Chapter 6
Infectious Diseases, Climate Change Effects on

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Glossary

Climate	The weather averaged over a long time or, succinctly, climate is what you expect, weather is what you get!
El Niño Southern Oscillation (ENSO)	A climate phenomenon whereby, following reversal of trade winds approximately every 4–7 years, a vast body of warm water moves slowly west to east across the Pacific, resulting in "an El Niño" event in the Americas and leading to a detectable change to climate (mostly disruption of normal rainfall patterns) across 70% of the earth's surface.
Emerging disease	An infection or disease that has recently increased in incidence (the number of cases), severity (how bad the disease is), or distribution (where it occurs).
Endemic stability	The counter-intuitive situation where the amount of disease rises as the amount of infection falls, such that controlling infection can exacerbate the problem.
Infection	The body of a host having been invaded by microorganisms (mostly viruses, bacteria, fungi, protozoa, and parasites).

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Infectious disease	A pathology or disease that results from infection. Note that many diseases are not infectious and not all infections result in disease.
Intermediate host	A host in which a parasite undergoes an essential part of its lifecycle before passing to a second host, and where this passing is passive, that is, not by direct introduction into the next host (see *vector*).
Vector	Usually, an arthropod that spreads an infectious pathogen by directly introducing it into a host. For diseases of humans and animals, the most important vectors are flies (like mosquitoes, midges, sandflies, tsetse flies), fleas, lice, and ticks. Aphids are important vectors of diseases in plants. In some instances, other means of carriage of pathogens, such as human hands, car wheels, etc., are referred to as vectors.
Vector competence	The proportion of an arthropod vector population that can be infected with a pathogen.
Zoonosis	An infection of animals that can spread to, and cause disease in, humans (plural, zoonoses).

Definition of the Subject and Its Importance

Infectious diseases of humans continue to present a significant burden to our health, disproportionately so in the developing world. Infectious diseases of livestock affect their health and welfare, are themselves important causes of human disease and, exceptionally, can threaten our food security. Wildlife infections again present a zoonotic risk to humans, but additionally, such diseases may threaten vulnerable populations and be a cause of extinction and biodiversity loss. Wild populations are inherently more susceptible to environmental change, largely lacking any human protective influence that domesticated species and human populations may benefit from.

Many infectious diseases of humans and farmed or wild animals are influenced by weather or climate, affecting where or when disease occurs, or how severe outbreaks are, and it is therefore likely that future climate change, whether human caused or natural, will have an impact on future disease burdens. Understanding the processes involved may enable prediction of how disease burdens will change in the future and, therefore, allow mitigative or adaptive measures to be put in place.

While climate change will likely be an important cause of change in some infectious diseases in the future, there are other disease drivers which will also change over similar time scales and which may exacerbate or counteract any effects of climate change. Assessment of the future importance of climate change as an influence over future disease burdens must therefore be considered alongside other causes of change.

Introduction

The impact of infectious diseases of humans and animals seems as great now as it was a century ago. While many disease threats have disappeared or dwindled, at least in the developed world, others have arisen to take their place. Important infectious diseases of humans that have emerged in the last 30 years, for a range of reasons, include Acquired immune deficiency syndrome (AIDS), variant Creutzfeldt-Jakob disease (vCJD), multidrug resistant tuberculosis, severe acute respiratory syndrome (SARS), *E. coli* O157, avian influenza, swine flu, West Nile fever, and Chikungunya [1, 2]. The same applies to diseases of animals: Indeed, all but one of the aforementioned human diseases have animal origins – they are zoonoses – and hence the two subjects of human and animal disease, usually studied separately by medical or veterinary scientists, are intimately entwined.

What will be the global impact of infectious diseases at the end of the twenty-first century? Any single disease is likely to be affected by many factors that cannot be predicted with confidence, including changes to human demography and behavior, new scientific or technological advances including cures and vaccines, pathogen evolution, livestock management practices and developments in animal genetics, and changes to the physical environment. A further, arguably more predictable, influence is climate change.

Owing to anthropogenic activities, there is widespread scientific agreement that the world's climate is warming at a faster rate than ever before [3], with concomitant changes in precipitation, flooding, winds, and the frequency of extreme events such as El Niño. Innumerable studies have demonstrated links between infectious diseases and climate, and it is unthinkable that a significant change in climate during this century will not impact on at least some of them.

How should one react to predicted changes in diseases ascribed to climate change? The answer depends on the animal populations and human communities affected, whether the disease changes in severity, incidence, or spatiotemporal distribution and, of course, on the direction of change: Some diseases may spread but others may retreat in distribution. It also depends on the relative importance of the disease. If climate change is predicted to affect mostly diseases of relatively minor impact on human society or global biodiversity/ecosystem function, while the more important diseases are refractory to climate change's influence, then our concerns should be tempered.

To understand climate change's effects on infectious diseases in the future it is necessary to first understand how climate affects diseases today. This entry begins by first presenting examples of climate's effects on diseases of humans and livestock today and, from the understanding gained, then describes the processes by which climate change might affect such diseases in the future. Diseases of wildlife are important, to some extent, for different reasons to those of humans and livestock, and are therefore considered separately. The relative importance of climate change as a disease driver, compared to other forces, is considered,

with examples provided of where climate change both is, and is not, the major force. Finally, the future prospects and the uncertainties surrounding them are considered.

Weather, Climate, and Disease

Many diseases are affected directly or indirectly by weather and climate. Remarkably, no systematic surveys of links between diseases and weather/climate seem to exist and, therefore, it is not possible to indicate whether these diseases represent a minority or majority.

The associations between diseases and weather/climate fall broadly into three categories. The associations may be *spatial*, with climate affecting the distribution of a disease; *temporal* with weather or climate affecting the timing of outbreaks; or they may relate to the *intensity* of an outbreak. Temporal associations can be further broken into at least two subcategories: *seasonal*, with weather or climate affecting the seasonal occurrence of a disease, and *interannual*, with weather or climate affecting the timing, or frequency of years in which outbreaks occur. Here a selection of these associations is presented, which is by no means exhaustive but is, rather, intended to demonstrate the diversity of effects. Furthermore, the assignment of diseases into the different categories should not be considered hard-and-fast as many diseases could come under more than one heading.

Spatial

- Schistosomosis is an important cause of human mortality and morbidity in Africa and, to a lesser extent, in Asia. The disease is caused by species of *Schistosoma* trematode parasite, for which water-living snails are intermediate hosts. The distribution of suitable water bodies is therefore important for its distribution. However, there must also be suitable temperature: In China, *Oncomelania hupensis* snail intermediate hosts cannot live north of the January 0°C isotherm (the "freezing line") while *Schistosoma japonicum* only develops within the snail at temperatures above 15.4°C. Shistosomosis risk in China is therefore restricted to the warmer southeastern part of the country [4].
- Diseases transmitted by tsetse flies (sleeping sickness, animal trypanosomosis) and ticks (such as anaplasmosis, babesiosis, East Coast fever, heartwater) impose a tremendous burden on African people and their livestock. Many aspects of the vectors' life cycles are sensitive to climate, to the extent that their spatial distributions can be predicted accurately using satellite-derived proxies for climate variables [5].
- Mosquitoes (principally *Culex* and *Aedes*) transmit several viruses of birds that can also cause mortality in humans and horses. Examples are West Nile

fever (WNF) and the viral encephalitides such as Venezuelan, western, and eastern equine encephalitis (VEE, WEE, and EEE, respectively) [6]. The spatial distributions of the mosquito vectors are highly sensitive to climate variables.

