Disease Emergence from Global Climate and Land Use Change

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KEYWORDS

• Climate change • Global warming • Emerging diseases
• Deforestation • Malaria • Urbanization

From the years 1906 to 2005, global average temperature has warmed by 0.74°C, and since 1961, sea level has risen on average by approximately 2 mm per year.[1] Arctic sea ice extent has declined by 7.4% per decade and snow cover and glaciers have diminished in both hemispheres. The rate of change in climate is faster now than in any period in the last 1000 years. According to the United Nations Intergovernmental Panel on Climate Change, in 90 years, average global temperatures will increase between 1.8°C and 4.0°C and sea level will rise between 18 and 59 cm (**Fig. 1**). Extremes of the hydrologic cycle (eg, floods and droughts) are also expected to accompany global warming trends.

The global rate of tropical deforestation continues at staggering levels, with nearly 2% to 3% of global forests lost each year. Land use change for agriculture represents the largest driver of land cover change across the earth. Together, croplands and pastures have become one of the largest terrestrial biomes on the planet, rivaling forest cover in extent, and occupying ~40% of the land surface.[2,3]

Emergence or resurgence of numerous infectious diseases are strongly influenced by environmental factors such as climate or land use change. The most sensitive diseases are those that are indirectly transmitted, that is, those requiring either a vehicle for transfer from host to host (eg, water- and food-borne disease) or an intermediate host or vector as part of its life cycle. Most vector-borne diseases involve arthropod vectors, such as mosquitoes, flies, ticks, or fleas. Because insects are cold blooded, a marginal change in temperature can have a potentially large biologic effect on

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Med Clin N Am 92 (2008) 1473–1491
doi:10.1016/j.mcna.2008.07.007 **medical.theclinics.com**
0025-7125/08/$ – see front matter

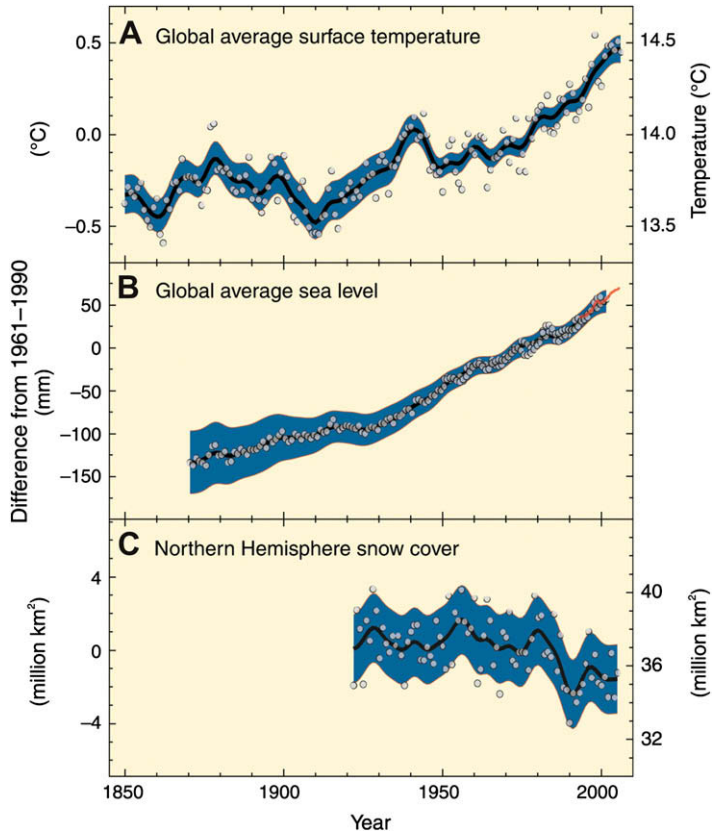

Fig. 1. Observed trends in global temperature, sea level, and extent of Northern Hemisphere snow cover. (*From* IPCC. Climate change 2007: impacts, adaptation and vulnerability: contribution of Working Group II to the Fourth Assessment Report of the IPCC. Cambridge (UK): Cambridge University Press; 2007; with permission.)

disease transmission. Therefore, climate change can alter the incidence, seasonal transmission, and geographic range of diseases such as malaria, dengue and yellow fever (mosquitoes), leishmaniasis (sand flies), Lyme disease (ticks), and onchocerciasis or "river blindness" (black flies). Schistosomiasis (involving water snails as the intermediate hosts) is also influenced by water temperature.[4]

CLIMATE EFFECTS ON WATER- AND FOOD-BORNE DISEASES

Waterborne Diseases

Heavy rainfall events can increase risk for waterborne disease outbreaks. Existing seasonal contamination of surface water in early spring in North America and Europe may explain some of the seasonality of many waterborne diseases. According to the North American chapter of the most recent Intergovernmental Panel on Climate Change (IPCC)[1] report, heavy precipitation events are expected to increase under climate change scenarios.

