

The risks of climate change from infectious diseases

Abstract

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

Keywords: climate change, heat-related mortality, infectious diseases, morbidity, world health organization

Volume 1 Issue 5 - 2017

Abd El-Aleem Saad Soliman Desoky

Department of Plant protection (Agriculture Zoology), Sohag University, Egypt

Correspondence: Abd El-Aleem Saad Soliman Desoky, Department of Plant protection (Agriculture Zoology), Sohag University, Egypt, Email abdelaalem2011@yahoo.com

Received: October 15, 2017 | **Published:** November 30, 2017

Introduction

Global climate change is expected to have broad health impacts. These could occur through various exposure pathways, such as the frequency or intensity of extreme heat waves, floods, and droughts. Warmer air temperatures could also influence local and regional air pollutants and aeroallergens. Less direct health impacts may result from climate-related alteration of ecosystems or water and food supplies, which in turn could affect infectious disease incidence and nutritional status. Finally, sea level rise could potentially lead to massive population displacement and economic disruption. Changes in temperature, humidity, rainfall, and sea level rise could all affect the incidence of infectious diseases. Mosquitoes, ticks, and fleas are cold-blooded and thus sensitive to subtle temperature and humidity changes. But vector-borne diseases are also dependent on many other interacting factors. Although there has been a resurgence of infectious diseases in recent years, it is unclear that climate change has played a significant role. Other factors such as the movement of human and animal populations, the breakdown in public health infrastructure, changes in land use, and the emergence of drug resistance have been contributory. The transmission of infectious diseases is strongly influenced by temperature, humidity, and rainfall. The distribution and seasonality of important infectious diseases are likely to be affected by climate change. Diseases transmitted by insect or rodent vectors have life cycles where much time is spent outside the human host, and therefore are more influenced by ambient conditions. There is a limited range of climatic conditions within which each such infective or vector species can survive and reproduce.

Mosquito-borne virus transmission is largely governed by vector population abundance, vector host-seeking behavior, and the dissemination of the virus through the vector's body to the salivary glands. The environment can be thought of as contextually providing suitable conditions for the persistence of infectious disease agents or more directly driving the variability of vector and host populations and interactions. Environmentally mediated mechanisms explaining

vector and host dynamics focus on nonlinear changes to time before an infectious mosquito can retransmit a virus or extrinsic incubation period, vector population explosions, or changing host-seeking behavior.¹⁻³

Infectious diseases

Water- and food-borne diseases

Water shortages, as mentioned above, contribute to diarrhoeal disease through poor hygiene, especially in poor countries. On the other hand, flooding can contaminate drinking water with run-off from sewage lines, containment lagoons (such as at animal-feeding operations), or conventional (non-point-source) pollution from across watersheds. The parasites in the genus *Cryptosporidium* are usually associated with domestic livestock but can contaminate water intended for human consumption, especially during periods of heavy precipitation. In 1993 a cryptosporidiosis outbreak in Milwaukee, which killed more than 50 people and potentially exposed over 400,000 more to *Cryptosporidium*, coincided with unusually heavy spring rains and run-off from melting snow.⁴ A review of outbreaks of any water-borne disease in the U.S.A. over a 50-year period demonstrated a distinct seasonality, a spatial clustering in the key watersheds, and a strong association with heavy precipitation.⁵ Certain food-borne diseases are also affected by fluctuations in temperature. Across much of continental Europe, for example, an estimated 30percent of reported cases of salmonellosis occur when air temperatures are 6°C above the mean.^{6,7} In the U.K., the monthly incidence of food poisoning is strongly correlated with air temperatures in the previous 2-5weeks.⁸

Coastal waters

One type of phytoplankton, the din flagellates, thrives in warm waters with adequate nitrogen, and they are the primary component of toxic "red tides." They can cause acute paralytic, diarrhetic, and amnesiac poisoning in humans, as well as extensive die-offs of fish and shellfish and the marine mammals and birds that depend on the

marine food-web. The frequency and global distribution of toxic algal incidents and the incidence of human intoxication from algal sources appear to be increasing.⁹ *Vibrio* species also proliferate in warm marine waters. Zooplankton that feed on algae can serve as reservoirs for *Vibrio cholerae* and other enteric pathogens of humans. In Bangladesh, cholera follows the seasonal increase in sea-surface temperatures that can enhance plankton blooms.¹⁰

