

Foresight

Infectious Diseases: preparing for the future

OFFICE OF SCIENCE AND INNOVATION

**T7.3: The Effects of Climate Change on
Infectious Diseases of Animals**

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Contents:

1 Introduction	3
2 Climate and animal diseases	4
3 Climate change and animal diseases	6
4 Climate change scenarios.....	7
4.1 The future climate of the UK	7
4.2 The future climate of Africa	7
5 How climate change affects animal diseases.....	8
5.1 Effects on pathogens	8
5.2 Effects on hosts	9
5.3 Effects on vectors	9
5.4 Effects on epidemiology.....	11
5.5 Other indirect effects.....	11
6 Evidence of climate change affecting animal disease.....	12
6.1 The emergence of bluetongue	12
7 Prospects for the future.....	13
7.1 The UK.....	13
7.2 Africa.....	14
8 Conclusions	16
Acknowledgements.....	17
References	18

1 Introduction

The impact of infectious animal diseases seems as great at the start of the 21st Century as it was at the start of the 20th. In the developing world, these diseases continue to limit productivity, constrain development and exacerbate poverty (Perry, 2002). In the developed world, infectious animal diseases still make animals suffer, harm the environment and cause financial loss. Worldwide, humans suffer a range of diseases originating from wild or domesticated animals. Topical examples include bovine spongiform encephalopathy (BSE), avian influenza, bovine tuberculosis, severe acute respiratory syndrome (SARS) and West Nile fever.

What will be the global impact of infectious animal diseases at the end of the 21st century? Any single disease is likely to be affected by many factors that cannot be predicted with confidence, including changes to livestock management practices, changes to the physical environment, developments in animal genetics, and new scientific or technological advances. 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 (Intergovernmental Panel on Climate Change, 2001), with concomitant changes in precipitation, flooding, winds and the frequency of extreme events such as El Niño. Innumerable studies have demonstrated links between infectious animal diseases and climate, and it is unthinkable that a significant change in climate during this century will not impact significantly on at least some of them.

How should we react to predicted changes in animal diseases ascribed to climate change? The answer depends on the animal populations and human communities affected, whether the disease changes in incidence or spatiotemporal distribution and, of course, on the direction of change. It also depends on the relative impact of the disease. If climate change is predicted to affect only diseases of relatively minor impact, then our concerns should be tempered.

With that in mind, this review focuses on the possible effects of climate change, by the 2020s and the 2080s, on the diseases that 'matter most'. We consider the effects of climate change both in the developed world, focusing on the UK, and in the developing world, focusing on Africa. For our purposes, diseases that matter most to the UK are notifiable diseases to the Department for Food and Rural Affairs, which in the main are exotic to the UK (<http://www.defra.gov.uk/animalh/diseases/notifiable/index.htm>)

and non-notifiable endemic diseases that cause the greatest economic losses (Bennett et al., 1999; Bennett and Ijpeelaar, 2005). Diseases that matter most in Africa are diseases endemic to the continent that impact most on the poor (Perry, 2002). These diseases are listed in Table 1. By virtue of these definitions, this review focuses on diseases of terrestrial livestock more than on diseases of wildlife (including birds) or invertebrates (including shellfish).

The possible impact of climate change on diseases of wildlife and invertebrates is reviewed elsewhere (Harvell et al., 2002).

2 Climate and animal diseases

Many important animal diseases are affected directly or indirectly by weather and climate. These links may be *spatial*, with climate affecting distribution, *temporal* with weather affecting the timing of an outbreak, or relate to the *intensity* of an outbreak. Here we present a selection of these associations, which is by no means exhaustive but is, rather, intended to demonstrate the diversity of effects.

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 (Parker et al., 2002). Blackleg, an acute infectious clostridial disease, mostly of young cattle, is also spore-forming, and disease outbreaks are associated with high temperature and heavy rainfall. (Hall, 1988)

Certain bacteria, such as *Dermatophilus congolensis*, the causative agent of dermatophilosis, and *Pasteurella multocida* that causes haemorrhagic septicaemia (pasteurellosis) in bovines, survive well outside the host in moist environments. Both diseases are associated with areas of high humidity and occur during rainy seasons (Hall, 1988).

Foot-and-mouth disease is a highly contagious, viral infection of cloven-footed animals, including cattle, sheep and pigs. It is a major threat to the UK's livestock and of considerable economic importance to Africa. 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 (Figure 1). The survival of the virus is low at relative humidity below 60% (Donaldson, 1972), and wind-borne spread is favoured by the humid, cold weather common to the UK. In warmer drier regions, such as Africa, wind-borne spread of FMD is considered unimportant (Sutmoller et al., 2003).

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 (Figure 2) or dry cold periods (Wosu et al., 1992), a pattern that may be related to viral survival. The closely related rinderpest virus survives best at low or high humidity, and least at 50-60% RH (Anderson, 1996).

Haemonchosis - infection with the nematode *Haemonchus contortus* - occurs worldwide in the guts of sheep and cattle. It can cause significant economic loss in terms of reduced productivity or, with heavy infestations, mortality. Eggs are excreted in droppings. Survival of the eggs and larvae, until they are ingested by another animal, depends on temperature and moisture: under appropriate conditions of warmth and moderate humidity, the larvae can survive for weeks or months.

Fascioliasis, caused by the *Fasciola* trematode fluke, is of economic importance to cattle and sheep producers in many parts of the world. The disease is a particular problem where environmental conditions favour the intermediate host, lymnaeid snails. These conditions include low lying wet pasture, areas subject to periodic flooding, and temporary or permanent bodies of water (Hall, 1988).

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) (Baylis et al., 1999) (Figure 3).

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 (Davies et al., 1985; Linthicum et al., 1987; Linthicum et al., 1999) or, in east Africa, with the combination of heavy rainfall following drought associated with ENSO (Anyamba et al., 2002; Linthicum et al., 1999). 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, causing the deaths of more than 2000 people and two-thirds of all small ruminant livestock (Little et al., 2001).

Diseases transmitted by tsetse flies (trypanosomiasis) and ticks (such as anaplasmosis, babesiosis, East Coast fever, heartwater) impose a tremendous burden on African livestock (Figure 4). Ticks, as ectoparasites, are a further direct burden. Many aspects of the vectors' life cycles are sensitive to climate, and spatial distributions can be predicted using satellite-derived proxies for climate variables (Rogers and Randolph, 1993).

Between 1998 and 2005, 1.5 - 2 million sheep died in Europe from bluetongue, a viral infection of ruminants transmitted by *Culicoides* biting midges (Purse et al., 2005). Vector distributions are largely dependent on environmental variables such as temperature, moisture and wind (Mellor et al., 2000). Both bluetongue and African horse sickness can be transported over hundreds of kilometres of sea by winds carrying virus-infected *Culicoides* vectors.

