



people's trust for **endangered species** |

# The State of **Britain's Mammals** a focus on disease

Written by David W. Macdonald, Tom Moorhouse  
and Merryl Gelling, WildCRU

## Contents

---

1.	Preface	3
2.	An introduction to disease	4
3.	Wildlife disease and conservation	7
4.	Disease in wildlife and domestic animals	11
5.	Badgers and bovine tuberculosis	15
6.	Disease and animal movement	18
7.	Zoonotics	20
8.	Monitoring and regulation of disease in British mammals	24
9.	The future	26
10.	References	29

**Acknowledgments** The authors would like to thank Eleanor Devenish-Nelson, Ann-Marie MacMaster, Tom August, Lisa Worledge, Helen Miller and Roisin Campbell-Palmer for their extremely generous input of time and expertise to this report.

# I. Preface

---

Wildlife conservation is, obviously, about treasuring that which is natural. This thought seems straightforward until one starts to ponder exactly what is natural and whether being natural is a clear-cut ticket to being treasured!

This is the 12th year that we have enjoyed the privilege of an invitation from PTES to offer a personal view on the State of Britain's Mammals. In most years we take an overview of all topics mammaliferous, but in 2010 David Macdonald and Dawn Burnham departed from that breadth to focus on just one issue: invasive species. These are a subset of Britain's nature that is not natural (insofar as the process by which they arrive, and the impacts that they have, fall outside of what is "normal") and, correspondingly, is generally not treasured. This year, for a second time, we select a special topic, that of wildlife diseases, and this brings us again into head-on collision with the question of what is natural and whether it is to be tolerated, far less treasured. Death, even illness, is natural and to that extent might not be thought the business of conservation (which is largely about fostering natural processes), but an infectious disease imported by people to wildlife that otherwise would remain unaffected, or a naturally occurring disease the impact of which is altered by human activities (such as fragmentation of populations), become the province of conservation, as indeed do diseases of wildlife that impact the human enterprise. Thus whether a given pathogen justifies conservation intervention is a matter of judgement and, as every conservation scientist knows, such judgements that lie between evidence and policy are the most difficult of the legion difficulties with which conservationists wrestle.

So, in this digest of diseases that affect British mammals, we open a deliciously seething can of (potentially parasitic) worms that burrow through the fundamental principles of epidemiology to entangle issues of biology, economics, ethics and animal welfare. We single out eight major themes, from the fundamental principles of disease ecology, through discussion of when disease becomes a conservation priority; to the relationship between diseases of wild and domestic mammal species, and the increasingly thorny issue of managing the impact of bTB on cattle; we discuss the disease implications of human-mediated movement of mammal species, and diseases transmissible to humans from wildlife and livestock; we describe the monitoring and regulation of wildlife disease and, finally, where the future of our collective relationship with disease is likely to lead. Each theme is illustrated by reference to examples and case histories from variously native British mammals, but also accompanied by a series of 11 stand-alone vignettes providing details of particularly intricate host-disease relationships.

A glance at our themes reveals that human interests are inextricably woven into the tapestry of relationships between disease-causing agents, wildlife populations and conservation. Wildlife disease has significant potential directly to impact human health and livelihoods. The

possibility of a global pandemic of zoonotic (ie passed from an animal species) origin remains very real, as the recent near-misses of SARS and avian influenza demonstrate; and the debate over the management of badgers for control of bTB (and the economic and social costs thereof) is underlain by the mind-bending complexities of bovine tuberculosis being transmitted to cows from badgers which very likely caught the disease from cattle in the first place. Mention of badgers brings to mind three topics on the role of evidence in policy. First, while evidence is essential to deciding what to do, even good evidence (of which there is a richness in the case of bTB) does not necessarily provide a simple solution; and this frustrating state of affairs raises the risk that a perceived imperative to do something blurs the distinction between doing the right thing or the wrong thing (as defined by their likely outcomes). Likelihood is the second topic: science is accustomed to dealing in probabilities and measures of uncertainty, which are incontrovertibly features of reality, but not ones readily embraced by the mass media (and politicians, who deal with uncertainty daily, need to be braver in presenting it to the public). Taking account of likelihood of achieving an outcome, and the marginal gain of doing so, are aspects of every crevice of human enterprise, and vividly relevant to formulating policy with regard to wildlife diseases. And thirdly it is important to understand that everything is linked to everything else - a truth encapsulated in the power of what economists call Full Life Cycle Analysis. For example a preoccupation with the expected marginal gain from killing badgers has led to the use of a figure of 16% for the expected reduction in the rate of increase of disease in cattle, often ignoring the wide confidence intervals around this figure (ranging from 8-24%), or the nine years of culling taken to achieve it. This preoccupation may have deflected attention from a perspective on the costs, financial and societal, of achieving that gain, and how large those costs might be in the context of, say, trading milk or beef on the Continent. The management of wildlife diseases necessitates this wider perspective which is as daunting as it is enthralling, and is the only protection against the paving with good intentions of the road to hell, strewn as it is with unintended consequences.