During the El Niño event in 1997-1998, winter temperatures in Lima increased to >5°C above normal, and the number of daily admissions for diarrhea rose to levels that were twice as high as recorded, over the same months, in the previous 5 years.¹¹ Although long-term studies of the El Niño Southern Oscillation (ENSO) have shown a consistent association with cholera and other diarrhoeal diseases, the oscillation appears to have played an increasing role in cholera outbreaks in recent years, perhaps because of concurrent climate change.¹² A detailed understanding of the inter-annual cycles of cholera and other infectious diseases, however, requires the combined analyses of both environmental exposure and the host's intrinsic immunity to a disease. When they considered these factors together,¹³ found that the inter-annual variability seen in cholera in Bangladesh was strongly correlated, across periods of <7 years, with sea-surface temperatures in the Bay of Bengal, ENSO and the extent of flooding in Bangladesh, and, across longer periods, with the monsoon rains and the discharge of the Brahmaputra river.

Vector-borne diseases: As the human pathogens transmitted indirectly by insect or rodent vectors spend considerable time outside of their vertebrate hosts, they may easily be affected by environmental conditions. The range of suitable climatic conditions within which each vector-borne pathogen and its vector can survive and reproduce is limited. The incubation time of a vector-borne infective agent within its vector is typically very sensitive to changes in temperature and humidity.¹⁴

Malaria: Between 700,000 and 2.7 million people—mostly children in sub-Saharan Africa—die each year of malaria (www.cdc.gov/malaria), and, thanks to climate and land-use change, drug resistance, ineffective control efforts, and various socio-demographic factors, there is no evidence that malaria-attributable mortality is falling. Malaria is an extremely climate-sensitive tropical disease, making the assessment of the potential change in malarial risk, caused by past or projected global warming, one of the most important topics in the field of climate change and health.¹⁵ The incidence of malaria varies seasonally in highly endemic areas, and malaria transmission has been associated with temperature anomalies in some African highlands.¹⁶ In the Punjab region of India, excessive monsoon rainfall and the resultant high humidity have been recognized for years as major factors in the occurrence of malaria epidemics. More recently in the region, the frequency of malaria epidemics was observed to increase approximately five-fold during the year following an El Niño event.¹⁷ In Botswana,¹⁸ recently showed that indices of El Niño-related climate variability can serve as the basis of malaria-risk prediction and early warning.

Highland malaria: Air temperatures decrease by a mean of 6°C for every 1,000m gained in elevation. In areas where human malaria is endemic, this effect usually precludes the transmission of malarial parasites at high altitudes, partly because the parasites cannot produce sporozoites in mosquitoes living at low temperatures. The minimum temperatures for the sporogony of *Plasmodium falciparum* and *P. vivax*, for example, are approximately 18°C and 15°C, respectively.

As seen in the African highlands Bodker, mosquito abundance tends to decrease with increasing altitude. Global warming is likely to result in an increase in the altitudes at which no malaria transmission occurs. In Africa,¹⁹ estimated that the risk of exposure to malaria, measured in person months, will be 16 percent-28 percent higher in 2100 than at present. Having compared climate suitability maps for malaria in the topographically diverse country of Zimbabwe,²⁰ concluded that the warming predicted from global-climate models could make the country's entire highland area climatologically suitable for malarial transmission by 2050.

The highland areas of Africa that is not currently endemic for malaria but are, as the result of global warming, at high risk of becoming areas where transmission occurs²¹ recently reported that the East African highlands had generally become warmer since 1950, over a period in which malaria incidence had also increased. There are well-recognized non-linear and threshold responses of malaria to the effect of regional temperature changes. In a form of biological 'amplification,' the response of mosquito populations to warming can be more than an order of magnitude larger than the measured change in temperature, an increase of just 0.5°C translating into a 30 percent-100 percent increase in mosquito abundance.²¹ In the African highlands, where mosquito populations are relatively small compared with those in lowland areas,²² such biological responses may be especially significant in determining the risk of malaria.

