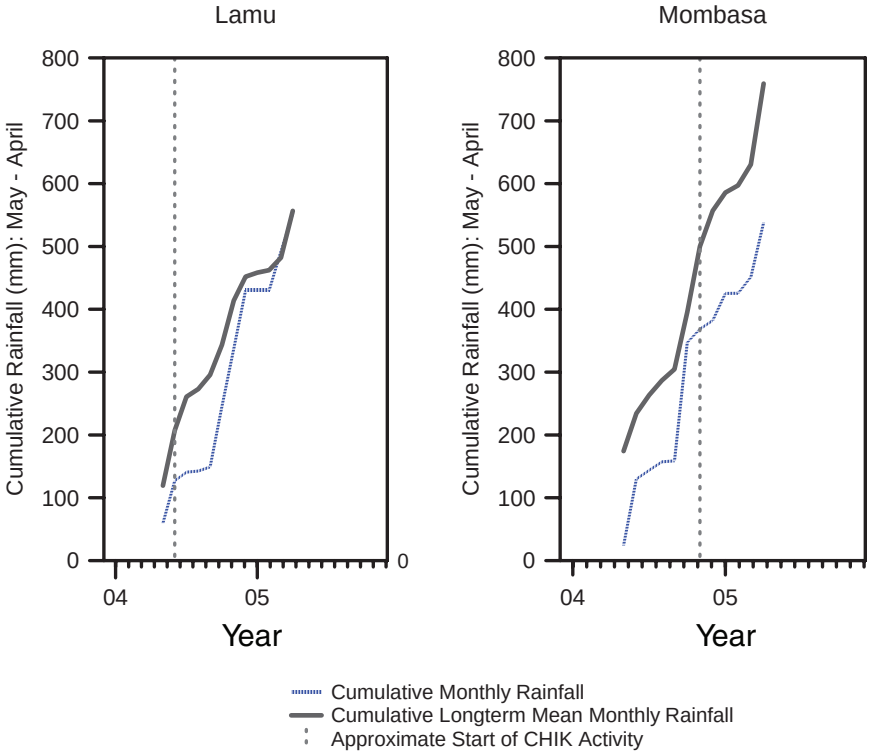

FIGURE 2-7 Cumulative monthly rainfall (dotted line) and long-term mean cumulative monthly rainfall (solid line) in Lamu and Mombasa. Vertical dashed line indicates approximate starting dates for the outbreaks (Lamu, June 2004; Mombasa, November 2004).

that, along with historically poor vector control, may have contributed to the abundance of mosquitoes in the affected area at the time of the outbreak (reported anecdotally; Rezza et al., 2007).

Developing Early Warning Systems for Extreme Weather-Linked Infections

In both the RVF and the chikungunya fever examples, climate appears to have interacted with other factors to facilitate the outbreaks (see Table 2-2), consistent with the "Convergence Model" of infectious disease emergence proposed by the Institute of Medicine's (IOM's) Committee on Microbial Threats to Health in the Twenty-First Century (Figure 2-9; IOM, 2003). For example, besides flooding of mosquito habitats, animal sacrificing and preparation practices may have

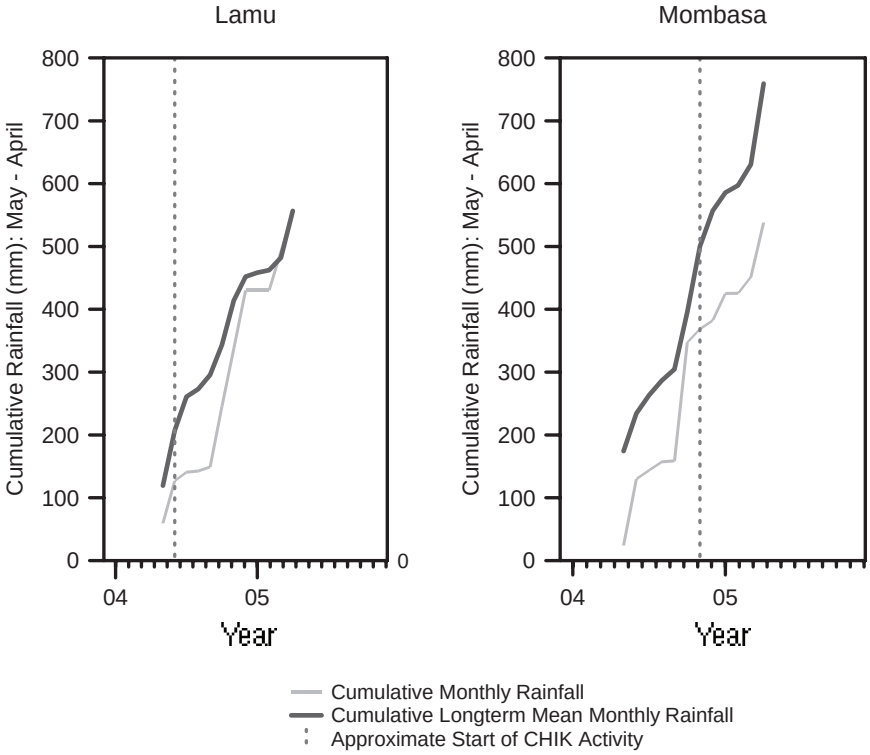

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TABLE 2-2 Factors in Emergence and Spread of Rift Valley Fever and Chikungunya Fever

	Rift Valley Fever	Chikungunya Fever
Climatic factors	• Flooding	• Warm, dry conditions
Biological factors	• Broad host and vector species range • Abundant livestock hosts	• Genetic adaptation to *Aedes albopictus* • Association of *Aedes albopictus* and *Aedes aegypti* to humans
Physical environment factors	• Dambos, other ground pools	• Vector breeding sites
Social, political, and economic factors	• Livestock trade • Herder and religious practices	• Travel • Delayed notification and control • Previous introduction of *Aedes albopictus* to Indian Ocean islands and Italy by trade

SOURCE: Adapted from Chretien and Linthicum (2007), IOM (2003), and Peters and Linthicum (1994).

contributed to the RVF epidemic in East Africa in 2006. In coastal Kenya in 2004, the availability of vector breeding sites (i.e., unprotected domestic water stores) appears to have facilitated the emergence of chikungunya fever. In developing early warning systems for outbreaks linked to extreme weather, consideration of the nonclimatic facilitating factors may enable more precise identification of populations at risk, with better targeting of risk communication.

The RVF and chikungunya fever outbreaks also suggest the need for infectious disease early warning systems to integrate with other natural disaster prediction and response programs. In both of these epidemics, climatic conditions facilitating disease emergence and transmission had other public health effects as well. Flooding in the Horn of Africa during late 2006-early 2007 affected more than 1 million people (WHO, 2007b), destroying homes, livestock, and crops; displacing families; causing hygiene breakdown and water-borne disease epidemics; and obstructing delivery of aid (Save the Children, 2007). Drought in Kenya during 2004 contributed to massive crop failure and food shortages. Coastal areas (where the chikungunya fever epidemics occurred) were particularly affected, since rainfall was well below normal during 2003 and the areas lacked community-based mechanisms for emergency intervention because they had not recently experienced severe drought (UN, 2004).

There are few operational early warning systems for climate-linked epidemics (WHO, 2004). But there is potential for developing such systems—WHO

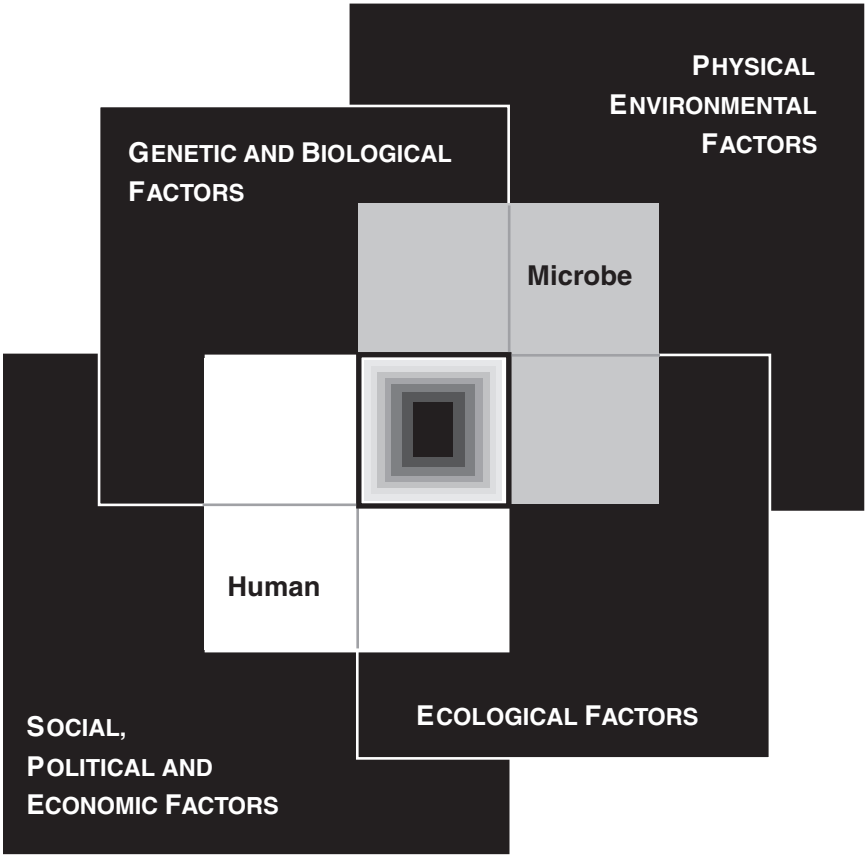

FIGURE 2-9 The Convergence Model.
SOURCE: IOM (2003).

has assessed climate-infectious disease links and recommended development of climate-based predictive models for cholera, malaria, and several other infectious diseases (WHO, 2004); and many countries maintain or are developing early warning systems for natural hazards. Citing the Indian Ocean tsunami of December 26, 2004, as a "wake-up call" about the role that early warning systems could play in reducing the human and physical impacts of natural hazards, United Nations (UN) Secretary General Kofi Annan called for the development of a global early warning system for all natural hazards (UN, 2006). The UN Platform for the Promotion of Early Warning,[7] initiated in 2004, is leading early warning

[7]See http://www.unisdr.org/ppew.

actors toward this goal. Integration of epidemic prediction with such related efforts could speed the development of epidemic prediction systems and facilitate more comprehensive risk communication to communities facing extreme weather events and other natural hazards.

PLAGUE AND CLIMATE

Nils Chr. Stenseth, Dr.philos.[8]
University of Oslo

Plague, caused by the bacterium *Yersinia pestis*, is found on all continents except Antarctica and Australia (Figure 2-10). The plague bacillus causes a rapidly progressing, serious illness that, in its bubonic form, is likely to lead to death by septicemia (40 to 70 percent mortality). Without prompt antibiotic treatment, pneumonic and bubonic plagues are nearly always fatal. For these reasons the plague bacterium *Y. pestis* is considered one of the most pathogenic bacteria for humans (Gage and Kosoy, 2005). Throughout history, it has played a dramatic role, and it continues to be a threat worldwide (Figure 2-10), particularly in Africa (Figure 2-11).

Plague is currently recognized as a reemerging disease increasing in frequency throughout the world (Duplantier et al., 2005; Schrag and Wiener, 1995; Stenseth et al., 2008; WHO, 2003, 2005) as well as being a potential agent of bioterrorism (Inglesby et al., 2000; Koirala, 2006). Throughout its geographic distribution, its main reservoir is composed of a variety of wild (and in some cases commensal) rodents and the bacterium is transmitted between individual hosts primarily by flea vectors (see "The (Full) Plague Eco-Epidemiological System" below). Understanding what determines the dynamics of plague necessitates an understanding of the dynamic rodent-flea-bacterium system in the wild.

The dynamics of the reservoir species are known to be profoundly influenced by climate variation (see Stenseth, 1999; Stenseth et al., 2002, 2006). Here, I summarize our findings from the analysis of long-term data monitoring in Kazakhstan. I both address what might happen should the climate change as expected (IPCC, 2007) and assess whether there has been a climate component underpinning the past plague pandemics.

The Three Big Historical Plague Pandemics

Plague has given rise to at least three major pandemics. The first ("the Justinian plague") spread around the Mediterranean Sea in the sixth century A.D., the second ("the Black Death") started in Europe in the fourteenth century and

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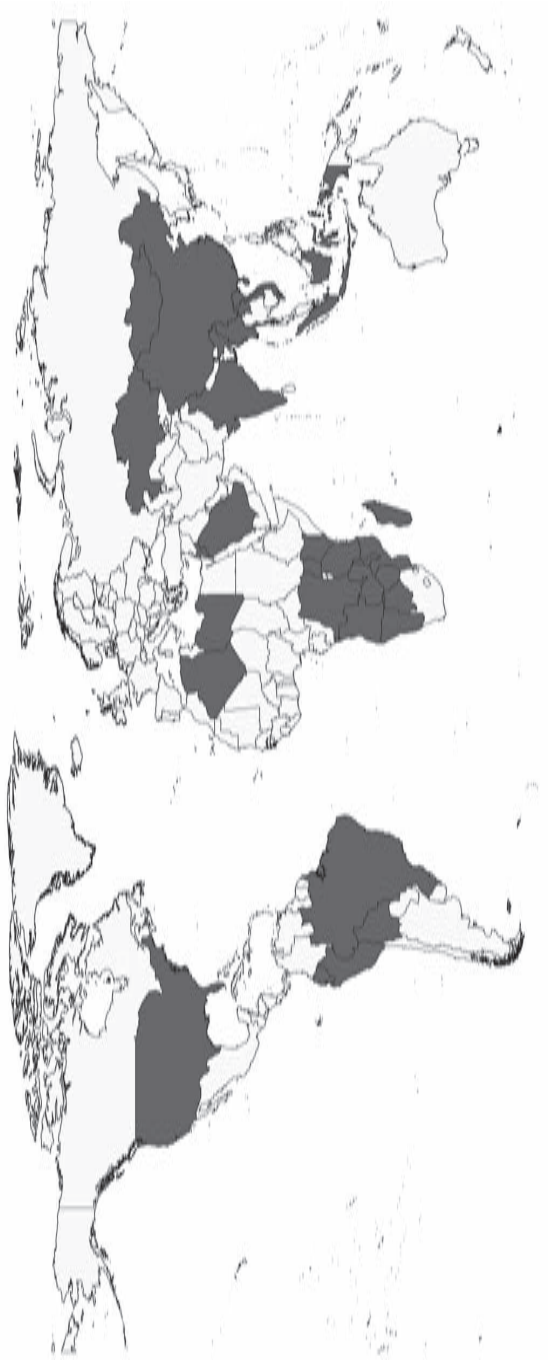

FIGURE 2-10 The global distribution of plague. The map shows countries with a known presence of plague in wild reservoir species (black) (after WHO, 2005). For the United States, only the mainland below 50°N is shown.
SOURCE: Stenseth et al. (2008).