Preliminary analysis from the authors' research shows that overall, climate models project that these extremely heavy precipitation events will become 10%

to 40% stronger in southern Wisconsin, resulting in greater potential for the flooding and waterborne diseases that often accompany high discharge into Lake Michigan.[5]

Community water systems are already overwhelmed by extreme rainfall events. Runoff can exceed the capacity of the sewer system or treatment plants, and these systems are designed to discharge the excess wastewater directly into surface water bodies.[6,7] Urban watersheds experience more than 60% of the annual loads of all contaminants during storm events.[8] Turbidity also increases during storm events, and studies have linked turbidity and illness in many communities.[9,10]

Waterborne disease outbreaks from all causes in the United States are distinctly seasonal, clustered in key watersheds, and associated with heavy precipitation.[11] In Walkerton, Ontario, in May 2000, heavy precipitation combined with failing infrastructure contaminated drinking water with *Escherichia coli 0157:H7* and *Campylobacter jejuni*, resulting in an estimated 2300 illnesses and seven deaths.[12]

Intense rainfall can also contaminate recreational waters and increase the risk for human illness[13] through higher bacterial counts. This association is strongest at the beaches closest to rivers.[14] Involvement of the respiratory tract, ears, nose, and throat and gastrointestinal illnesses are commonly associated with recreational swimming in fresh and salt waters. High-risk groups for exposure to pathogens include frequent water users such as lifeguards or water sport enthusiasts, whereas young children, the elderly, pregnant women, and the immunocompromised have the greatest risk for suffering serious complications.[15,16] Population trends in the United States toward an older and more immunocompromised population suggest that the United States' vulnerability to waterborne pathogens will continue to increase.

Pathogens tend to co-occur with indicator bacteria but indicators are prone to false-positive readings. Indicator bacteria may survive in soil sediments or beach sand, resuspended during a precipitation event, and confound waterborne disease risk estimates.[17–19] *E coli* indicator bacteria are influenced by precipitation events up to a week before sample collection, although recent precipitation (0–3 days) tends to exhibit the strongest relationships with their numbers.[11] The spacing of rainfall events can also increase pollutant accumulation and subsequent loading into water bodies.[20,21] Longer interval processes such as the El Niño Southern Oscillation (ENSO) strongly influence interannual precipitation and therefore must be taken into account.

During periods of heavy precipitation, *Cryptosporidium parvum*, a protozoan associated with domestic livestock, can readily contaminate drinking water. The 1993 cryptosporidiosis outbreak in Milwaukee killed more than 50 people and potentially exposed more than 400,000; this epidemic coincided with unusually heavy spring rains and runoff from melting snow.[22] The authors' review of all-cause waterborne disease outbreaks in the United States over a 50-year period demonstrated a distinct seasonality, a spatial clustering in key watersheds, and a strong association with heavy precipitation.[11]

Marine organisms

In warm marine waters, *Vibrio* species proliferate. Copepods (or zooplankton), which feed on algae, can serve as reservoirs for *Vibrio cholerae* and other enteric pathogens. In Bangladesh, for example, cholera follows seasonal warming of sea surface temperature that can enhance plankton blooms.[23] After including intrinsic host immunity factors, interannual variability of cholera is strongly correlated to (1) sea surface temperatures in the Bay of Bengal; (2) ENSO; (3) the extent of flooding in Bangladesh across short time periods (<7 years), and (4) monsoon rains and Brahmaputra river discharge, for longer-period climate patterns (>7 years).[24] ENSO has had an increasing

role in explaining cholera outbreaks in recent years, perhaps because of concurrent climate change.[25]

Similarly, during the 1997 to 1998 El Niño event, winter temperatures in Lima increased more than 5°C above normal, and the number of daily admissions for diarrhea increased by more than 200% compared with expected levels based on the prior 5 years.[26] For every degree centigrade rise in air temperature above normal, an 8% increase in hospital admissions for childhood diarrhea was observed (**Fig. 2**).

Food-Borne Diseases

Some food-borne diseases are impacted by fluctuations in temperature. An estimated 30% of reported cases of salmonellosis across much of continental Europe have been attributed to warm temperatures, especially when they exceed a threshold of 6°C above average.[27] Monthly incidence of food poisoning in Britain is most strongly associated with temperatures of the previous 2 to 5 weeks.[28] Other food-borne agents, such as campylobacter, are also seasonal but are not as strongly linked to temperature fluctuations.