Mosquitoes (principally *Culex* and *Aedes*) transmit several bird viruses which 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) (Weaver and Barrett,

2004). The spatial and temporal distributions of the mosquito vectors are highly sensitive to climate variables.

The time period between a vector feeding on an infected host and being able to transmit the infection onward to a susceptible host is called the extrinsic incubation period (EIP). The EIP lengthens at lower temperatures. In colder areas, some short-lived vectors, such as mosquitoes and biting midges, tend to die before the EIP is complete and transmission does not occur (Lines, 1995; Reeves et al., 1994).

The susceptibility of some insect vectors to arboviruses is linked to temperature. Adult *Culicoides nubeculosus*, a UK biting midge, is refractory to infection with bluetongue virus when reared as a juvenile at temperatures below 30 °C, but is a competent vector when reared at higher temperatures (Wittmann and Baylis, 2000) (Figure 5). By contrast, *Culex tarsalis* can suppress infection with WEE virus when maintained as an adult at higher temperatures (Reeves et al., 1994).

3 Climate change and animal diseases

There is a substantial scientific literature on the effects of climate change on health and disease, but it has a strong focus on human health and vector-borne disease (Githeko et al., 2000; Hay et al., 2002; Kovats et al., 1999; Kovats et al., 2001; Lines, 1995; McMichael and Githeko, 2001; Patz and Kovats, 2002; Randolph, 2004; Reeves et al., 1994; Reiter et al., 2004; Rogers and Randolph, 1993, 2000; Rogers et al., 2001; Sutherst, 1998; WHO, 1996; Wittmann and Baylis, 2000; Zell, 2004). By contrast, the effects of climate change on animal- or non-vector-borne disease has received comparatively little attention (but with notable exceptions: Cook, 1992; Harvell et al., 1999; Harvell et al., 2002). Given the global burden of diseases that are not vector-borne, and the contribution made by animal diseases to poverty in the developing world (Perry, 2002), attention to these areas is overdue.

Section 2 shows clearly that many animal diseases of significant impact in the UK and Africa, are influenced by climate. Such influences are not the sole preserve of vector-borne diseases. Indeed, certain directly transmitted, food/waterborne 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 and blackleg; the wind-borne aerosol droplets that spread FMD virus in temperate regions; and the moisture-dependent survival of the agents of dermatophilosis, haemorrhagic septicaemia and haemonchosis.

By contrast, diseases transmitted directly between animals in close contact have few reported associations with climate. Examples here include avian influenza, bovine tuberculosis, brucellosis, BVD, CSF, CBPP, CCPP, FMD in Africa, Newcastle's disease, coccidiosis, rabies, rinderpest, salmonellosis, scrapie and SVD.

Additionally, climate appears to be more frequently associated with the seasonal occurrence of non-vector borne animal diseases than their spatial distribution. By contrast, the associations of vector-borne diseases with climate are equally apparent in time and space - a reflection of the strong influence of climate on both the spatial and temporal distributions of the intermediate vectors.

4 Climate change scenarios

4.1 The future climate of the UK

The UKCIP (2002) report (Hulme, 2002) predicts that the UK climate will warm by 0.5 to 1.5 °C by the 2020s, relative to a 1961-1990 baseline, with greater warming in summer than winter. One year in every three or four will be very warm, and one in a hundred will have an extremely hot summer. Night-time temperatures will rise more than daytime temperatures. Summer rainfall will decrease by up to 10% while winter rainfall will increase by the same amount, with little net change overall. One year in ten will have a very dry summer and one in a hundred will have a very wet winter, with a concomitant increase in the risk of flooding.

Changes are predicted to be more extreme by the 2080s. The mean annual temperature will rise by 2 – 4 °C (greater again in summer), relative to the 1961-1990 baseline. All years will be very warm and nearly two thirds will have extremely hot summers. Summer rain will decrease by 35-50% and winter precipitation will increase by 10-35%, a slight net decrease annually. Half of all years will have very dry summers; one in fifteen will have very wet winters. Relative humidity is predicted to decrease by 10% and soil moisture by 20-40% during summer throughout the 2080s. It may be windier during winter.

4.2 The future climate of Africa

The African climate is far more diverse than that of the UK, ranging as it does from snow-capped mountains to deserts. Major influences are the surrounding oceans and Mediterranean Sea, different types of land cover, lakes, and varied topography. The Intertropical Convergence Zone (ITCZ) primarily controls the rainy seasons. Mean annual rainfall ranges from 10 mm in parts of the Sahara desert to >2,000 mm in the tropical regions and other parts of west Africa. Inter-annual variability in rainfall depends on sea surface temperatures (SST), atmospheric winds, ENSO, and regional climate fluctuations in the Indian and Atlantic Oceans.

Africa is generally warm, with mean daily temperatures >25°C. However, the extreme north and south, and areas of higher altitude, experience some cooler weather (mean daily temperature <20°C).

Continent-wide changes in temperature and rainfall in Africa, for the period 2020-2080, have been modelled (Hulme et al., 2001) based on low or high carbon-emission scenarios. The predictions are that, by the 2020s, Africa will

warm by 0.6 – 2.4 °C, relative to the 1961-1990 baseline, and, by 2080, by 1.2 - 7 °C. Temperature will rise most in West and central southern Africa, and least in east Africa.

Rainfall predictions are somewhat patchy and variable. In the broadest terms, central Africa and southern west Africa will become wetter in both the December-February (DJF) and June-August (JJA) seasons, while north and southern Africa will be drier in both seasons. east Africa and the sahelian west Africa will become wetter in DJF but drier in JJA.

Some global climate models predict that ENSO will become more frequent as a result of climate change. However, current models are rather poor at describing the effects of ENSO in Africa today (Hulme et al., 2001) so their predictive power may be limited.

5 How climate change affects animal diseases

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/parasites directly or indirectly, the hosts, the vectors (if there is an intermediate host), epidemiological dynamics or the natural environment. Only some of these processes can be expected to apply to any single infectious disease.

5.1 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 life cycle stages outside their animal host. This may shorten generation times and, possibly, increase the total number of generations per year, leading to higher pathogen/parasite population sizes (Harvell et al., 2002). Conversely, some pathogens are sensitive to high temperatures and their survival may decrease with climate warming.

Phenological evidence indicates that spring is arriving earlier in temperate regions (Walther et al., 2002). Lengthening of the warm season may increase or decrease the number of cycles of infection possible within one 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/parasites and many vectors experience significant mortality during cold winter conditions; warmer winters may increase the likelihood of successful overwintering (Harvell et al., 2002; Wittmann and Baylis, 2000).

Pathogens and parasites 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.