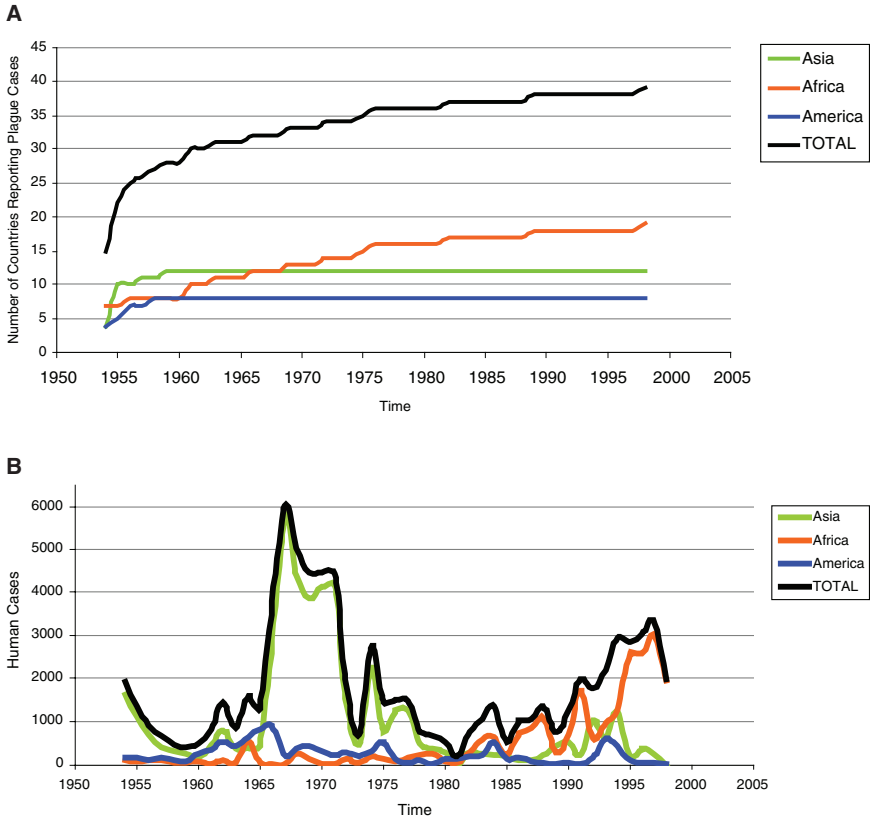

FIGURE 2-11 The global distribution of plague: (A) cumulative number of countries that reported plague to WHO per continent, from 1954-1998; (B) the temporal distribution of plague cases by continent, from 1954-1998, also from WHO. (Panel B is corrected relative to a similar one given in Stenseth et al. (2008): for 1997 and 1998 the numbers have, in dialogue with WHO, been corrected for Madagascar.)
SOURCE: Modified from Stenseth et al. (2008).

recurred intermittently for more than 300 years, and the third started in China during the middle of the nineteenth century and spread throughout the world. Purportedly, each pandemic was caused by a different biovar of *Yersinia pestis*, respectively Antiqua (still found in Africa and central Asia), Medievalis (currently limited to central Asia), and Orientalis (nearly worldwide; Guiyoule et al., 1994; Twigg, 1984; see Figure 2-12).

The Black Death decimated medieval Europe, and as a result, had a major impact on the continent's socioeconomic development, culture, art, religion, and

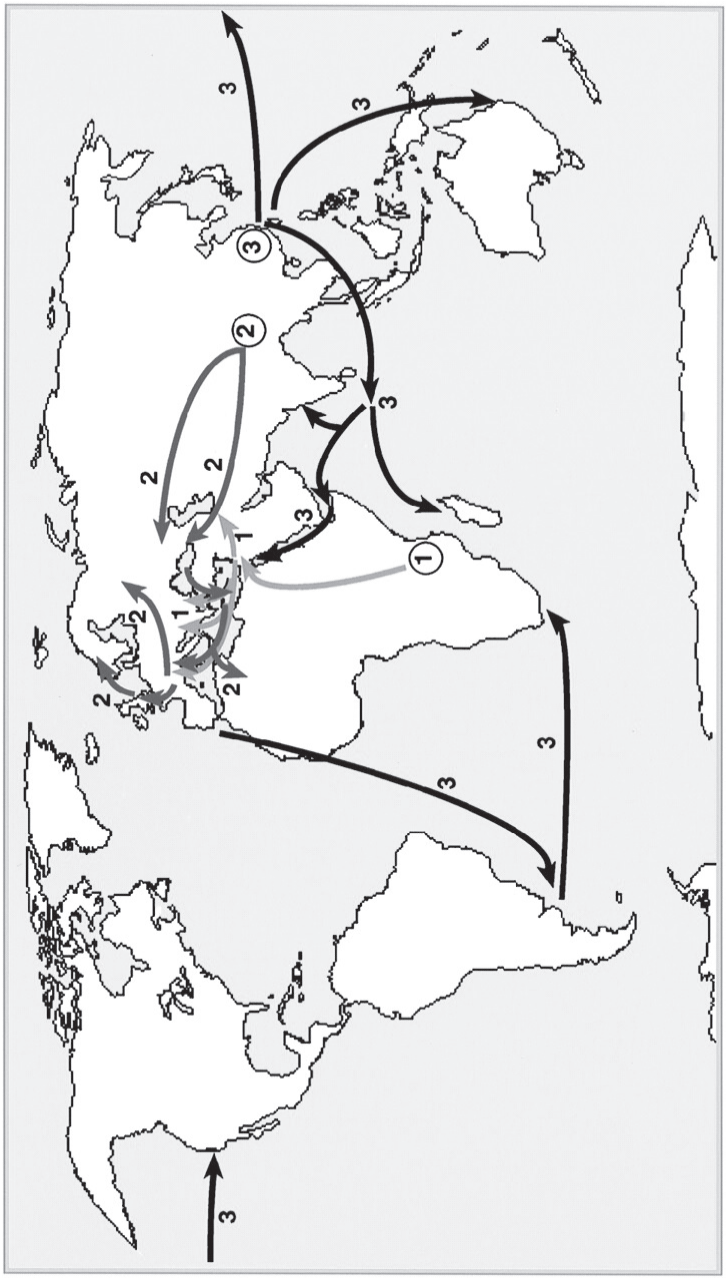

FIGURE 2-12 Routes followed by the three plague pandemic waves (labeled 1, 2, and 3). Circled numbers indicated the regions thought to be the origin of each of the three pandemics: the Justinian plague (541 A.D. to 767 A.D.); the Black Death and subsequent epidemics from 1346 to the early nineteenth century; and the Third Pandemic, in the mid-nineteenth century in the Yunnan region of China, started in 1855. SOURCE: Achtman et al. (1999).

politics (Twigg, 1984; Ziegler, 1969). Although some have questioned whether the Black Death (as well as the first pandemics) was caused by *Y. pestis* (Cohn, 2002; Scott and Duncan, 2001), it seems settled today (Stenseth et al., 2008). It is generally accepted that the epidemiology of the Black Death plague, as reflected in historical records, does not always match the "classical" rat-flea-human plague cycle, but the reported medical symptoms were very similar during each historical pandemic. It should be appreciated, however, that "classical" plague epidemiology is only one of several possibilities to explain the Black Death and may not even be the most typical of the different plague ecology systems (Drancourt et al., 2006). The discovery of *Y. pestis* genetic material in those who died from the Black Death and are buried in medieval graves (Raoult and Aboudharam, 2000) further supports the view that *Y. pestis* was the causative agent of the Black Death.

The (Full) Plague Eco-Epidemiological System

Soon after Yersin's discovery of the plague bacillus (Yersin, 1894), it became clear that urban outbreaks were linked to transmission among commensal rats and their fleas. In this classic urban plague scenario, infected rats (transported, for example, by ships) arrive in a new city and transmit the infection to local house rats and their fleas, which then serve as sources of human infection. Occasionally, humans develop a pneumonic form of plague, which is then directly transmitted from human to human through respiratory droplets.

The epidemiology of plague, however, is much more complicated than this urban plague scenario suggests, involving several other pathways of transmission. This complicated epidemiology necessitates reconsidering plague ecology within its full ecological web (Figure 2-13).

Maintenance of plague foci depends on a whole suite of rodent hosts and their associated fleas. Under favorable conditions, the plague bacillus might survive in the environment, essentially in rodent burrows (Baltazard et al., 1963). When an infected flea happens to feed on a commensal rodent, the cycle continues in the latter. As commensal rodents die, their fleas are forced to move to alternate hosts (e.g., humans). If humans develop pneumonic plague, the infection may transmit from person to person through exposures to respiratory droplets spread by coughing. Humans may also become infected through handling infected animals (or meat), including rodents, camels, or cats. Cats may also develop pneumonic plague, passing their infection to their owners through coughing. Finally, there is evidence that the human flea, *Pulex irritans*, can be involved in human-to-human transmission (Blanc, 1956; Laudisoit et al., 2007). Mammalian predators, birds of prey, and other birds that use rodent burrows for nesting may move over larger areas than the rodents themselves, spreading the infection over longer distances. Infected commensal rats or humans may also travel over long distances.

Because of its widespread occurrence in wildlife rodent reservoir species one must recognize that plague cannot be eradicated. There is a critical need,

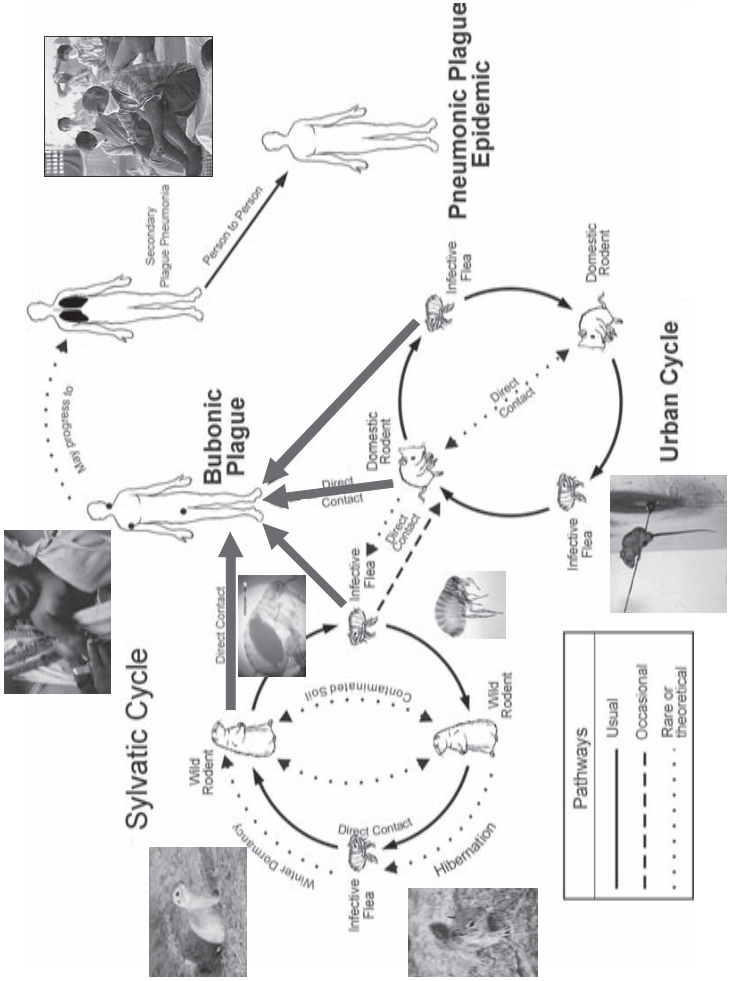

FIGURE 2-13 Possible transmission pathways for the plague bacterium, *Yersinia pestis*. Thick arrows indicate pathways to people. SOURCE: Adapted from Chamberlain (2004) and printed with permission from Neal R. Chamberlain, Ph.D., A.T. Still University of Health Sciences.

therefore, to understand how human risks are affected by the dynamics of these reservoirs and how people interact with them.

The capacity of the plague bacillus to form permanent foci under highly diverse ecological conditions attests to its extraordinary adaptability. During its emergence in central Asia, *Y. pestis* accumulated copies of insertion sequences rendering its genome highly plastic (Parkhill et al., 2001). The capacity to undergo genomic rearrangements may thus be an efficient means for the plague bacillus to adapt to new ecological niches. *Y. pestis* was recently shown to be able to acquire antibiotic resistance plasmids under natural conditions (Galimand et al., 1997; Guiyoule et al., 2001), probably during its transit in the flea midgut (Hinnebusch et al., 2002). Obviously, the emergence and spread of multidrug-resistant strains of *Y. pestis* would represent a major threat to human health.