So, in this digest of the infections of British mammals we seek not merely to provide a ready and durable reference for facts and figures to inform policy-makers, and the wider public for whom they make the policy, but more broadly to illustrate the underlying principles that inform the challenging journey from evidence to action that is the nub of modern conservation.



David Macdonald



Tom Moorhouse



Merryll Gelling

## 2. An introduction to disease

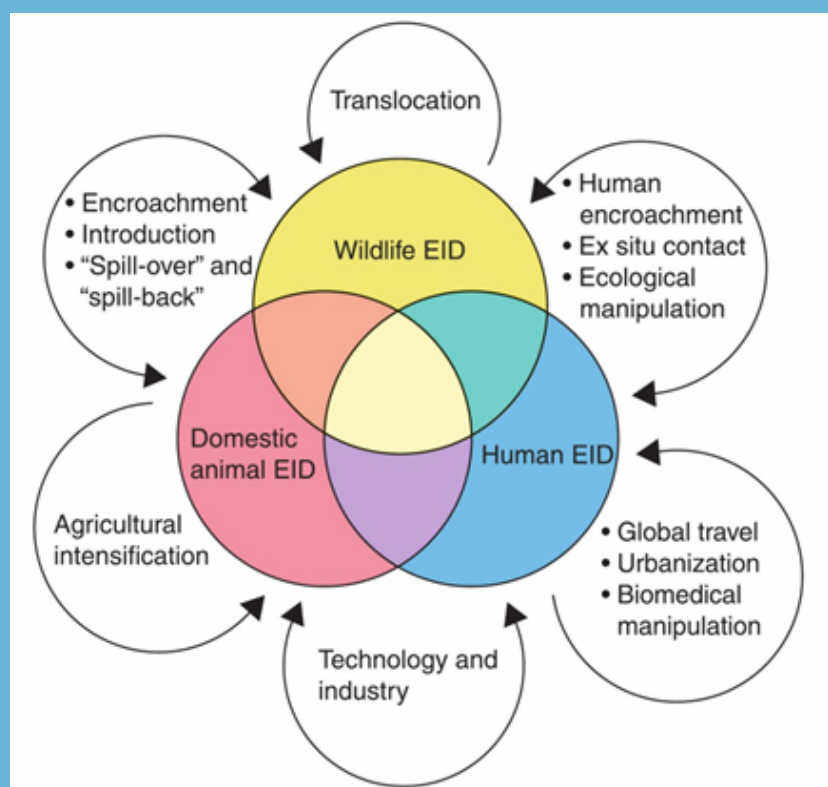
Diseases are a normal and unavoidable part of life for almost every individual<sup>1</sup>. They affect every species whether it be mammal, bird, fish, arthropod, plant, fungus or bacterium. Life, for most if not all individuals, is a continual challenge from an array of naturally occurring parasites and pathogens. Indeed, in line with the ditty that 'big fleas have little fleas upon their back to bite them, and little fleas have lesser fleas, and so ad infinitum', many of the hosts of an infective agent may themselves cause diseases in other, larger-bodied taxa - bacteria, for example, may be hosts for viruses, and parasitic worms might be infected by bacteria, fungi or viruses.

Diseases can be caused by any of a number of infectious agents, including viruses, bacteria, fungi or single or multi-cellular parasites<sup>2</sup>. These infectious agents can be broadly grouped into parasites and pathogens. Parasites live either *in* (microparasites, such as bacteria and fungi) or *on* (macroparasites, such as arthropods and parasitic worms) the living tissue of a host organism<sup>1</sup>. For the purposes of this report the host organisms we are concerned with are animals, not plants, and typically British wild mammals or domestic species capable of passing diseases to them. Microparasites reproduce within the host animal, and from the perspective of the microparasite, hosts are either susceptible to infection, infected, or recovered and immune<sup>1</sup> (Box 1). Macroparasites differ in having a life stage outside their host, and infection occurs through the acquisition, and subsequent maturation, of eggs or larvae<sup>1</sup>. A pathogen is any disease-producing micro-organism or material (which does not necessarily have to be alive:

diseases may also be caused by toxins). By definition, a parasite becomes pathogenic only when its presence negatively impacts upon the host's health and well-being. In the terminology of infectious disease this distinction is the difference between *infection*, the presence or absence of a micro- or macroparasite, and *disease*, which is a measurable, clinical condition in an individual or a population<sup>3</sup>. In what follows we will principally consider infectious disease, mainly caused by microparasites, as the most likely to affect the conservation status of British mammal populations.

The ultimate effect of a disease is to cause a decrease in either the survival probability or the reproductive capacity of individuals in a host species<sup>4</sup> (Box 1). A disease affecting many individuals could negatively impact on a species' population, community and, potentially, evolution<sup>5</sup>. An obvious severe consequence would be widespread direct mortality of individuals within a population. Numerous examples of this exist from humans, livestock and wildlife. Notable human examples are the ~50 million deaths among native South Americans which resulted from their first contact with smallpox, typhus and measles introduced by the conquistadors in the 15<sup>th</sup> and 16<sup>th</sup> centuries<sup>6</sup> and the 1918-20 influenza pandemic which infected over 500 million people and killed between 50-100 million<sup>7</sup> across North America, India, Africa, Australia and Europe, largely transferred between countries by the movements of American and British military personnel<sup>8</sup>. In this report we will explore examples relevant to wild mammals in the UK (chapters 3, 4, 5, 6).

Figure 1, redrawn from Danszak<sup>10</sup>. Reprinted with permission from the American Association for the Advancement of Science: the host-parasite ecological continuum (parasites including viruses and parasitic prokaryotes). Most emerging infectious diseases (EID) exist within a host and parasite continuum between wildlife, domestic animal, and human populations. Few diseases affect exclusively any one group, and the complex relations between host populations set the scene for disease emergence. Examples of EIDs that overlap these categories are canine distemper (domestic animals to wildlife), Lyme disease (wildlife to humans), cat scratch fever (domestic animals to humans) and rabies (all three categories). Arrows denote some of the key factors driving disease emergence.



It is not necessary, however, for diseases to cause death to impact on a population. Endemic (or 'enzootic' when not referring to humans) diseases, where the infection causes sickness (morbidity) rather than immediate mortality, can, for example, delay the onset of sexual maturity or reduce individual growth and survival<sup>3</sup> (see also Box 1), and these often invisible effects can have severe consequences for the long-term endurance of a population, particularly if that population already faces other challenges. Populations which are isolated or fragmented - a situation which of itself may lead to precarious loss of genetic variation and could thereby diminish disease resistance<sup>5</sup> - may become extinct locally as a direct result of infection with enzootic diseases if these result in chronic population depression<sup>9-10</sup>. Although infectious disease occurs naturally in populations unaffected by people, and under those circumstances are not the business of conservation, very often nowadays they interact with human influences and therefore fall within the ambit of conservation, as we explore in chapter 3.

The distinction between diseases in a population that are long established (endemic or enzootic) and those that are new (or 'emergent') is a crucial one. Within a stable ecosystem, hosts and pathogens co-evolve, whereby the host species develops individual and/or population immunity to protect it from the disease<sup>5</sup>. Typically, consequently, populations demonstrate substantial levels of immunity to endemic diseases. An emerging infectious disease is one that is in some way novel and also either 'epidemic' (in humans) or 'epizootic' (in animals)<sup>10</sup>, such that the number of new cases is substantially greater than what would normally be expected in a given population. While endemic and enzootic diseases may certainly result in a steady death toll (Box 1), emerging infectious diseases pose considerably greater risks of mortality in populations of humans, livestock and wildlife (chapter 4,5,6,7, 9).

#### BOX 1 Diseases and host populations

Diseases (ie microparasites and macroparasites) exist in balance with their hosts. If a hypothetical virus were quickly to kill 100% of infected individuals it probably would not spread far enough to become an epidemic because infected hosts would die before they were able to transmit it. A less virulent virus, however, might spread further and persist longer because hosts remain alive for long enough to pass it on. Whether an epidemic can be triggered, or an endemic disease can spread, depends in both cases upon whether a given host passes the parasite to more than one other host (the parasite spreads), or less than one other host (the parasite will not spread). One complicating factor is that individuals in real populations are rarely, if ever, all equally susceptible to a given infection. Even parasites that easily pass between hosts may still fail to spread if the majority of the host population is immune to it. The impact that a microparasite has on a population is therefore governed by not only the parasite's ability to spread but also the population's composition in terms of the proportion of individuals that are susceptible, infected and recovered (and now immune), as well as the contact rate between host individuals (which is related to population density) and the death rate due to the parasite.

