The role of climate change in a developing threat: the case of bluetongue in Europe

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Summary
There is a solid theoretical basis for expecting climate change to have a considerable effect on the infectious diseases of humans, animals and plants. Vector-borne diseases are the most likely to be affected. It is, however, rare to observe such impacts, as diseases are also influenced by many other drivers, some of which may have stronger effects over shorter time scales than climate change. Nevertheless, there is evidence that our warming climate has already influenced some animal diseases, of which bluetongue is considered a prime example.
Bluetongue emerged dramatically in southern Europe after 1998 and in northern Europe from 2006. While the speed and scale of this emergence is a challenge to explain, there is evidence, principally from the development of climate-driven models, that recent climate change has played a significant role. Climate-driven models point to an increase in the risk of bluetongue transmission in Europe in recent decades, caused by an increased suitability of parts of southern Europe for the Afro-tropical biting midge, *Culicoides imicola*, as well as an increase in the vectorial capacity of indigenous *Culicoides* vectors in northern Europe. Farm-to-farm transmission models of bluetongue in England and Wales under predicted climatic conditions further suggest that, under high-emission scenarios, the scale of future outbreaks could far exceed those experienced to date. The role of climate change in the developing threat of animal disease is, therefore, likely to be economically and socially costly, unless lower emission targets can be set and followed.

Keywords
Basic reproduction number – Bluetongue – Climate change – *Culicoides* – Europe – Model – Vector-borne disease.

Introduction

Climate change is widely believed to be an important driver of the emergence and spread of infectious diseases (1, 2, 3, 4, 5, 6) and, therefore, a major threat to human, animal and plant health. This belief comes from the observation that many diseases are sensitive to the effects of climate, coupled with the supposition that such diseases will therefore be affected as the climate changes.

The link between specific diseases and climate is strongly supported by the evidence. In general, climate affects diseases in five ways. It may limit the spatial distribution of a disease; for example, many vector-borne diseases are restricted to warmer parts of the world because climate constrains the geographic range of ectothermic insects and ticks. Second, it may influence the scale of outbreaks by affecting the survival and spread of pathogens in the environment or vectors. Third, it may affect seasonal disease occurrence. For example, the common cold and influenza tend to occur in winter because of indoor crowding, facilitating the rapid spread of viruses among people and, possibly, because of weaker immune systems. Fourth, climate may determine the years in which outbreaks of disease occur; the periodic occurrence of the El Niño Southern Oscillation (ENSO), for example, induces drought and more intense rainfall in various parts of the world, and these climatic changes are associated with outbreaks of many diseases. Finally, in some cases, climate may affect the clinical severity of disease in

the affected individual. This is most common for diseases whose severity is linked to the size of inoculum which, in turn, is affected by climate (parasites acquired from the environment, for example).

The scale of the link between climate and disease is formidable. Studies have found that about half (7) to two-thirds (8) of clinically important infectious diseases of humans and animals in Europe are sensitive to a range of climate variables. However, it is worth asking whether sensitivity to climate change is an inevitable consequence of sensitivity to climate variability. The answer is probably no. The seasonal occurrence of diseases such as influenza, for example, may not change significantly with climate change. It remains unclear whether periodic cycles occurring over shorter time scales, such as ENSO, will be affected by climate change. If not, then the many diseases susceptible to such cycles may not respond to climate change. There is, nevertheless, a strong theoretical basis for believing that many climate-sensitive diseases will respond, in one or more ways, to climate change. Systematic review has identified vector-borne diseases as particularly likely to be driven by climate, although water-, food-, soil- and air-borne diseases were also important (8). It is possible, therefore, that a variety of diseases, with a range of transmission routes, may respond to future climate change.

While many diseases may respond to climate change, it is arguable that in many, perhaps most, cases, we will not see the response. A major issue is that diseases are affected by a range of factors, called drivers, which affect their emergence or spread. Climate is just one of these (9). Other drivers are changes to the environment through deforestation and urbanisation; developments in agriculture and food production; changes in how people live, behave, eat, travel and trade; changes in human and veterinary medicine; public health; the use of antimicrobials and insecticides and the development of resistance to these; and the occurrence of 'shocks' such as war or famine. In a major review of the causes of over 300 human disease outbreaks over 65 years, climate was only implicated in ten: of these, six were water-borne diseases, one was fungal and only three were insect-borne (9).

