CHAPTER 28

Climate, Ecology, and Infectious Human Disease

James L. A. Webb

28.1 Introduction

Climate has had a profound influence on evolving patterns of human disease. From the early eras of human history to the present, climate forces have been determinative in establishing the ecological parameters within which human beings and the pathogens that afflict us have coexisted. As early human societies became more complex, population densities increased, and networks of exchange thickened, possibilities for the transmission of pathogens broadened. Over the past few millennia, previously discrete zones of disease transmission became integrated, with devastating demographic consequences.

Shifts in climate phases—between eras of warming and cooling or between eras of increasing or diminishing precipitation—have had significant impacts on human communities. At some times and places, climate shifts have provoked transformations in patterns of land use and thus the environments for animal and insect vectors that could transmit disease. At other times and places, climate change has provoked transformations in regional balances of political power. Some of these changes, in turn, have forced migrants into new environments and exposed them to diseases and nutritional stresses that have compromised their health.

At shorter timescales, extreme seasons and unique weather events have disrupted agriculture and created food shortages that promoted the transmission of disease. Floods, earthquakes, volcanic explosions, droughts, and unseasonal freezes have wreaked havoc on human communities. These threats remain of great concern, even as over the past century or two human beings have developed technologies and medicines that are able to limit or mitigate some of the

J. L. A. Webb (✉)
Colby College, Waterville, ME, USA

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consequences of disease transmission. The long-term result of these achievements is that human beings in many areas of the world—even in an era of anthropogenic global warming—are now less susceptible to infectious disease than at any earlier point in human history, and this trend toward greater security is likely to continue. This remains true even as newly emerging and reemerging disease threats attract the attention of researchers trying to estimate the future health impacts of climate change.

This brief chapter presents a synthetic overview of the relationships between climate, ecology, and human disease over time. It draws upon research in diverse fields, including historical climatology, epidemiology, ecology, and biomedicine. It emphasizes that our biomedical and ecological understandings of disease processes and the widespread use of effective medicines and vaccines have substantially changed the nature of the threats from infectious disease in many areas of the world. This historical contextualization is important to consider when evaluating future disease scenarios.

28.2 Climate Forces and the Ecological Parameters of Disease History

Over the immensely long eras during which our ancestors walked the earth, the forces of climate shaped and reformed the natural world. Over the roughly 200,000 years of the human past, geophysical processes created eras with starkly different temperature zones and levels of carbon dioxide; shifted patterns of global distributions of flora and fauna; dramatically raised and lowered the level of the oceans; and lavished or scanted the freshwater resources upon which our ancestors depended. Climate change has successively configured and reconfigured the earth's ecological zones as all forms of life have continued to evolve, including the pathogens that cause human illness and death.

Research in the genetic and molecular sciences has shown that humans and our hominin ancestors were afflicted with infectious diseases from the very earliest times, and that humans continue to suffer from some of these infections to the present day. The long chains of infections are sometimes referred to as heirloom diseases, either because they have been passed down from one generation to the next (as in the case of various herpes viruses) or because transmission was possible between primates and humans (as in some forms of hepatitis).[1] Yet other heirloom pathogens, such as intestinal worms probably first acquired from eating the meat of wild animals, have gone on to infect human beings and domesticated animals such as pigs, dogs, and cats.[2]

Many infections have proven to be remarkably resilient. They have continued even through intermittent, recurrent crises of dwindling resources and through transitions between Ice Ages and eras of global warming. The ancestors of many infectious pathogens such as mumps, chickenpox, and smallpox originated as zoonoses—that is, infections of non-human animals that jumped species only in the past several thousand years and then evolved to become

human infectious diseases without non-human hosts. Measles, a pathogen that was once a zoonosis, emerged as human disease about 1000 years ago from the cattle virus that caused rinderpest. Other infectious pathogens continue to emerge from animal hosts. Influenza epidemics, powered by novel recombinations of swine and avian viruses, appear seasonally; once they produce illness and death, and their survivors develop specific immunity, they become self-limiting. Other originally zoonotic diseases, such as HIV, evolved into human scourges only in the past several decades.

