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Extreme weather events and infectious disease outbreaks

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Human-driven climatic changes will fundamentally influence patterns of human health, including infectious disease clusters and epidemics following extreme weather events. Extreme weather events are projected to increase further with the advance of human-driven climate change. Both recent and historical experiences indicate that infectious disease outbreaks very often follow extreme weather events, as microbes, vectors and reservoir animal hosts exploit the disrupted social and environmental conditions of extreme weather events. This review article examines infectious disease risks associated with extreme weather events; it draws on recent experiences including Hurricane Katrina in 2005 and the 2010 Pakistan mega-floods, and historical examples from previous centuries of epidemics and 'pestilence' associated with extreme weather disasters and climatic changes. A fuller understanding of climatic change, the precursors and triggers of extreme weather events and health consequences is needed in order to anticipate and respond to the infectious disease risks associated with human-driven climate change. Post-event risks to human health can be constrained, nonetheless, by reducing background rates of persistent infection, preparatory action such as coordinated disease surveillance and vaccination coverage, and strengthened disaster response. In the face of changing climate and weather conditions, it is critically important to think in ecological terms about the determinants of health, disease and death in human populations.

In late 1987 Rwanda, in central-eastern Africa, was drenched by heavy rainfall along with warmer temperatures. These extreme climatic conditions arose during the El Niño event of 1987–88. Soon after, in a region of Rwanda where malaria occurrence was well monitored and where non-climatic variables were also recorded and able to be taken into account, the malaria infection rate increased more than threefold compared to the preceding 3 years, particularly at higher altitudes.[1]

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The 2 opposite poles of the Pacific Ocean-based El Niño Southern Oscillation (ENSO) are the El Niño and La Niña phases. ENSO is a quasi-periodic oscillation (typically reversing approximately every 6–7 years) of coupled changes in atmospheric pressure and sea-surface temperature and flow direction, and hence of convective atmospheric circulation that affects climatic conditions around much of the low-to-mid latitude world. Some evidence and modeled projections suggest an increasing frequency of ENSO events under medium-scenario climate change conditions by around 2050.[2]

ENSO, meanwhile, provides an analog for us to learn more about climate system dynamics, extreme weather manifestations and their environmental and human health consequences. More such information about climatic variations and health consequences is needed from research teams around the world to gain a fuller understanding and anticipation of the infectious disease risks from impending human-driven climate change. Once gained, that knowledge can then be infused into public awareness and policy making.

Throughout recorded history infectious disease outbreaks have very often followed in the wake of extreme weather events (EWEs), particularly from river-basin flooding and from acute food shortages due to destructive storms or severe but short-lived.[3] For as long as there are microbes and, where relevant, vectors and reservoir animal hosts living among humans (not yet bioengineered beyond the natural constraints of the biosphere!), that relationship will continue. Microbes, with a metabolism and replication to support, are naturally opportunistic in exploiting conditions that arise from the physical disruptions of EWEs, the social chaos, displacement and, often, under-nutrition and starvation.

As populations and their densities, interconnectivity and mobility continue to grow, and as humankind encroaches increasingly and disruptively on the natural environment, so the opportunities for contact, infection and its onward transmission will increase. Surveillance, better coordinated across national borders, and rapid public health response can restrict infectious disease spread; vaccination can prevent both individual infection and 'herd' epidemic; while antibiotics remain, for the moment, a faltering backup option.

Extreme Weather Events: Rising Future Risks

Supplementing the health risks associated with this ongoing background uptrend in potential transmission of many infectious

diseases are the additional risks due to EWEs, or 'weather disasters'. Those are projected to increase as the process of human-driven climate change advances. The greater the amount of heat trapped within the lower atmosphere, the greater the energy flux within the atmosphere, and the more energetic and variable the weather patterns become. The physics of this is well understood – illustrated, for example, by the exponential heightening of cyclonic intensity by sea-surface warming of just 1–2°C above the threshold temperature of around 27°C.

The complexities of topography, regional atmospheric (and ocean) circulation systems and the seeming stochastic element in climate system behavior means that climate scientists cannot yet forecast accurately where and what types of EWEs will increase. Nor can current modeling forecast whether a regional increase will be in the frequency or intensity of the event – or both.

