GLOBAL WARMING AND ANIMAL DISEASE

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Eight of the warmest years on record have occurred during the last decade, thereby, superficially at least, seeming to support the concept of imminent climate change. It is now accepted by most authorities that 'the balance of evidence suggests that human activities are warming the planet' (Inter Governmental Panel on Climate Change, 1996). Climate models project an increase in global mean temperature of between 1 and 3.5˚C during the next century. Maximum warming will occur at high northern latitudes and during the winter. Night time temperatures will increase more than daytime temperatures.

Environmental factors affecting disease agents and their vectors

The ability of an infectious agent or its vector to survive in the environment and transmit from one animal host to another is dependent on a combination of factors such as:

Macro-climate
- Temperature range
- Rainfall (amount and distribution)
- Humidity

Micro-climate[1]
- Temperature range
- Humidity

Environment
- Vegetation (eg forest, bush, tundra)
- Water (eg rivers, lakes, swamps)

Infectious disease agents are parasites of their animal and human hosts and are most successful when they have little effect on the host they infect and least successful when they kill their host. Often their best strategy is persistent infection, enabling them to multiply and survive for many generations in one host while evading the host's innate and acquired resistance mechanisms.

Innate resistance is the ability to resist damaging effects of an infectious agent that has been gained through generations of exposure to it. This allows selection of those animals that are most resistant (survival of the fittest). Though relatively resistant to clinical disease, animals will become infected and develop immune responses to the disease. This is known as *acquired resistance*. The following presentation will consider those infectious agents of animals that are restricted geographically by climate. In the regions where they occur, their ability to cause disease is restricted by the development of innate and acquired resistance in their hosts.

With regard to the infectious agent/host relationship, these regions are known as areas of *endemic stability*. Climate change, which allows the wider distribution of infectious agents

[1] The microclimate is that which may occur at ground level in vegetation, which may differ markedly with regard to temperature and humidity when compared to the climate generally.

and the vectors that spread them, will allow them to reach naïve populations of animals with no innate or acquired resistance. In these, the infectious agent may cause an epidemic with severe clinical signs of disease. A region where this happens is referred to as an area of *epidemic instability*.

Tickborne diseases

With regard to tickborne diseases, where there is endemic stability, there are usually few or no signs of clinical disease in livestock populations, unless exotic breeds of animals are introduced which have had no experience of the prevalent tickborne diseases.

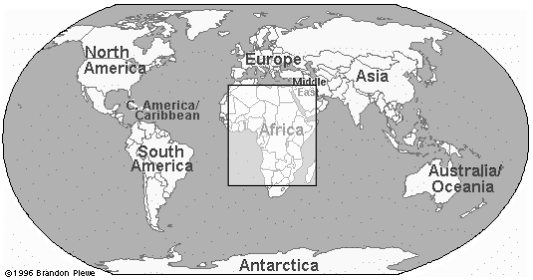
Tropical ticks transmit the worst tick-borne diseases, for example in Africa, *Rhipicephalus appendiculatus* (the brown ear tick) transmits East Coast Fever and *Amblyomma variegatum* (the tropical bont tick) transmits heartwater. In Africa, Australasia and South America, *Boophilus spp*. (blue ticks) transmit babesiosis (redwater) and anaplasmosis.

Tropical ticks cannot survive cold winters. However, in Australia *Boophilus microplus* has been extending its range southwards and causing outbreaks of babesiosis and anaplasmosis in areas previously free from infection. This may be due to global warming or to change in attitude to tick control in Australia which places more reliance on maintaining endemic stability to tickborne diseases rather than controlling them with acaricides.

African Horse Sickness

Global warming is a gradual process. While infectious agents which thrive in warm climates may increase their range, this slow process should not lead to major epidemics. The latter might be caused by sudden climatic changes which gave rise to rapid redistribution of an organism. Phenomena like El Niño can cause rapid climatic change. Changes brought on by El Niño may be responsible for epidemics of African Horse Sickness (AHS). Figure 1 shows the normal distribution of AHS which is limited by the distribution of its vector, the biting midge *Culicoides imicola*. In the northern hemisphere this midge extends up to latitude 40°N. As well as transmitting AHS, it also transmits bluetongue and akabane viruses which cause disease in ruminants. *C. imicola* can travel long distances via wind. AHS is endemic in zebra in southern Africa and when zebra were taken from there to Spain, AHS entered that country. It is also believed that AHS is endemic in donkeys in North Africa.

Figure 1 The shaded box shows approximately the normal distribution of African Horse Sickness in Africa.



A link has been shown between the timing of epidemics of African Horse Sickness in South Africa and the warm phase of the El Niño/Southern Oscillation. This involves a combination of rainfall and drought which gives rise to a population explosion of *C. imicola*. Of the 14 major epidemics of African Horse Sickness that have occurred in South Africa since 1803, 13 were during El Niño periods. With global warming, it is predicted that there will be increasing frequency of El Niño periods. One scenario is that El Niño could become an annual event. If similar events associated with global warming frequently occur, we might experience more epidemics of vector borne diseases like these of AHS. As well as causing epidemics in South Africa, abnormal weather conditions associated with global warming might enable epidemics of AHS in Europe. The disease could even become endemic in southern Europe and might extend further north in other less efficient transmitters, for example, *Culicoides nubeculosus*.