MALNUTRITION RISKS

Global climate change is expected to cause extremes of the hydrologic cycle (more floods and droughts). Droughts will exacerbate malnutrition, still one of the world's largest health challenges, with 800 million undernourished.[29] Climate extremes have direct impacts on food crops and can indirectly influence food supply by altering the ecology of plant pathogens, and higher soil temperatures can promote fungal growth that kills seedlings. According to the IPCC, reduced yields will occur throughout the tropics because of heat stress, and crops can be damaged from flooding, erosion, and wildfires.[1] Malnutrition increases the risk for death from infectious diseases, especially diarrhea, and micronutrient deficiencies are related to drought.

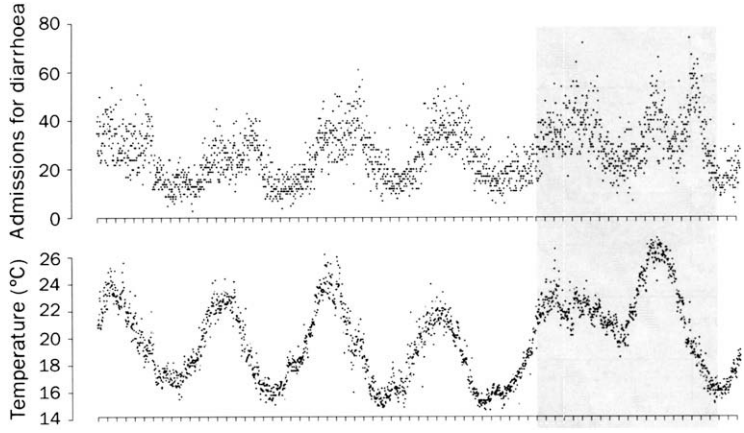

Fig. 2. Daily hospitalizations for diarrhea, by daily temperature, Lima, Peru. Top graph shows hospital admission over time for childhood diarrhea in Lima, Peru. Bottom graph is ambient temperature for Lima. Note the marked seasonality of disease incidence, and that during the winter of 1997–98, a strong El Niño (*shaded region*) made winter temperatures 5°C above normal, associated with a higher than expected incidence of diarrheal disease. For every 1 °C rise in ambient temperature, admissions increased by 8%. (*From* Checkley W, Epstein LD, Gilman RH, et al. Effect of El Nino and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children. Lancet 2000;355:442–50; with permission.)

Although malnutrition is complex in cause, one study has estimated that by the 2050s, climate change will increase the percentage at risk for hunger from a current 34% to a level of 64% to 72%, unadjusted for potential adaptive interventions.[30]

Droughts also can increase diarrhea and diseases such as scabies, conjunctivitis, and trachoma that are associated with poor hygiene and result from a breakdown in sanitation if water resources become depleted.[31]

Biofuels and Malnutrition?

The global biofuel industry is growing rapidly as rising oil prices and government mandates encourage increased production of these alternative fuels. Global biofuel production may quadruple within the next 15 to 20 years[32–34] and it has already been implicated in changing world food supplies and price.[35] Rising prices for food staples resulting from an unregulated biofuels boom could place undue burden on poor or malnourished populations in a potential scenario. According to one estimate, for every percentage increase in the real prices of staple foods, 16 million more people could become food insecure.[36] Further, the amount of humanitarian food aid available for extremely impoverished countries will be affected in the short term because food aid shipments from the United States are inversely correlated to commodity prices.[37]

Demand for crop-derived ethanol or biodiesel also could have devastating effects on the fate of the world's tropical forests. Expansion of the leading biofuel crops is already evident in South America and insular Southeast Asia as large-scale fields of soybean and oil palm, respectively, expand in these regions, leading to forest clearing, expulsion of subsistence farmers, and large emissions of carbon dioxide to the atmosphere.[38–41] The authors' center has found that most recently expanding oil palm fields have replaced forests in parts of Malaysia and Indonesia and that increases in soybean production in Brazil coincide with more forest conversion (Gibbs, unpublished data, 2008).

CLIMATE VARIABILITY AND CHANGE EFFECTS ON VECTOR-BORNE DISEASES

The transmission dynamics and geographic distribution of most insect- or rodent-borne (vector-borne) diseases are highly climate sensitive. Vector-borne pathogens spend part of their life cycle in cold-blooded arthropods that are subject to many environmental factors. Changes in weather and climate that can affect transmission of vector-borne diseases include temperature, rainfall, wind, extreme flooding or drought, and sea level rise. Rodent-borne pathogens can be affected indirectly by ecologic determinants of food sources affecting rodent population size, and floods can displace and lead them to seek food and refuge. See **Box 1**. The extrinsic incubation time of an infective agent within its vector organism is typically sensitive to changes in temperature and humidity.[42] Some examples of temperature thresholds are included in **Table 1**.