Climate forces set the ecological parameters for the survival of the multitudes of pathogens that have caused human disease.[3] Two essential biophysical parameters—precipitation and temperature—have had a determinative influence on global distributions of protozoa, bacteria, viruses, and their various hosts, whether insect, rodent, domesticated and wild animal, or human. Over millennia, humid and drying phases of climate reorganized the zones in which diseases could be transmitted. Consider, for example, the case of the Sahara. During a humid era that peaked *c.* 7000–4000 BCE, the Sahara was a land of vegetation and lakes. The decisive drying out of the Sahara that followed brought transformations in ways of life, as climate migrants were forced either north or south into moister zones. This climatic shift created conditions that prevented the transmission of certain pathogens. In the Sahara, aridity and extreme daily temperature variations produced a healthier human environment than in sub-Saharan Africa. Today, as throughout history, warm and humid environments enable the transmission of the greatest number of diseases.

In a broad biogeographical sense, cold temperatures set the northern and southern limits within which most pathogens can survive. The ecology of contemporary malaria offers a good example. The mosquito species that host falciparum malaria parasites could survive during the summer season even above the Arctic Circle, but even summertime Arctic temperatures would be too low for malaria parasites to reproduce in their guts. There is no falciparum malaria transmission in the extreme North. Similarly, the zone of malaria transmission has never extended into the Antarctic, because mean temperatures there fall below the threshold for mosquito reproduction as well as the reproduction of the parasites in mosquito guts.

28.3 New Pathogens and Centers of Transmission

The rapid end of the last Ice Age and start of the warmer Holocene era about 12,000 years ago, followed by rising aridity in southern Eurasia and North Africa from *c.* 4000 to 3000 BCE, established some of the baseline ecological conditions that allowed for the flourishing of seed-based agriculture. In this sense, climate forces ushered in the age of modern humanity. The different lateral bands of climate that ring the earth—the tropical, subtropical, temperate, and Arctic and Antarctic zones—have been relatively stable since *c.* 3000 BCE (see Chap. 15).

In the river basins of North Africa and southern Eurasia, those who farmed eventually produced food surpluses that allowed for impressive increases in human numbers. The farming communities also supported populations of insects, rodents, and dogs who lived off the stored food supplies and human wastes. The early phases of animal domestication took place in the same regions, and newly acquired zoonotic infectious diseases greatly contributed to human morbidity and mortality.[4] The early river basin diseases such as whooping cough, mumps, chickenpox, rubella, and smallpox jumped from animal species and accommodated themselves to human hosts. They spread from infected persons to healthy persons without an intermediary vector or host, much as the common cold does today. Many of these pathogens—particularly smallpox and measles—could have an extraordinarily destructive power when introduced to epidemiologically naïve populations.

The greater population density of these farming communities facilitated new levels of exposure to infectious pathogens. In regional hinterlands with uneven population densities, these pathogens circulated intermittently. Everywhere, they hit the non-immune populations hardest, and these tended to be the youngest generations and newest immigrants. Although the farming communities were repeatedly hard hit, they became "disease-experienced" in the sense that the survivors of the lethal diseases generally gained a life-long immunity to them. This immunity provided them with an epidemiological advantage over surrounding populations, which helps to explain the expansion of "river basin cultural zones" into the surrounding hinterlands.[5]

A similar process probably took place in tropical Africa, where the first farmers cultivated yam tubers rather than grain seeds. As in the river basin societies of North Africa and southern Eurasia, the surplus in food calories allowed for increasing populations of farmers. Yam farmers first expanded into rainforest areas, where ecological conditions were propitious for the proliferation of a species of particularly efficient malaria-transmitting mosquitoes. The high densities of village farmers and vector mosquitoes allowed for the intense transmission of falciparum malaria. Those who survived their first encounters gained a partial immunity that accorded them an epidemiological advantage over hunting and gathering peoples. Over time, these "disease-experienced" communities expanded throughout West and West Central Africa in an unfolding demographic process known as the Bantu expansions.[6]

In tropical Africa other lethal pathogens continued to cross from wild animals into human communities and their herds of livestock. Seasonal weather conditions modulated transmission of some pathogens, such as trypanosomiasis (also known as sleeping sickness), a deadly infection transmitted by the bite of *Glossina* flies from wild animal reservoirs to human communities and livestock. Outbreaks of sleeping sickness were in part a function of abundant rainfall that promoted the growth of bush habitat in which the flies bred.[7]