Although the number of human cases of plague is relatively low, it would be a mistake to overlook its threat to humanity because of the disease's inherent communicability, rapid spread, rapid clinical course, and high mortality if left untreated. A plague outbreak may also cause widespread panic, as occurred in 1994, when a relatively small outbreak, with 50 deaths, was reported in the city of Surat, India (Mudur, 1995), which led to a nationwide collapse in tourism and trade, with an estimated cost of $600 million U.S. dollars (Fritz et al., 1996).

Studying the Plague Dynamics of Central Asia: The Effect of Climate Variation

Together with colleagues, I have been studying the dynamics of the plague ecological system based on long-term monitoring data from the former Soviet Union (specifically from Kazakhstan), some of which have been published (Davis et al., 2004, 2007; Frigessi et al., 2005; Kausrud et al., 2007; Park et al., 2007; Samia et al., 2007; Stenseth et al., 2006) but much more is to come, including information on human plague cases. Currently, we are expanding our geographic area of interest to include China, India, Madagascar, and the United States.

Our core set of monitoring data comes from southeastern Kazakhstan (74-78°E and 44-47°N; see Figure 2-14). Each spring and autumn, between 1949 and 1995, a proportion of inhabited burrows and site-count observations were done at different locations within the PreBalkhash area (see Figure 2-14; for details, see Stenseth et al., 2006).

For monitoring purposes, the area was divided into 10×10 km^2 sectors. Four sectors constitute a 20×20 km^2 *primary square* (PSQ), and four PSQs constitute a *large square* (LSQ; Figure 2-14). At a given site, the great gerbil population densities were estimated at most twice per year. On approximately 85 percent of these occasions, up to 8,576 gerbils (median = 604) were trapped per LSQ, based on independent plague prevalence data (see Stenseth et al., 2006) and season, and tested for *Y. pestis* infection. The LSQs chosen had the longest regular and continuous time-series data required by our analysis. We also have access to

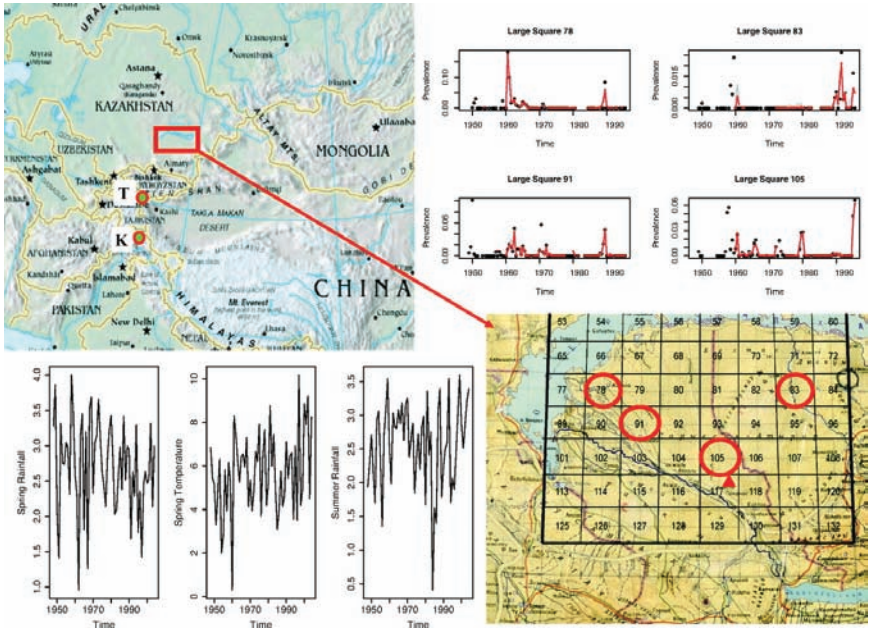

FIGURE 2-14 The field data used in Stenseth et al. (2006) were collected in a natural plague focus in Kazakhstan. The data are plague prevalence in great gerbils, counts of fleas collected from trapped gerbils, and meteorological observations. *Left Upper*: Kazakhstan on a map of Central Asia with the PreBalkhash focus (between 74 and 78°E and 44 and 47°N) marked as a square. The historic climate (tree-ring) measurement sites are circles marked K (Karakorum) and T (Tien Shan). These sites are located approximately 1,000 and 600 km from the research area, respectively. *Lower Right*: The LSQ in the PreBalkhash focus from which we have prevalence. The four LSQs (40 × 40 km) circled in red, namely LSQs 78, 83, 91, and 105, represent key sites where collection of samples for testing the presence of plague was more regular and continuous. The Bakanas meteorological station is located in LSQ 117, marked by a red triangle. *Upper Right*: The time-series plots of the observed prevalence per LSQ. Open and filled circles denote the observed prevalence during the spring and fall, respectively. The time series of the prevalence fitted by using the model defined by the model is shown in red. Using the same model but without any climatic covariates gives the time series shown in gray. Note that owing to the presence of missing values in some covariates (occupancy) and prevalence data, the curves of the fitted values are discontinuous. The fitted values from the model provide a closer fit and reproduce the peaks in prevalence far better than the model without the climatic variables. *Lower Left*: Time-series plots of the climate variables, spring rainfall, spring temperature, and summer rainfall (from left to right).
SOURCE: Stenseth et al. (2006).

plague prevalence data: gerbils caught were tested for plague through isolation of *Y. pestis* from blood, spleen, or liver smears.

Spring climatic variables used were the average monthly temperature during the spring (i.e., March and April) and the log average of the spring rainfall. The fall climatic variable used is the log average of summer rainfall over June, July, and August. Incorporating the climatic effects in the model resulted in fitted values that track the peak occurrences in prevalence more closely than the model without the climatic variables.

Climate variability over the past millennium was estimated by using a large data set of 203 *Juniperus turkestanica* tree-ring width series to reconstruct temperature variations in the Tien Shan Mountains (Kirghizia) (Esper et al., 2003) and a total of 40 stable oxygen isotope ($\delta^{18}O$) series to reconstruct precipitation variations in the Karakorum Mountains (Pakistan) (Treydte et al., 2006). Climatic variations at these sites are found to be correlated with those in the study area.

We also used the NDVI (Hall et al., 2005; Los et al., 2000; see also Pettorelli et al., 2005), which is based on the difference between near-infrared and visible light reflected from the ground, thereby giving an index of light absorbed by chlorophyll on the ground, an index we also extended through proxy data back in time (see Kausrud et al., 2007).

The following discussion summarizes our findings to date. Davis et al. (2004) demonstrated that plague within an area invades, fades out, and reinvades in response to fluctuations in the abundance of its main reservoir host, the great gerbil. Broadly speaking, they found that infection spreads and persists when total abundance is above a single threshold value and fades out when it is below (see Figure 2-15).

Stenseth et al. (2006) reported that a 1°C increase in spring temperatures is predicted to lead to a >50 percent increase in prevalence (see also Samia et al., 2007). Changes in spring temperature were found to be the most important environmental variable determining the prevalence level, leading to the following scenario: Warmer spring conditions result in an elevated vector-host ratio, which leads to a higher prevalence level in the gerbil host population. Moreover, the climatic conditions that support increased prevalence among gerbils, given unchanged gerbil abundance, also favor increased gerbil abundance (see Kausrud et al., 2007), implying that the threshold density (as found by Davis et al., 2004) condition for plague will be reached more often, thereby increasing the frequency with which plague can occur.

Kausrud et al. (2007), focusing on rodent-host dynamics, drew the following five main conclusions from their analyses:

1. Density fluctuations of the great gerbil, the main host, are highly correlated over large areas, suggesting that climate may be a synchronizing agent. This is probably an important factor causing large-scale plague epizootics in the region.

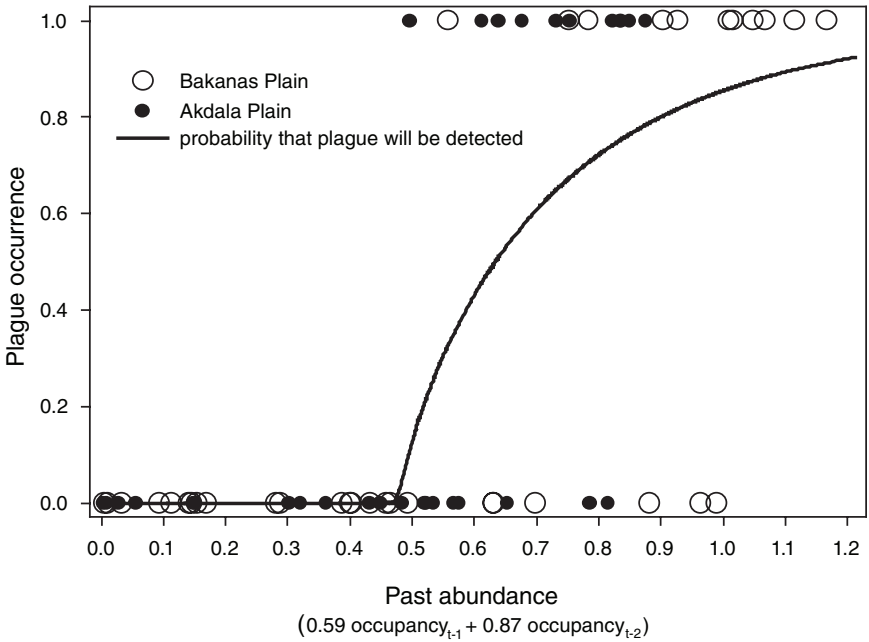

FIGURE 2-15 Relationship between the likelihood of detecting plague (solid line) in gerbils and past burrow occupancy rates together with data on presence or absence of plague at two sites: Bakanas plain (open circles) and Akdala plain (filled circles); see Davis et al. (2004) for details. The likelihood of detecting *Y. pestis* is 0 below a threshold value of 0.476 (95 percent confidence interval: 0.355, 0.572) but rises rapidly once the threshold is attained and continues to increase for even higher values. The seasonal data on abundance and presence of infection are pooled such that presence in a particular year means the disease was detected in either spring or autumn (or both seasons) of that year. Occupancy data represent averages of spring and autumn estimates.
SOURCE: Reprinted from Davis et al. (2004) with permission from AAAS.

2. Great gerbil population densities at large spatial scales can be well predicted 6 to 12 months in advance when combining spatial environmental effects and intrinsic dynamics. This insight is certainly important for predicting plague dynamics.

3. While great gerbil population growth rates exhibit greater variability in areas with low April NDVI index, average population density is not strongly correlated to average vegetation productivity. This suggests that the gerbils will be capable of maintaining population densities where plague can persist over most of their range even if, as predicted, the climate in central Asia becomes increasingly arid.

4. While the presence of plague infection in an area is associated with population decrease over the following months, plague seems unlikely to be the main driving force behind great gerbil density fluctuations.

5. The magnitude of plague epizootics associated with the great gerbil may be expected to increase under predicted effects of ongoing climate change, a confirmation of the conclusion previously drawn by Stenseth et al. (2006).

Altogether, the model reported by Stenseth et al. (2006) suggests that warmer springs (and wetter summers) can trigger a cascading effect on the occurrence and level of plague prevalence, in years with above-threshold great gerbil abundance during the fall 2 calendar-years earlier and in a region that is itself dry and continental (hot summers, cold winters; see IPCC, 2007). Our analyses, moreover, favor the suggestion that enhanced flea survival and reproduction are critical to this effect. Given the multiple routes of plague transmission (flea-borne, direct via several pathways, transfer from other reservoirs), climatic influences on other epidemiological processes cannot be precluded. More generally, it is widely accepted that the distribution and dynamics of vector-borne infections are particularly sensitive to climatic conditions, by virtue of the sensitivity of the (arthropod) vectors themselves to variations in temperature, humidity, and often, quantities of standing water used as breeding sites.

The model reported by Stenseth et al. (2006) may also shed light on the emergence of the Black Death and the plague's Third Pandemic, thought to have spread from an outbreak "core region" in central Asia. Analyses of tree-ring proxy climate data demonstrated that conditions during the period of the Black Death (1280-1350) were both warmer and increasingly wet. The same was true during the origin of the Third Pandemic (1855-1870), when the climate was wetter and underwent an increasingly warm trend. Our analyses are thus in agreement with the hypothesis that the medieval Black Death and the mid-nineteenth-century plague pandemic may have been triggered by favorable climatic conditions in central Asia. Figure 2-16 summarizes the link between climate and the two last plague pandemics.

Such climatic conditions have recently become more common (IPCC, 2007), and whereas regional scenarios suggest a decrease in annual precipitation with increasing variance, mean spring temperatures are predicted to continue increasing (Huntington, 2006). Indeed, during the period from the 1940s, plague prevalence has been high in its host-reservoir in Kazakhstan (see Stenseth et al., 2006). Effective surveillance and control during the Soviet period resulted in few human cases. However, recent changes in the public health systems, coupled with a period of political transition in central Asia and an increased prevalence of plague in its natural reservoir in the region, shadow a future of increased risk of human infections.