Malaria

Malaria kills between 700,000 and 2.7 million persons each year, mostly children in sub-Saharan Africa.[43] Although malaria's resurgence involves multiple factors, from climate and land use change to drug resistance, variable disease-control efforts, and other sociodemographic factors, malaria is an extremely climate-sensitive tropical disease, and one of the most important climate change/health questions to resolve.[44]

Box 1
Effects of weather and climate on vector- and rodent-borne diseases[a]

Examples of temperature effects on selected vectors and vector-borne pathogens

Vector

Survival can decrease or increase, depending on the species.

Some vectors have higher survival at higher latitudes and altitudes with higher temperatures.

Changes are possible in the susceptibility of vectors to some pathogens (eg, higher temperatures reduce the size of some vectors but reduce the activity of others).

Changes occur in the rate of vector population growth.

Changes occur in feeding rate and host contact (which may alter the survival rate).

Changes occur in the seasonality of populations.

Pathogen

Extrinsic incubation period of pathogen is decreased in vector at higher temperatures,

Changes occur in the transmission season.

Changes occur in distribution.

Viral replication is decreased.

Examples of effects of changes in precipitation on selected vector-borne pathogens

Vector

Increased rain may increase larval habitat and vector population size by creating a new habitat.

Excess rain or snow pack can eliminate habitat by flooding, thus decreasing the vector population size.

Low rainfall can create habitat by causing rivers to dry into pools (dry season malaria).

Decreased rain can increase container-breeding mosquitoes by forcing increased water storage.

Epic rainfall events can synchronize vector host seeking and virus transmission.

Increased humidity increases vector survival; decreased humidity decreases vector survival.

Pathogen

Few direct effects are evident but some data indicate humidity effects on malarial parasite development in the anopheline mosquito host.

Vertebrate host

Increased rain can increase vegetation, food availability, and population size.

Increased rain can also cause flooding and decrease population size but increase contact with humans.

Decreased rain can eliminate food and force rodents into housing areas, increasing human contact, but it can also decrease population size.

Increased sea level

Increased levels alter estuary flow and change existing salt marshes and associated mosquito species, decreasing or eliminating selected mosquito breeding sites (eg, reduced habitat for *Culiseta melanura*).

[a] The relationship between ambient weather conditions and vector ecology is complicated by the natural tendency for insect vectors to seek out the most suitable "microclimates" for their survival (eg, resting under vegetation or pit latrines during dry or hot conditions or in culverts during cold conditions).

From Gubler DJ, Reiter P, Ebi KL, Yap W, Nasci R, Patz JA. Climate variability and change in the United States: potential impacts on vector- and rodent-borne diseases. Environ Health Perspect 2001;109:223–33; with permission.

Table 1
Temperature thresholds of pathogens and vectors

Disease	Pathogen	T_{min}	T_{max}	Vector	T_{min} for Vector
Malaria	*Plasmodium falciparum*	16–19	33–39	*Anopheles*	8–10 biologic activity
Malaria	*Plasmodium vivax*	14.5–15	33–39	*Anopheles*	8–10 biologic activity
Chagas disease	*Trypanosoma cruzi*	18	38	*Triatomine* bugs	2–6 survival 20 biologic activity
Schistosomiasis	*Cercaria*	14.2	>37	Snails (*Bulinus* and others)	5 biologic activity 25±2 optimum range
Dengue fever	Dengue virus	11.9	Not known	*Aedes*	6–10
Lyme disease	*Borrelia burgdorferi*	No yet determined		*Ixodes* ticks	5–8

T_{min} is the minimum temperature required for disease transmission. T_{max} for the pathogen is the upper threshold beyond which temperatures are lethal. T_{max} for vectors are not provided. Temperatures in °C. Note: temperatures assume optimum humidity, vector survival decreases rapidly as dryness increases. Considerable variation exists in these thresholds within and among species.

Sources: Purnell, 1966; Pfluger, 1980; Molineaux, 1988; Curto de Casas and Carcavallo, 1984; Rueda et al, 1990.

Reprinted from IPCC. Climate change 2007: impacts, adaptation and vulnerability: contribution of Working Group II to the Fourth Assessment Report of the IPCC. Cambridge (UK): Cambridge University Press; 2007; with permission.