Non-climate drivers may therefore be more important than climate drivers for the vast majority of diseases. Crucially, these non-climate drivers may also be changing far more rapidly than climate, so that any effects of climate change may get lost in the greater effects that other drivers have over the same time scale (10). Malaria presents an excellent example. While malaria is strongly sensitive to climate, and climate change is therefore expected to be a force in increasing its incidence or expanding its distribution in some tropical highland areas (11), in reality malaria is rapidly declining (12). This is as a result of a range of negative drivers – the integrated reduction of mosquito

populations, the development of rapid diagnostic tests and prophylactic control of the malaria-carrying parasites.

These considerations have not stopped many researchers from developing models for the future distribution of infectious diseases, especially vector-borne diseases (or the vectors themselves), at future times and climates (11, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23). These models are rarely able to include the influence of other drivers (though see 24, 25) for two main reasons. First, there is often little or no quantification of how change in a non-climate driver affects a disease; and second, there are rarely scenarios or models for how these non-climate drivers will change in the future (although human population size estimates are an exception). This contrasts with the relative ease of climate-disease modelling: experimental or statistical studies can be used to associate levels of disease with weather or climate variables, and global climate models are used to project those or similar variables into the future. Combining the two allows levels or distributions of climate-sensitive diseases to be projected under future climate scenarios.

Given the importance of other drivers, it is perhaps not surprising that there is as yet little empirical evidence for the effects of climate change on diseases, despite the world approaching 1°C above pre-industrial temperatures. This was also the case for vector-borne diseases, 15 years ago (26). In recent years, however, a number of vector-borne diseases have emerged in different parts of the world, including dengue, Chikungunya, Zika, yellow fever, West Nile, tick-borne encephalitis, Lyme disease, Crimean-Congo haemorrhagic fever, bluetongue, epizootic haemorrhagic disease and Schmallenberg. The emergence of bluetongue in Europe has been particularly strongly attributed to climate change (18, 27). Given its position as the prime example, it is useful to look in detail at the evidence for and against this theory, as it provides us with information on the importance of climate change when considering the developing threat of emerging infectious diseases in general.

Bluetongue

Bluetongue is a disease of ruminants (cattle, sheep, deer) caused by bluetongue virus (BTV), which has more than 25 serotypes. It was first discovered in South Africa in the early 20th century (28) and considered an African disease until 1943, when an outbreak occurred in Cyprus (29). Further outbreaks followed in the Middle East. Soon after, it was discovered that the virus was present in North America and, later, in South Asia and Australia. After the initial outbreak in Cyprus, there were two further outbreaks in Europe before the turn of the century. The first was a severe outbreak of serotype 10 (BTV-10) between 1956 and 1960 in southern Iberia (Spain and Portugal). The second

was an outbreak of BTV-4 from 1979 to 1980, at the other end of the Mediterranean Sea, on the Greek islands of Lesbos and Rhodes, close to the west coast of Turkey.

BTV-4 then reappeared another 20 years later (1998), also on the Greek islands. This time, however, the outbreak heralded the start of the biggest bluetongue outbreak seen to date. Over the ensuing few years, southern Europe was invaded by several different serotypes of BTV (most notably: 1, 2, 4, 9 and 16). These incursions occurred in many countries that had never experienced bluetongue before and 800 km further north than had been previously reported (30). Then, in 2006, BTV-8 appeared unexpectedly in northern Europe and, over the next three years, spread over a huge area of the continent, including the United Kingdom (UK) and reaching southern Scandinavia (31). The outbreak of BTV-8 alone affected hundreds of thousands of farms, at a huge financial cost (an estimated €164–175 million in the Netherlands in 2007 alone) (32), until it was brought to a stop with a newly developed vaccine.

Eighteen years after the start of these unprecedented bluetongue outbreaks, BTVs are still circulating in Europe. In 2015, BTV-1, 2, 4, 8 and 16 were all detected in different parts of the continent and, as of 2016, BTV-8 had spread significantly through France and threatened the UK once more.

Therefore, after most of a century in which only three bluetongue outbreaks were recorded in the southern corners of Europe, the last 18 years have seen an upsurge in the disease's epidemiology in the region.