Epidemics in host populations start with a high density of susceptible individuals and for a disease with a high spreading ability contacts between infected and susceptible individuals are frequent, and the virus spreads quickly between them. With each new infection, however, the density of susceptibles is reduced as they become infected and then either die or recover (becoming immune). Eventually the density of susceptibles will decrease to a point where the parasite is on average being passed to less than one other susceptible host, the number of infected cases starts to fall and the epidemic dies out.

For endemic diseases the relationship between host and parasite can be more complex. It is possible for a microparasite to persist at very low levels in a host population but nevertheless to limit the host's numbers<sup>17</sup>. Imagine a lethal disease that can spread faster than a host population can grow. At low host densities it may kill < 1% of individuals, and an observer might therefore be tempted to conclude that the disease plays little role in suppressing the population, compared with other sources of mortality. If the density of hosts increases, however, contact rates between them will increase and the disease will spread, killing ever larger proportions of the hosts until the population begins to decline and is reduced to its original size. In this instance the microparasite regulates the host population size to an equilibrium level.

A major caveat is that the above relationships exist for diseases that are transmitted directly from host to host (eg by coughs and sneezes) and for which transmission rates are concomitantly dependent upon host density. While epidemics of such diseases may cause widespread mortality, as hosts become rarer the chain of disease transmission will eventually break, meaning the population, while potentially critically depleted (and therefore vulnerable to other factors associated with having a small population size, including infection by other diseases), is unlikely to be wiped out. For diseases that are transmitted at rates that are unlinked to host density - for example if the disease has a reservoir in another, abundant species, or is transmitted by an abundant vector, or if the disease is passed on by sexual contact between hosts - high rates of transmission can still occur at low host densities, and the disease has the potential to drive the host to extinction. For conservation purposes, therefore, it is often critical to identify whether diseases are directly transmitted or if there is a reservoir<sup>18</sup>.

---

An intriguing aspect of emergent infectious disease is that a pathogen which may be endemic or enzootic in one population may be 'novel', and therefore a severe epidemic or epizootic risk to immunologically naïve individuals, in another. Within a given species, this may occur when geographically separated populations meet, as in the devastating impact of measles from the conquistadors on native South Americans. When infections pass between species the outcome depends not only on the severity of the disease in the infected individual but also on the ability of the disease to pass between individuals in the new host. Many enzootic infections (ie established and stable in animal populations) can be transmitted from animals to humans (such diseases are termed 'zoonoses'), but with little or no subsequent person-to-person transmission - eg rabies or trypanosomiasis - whereas other zoonotic pathogens can spread efficiently between people once introduced, leading to localised outbreaks (eg Ebola virus) or global spread (eg pandemic influenza)<sup>11</sup> (chapter 7; see also Box 1).

Wild animal populations represent a significant reservoir of potential emergent infectious diseases in humans, and vice versa (Figure 1). Pathogens shared with wild or domestic animals cause more than 60% of infectious diseases in man<sup>12</sup>, and enzootic zoonoses cause about a billion cases of illness in people and millions of deaths every year<sup>11</sup>. Wild animal species are a primary reservoir for emerging zoonoses<sup>13</sup>, and in particular wildlife species which are directly consumed (eg as bushmeat; chapter 7) or which can pass diseases to livestock (chapter 4)<sup>11</sup>.

Diseases passed to livestock from wildlife may also represent a significant financial burden (chapters 3, 8) and diseases passed from humans and domesticated animals to wildlife may represent a considerable conservation risk for a large number of species. To provide an indication of the degree to which diseases are shared between humans, livestock and wildlife, a study of 1 922 diseases (the majority of which were principally human diseases), considering these categories of host, found that 1 115 (58.0%) of hosts fell into more than one of these categories, and 392 (20.4%) fell into all three (see Figure 1).