In the Americas agricultural practices developed first in the Mesoamerican and Andean regions, supporting larger population growth in those centers of civilization. However, these regions contained few large animals suitable for

domestication or farming, sparing human populations the same onslaught of zoonotic diseases as in North Africa and Eurasia. American populations were nevertheless subject to the forces of climate, and severe and protracted droughts in the early centuries of the second millennium CE are thought to have brought about the collapses of the Mayan civilization in what is today Guatemala and the Hohokam civilization in what is today the state of Arizona.[8]

28.4 Processes of Epidemiological Integration

The growth of agrarian empires brought raids against vulnerable neighbors and warfare against regional rivals, as well as new trade relationships. The increases in political violence and long-distance commerce were key motors for the epidemiological integration of Eurasia. "Natural disasters" almost certainly had a role in these processes, but the relationships between many epidemic diseases and climate, weather, and ecological change are difficult to establish with certainty. Such is the case with the Plague of Justinian, an epidemic of the bubonic plague in the sixth century CE that created havoc in the Byzantine world. It is possible that this sixth-century event was linked to a volcanic explosion that cast an enormous volume of dust into the atmosphere and caused the failure of harvests. In this view, extreme weather conditions created food shortages, a subsequent famine, and a heightened biological vulnerability to pathogens. It is also possible that the epidemic contributed to the inability of the population to harvest crops (see Chap. 32). Natural disasters and weather anomalies in earlier eras are difficult to invoke with precision as a direct cause or intensifier of infectious disease, because the evidence is frequently suggestive rather than definitive.

In some cases, climate events may have helped to determine the timing of epidemic outbreaks. A catastrophic bubonic plague epidemic ripped through Europe in the mid-fourteenth century and smote European populations in intermittent waves for centuries thereafter. New research findings have established a correlation with wet spring seasons in China. This new evidence supports an alternative, climate-based explanation for the recurrent plagues that may replace the previous consensus that plague continued to circulate in black rat populations in Europe. The new climate-based interpretation argues that maritime trade (rather than overland caravans) introduced the plague bacillus, borne into Europe by gerbils (rather than rats). In this view, long-distance trade, rather than extreme weather events, may have been the primary mechanism of diffusion across Eurasia, although wet spring seasons contributed to larger populations of the gerbil reservoir of the pathogen.[9]

Extreme weather events such as drought, cooling from volcanic explosions, flooding from high rainfalls, and unseasonal frosts could wreak havoc on harvests, and one of the most frequent impacts was famine. Shortages of food caused nutritional stress and reduced the resiliency of the sufferers, who were more liable to fall ill, particularly to diseases associated with poor sanitation.[10] When shortages induced migrations, famine refugees suffering from contagious

diseases could introduce infections to new populations.[11] The number of unusual weather events increased during climate shifts such as the Little Ice Age that afflicted Europe and North America from the fourteenth through the mid-nineteenth centuries (see Chap. 23) and the period of low rainfall along the western Sahel from the seventeenth until the mid-nineteenth centuries (see Chap. 20).[12]

The voyages of discovery and conquest, initiated by Christopher Columbus, unleashed an epidemiological disaster in the New World.[13] The Old World pathogens, once introduced across the Atlantic Ocean, had an even more destructive demographic impact on New World populations than had the bubonic plague in Europe or elsewhere in Eurasia (even though the millennia-long process of epidemiological integration in Eurasia had itself been a profoundly destructive process). In the first century following European contact, the Old World pathogens reduced the American peoples—none of whom had acquired any immunities to the invaders—to roughly 10% of their pre-contact population sizes.[14]

Many of these virulent pathogens were viruses rather than bacteria or protozoa, and they were transmitted directly from person to person, without an intermediate non-human vector or host. Smallpox wrought the most damage as it tore through densely populated areas of the Americas. The principal limitation of these epidemics—including smallpox, measles, chickenpox, and mumps—was population density, because these viruses left survivors with lifetime immunity to reinfection. In the case of low population densities, the viruses ran out of non-immunes to infect and became self-limiting, disappearing for a time only to flare out of control among later generations born without immunity.