Temporal-Seasonal

All of the previous examples of spatial associations between diseases and climate can also be classified as temporal-seasonal, as the effects of climate on the seasonal cycle of the intermediate hosts (snails, tsetse flies, and mosquitoes, respectively) also determines in part the seasonal cycle of disease. There are other diseases where the associations can be described as seasonal-temporal.

- Salmonellosis is a serious food-borne disease caused by *Salmonella* bacteria, most often obtained from eggs, poultry, and pork. Salmonellosis notification rates in several European countries have been shown to increase by about 5–10% for each 1°C increase in ambient temperature [7]. Salmonellosis notification is particularly associated with high temperatures during the week prior to consumption of infected produce, implicating a mechanistic effect via poor food handling.
- Foot-and-mouth disease (FMD) is a highly contagious, viral infection of cloven-footed animals, including cattle, sheep, and pigs. Most transmission is by contact between infected and susceptible animals, or by contact with contaminated animal products. However, FMD can also spread on the wind. The survival of the virus is low at relative humidity (RH) below 60% [8], and wind-borne spread is favored by the humid, cold weather common to temperate regions. In warmer drier regions, such as Africa, wind-borne spread of FMD is considered unimportant [9].
- Peste des petits ruminants (PPR) is an acute, contagious, viral disease of small ruminants, especially goats, which is of great economic importance in parts of Africa and the Near East. It is transmitted mostly by aerosol droplets between animals in close contact. However, the appearance of clinical PPR is often associated with the onset of the rainy season or dry cold periods [10], a pattern that may be related to viral survival. The closely related rinderpest virus survives best at low or high relative humidity, and least at 50–60% [11].
- Several directly transmitted human respiratory infections, including those caused by rhinoviruses (common colds) and seasonal influenza viruses (flu) have, in temperate countries, seasonal patterns linked to the annual temperature cycle. There may be direct influences of climate, such as the effect of humidity on survival of the virus in aerosol [12], or indirect influences via, for example, seasonal changes in the strength of the human immune system or more indoor crowding during cold weather [13].

Temporal-Interannual

The previous examples of spatial associations between diseases and climate, which were further categorized as temporal-seasonal, can also be classified as temporal-interannual, as the effects of climate on the intermediate hosts (snails, tsetse flies, and mosquitoes, respectively) will determine in part the risk or scale of a disease outbreak in a given year. There are other diseases where the associations can be described as seasonal-interannual.

- Anthrax is an acute infectious disease of most warm-blooded animals, including humans, with worldwide distribution. The causative bacterium, *Bacillus anthracis* forms spores able to remain infective for 10–20 years in pasture. Temperature, relative humidity, and soil moisture all affect the successful germination of anthrax spores, while heavy rainfall may stir up dormant spores. Outbreaks are often associated with alternating heavy rainfall and drought, and high temperatures [14].
- Cholera, a diarrheal disease which has killed tens of millions of people worldwide, is caused by the bacterium *Vibrio cholerae*, which lives amongst sea plankton [15]. High temperatures causing an increase in algal populations often precede cholera outbreaks. Disruption to normal rainfall helps cholera to spread further, either by flooding, leading to the contamination of water sources, such as wells, or drought which can make the use of such water sources unavoidable. Contaminated water sources then become an important source of infection in people.
- Plague is a flea-borne disease caused by the bacterium *Yersinia pestis*; the fleas' rodent hosts bring them into proximity with humans. In Central Asia, large scale fluctuations in climate synchronize the rodent population dynamics over large areas [16], allowing population density to rise over the critical threshold required for plague outbreaks to commence [17].
- African horse sickness (AHS), a lethal infectious disease of horses, is caused by a virus transmitted by *Culicoides* biting midges. Large outbreaks of AHS in the Republic of South Africa over the last 200 years are associated with the combination of drought and heavy rainfall brought by the warm phase of the El Niño Southern Oscillation (ENSO) [18].
- Rift Valley Fever (RVF), an important zoonotic viral disease of sheep and cattle, is transmitted by *Aedes* and *Culex* mosquitoes. Epizootics of RVF are associated with periods of heavy rainfall and flooding [19–21] or, in East Africa, with the combination of heavy rainfall following drought associated with ENSO [20, 22]. ENSO-related floods in 1998, following drought in 1997, led to an epidemic of RVF (and some other diseases) in the Kenya/Somalia border area and the deaths of more than 2000 people and two-thirds of all small ruminant livestock [23]. Outbreaks of several other human infections, including malaria and dengue fever have, in some parts of the world, been linked to ENSO events.

Intensity

In addition to associations between climate and the spatial and temporal distributions of disease outbreaks, there are some examples of associations between climate and the intensity or severity of the disease that results from infection. It is theoretically possible, for example, that climate-induced immunosuppression of hosts may favor the multiplication of some microparasites (viruses, bacteria, rickettsia, fungi, protozoa), thereby increasing disease severity or, alternatively, reduce the disease-associated immune response to infection, thereby reducing disease severity.

However, the clearest examples pertain to macroparasites (helminth worms) which, with the notable exception of *Strongyloides* spp., do not multiply within the host. Disease severity is therefore directly correlated with the number of parasites acquired at the point of infection or subsequently, and in turn this is frequently associated with climate, which affects both parasite survival and seasonal occurrence.

- Fasciolosis, caused by the *Fasciola* trematode fluke, is of economic importance to livestock producers in many parts of the world and also causes disease in humans. In sheep, severe pathology, including sudden death, results from acute fasciolosis which occurs after ingestion of more than 2,000 metacercariae (larval flukes) of *Fasciola hepatica* at pasture, while milder pathology associated with subacute and chronic fasciolosis occurs after ingestion of 200–1,000 metacercariae [24]. Acute fasciolosis is therefore most common in places or in years when rainfall and temperature favor the survival of large numbers of metacercariae.
- *Nematodirus battus* is a nematode parasite of the intestine of lambs. Eggs deposited in the feces of one season's lambs do not hatch straightaway; instead, they build up on the pasture during summer and remain as eggs over winter, not hatching until temperatures the following spring exceed 10°C [25]. Once the mean daily temperature exceeds this threshold the eggs hatch rapidly, leading to a sharp peak in the number of infective larvae on the pasture. If this coincides with the new season's lambs grazing on the pasture, there is likely to be a large uptake of larvae and severe disease, called nematodiriasis. If, however, there is a warm spell early in the year, the peak in larvae on pasture may occur while lambs are still suckling rather than grazing, such that fewer larvae are ingested and the severity of nematodiriasis is reduced.