Malaria and local effects on climate from land-use change

Changing landscapes can significantly affect local climate more acutely than long-term global warming. Land-cover change, for example, can influence the micro-climatic conditions, including temperature, evapo-transpiration and surface run-off,²³ that are key to determining mosquito abundance and survivorship. In Kenya,²⁴ observed that open treeless habitats had warmer mean midday temperatures than forested habitats, and that deforestation also affected indoor hut temperatures. As a result, the gonotrophic cycle of female *Anopheles gambiae* s.l. during the dry and rainy seasons was found to be 2.6 days (52 percent) and 2.9 days (21 percent) shorter, respectively, in the deforested sites than in the forested. Similar findings have been documented in Uganda, where temperatures in communities bordering cultivated fields have been found higher than those in communities adjacent to natural wetlands, and the number of *An. Gambiae* s.l./ house has been found to increase with increasing minimum temperature, after adjustment for potentially confounding variables Lindblade. In Kenya, mosquito breeding sites in farmland have been found to be relatively warm and this warmth speeds up the development of the immature insects.²⁵ Increased canopy cover in western Kenya is negatively associated with the presence of larval *An. Gambiae* s.l. and *An. Funestus* in natural aquatic habitats.²⁶ In artificial pools, survivorship of the larvae of *An. Gambiae* s.s. in sunlit open areas was 50-fold higher than that in forested areas, and also related to assemblages of predatory species.²⁶

In short, deforestation and cultivation of natural swamps in the African highlands creates conditions favorable for the survival of *An. Gambiae* larvae, making an analysis of the effects of land-use change on local climate, habitat, and biodiversity key to any malaria-risk assessments. Deforestation has also affected malaria in other regions, such as the Amazon basin Guerra.²⁷ found a strong association between the biting rates of *An. Darlingi* and the extent of deforestation in the Amazon; after controlling for the variation in human population

densities, the biting rates of *An. Darlingi* were still >200-fold higher in sites experiencing >80 percent deforestation than in sites with <30 percent deforestation. Human activities have the capacity to shift the biodiversity of local ecosystems rapidly, intentionally and unintentionally increasing or decreasing malarial risk factors by altering the environment and mosquito habitat. The direction of the trend depends heavily on the *Anopheles* species present and on local conditions.²⁸ In north-eastern India, expansive deforestation has caused the numbers of *An. Dirus* and *An. Culicifacies* to decline.²⁹ The effects of changing land-use patterns on the regulation of malaria (or other infectious disease) across a large area are species and site-specific, and therefore cannot be generalized.

Arboviruses

Although *Aedes aegypti* is known to be strongly affected by ecological and human 'drivers' in urban settings, this species is also influenced by climate, including variability in temperature, moisture and solar radiation. Similar to the extrinsic incubation periods of malarial parasites, the rate of replication of dengue virus in *Ae. Aegypti* increases directly with air temperature, at least in the laboratory. Biological models have been developed to explore the influence of projected temperature change on the incidence of dengue fever. These models indicate that, given viral introduction into a susceptible human population, relatively small increases in temperature could significantly increase the potential for epidemics of dengue.³⁰ In addition, for relatively small countries with presumably some climate uniformity, a climate-based dengue model has been developed that strongly correlates with the inter-annual variability seen in the incidence of dengue reported at the national level.³¹

Certain other arboviruses, such as Saint Louis encephalitis virus (SLEV), are also associated with climatic factors. In Florida, the appearance of SLEV in sentinel chicken flocks is preceded by a wet period followed by drought.³² It has been suggested that spring drought forces the mosquito vector, *Culex nigripalpus*, to converge with immature and adult wild birds in restrictive, densely vegetated, hammock habitats. This forced interaction of mosquito vectors and avian hosts then creates an ideal setting for rapid transmission and amplification of SLEV. Once the drought ends and water sources are restored, the infected vectors and hosts disperse and transmit SLEV to a much broader geographical area.³² Climate variability may also have an effect on West Nile virus (WNV), a pathogen only recently introduced into the New World.³ found that the strain of WNV that entered New York, during the record hot July of 1999, differed from the South African strain in that it required warmer temperatures for efficient transmission. It seems likely that, during the epidemic summers of 2002-2004 in the U.S.A., epicenters of WNV were linked to above-average temperatures.