Vector transmission of bluetongue virus in Europe

The historical distribution of BTV in Europe before the recent outbreak was almost identical to that of the Afro-tropical species of biting midge, *Culicoides imicola*. This species occurs throughout sub-Saharan Africa where it is the major vector of BTV. Its distribution extends to North Africa, the Middle East and southern Asia, as far east as Viet Nam, as well as into Europe. Before the 1998 outbreak, it was known only from southern Spain (33) and Portugal (34), Cyprus (35) and the Greek islands of Rhodes (36) and Lesbos (37), as mentioned above. It is important to recognise that many of these surveys were undertaken in response to outbreaks of bluetongue. Few surveys of *Culicoides* in southern Europe were published in regions that had not experienced the disease, although one survey of Spain in the early 1990s failed to record a single *C. imicola* at over 30 sites in the northern half of the country (38). Remarkably, a survey of the *Culicoides* of Morocco in 1970

recorded *C. imicola* (under its old name, *C. pallidipennis*) in the south of France (39); but when an author (M. Kremer) was asked about this in the early 1990s (by M. Baylis), he described it as a mistake.

The outbreak that began in 1998 occurred over a much larger area of southern Europe, including mainland Greece, mainland Italy, Sicily, Sardinia and the French island of Corsica. *Culicoides imicola* was subsequently found in all of these regions, suggesting an expansion of its distribution.

Bluetongue (serotype 9) also occurred extensively in Bulgaria from 1999; yet, in a survey of 119 farms and 300 trap-nights, and catching more than 70,000 *Culicoides*, not a single *C. imicola* was caught (40). Instead, catches were dominated by the Palearctic *C. obsoletus* and *C. pulicaris* groups. These species groups had, in fact, previously been implicated as possible vectors of BTV. In 1979, BTV-4 was isolated from Cypriot *C. obsoletus* (41), and the closely related African horse sickness virus, serotype 4, was isolated from mixed pools of the two species groups (and one to two other species of *Culicoides*) from Spain in 1988 (42). These species groups occur throughout Europe and, as immediately recognised in 1979, the findings were potentially important for mainland Europe and the UK (41). These field isolations were accompanied by demonstrations showing that wild-caught individuals of both species groups could also be infected with BTV by membrane feeding in the laboratory (43). In the period after 2006, many studies have provided strong evidence for the role of both species groups in the transmission of BTV-8 in northern Europe, as well as the transmission of other serotypes, both outside and inside (44, 45) the range of *C. imicola*.

Evidence for the role of climate change in the European emergence of bluetongue

The emergence of bluetongue in Europe appears to have been underpinned by three developments: first, incursions by multiple serotypes of the virus; second, a wider distribution of *C. imicola* than was previously known; and third, effective transmission of BTV, most clearly in northern Europe, by indigenous species of *Culicoides*, such as the *obsoletus* and *pulicaris* groups.

Evidence has been presented that climate change was a factor in this emergence. Temperature is critically important for the *Culicoides* life cycle and for the development of BTV within adult midges. It was argued that bluetongue's incidence in the first years of its emergence was most marked where European temperature had increased the

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most between the 1980s to 1990s (27). This assessment was made, however, before bluetongue occurred over a huge area of northern Europe, and no evidence was presented that the degree of warming was sufficient to explain the dramatic emergence in the south.

A more detailed exploration of the possible role of climate change in Europe used a mathematical model that described the risk (based on R_0, the basic reproduction number) of bluetongue transmission. The model included four climate-sensitive variables:

– the number of *C. imicola* and *C. obsoletus* group midges

– their longevity

– their biting rate

– the time required for a midge to become infectious after taking an infected blood meal (termed the extrinsic incubation period or EIP).

The last three combine to describe the average number of blood meals taken by a midge on a suitable host during the portion of its life for which it is infectious; this combination is a measure of the risk of virus transmission by infectious midges. The model was run using observed climate data for Europe from 1961 to 2009, and using modelled climate data up to 2050, based on 11 regional climate models driven by the IPCC *Special Report on Emissions Scenarios* 1B emission scenario (46).