Climate Change and Disease

There is a substantial scientific literature on the effects of climate change on health and disease, but with strong focus on human health and vector-borne disease [5, 26–42]. By contrast, the effects of climate change on diseases spread by other means, or animal diseases in general, have received comparatively little attention

[43–48]. Given the global burden of diseases that are not vector-borne, and the contribution made by animal diseases to poverty in the developing world [49], attention to these areas is overdue.

The previous section demonstrates the range of climate influences upon infectious disease. Such influences are not the sole preserve of vector-borne diseases: Food-borne, water-borne, and aerosol-transmitted diseases are also affected. A common feature of non-vector-borne diseases affected by climate is that the pathogen or parasite spends a period of time outside of the host, subject to environmental influence. Examples include the infective spores of anthrax; FMD viruses in temperate regions; the *Salmonella* bacteria that contaminate food products; the cholera-causing *vibrio* bacteria in water; and the moisture- and temperature-dependent survival of the parasites causing schistosomosis and fasciolosis.

By contrast, most diseases transmitted directly between humans (for example, human childhood viruses, sexually transmitted diseases (STDs), tuberculosis) have few or no reported associations with climate. This is also the case for animal infections such as avian influenza, bovine tuberculosis, brucellosis, Newcastle's disease of poultry, and rabies. Clear exceptions are the viruses that cause colds and seasonal flu in humans, and PPR in small ruminants; these viruses are spread by aerosol between individuals in close contact but are nevertheless sensitive to the effects of ambient humidity and possibly temperature.

The influence of climate on diseases that are not vector-borne appears to be most frequently associated with the timing (intra- or interannual) of their occurrence rather than their spatial distribution. There are examples of such diseases that occur only in certain parts of the world (for example, PPR) but most occur worldwide. By contrast, the associations of vector-borne diseases with climate are equally apparent in time and space, with very few vector-borne diseases being considered a risk worldwide. This is a reflection of the strong influence of climate on both the spatial and temporal distributions of intermediate vectors. If there are exceptions to this rule, the vectors are likely to be lice or fleas, with lives so intimately associated with humans or animals that they are relatively protected from climate's influences.

In the scientific literature, many processes have been proposed by which climate change might affect infectious diseases. These processes range from the clear and quantifiable to the imprecise and hypothetical. They may affect pathogens directly or indirectly, the hosts, the vectors (if there is an intermediate host), epidemiological dynamics, or the natural environment. A framework for how climate change can affect the transmission of pathogens between hosts is shown in Fig. 6.1. Only some of the processes can be expected to apply to any single infectious disease.

Effects on Pathogens

Higher temperatures resulting from climate change may increase the rate of development of certain pathogens or parasites that have one or more lifecycle stages outside their human or animal host. This may shorten generation times and,

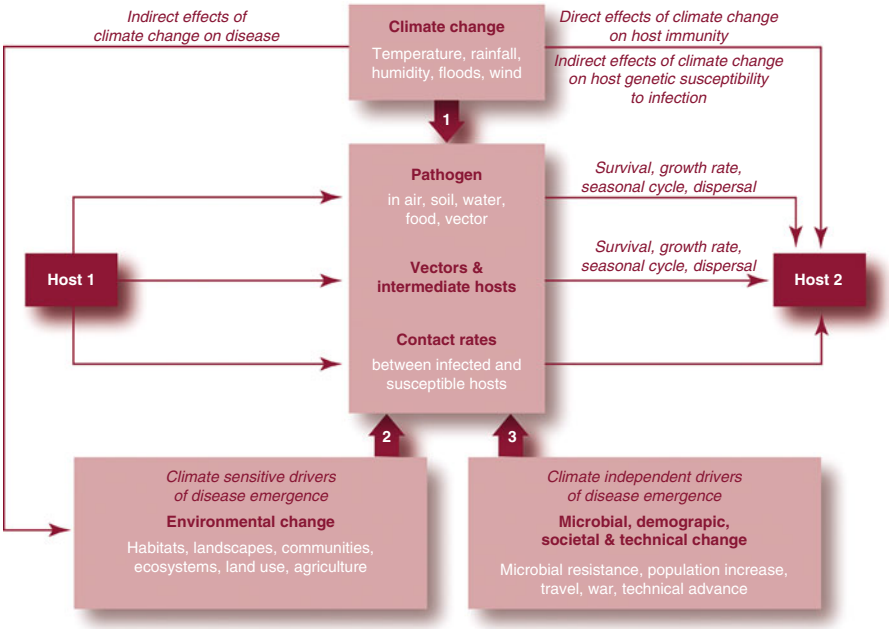

Fig. 6.1 A schematic framework of the effects of climate change on the transmission of diseases of humans and animals. Climate change can act directly on pathogens in a range of external substrates, or their vectors and intermediate hosts, thereby affecting the processes of survival, growth, seasonality, and dispersal. It can also directly affect hosts themselves or the contact rates between infected and susceptible individuals. Climate change can have indirect effects on disease transmission via its effects on the natural or anthropogenic environment, and via the genetics of exposed populations. Environmental, demographic, social, and technical change will also happen independently of climate change and have as great, or much greater, influence on disease transmission than climate change itself. The significance of climate change as a driver of disease will depend on the scale of *arrow 1*, and on the relative scales of *arrows 2* and *3*

possibly, increase the total number of generations per year, leading to higher pathogen population sizes [48]. Conversely, some pathogens are sensitive to high temperature and their survival may decrease with climate warming.

Phenological evidence indicates that spring is arriving earlier in temperate regions [50]. Lengthening of the warm season may increase or decrease the number of cycles of infection possible within 1 year for warm or cold-associated diseases respectively. Arthropod vectors tend to require warm weather so the infection season of arthropod-borne diseases may extend. Some pathogens and many vectors experience significant mortality during cold winter conditions; warmer winters may increase the likelihood of successful overwintering [41, 48].

Pathogens that are sensitive to moist or dry conditions may be affected by changes to precipitation, soil moisture, and the frequency of floods. Changes to winds could affect the spread of certain pathogens and vectors.

Effects on Hosts

A proposed explanation for the tendency for human influenza to occur in winter is that the human immune system is less competent during that time; attributable to the effects of reduced exposure to light on melatonin [51] or vitamin D production [52]. The seasonal light/dark cycle will not change with climate change, but one might hypothesize that changing levels of cloud cover could affect exposure in future. A second explanation, the tendency for people to congregate indoors during wintertime, leads to a more credible role for a future influence of climate change.

Mammalian cellular immunity can be suppressed following heightened exposure to ultraviolet B (UV-B) radiation – an expected outcome of stratospheric ozone depletion [53, 54]. In particular, there is depression of the number of T helper 1 lymphocytes, cells which stimulate macrophages to attack pathogen-infected cells and, therefore, the immune response to intracellular pathogens may be particularly affected. Examples of such intracellular pathogens include many viruses, rickettsia (such as *Cowdria* and *Anaplasma*, the causative agents of heartwater and anaplasmosis), *Brucella, Listeria monocytogenes* and *Mycobacterium tuberculosis*, the bacterial agents of brucellosis, listeriosis, and tuberculosis, respectively, and the protozoan parasites *Toxoplasma gondii* and *Leishmania* which cause toxoplasmosis and visceral leishmanosis (kala-azar), respectively, in humans [55].