Rodent-borne diseases

Hantavirus is transmitted to humans largely by exposure to infectious rodent excreta, and may then cause serious disease, with a high level of mortality. In the emergence of hantavirus pulmonary syndrome in the southwestern U.S.A., in 1993, it was the weather conditions, especially El Nino-driven heavy rainfall, that appear to have led to a growth in rodent populations and subsequent viral transmission.³³ Extreme flooding or hurricanes can lead to outbreaks of leptospirosis. In 1995, an epidemic of this disease occurred in Nicaragua after heavy flooding, and a major risk factor for the disease was found to be walking through the flood waters.³⁴

Attribution of disease burden resulting from climate change

The World Health Organization (WHO) has examined the global burden of disease already attributable to anthropogenic climate change up to the year 2000 and made model-based forecasts of the health risks from global climate change up to the year 2030.⁷ Conservative assumptions were made about climate-health relationships (e.g., that socio-economic conditions would prevent a climate-driven spread of vector-borne disease from endemic tropical regions to temperate regions) and many plausible health impacts were excluded for lack of quantitative models. The results indicate that the current burden from climate-sensitive diseases such as diarrhea, malaria and malnutrition is so large that even the subtle climatic changes that have occurred since the mid-1970s could already be causing > 150,000 deaths and approximately 5million disability-adjusted life-years (DALY) each year. Although climate change is a global threat to public health, the WHO's assessment also revealed that the poorer regions of the world may be the most vulnerable. When the WHO's estimates of morbidity and mortality caused by human-induced climate change were extrapolated to 2030, it was found that the climate-change-induced excess risk of the various health outcomes considered could more than double by that year.^{35,36}

Conclusion

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

The regions with the greatest burden of climate-sensitive diseases are often the regions with the lowest capacity to adapt to the new risks. Many of the regions most vulnerable to climate change are also those least responsible for causing the problem. Africa, for example, is thought to harbor about 70percent of all malaria cases but has the lowest per-capita emissions of the 'greenhouse' gases that cause global warming. In today's globalized world, with its international trade and travel, an increase in disease anywhere on the globe can affect every country. Health is just one of the many sectors expected to be affected by climate change. It represents just a part of the interconnected context in which decision makers must implement strategies to prevent or reduce the adverse effects of such change. To achieve the greatest disease prevention, "upstream" environmental approaches, rather than assaults on single agents of disease, must form part of any intervention. If the truly global public-health challenge of climate change is to be adequately addressed, an unprecedented co-operation between natural and social/health scientists, as well as between rich and poor countries, must occur.

Acknowledgements

None.

Conflict of interest

Author declares that there is no conflict of interest.