Malaria incidence varies seasonally in highly endemic areas, and malaria transmission has been associated with temperature anomalies in some African highland areas.[45] In the Punjab region of India, excessive monsoon rainfall and resultant high humidity have been recognized for years as major factors in the occurrence of malaria epidemics. Malaria epidemics have increased approximately fivefold during the year following an El Niño event[46] and recently, indices of El Niño–related climate variability predicted malaria incidence in Botswana.[47]

Special case of malaria in the African highlands

For every 1000-meter gain in elevation, temperatures decrease by 6°C. Minimum temperature for parasite development of *Plasmodium falciparum* and *Plasmodium vivax* approximates 18°C and 15°C, respectively, limiting the spread of malaria at higher altitudes. Increasing altitude also results in decreasing mosquito abundance in African highlands.[48]

A warming trend from 1950 to 2002 was documented in the East African highlands, coinciding with increases in malaria incidence.[49] Well-recognized nonlinear and threshold responses of malaria (a biologic system) are established for regional temperature fluctuations. As a biologic system, the response of mosquito populations to warming can be more than an order of magnitude larger than the measured change in temperature; just half a degree centigrade increase in temperature trend can translate into a 30% to 100% increase in mosquito abundance, demonstrating a ''biologic amplification'' of temperature effects. In the African highlands, where mosquito populations are small compared with lowland areas,[50] such biologic responses may be especially significant in determining the risk for malaria.

Arboviruses

Dengue fever

The peridomestic urban mosquito, *Aedes aegypti*, is also strongly influenced by climate, including variability in temperature, moisture, and solar radiation. Similar to the extrinsic incubation period of the malaria parasite, the rate of dengue virus replication in *A aegypti* mosquitoes increases directly with temperature in the laboratory. When linked to future climate change projections, biologic models of dengue transmission suggest that small increases in temperature, given viral introduction into a susceptible human population, could increase the potential for epidemics.[51] For small countries with presumably some climate uniformity, a climate-based *Aedes* mosquito population model strongly correlates climate conditions with the variability in dengue cases reported at the national level.[52]

West Nile virus

Climate variability has an effect on West Nile virus (WNV), a disease that rapidly spread across the Western hemisphere. Reisen and colleagues[53] found that the strain of WNV that entered New York (during the record hot July of 1999) differed from the South African strain in that it required warmer temperatures for efficient transmission. The investigators concluded that during the epidemic summers of 2002 and 2004 in the United States, epicenters of WNV were linked to above-average temperatures. Temperature influences other important components of the WNV transmission cycle, such as the development rate and fitness of immature mosquitoes and the biting rate and survival of adult female mosquitoes.[54–58] Anomalously hot summer temperatures are also linked to international WNV outbreaks in South Africa and Russia.[59–61]

Variability of precipitation may affect WNV transmission by (1) inducing a large increase in disease-transmitting mosquito abundance or (2) killing mosquito predators and competitors (3) vector–avian host contact. Multiple North American WNV vector mosquito population sizes tend to mirror the total amount of summer season precipitation.[62–65] Above-average summer precipitation likely activates new larval breeding habitats and temporarily increases the number of disease vectors, which may increase the level of WNV transmission. This mechanism appears to be behind the 1974 South African WNV epidemic, which infected more than 18,000 people.[60] In the United States, below-average rainfall the previous year tended to increase WNV transmission the following year.[66] Drought over multiple time periods has inconsistently been reported as a potential driver of multiple individual WNV outbreaks.[61,67,68] Disease-transmitting mosquito populations recover more rapidly than mosquito competitors and predators from a disturbance like drought, which may increase disease transmission.[69] In humid Florida, spring drought increases vector and avian host contact and WNV transmission, and wet summer conditions foster mosquito dispersal and subsequent disease transmission.[70]

Similar to WNV, Saint Louis encephalitis virus (SLEV) is also associated with climatic factors. In Florida, SLEV appearance in sentinel chicken flocks is preceded by a wet period followed by drought.

Chikungunya

During July 2004, amidst a severe drought in East Africa, an epidemic of chikungunya virus erupted in Lamu, Kenya, where an estimated 13,500 people (75% of the population) were infected.[71] Climate analysis showed that unseasonably warm and dry conditions, especially over coastal Kenya, occurred during May 2004.[72] Such conditions may have (1) led to unsafe domestic water storage practices and infrequent changing of water storage and (2) hastened viral development in the *Aedes* mosquito.