A third host-related effect worthy of consideration is genetic resistance to disease. Some human populations and many animal species have evolved a level of genetic resistance to some of the diseases to which they are commonly exposed. Malaria presents a classic example for humans, with a degree of resistance to infection in African populations obtained from heterozygosity for the sickle-cell genetic trait. Considering animals, wild mammals in Africa may be infected with trypanosomes, but rarely show signs of disease; local Zebu cattle breeds, which have been in the continent for millennia, show some degree of trypanotolerance (resistance to disease caused by trypanosome infection); by contrast, recently introduced European cattle breeds are highly susceptible to trypanosomosis. In stark contrast, African mammals proved highly susceptible to rinderpest which swept through the continent in the late nineteenth century, and which they had not previously encountered. It seems unlikely that climate change will directly affect genetic or immunologic resistance to disease in humans or animals. However, significant shifts in disease distributions driven by climate change pose a greater threat than simply the exposure of new populations. Naïve populations may, in some cases, be particularly susceptible to the new diseases facing them.

Certain diseases show a phenomenon called *endemic stability*. This occurs when the severity of disease is less in younger than older individuals, when the infection is common or endemic and when there is life-long immunity after infection. Under these conditions most infected individuals are young, and experience relatively mild disease. Counter-intuitively, as endemically stable infections become rarer, a higher proportion of cases are in older individuals (it takes longer, on average, to acquire infection) and the number of cases of severe disease rises. Certain tick-borne diseases of livestock in Africa, such as anaplasmosis, babesiosis, and cowdriosis,

show a degree of endemic stability [56], and it has been proposed to occur for some human diseases like malaria [57]. If climate change drives such diseases to new areas, nonimmune individuals of all ages in these regions will be newly exposed, and outbreaks of severe disease could follow.

Effects on Vectors

Much has already been written about the effects of climate change on invertebrate disease vectors. Indeed, this issue, especially the effects on mosquito vectors, has dominated the debate so far. It is interesting to bear in mind, however, that mosquitoes are less significant as vectors of animal disease than they are of human disease (Table 6.1). Mosquitoes primarily, and secondarily lice, fleas, and ticks, transmit between them a significant proportion of important human infections. By contrast, biting midges, brachyceran flies (e.g., tabanids, muscids, myiasis flies, hippoboscids), ticks, and mosquitoes (and, in Africa, tsetse) all dominate as vectors of livestock disease. Therefore, a balanced debate on the effects of climate change on human and animal disease must consider a broad range of vectors.

There are several processes by which climate change might affect disease vectors. First, temperature and moisture frequently impose limits on their distribution. Often, low temperatures are limiting because of high winter mortality, or high temperatures because they involve excessive moisture loss. Therefore, cooler regions which were previously too cold for certain vectors may begin to allow them to flourish with climate change. Warmer regions could become even warmer and yet remain permissive for vectors if there is also increased precipitation or humidity. Conversely, these regions may become less conducive to vectors if moisture levels remain unchanged or decrease, with concomitant increase in moisture stress.

For any specific vector, however, the true outcome of climate change will be significantly more complex than that outlined above. Even with a decrease in future moisture levels, some vectors, such as certain species of mosquito, could become more abundant, at least in the vicinity of people and livestock, if the response to warming is more water storage and, thereby, the creation of new breeding sites. One of the most important vectors of the emerging Chikungunya virus (and to a lesser extent dengue virus) is the Asian tiger mosquito (*Aedes albopictus*) which is a container breeder and therefore thrives where humans store water. Equally, some vectors may be relatively insensitive to direct effects of climate change, such as muscids which breed in organic matter or debris, and myiasis flies which breed in hosts' skin.

Changes to temperature and moisture will also lead to increases or decreases in the abundance of many disease vectors. This may also result from a change in the frequency of extreme weather events such as ENSO. Outbreaks of several biting midge and mosquito-borne diseases, for example, have been linked to the occurrence of ENSO [18, 22, 59–62] and mediated, at least in part, by increase in the vector population size in response to heavy rainfall, or rainfall succeeding drought, that ENSO sometimes brings [18, 22]. Greater intra- or interannual variation in rainfall, linked or unlinked to ENSO, may lead to an increase in the frequency or scale of outbreaks of such diseases.

Table 6.1 The major diseases transmitted by arthropod vectors to humans and livestock (Adapted from [58])

Vector	Diseases of humans	Diseases of livestock
Phthiraptera (Lice)	Epidemic typhus Trench fever Louse-borne relapsing fever	
Reduvidae (Assassin bugs)	Chagas' disease	
Siphonaptera (Fleas)	Plague Murine typhus Q fever Tularaemia	Myxomatosis
Psychodidae (Sand flies)	Leishmanosis Sand fly fever	Canine leishmanosis Vesicular stomatitis
Culicidae (Mosquitoes)	Malaria Dengue Yellow fever West Nile Filiariasis Encephalitides (WEE, EEE, VEE, Japanese encephalitis, Saint Louis encephalitis) Rift Valley fever	West Nile fever Encephalitides Rift Valley fever Equine infectious anemia
Simulidae (Black flies)	Onchocercosis	Leucocytozoon (birds) Vesicular stomatitis
Ceratopogonidae (Biting midges)		Bluetongue African horse sickness Akabane Bovine ephemeral fever
Glossinidae (Tsetse flies)	Trypanosomosis	Trypanosomosis
Tabanidae (Horse flies)	Loiasis	Surra Equine infectious anemia *Trypanosoma vivax*
Muscidae (Muscid flies)	Shigella *E. coli*	Mastitis Summer mastitis Pink-eye (IBK)
Muscoidae, Oestroidae (Myiasis-causing flies)	Bot flies	Screwworm Blow fly strike Fleece rot
Hippoboscoidae (Louse flies, keds)		Numerous protozoa
Acari (Mites)	Chiggers Scrub typhus (tsutsugamushi) Scabies	Mange Scab Scrapie?
Ixodidae (Hard ticks)	Human babesiosis	Babesiosis
Argasidae (Soft ticks)	Tick-borne encephalitis Tick fevers Ehrlichiosis Q fever Lyme disease	East coast fever (Theileriosis) Louping ill African Swine Fever Ehrlichiosis Q fever

(continued)

Table 6.1 (continued)

Vector	Diseases of humans	Diseases of livestock
	Tick-borne relapsing fever Tularaemia	Heartwater Anaplasmosis Borreliosis Tularaemia

The ability of some insect vectors to become or remain infected with pathogens (their vector competence) varies with temperature [63, 64]. In addition to this effect on vector competence, an increase in temperature may alter the balance between the lifespan of an infected vector, its frequency of feeding, and the time necessary for the maturation of the pathogen within it. This balance is critical, as a key component of the risk of transmission of a vector-borne disease is the number of blood meals taken by a vector between the time it becomes infectious and its death [65]. Accordingly, rising ambient temperature can increase the risk of pathogen transmission by shortening the time until infectiousness in the vector and increasing its feeding frequency at a faster rate than it shortens the vector's lifespan, such that the number of feeds taken by an infectious vector increases. However, at even higher temperatures this can reverse [66] such that the number of infectious feeds then decreases relative to that possible at lower temperatures. This point is extremely important, as it means that the risk of transmission of vector-borne pathogens does not uniformly increase with rising temperature, but that it can become too hot and transmission rates decrease. This effect will be most important for short-lived vectors such as biting midges and mosquitoes [30].