5.2 Effects on hosts

Mammalian cellular immunity can be suppressed following heightened exposure to ultraviolet B (UV-B) radiation - an expected outcome of stratospheric ozone depletion (Aucamp, 2003; de Gruijl et al., 2003). In particular, there is depression of the number of T helper 1 lymphocytes, the cells involved in the immune response to intracellular pathogens. In terms of animal disease, such pathogens include viruses, rickettsia (such as *Cowdria* and *Anaplasma*, the causative agents of heartwater and anaplasmosis) and some bacteria, such as *Brucella*, the organism causing brucellosis. Furthermore, increased UV-B exposure may diminish the host's response to certain vaccinations (de Gruijl et al., 2003). Continued ozone depletion could possibly impact on certain animal diseases, but it should be borne in mind that the association of UV-B with immunity has only been investigated in humans and mice. Diurnal animals that live outdoors may be less susceptible to heightened UV-B exposure.

A second host-related effect worthy of consideration is genetic resistance to disease. Many animals have evolved a level of genetic resistance to some of the diseases to which they are commonly exposed. For example, 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) whereas recently introduced European cattle breeds are highly susceptible to trypanosomiasis. In stark contrast, African mammals proved highly susceptible to rinderpest which swept through the continent in the late 19th century, and which they had not previously encountered. It seems unlikely that climate change will directly affect genetic or immunologic resistance to disease in livestock. However, significant shifts in disease distributions driven by climate change pose a greater threat than simply that of 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 disease is less severe in younger than older individuals, when the infection is common or endemic and when there is lifelong 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 (Eisler et al., 2003). If climate change drives such diseases to new areas, non-immune individuals of all ages in these regions will be newly exposed, and outbreaks of severe disease could follow.

5.3 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. Remember, though, that

mosquitoes are less significant as vectors of animal disease than they are of human disease (Table 2). Mosquitoes primarily, and secondarily lice, fleas and ticks, transmit 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 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 and a relatively slow rate of population recovery during warmer seasons. By contrast, high temperatures are limiting 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. 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 (Anyamba et al., 2002; Baylis et al., 1999; Gagnon et al., 2001; Gagnon et al., 2002; Hales et al., 1999; Kovats, 2000). They have been 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 (Anyamba et al., 2002; Baylis et al., 1999). Greater intra- or inter-annual variation in rainfall, linked or unlinked to ENSO, may lead to an increase in the frequency or scale of outbreaks of such diseases.

The ability of some insect vectors to become or remain infected with viruses varies with temperature. In addition to this effect on vector competence, an increase in temperature may alter the balance between lifespan and the EIP, increasing or decreasing the proportion of infected vectors that live long enough to transmit the infection onward. This effect will be most important for short-lived vectors such as biting midges and mosquitoes (Lines, 1995). In one example, the likelihood of bluetongue virus transmission by *Culicoides sonorensis* increases as temperature rises to 27 – 30 °C, because the

reduction in mean longevity is outweighed by the shortening of the EIP (Wittmann and Baylis, 2000).

The feeding frequency of arthropod vectors may also increase with rises in temperature. Many vectors must feed twice on suitable hosts before transmission is possible - once to acquire the infection and, after the EIP, once to transmit it. For many blood-feeding arthropods, feeding frequency is determined by the time required for egg development. For example, *C. sonorensis* females feed every three days at 30 °C but only every ~14 days at 13 °C (Wittmann and Baylis, 2000). At the warmer temperature, the vector is more likely to take the two feeds on suitable hosts that are required for successful transmission.

Transovially-infectable vectors need feed only once for successful transmission. Nevertheless, a higher feeding frequency may increase the number of feeds taken and thereby increase the likelihood of transmission.

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 (Sellers et al., 1982; Sellers and Pedgley, 1985; Sellers and Maarouf, 1990, 1991, 1993).

5.4 Effects on epidemiology

Climate change may alter transmission rates between hosts by affecting the survival of the pathogen/parasite or the intermediate vector, but also by other, indirect, forces that may be hard to predict with accuracy. Climate change may be one of the forces that leads to changes in future patterns of international trade, local animal transportation and farm size - all of which may affect the chances of an infected 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 (Kock et al., 1999).

5.5 Other 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 (Davis et al., 1998), and parasites and predators of vectors themselves, could have important effects (Harvell et al., 2002). 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 loss 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 (WHO, 1996). A possible 'example in progress' is the re-emergence 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 etc., is a risk factor for the disease, affecting the rate of contact between cattle and badger (White et al., 1993). Climate change will be a force for modifying future landscapes and habitats, with indirect and largely unpredictable effects on diseases.

6 Evidence of climate change affecting animal disease

Climate warming has already occurred in recent decades. If diseases, including animal diseases, are sensitive to such warming, then we should already have witnessed dramatic changes to certain infections. The standard for linking disease change to climate change has been set: there must be change in both at the same time, in the same place, and in the 'right' direction (Rogers and Randolph, 2003). To date, evidence of this nature is remarkably scarce, but it is beginning to appear.

6.1 The emergence of bluetongue

In 1998, bluetongue was reported in Europe for the first time in 20 years, following the detection of infected sheep on some Greek islands close to Turkey. Since the disease was first recorded in Europe 75 years ago, there have been several outbreaks in the south east of the Mediterranean region and one in the south west. No other parts of Europe experienced this or other *Culicoides*-borne arboviral disease. Before 1998, these regions were also the only parts of Europe where the primary Old World vector of bluetongue, *Culicoides imicola*, was detected.

Between 1998 and 2005 bluetongue has accounted for the deaths of more than 1.5 million sheep in Europe making it the longest and largest outbreak on record. Five serotypes of bluetongue virus (BTV) have invaded Europe. Disease has occurred in numerous countries or regions that have never previously reported any *Culicoides*-borne arboviral disease, namely: European Turkey; mainland Greece; Bulgaria; several Balkan countries; mainland Italy; Sicily and Sardinia; the French island of Corsica; the Balearic islands; and Tunisia.

There have been at least two key developments. First, there was transmission of BTV in the Balkans by novel *Culicoides* vectors, probably of the *C. obsoletus* and *C. pulicaris* complexes. Second, *C. imicola* has spread dramatically, being newly found in affected areas of mainland Greece, mainland Italy, Sicily, Sardinia, Corsica and the Balearic islands, and several areas that have not yet experienced bluetongue such as north-eastern Spain, southern continental France and northern Italy (Figure 6). The vector's range

has extended significantly northward. However, there is no evidence of spread of *C. imicola* in western Iberia. Extensive trapping in the early and late 1990s detected no change.