The model suggests that there was an increase in the risk of bluetongue transmission in much of western Europe in the 1990s and from 2000 to 2009 (Figs 1a & 1b), relative to the long-term average (1960–2009). It then provides some insight into possible explanations. First, the increase in risk in south-western Europe is consistent with a recent increase in the suitability of much of southern Europe for *C. imicola* (Fig. 1c); and second, the increase in risk
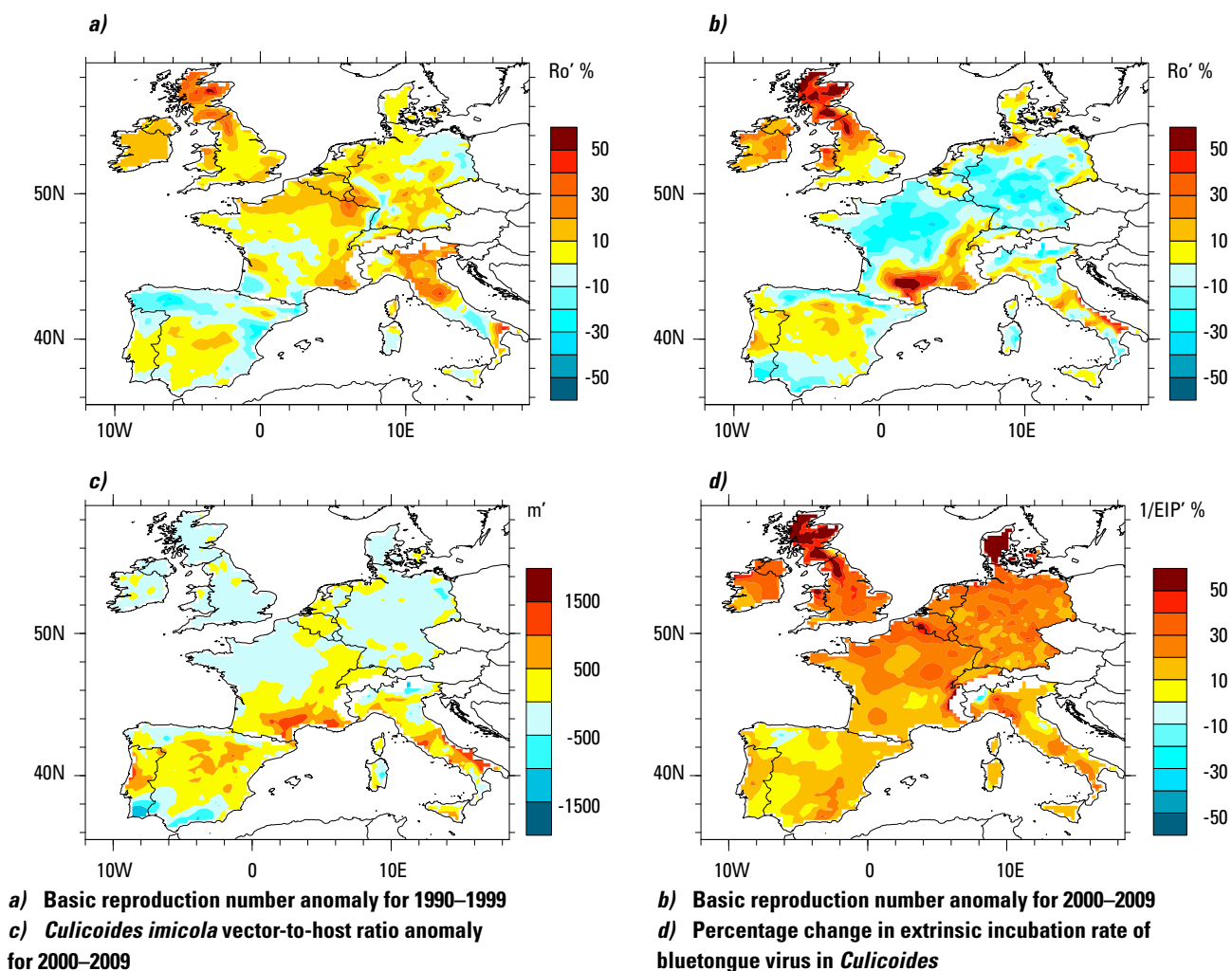

a) **Basic reproduction number anomaly for 1990–1999**
c) ***Culicoides imicola* vector-to-host ratio anomaly for 2000–2009**

b) **Basic reproduction number anomaly for 2000–2009**
d) **Percentage change in extrinsic incubation rate of bluetongue virus in *Culicoides***

Fig. 1.

Modelled impact of climate on the risk of bluetongue or its vectors

All figures show anomalies for the stated time period relative to the 1961–2009 long-term average. Adapted from (18), updated with more recent climate data from E-OBS version 11

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in north-western Europe is consistent with a temperature-associated increase in the rate of virus replication (Fig. 1d).