Diseases transmitted between humans and wild and domestic animal species have important impacts on public health, livestock economies and wildlife conservation<sup>14</sup>. These impacts are not restricted to low-income countries<sup>11</sup> - the cost in the USA of introduced disease to human, livestock, and crop plant health is estimated as \$41 - 47 billion per year<sup>15-16</sup> - and arise from a complex series of interactions all ultimately deriving from human landuse practices. Figure 1 details the inter-relationship between the key players, and illustrates the underlying drivers of disease emergence. In this edition of the SOBM we explore these drivers in the context of British mammal species, from a starting point of concern for wild species but inevitably encompassing impacts on domestic ones, to give an account of the issues facing the health of British wildlife, humans and livestock and the challenges for our collective future.

### 3. Wildlife disease and conservation

Diseases are present in all wildlife populations and do not necessarily either imperil them or qualify as a conservation issue (see Box 2). A recent small-scale health study of free-living adult American mink (*Neovision vision*) in the UK found that six of 12 individuals tested were positive for antibodies to *Toxoplasma gondii*, and eight of 12 were positive for antibodies to Alutians Disease Virus (ADV)<sup>19</sup>. But there is, in this case perhaps unfortunately, no suggestion that the presence of these diseases has caused any population decline in the invasive UK population of mink. Similarly, even in water voles (*Arvicola amphibius*), a species which certainly is a UK conservation concern<sup>20</sup>, over 6% of 120 individuals sampled across 11 sites in the UK were found to have antibodies for *Leptospira* spp. (the bacteria that cause Leptospirosis, or Weil's disease; chapter 7), with 32% of the voles being infected with two or more different pathogens, including *Campylobacter* spp., *Escherichia coli*, *Salmonella enterica*, *Toxoplasma gondii* and *Giardia* spp.<sup>21</sup>. These co-infected voles did not differ in condition to those that were singly infected. This is perhaps unsurprising given that a host of other unrecorded infections may also have been present (ie because only 21 parasites and pathogens were screened-for, other infections may have been present but undetected, potentially meaning that all voles were, in fact, co-infected), because these infections are of unknown pathogenicity in voles (ie their presence may not imply disease)<sup>21</sup>, and because co-infections do not always increase host mortality if they result in cross-immunity

to a range of related infections<sup>5,22</sup>. In short, infection (and indeed co-infection, although the results of an interaction between different parasites and pathogens in a host can be unpredictable<sup>23</sup>) is 'normal' for wildlife populations and not necessarily a conservation concern (Box 2).

For a given parasite or pathogen to have conservation implications requires some combination of two factors. Firstly, the disease should be sufficiently novel that many susceptible individuals exist, so that it can cause widespread morbidity and/or mortality (Box 1; chapter 2). A population can encounter a pathogen to which it has no resistance through intraspecific transmission (ie be passed from individuals in a separate population of the same species). For example the phocine distemper virus (PDV) outbreak of 1988 in which 18000 harbour seals (*Phoca vitulina*) were washed up along the shores of Europe and the UK, and the second outbreak in 2002, both originated in seals from the Danish island of Anholt and were spread by natural movements within and between populations<sup>24-25</sup> (although there is some suspicion that grey seals (*Halichoerus grypus*) may have

#### Leptospirosis in rodents

Leptospirosis is a water-borne, emerging infectious zoonotic disease which can give rise to potentially fatal Weil's disease in humans and can be a significant problem for people who regularly use waterways in some capacity<sup>30</sup>. Leptospire (the spirochete bacteria that cause leptospirosis) are excreted from infected animals in urine. Early studies in wild rats (*Rattus norvegicus*) suggested a prevalence of 70 - 90%<sup>31</sup>, however later work found only 14% of rats to be infected with *Leptospira* spp., approximately half of which were infected with *Leptospira bratislava* (a bacterium that is much less serious for human health). The role of rats in the spread of leptospirosis to humans may therefore be less important than commonly believed<sup>32-33</sup> and other mammals are likely to play larger roles. Recent work investigating the incidence of Leptospirosis in wild water voles (*Arvicola amphibius*) throughout the UK found a 6% prevalence<sup>21</sup>, which increased to 43% in captive-bred and reintroduced water voles<sup>34</sup>, indicating a possible lack of immunocompetence in reintroduced animals<sup>35</sup>. Nevertheless, identification of further wild mammalian reservoirs for leptospirosis does not increase the risk of this disease for humans (the risks have not changed, merely our understanding of them), which is mitigated by following standard hygiene precautions after coming into contact with bodies of water.