In a yet-to-be-published study, Kausrud et al. (2008), using the same surveillance data from 1950 to 1995, together with regional climate indices, have

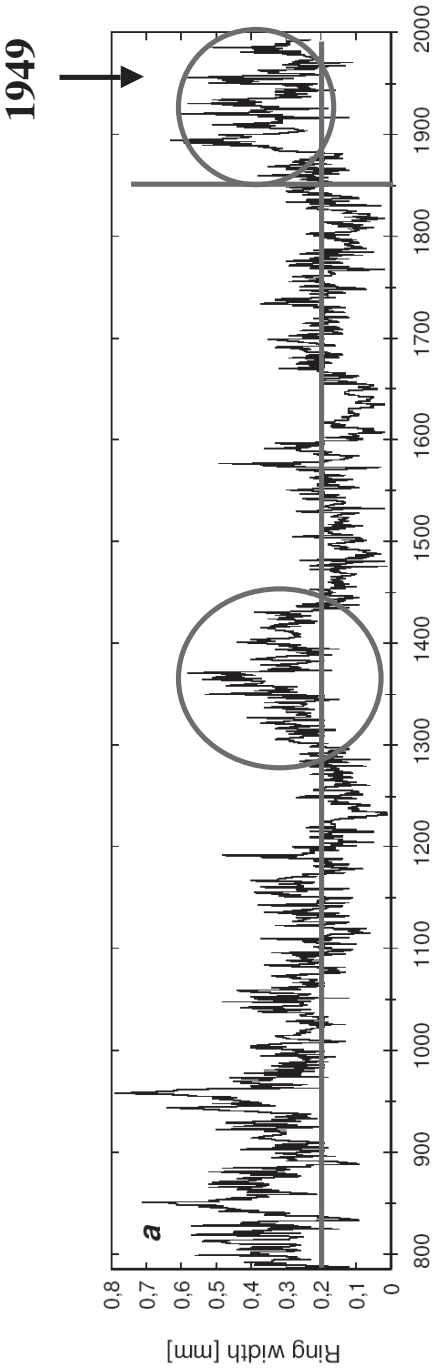

FIGURE 2-16 Tree-ring data suggesting that conditions during the Black Death and the Third Pandemic were similar. The two circles highlight the start of the Black Death and the Third Pandemic; the horizontal line is inserted for the purpose of baseline reference; the vertical gray line indicates the very start of the Third Pandemic (1855); 1949 is the year for which the monitoring and intervention program started in Kazakhstan.
SOURCE: Based on data in Stenseth et al. (2006).

found that climate influences plague dynamics through the rodent-host and flea-vector relationship. Simulating backwards, Kausrud et al. (2008) successfully predicted human plague patterns in Kazakhstan from 1904 to 1950. Using tree-ring data extending back in time to 1000 A.D., this model allows us to compare model predictions with historical plague epidemiology. Analysis suggests an eco-epidemiological basis for considering the Black Death epidemic as having originated in central Asia during climatically favorable conditions (for the plague system). The same model, used for prediction forward, suggests that expected climate change will sustain and possibly increase plague activity in central Asia.

Effect of Climate on Plague Dynamics in Other Regions of the World

Based upon our work on the Kazakh data, we are now extending our interest to other parts of the world. Together with Zhang et al. (2007), I have been involved in some preliminary analysis of data on human plague cases from China. These show a clear effect of large-scale climate influence. Unpublished work that I have done in cooperation with a student of mine (Ben Ari et al., 2008) similarly shows that the number of human plague cases in the western United States is strongly influenced by the Pacific Decadal Oscillation (PDO) and the number of days with above-normal temperatures. In short, a warmer and wetter climate is associated with increased prevalence level of the plague bacterium in the rodent reservoir, which subsequently might lead to an increased number of human cases. These results match up nicely with the previously published cascade model by Parmenter et al. (1999) emphasizing that the climate connection works partly through the rodent-host dynamics and the flea-vector dynamics (see Figure 2-17) in the same region.

Additional Reasons for Being Concerned: Bioterror

As indicated in the introduction, we should not overlook the fact that plague has been weaponized throughout history—from catapulting diseased corpses over city walls, to dropping infected fleas from airplanes, to refined modern aerosol formulations (Inglesby et al., 2000; Koirala, 2006). The weaponization research carried out on plague from the 1930s through the 1990s fueled biological warfare fears that may actually have stimulated research on infectious disease surveillance and response strategies. More recently, however, the fears of small-scale bioterrorism and a desire by government authorities to more fully control all access to plague materials increase the danger of stifling basic research on plague ecology, epidemiology, and pathophysiology that is required to improve its clinical management in endemic areas. Terrorist use of an aerosol released in a confined space could result in significant mortality and widespread panic (Inglesby et al., 2000; Koirala, 2006), and no one would want the knowledge and materials for weaponizing plague to fall into the hands of non-state actors. However, the

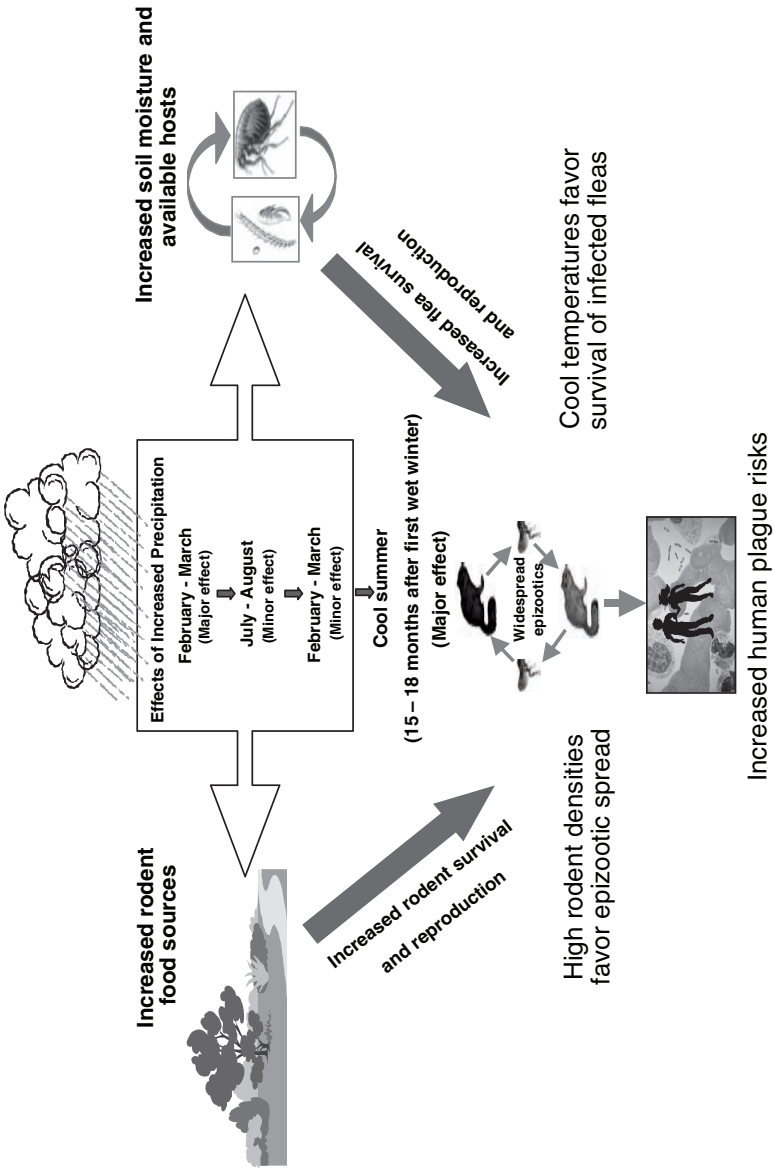

FIGURE 2-17 The modified trophic cascade model of Parmenter et al. (1999).
SOURCE: Adapted from Parmenter et al. (1999) with permission from the *American Journal of Tropical Medicine and Hygiene*.

need for scientifically sound studies of the dynamics of infection, transmission, outbreak management, and improved surveillance and monitoring systems has never been greater.

Conclusion: It Is Unwise to Neglect Plague

In conclusion, it should be noted that although plague may not match the so-called big three diseases (malaria, HIV/AIDS, and tuberculosis; see Hotez et al., 2006) in numbers of human cases, it by far exceeds these diseases in pathogenicity and rapid spread under the right environmental conditions. Plague should be seen not only as a historical curiosity but as a reemerging disease of the twenty-first century. Plague should not continue to be neglected and relegated to the sidelines; it is a disease which should concern us today.

Plague remains a fairly poorly understood threat that we cannot afford to ignore. Nevertheless, much progress has recently been made toward understanding the dynamics of the full plague eco-epidemiological system, and not the least how it responds to climate variation and change. We know that climate does affect the dynamics (and indeed the level) of plague. However, it is difficult at present to say what that effect will be. For example, in central Asia there might be higher levels of plague in the rodent reservoir populations, if current climate prognoses for the region materialize. Also, higher levels in the wildlife reservoir will automatically lead to a greater chance of people being infected by the plague bacillus. In other places of the world there might be lower plague levels in the reservoirs—we simply do not know, but we ought to know if we are to be maximally prepared for what happens should climate change. It is certain, though, that the picture regarding plague might be much more serious than conveyed by Anyamba et al. (2006). Only by knowing more about how the eco-epidemiological plague systems in different parts of the world will respond to given climate scenarios can we take the necessary precautionary measures to reduce the risks associated with human infections. Indeed, knowing how climate is affecting the components of the eco-epidemiological system depicted in Figure 2-13, and subsequently how these climate drivers might change the dynamics of the system, will put us in a greatly improved position for predicting where and under what environmental conditions the risk of human plague infections might increase and where and under what conditions it might decrease (or remain unchanged). Much of the insight derived from studying particular plague systems will be general and applicable to other plague systems—and indeed to other vector-borne infectious disease systems. However, since the involved host and vector species are different from one part of the world to another (indeed, the plague eco-epidemiological system is characterized by a whole suite of rodent host species and their associated fleas, differing from one place to another), studies similar to those that I have summarized for central Asia are greatly needed. Such additional studies may help us to understand which insights derived from the central-Asian studies may or may not be generalized to other places where

plague occurs. Such additional studies will further help us develop more region-specific prediction regarding what might happen should climate change in some specific way.

Acknowledgments

I thank Tamara Ben Ari for having read and commented on an earlier version of this paper; furthermore, I thank her and Kyrre Linné Kausrud for allowing me to summarize yet unpublished work. Over the years working on plague dynamics, I have benefited enormously from collaboration with several colleagues, most importantly Herwig Leirs, Hildegunn Viljugrein, Mike Begon, Kung-Sik Chan, Noelle I. Samia, Stephen Davis, Kyrre Linné Kausrud, Tamara Ben Ari, Lise Heier, Elisabeth Carniel, Mark Achtman, Kenneth L. Gage, Vladimir S. Ageyev, Nikolay L. Klassovskiy, and Sergey B. Pole. I have learned a lot from them—any misunderstandings of what they have tried to teach me is due solely to my own shortcomings. On a more administrative side, I would like to thank Dr. M. Pletschette for his stimulating encouragement, which made me start working on plague in the first case. My work on plague has been generously funded over the years through the European Union Projects (ISTC K-159, STEPICA [INCO-COPERNICUS, ICA 2-CT2000-10046], as well as Marie Curie Early Stage Training grant to CEES), the Norwegian Research Council, and my own university and center. Last, but not least, I extend my thanks to the many hundreds of Kazakh plague zoologists who collected so many data over all these years.

CLIMATE CHANGE AND PLANT DISEASE RISK

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Plant Disease and Ecosystem Services

One of the most important effects of plant disease is its impact on crop plant productivity. Oerke et al. (1994) estimated that damage by disease and insect pests resulted in a 42 percent loss in the eight most important food and cash crops. Pimentel et al. (2000) estimated that 65 percent of U.S. crop losses, $137 billion, were due to introduced pathogens. The effects of plant disease can also be considered within the broader context of ecosystem services, defined as the benefits provided to humans by ecosystems, including services provided by plants and their pathogens (Daily, 1997). Ecosystem services include the following: (1) provisioning services, such as the more obvious provisioning of food, fiber, fuel, and also the provisioning of genetic resources; (2) supporting services, such as

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soil formation, nutrient cycling, and primary productivity by plants, all of which have great economic value but tend to be appreciated only when there are breakdowns, such as the loss of soil during the U.S. dust bowl; (3) regulating services, such as regulation of climate, disease, and insect herbivory, and water purification; and (4) cultural services, such as opportunities for education, recreation, tourism, and inspiration. The Millennium Ecosystem Assessment[10] provides an example of system evaluation based on ecosystem services. Cheatham et al. (in revision) have synthesized perspectives on plant disease and its management in the context of ecosystem services. In addition to the direct effects of disease on crop production, disease and its management by increased tillage, pesticide use, and other methods may reduce services provided by plants such as soil formation and climate and water regulation. Disease may also remove plants that provide important cultural services in addition to the range of other potential services.

Some examples among the many notorious plant diseases illustrate the issues for disease management and the potential impact when diseases cannot be managed effectively. Chestnut blight has had one of the most definitive effects, essentially removing the once common American chestnut from the landscape of eastern North America (Anagnostakis, 2000). Potato late blight is infamous as the proximate cause of the Irish potato famine and continues as a major constraint to potato production, making the use of pesticides a typical part of potato management in many areas (Hijmans et al., 2000). Karnal bunt of wheat offers an example of a disease that does not cause major yield loss, but has an important economic impact on regions where it is present through limits on trade with Europe and other parts of the world where the pathogen has not been detected (Rush et al., 2005). Sudden oak death has changed the structure of some western U.S. forests and threatens to impact forests throughout a much wider area (Rizzo et al., 2005). Soybean rust is a new pathogen to the United States, with the potential to become established throughout much of the U.S. soybean production areas (Pivonia and Yang, 2004). Wheat stem rust was an important pathogen in the United States in the 1900s, motivating the removal of barberry plants that served as an alternate host and supported sexual reproduction of the pathogen. Disease resistance in U.S. wheat has been effective against this pathogen, but now new pathogen types for which this resistance is not useful have arisen in Africa and are likely to arrive in the United States in the near future (Stokstad, 2007).