Lastly, there may be important effects of climate change on vector dispersal, particularly if there is a change in wind patterns. Wind movements have been associated with the spread of epidemics of many *Culicoides*- and mosquito-borne diseases [67–72].

Effects on Epidemiological Dynamics

Climate change may alter transmission rates between hosts by affecting the survival of the pathogen or the intermediate vector, but also by other, indirect, forces that may be hard to predict with accuracy. Climate change may influence human demography, housing, or movement or be one of the forces that leads to changes in future patterns of international trade, local animal transportation, and farm size. All of these can alter the chances of an infected human or animal coming into contact with a susceptible one. For example, a series of droughts in East Africa between 1993 and 1997 resulted in pastoral communities moving their cattle to graze in areas normally reserved for wildlife. This resulted in cattle infected with a mild lineage of rinderpest transmitting disease both to other cattle and to susceptible wildlife, causing severe disease, for example, in buffalo, lesser kudu, and impala, and devastating certain populations [73].

Indirect Effects

No disease or vector distribution can be fully understood in terms of climate only. The supply of suitable hosts, the effects of co-infection or immunological cross-protection, the presence of other insects competing for the same food sources or breeding sites as vectors [74], and parasites and predators of vectors themselves, could have important effects [48]. Climate change may affect the abundance or distribution of hosts or the competitors/predators/parasites of vectors and influence patterns of disease in ways that cannot be predicted from the direct effects of climate change alone.

Equally, it has been argued that climate change-related disturbances of ecological relationships, driven perhaps by agricultural changes, deforestation, the construction of dams, and losses of biodiversity, could give rise to new mixtures of different species, thereby exposing hosts to novel pathogens and vectors and causing the emergence of new diseases [40]. A possible "example in progress" is the reemergence in the UK of bovine tuberculosis, for which the badger (*Meles meles*) is believed to be a carrier of the causative agent, *Mycobacterium bovis*. Farm landscape, such as the density of linear features like hedgerows, is a risk factor for the disease, affecting the rate of contact between cattle and badger [75]. Climate change will be a force for modifying future landscapes and habitats, with indirect and largely unpredictable effects on diseases.

Other Drivers of Disease

The future disease burden of humans and animals will depend not only on climate change and its direct and indirect effects on disease, but also on how other drivers of disease change over time. Even for diseases with established climate links, it may be the case that in most instances these other drivers will prove to be more important than climate. A survey of 335 events of human disease emergence between 1940 and 2004 classified the underlying causes into 12 categories [2]. One of these, "climate and weather," was only listed as the cause of ten emergence events. Six of these were non-cholera *Vibrio* bacteria which cause poisoning via shellfish or exposure to contaminated seawater; the remaining four were a fungal infection and three mosquito-borne viruses. The other 11 categories included, however, "land use changes" and "agricultural industry changes," with 36 and 31 disease emergence events, respectively, and both may be affected by climate change. The causes of the remaining 77% of disease emergence events – "antimicrobial agent use," "international travel and commerce," "human demography and behavior," "human susceptibility to infection," "medical industry change," "war and famine," "food industry changes," "breakdown of public health," and "bushmeat" – would be expected to be either less or not influenced by climate change. Hence, climate change's indirect effects on human or animal disease may exceed its direct effects, while drivers unsusceptible to climate change may be more important still at determining our disease future.

Climate Change and Disease in Wildlife

Wildlife disease is important for different reasons to those which make disease in humans and domestic animals important. It has the potential for endangerment of wildlife and can be a source of zoonoses and livestock disease.

Wildlife Disease as a Cause of Endangerment

Disease in wild populations may limit or cause extreme fluctuations in population size [76] and reduce the chances of survival of endangered or threatened species [77]. Indeed, disease can be the primary cause of extinction in animals or be a significant contributory factor toward it. For example:

- The Christmas Island rat, *Rattus macleari*, is believed to have been extinct by 1904. There is molecular evidence that this was caused by introduction of murine trypanosomes apparently brought to the island by black rats introduced shortly before 1904 [78].
- Similarly, the last known Po'o-uli bird (*Melamprosops phaeosoma*) in Hawaii died from avian malaria brought by introduced mosquitoes [79].
- Canine Distemper in the Ethiopian Wolf (*Canis simensis*) has brought about its decline [80].
- Devil facial tumor disease, an aggressive nonviral transmissible parasitic cancer, continues to threaten Tasmanian Devil (*Sarcophilus harrisii*) populations [81].
- The white nose fungus G*eomyces destructans* is decimating bat populations in Northeastern US states and is currently spreading in Europe [82]. This is perhaps the most recent emergence of a disease of concern to wildlife endangerment.

Although disease can cause endangerment and extinction, its importance relative to other causes is uncertain. A review of the causes of endangerment and extinction in the International Union for Conservation of Nature (IUCN) red list of plant and animal species found that disease was implicated in a total of 254 cases, some 7% of the total examined [83]. Although the other factors may be more important overall, disease remains an important cause of endangerment and extinction for certain animal groups. A contender for the single issue of greatest current conservation concern is the epidemic of the chytrid fungus *Batrachochytrium dendrobatidis* in amphibians. With a broad host range and high mortality, this pathogen is likely to be wholly or partly responsible for all recent amphibian extinction events, which, remarkably, comprise 29% of all extinctions attributable to disease since the year 1500 [77].

Wildlife Disease as a Source of Infections for Humans and Livestock

Many diseases of wildlife frequently cross the species barrier to infect humans or domestic animals [84, 85]. Closely related organisms often share diseases. A particular risk to humans is presented by diseases of primates: The human immunodeficiency viruses (HIV) that cause AIDS originated as simian immunodeficiency viruses in African monkeys and apes. Humans acquire or have acquired many other pathogens from mammals other than primates, especially those that humans choose to live close to (livestock, dogs, cats), or that choose to live close to us (rodents, bats) [86]. In addition, there are examples of human infections shared with birds (e.g., avian influenza), and reptiles and fishes (e.g., *Salmonella* spp.). Insects are frequent carriers of pathogens between vertebrate hosts, and there may even be a pathogen transmissible from plants to humans [87].

Wildlife populations are the primary source of emerging infectious diseases in humans. A search of the scientific literature published between 1940 and 2004 attempted to quantify the causes of disease introductions into human populations and found that about 72% were introduced from wildlife [2].