A severe drought in the mid-sixteenth century struck the highlands of Mexico, which suffered severe epidemics in 1545–8 and 1576–8. The highland epidemics have generally been attributed to typhus, a disease caused by *Rickettsia* bacteria transmitted by fleas or ticks.[15] A recent reassessment of the sixteenth-century highland epidemics and later outbreaks in the seventeenth, eighteenth, and early nineteenth centuries, however, suggests that the epidemics may have been caused by indigenous hemorrhagic fevers.[16] The mid-sixteenth-century drought may have brought a rodent host into contact with a highland population weakened by crop failures and the excessive labor demands of Spanish colonists.[17] Otherwise, climatic conditions in the New World appear to have played a minor role in the viral epidemics caused by Old World pathogens, although extreme weather events, as always, could increase the susceptibility of the affected populations to more severe encounters with disease.

Climate and weather had other effects on vector-borne disease. Mosquito-borne diseases such as falciparum malaria (a protozoal infection) and yellow fever (a viral infection) first emerged in tropical Africa.[18] Unlike the person-to-person infections described above, these mosquito-borne diseases could only spread to regions with similar climates. For example, yellow fever and its principal vector, *Aedes aegypti*, were transferred laterally into the Americas, and became established in the same tropical latitudes. Weather conditions played a

pivotal role, because rainy seasons produced denser populations of vector mosquitoes, and the density of the vectors was a critical variable in the intensities of transmission. Another major variable was the immunological status of the populations. The survivors of yellow fever infections gained a life-long immunity. The survivors of falciparum malaria gained some degree of acquired immunity that did not protect them from future infections but did lessen the severity of those infections. Most malarial deaths occurred at the first encounter.[19]

28.5 Biomedicine, Emerging Diseases, and Climate Change

Over the past two centuries, advances in biomedicine and improvements in standards of living have greatly reduced the incidence and mortality of infectious disease among populations in economically advanced states.[20] Some programs for the control of infectious diseases in economically less-developed states have also had major successes. In recent decades, global health initiatives have dramatically reduced childhood deaths through immunization programs across the world. Deaths from the scourge of malaria are now largely restricted to tropical Africa, where major efforts are currently underway to reduce transmission.[21]

These developments have coincided with a rapid increase in passenger air travel that has facilitated the global diffusion of pathogens. The greatest concern is for the spread of viral pathogens such as influenza that can be transmitted via human respiration, because our ability to make vaccines and administer doses at the population level falls far short of what is needed. This concern, however, is largely independent from the anticipated increase in extreme climate events that are expected to accompany anthropogenic forcing of climate change.

There are also major concerns that global climate change will increase the transmission of vector-borne diseases. The West Nile virus, introduced into the United States in 1999, has been found in a large number of mosquito species, and it is likely that global warming will extend the range of many of these species and may increase transmission. These possibilities are real, although at present the total number of people affected is small. There is no antidote or vaccine for West Nile virus, although insecticides, screens, and repellents are highly effective. The greater health concerns are that warming may increase the transmission of mosquito-borne diseases such as malaria, dengue fever, and chikungunya fever. In tropical Africa, where transmission rates are highest, continued warming will likely extend the range of the vector mosquitoes to higher altitudes in mountainous regions of eastern and central Africa, although some experts believe this concern is overblown.[22]

Further vulnerabilities come from rising sea levels and storm surges, which could compromise the integrity of coastal water and sanitation systems. Failure of sanitation systems and subsequent pollution of water supplies with fecal

matter has in the past set off large-scale epidemics, such as in mid-twentieth-century New Delhi.[23]

28.6 Conclusion

The relationships between climate change, ecological change, and human infectious diseases are complex, and our understandings of these relationships will continue to be refined by the development of new data and perspectives from a wide range of investigations.[24] A major challenge will be for researchers to incorporate insights from different disciplinary perspectives. A fuller understanding of the importance of climate in the epidemiological past can only be won from an evolving integration of the biological, social, and historical sciences.