The effects of climate on plant disease have been a direct object of study for decades. In contrast to many human diseases, the pathogens causing important plant diseases are often present on and around plants, ready to infect when environmental conditions become conducive. This has motivated the development of plant disease forecasting systems based on climatic variables such as

[10]The Millennium Ecosystem Assessment is an evaluation of the effects of ecosystem change on human well-being assembled from the work of more than 1,360 scientists (see http://www.millenniumassessment.org).

temperature and precipitation (De Wolf and Isard, 2007). Although such models are not new, the need to address climate change has placed new demands on these models, the research underpinning them, and policy drawing upon them. This paper begins with a brief introduction to the typical methods for managing disease. The observed and potential effects of climate change on plant disease are then reviewed, with an emphasis on biological thresholds and interactions that may lead to particularly large impacts from climate shifts. The paper concludes with a discussion of research and policy needs for plant disease management in response to climate change.

The Usual Challenges for Managing Plant Disease

Pesticides are a common tool for managing plant disease. For some plant diseases such as potato late blight, crop production without pesticides is currently impractical in many systems. In regions where education about pesticide safety is lacking, some farmers and their families experience chronic pesticide exposure. There are estimated to be between 1 and 5 million cases of pesticide poisoning each year, including many thousands of fatalities (UNEP, 2004). Shifts in pesticide use may thus result in shifts in unmanaged pesticide exposure, so that changes in demand for pesticides due to climate or other factors may have unexpected impacts on human health as well. Other disease management methods may be useful for specific diseases, such as removal of infected plant materials, introduction of biocontrol agents, management for disease-suppressive soils, or use of certified seed to avoid introduction of pathogens.

Deployment of disease resistance genes is often the most attractive option for disease management in agricultural systems. For some diseases, resistance offers completely effective management, whereas for others, effective resistance is not known although partial resistance may still be a useful management component. There is little cost from use of resistance genes to growers or consumers, except that in some cases it may be challenging for plant breeders to combine desired resistance genes with other desirable plant characteristics. Breeding crops for disease resistance also offers challenges in terms of identifying resistance that is durable. The deployment of resistance genes is much more efficient if the genes are useful against pathogen populations for long periods of time even if exposed to large pathogen populations under disease-conducive environmental conditions. Pathogen adaptation to overcome disease resistance is an ongoing problem for the management of many diseases (McDonald and Linde, 2002).

The use of cultivar mixtures is one method of resistance gene deployment that may increase the useful life of resistance genes in some cases. The management of rice blast in China offers a particularly dramatic example of the utility of mixtures for disease management, applied to over a million hectares. Higher-value susceptible rice varieties were grown in strips mixed with strips of lower-value resistant varieties. Both resistant and susceptible varieties experienced a

decrease in disease pressure compared to test plots where single varieties were grown for comparison (Zhu et al., 2000). In this case, it seems that microclimate was an important factor, such that the taller susceptible varieties experience relatively drier conditions when surrounded by the shorter resistant varieties (Zhu et al., 2005).

The fact that agriculturalists have the ability to manipulate crop plant genetics makes plant disease management in agriculture much easier, in some respects, than human disease management. Problems can also arise from this ability, however, as particularly successful crop varieties become widespread. Thus, a common challenge for plant disease management is the general homogeneity of cropping systems in the United States and trends toward greater crop homogeneity in most regions of the world. This homogeneity makes it easier for plant pathogens adapted to the common crop varieties to spread rapidly throughout crop plant populations. Margosian et al. (in revision) have evaluated the connectivity of the four major crop plants in the United States in terms of availability of the crop host species. The connectivity of a landscape for a particular organism, in this case a plant pathogen, is a measure of the ease with which the organism can move through the landscape. Maize and soybean are strongly connected throughout much of their range. Wheat and cotton production are more fragmented, so that pathogen populations cannot move as readily through all production areas. Conversion to biofuel production has the potential to increase crop homogeneity.

Maps of disease risk based on climate can be generated for diseases with reliable and widely applicable forecasting models. For example, Hijmans et al. (2000) mapped the risk of potato late blight based on climate parameters. Using updated forecasting models for potato late blight risk, Villanueva et al. (in preparation) estimated disease risk in the Altiplano region around Lake Titicaca (Figure 2-18). Such models are available for only very well-studied diseases, but Magarey et al. (2007) have developed a general model of infection risk for application in mapping the risk of new pathogens for which detailed models are not yet available. The combination of maps of current and future climatic conditions with models of pathogen risk can be adapted to evaluate changes in global risk in response to climate change. For example, Bergot et al. (2004) predicted the spread of the host-generalist pathogen *Phytophthora cinnamomi* in Europe.

Implications of Climate Change

Climate change will impact the productivity of agricultural and wildland plant populations through many mechanisms. One method for studying climate change effects on crop productivity is to study the correlation between climate variables and yield to date. Yield is the product of a number of factors, including losses to plant disease; partitioning the effects of these different factors will be necessary to develop a full understanding of the impacts of climate change.

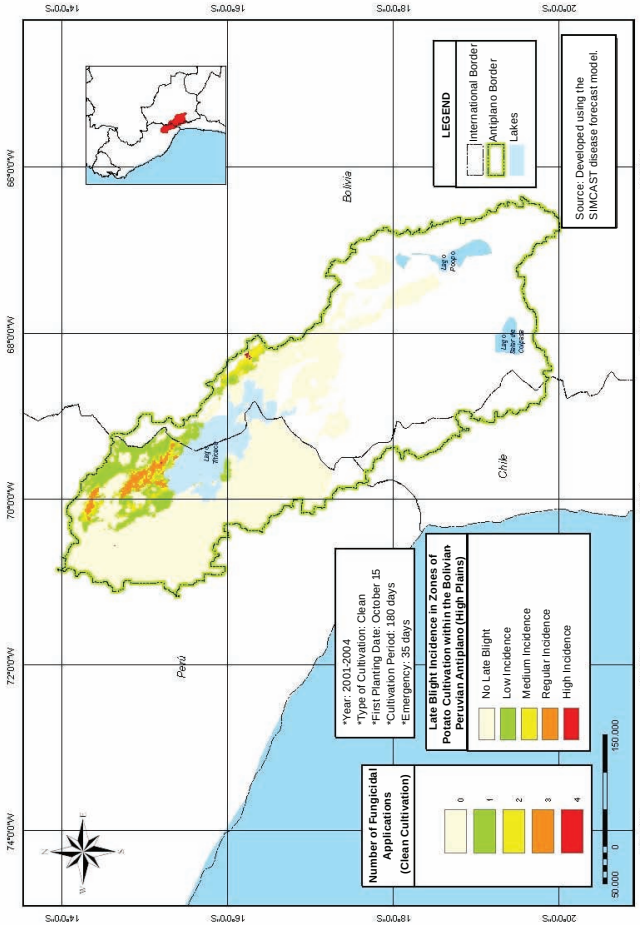

FIGURE 2-18 Estimated potato late blight severity in the Altiplano area of Peru and Bolivia based on weather measures during 2001-2004 used in a late blight forecasting model. The comparable estimates for disease severity in 1995-1998 were for no late blight occurrence in the region. As temperatures increase in the region, the risk of potato late blight may be expected to increase at higher altitudes. Color indicates the level of estimated disease, ranging from green = low to red = high, with a corresponding range in the number of fungicide applications needed for successful potato production. This figure was translated into English from the original (in Spanish) by Mila Gonzalez. SOURCE: Map courtesy of H. Villanueva, R. Raymundo, H. Juarez, W. Perez, and G. Forbes, International Potato Center.

The general trend toward warmer temperatures in recent years in many regions has facilitated study of the correlation between climate variables and yield. For example, rice productivity in the Philippines has shown a negative correlation with night temperature from 1979 to 2003 (Peng et al., 2004). Of course a challenge for such analyses is to account well for all of the other potential causal factors of the system that may vary along with climatic parameters. Lobell et al. (2008) have analyzed which regions of the world are most likely to be confronted with food security issues resulting from climate change, concluding that south Asia and southern Africa are particularly at risk. In another analysis of global agriculture, Cline (2007) points out that potential benefits to agriculture in some areas in the first decades of global temperature change may give the public a false sense of security and make it more difficult to put policies in place to avoid problems from more extreme changes in later decades.

A first step toward understanding wildland plant responses to climate change and the potential for adaptation to new climatic conditions is to address gene expression and underlying genetic diversity in wild plant populations. Travers et al. (2007) studied the effects of simulated precipitation change on big bluestem, the dominant grass of tallgrass prairie of the U.S. Great Plains. Under the predicted future precipitation patterns with fewer and larger precipitation events leading to longer periods of drought stress, they observed lower expression of a gene associated with the hypersensitive response, a disease resistance reaction. Frank (2007) also studied big bluestem, finding higher infection rates and dampened phytohormonal responses to infection when plants experienced severe drought stress. Studying the diversity of resistance genes in wild plant populations is still challenging because little is known about them and for the moment there are few tools available. Rouse (2007) studied a gene in big bluestem that is related to genes conferring disease resistance in sorghum, finding evidence for historical disease patterns in natural populations that vary in diversity for these genes across a gradient of disease conduciveness.

The effectiveness of disease resistance genes may vary with climatic parameters. For example, Webb et al. (in preparation) found that rice genes conferring resistance to rice blast have different effectiveness depending on temperature. Most resistance genes tested were less effective at higher temperatures, but one of the most effective genes was actually more effective at 35-29ºC day-night temperatures than at 29-21ºC. These differential responses will influence the selection pressures experienced by pathogen populations as temperatures fluctuate annually and shift over years (Webb et al., in preparation).

Climatic changes and changes in CO_2 concentrations can affect plant physiology, growth, and architecture in several ways that influence plant disease risk. On shorter time scales, stomatal closure in response to drought stress makes it more difficult for some pathogens to enter leaves. If plant canopies close earlier in the season due to changed conditions, the increased humidity in canopy microclimates may favor many pathogens. CO_2 concentrations are expected to

impact pathogens directly as well, although a model for this impact is unlikely to be simple. For example, in a study of a set of fungal pathogens, Chakraborty et al. (2000) found that some species reproduced more rapidly under increased CO_2, while other species reproduced more slowly.

In wildland systems, climate change and increased CO_2 concentrations may also have mixed effects. Mitchell et al. (2003) found that the fungal pathogen load in tallgrass prairie increased overall in response to higher ambient CO_2. In montaine prairie, Roy et al. (2004) found mixed effects of simulated temperature increases, with some pathogens increasing in abundance and others decreasing. Desprez-Loustau et al. (2007) predicted that the effect of climate change on a set of forest pathogens in Europe will be to increase favorability for the majority of pathogens. In general, rising temperatures may favor soil fungi that cause damping-off in seedlings, sometimes with high rates of mortality, a trend unlikely to be observed in the short term unless studies are designed specifically to look for such effects.

Range shifts in pathogens are frequently observed. As others have discussed at this workshop, such range shifts can be difficult to interpret. For example, needle blight is moving northward in North America as temperature and precipitation patterns shift (Woods et al., 2005). It is reasonable to think that such range shifts may be driven by changing climatic conditions, but the correlative nature of the data makes it impossible to determine this conclusively. Ultimately these relationships will have to be addressed in projects that combine the full range of factors in field studies as well as more limited and controlled experiments that allow clear conclusions about the effects of factors to partition effects.

The potential importance of extreme weather events is illustrated by the introduction of soybean rust to the United States. It is likely that spores of soybean rust entered the United States via Hurricane Ivan (Isard et al., 2005).[11] If such extreme weather events become more common, global movement of pathogens will be accelerated. Soybean rust also offers an interesting example of the potential interactions between two invasive species. The widely introduced and problematic kudzu vine is another host of this pathogen and has the potential to play an important role as a pathogen reservoir during seasons when soybeans are not available for infection. Until now, however, movement of soybean rust has been slower than expected based on some predictions, probably due to environmental conditions that have not been conducive to disease. If the public becomes too complacent about the slower-than-predicted progress of soybean rust across the United States, this may result in more substantial problems if there is not support for needed research and if soybean growers do not prepare adequately.

The ultimate impact of changes in plant disease pressure, in either agricultural systems or wildland systems, will be determined in part by what plant

[11]Of course, spores of this pathogen may well have entered the United States previously but been unsuccessful in establishing infection. Entry of large numbers of spores may be necessary for an invasive pathogen to "beat the odds."

genotypes or species replace those that have experienced more damage by disease. Eviner and Likens (2008) summarize factors important for predicting the effects of disease on ecosystems, where one of the most important factors may be the functional similarities of infected host individuals versus the species that replace them. Through a broad ecosystem science lens, plant species may be generalized as composing "a single giant photosynthesizing leaf." From this standpoint, damage to one or a few plant species may not be important if other species can play the same role. In eastern U.S. forests, while other tree species increased in abundance to photosynthesize in the place of American chestnuts, they did not provide other important ecosystem services such as production of chestnuts as food for humans and wildlife. Likewise, most agricultural systems are not diverse enough to readily accommodate removal of an important species such as soybeans, if soybean production were to become uneconomical due to a new disease such as soybean rust.

Potential Interactions, Thresholds, and Positive Feedback Loops

If a small change in average temperature or precipitation patterns results in a small change in plant disease risk, this may be relatively easy to accommodate in agricultural disease management and may have little impact on wildland systems. Climate change is a greater concern when interactions serve to amplify the effects on biological systems or when systems are currently near thresholds such that small changes in abiotic drivers may push them beyond the threshold and thus have important effects. Effects may also be exacerbated if positive feedback loops are in place so that increased disease pressure further increases disease risk.

Abiotic environmental conditions are understood to be critically important in plant disease epidemiology, as commonly represented in the "plant disease triangle" (Figure 2-19). The three components of this triangle are a susceptible host, a virulent pathogen (and effective vector, as needed), and a conducive abiotic environment. For example, many fungal and oomycete pathogens benefit from higher levels of humidity. Surprising new disease problems may occur if the susceptible host and virulent pathogen have been present all along and the environment shifts to become more conducive. For example, potato late blight became an extreme problem for Irish food security during the potato famine when wetter years supported rapid disease development. The further interaction between high losses to disease and widespread reliance on potatoes as a primary food led to a disastrous situation.