The virus spread to islands of the western Indian Ocean, then to India, and most recently to Italy during the summer of 2007. Although the role of climatic conditions in Italy is not clear, southern Europe was experiencing an unusually warm and dry summer.[73]

Rift valley fever

All known Rift Valley fever virus outbreaks in East Africa from 1950 to May 1998, and probably earlier, followed periods of abnormally high rainfall. Analysis of this record and Pacific and Indian Ocean sea surface temperature anomalies, coupled with satellite normalized difference vegetation index data, shows that prediction of Rift Valley fever outbreaks may be made up to 5 months in advance of outbreaks in East Africa. Concurrent near–real-time monitoring with satellite normalized difference vegetation data may identify actual affected areas.[74]

Dams and irrigation can increase breeding sites, exacerbating the effect of extreme rainfall. Extensive human disease outbreaks were not reported until 1951, when an estimated 20,000 persons were infected during an epidemic in cattle and sheep in South Africa. Outbreaks were reported exclusively from sub-Saharan Africa until 1977–78, when 18,000 persons were infected and 598 deaths were reported in Egypt.[75]

Lyme Disease

Lyme disease is a prevalent, tick-borne disease in North America that new evidence suggests has an association with temperature[76] and precipitation.[77] In the field, temperature and vapor pressure contribute to maintaining populations of the tick *Ixodes scapularis* which, in the United States, is the microorganism's secondary host. A monthly average minimum temperature above -7°C is required for tick survival.[78]

The northern boundary of tick-borne Lyme disease is limited by cold temperature effects on the tick, *I scapularis*. The northern range limit for this tick could shift north by 200 km by the 2020s, and 1000 km by the 2080s (based on projections from the CGCM2 and HadCM3 atmosphere-ocean global circulation models under *the Special Report on Emissions Scenarios* A2 emissions scenario).[79]

Rodent-Borne Diseases

For hantavirus pulmonary syndrome, which newly emerged in the Southwest in 1993, weather conditions led to a growth in rodent populations and subsequent disease transmission, all following unusually heavy El Niño–driven rainfall.[80] Hantavirus infections are transmitted largely by exposure to infectious excreta, and may cause serious disease in humans and a high fatality rate.

Extreme flooding or hurricanes can lead to outbreaks of leptospirosis. In Nicaragua, for example, an epidemic of leptospirosis followed heavy flooding in 1995. From a case-control study, a 15-fold risk for disease was associated with walking through flooded waters.[81]

Plague is another climate-sensitive disease that is carried by fleas, and it is associated with populations of rodents, the primary reservoir hosts of the *Yersinia pestis* bacterium. In the desert southwestern United States, plague bacterial levels in rodents have been found to increase in the wake of wet climate conditions following El Niño and Pacific Decadal Oscillation–driven wet weather conditions.[82] Historically, according to tree-ring proxy climate data, during the major plague epidemics of the Black Death period (1280–1350), climate conditions were becoming warmer and wetter.[83]

Other Vector-Borne Diseases and Climate

Bluetongue

Bluetongue disease, a viral illness that is fatal to sheep and other ruminants, is spread by *Culicoides* spp (midges) and historically, only rarely reached north into Europe. But since 1998, several strains of bluetongue virus have advanced 800 km further into Europe than previously reported. Warming temperatures in the region have allowed enhanced survival of viruses through winter and a northern expansion of the insect vector of the disease.[84] Warmer winter temperatures projected for the future may further the geographic range of this serious livestock disease; the warm temperatures of 2007 already have allowed establishment of bluetongue in Northern Europe (**Fig. 3**).[85]

LAND USE CHANGE AND DISEASE EMERGENCE

Disturbance of habitats due to land cover change is likely the largest environmental cause of altered risk for infectious diseases. Habitat change, in turn, may affect the breeding sites of disease vectors or the biodiversity of vectors or reservoir hosts. Major drivers of land use change include agricultural development or water projects, urbanization and sprawl, and deforestation. These changes, in turn, cause a cascade of factors that exacerbate infectious disease emergence, such as forest fragmentation, pathogen introduction, pollution, poverty, and human migration. These issues are important but complex and are only understood for a few diseases. For example, recent research has shown that forest fragmentation, urban sprawl, and loss of biodiversity are linked to increased Lyme disease risk in the northeastern United States.[86]