How Climate Change Can Influence Wildlife Disease

The effects of climate change on wildlife disease are important when the changes produced lead to increased risk of endangerment or extinction of the wildlife, or increased transmission risk to humans or domestic animals.

Climate change can increase the threat of endangerment or extinction, via reduction in population size of the wildlife host (by altering habitats, for example), or increase in pathogen range or virulence, such that the persistence of a host population is at risk, and climate change can increase the risk of disease emergence and spread to humans or livestock via change to the distribution of wildlife hosts, such that encroachment on human or livestock populations is favored.

Changes in species' distribution may arise directly under climate change as a result of an organism's requirement for particular climatic conditions or indirectly via ecological interactions with other species which are themselves affected. Climate change can cause the appearance of new assemblages of species and the disappearance of old communities [88], which can create new disease transmission opportunities or end existing ones.

Climatic factors potentially affecting wildlife disease transmission more directly include the growth rate of the pathogen in the environment or in a cold-blooded (ectothermic) wildlife host (e.g., fish, amphibian, reptile). Therefore, effects which are more marked in wildlife disease in comparison to human or livestock disease include the occurrence of ectothermic hosts, and also the vast range of potential

vectors that may transmit disease. It is therefore more difficult to generalize about the effects of climate change on wildlife.

In colder climates, the parasite that causes the most severe form of human malaria, *Plasmodium falciparum* does not develop rapidly enough in its mosquito vectors for there to be sufficient transmission to maintain the parasite. Avian malaria (*Plasmodium relictum*) exhibits an elevational gradient due mainly to temperature and is subject to similar constraints [89].

An example of a wildlife pathogen constrained due to its dependence on environmental transmission is anthrax. Infective spores of anthrax bacilli can lie dormant in soils for decades, becoming dangerous when climatic conditions, particularly precipitation, favor it. It is well established that when a host population size is reduced, the pool of susceptible individuals may be too small for pathogen survival. This effect is particularly acute for host-specific diseases, such as the transmissible cancer of Tasmanian devils, *Sarcophilus harrisii*, DFTD or Devil facial tumor disease (discussed in [81]); at low host population sizes, DFTD may become extinct. By contrast, diseases with a broad host range may threaten individual species down to the last individual. As anthrax has both a broad host range and can lie dormant in the environment, it is a particular threat for species with very low numbers, and is currently a conservation consideration for many species. For example the Javan rhinoceros population is down to fewer than 60 individuals and identified as a priority for conservation (an "EDGE" species) because of its uniqueness and scarcity [90].

Climate change may have particular impact on marine animals, because of the preponderance of ectothermic animals in the sea, the multiple ways in which climate change is predicted to affect the marine environment, and the multiple stresses that marine organisms and ecosystems are already experiencing due to anthropogenic influence. Disease is an important part of this impact. For example, warming of the Pacific in the range of the oyster *Crassostrea virginica* caused range expansion of the protozoan parasite *Perkinsus marinus* probably due to a combination of increased overwinter survival, greater summer proliferation, and increased host density [91]. Coral reefs are also sensitive to at least 12 potential factors associated with climate change: [92] CO_2 concentration, sea surface temperature, sea level, storm intensity, storm frequency, storm track, wave climate, run off, seasonality, overland rainfall, and ocean and air circulation [92]. Although these factors might not all increase levels of disease, the synergism between disease, climate, and other stressors might lead to accelerating degradation of the coral reef habitat.

From a geographic perspective, there is evidence that the greatest change in ecosystems attributable to climate change is likely to be in the tropics; the second being the arctic [88]. The impacts of this change on wildlife disease and its consequences may be particularly great in these two regions, and there is evidence that it is already occurring. The tropics have the most species in imminent danger of extinction [93] while tropical coral reefs comprise much of the biodiversity of the oceans. In addition to extinction risks, tropical forests may also pose a zoonosis risk. An increase in animal-human interaction is likely in tropical forests, which have a diverse fauna subject to increasing human encroachment.

With regard to the Arctic, a model of the effect of global warming on a protostrongylid lung-dwelling nematode *Umingmakstrongylus pallikuukensis*, in Canadian Arctic muskoxen *Ovibos moschatus*, found that warming was likely to significantly influence infection, making the muskoxen more susceptible to predation [94]. Muskoxen were also subject to climate-influenced outbreaks of fatal pneumonia [95]. Indeed, the combination of climate change's effects on pathogen survival and transmission, and the stress to host species from changing environmental conditions, may have serious impact on arctic populations of fish, muskoxen, sheep, and others [96].

Dependency of Disease on Climate: The Example of Chytridiomycosis in Amphibians

In wildlife epidemiology, the host may be of equal importance to the pathogen and vector when considering the impact of climate, as wildlife may be impacted by climate in more diverse ways than humans or domestic animals, and are subject to much reduced human mitigation of those impacts. The importance of climate in *Batrachochytrium dendrobatidis* epidemiology, the cause of chytridiomycosis, and numerous amphibian extinctions is fiercely debated. Although the pathogen *B. dendrobatidis* is neither vector-borne nor has a prolonged environmental phase, it is affected by temperature because the environment of its ectothermic hosts is not kept constant when external temperature varies. It belongs to a basal group within the fungi and has a brief motile zoospore stage for dispersal, followed by the penetration of the outer layers of amphibian skin and asexual intracellular multiplication [97]. Its growth is limited by warmer temperatures, perhaps because amphibians shed their outer layers of skin more frequently in warmer temperatures [97]. Pounds et al. [98] were the first to make a connection between climate and *B. dendrobatidis*-mediated amphibian extinctions, reporting spatiotemporal associations between warming and last year of detection of frog species. The development rate of the *B. dendrobatidis* fungus depends on summer temperature [99], and its survival is dependent on winter freezing [100]. The fungus appears to cause more mortality in mountainous regions [101], yet may be limited at the upper extremes of altitude. Climate may also affect the impact of the disease due to host factors. The habitat of the golden toad *Bufo periglenes* in 1987, the last year of its existence, was much reduced due to an especially dry summer. This may have caused crowded conditions in the remaining ponds, facilitating the spread of chytridiomycosis [102].

In addition, climate may affect mortality associated with the disease. The mortality of frogs exposed to *B. dendrobatidis* spores as adults has been shown to be dependent on the condition of the frogs [103]. In a changing climate where amphibians are shifting their ranges into suboptimal areas, hosts are likely to be more susceptible to the damaging effects of *B. dendrobatidis* infection.

On the other hand, it has been argued that climate is not important in *B. dendrobatidis* outbreaks [104]. The authors contrast the climate-linked epidemic hypothesis with the hypothesis that disease outbreaks occur largely due to introduction into unexposed, naive populations, and describe spatiotemporal "waves" of declines across Central America as evidence that it is disease introduction (and not climatic variables) causing declines.

