Russian heat wave led Russia to halt grain exports, causing higher grain prices on the world market and food riots in developing nations.

EFFECTS OF CLIMATE CHANGE ON INFECTIOUS DISEASE

The incidence of most, if not all, infectious diseases depends on climate. For any given infection, however, climate change is but one of many factors that determine disease epidemiology, and often it is not the most influential factor. Even in instances in which climate change creates conditions favorable to the spread of infections, diseases may be kept in check through interventions such as vector control or antibiotic treatment.

Detecting climate-change influence on an emerging human disease can be challenging. Research with animal pathogens, which in most instances is less well monitored and intervened upon than that with their human counterparts, has suggested how climate change may influence disease spread. For example, the life cycle of nematode parasites of caribou and musk oxen shortens as temperatures rise. As the Arctic has warmed, higher nematode burdens and consequently higher rates of morbidity and mortality have been observed. Other examples from animals, such as the spread of the protozoan parasite *Perkinsus marinus* in oysters, demonstrate how warming can enable range expansion of pathogens previously held in check by colder temperatures.

As these and other examples from studies of animals make clear, the influence of climate change on infectious diseases can be pronounced. The following sections deal with the infectious diseases for which research has explored the influence of climate change.

VECTOR-BORNE DISEASE

Because insects are cold-blooded, ambient temperature dictates their geographic distribution. With increases in temperatures (in particular, nighttime minimum temperatures), insects are freed to move poleward and up mountainsides. At the same time, as new areas become climatically suitable, current mosquito habitats may become unsuitable as a result of heat extremes.

In addition, insects tend to be sensitive to water availability. Mosquitoes that transmit malaria, dengue, and other infections may breed in pools of water created by heavy downpours. As has been observed in the Amazon, breeding pools can also appear during periods of drought when rivers recede and leave behind stagnant pools of water for *Anopheles* mosquitoes. These circumstances have raised interest in the potentially favorable impact of water-cycle intensification on the spread of mosquito-borne disease.

Malaria • ***TEMPERATURE*** Higher temperatures promote higher mosquito-biting rates, shorter parasite reproductive cycles, and the potential for the survival of mosquito vectors of *Plasmodium* infection in locations previously too cold to sustain them. Recent modeling experiments identify highland areas of East Africa and South America as perhaps most vulnerable to increased malarial incidence as a result of rising temperatures. In addition, a recent analysis of interannual malaria in Ecuador and Colombia has documented a greater incidence of malaria at higher altitudes in warmer years. Highland populations may be more vulnerable to malaria epidemics because they lack immunity.

Although rising temperature has the potential to expand the viable range of disease, malaria incidence is not associated with temperature in a strictly linear fashion. While mosquitoes and parasites may adapt to a warming climate, the present optimal temperature for malaria transmission is ~25°C, with a range of transmission temperatures between 16°C and 34°C. Rising temperatures also can have differential effects on parasite development during external incubation and on the mosquitoes' gonotrophic cycle. Asynchrony between these two temperature-sensitive processes has been shown to decrease the vectorial capacity of mosquitoes.[1]

PRECIPITATION The abundance of *Anopheles* mosquitoes is strongly correlated with the availability of surface water pools for mosquito breeding, and biting rates have been linked to soil moisture (a surrogate for breeding pools). Research in the East African highlands has documented that increased variance in rainfall over time has strengthened the association between precipitation and disease incidence. These disease-promoting effects of precipitation may be countered by the potential for extreme rainfall to flush mosquito larvae from breeding sites.

PROJECTIONS Climate models have begun to deliver output on regional scales, permitting projections of climate-suitable regions to assist national and local health authorities. Climate models speak to the temperature and precipitation ranges necessary for malaria transmission but do not—and cannot—account for the capacity of malaria control programs to halt the spread of disease. The global reduction in malaria distribution over the past century makes it clear that, even with climate change, malaria occurs in far fewer places today because of public health interventions.

Despite intensive efforts, malaria remains the single greatest vector-borne disease cause of morbidity and death in the world. Particularly in regions that are most affected by malaria and where the public health infrastructure is inadequate to contain it, climate modeling may provide a useful tool in determining where the disease may spread. Recent modeling studies in sub-Saharan Africa have suggested that, although East African nations may encompass regions that will become more climatically suitable for malaria over this century, West African nations may not. By 2100, temperatures in West Africa may largely exceed those optimal for malaria transmission, and the climate may become drier; in contrast, higher temperatures and changes in precipitation may allow malaria to move up the mountainsides of East African countries. Climate change may create conditions favorable to malaria in subtropical and temperate regions of the Americas, Europe, and Asia as well.

Dengue Like malaria epidemics, dengue fever epidemics depend on temperature **(Fig. 151e-5)**. Higher temperatures increase the rate of larval development and accelerate the emergence of adult *Aedes* mosquitoes. The daily temperature range may also influence dengue virus transmission, with a smaller range corresponding to a higher transmission potential. Temperatures <15°C or >36°C also substantially reduce mosquito feeding. In a *Rhesus* model of dengue, viral replication can occur in as little as 7 days with temperatures of >32–35°C; at 30°C, replication takes ≥12 days; and replication does not reliably occur at 26°C. Research on dengue in New Caledonia has shown peak transmission at ~32°C, reflecting combined effects of a shorter extrinsic incubation period, a higher feeding frequency, and more rapid development of mosquitoes. Along with temperature, peak relative humidity is a strong predictor of dengue outbreaks.

The association between dengue epidemics and precipitation is less consistent in the peer-reviewed literature, possibly because of the mosquito vector's greater reliance on domestic breeding sites than on natural pools of water. For instance, in some studies, increased access to a piped water supply has been linked to dengue epidemics, presumably because of associated increased domestic water storage. Nonetheless, several studies have established rainfall as a predictor of the seasonal timing of dengue epidemics.

The current global distribution of dengue largely overlaps the geographic spread of *Aedes* mosquitoes **(Fig. 151e-6)**. The presence of *Aedes* without dengue endemicity in large regions of North and South America and Africa illustrates the relevance of variables other than climate to disease incidence. Nevertheless, coupled climatic–epidemiologic modeling suggests dramatic shifts in the relative vectorial capacity for dengue by the end of this century should little or no mitigation of greenhouse gas emissions occur **(Fig. 151e-7)**.

Other Arbovirus Infections Climate change may favor increased geographic spread of other arboviral diseases, including Chikungunya virus disease, West Nile virus disease, and eastern equine encephalitis. Chikungunya virus disease emerged in Italy in 2007, having previously

[1] *rVc* is the vectorial capacity relative to the vector-to-human population ratio and is defined by the equation $rVc = a^2 b_h b_m e^{-\mu_m n}/\mu_m$ where a is the vector biting rate; b_h is the probability of vector-to-human transmission per bite; b_m is the probability of human-to-vector infection per bite; n is the duration of the extrinsic incubation period; and μ_m is the vector mortality rate.