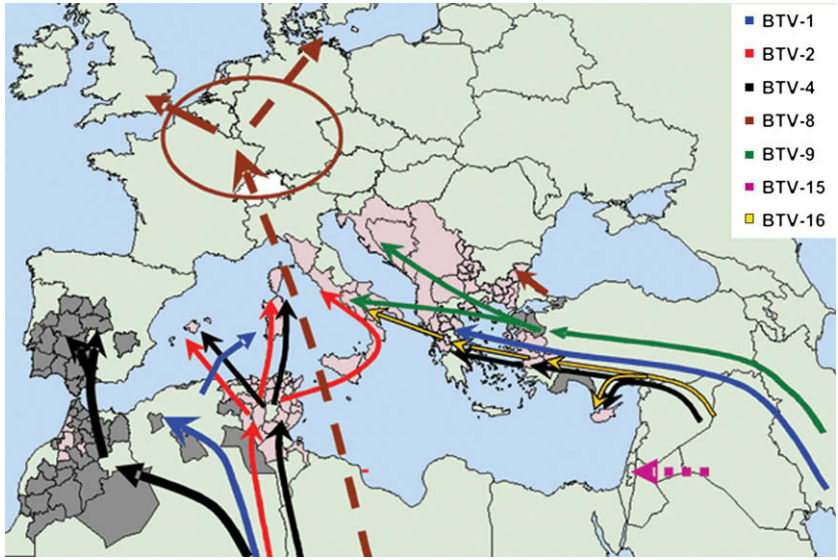

Fig. 3. Map of bluetongue virus (BTV) across the European Union. The molecular epidemiology of BTV since 1998: routes of introduction of different serotypes and individual virus strains. The presence of BTV-specific neutralizing antibodies in animals in Bulgaria is shown, but the presence of BTV serotype 8 cannot yet be confirmed. (*From* Saegerman C, Berkvens D, Mellor PS. Bluetongue epidemiology in the European Union. Emerg Infect Dis 2008;14:539–44; with permission.)

Agricultural Development

Land use change for agricultural expansion represents the largest driver of land cover change across the earth, and has continued to be the dominant cause of tropical deforestation well into this decade.[87] Together, croplands and pastures have become one of the largest terrestrial biomes on the planet, rivaling forest cover in extent, and occupying ~40% of the land surface.[2,3] The area of cultivated land is expected to increase dramatically across the tropics because of unprecedented increases in global demand for food, feed, and fuel. Indeed, estimates suggest that agricultural land area in developing countries may increase considerably (25%) to meet this demand.[88] Already, agriculture uses over two thirds of the world's fresh water.[89] Agricultural development in many parts of the world has resulted in an increased requirement for crop irrigation, which reduces water availability for other uses and increases breeding sites for disease vectors. An increase in soil moisture associated with irrigation development in the southern Nile Delta following the construction of the Aswan High Dam has caused a rapid rise in the mosquito, *Culex pipiens*, and a consequential increase in the arthropod-borne disease, Bancroftian filariasis.[90,91] Onchocerciasis and trypanosomiasis are further examples of vector-borne parasitic diseases that may be triggered by changing land-use and water-management patterns. In addition, large-scale use of pesticides has had deleterious effects on farm workers, including hormone disruption and immune suppression.[92]

Urbanization and Urban Sprawl

On a global basis, the proportion of people living in urban centers will increase to an unprecedented 65% by the year 2030.[93] The 2000 census shows that 80% of the American population now lives in metropolitan areas, with 30% in cities of 5 million or more. The environmental issues posed by such large population centers have profound impacts on public health beyond the city limits.[94]

Alterations of ecosystems and natural resources contribute to the emergence and spread of infectious disease agents. Human encroachment of wildlife habitats has broadened the interface between wildlife and humans, resulting in increased opportunities for the emergence of novel infectious diseases in wildlife and their transmission to people. Rabies is an example of a zoonotic pathogen carried by animals that has become habituated to urban environments. Bats colonize buildings, skunks and raccoons scavenge human refuse, and in many countries, feral dogs in the streets are common and the major source of human infection.[95]

Periurban slums provide the ideal conditions for major epidemics, combining dense housing, poor sanitation and shelter, and open breeding sites for vectors that can transmit pathogens. Peridomestic dengue fever, infecting an estimated 50 million people each year, is one ramification of such poor urban conditions.