Evidence has strengthened, however, that climate change has contributed to the recent evident uptrend in recorded EWEs.[4,5] Attribution of causal influence remains a topic of public contention, however; and the amplifying role played by a warmer and less stable climate system does not contribute similarly to all such events. The Intergovernmental Panel on Climate Change, recognizing the rising importance of weather disasters as a source of diverse risks to human communities, published a comprehensive scientific assessment of climate change and EWEs, their potential impacts and costs, and the options for impact-reducing adaptation.[6]

Recent Examples

Recent experiences give a measure of the extent and range of infectious disease consequences that often follow EWEs. Consider, for example, Hurricane Katrina in the southern USA in late August 2005 and the mega-floods in northwest Pakistan in 2010.

The US Centers for Disease Control and Prevention lists a wide range of infectious diseases that are likely to occur following weather disasters.[7] A number of these, broadly classified, were identified in the aftermath of Hurricane Katrina's impact on New Orleans, from late August through September 2005. They were predominantly infections of the gastrointestinal tract, respiratory system, skin and open wounds. Surveillance at New Orleans Area hospitals for conditions with infectious disease epidemic potential, by the Louisiana Department of Health and New Orleans Public Health Response Team during September 9–10, around 12 days after the hurricane hit, identified 869 presumptive cases.[8]

- Non-infectious rash 299
- Respiratory infections 188
- Vomiting 142
- Fever 98
- Dehydration 87
- Watery diarrhea 55

There were also 17 cases of Vibrio infections of the skin: V. vulnificus (14 cases, with 3 deaths) and V. parahemolyticus (3 cases, with 2 deaths).[9] A regional peak of 31 cases of mosquito-borne West Nile Virus occurred during September 2–18, and then just one case occurred in the third week after Katrina. Quantitative attribution was made difficult by the fact that the case numbers had been rising during the month before the event, presumably reflecting an underlying seasonal fluctuation.

In July 2010 nationwide floods in Pakistan caused huge damage and suffering after unprecedented monsoon rains overwhelmed the expansive Indus River basin. A vast cascade of waters, triggered by heavy monsoon rains in late July, swept through the basin, flushing away homes, roads, bridges, crops and livestock. The flooding left a swathe of destruction from north to south, and one-fifth of the country was inundated. Those floods were Pakistan's worst in recorded history. The flooding killed around 2,000 people, injured another 3,000 and affected over 20 million people – more than a tenth of the population. Millions were left homeless, as housing, schools, health facilities, communication networks, power plants, grids and irrigation channels were damaged or destroyed.[10] During the ensuing year, more than 37 million medical consultations were reported with acute respiratory infection, acute diarrhea, skin diseases, and suspected malaria being the most common presentations.[11]

In poor, crowded and unhygienic settings, cholera is lurking in the background as the most feared source of an outbreak of diarrheal disease; and often outbreaks to occur – especially in crowded, poor and unhygienic living conditions, where faecal contamination of drinking water often occurs. The disease displays a complex relationship to extremes of weather conditions, flooding being the more recognized and intuitively likely cause.

Interestingly, though, an analysis of the systematic colonial records from British India, for the districts of Madras (now Chennai) during 1901–1940, found that cholera had a dual relationship to water extremes – flooding and drought – linked to seasonally-related rhythms of periods with and without cholera mortality.[12] These results suggested a model of cholera seasonality with 2 different routes of transmission. One route, person-to-person transmission in areas of south-east India with typically abundant water, is amplified by excessive rainfall (causing flooding, social disruption and crowding). In other areas that are typically dry, and which often have a concentrated cholera-bacterial contamination of the limited drinking water sources, is buffered by increasing, diluting, rainfall. Temperature, though, is not without influence. A much more recent analysis of a monthly 18-year time series of cholera incidence in nearby Bangladesh found that cholera variability over time includes an influence at the typical 5–9 year periodicity of the El Niño–Southern Oscillation (ENSO), a component that was best explained by variations in temperature.[13]

Many more details exist in relation to a diversity of other EWEs that have occurred widely in recent years. Taken in aggregate, they underscore the potentially great health risks and demands on the health-care and public health systems and on national budgets that will result from increased incidences and epidemic outbreaks of infectious diseases following future, and often more severe, weather disasters as the world's climate continues to change.