also contributed to the dispersal of the PDV among harbour seal populations<sup>26</sup>). Diseases likely to result in severe population declines or extinction, however, are far more likely to result from interspecific transmission, ie between different, but similar, species living sympatrically (in the same geographical area)<sup>14</sup>. This is because pathogens that are a major threat are highly unlikely to persist in small populations of endangered hosts<sup>27</sup>, and so the majority of outbreaks of disease in endangered species originate from pathogens that infect multiple species and which persist in another species with a larger host population<sup>10,14</sup> (see Box 1). Often, therefore, outbreaks of conservation importance result from spill-over (see chapter 4) of disease from domestic animals, brought into contact with wild populations as a result of human encroachment. Domestic dogs, for example, were the probable source of rabies outbreaks that decimated populations of both African wild dogs (*Lycaon pictus*)<sup>28</sup> and Ethiopian wolves (*Canis simensis*)<sup>29</sup>.

The second pre-requisite for a disease to cause wildlife conservation concern is that a given population must usually already be facing some other challenge, and that either this challenge or the cause of the disease's spread is anthropogenic in origin (Box 2). How severely an epizootic event affects a given wild population of animals depends to a very large degree on a raft of additional factors influencing the size, degree of isolation or some other constituent of that population's conservation status<sup>10,14,36-38</sup>. Populations that are spread over large geographical areas are unlikely to face long term threats to their viability from epizootic disease, whereas small, fragmented and isolated populations are considerably

more likely to be driven to (at least local) extinction by an epizootic event<sup>10,14,36-38</sup>. The UK population of rabbits (*Oryctolagus cuniculus*) remains substantial and widespread despite the myxoma virus having been deliberately introduced to Britain in the 1950s in a bid to control their numbers. The virus, which causes myxomatosis, resulted in 99% mortality<sup>39</sup> but the survival of resistant individuals, combined with a subsequent reduction in the virulence of the virus, has provided rabbits the opportunity to recover their numbers, although perhaps not to quite their pre-1950s levels<sup>40</sup>. (Interestingly, and providing the exception that proves our point, evidence suggests that rabbit populations infected with rabbit haemorrhagic disease virus - which broke out in the UK in the early 1980s - are slower to recover when also under pressure from myxomatosis<sup>41</sup>.) Similarly a 1994 outbreak of sarcoptic mange - a disease caused by the *Sarcoptes scabiei* mite which is globally widespread, affecting over 100 different domestic and wild species<sup>42</sup> - amidst UK red fox (*Vulpes vulpes*) populations caused over 95% mortality of individuals in areas of Bristol<sup>43</sup> but did not lead to even local extinction of foxes. By contrast the effects of disease when added to other sources of pressure on a population can be devastating. Populations of the African wild dog (*Lycaon pictus*) have been declining since the 1960s and are now severely fragmented<sup>44-45</sup>. The probable transmission of canine distemper virus, which was epizootic amongst domestic dogs, to the population of African wild dogs in the Serengeti national park in 1991, resulted in the extinction of this population<sup>46-47</sup>.

The combination of novel disease and additional pressures can lead to population extinction even in

## Box 2: When does infectious disease become a conservation problem?

A glib answer to this question might be 'when an individual of an endangered species becomes ill'. A more thoughtful response may be that infectious diseases are hazards to ecosystems when they affect keystone species such as top predators, or when they undermine ecosystem support systems<sup>71</sup>. Both responses, however, ignore the fact that infectious disease is a natural phenomenon, and a general tenet of biological conservation might be not to meddle where natural processes operate naturally. Compassion might prompt the rescue, or even euthanasia, of a sick animal, but such an intervention could be said to have little relevance to conservation, which is focused on the viability of populations and ecological communities. Of course, even before it was formalised mathematically<sup>72</sup>, ecologists realised that diseases were not merely a source of individual morbidity and mortality in nature, but could also limit, even regulate, populations (Box 1). In that sense diseases are clearly relevant to conservation biology, as part of natural processes, but this neither qualifies them as a problem nor constitutes a justification for meddling

in population processes. So what, then, would justify an intervention on conservation grounds? The litmus test is of exactly how natural the population effects of a given disease are. In this test, disease becomes the business of conservation if it arises naturally but affects individuals of a species threatened by anthropogenic factors, or, conversely, if anthropogenic factors brought a disease into contact with a previously unthreatened population. This outlook leads sometimes to conservationists being disquieted by a too-ready eagerness to intervene when disease afflicts wildlife. On the other hand, there are clear and pressing cases where infectious disease in wildlife conspicuously affects, or is affected or caused by, humans; and human involvement is an operational definition of topics within the ambit of conservation<sup>73</sup>. So, as is characteristic of conservation issues, the decision of when an infectious disease justifies intervention is not always straightforward, and indeed the position of infectious disease within conservation is both technically and philosophically challenging<sup>24</sup>.