Tsetse fly and trypanosomiasis

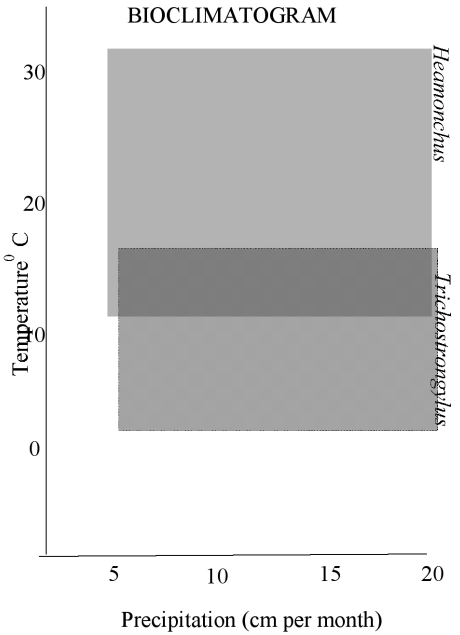
The range of the tsetse fly in Africa is not static but is affected, for example, by cultivation destroying the habitat of the fly and causing the infested area to shrink, or by regeneration of bush allowing the fly to recolonise (for example, abandoned sisal plantations in East Africa colonised by *Lantana* and re-infested by fly).

However, the fly can survive only within a tropical temperature range (15 – 35°C). If other environmental factors are right, it seems that with global warming, the fly could extend its range in Africa southwards in the Okovango Delta in Botswana and northwards in Ethiopia. It could also get 'burned off' in the more arid areas of its range in tropical Africa because of high temperatures. The effects of global warming on the range of tsetse fly are being modelled in Nairobi at the International Livestock Research Institute.

Parasitic gastro enteritis (PGE)

Internal parasites of livestock which cause PGE have a terrestrial larval phase which is affected by the climate. Thus, different parasites are prevalent in different regions dependent on temperature and precipitation. An approximation of the times when livestock are at risk from different parasites causing PGE can be obtained through the use of a bioclimatogram, an example of which (Figure 2) indicates when livestock may be challenged by *Haemonchus*, a nematode which favours a tropical climate, and *Trichostrongylus*, which favours a temperate climate.

Figure 2 A bioclimatogram showing average temperature and rainfall conditions which are favourable for the development of *Haemonchus* (top) and *Trichostrongylus* (bottom) infective larvae.



With global warming, there will be more months of the year over a wider geographical distribution in which livestock will be at risk to PGE caused by *Haemonchus*. Similarly, the range of temperate parasites such as *Trichstrongylus* may be changed because average temperatures may become too hot for development of its terrestrial stages in some areas where it is currently present.

While PGE is dependent on temperature, it is also very dependent on rainfall. The level of PGE in lambs in late summer depends on the date soil moisture returns to field capacity (the autumn return date) in the previous year, together with rainfall from May to July in the current year. In autumn, with decreasing evapotranspiration and often increasing rainfall, the soil eventually becomes saturated and any more rain results in run-off. This is termed the “autumn return date”. If this is late, three generations of parasites can occur in lambs. The first in May-June from over-wintered larvae on herbage. The second from a July-August peak of herbage infection arising mainly from the ewes’ post parturient rise but also auto-infection from the lambs. A third generation due to auto infection only, results from a second usually smaller herbage peak in September-October. If climate change results in later autumn return dates, then PGE will become more prevalent.

There are other examples of the potential for global warming to influence the prevalence and severity of PGE. For instance, herbage infestation with *Nematodirus battus* correlates with temperatures in January to March and amount of rainfall during this period. Overwintering *Nematodirus* eggs require a cold period followed by a warm period in order to hatch. For agricultural and horticultural purposes, it is sometimes accepted that the first day of Spring is when earth temperatures 30cm below ground level reach 6°C. When March temperatures are low and Spring is late, incidence of *Nematodirus* infestation is high, especially if following a particularly cold winter.

Date of spring:

After March 27th	high incidence
March 15th to 27th	above average incidence
March 1st to 15th	below average incidence
February	low incidence

With higher ground temperatures associated with global warming, outbreaks of PGE associated with *Nematodirus battus* are likely to be less.

Parasitic flies

Oestrus ovis (nasal bot) is widely distributed through the tropics and Britain is at the edge of the range of this parasite. It is therefore only important in southern England in warm summers. With global warming, this is an example of a parasitic fly that could become much more widely distributed in UK. Similarly, screw worms *Cochliomyia hominivorax* (New world screw worm) and *Chrysomia bezziana* (Old world screw worm) could become established in Europe as temperatures rise.

Hot, dry summer of 1976

The UK experienced a long, hot and very dry summer in 1976. When considering the effects of global warming in the UK it is interesting to look back at the effects the climate had that summer. Milk yields and weight gains of livestock were depressed. Fascioliasis and parasitic bronchitis were absent. PGE was not a problem in Summer (due to low rainfall) but reappeared in Autumn with Type 2 ostertagiosis. Myiasis and warble fly were more prevalent but head fly was less common. Toxicity from poisonous plants was common, as was bovine keratoconjunctivitis.

Swayback

A warmer climate may also affect indirectly the prevalence of some non-infectious diseases. For example, swayback (copper deficiency in sheep). During warm winters, grazing remains available and it is not necessary to give supplementary feed. Pasture in some areas is deficient in copper, but where supplementary feed is given, livestock receive adequate copper from the supplement. When it is not given outbreaks of swayback can be expected. Thus, a forecast of the incidence of swayback can be given from recordings of winter temperature.

Conclusions

Conclusions which may be drawn are that global warming will certainly change the patterns of disease and while some tropical diseases may extend their range into temperate regions, some other diseases may retreat from areas where they are endemic in the face of global warming. However, global warming will never compete with long distance movement of livestock as a means of spreading diseases to areas where they have not been encountered previously.