Notes

1. Barrett and Armelagos, 2013, 29–41; Torrey and Yolken, 2005, 14–19.
2. On the tapeworm, see Hoberg et al., 2001; on the roundworm, see Peng and Criscione, 2012.
3. For an impressive effort to synthesize the scientific literature on climate change and its impact on the human past, see Brooke, 2014.
4. Diamond, 1997, 195–214.
5. McNeill, 1976.
6. Webb, 2009, 18–41.
7. This inference is based upon historical evidence from the twentieth and twenty-first centuries. During the era of European colonization of tropical Africa, European colonial governmental policies and medical campaigns that included the forced relocation of African populations also influenced the distribution of sleeping sickness. See Courtin et al., 2008; Hoppe, 1997; Lyons, 1992.
8. The explanations of the social collapses are multicausal and contested. See Redman, 1999; Diamond, 2005; McAnany and Yoffee, 2009.
9. Schmid et al., 2015.
10. The influence of famine conditions could persist for several decades. The Great Famine of 1315–17 and the Great Bovine Pestilence of 1319–20 (which produced a prolonged dearth of dairy products) in England and northern Europe rendered the populations more susceptible to the ravages of the bubonic plague (DeWitte and Slavin, 2013). On the susceptibility to infectious diseases associated with poor sanitation, see Mokyr and Ó Gráda, 2002.
11. Schellekens, 1996; Post, 1984.
12. For a recent discussion of the evidence for the Little Ice Age, see White, 2014; on the western Sahel, Webb, 1995.
13. Crosby, 1972.
14. Stannard, 1993.
15. Nothing is known about the geographical origins of typhus, including whether it is an Old World or New World pathogen (Wolfe et al., 2012, 358).
16. Acuña-Soto et al., 2000.
17. Acuña-Soto, 2002; Marr and Kiracofe, 2000.

18. Bryant et al., 2007; Liu et al., 2010.
19. Webb, 2009, 66–91; McNeill, 2010.
20. In the early nineteenth century, researchers isolated medically active compounds such as quinine, a highly effective anti-malarial that was the first disease-specific drug in the Western *materia medica*. See Webb, 2009.
21. Webb, 2014.
22. Chaves and Koenraadt, 2010.
23. Dennis and Wolman, 1959.
24. The National Academy of Sciences has convened three workshops to explore the relationships between weather events, disease outbreaks, and emerging infections and another workshop to improve our understandings of the relationships between vector-borne disease and environmental and ecological change and human health. See Choffnes and Mack, 2014; Mack et al., 2008; National Research Council, 2001; Lemon, 2008.

References

Acuña-Soto, Rodolfo. "Megadrought and Megadeath in 16th-Century Mexico." *Emerging Infectious Diseases* 8 (2002): 360–62.

Acuña-Soto, Rudolfo et al. "Large Epidemics of Hemorrhagic Fevers in Mexico 1545–1815." *The American Journal of Tropical Medicine and Hygiene* 62 (2000): 733–39.

Barrett, Ron, and George J. Armelagos. *An Unnatural History of Emerging Infections*. Oxford: Oxford University Press, 2013.

Brooke, John L. *Climate Change and the Course of Global History: A Rough Journey*. New York: Cambridge University Press, 2014.

Bryant, Juliet E. et al. "Out of Africa: A Molecular Perspective on the Introduction of Yellow Fever Virus into the Americas." *PLoS Pathog* 3 (2007): e75.

Chaves, Luis Fernando, and Constantianus J.M. Koenraadt. "Climate Change and Highland Malaria: Fresh Air for a Hot Debate." *The Quarterly Review of Biology* 85 (2010): 27–55.

Choffnes, Eileen R., and Alison Mack. *The Influence of Global Environmental Change on Infectious Disease Dynamics: Workshop Summary*. Washington, DC: National Academies Press, 2014.

Courtin, F. et al. "Sleeping Sickness in West Africa (1906–2006): Changes in Spatial Repartition and Lessons from the Past." *Tropical Medicine & International Health* 13 (2008): 334–44.

Crosby, Alfred W. *The Columbian Exchange: Biological and Cultural Consequences of 1492*. Westport, CT: Greenwood Press, 1972.

Dennis, Joseph M., and Abel Wolman. "1955–56 Infectious Hepatitis Epidemic in Delhi, India [with Discussion]." *Journal American Water Works Association* 51 (1959): 1288–98.

DeWitte, Sharon, and Philip Slavin. "Between Famine and Death: England on the Eve of the Black Death—Evidence from Paleoepidemiology and Manorial Accounts." *Journal of Interdisciplinary History* 44 (2013): 37–60.

Diamond, Jared M. *Guns, Germs, and Steel: The Fates of Human Societies*. New York: W.W. Norton, 1997.