References

- Jupp PG, Blackburn NK, Thompson DL, et al. Sindbis and West Nile virus infections in the Witwatersrand–Pretoria region. *South African Medical Journal*. 1986;70(4):218–220.
- Kilpatrick AM, Kramer LD, Jones MJ, et al. West Nile virus epidemics in North America are driven by shifts in mosquito feeding behavior. *PLoS Biology*. 2006;4(4):e82.
- Reisen WK, Fang Y, Martinez VM. Effects of temperature on the transmission of West Nile virus by *Culex tarsalis* (Diptera: Culicidae). *J Med Entomol*. 2006;43(2):309–317.
- Mac Kenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. *N Engl J Med*. 1994;331(3):161–167.
- Curriero F, Patz J, Rose J, et al. The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948–1994. *Am J Public Health*. 2001;91(8):1194–1199.
- Kovats RS, Edwards SJ, Hajat S, et al. The effect of temperature on food poisoning: a time–series analysis of salmonellosis in ten European countries. *Epidemiol Infect*. 2004;132(3):443–453.
- McMichael AJ, Campbell–Lendrum DH, Kovats S, et al. Global climate change. In *Comparative quantification of health risks: global and regional burden of disease due to selected major risk factors*. In: M Ezzati, AD Lopez, et al. editors. Geneva, World Health Organization, Switzerland; 2004. p. 1543–1649.
- Bentham G, Langford IH. Climate change and the incidence of food poisoning in England and Wales. *International Journal of Biometeorology*. 1995;39(2):81–86.
- Van Dolah FM. Marine algal toxins: origins, health effects, and their increased occurrence. *Environ Health Perspect*. 2000;108(Suppl 1):133–141.
- Colwell RR. Global climate and infectious disease: the cholera paradigm. *Science*. 1996;74(5295):2025–2031.
- Checkley W, Epstein L, Oilman R, et al. Effects of El Niño and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children. *Lancet*. 2000;355(9202):442–450.
- Rodo X, Pascual M, Fuchs G, et al. ENSO and cholera: a non–stationary link related to climate change? *Proceedings of the National Academy of Sciences*. 2002;99(20):12901–12906.
- Koelle K, Rodó X, Pascual M, et al. Refractory periods and climate forcing in cholera dynamics. *Nature*. 2005;436(7051):696–700.
- Gubler DJ, Reiter P, Ebi KL, et al. Climate variability and change in the United States: potential impacts on vector– and rodent–borne diseases. *Environ Health Perspect*. 2001;109(Suppl 2):223–233.
- Patz JA, Campbell–Lendrum D, Holloway T, et al. Impact of regional climate change on human health. *Nature*. 2005;438(7066): 310–317.
- Zhou G, Minakawa N, Githeko AK, et al. Climate variability and malaria epidemics in the highlands of East Africa. *Trends Parasitol*. 2005;21(2):54–56.
- Bouma MJ, HJ van der Kaay. The El Niño Southern Oscillation and the historic malaria epidemics on the Indian subcontinent and Sri Lanka: an early warning system for future epidemics? *Tropical Medicine and International Health*. 1996;1(1):86–96.
- Thomson MC, Doblas–Reyes FJ, Mason SJ, et al. Malaria early warnings based on seasonal climate forecasts from multimodel ensembles. *Nature*. 2006;439(7076):576–579.
- Tanser FC, Sharp B, le Sueur D. Potential effect of climate change on malaria transmission in Africa. *Lancet*. 2003;362(9398):1792–1798.
- Ebi KL, Hartman J, Chan N, et al. Climate suitability for stable malaria transmission in Zimbabwe under different climate change scenarios. *Climatic Change*. 2005;73(3):375–393.
- Pascual M, Ahumada JA, Chaves LF, et al. Malaria resurgence in East African highlands: temperature trends revisited. *Proc Natl Acad Sci USA*. 2006;103(15):5829–5834.
- Foley JA, Defries R, Asner GP, et al. Global consequences of land use. *Science*. 2005;309(5734):570–574.
- Minakawa N, Sonye G, Mogi M, et al. The effects of climatic factors on the distribution and abundance of malaria vectors in Kenya. *J Med Entomol*. 2002;39(6):833–841.
- Afrane YA, Lawson BW, Githeko AK, et al. Effects of microclimatic changes caused by land use and land cover on duration of gonotrophic cycles of *Anopheles gambiae* (Diptera: Culicidae) in western Kenya highlands. *J Med Entomol*. 2005;42(6): 974–980.
- Munga S, Minakawa N, Zhou G, et al. Association between land cover and habitat productivity of malaria vectors in western Kenyan highlands. *Am J Trop Med Hyg*. 2006;74(1):69–75.
- Tuno N, Okeka W, Minakawa N, et al. Survivorship of *Anopheles gambiae sensu stricto* (Diptera: Culicidae) larvae in western Kenya highland forest. *Journal of Medical Entomology*. 2005;42(3):270–277.
- 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(1):3–11.
- Guerra CA, Snow RW, Hay SI. A global assessment of closed forests, deforestation and malaria risk. *Ann Trop Med Parasitol*. 2006;100(3):189–204.
- Dev V, Bhattacharyya PC, Talukdar R. Transmission of malaria and its control in the northeastern region of India. *Journal of the Association of Physicians of India*. 2003;51:1073–1076.
- Patz JA, Martens WJ, Focks DA, et al. Dengue fever epidemic potential as projected by general circulation models of global climate change. *Environ Health Perspect*. 1998;106(3):147–153.
- Hopp MJ, Foley JA. Worldwide fluctuations in dengue fever cases related to climate variability. *Climate Research*. 2003;25(1):85–94.
- Jeffrey Shaman, Marc Stieglitz, Colin Stark, et al. Using a dynamic hydrology model to predict mosquito abundances in flood and swamp water. *Emerging Infectious Diseases*. 2002;8(1):6–13.
- Glass GE, Cheek JE, Patz JA, et al. Using remotely sensed data to identify areas at risk for hantavirus pulmonary syndrome. *Emerg Infect Dis*. 2000;6(3):238–247.
- Trejevo RT, Rigau–Pérez JG, Ashford DA, et al. Epidemic leptospirosis associated with pulmonary hemorrhage–Nicaragua, 1995. *J Infect Dis*. 1998;178(5):1457–1463.
- Bødker R, Akida J, Shayo D, et al. Relationship between altitude and intensity of malaria transmission in the Usambara Mountains, Tanzania. *J Med Entomol*. 2003;40(5):706–717.
- Lindblade KA, Walker ED, Onapa AW, et al. Land use change alters malaria transmission parameters by modifying temperature in a highland area of Uganda. *Trop Med Int Health*. 2000;5(4):263–274.