Allee effects represent one type of threshold. An Allee effect occurs when a species experiences greater limitations on per capita reproduction for small population sizes. Quorum sensing provides an interesting potential mechanism for this type of phenomenon, where bacterial populations may become pathogenic only when intraspecific signaling indicates that a sufficiently large population is present for infection. Smaller population sizes may also make it less likely that

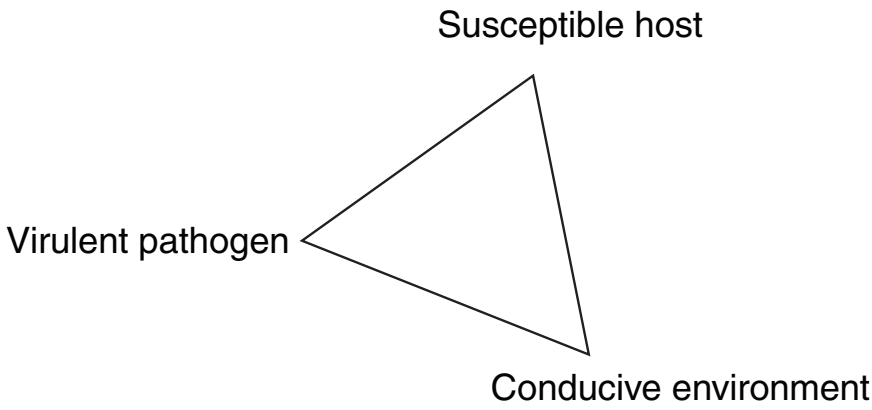

FIGURE 2-19 The plant disease triangle, illustrating the relationship between host, pathogen, and environment necessary for disease to occur.

individuals encounter mates. For example, the Karnal bunt pathogen requires encounters between two mating types for reproduction to occur, yet its propagules are wind dispersed, making encounters between individuals of different mating types unlikely when populations are small. The resulting Allee effect may help to explain why this species has not been more invasive, since encounters between mating types will be even less likely when dispersed by wind over larger areas (Garrett and Bowden, 2002). For species that experience them, such Allee effects interact with disease nonconducive environmental conditions to reduce the chance of infection still further. As a result, if climatic conditions become more conducive to disease so that pathogens are released from the constraint of the Allee effect, pathogen populations may increase much more rapidly than anticipated.

The typical "compound interest" development of plant disease epidemics for pathogens with multiple generations per season can also result in important threshold structures. Infection levels can often increase by orders of magnitude toward the end of the season. If the length of the growing season increases, regional production of particular crop species may expand over time, with the longer season length allowing for huge increases in pathogen populations toward the end of the growing season. These populations may reduce yields during that season and also serve as large sources of inoculum for upcoming cropping seasons. Such higher regional inoculum loads may produce positive feedback loops, rendering local application of some management techniques less useful. For example, local application of techniques such as sanitation (removal of infected plant materials), use of cultivar mixtures, and use of disease resistance based on lower inoculum production all rely, at least to some extent, on an ability to control

local inoculum loads. If regional inoculum loads are too high, the contributions of these methods will be diminished (Garrett et al., in revision). Likewise, in wildland systems, plant diversity probably provides baseline regulation of plant disease that is unappreciated but may be diminished if systems become saturated with inoculum. Conversely, if seasons become shorter or if climatic conditions during significant parts of growing seasons become less disease conducive, greater benefits may be obtained from some management techniques.

Pathogen range shifts may occur as climatic conditions change to allow infection and overwintering or oversummering in new areas. The effects of climatic shifts may interact with other phenomena, such as the introduction of new pathogen species or pathogen genotypes. For example, overwintering of the potato late blight pathogen is facilitated by the presence of different mating types, which allows sexual production of a much hardier oospore and the potential for adaptation through production of new genotypes (Widmark et al., 2007). The combination of milder winters and introduction of new mating types may greatly increase problems with such pathogens. Range shifts and pathogen introductions will also result in new encounters between pathogen species, with the potential for hybridization to produce new pathogens (Brasier, 2001). Likewise, the introduction or range shift of new vector species may make diseases much more important, such as in the case of the movement of the glassy-winged sharpshooter and resulting increased risk of Pierce's disease of grapevines (Redak et al., 2004).

Phenological shifts and range shifts in response to climate change may not follow the same patterns for plant hosts and pathogens. Some pathogens can only infect particular plant growth stages or organs, such as flowers. For example, the *Fusarium* head blight pathogen infects wheat anthers or other floral organs (Bai and Shaner, 2004). Shifts in flowering time phenology in response to climate change may not match shifts in pathogen phenology such that infection rates may unexpectedly rise or fall. Different patterns of geographic range shifts may result in new pathogen-host combinations (Parker and Gilbert, 2004). The genetic potential for adaptability of pathogen populations will be important in determining whether any resulting reductions in infection will be short term or lasting. In general, the timeline of pathogen adaptation is likely to be much shorter than the timeline for plant adaptation. This will be especially true for long-lived plant species in wildlands, but also for annual crop species even with the full attention of agricultural scientists.

Policy may also interact in important ways with abiotic conditions. Along with the Irish potato famine, another dramatic example is the dustbowl in the central United States. Policies that supported extensive plowing of lands in this area coincided with climatic conditions favoring wind erosion of soils. Either factor alone might have caused problems, but the combination of the two led to conditions devastating to the region. The interaction of biological and sociological factors may also result in amplified effects of climate change. For example, if

plant pathogens are intentionally introduced (Fletcher and Stack, 2007; Madden and Wheelis, 2003), bioterrorists using them might seek out the most environmentally conducive conditions for their establishment in vulnerable cropping systems. Regions where local food security is closely tied to local food production will be particularly vulnerable to changes in crop disease pressure. Yet societies in these regions may also tend to rely on crop species that are less well supported by research and development. These "orphan crops," such as millet, quinoa, cassava, and teff, need more research support to buffer the vulnerability of societies to which they are important (Nelson et al., 2004).

Responding to Climate Change

The good news for formulation of strategies for plant disease management under changing climate conditions is that much of what needs to be done is the same with or without climate change. Even if there were no long-term trend in climatic parameters, climatic variation from season to season, year to year, and region to region requires knowledge and tools for adapting to the different scenarios. However, the potential for new combinations of climatic variables, along with the potential for interactions and for more rapid variation in conditions, reinforces the need for research and policy responses to plant disease risk (Coakley et al., 1999; Garrett et al., 2006). Research directed explicitly toward understanding the complexity of system responses to climate change is needed.

A mechanistic understanding of plant and pathogen responses to climate change will be based on characterizing current populations and their potential for adaptation. New genomic tools make it possible to characterize gene expression and genotypic diversity much more readily in both wildland and agricultural plant communities. These tools can be applied in concert with other "-omics" approaches to link responses in gene expression (transcriptomics), lipidomics, and metabolomics for a fuller mechanistic understanding of adaptive potential. These approaches will have to be applied in multifactor studies of climate change effects, so that the interactions between the effects of changes in temperature, precipitation, CO_2, and other environmental factors can be understood, along with the potential for adaptation.

Tools for the study of pathogen population and community structure, gene expression, and other responses are evolving rapidly. Advances in sequencing technologies make the routine characterization of microbial communities feasible (Riesenfeld et al., 2004; Roesch et al., 2007) and will eventually make it inexpensive. Microarrays, such as the GeoChip (He et al., 2007), are being designed to study microbial gene function in soils. New microarrays are needed to study the presence and expression of microbial genes related to plant disease. It will be important to collect baseline information about microbial community structure and function soon, so that changes in microbial communities under new climatic conditions can be studied. Experiments to compare responses of microbial com-

munities to new environments will also be important (e.g., Waldrop and Firestone, 2006). Undoubtedly there are many forms of disease suppressiveness provided by microbial communities in soils that offer benefits to agriculture and regulate disease in natural systems and are currently underappreciated.

Research to clarify the effects of host landscape structures will help to improve strategies and will be necessary for studying changes at regional, continental, and global scales. Current regional analyses of climatic effects on disease risk tend to be calculated for disease risk in individual "pixels," important for developing a first-approximation estimate of risk. The next stage for such models will be to incorporate risk neighborhoods to improve estimates, where the risk for any given location will increase with proximity to higher-risk areas. Finally, regional and global models will need to incorporate pathogen evolution. Formulating and parameterizing these models will require advances in epidemiological theory and experimentation. For example, better data and models related to pathogen and host dispersal, current levels of intraspecific diversity, and the strength of selection under different climate change scenarios are needed.

Long-term geographically representative records of disease occurrence and the distribution of pathogens and hosts are rare, despite their importance for understanding epidemiology and trends in epidemics (Jeger and Pautasso, 2008). Global networks supporting the analysis of epidemics are needed. Progress toward this goal is in place; for example, the United States has developed a National Plant Diagnostic Network to facilitate data collection and analysis (Stack and Fletcher, 2007). To be most effective, this network ultimately needs to be linked with comparable national networks in other countries. It is to the advantage of the United States to assist other countries in setting up such networks for gathering and analyzing data, so that we can all benefit from more complete information. The use of model predictions for modifying agricultural management has proven useful in many parts of the world, including applications by resource-poor farmers based on climate predictions in Zimbabwe (Patt et al., 2005).

One of the most important investments we can make is in conservation, characterization, and the development of strategies for optimal use of plant genetic resources. In wildland systems, conservation is necessary to increase the chances that plant populations are large enough to include individual genotypes adapted to new climate scenarios. In agricultural systems, conservation of diversity in crop species and their wild relatives is necessary to increase the chances that genes needed for resistance and tolerance to new biotic and abiotic stresses are maintained (Johnson, 2008). *In situ* conservation allows natural selection to continue acting on these species. *Ex situ* conservation is a useful backup strategy and simplifies some analyses of accessions. International networks for conservation of crop genetic diversity, such as the institutions in the Consultative Group for International Agricultural Research (CGIAR), are critical for ensuring conservation and analysis of accessions. The funding currently available for such programs is very low compared to the importance of their mission. While investments such

as the Svalbard Global Seed Vault provide a last resort, active investigation of plant resources is needed.

Ultimately our best response to the challenge of climate change in agriculture will be to develop diverse, flexible, and resilient agricultural systems that can adapt more readily to new climatic conditions. These systems will have to include well-prepared and well-funded agricultural scientists working globally to develop new strategies. In wildland systems, replacing plant species or genotypes at risk is a less attractive option. Since invasive pathogens can have the most important effects and have the potential to exacerbate the effects of climate change, policies to better reduce the spread of exotic pathogens will be important (Anderson et al., 2004; Burdon et al., 2006).

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CLIMATE CHANGE AND INFECTIOUS DISEASE: IMPACT ON HUMAN POPULATIONS IN THE ARCTIC[12]

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Introduction: The Arctic Environment

The circumpolar region is defined as the region that extends above 60°N latitude, borders the Arctic Ocean, and includes all of or the northern parts of eight nations: the United States (Alaska), Canada, Greenland, Iceland, Norway,

[12]The findings and conclusions in this report are those of the author and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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Finland, Sweden, and the Russian Federation (see Figure 2-20). The climate in the Arctic varies geographically from severe cold in arid uninhabited regions to temperate forests bordering coastal agrarian regions. Approximately 4 million people live in the Arctic and almost half reside in northern regions of the Russian Federation. Peoples of the Arctic and sub-Arctic regions live in social and physical environments that differ substantially from those of their more southern dwelling counterparts. These populations are comprised of varying proportions of indigenous and nonindigenous peoples (Stephansson Arctic Institute, 2004; see Figure 2-21).

The indigenous populations of northern Canada (Northwest Territories, Yukon, Nunavut, northern Quebec, and Labrador), Alaska, and Greenland generally reside in small communities in remote regions. They have little economic infrastructure and depend on subsistence hunting, fishing, and gathering of food for a significant proportion of their diet. In these remote areas, access to public health and acute care systems is often marginal and poorly supported. Life expectancy of the indigenous peoples of Alaska, northern Canada, and Greenland is lower than that of the general populations of the United States, Canada, and Nordic countries (Young, 2008). Similarly the infant morality rate for the indigenous segments of these populations is higher than that of the comparable national populations. Mortality rates for heart disease and cancer, once much lower among the indigenous populations of the United States, Canada, and northern European countries, are now similar to their respective national rates. The indigenous populations of Alaska, Canada, and Greenland have higher mortality rates for unintentional injury and suicide. Other health concerns of the indigenous peoples of the Arctic include the high prevalence of certain infectious diseases, such as hepatitis B, *Helicobacter pylori*, respiratory syncytial virus (RSV) infections in infants, and sexually transmitted diseases, as well as heath impacts associated with exposures to environmental pollutants, rapid economic change and modernization, and climate change (Bjerregaard et al., 2004).

Climate Change and the Arctic Environment

The Arctic, like most other parts of the world, warmed substantially over the twentieth century, principally in recent decades. Arctic climate models project continued warming with a 3-5ºC mean increase by 2100. The winters will warm more than summers, the mean annual precipitation is projected to increase, and continued melting of land and sea ice is expected to increase river discharge and contribute to rising sea levels. These changes will be accompanied by greater overall climate variability and an increase in extreme weather events (Arctic Council, 2005).