The types and severity of post-EWE increases in infectious disease incidence or in actual epidemics depend, of course, on geography, demography, economic conditions, the pre-existing profile of infectious diseases in the affected population, and cultural and political context. Vector-borne diseases will prove most problematic in tropical and sub-tropical regions, where mosquitoes often thrive, although some other vectors such as ticks inhabit temperate regions. Cholera, noted above, is primarily a disease of poverty and unhygienic crowding. Respiratory infections occur widely, and are of many types – some are enhanced by warmer conditions, some by cooler conditions.

Historical Examples

The experiences of the past 5 thousand years, since historical records first emerged, contain many examples of weather disasters as harbingers of epidemics, or 'pestilence'. The presumed agency of such disease was, variously, one arising from theology, cosmology, rank speculation and politics. Unavoidably, most of what we can learn from history about this relationship comes from the Eurasian super-continent and, later, from North America. Few specific records of epidemics are available from other regions.

Four examples will illustrate that long historical experience of weather extremes and (what we now recognize were) infectious diseases.

First, the first 2 great pandemics of bubonic plague: (i) the Plague of Justinian that brought the now Constantinople-based eastern rump of the once mighty Roman Empire to its knees in mid-sixth century CE, and its subsequent regional spread for more than 2 centuries; and (ii) the 'Black Death' that killed around one third of the European population during the initial onslaught from 1347 to the early 1360s, and then many more over 3 more centuries.

The Plague of Justinian has a seemingly direct connection with a half-decade of extreme and very unusual weather in the greater Mediterranean region, part of an acute global cooling that began abruptly in 536 CE due to atmospheric shrouding caused by a massive volcanic eruption in Indonesia.[3] 'Plague' was known, and documented by several Roman historians of the time, to have been circulating in north-eastern Africa.[14] Then in 541 CE the plague broke out in Egypt's Mediterranean coastal port of Pelusium, apparently introduced via the vast grain shipments from Ethiopia ('Axum') or the Horn of Africa, much of destined for Constantinople, and which usually had to pass through the extreme heat of either the Nubian and Upper Egypt desert or the arid northern coastal stretch of the Red Sea. The heat that typically prevailed in that region during the inland (Nile river) or coastal shipping season was sufficient to impede flea reproduction and rat survival; but now, for several years, that barrier was loosened and presumably the probabilities duly shifted.[3,15] Before long the plague reached Constantinople where up to half the population died, horribly, within the first 3 months or so. The Emperor Justinian was one of the lucky minority of people infected, but who managed to survive.

The Plague of Justinian entailed, apparently, an unusual acute disruption of climatic conditions due to a relatively infrequent cause, a massive volcanic eruption. In contrast, the connection of the Black Death pandemic with extremes of weather was of a different kind.

The account of that great outbreak begins in Central Asia and then on to China. Regional changes in climatic conditions in the early fourteenth century are thought to have unsettled and mobilized populations of the plague bacterium's natural wild host, ground-burrowing rodents (marmots) in the Kazakhstan region.[16,17] Via heightened contacts of those infected rodents with the black rats that cohabit with humans, or, more directly, with the humans themselves, the disease spread east into China. There it appears to have been the cause of the great, death-dealing, epidemic that broke out in China in the 1330s in response to massive and destructive river floods that displaced vast numbers of rats and humans, crowding into drier and safer localities.[18,3]

Subsequently, via the medium of either, or both of, east-to-west trade along the Silk Road and the movement of horse-borne Mongol armies (and stowaway rats) marauding their way west to the Black Sea region, the plague bacterium gained entry to Europe via the Mediterranean coast in 1347. It then spread relentlessly, flaring up recurrently, for the next 3 centuries.