There is a dramatic increase in transmission risk in north-western Europe, relative to the long-term average, from the mid-1990s onwards (Fig. 2a). Remarkably, the year of highest risk, out of the previous 50, was 2006, which was the year that bluetongue first appeared in the region. An increasing transmission risk over time is also apparent in south-western Europe (Fig. 2b). Interestingly, all three outbreaks of midge-borne disease in south-western Europe since 1960 (African horse sickness in 1965–1966 and 1987–1990, and bluetongue from 2000–2006) occurred during periods of positive anomaly in transmission risk.

In the authors' view, these results provide the strongest evidence to date that recent changes to climate have played a role in the emergence of bluetongue in Europe. Specifically, they show that climate variability affects the risk of virus transmission by *Culicoides* in Europe and that recent climate change has increased that risk. They indicate that disease outbreaks in Europe occur during periods of high risk induced by favourable climatic conditions. They suggest that the changing climate has increased the suitability of southern Europe for the exotic vector *C. imicola*; and increased the ability of native vectors (especially the *C. obsoletus* group) to transmit the virus, in particular by shortening the EIP, over northern Europe.

Evidence from other sources

It is important to ask whether these inferences from models are supported by empirical observation. Specifically, has *C. imicola* recently spread in southern Europe, and are the *C. obsoletus* group more able to transmit BTV than they were before?

There is clear evidence from entomological surveys that *C. imicola* has recently colonised at least three areas: Catalonia in north-eastern Spain (47), the Var region of southern mainland France (48), and the north Pyrenees region, also in mainland France (49). These areas of range expansion largely agree with the modelled outputs. Figure 1c shows increased suitability for *C. imicola* around the Mediterranean coast of France, extending west along the Pyrenees and touching Catalonian Spain. However, a suite of recent genetic population studies has demonstrated that *C. imicola* is anything but a new arrival in the Mediterranean region (50, 51, 52) – in fact, it appears to have been present for thousands of years (50). *C. imicola* may have existed in southern Europe in isolated populations that, in the absence of midge-borne diseases, managed to avoid detection (except, perhaps, by 39). Recent climate change may then have facilitated range expansion of these existing populations at the

extremes of their range, but cannot be credited with the initial introduction of *C. imicola* into the region. There is no evidence to address the question of whether the *C. obsoletus* group in northern Europe has increased in vectorial capacity as a result of climate change since, before 2006, hardly any studies were undertaken on the ability of this species to transmit viruses. It is interesting to observe that, in the same period that BTV-8 was spreading through northern Europe, a second BTV serotype (BTV-1) spread northwards from southern Europe (53), reaching Brittany in northern France by November 2008 and affecting over 4,000 farms (48). The *C. obsoletus* group dominates *Culicoides* catches in the region (48).

Three years later, northern European *C. obsoletus* (54) were identified as major vectors of a novel Orthobunyavirus, called Schmallenberg virus (SBV), that appeared suddenly in Germany in the summer of 2011 (55) and spread rapidly across the continent (56). SBV causes negligible clinical disease in adult cattle and sheep and is therefore difficult to detect at the time of spread; however, it is able to damage developing fetuses when female animals are infected early in pregnancy. In late 2011 and 2012–2013, northern Europe experienced a devastating wave of abortions and birth defects in both cattle and sheep caused by SBV.

There have, therefore, been at least three separate occurrences of arbovirus transmission by *C. obsoletus* group midges in northern Europe in the last decade, while there is no evidence of any transmission in the region in the previous 100 years. It appears that something has changed recently – but what? The increase in vectorial capacity in recent years (Fig. 2a) in models driven by observed climate data suggests that recent climate change should be considered a likely cause, although this cannot be considered proven.

Impact of climate change on outbreak dynamics

Models suggest that climate change will continue to increase the risk of bluetongue outbreaks in the future (18). It is useful to ask how severe future outbreaks might be (in terms of incidence, duration, rate of spread, etc.), in comparison to those of today, and what additional measures might therefore be required to control them. Addressing such questions requires a different modelling approach; namely, full transmission models.