Diamond, Jared M. *Collapse: How Societies Choose to Fail or Succeed*. New York: Viking, 2005.

Hoberg, Eric et al. "Out of Africa: Origins of the Taenia Tapeworms in Humans." *Proceedings: Biological Sciences* 268 (2001): 781–87.

Hoppe, Kirk A. "Lords of the Fly: Colonial Visions and Revision of African Sleeping-Sickness Environments on Ugandan Lake Victoria, 1906–61." *Africa* 67 (1997): 86–105.

Lemon, Stanley M. *Vector-Borne Diseases: Understanding the Environmental, Human Health, and Ecological Connections, Workshop Summary*. Washington, DC: National Academies Press, 2008.

Liu, Weimin et al. "Origin of the Human Malaria Parasite Plasmodium Falciparum in Gorillas." *Nature* 467 (2010): 420–25.

Lyons, Maryinez. *The Colonial Disease: A Social History of Sleeping Sickness in Northern Zaire, 1900–1940*. Cambridge; New York: Cambridge University Press, 1992.

McAnany, Patricia Ann, and Norman Yoffee, eds. *Questioning Collapse: Human Resilience, Ecological Vulnerability, and the Aftermath of Empire*. New York: Cambridge University Press, 2009.

Mack, Alison et al. *Global Climate Change and Extreme Weather Events: Understanding the Contributions to Infectious Disease Emergence, Workshop Summary*. Washington, DC: National Academies Press, 2008.

McNeill, John Robert. *Mosquito Empires: Ecology and War in the Greater Caribbean, 1620–1914*. New York: Cambridge University Press, 2010.

McNeill, William Hardy. *Plagues and Peoples*. Garden City, NY: Anchor Press, 1976.

Marr, John S., and James B. Kiracofe. "Was the Huey Cocoliztli a Hemorrhagic Fever?" *Medical History* 44 (2000): 341–62.

Mokyr, J., and C. Ó Grada. "What Do People Die of During Famines: The Great Irish Famine in Comparative Perspective." *European Review of Economic History* 6 (2002): 339–63.

National Research Council (U.S.), and Ecosystems Committee on Climate Infectious Disease, and Human Health. *Under the Weather: Climate, Ecosystems, and Infectious Disease*. Washington, DC: National Academy Press, 2001.

Peng, W., and C.D. Criscione. "Ascariasis in People and Pigs: New Inferences from DNA Analysis of Worm Populations." *Infection, Genetics and Evolution: Journal of Molecular Epidemiology and Evolutionary Genetics in Infectious Diseases* 12 (2012): 227–35.

Post, J.D. "Climatic Variability and the European Mortality Wave of the Early 1740's." *The Journal of Interdisciplinary History* 15 (1984): 1–30.

Redman, Charles L. *Human Impact on Ancient Environments*. Tucson: University of Arizona Press, 1999.

Schellekens, Jona. "Irish Famines and English Mortality in the Eighteenth Century." *The Journal of Interdisciplinary History* 26 (1996): 29–42.

Schmid, Boris V. et al. "Climate-Driven Introduction of the Black Death and Successive Plague Reintroductions into Europe." *Proceedings of the National Academy of Sciences* 112 (2015): 3020–25.

Stannard, David E. *American Holocaust: Columbus and the Conquest of the New World*. New York: Oxford University Press, 1993.

Torrey, E. Fuller, and Robert H. Yolken. *Beasts of the Earth: Animals, Humans, and Disease*. New Brunswick, NJ: Rutgers University Press, 2005.

Webb, James L.A. *Desert Frontier: Ecological and Economic Change along the Western Sahel, 1600–1850*. Madison: University of Wisconsin Press, 1995.

Webb, James L.A. *Humanity's Burden: A Global History of Malaria*. Cambridge; New York: Cambridge University Press, 2009.

Webb, James L.A. *The Long Struggle Against Malaria in Tropical Africa*. New York: Cambridge University Press, 2014.

White, Sam. "The Real Little Ice Age." *Journal of Interdisciplinary History* 44 (2014): 327–52.

Wolfe, N.D. et al. "Origins of Major Human Infectious Diseases." In *Improving Food Safety Through a One Health Approach*. Washington, DC: National Academies Press, 2012.