Far away, and 2 centuries after that fateful entry of Yersinia pestis into Europe, a sequence of virulent epidemics, associated with a sequence of severe droughts in the sixteenth century, struck the Aztec survivors of the Spanish conquest. The cumulative death toll of these epidemics was huge, almost 90 percent, and the native Mexican population did not recover to pre-Hispanic levels until the twentieth century. The persistent drought conditions punctuated by occasional years of intense rainfall were associated with the spill-over of indigenous rodent-borne infections into human communities. Indeed, the coincident timing of the great drought of 1540–1580 and the 2 large epidemics of 1545 and 1576 suggests that they were caused by 'Cocoliztli' infection – indigenous hemorrhagic viral fevers transmitted by infected rodents whose food-seeking activity during drought and then proliferation during the rains would have increased human contact with the virus.[19]

Analogous contemporary evidence for this apparent scenario comes from the acute epidemic of rodent-borne hantavirus pulmonary syndrome in southwest USA in 1993, after the rains that followed the El Niño-related drought-years of the early 1990s.[20] In that case, the rapid post-drought proliferation of the field-mouse population (the natural hosts of the hantavirus), feeding on the restored abundance of piñon nuts, greatly increased human contact with aerosolised hantavirus from mouse excreta.

Returning now to Europe, the early decades of the seventeenth century provide a different, and politically more complex, example of extremes of climate causing increased outbreaks of epidemics. This period was the nadir of the Little Ice Age, when life for the majority became increasingly and often freezingly miserable, when harvest failures (and associated witch-burning) were

common, and when fledgling nation-states were struggling to gain and hold territory. Historians refer to it as 'the General Crisis of the Seventeenth Century'.[21]

The chaotic and destructive Thirty Years War broke out in 1618, with religious conflict within the aging Holy Roman Empire and the dynasties of France and Austria-Hungary (Bourbons and Habsburgs, respectively) striving for supremacy in Europe, along with an overlay of local territorial grabs. During those several climatically dire decades, rates of displacement, migration, food price spikes and starvation increased and the frequency of recorded epidemic outbreaks and resultant deaths increased 3-fold.[22]

A final historical example comes from the very early years of European occupation of Australia. The First Fleet arrived in Sydney Cove in early 1788, and then faced 2 years of violent and cold weather, with heavy rainfalls. This was followed from mid-1790 by a switch in climatic conditions that ushered in drought and great heat, sufficient to cause serious food shortage and stringent rationing, and, nearly, the collapse of the young colony. Some have concluded that this extreme of weather, which persisted for over 2 years, was due to the latter stage of the extraordinary El Niño event – the greatest such event of the eighteenth century – that caused much damage, strife and adverse health around much of the world.[23]

During this oppressive change in climate an initial surge in illnesses and deaths occurred among convicts during August-September of 1790. Many deaths were from dysentery and scurvy, although there may well have been an acute epidemic of imported infectious disease, most probably typhoid or typhus.[24] The suffering was not equally shared; a majority of the recorded deaths occurred in debilitated survivors of the seriously mismanaged Second Fleet (with its 25% on-board mortality among convicts).

The Future

The inevitability and heterogeneity of infectious disease clusters or epidemics occurring after most extreme weather events that damage human communities, their infrastructure and physical environment, and their daily sources of food, water, power and shelter are evident from both recent and historical experience. And yet it remains difficult to forecast the extent and pattern of such outcomes in a future, climatically changed, world.

Climate scientists do not yet know enough about the precursors and triggers of EWEs, region by region, to be able to build valid and useful forecasting models for this purpose. Meanwhile, much is known about the conditions that predispose to infectious disease outbreaks, including poverty, hygiene, crowding, freely-breeding mosquito populations, food insecurity, and lack of (or resistance to) vaccination. If local background rates of persistent infections, such as diarrheal diseases, acute respiratory infections in children, malaria and dengue can be lowered – as they should be anyway – then the multiplying impact of EWEs will be greatly reduced.

Local communities and governments at all levels can, nevertheless, constrain the post-event risks to health via many preparatory actions. These include, as mentioned earlier, improved and better coordinated surveillance systems (not just of the disease itself, but of precursor signals, especially vector populations and activity) and systematic vaccination coverage. Health-care facilities may need expansion and upgrading, and professional staff will require in-service training in relation to disaster response and likely shifts in the patterns of post-event community demands.

Conclusion

Extreme weather events create the sorts of environmental and social conditions in which many infectious diseases of humans and, often, of other animals thrive. As outbreaks of infectious diseases occur in particular locations in the wake of extremes of climate and weather conditions, we will hopefully also re-acquire much of the earlier, if cruder, wisdom that led communities to think in more ecological terms about the determinants of health, disease and death in human populations. Human-driven climate change will necessarily influence the re-framing of our concepts of health in whole communities, whole populations; experiences with infectious diseases will play a key role in that process.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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