These events have now been linked to recent climate warming in Europe (Purse et al., 2005). A mean annual minimum temperature surface map of Europe for the 1980s was subtracted from the equivalent surface map for the 1990s, to create a 'difference map', showing which areas have warmed or cooled most during the period in which bluetongue began to emerge (Figure 7). The results indicate significant warming in the very parts of Europe which have seen the invasion of *C. imicola* or transmission by novel vectors. By contrast, the region where no detectable change in bluetongue vectors has taken place - western Iberia - has not warmed. The changes in bluetongue and surface temperature in Europe, concurrent in time and space, and in the 'right' direction suggest that there is a direct, causal link between the emergence of bluetongue in Europe and climate warming.

7 Prospects for the future

7.1 The UK

One way to predict the future for animal disease in the UK is to learn from countries that, today, have the UK's future climate (Rogers et al., 2001; Sutherst, 1998). 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.

In broad terms, the UK's climate is predicted to get warmer, with drier summers and wetter winters. In other words, it will become increasingly 'Mediterranean'. This is shown clearly in Figure 8. Under the HadCM2 medium-high scenario, by the 2020s, the climate of much of England is predicted to be that experienced today in the northern half of France, while the south west of the UK is predicted to be similar to that of today's Italy and southern France. Under the same scenario, by the 2080s, the climate of southern England is predicted to be similar to that of Sicily today. The recent discovery of *Culicoides imicola* in southern France and Italy indicates that the future, warmer UK could be successfully invaded by this species, thereby putting the UK at significant risk of bluetongue and AHS.

The risk of transmission of West Nile virus, which has occurred in recent years in both Italy and the south of France, will also increase. Phlebotomous sandflies, vectors of canine leishmaniasis, do not currently occur in the UK but they are found widely in southern continental Europe, including France, and have recently been detected in Belgium (Depaquit, 2005).

The risk of Rift Valley Fever will rise but, as the disease does not yet occur in Europe, the UK may not be directly threatened in the near future.

Many pathogens or parasites, such as those of anthrax, haemonchosis and summer mastitis, may be subject to the opposing forces of higher

temperatures promoting pathogen or vector development, and increased summer dryness leading to more pathogen or vector mortality. For such diseases, it is not possible to predict the direction of future change in risk, incidence or distribution.

The risk of certain diseases may decline. Warmer air temperatures may reduce the likelihood of the wind-borne spread of foot-and-mouth disease. 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 fascioliasis. However, where moisture levels are maintained by natural or artificial means, higher temperatures may lead to an increased challenge from the disease.

Finally, many of the livestock diseases deemed most important to the UK may be little affected, or totally unaffected, by climate change by the 2020s, and even the 2080s. These are diseases generally transmitted by close contact between hosts, or by other routes such as food, biting etc. Examples include avian influenza, brucellosis, BSE, CSF, most transmission of FMD, mastitis, Newcastle's disease, rabies and the salmonellosis.

7.2 Africa

Africa is expected to get warmer, with some regions predicted to become drier, some wetter and others more seasonally variable. Livestock production in most of sub-Saharan Africa is determined by moisture (FAO, 1978), and these changes to the continent's climate could have dramatic effects on the distribution of animals themselves, in addition to patterns of infectious diseases.

Warming and changes in rainfall distribution in Africa will lead to changes in the spatial or temporal distributions of those diseases sensitive to moisture, such as anthrax, blackleg, dermatophilosis, haemorrhagic septicaemia, PPR, haemonchosis and vector-borne diseases. These diseases, as is clearly shown by climate-driven models of disease vectors in Africa, may decline in some areas and spread to others.

Tsetse-transmitted trypanosomiasis is one of the greatest disease constraints on livestock production in a large part of sub-Saharan Africa. The many species of tsetse, each with its own climatic requirements, are classified into three groups: savannah (*morsitans* group), riverine (*fusca* group) and forest (*palpalis* group). The probability of the presence/absence of each group in grid cells across sub-Saharan Africa has been modelled in terms of the current Length of the Growing Period (LGP), a composite of temperature and moisture (McDermott et al., 2001). Threshold values of LGP for a group's presence or absence were found, and future distributions of habitat suitability for each group were predicted for future LGP scenarios. By 2050, climate change-driven decline in habitat suitability is predicted for all three groups in parts of their northern and southern fronts, together with an increase in habitat suitability in parts of east Africa. Additionally, there is predicted to be an increase in habitat suitability for the *morsitans* group along its southern front in west Africa.

A model of the current southern African distribution of a single tsetse species, *Glossina morsitans*, based on a number of climate variables, was used to predict future distributions of the species, again for the 2050s (Rogers, 1996). A net decline in habitat suitability for this species in southern Africa was predicted, but with localized gains in some, particularly highland, areas.

Ticks transmit many important livestock diseases in Africa and, like tsetse, impose significant constraints on productivity. A model similar to that for *Glossina morsitans* was developed for the brown-ear tick, *Rhipicephalus appendiculatus*, the primary vector of ECF in Eastern and southern Africa (Rogers, 1996). By the 2050s, suitable habitat is predicted to have largely disappeared from the south-eastern part of its range (south-eastern Zimbabwe and southern Mozambique). On the other hand, newly permissive areas appear in more western and central parts of southern Africa.

These modelling exercises lend quantitative support - and spatiotemporal detail - to our intuitions. Moisture-stress, exacerbated by high temperature, limits the distribution of many disease vectors in Africa. A general decline in habitat suitability for disease vectors may be witnessed in the hotter and drier southern Africa of the future, but with a possible increase seen in highland areas which are currently too cold. Distributional limits may be relaxed in east Africa and sahelian west Africa, as increased rain falls during the current DJF hot season, and in central Africa and southern west Africa which are predicted to get more rainfall during two seasons.

Similar predictions might be offered for all moisture-limited disease agents, but with important complexities. Fascioliasis in Africa is caused by two species, *Fasciola hepatica* (transmitted primarily by the lymnaeid snail vector, *Galba truncatula*) and *F. gigantica* (transmitted primarily by the lymnaeid *Radix natalensis*). *Galba truncatula* prefers lower temperatures and small, temporary water bodies, while *R. natalensis* prefers higher temperatures and large, deep, permanent water bodies (Mas-Coma, 2004). Fascioliasis hepatica may increase in areas of Africa that experience more rainfall. Both snails (and parasites) might be expected to decline in areas that become hotter and drier. However, in such regions, fascioliasis gigantica could, perhaps, increase if more dams or reservoirs are constructed to help maintain the water supply.

After drought in 1997, the 1998 ENSO brought excessive rainfall to east Africa which led to an 'explosion' of many diseases, notably RVF and bluetongue, transmitted by mosquitoes and biting midges respectively. The combination of drought and rainfall, brought by ENSO, has also been linked to large epidemics of African horse sickness in the Republic of South Africa. Some global climate models predict that ENSO will occur more frequently in future. Even discounting ENSO, it is widely predicted that climate extremes -drought and floods - will become more frequent. It is possible, therefore, that significant epizootics of certain vector-borne diseases will become more common in parts of Africa.