previously abundant species with widespread geographical ranges. The white-clawed crayfish (*Austropotamobius pallipes*), which until the last 20 years of the 20<sup>th</sup> century was ubiquitous in British freshwaters, has undergone a catastrophic decline due to the twin effects of competition from the larger and more aggressive invasive American signal crayfish (*Pacifastacus leniusculus*), and from crayfish plague (*Aphanomyces astaci*), a fungal infection to which signal crayfish are immune and for which they act as a vector<sup>48-49</sup>. It is plausible that in the absence of the competition from signal crayfish that white-clawed crayfish populations could recover, but with the additional outcompeting of white clawed crayfish by signal crayfish<sup>50-51</sup> the native species is increasingly restricted to isolated water bodies that have not been colonised by signal crayfish and/or infected with crayfish plague.

The mammalian analogue of the crayfish example is the UK relationship between red squirrels (*Sciurus vulgaris*) and grey squirrels (*Sciurus carolinensis*). Red squirrels were historically widespread throughout Britain, but over the last 50 years have suffered a decline of 50%<sup>52</sup>, despite expanding their range within Scotland, where more than 75% of the red squirrel population is currently located. Grey squirrels carry the squirrel poxvirus (SQPV) which is fatal to red squirrels<sup>24, 53-54</sup> and which is transmitted

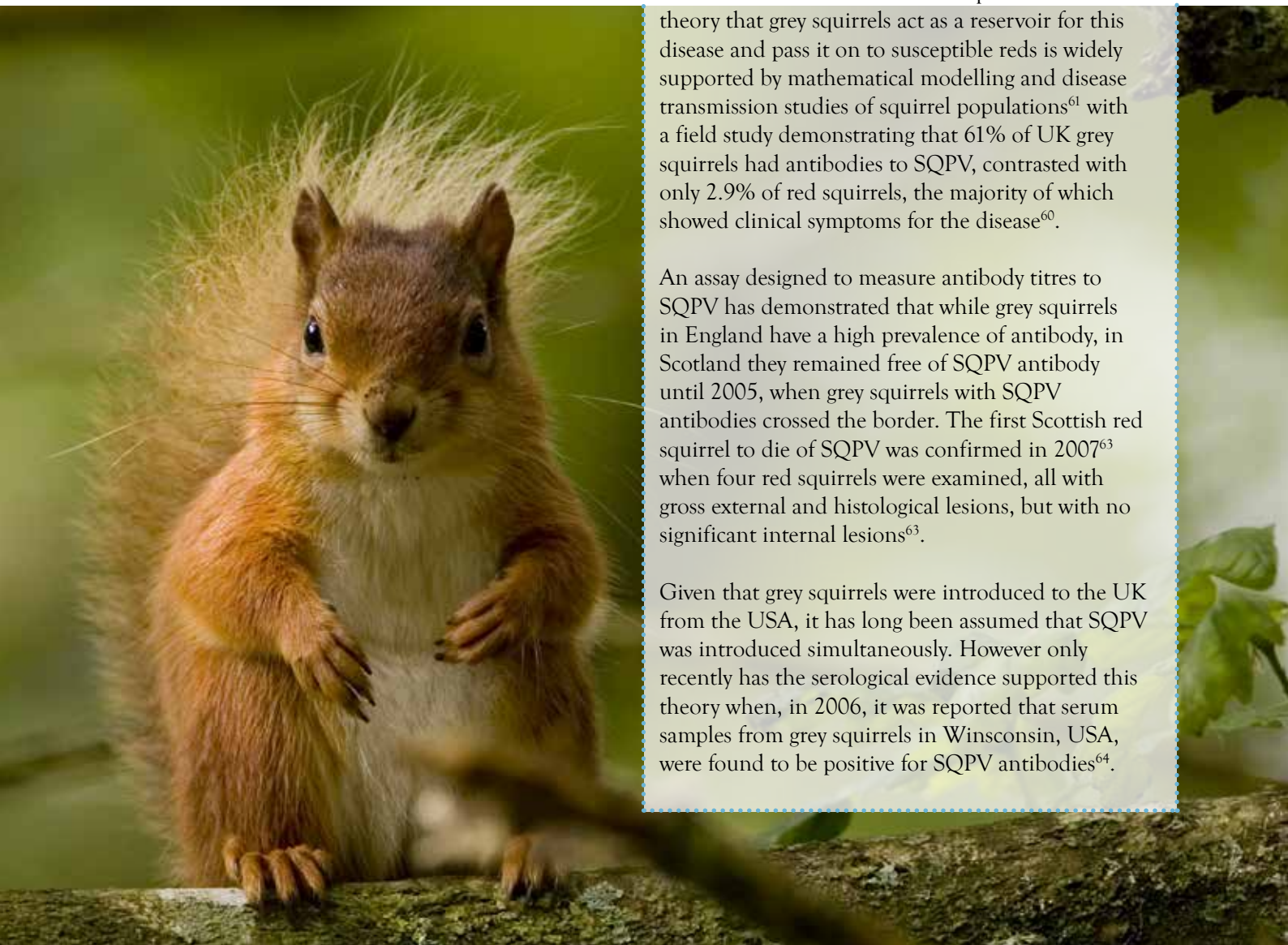
either through direct contact between individuals<sup>55</sup> or through environmental contamination (eg infection vectored through the common use of dreys by members of both species<sup>55-56</sup>). Grey squirrels are also able to out-compete red squirrels, partially through their ability to use acorns as a food source, which the red squirrels are unable to do. In the absence of SQPV, numbers of red squirrels decline when in contact with grey squirrels (eg in Scotland and Italy), but in the presence of both SQPV and grey squirrels (eg in Cumbria) the rate of decline is 17-25 times faster<sup>57</sup>. With the first detection of seropositive grey squirrels in southern Scotland in 2005, and the first cases of disease in Scottish red squirrels two years later, SQPV disease represents a significant