The rapid warming in the Arctic is already bringing about substantial ecological and socioeconomic impacts, many of which result from the thawing of permafrost, flooding, and shoreline erosion resulting from storm surges and

FIGURE 2-20 The circumpolar region showing administrative jurisdictions.
SOURCE: Map by W. K. Dallmann. Reprinted from Young (2008) with permission from W. K. Dallmann and the *International Journal of Circumpolar Health*. Copyright 2008.

loss of protective sea ice. In many communities, the built infrastructure is supported by permafrost. Loss of this permafrost foundation will result in damage to water intake systems and pipes, and may result in contamination of the community water supply. In addition, loss of foundation support for access roads, boardwalks, water storage tanks, and wastewater treatment facilities will render water distribution and wastewater treatment systems inoperable. Several villages already face relocation because village housing, water system, and infrastructure are being undermined (Warren et al., 2005).

Rapid warming has resulted in the loss of annual Arctic sea ice. On September 11, 2007, the Arctic sea ice cover reached the lowest extent recorded since

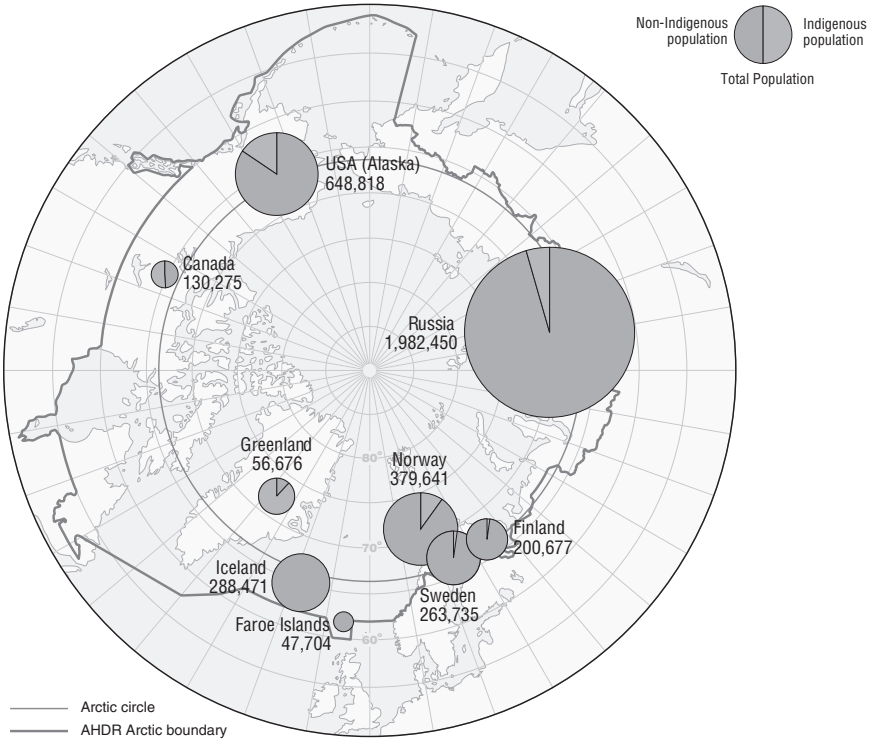

FIGURE 2-21 The circumpolar region showing indigenous and nonindigenous population distributions.
SOURCE: Reprinted from Stefansson Arctic Institute (2004) with permission from W. K. Dallmann, Norwegian Polar Institute and the Stefansson Arctic Institute. Copyright 2004.

observations began in the 1970s, exceeding the most pessimistic model predictions of an ice-free Arctic by 2050 (Richter-Menge et al., 2008; Figure 2-22). This dramatic reduction in sea ice will have widespread effects on marine ecosystems, coastal climate, human settlements, and subsistence activities. For the first time the reduction in annual sea ice has created ice-free shipping lanes to the northwest, from northern Labrador through the Arctic archipelago in northern Canada, to the Bering Strait, and has almost completely cleared a passage to the northeast, from the Bering Strait along the northern coast of the Russian Federation to Norway (see Figure 2-23). Both routes represent time- and fuel-saving shortcuts between the Pacific and Atlantic Oceans and will bring an increase in marine transport and access to vast oil, gas, and mineral reserves once inaccessible to exploration and exploitation.

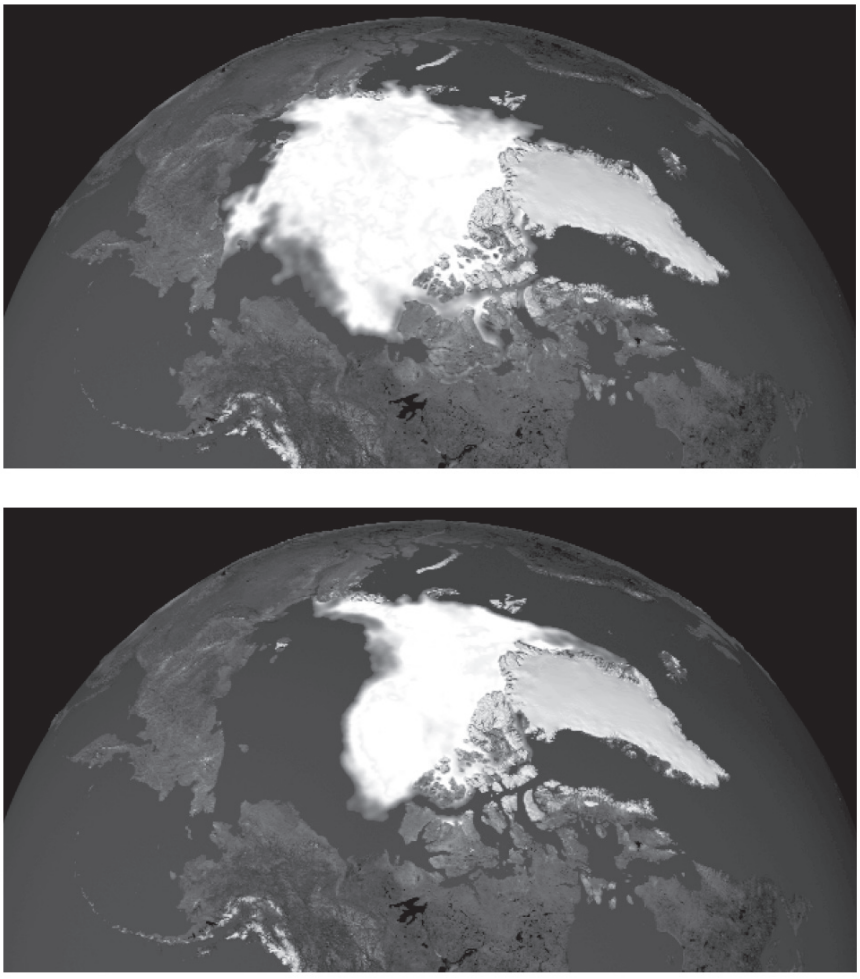

FIGURE 2-22 The Arctic ice cap, September 2001 (Top) and September 2007 (Bottom).
SOURCE: NASA, as printed in Borgerson (2008).

Such access will bring many benefits as well as risks to once isolated Arctic communities. Construction of new coast guard or military bases and other industrial ventures will bring employment opportunities to local populations, but will also affect population distribution, dynamics, culture, and local environments. Tourism will most likely increase. Public sector and government services will then increase to support the new emerging economies. These events will greatly

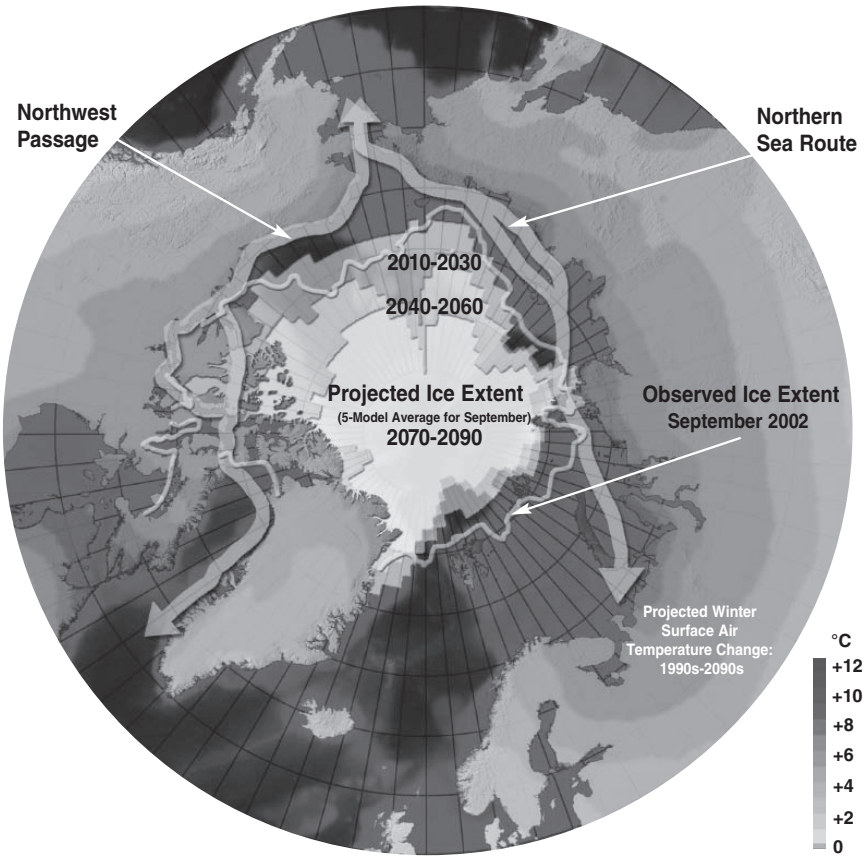

FIGURE 2-23 Proposed northwest and northeast shipping lanes through the Arctic Ocean joining the Atlantic and Pacific Oceans.
SOURCE: Map by C. Grabhorn Reprinted from ACIA (2004) with permission from Cambridge University Press and C. Grabhorn.

challenge the traditional subsistence way of life for many communities and lead to rapid and long-term cultural change, which will create additional stress on an already vulnerable population (Curtis et al., 2005).

Climate Change and Human Health

The direct health effects of climate change will result from changes in ambient temperature, altered patterns of risk from outdoor activities, and changes in the incidence of infectious diseases. As ambient temperature increases, the

incidence of hypothermia and associated morbidity and mortality may decrease. Conversely hyperthermia may increase, particularly among the very young and the elderly (Nayha, 2005). However, because of the low mean temperature in many Arctic regions, the likelihood of such events having large impacts on public health for the general population is low. More significantly, unintentional injury, mostly related to subsistence hunting and fishing—already a significant cause of mortality among Arctic residents—may increase (Arctic Council, 2005). The reduction in river and sea ice thickness, curtailed ice season, reduced snow cover, and permafrost thawing will make hunting and gathering more difficult, dangerous, and less successful, thereby increasing the risk of injuries and death by drowning.

Permafrost thawing erosion or flooding can force relocation. Communities and families undergoing relocation will have to adapt to new ways of living, may face unemployment, and will have to integrate and create new social bonds. Relocation may also lead to rapid and long-term cultural change and loss of traditional culture, which will increase individual and community stress, leading to mental and behavioral health challenges (Hess et al., in press).

Climate change already poses a serious threat to the food security of many Arctic communities because of their reliance on traditional subsistence hunting and fishing for survival. Populations of marine and land mammals, fish, and waterfowl may be reduced or displaced by changing habitats and migration patterns, further reducing the traditional food supply. Release of environmental contaminants from the atmosphere and melting glaciers and sea ice may increase the levels of these pollutants entering the food chain, making traditional foods less desirable (AMAP, 2003). Reduction in traditional food supply will force indigenous communities to depend increasingly on nontraditional and often less healthy Western foods. This will most likely result in increasing rates of modern diseases associated with processed foods, such as obesity, diabetes, cardiovascular diseases, and outbreaks of food-borne infectious diseases associated with imported fresh and processed foods (Bjerregaard et al., 2004; Orr et al., 1994).

Many host-parasite systems are particularly sensitive to climate change. Specific stages of the life cycles of many helminths may be greatly affected by temperature. For example, small increases in temperature can substantially increase the transmission of lung worms and muscle worms pathogenic to wildlife that are important as a food source for many northern communities (Hoberg et al., 2008).

Climate Change and Infectious Diseases in the Arctic

It is well known that climate and weather affect the distribution and risk of many vector-borne diseases, such as malaria, RVF, plague, and dengue fever in tropical regions of the globe. Weather also affects the distribution of food- and water-borne diseases and emerging infectious diseases, such as West Nile virus,

hantavirus, and Ebola hemorrhagic fever (Haines et al., 2006). Less is known about the impact of climate change and the risk and distribution of infectious diseases in Arctic regions. It is known that Arctic populations have a long history of both endemic and epidemic infectious diseases (Parkinson et al., 2008). However, with the introduction of antimicrobial drugs, vaccines, and public health systems, morbidity and mortality due to infectious diseases have been greatly reduced. Despite these advances, high rates of invasive diseases caused by *Streptococcus pneumoniae, Haemophilus influenzae,* and *Mycobacterium tuberculosis* persist (Bruce et al., 2008a,b; Christensen et al., 2004; Dawar et al., 2002; Degani et al., 2008; Gessner et al., 1998; Meyer et al., 2008; Netesov and Conrad, 2001; Nguyen et al., 2003; Singleton et al., 2006; Søborg et al., 2001). Sharp seasonal epidemics of viral respiratory infections also commonly occur (Bulkow et al., 2002; Karron et al., 1999; Van Caeseele et al., 2001). The overuse of antimicrobial drugs in some regions has led to the emergence of multidrug-resistant *S. pneumoniae*, *Helicobacter pylori*, and methicillin-resistant *Staphylococcus aureus* (Baggett et al., 2003, 2004; McMahon et al., 2007; Rudolph et al., 1999, 2000).