Finally, the east African droughts in the 1990s that led, via the translocation of pastoralist cattle herds, to a rinderpest epidemic in wildlife provide an

extremely important lesson: climate change-driven alterations to livestock husbandry in Africa, if they occur, could have many indirect and unpredictable impacts on infectious animal disease in the continent.

8 Conclusions

*Oh, happy he who still can hope in our day
to breathe the truth while plunged in seas of error!
What we don't know is really what we need,
and what we know is of no use whatever!*

(Faust lamenting the difficulty of halting the plague: from Goethe's *Faust*).

What we do know is that the spatiotemporal distributions and incidences of many animal diseases are associated with climate, and that some of these infections at least will respond to climate change. The most responsive diseases will be those where the causative agent spends a period of time outside an animal host, exposed to environmental influence, the prime example being vector-borne diseases.

What we do not know, at least with confidence, is what the responses will be. Climate change will affect diseases through forces operating on the pathogen, the host, the vector, epidemiological processes and other, indirect routes. Other forces will also be at play. The number and variety of diseases, our poor understanding of many of them, the number of climate change (and other) processes affecting them, the uncertainties in climate change predictions, and the spatial heterogeneity of these predictions, all combine to make attempts to predict our disease future bewilderingly complex.

The high rainfall and wealth of the UK probably ensure that livestock production will, as far ahead as the 2080s, be similar to now, even if the spectrum of animal diseases changes. This, combined with the country's small size, makes predicting the future simpler than for Africa. The UK climate is predicted to become increasingly 'Mediterranean', so animals in the UK may become threatened by diseases currently facing their Mediterranean counterparts. Bluetongue, African horse sickness, West Nile fever and canine leishmaniasis are among the most obvious new threats. There may be a decline in certain, highly moisture-dependent diseases, while some of the most important infectious diseases of animals in the UK may be little affected, or quite unaffected, by predicted climate change this century.

Livestock production in Africa is already moisture-limited. Regional increases or decreases in rainfall may be expected to have fundamental effects on this sector, and disease models based on climate variables alone are likely to be over-simplified. Models of disease vectors, however, suggest that, in the broadest terms, there may be decreases in habitat suitability in areas that become drier, and increases where it becomes wetter. In drier areas, fewer vectors may be some compensation for the greater difficulty of keeping animals at all.

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Table 1. Major diseases of animals in the UK and Africa

	Direct transmission	Food/waterborne	Airborne	Vector-borne	Other/unknown
<p>Notifiable diseases of the UK (from http://www.defra.gov.uk/animalh/diseases/notifiable/index.htm)</p> <p>The last year of occurrence of a disease, if within the last 30 years, is shown in parentheses.</p> <p>Endemic diseases are marked with a +.</p> <p>Diseases which have occurred more than 30 years ago or never in the UK are unmarked.</p>	<p>Anthrax (2002)</p> <p>Aujeszky's disease (1989)</p> <p>Avian influenza (1992)</p> <p>Bovine tuberculosis (+)</p> <p>Brucella abortus, (2004)</p> <p>Brucella melitensis</p> <p>Classical swine fever (CSF) (2000)</p> <p>Contagious bovine pleuro-pneumonia (CBPP)</p> <p>Enzootic bovine leucosis (1996)</p> <p>Epizootic lymphangitis</p> <p>Equine viral arteritis (EVA) (2004)</p> <p>Foot-and-mouth disease (FMD) (2001)</p> <p>Glanders/Farcy</p> <p>Newcastle disease (+)</p> <p>Paramyxovirus of pigeons (+)</p> <p>Peste des petits ruminants (PPR)</p> <p>Rinderpest</p> <p>Scrapie (+)</p> <p>Sheep and goat pox</p> <p>Swine vesicular disease (SVD)</p> <p>Teschen disease</p> <p>Vesicular stomatitis</p>	<p>Bovine spongiform encephalopathy (BSE) (+)</p>	<p>Foot-and-mouth disease (FMD) (2001)</p>	<p>African horse sickness (AHS)</p> <p>African swine fever (ASF)</p> <p>Bat rabies (+)</p> <p>Bluetongue (BT)</p> <p>Epizootic haemorrhagic disease of deer</p> <p>Equine viral encephalomyelitis</p> <p>Equine infectious anaemia (1976)</p> <p>Lumpy skin disease</p> <p>Rift Valley fever</p> <p>Vesicular stomatitis</p> <p>Warble fly (1990)</p> <p>West Nile fever</p>	<p>Contagious agalactia</p> <p>Contagious epididymitis</p> <p>Contagious equine metritis (2005)</p> <p>Dourine</p> <p>Equine viral arteritis (EVA) (2004)</p> <p>Classical Rabies (1970)</p>

Endemic diseases of the UK (ten most economically important – Bennett & Ijpelaar 2003)	Mastitis Bovine tuberculosis Bovine viral diarrhoea (BVD) Infectious bronchitis	Salmonellosis Coccidiosis Toxoplasmosis		Mastitis Summer mastitis Fascioliasis Infectious bovine keratoconjunctivitis (IBK) (Pinkeye)	
Endemic diseases of West Africa, and East/Central/Southern Africa, with high impact on the poor (combined top 20 lists from both regions) (http://www.ilri.cgiar.org/InfoServ/Webpub/fulldocs/investinginaimal/index.htm)	Anthrax Brucellosis Dermatophilosis Foot-and-mouth disease (FMD) Fowl pox Newcastle disease Pasteurellosis (Haemorrhagic septicaemia) Respiratory complexes Contagious bovine pleuropneumonia (CBPP) Contagious caprine pleuropneumonia (CCPP) Peste des petits ruminants (PPR) Sheep and goat pox	Coccidiosis GI helminths (e.g. Strongyloides, Trichostrongylus, Trichuris) Haemonchosis Infectious coryza Blackleg		Anaplasmosis Babesiosis Ectoparasites Fascioliasis GI helminths (eg Onchocerca) Trypanosomiasis Heartwater Rift Valley fever East coast fever (Theileriosis)	GI helminths (e.g. Bunostomum, Strongyloides)

Table 2. The major diseases transmitted by arthropod vectors to humans and livestock (from Mullen and Durden, 2002)