Full transmission models describe the spread of infection between individuals or, in the case of livestock, between farms. A stochastic transmission model for bluetongue in the UK (57) describes the spread of infection between farms by both the movement of infected cattle or sheep (if not yet detected in these animals), and by the dispersal of
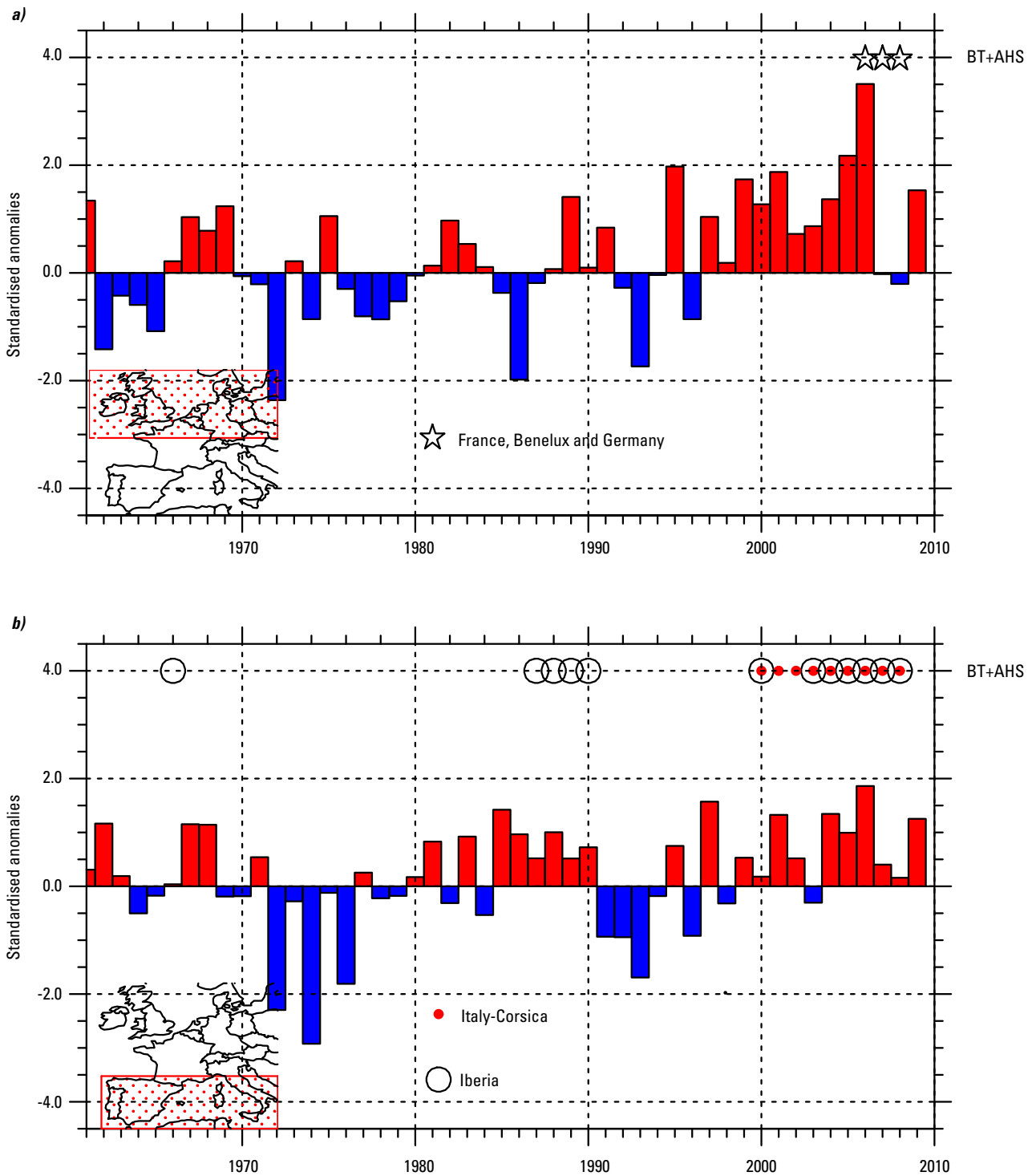
BT: bluetongue
AHS: African horse sickness

Fig. 2

Standardised anomalies of a reduced model of the basic reproduction number of bluetongue in *a)* north-western and *b)* south-western Europe

The reduced model describes the ability of the average *Culicoides* vector to transmit the virus (combining longevity, biting rate and extrinsic incubation period) but does not include dynamic vector-to-host ratio

Red bars are positive anomalies, indicating higher-than-average transmission ability

Blue bars are negative anomalies, indicating lower-than-average transmission ability

Stars and circles show years of recorded occurrence of *Culicoides*-borne diseases (bluetongue or African horse sickness) in the region

Adapted from (18), updated with more recent climate data from E-OBS version 1

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infected *Culicoides* between farms. The model incorporates the effects of temperature in determining the number of midges present on a farm, and their vectorial capacity (thus affecting a farm's 'infectiousness').