The impact of climate on the incidence of these existing infectious disease challenges is unknown. In many Arctic regions, however, inadequate housing and sanitation are already important determinants of infectious disease transmission. The cold northern climate keeps people indoors amplifying the effects of household crowding, smoking, and inadequate ventilation. Crowded living conditions increase person-to-person spread of infectious diseases and favor the transmission of respiratory and gastrointestinal diseases and skin infections. Many homes in communities across the Arctic lack basic sanitation services (e.g., flush toilet, shower or bath, kitchen sink). Providing these services is difficult in remote villages where small isolated populations live in a harsh cold climate. A recent study in western Alaska demonstrated two to four times higher hospitalization rates among children less than 3 years of age for pneumonia, influenza, and childhood RSV infections in villages where the majority of homes had no in-house piped water, compared with villages where the majority of homes had in-house piped water service. Likewise, outpatient *Staphylococcus aureus* infections and hospitalization for skin infections among persons of all ages were higher in villages with no in-house piped water service compared to villages without water service (Hennessy et al., 2008). Damage to the sanitation infrastructure by melting permafrost or flooding may therefore result in increased rates of hospitalization among children for respiratory infections, as well as an increased rate of skin infections and diarrheal diseases caused by bacterial, viral, and parasitic pathogens.

Some infectious diseases are unique to the Arctic and lifestyles of the indigenous populations and may increase in a warming Arctic. For example, many Arctic residents depend on subsistence hunting, fishing, and gathering for food, and on a predictable climate for food storage. Food storage methods often include above ground air-drying of fish and meat at ambient temperature, below ground

cold storage on or near the permafrost, and fermentation. Changes in climate may prevent the drying of fish or meat, resulting in spoilage. Similarly, loss of the permafrost may result in spoilage of food stored below ground. Outbreaks of food-borne botulism occur sporadically in communities in the United States, Canadian Arctic, and Greenland and are caused by ingestion of improperly prepared fermented traditional foods (CDC, 2001; Proulx et al., 1997; Sobel et al., 2004; Sørensen et al., 1993; Wainwright et al., 1988). Because germination of *Clostridium botulinum* spores and toxin production will occur at temperatures greater than 4°C, it is possible that warmer ambient temperatures associated with climate change may result in an increased rate of food-borne botulism in these regions. Preliminary studies have shown that fermentation of aged seal meat challenged with *C. botulinum* at temperatures above 4ºC results in toxin production (Leclair et al., 2004).

Outbreaks of gastroenteritis caused by *Vibrio parahaemolyticus* have been related to the consumption of raw or inadequately cooked shellfish collected from seawater at temperatures of higher than 15ºC. Prior to 2004, the most northerly outbreak occurred in northern British Columbia in 1997. However, in July 2004, an outbreak of gastroenteritis caused by *V. parahaemolyticus* was documented among cruise ship passengers consuming raw oysters while visiting an oyster farm in Prince William Sound, Alaska (McLaughlin et al., 2005). The outbreak investigation documented an increase of 0.21ºC per year in the July-August water temperature since 1997, and reported that 2004 was the first year that the oyster farm water temperature exceeded 15ºC in July. This event provides direct evidence of an association between rising seawater temperature and the onset of illness.

Warmer temperatures may allow infected host animal species to survive winters in larger numbers, increase in population, and expand their range of habitation, thus increasing the opportunity to pass infections to humans. For example, milder weather and less snow cover may have contributed to a large outbreak of Puumala virus infection in northern Sweden in 2007. Puumala virus is endemic in bank voles, and in humans causes hemorrhagic fever with renal syndrome (Pettersson et al., 2008). Similar outbreaks have been noted in the Russian Federation (Revich, 2008). The climate-related northern expansion of the boreal forest in Alaska and northern Canada has favored the steady northward advance of the beaver, extending the range of *Giardia lamblia*, a parasitic infection of beaver that can infect other mammals, including humans who use untreated surface water (Arctic Council, 2005). Similarly, warmer temperatures in the Arctic and sub-Arctic regions could support the expansion of the geographical range and populations of foxes and voles, common carriers of *Echinococcus multilocularis,* the cause of alveolar echinococcus in humans (Holts et al., 2005). The prevalence of alveolar echinococcus has risen in Switzerland as fox populations have increased in size and expanded their geographic ranges into urban areas (Schweiger et al., 2007). Alveolar echinococcus was common in two

regions of northwestern Alaska prior to 1997. Disease in humans was associated with contact with dogs; however, improvements in housing and dog lot management have largely eliminated dog-to-human transmission in Alaska. This may not be the case, however, in other parts of the Arctic where human infections with *Echinococcus granulosis*, and *E. multilocularis* are still reported, particularly in association with communities dependent on reindeer herding and dog use (Castrodale et al., 2002; Rausch, 2003).

Climate change may also influence the density and distribution of animal hosts and mosquito vectors, which could result in an increase in human illness or a shift in the geographical range of disease caused by these agents. The impact of these changes on human disease incidence has not been fully evaluated, but there is clearly potential for climate change to shift the geographical distribution of certain vector-borne and other zoonotic diseases. For example, West Nile virus entered the United States in 1999 and in subsequent years infected human, horse, mosquito, and bird populations across the United States and as far north as northern Manitoba, Canada (Parkinson and Butler, 2005). In the Russian Federation infected birds and humans have been detected as far north as the region of Novosibirsk (Revich, 2008). Although there is, at present, insufficient information about the relationship between climate and the spread of West Nile virus, a number of factors may contribute to its further northward migration. Milder winters could favor winter survival of infected *Culex spp*. mosquitoes, the predominant vector of West Nile virus, which since the 1970s have migrated as far north as Prince Albert, Saskatchewan in Canada. Longer, hotter summers increase the transmission season leading to higher numbers of infected mosquitoes and greater opportunities for human exposure. Climate change may alter the disease ecology and migration patterns of other reservoirs such as birds. These factors may affect disease incidence and result in expansion of the range of other arthropod vector-borne diseases.

A number of mosquito-borne viruses that cause illness in humans circulate in the U.S. Arctic and northern regions of the Russian Federation (Walters et al., 1999). Jamestown Canyon and Snowshoe Hare viruses are considered emerging threats to the public health in the United States, Canada, and the Russian Federation, causing flu-like symptoms and central nervous system diseases, such as aseptic meningitis and encephalitis (Walters et al., 1999). Sindbis virus also circulates in northern Europe. The virus is carried northward and amplified by migratory birds. In the late summer, ornithophilic mosquitoes pass the virus onto humans causing epidemics of Pogosta disease in northern Finland, an illness characterized by a rash and arthritis (Kurkela et al., 2008). In Sweden, the incidence of tick-borne encephalitis (TBE) has substantially increased since the mid-1980s (Lindgren and Gustafson, 2001). This increase corresponds to a trend of milder winters and an earlier onset of spring, resulting in an increase in the tick population (*Ixodes ricinus*) that carries the virus responsible for TBE and other potential pathogens (Skarphédinsson et al., 2005). Similarly in north-

eastern Canada, climate change is projected to result in a northward shift in the range of *Ixodes scapularis,* a tick that carries *Borrelia burgdorferi,* the etiologic agent of Lyme disease. The current northern limit of *Ix. scapularis* is southern Ontario including the shoreline of Lake Erie and southern coast of Nova Scotia. Some temperature-based models show the potential for a northward expansion of *Ix. scapularis* above 60ºN latitude and into the Northwest Territories by 2080 (Ogden et al., 2005). However, it should be noted that tick distribution is influenced by additional factors such as habitat suitability and dispersal patterns which can affect the accuracy of these predictions. Whether or not disease in humans is a result of these climate change-induced alterations in vector range depends on many other factors, such as land-use practices, human behavior (suburban development in wooded areas, outdoor recreational activities, use of insect repellents, etc.), human population density, and adequacy of the public health infrastructure.

Response to Climate Change in the Arctic

In 1992, the IOM published a report titled *Emerging Infections: Microbial Threats to Health in the United States*. This report uncovered major challenges for public health in the medical community primarily related to detecting and managing infectious disease outbreaks and monitoring the prevalence of endemic infectious diseases. It stimulated a national movement to reinvigorate the U.S. public health system to address the HIV/AIDS epidemic, the emergence of new diseases, the resurgence of old diseases, and the persistent evolution of antimicrobial resistance. In a subsequent report, the IOM provided an assessment of the capacity of the public health system to respond to emerging threats and made recommendations for addressing infectious disease threats to human health (IOM, 2003).

Because climate change is expected to exacerbate many of the factors contributing to infectious disease emergence and reemergence, the recommendations of the 2003 IOM report can be applied to the prevention and control of emerging infectious disease threats resulting from climate change. A framework for public health response to climate change in the United States has recently been proposed (Frumkin et al., 2008; Hess et al., in press). The framework emphasizes the need to capitalize on and enhance existing essential public health services and to improve coordination efforts between government agencies (federal, state, and local), academia, the private sector, and nongovernmental organizations.

Applying this framework to Arctic regions requires enhancing the public health capacity to monitor diseases with potentially large public health impacts, including respiratory diseases in children, skin infections, and diarrheal diseases, particularly in communities with failing sanitation systems. Monitoring certain vector-borne diseases, such as West Nile virus, Lyme disease, and TBE, should be priorities in areas at the margins of focal regions known to support both animal and insect vectors, and where climate change may promote the geographic

expansion of vectors. Because Arctic populations are relatively small and widely dispersed over a large area, region-specific detection of significant trends in emerging climate-related infectious diseases may be delayed. This difficulty may be overcome by linking regional monitoring systems together for the purposes of sharing standardized information on climate-sensitive infectious diseases of mutual concern. Efforts should be made to harmonize notifiable disease registries, laboratory methods, and clinical surveillance definitions across administrative jurisdictions to allow comparable disease reporting and analysis. An example of such a network is the International Circumpolar Surveillance system for emerging infectious diseases. This network links hospital and public health laboratories together for the purposes of monitoring invasive bacterial diseases and tuberculosis in Arctic populations (Parkinson et al., 2008).

Public health capacity should be enhanced to respond to infectious disease food-borne outbreaks (e.g., botulism, gastroenteritis caused by *Giardia lamblia* or *Vibro parahaemolyticus*). Public health research is needed to determine the baseline prevalence of potential climate-sensitive infectious diseases (e.g., West Nile virus, *Borrelia burgdorferi*, *Brucella spp.*, *Echinococcus spp., Toxoplasma spp.*) in both human and animal hosts in regions where emergence may be expected. Such studies can be used to accumulate additional evidence of the effect of climate change or weather on infectious disease emergence, to guide early detection and public health intervention strategies, and to provide science-based support for public health actions on climate change. The circumpolar coordination of research efforts will be important not only to harmonize research protocols, laboratory methods, data collection instruments, and data analysis, but also to maximize the impact of scarce resources and to minimize the impact of research on affected communities. Coordination can be facilitated through existing international cooperatives, such as the Arctic Council,[14] the International Union for Circumpolar Health,[15] and the newly formed International Network of Circumpolar Health Researchers.[16]

The challenge in the Arctic, however, will be to ensure sufficient public health capacity to allow the detection of disease outbreaks and monitor infectious disease trends most likely to be influenced by climate. The remoteness of many communities from clinical or public health facilities, and the harsh weather conditions of Arctic regions, often preclude appropriate specimen and epidemiologic data collection during an outbreak investigation, research, or ongoing surveillance activities. Staffing shortages are frequent in many in local clinics and regional hospitals that are already overwhelmed by routine and urgent care priorities, leaving little capacity for existing staff to assist public health personnel in outbreak investigations, research, or maintenance of routine surveillance activities.

[14]See http://www.arctic-council.org.
[15]See http://www.iuch.org.
[16]See http://www.inchr.org.

Additional resources and training may be needed to ensure adequate staffing at these facilities, to address existing gaps between regional clinics and hospitals and public health departments, and to ensure a sufficiently trained staff to address the emerging public health impacts posed by climate change.

A key aspect of the public health response to climate change in Arctic regions will be the formation of community-based partnerships with tribal governments to identify potential threats to the community and develop strategies to address those threats. Communities at greatest risk should be targeted for education, outreach, and assessment of existing or potential health risks, vulnerabilities, and engagement in the design of community-based monitoring and the formulation of intervention strategies. The identification, selection, and monitoring of basic indicators for climate change and community health will be important for any response to climate change at the community level (Furgal, 2005). The selection of site- or village-specific indicators should be guided by local concerns and may include activities such as the surveillance of a key wildlife or insect species in a region where climate changes may contribute to the emergence of new zoonotic diseases or the measurement of weather (i.e., precipitation and temperature), water quality (i.e., turbidity, pathogens), and gastrointestinal illness (i.e., clinic visits) in a community. Linking communities across regions and internationally should facilitate the sharing of standard protocols, data collection instruments, and data for analysis. These linkages will be important for the detection of trends over larger geographic regions, should enhance a community's ability to detect changes that impact health, and will allow the development of strategies to minimize the negative health impacts of climate change on Arctic residents in the future.

Conclusion

Resident indigenous populations of the Arctic are uniquely vulnerable to climate change because of their close relationship with, and dependence on, the land, sea, and natural resources for their cultural, social, economic, and physical well-being. The increasing mean ambient temperature may lead to an increase in food-borne diseases, such as botulism and gastrointestinal illnesses. An increase in mean temperature may also influence the incidence of zoonotic and arboviral infectious diseases by changing the population density and range of animal hosts and insect vectors. The public health response to these emerging microbial threats should include enhancing the public health capacity to monitor climate-sensitive infectious diseases with potentially large public health impacts; the prompt investigation of infectious disease outbreaks that may be related to climate change; and research on the relationship between climate and infectious disease emergence to guide early detection and public health interventions. The development of community-based monitoring networks with links to regional and national public health agencies as well as circumpolar health organizations will facilitate method

standardization, data-sharing, and the detection of infectious disease trends over a larger geographic area. This capacity is essential for the development of strategies to minimize the negative effects of climate change on the health of Arctic residents in the future.

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