Vector	Diseases of humans	Diseases of livestock
Phthiraptera (Lice)	Epidemic typhus Trench fever Louse-borne relapsing fever	
Reduviidae (Assassin bugs)	Chagas' disease	
Siphonaptera (Fleas)	Plague Murine typhus Q fever Tularaemia	Myxomatosis
Psychodidae (Sand flies)	Leishmaniasis Sand fly fever	Canine leishmaniasis Vesicular stomatitis
Culicidae (Mosquitoes)	Malaria Dengue Yellow fever West Nile fever Filiariasis Encephalitides ((WEE, EEE, VEE, Japanese encephalitis, Saint Louis encephalitis) Rift Valley fever	West Nile fever Encephalitides Rift Valley fever Equine infectious anaemia
Simuliidae (Black flies)	Onchocerciasis	Leucocytozoon (birds) Vesicular stomatitis
Ceratopogonidae (Biting midges)		Bluetongue African horse sickness Akabane Bovine ephemeral fever
Glossinidae (Tsetse flies)	Trypanosomiasis	Trypanosomiasis
Tabanidae (Horse flies)	Loiasis	Sura Equine infectious anaemia 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) Argasidae (Soft ticks)	Human babesiosis Tick-borne Encephalitis Tick fevers Ehrlichiosis Q fever Lyme disease Tick-borne relapsing fever Tularaemia	Babesiosis East coast fever (Theileriosis) Louping ill African Swine Fever Ehrlichiosis Q fever Heartwater Anaplasmosis Borreliosis Tularaemia

Figure 1. In 1981, FMD spread on winds from France to the UK (Isle of Wight)
Donaldson, 1982.



Figure 2. Monthly incidence of PPR in a goat herd in Nigeria from 1982-1986. PPR incidence rises during dry season and falls at the height of the rains. Redrawn from Wosu et al., 1992.

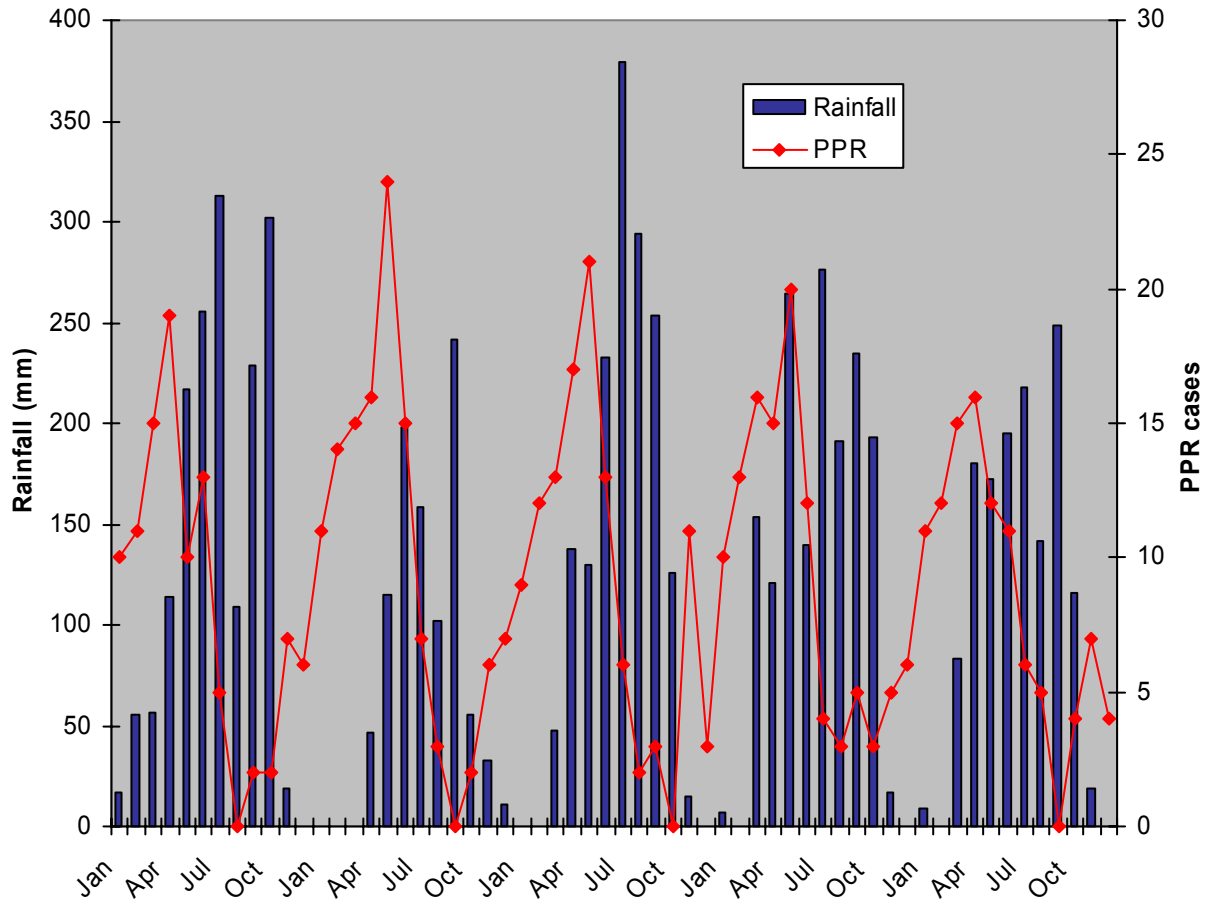


Figure 3. Epidemics of African horse sickness (A) in South Africa are associated with the combination of drought followed by rainfall brought by the El Niño Southern Oscillation (B). Figure B shows the average rainfall anomaly in South Africa during major AHS epizootic years, over the typical 2-year duration of an ENSO event. Reproduced from Baylis et al., 1999.

A



B

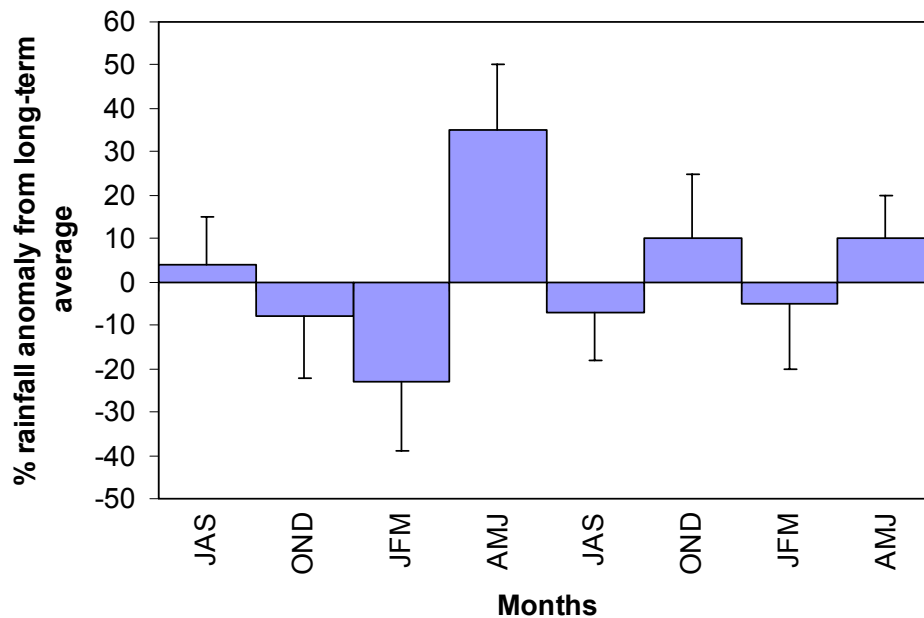


Figure 4. *Tsetse flies (A) transmit animal trypanosomiasis in sub-Saharan Africa, one of the greatest constraints on livestock productivity in the region.*