Evidence of Climate Change's Impact on Disease

Climate warming has already occurred in recent decades. If diseases are sensitive to such warming, then one might expect a number of diseases to have responded by changing their distribution, frequency, or intensity. A major difficulty, however, is the attribution of any observed changes in disease occurrence to climate change because, as shown above, other disease drivers also change over time. It has been argued that the minimum standard for attribution to climate change is that there must be known biological sensitivity of a disease or vector to climate, and that the change in disease or vector (change in seasonal cycle, latitudinal or altitudinal shifts) should be statistically associated with observed change in climate [28]. This has been rephrased as the need for there to be change in both disease/vector and climate at the same time, in the same place, and in the "right" direction [105]. Given these criteria, few diseases make the standard: Indeed, only a decade ago one group concluded that the literature lacks strong evidence for an impact of climate change on even a single vector-borne disease, let alone other diseases.

This situation has changed. One disease in particular, bluetongue, has emerged dramatically in Europe over the last decade and this emergence can be attributed to recent climate change in the region. It satisfies the right time, right place, right direction criterion [64], but in fact reaches a far higher standard: A model for the disease, with variability in time driven only by variation in climate, produces quantitative estimates of risk which fit closely with the disease's recent emergence in both space and time.

Bluetongue

Bluetongue is a viral disease of sheep and cattle. It originated in Africa, where wild ruminants act as natural hosts for the virus, and where a species of biting midge, *Culicoides imicola*, is the major vector [106]. During the twentieth century bluetongue spread out of Africa into other, warm parts of the world, becoming endemic in the Americas, southern Asia, and Australasia; in most of these places, indigenous *Culicoides* became the vectors. Bluetongue also occurred very infrequently in the far extremes of southern Europe: once in the southwest (southern Spain and Portugal, 1955–1960), and every 20–30 years in the southeast (Cyprus, 1924,

1943–46); Cyprus and Greek islands close to Turkey (1977–1978); the presence of *C. imicola* was confirmed in both areas and this species was believed to be the responsible vector. Twenty years after this last 1977–1978 outbreak, in 1998 bluetongue once again reappeared in southeastern Europe [107]. Subsequent events, however, are unprecedented.

Between 1998 and 2008 bluetongue accounted for the deaths of more than one million sheep in Europe – by far the longest and largest outbreak on record. Bluetongue has occurred in many countries or regions that have never previously reported this disease or its close relatives. There have been at least two key developments. First, *C. imicola* has spread dramatically, now occurring over much of southern Europe and even mainland France. Second, indigenous European *Culicoides* species have transmitted the virus. This was first detected in the Balkans where bluetongue occurred but no *C. imicola* could be found [108]. In 2006, however, bluetongue was detected in northern Europe (The Netherlands) from where it spread to neighboring countries, the UK and even Scandinavia. The scale of this outbreak has been huge, yet the affected countries are far to the north of any known *C. imicola* territory [109].

Recently, the outputs of new, observation based, high spatial resolution (25 km) European climate data, from 1960 to 2006 have been integrated within a model for the risk of bluetongue transmission, defined by the basic reproduction ratio R_0 [110]. In this model, temporal variation in transmission risk is derived from the influence of climate (mainly temperature and rainfall) on the abundance of the vector species, and from the influence of temperature alone on the ability of the vectors to transmit the causative virus. As described earlier, this arises from the balance between vector longevity, vector feeding frequency, and the time required for the vector to become infectious. Spatial variation in transmission risk is derived from these same climate-driven influences and, additionally, differing densities of sheep and cattle. This integrated model successfully reproduces many aspects of bluetongue's distribution and occurrence, both past and present, in Western Europe, including its emergence in northwest Europe in 2006. The model gives this specific year the highest positive anomaly (relative to the long-term average) for the risk of bluetongue transmission since at least 1960, but suggests that other years were also at much higher-than-average risk. The model suggests that the risk of bluetongue transmission increased rapidly in southern Europe in the 1980s and in northern Europe in the 1990s and 2000s.

These results indicate that climate variability in space and time are sufficient to explain many aspects of bluetongue's recent past in Europe and provide the strongest evidence to date that this disease's emergence is, indeed, attributable to changes in climate. What then of the future? The same model was driven forward to 2050 using simulated climate data from regional climate models. The risk of bluetongue transmission in northwestern Europe is projected to continue increasing up to at least 2050 (Fig. 6.2). Given the continued presence of susceptible ruminant host populations, the models suggest that by 2050, transmission risk will have increased by 30% in northwest Europe relative to the 1961–1999 mean. The risk is also projected to increase in southwest Europe, but in this case only by 10% relative to the 1961–1999 mean.

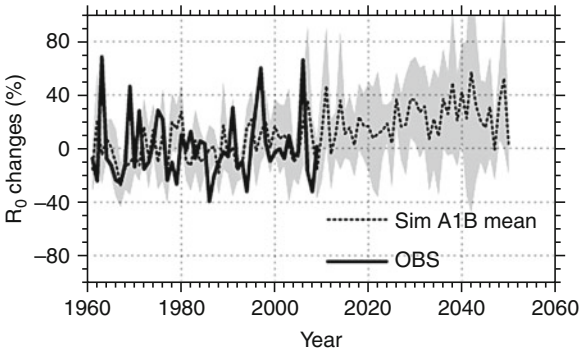

Fig. 6.2 Projections of the effect of climate change on the future risk of transmission of bluetongue in northern Europe. The *y-axis* shows relative anomalies (%) with respect to the 1961–1999 time period for the risk of bluetongue transmission, during August-October in north-west Europe, as defined by the basic reproductive ratio, R_0. R_0 was estimated from climate observations (*OBS – thick black line*), and an ensemble of 11 future climate projections (*SimA1B*), for which the *dashed line* presents the mean and the *grey* envelope the spread (Adapted from [110])

The matching of observed change in bluetongue with quantitative predictions of a climate-driven disease model provides evidence for the influence of climate change far stronger than the "same place, same time, right direction" criterion described earlier. Indeed, it probably makes bluetongue the most convincing example of a disease that is responding to climate change. In this respect, bluetongue differs remarkably from another vector-borne disease, malaria.

Malaria

Some 3.2 billion people live with the risk of malaria transmission, between 350 and 500 million clinical episodes of malaria occur each year and the disease kills at least one million people annually [111]. Of these, each year about 12 million cases and 155,000–310,000 deaths are in epidemic areas [112]. Interannual climate variability primarily drives the timing of these epidemics.

Malaria is caused by *Plasmodium* spp. parasites. Part of the parasite's life cycle takes place within anopheline mosquitoes while the remainder of the life cycle occurs within the human host. The parasite and mosquito life cycles are affected by weather and climate (mainly rain, temperature, and humidity), allowing models of the risk of malaria transmission to be driven by seasonal forecasts from ensemble prediction systems [113], thereby permitting forecasts of potential malaria outbreaks with lead times of up to 4–6 months [114, 115].

Among scientists there are contrasting views about the overall importance of climate on the transmission of malaria, and therefore on the importance of future climate change. Some argue that climate variability or change is the primary actor in any changing transmission pattern of malaria, while others suggest that any

changing patterns today or in the foreseeable future are due to non-climate factors [35, 116, 117].