With this modelling framework, it is possible to explore the potential scale of outbreaks in different years, according to their various recorded temperatures, and also to explore the potential outbreak scale under future climates. Figure 3 summarises the scale of simulated outbreaks, following the seeding of infection into a single farm in the south of England, under the recorded temperatures of 2006, 2007 and 2014 (58). Potential outbreak scales are seen to vary significantly from year to year, depending on temperature conditions. Figure 3 also shows that 2006 was much more favourable for bluetongue in the UK than 2007. 2006 was the year of introduction of BTV-8 into northern Europe and it reached the UK in 2007; the less favourable temperature conditions that year may have prevented a larger outbreak in the UK.

The model further shows that the scale of future outbreaks may be similarly large or larger. Under a scenario of medium low emissions, the representative concentration pathway (RCP)
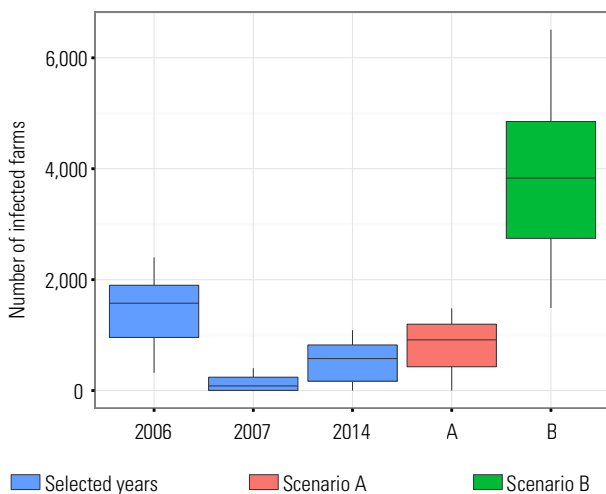

Fig. 3

Number of infected farms across 100 model simulations with infection introduced into a randomly selected farm in Hampshire, the United Kingdom, on 1 June

Examples are shown for selected years (2006, 2007 and 2014), using temperature data from the UKCP09 5-km archive (58), together with two example future years for the last two decades of the 21st century, using temperature projections from the NASA Earth Exchange global daily downscaled projections data set:

Scenario A: 'Typical' year (mean temperature = 11.1°C, mean temperature amplitude = 7.2°C, both close to the medians for 2080–2099, representative concentration pathways or RCP 4.5)

Scenario B: 'Exceptional' year (mean temperature = 13.3°C, mean temperature amplitude = 10.0°C, the maxima for 2080–2099, RCP 8.5)

Box and whisker plot of outbreak size (number of farms infected) shows median (line), interquartile range (box), and 95% range of data (whiskers)

4.5 emission scenario (Scenario A) (59), the average outbreak size at the end of the current century is slightly smaller than that simulated for 2006, but much larger than that in 2007. Under a higher RCP 8.5 emission scenario, and employing extremes of temperature (Scenario B), the scale of an outbreak at the end of the century is, on average, more than two times larger than that simulated for 2006. Indeed, the 95th percentile of outbreak sizes in 2006 is less than the 25th percentile in Scenario B. These simulations show that under future climate conditions the UK will not only be at greater risk of bluetongue outbreaks, but outbreaks that occur will be substantially larger than equivalent outbreaks would be today, and more stringent measures may be required to control them.

Conclusions

The extremely rapid and dramatic emergence of bluetongue in Europe between 1998 and 2008 remains a challenge to explain. The abrupt transition from periodic epizootics in the southern corners of Europe to regular viral invasions, continent-wide transmission, and a state of endemicity, cannot readily be accounted for. Nevertheless, modelling suggests that warming in Europe in recent decades has played a role, allowing *C. imicola* to expand beyond its range and indigenous *Culicoides* to increase in vectorial capacity.

Modelling suggests that the risk of bluetongue will increase in the future, as the climate continues to warm; and other *Culicoides*-borne diseases, such as African horse sickness and epizootic haemorrhagic disease, will probably increase in risk too. Transmission modelling indicates that future outbreaks (at least in northern Europe), when they happen, should also be expected to be substantially larger than those experienced so far, but also that this largely depends on the emission scenario. This is an important message for policy-makers: pursuing lower emission targets can lead to financial benefits from smaller, less frequent animal disease outbreaks.