A



B



Figure 5. Adults of the biting midge *Culicoides nubeculosus* are increasingly competent vectors for bluetongue virus when reared as larvae or pupae at warmer temperatures.

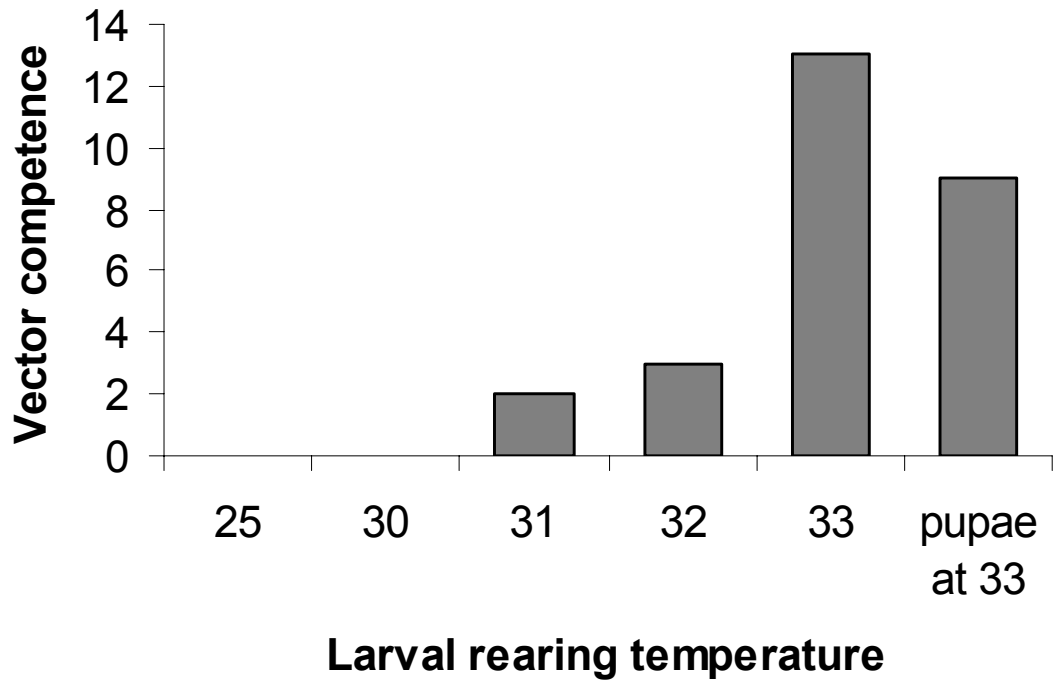


Figure 6. The emergence of bluetongue in Europe from 1998-2005. Yellow regions were affected by bluetongue or AHS before 1998. Blue regions have been affected in the current epidemic. Red circles indicate sites where *Culicoides imicola*, the major old world bluetongue vector, has been recently trapped. The lower black line indicates the northern limit of the vector prior to 1998. The upper line indicates its current northern limit. Reproduced from Purse et al., 2005.

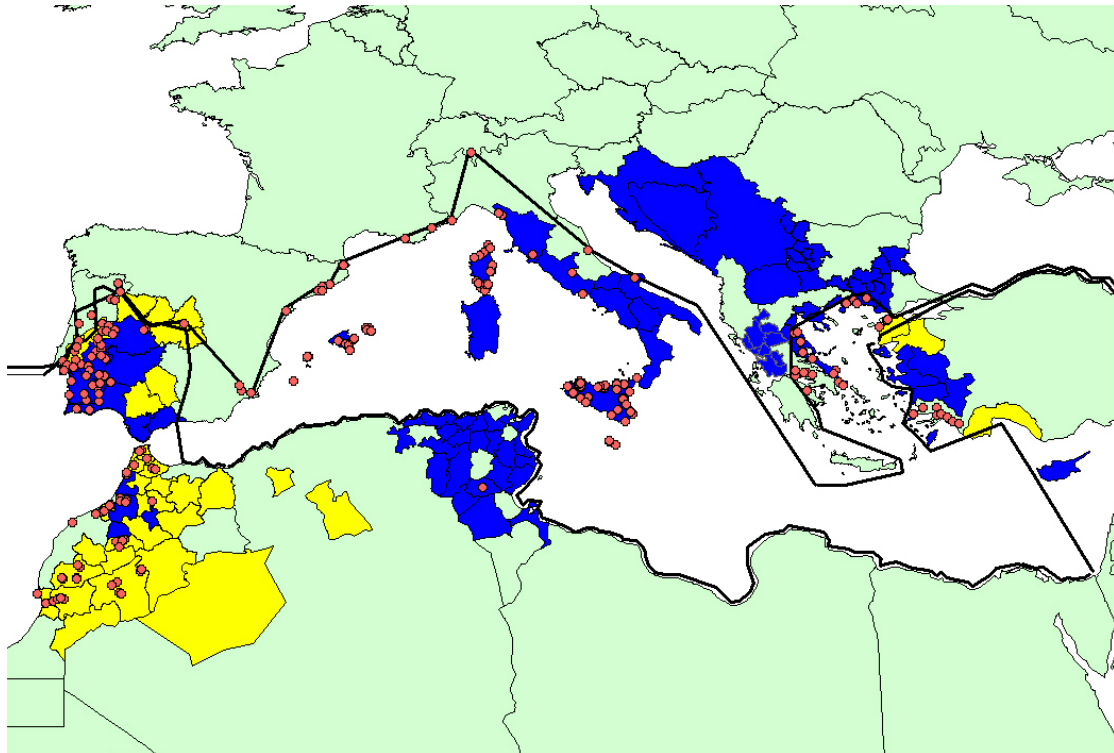


Figure 7. Climate change in Europe between the 1980s and 1990s. A surface map of mean annual minimum temperature was subtracted from a similar map for the 1990s, to show regions of warming or cooling. The scale for temperature change is $\times 10$ (range = -2 to +2 °C). All data from the Climate Research Unit, Norwich, UK. Reproduced from Purse et al., 2005.