Deforestation

Rates of deforestation have grown explosively since the beginning of the twentieth century. Driven by local to global demand for agricultural and forest products and expanding human population centers, large swaths of species-rich tropical and temperate forests, and prairies, grasslands, and wetlands, have been converted to species-poor agricultural and ranching areas. The global rate of tropical deforestation is continuing at staggering levels well into this decade, with more than 2.3% of humid tropical forests cleared between 2000 and 2005 alone.[87] Parallel to this habitat destruction is an exponential growth in human–wildlife interaction and conflict, which has resulted in exposure to new pathogens for humans, livestock, and wildlife.[96]

Case study: malaria and deforestation

Land cover change can significantly affect local climate more acutely than long-term global warming. Surface change can influence microclimatic conditions including temperature, evapotranspiration, and runoff,[97] all potentially important determinants of mosquito abundance and survivorship. In Kenya, open, treeless habitats average warmer midday temperatures than forested habitats, and also affect indoor hut temperatures.[98] Subsequently, the gonadotropic cycle of female *Anopheles gambiae* was shortened by 2.6 days (52%) and 2.9 days (21%) during the dry and rainy seasons, respectively, compared with that of forested sites. In Uganda, similarly higher temperatures have been measured in communities bordering cultivated fields compared with those adjacent to natural wetlands, and the number of *A gambiae s.l.* per house increased along with minimum temperatures after adjustment for potential confounding variables.[99]

In aquatic breeding sites found in farmlands, higher maximum and mean temperatures also hasten larval development and pupation rates.[100] In western Kenya, heavier canopy cover decreases the abundance of *A gambiae* complex and *Anopheles funestus* larvae in natural aquatic habitats.[50] In artificial pools, survivorship of *A gambiae* larvae in sunlit open areas was 50 times the survivorship in forested areas and also related to assemblages of predatory species.[101] In short, deforestation and cultivation of natural swamps in the African highlands create conditions favorable for the survival of *A gambiae* larvae, making analysis of land use change on local climate, habitat, and biodiversity key to malaria risk assessments.

In the Amazon Basin, deforestation has also altered the risk for malaria. Vittor and colleagues[102] have found a strong association between the biting rates of *Anopheles darlingi* and the extent of deforestation in the Amazon. Controlling for human population density, the biting rates of *A darlingi* were more than 200-fold higher in sites experiencing greater than 80% deforestation versus those with less than 30% deforested landscape (**Fig. 4**).

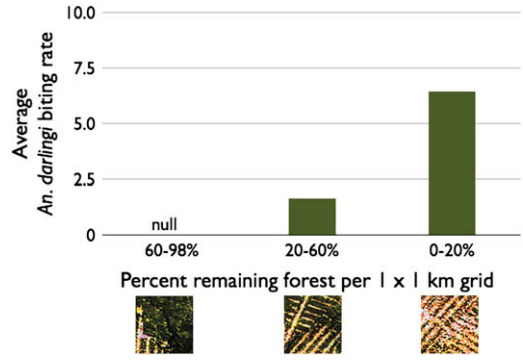

Fig. 4. Deforestation and *A darlingi* biting rates, Peruvian Amazon. Average human biting rates of the Anopheles darlingi mosquito according to the percentage of forest within a 1 × 1 km pixel. Biting rates rise dramatically with deforestation, even after controlling for human population density. Mean human-biting rate if determined per 6 hours per person; 15 or 16 collection nights per site during 1 year (total: 888 6-hour nights). (*Data from* Vittor AY, Gilman RH, Tielsch J, et al. The effect of deforestation on the human-biting rate of Anopheles Darlingi, the primary vector of falciparum malaria in the Peruvian Amazon. Am J Trop Med Hyg 2006;74:3–11.)

Host immunity against malaria can be affected indirectly by another form of land use change. Gold mining is an extractive industry that damages local and regional environments and has adverse human health effects because mercury is used to extract gold from riverbeds in the tropical forests. Not only does mercury accumulate in local fish populations, making them toxic to eat,[103,104] but mercury also suppresses the human immune system. In gold-mining areas, more mosquito-breeding sites and increased malaria risk result from digging gem pits in the forest and the craters resulting from logging; broader disease spread occurs as populations disperse throughout the region.[105]

SUMMARY

Climate change and land use change can affect multiple infectious diseases of humans, acting either independently or synergistically. Although in isolated cases, disease resurgence has been attributed to recent warming trends, some of the long-term and complex problems posed by climate change may not be readily discernible from other causal factors. Expanded efforts, therefore, in empiric and future scenario-based risk assessment are required to anticipate these problems. Moreover, the many health impacts of climate and land use change must be examined in the context of the myriad other environmental and behavioral determinants of disease.

Health risks are but one of many sectors expected to be affected by climate and ecologic change and represent the interconnected context in which decision makers must implement strategies. To optimize prevention capabilities, upstream environmental approaches must be part of any intervention, rather than assaults on single agents of disease. Clinicians must develop stronger ties, not only to public health officials and scientists, but also to earth and environmental scientists and policy makers. Without such efforts, we risk practicing medicine in an unsustainable manner and will inevitably benefit our current generation at the cost of generations to come.

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