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L'influence du changement climatique dans les menaces sanitaires évolutives : l'exemple de la fièvre catarrhale ovine en Europe

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Résumé

Nombre d'arguments théoriques sérieux confirment l'ampleur des effets du changement climatique sur les maladies infectieuses affectant l'être humain, les animaux et les végétaux. Les maladies à transmission vectorielle sont probablement les plus sujettes à cette influence. Toutefois, il est très rare de pouvoir observer ces effets directement, dans la mesure où d'autres facteurs exercent également une influence sur les maladies, dont certains ont des effets plus marquants et plus rapides que le changement climatique. L'influence du réchauffement climatique sur certaines maladies animales a néanmoins été prouvée ; à ce titre, le cas de la fièvre catarrhale ovine est considéré comme exemplaire.

La fièvre catarrhale ovine a fait son apparition en Europe méridionale après 1998, puis en Europe du Nord à partir de 2006. Si la rapidité et l'envergure de cette émergence sont difficiles à expliquer, plusieurs démonstrations, recourant pour la plupart à des modèles axés sur le climat font état du rôle important joué par le changement climatique. Les modèles axés sur le climat font ressortir un risque accru de transmission de la fièvre catarrhale ovine en Europe au cours des dernières décennies, associé, d'une part, à l'adéquation croissante de régions entières de l'Europe méridionale vis-à-vis du moucheron afro-tropical *Culicoides imicola* et, d'autre part, à l'accroissement de la capacité vectorielle des vecteurs *Culicoides* autochtones dans le nord de l'Europe. D'après les études basées sur des modèles de transmission de la fièvre catarrhale ovine entre exploitations en Angleterre et au Pays de Galles, dans les conditions climatiques prévisibles, il apparaît qu'en cas de fortes émissions, l'ordre de grandeur des foyers futurs serait considérablement plus élevé que dans les épisodes que nous avons connus jusqu'à présent. Par conséquent, le rôle du changement climatique dans les menaces évolutives de santé animale risque d'avoir un coût économique et social élevé, à moins que des objectifs de réduction de l'émission soient mis en place et fassent l'objet d'un suivi approprié.

Mots-clés

Changement climatique – *Culicoides* – Europe – Fièvre catarrhale ovine – Maladie à transmission vectorielle – Modèle – Taux de reproduction de base.

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La influencia del cambio climático en una amenaza creciente: el caso de la lengua azul en Europa

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Resumen

Existen sólidas bases teóricas para prever que el cambio climático tendrá efectos considerables en las enfermedades infecciosas que afectan al hombre, los animales o las plantas. Las que más probablemente se verán afectadas son las enfermedades transmitidas por vectores. Sin embargo, rara vez pueden observarse tales efectos, pues hay otros muchos factores que influyen en las enfermedades, algunos de los cuales, a una escala temporal más breve, pueden tener una influencia más marcada que el cambio climático. Aun así, hay pruebas de que el clima, en pleno proceso de calentamiento, ya ha incidido en algunas enfermedades animales, de las que la lengua azul se considera un perfecto ejemplo.

La lengua azul hizo una espectacular aparición en el sur de Europa a partir de 1998, y en la Europa septentrional a partir de 2006. Aunque resulta difícil explicar la velocidad y las proporciones de tal aparición, existen sólidos indicios, obtenidos principalmente de la elaboración de modelos regidos por variantes climáticas, de que el reciente cambio climático ha cumplido una función importante. Estos modelos apuntan a un incremento del riesgo de transmisión de la lengua azul en Europa en los últimos decenios, lo que se explica por las condiciones más propicias al jején afrotropical, *Culicoides imicola*, que ofrecen ciertas partes de Europa meridional y por un aumento de la capacidad vectorial de los *Culicoides* autóctonos del norte de Europa. Los modelos de transmisión de la lengua azul entre explotaciones agropecuarias de Inglaterra y Gales en las condiciones climáticas predichas indican además que, en la hipótesis de un elevado volumen de emisiones, los futuros brotes pueden revestir una escala muy superior a cuanto hemos conocido hasta ahora. Por consiguiente, a menos que se logre establecer y cumplir objetivos de emisiones menos cuantiosas, es probable que el cambio climático resulte económica y socialmente gravoso por su incidencia en la creciente amenaza que plantean las enfermedades animales.

Palabras clave

Cambio climático – *Culicoides* – Enfermedad transmitida por vectores – Europa – Lengua azul – Modelo – Número básico de reproducción.

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