A key insight is that while global temperatures have risen, there has been a net reduction in malaria in the tropics over the last 100 years and temperature or rainfall change observed so far cannot explain this reduction [118]. Malaria has moved from being climate sensitive (an increasing relationship between ambient temperature and the extent of malaria transmission) in the days before disease interventions were widely available to a situation today where regions with malaria transmission are warmer than those without, but within the malaria-affected region, warmer temperatures no longer mean more disease transmission. Instead, other variables affecting malaria, such as good housing, the running of malaria control schemes, or ready access to affordable prophylaxis, now play a greater role than temperature in determining whether there are higher or lower amounts of transmission. This would suggest that the importance of climate change in discussions of future patterns of malaria transmission is likely to have been significantly overplayed.

What is clearly recognized, by all sides in the malaria and climate debate, is that mosquitoes need water to lay their eggs in, and for larval development, and that adult mosquitoes need to live long enough in an environment with high humidity and with sufficiently high temperature for transmission to be possible to the human host. Hence, while the spatial distribution of higher versus lower degrees of malaria transmission appears to have become, in a sense, divorced from ambient temperature, it seems likely that the weather plays as important a role as ever in determining when seasonal transmission will start and end. Climate change may therefore still have a role to play in malaria: not so much affecting where it occurs but, via changing rainfall patterns and mosquito numbers, when or for how long people are most at risk.

Malaria has only recently become confined to the developing world and tropics. It is less than 40 years since malaria was eradicated in Europe and the United States; and the 15°C July isotherm was the northern limit until the mid nineteenth century [119]. Changes in land use and increased living standards, in particular, acted to reduce exposure to the mosquito vector in these temperate zones, leading ultimately to the final removal of the disease. In the UK, a proportion of the reduction has been attributed to increasing cattle numbers and the removal of marshland [120]. In Finland, changes in family size, improvements in housing, changes in farming practices, and the movement of farmsteads out of villages lead to the disappearance of malaria [121], where it had formerly been transmitted indoors in winter. While future increases in temperature may, theoretically, lead to an increased risk of malaria transmission in colder climes than at present [120, 122], the much-altered physical and natural environment may preclude this risk increasing to a level that merits concern. Once again, a more important future driver of malaria risk, in the UK at least, may be the pressure to return some of our landscape to its former state, such as the reflooding of previously drained marshland.

Future Directions

Climate change is widely considered to be a major threat to human and animal health, and the viability of certain endangered species, via its effects on infectious diseases. How realistic is this threat? Will most diseases respond to climate change, or just a few? Will there be a net increase in disease burden or might as many diseases decline in impact as increase?

The answers to these questions are important, as they could provide opportunities to mitigate against new disease threats, or may provide the knowledge-base required for policy makers to take necessary action to combat climate change itself. However, both the methodology to accurately predict climate change's effects on diseases and, in most cases, the data to apply the methodology to a sufficiently wide-range of pathogens is currently lacking.

The majority of pathogens, particularly those not reliant on intermediate hosts or arthropod vectors for transmission, either do occur, or have the potential to occur, in most parts of the world already. Climate change has the capacity to affect the frequency or scale of outbreaks of these diseases: Good examples would be the frequency of food poisoning events from the consumption of meat (such as salmonellosis) or shellfish (caused by *Vibrio* bacteria).

Vector-borne diseases are usually constrained in space by the climatic needs of their vectors, and such diseases are therefore the prime examples of where climate change might be expected to cause distributional shifts. Warmer temperatures usually favor the spread of vectors to previously colder environments, thereby exposing possibly naïve populations to new diseases.

However, altered rainfall distributions have an important role to play. Many pathogens or parasites, such as those of anthrax, haemonchosis, and numerous vector-borne diseases, may in some regions be subject to opposing forces of higher temperatures promoting pathogen or vector development, and increased summer dryness leading to more pathogen or vector mortality. Theoretically, increased dryness could lead to a declining risk of certain diseases. A good example is fasciolosis, where the lymnaeid snail hosts of the *Fasciola* trematode are particularly dependent on moisture. Less summer rainfall and reduced soil moisture may reduce the permissiveness of some parts of the UK for this disease. The snail and the free-living fluke stages are, nevertheless, also favored by warmer temperatures and, in practice, current evidence is that fasciolosis is spreading in the UK [123].

One way to predict the future for disease in a specific country is to learn from countries that, today, are projected to have that country's future climate [37, 39]. At least some of the complexity behind the multivariate nature of disease distributions should have precipitated out into the panel of diseases that these countries currently face.

For example, in broad terms, the UK's climate is predicted to get warmer, with drier summers and wetter winters, becoming therefore increasingly "mediterranean." It would seem reasonable, therefore, to predict that the UK of the future might experience diseases currently present in, or that occur periodically in, southern Europe. For humans, the best example would be leishmanosis (cutaneous and

visceral) [124], while for animals, examples include West Nile fever [125], *Culicoides imicola*–transmitted bluetongue and African horse sickness [41], and canine leishmanosis [126]. The phlebotomid sandfly vectors of the latter do not currently occur in the UK, but they are found widely in southern continental Europe, including France, with recent reports of their detection in Belgium [127]. The spread of the Asian tiger mosquito into Europe and the recent transmission in Europe of both dengue fever [128] and Chikungunya [129] by this vector are further cause for alarm.

However, the contrasting examples of bluetongue and malaria – one spreading because of climate change and one retreating despite it – show that considerations which focus entirely on climate may well turn out to be false. Why are these two diseases, both vector-borne and subject to the similar epidemiological processes and temperature dependencies, so different with respect to climate change? The answer lies in the relative importance of other disease drivers. For bluetongue, it is difficult to envisage epidemiologically relevant drivers of disease transmission, other than climate, that have changed significantly over the time period of the disease's emergence [64]. Life on the farm for the midges that spread bluetongue is probably not dramatically different today from the life they enjoyed 30 years ago. Admittedly, changes in the trade of animals or other goods may have been important drivers of the increased risk of introduction of the causative viruses into Europe, but after introduction, climate change may be the most important driver of increased risk of spread.

For malaria, change in drivers other than climate, such as land use and housing, the availability of prophylaxis, insecticides and, nowadays, insecticide-treated bed nets, have played far more dominant roles in reducing malaria occurrence than climate change may have played in increasing it. Two key reasons, then, for the difference between the two diseases are, first, that life for the human hosts of malaria has changed more rapidly than that of the ruminant hosts of bluetongue, and second, the human cost of malaria was so great that interventions were developed; while the (previously small) economic burden of bluetongue did not warrant such effort and our ability to combat the disease 5 years ago was not very different from that of 50 years before. The very recent advent of novel inactivated vaccines for bluetongue may now be changing this situation.

This entry began by asking whether climate change will affect most diseases or just a few. The examples of malaria and bluetongue demonstrate that a better question may be as follows: Of those diseases that are sensitive to climate change, how many are relatively free from the effects of other disease drivers such that the pressures brought by a changing climate can be turned into outcomes?

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