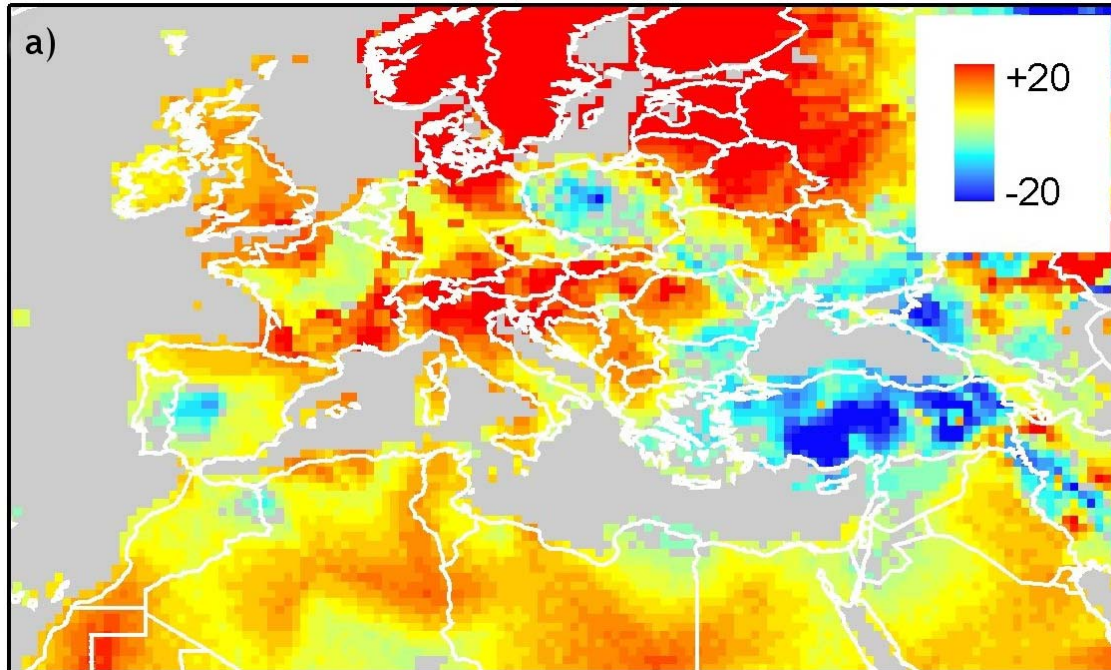
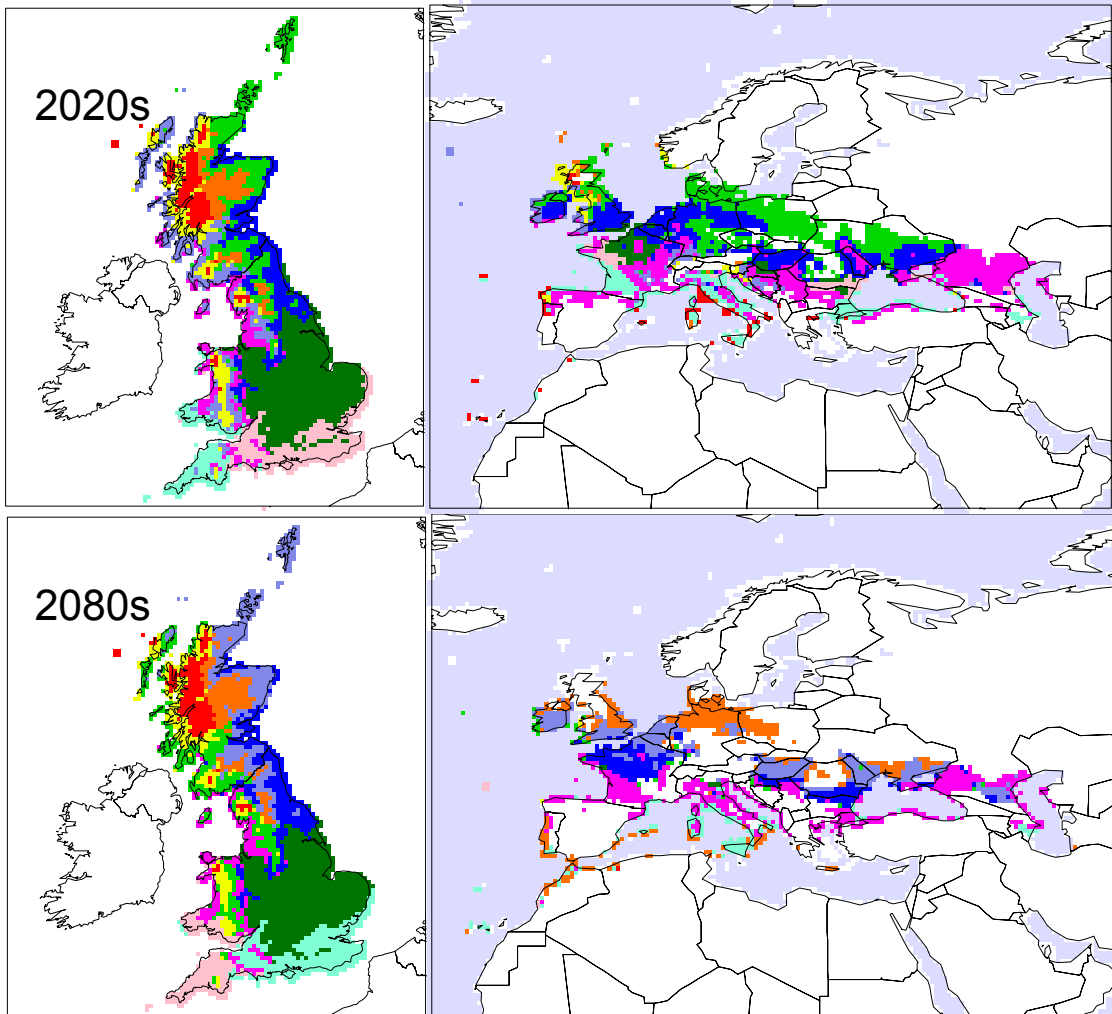


Figure 8. Matching of the predicted UK climate for the 2020s and 2080s with current European climates. Future UK climates are shown as 10 climatic zones defined in terms of temperature, rainfall and vapour pressure (the mean, maxima and minima) for the HadCM2 2020 and HadCM2 2080 medium-high scenarios. The European maps to the right show the current distribution of these same zones at the present time (1961-90). Note that climate zones of equivalent colours in the 2020s and 2080s are not identical. Reproduced from Rogers et al., 2001.



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