### Squirrelpox

Squirrelpox virus (SQPV) continues to be a major threat to remaining UK red squirrel populations<sup>59-60</sup>. Experimental infection of squirrels with SQPV has confirmed that grey squirrels are hosts for the disease but remain clinically asymptomatic, whereas the disease is characterised by the formation and ulceration of haemorrhagic scabs around the eyes, nose and mouth of infected red squirrels<sup>61-62</sup>. The theory that grey squirrels act as a reservoir for this disease and pass it on to susceptible reds is widely supported by mathematical modelling and disease transmission studies of squirrel populations<sup>61</sup> with a field study demonstrating that 61% of UK grey squirrels had antibodies to SQPV, contrasted with only 2.9% of red squirrels, the majority of which showed clinical symptoms for the disease<sup>60</sup>.

An assay designed to measure antibody titres to SQPV has demonstrated that while grey squirrels in England have a high prevalence of antibody, in Scotland they remained free of SQPV antibody until 2005, when grey squirrels with SQPV antibodies crossed the border. The first Scottish red squirrel to die of SQPV was confirmed in 2007<sup>63</sup> when four red squirrels were examined, all with gross external and histological lesions, but with no significant internal lesions<sup>63</sup>.

Given that grey squirrels were introduced to the UK from the USA, it has long been assumed that SQPV was introduced simultaneously. However only recently has the serological evidence supported this theory when, in 2006, it was reported that serum samples from grey squirrels in Wisconsin, USA, were found to be positive for SQPV antibodies<sup>64</sup>.



---

threat to the conservation of red squirrels in the UK<sup>58</sup>. Scottish red squirrel populations, the last remaining substantial population of native British squirrels, are likely to suffer numerous disease outbreaks over the next 25 years<sup>58</sup>, leaving the conservation status of this endangered mammal in the balance in Britain.

The above analysis raises the point that species faced with multiple threats will require their conservation action to be carefully considered. For example species whose populations are reduced by population fragmentation might be expected to respond favourably to targeted habitat restoration to provide linkages between sub-populations. Counterintuitively, however, where these populations are also threatened by invasive species and/or the spread of an epidemic disease, joining up these fragments might facilitate the invasion of the inimical pathogen or species into the endangered species' final strongholds. In this case an action that is desirable to combat a particular threat is rendered extremely unwise by the existence of another, concurrent, threat. In such cases it would be necessary to deal first with the disease/invasive and only then to implement the required habitat restoration, but this example highlights the importance of a firm understanding of all of the causes of a species decline prior to remedial action.

Another factor that may interact with disease to affect the conservation status of wildlife populations is environmental pollution. Pesticide pollution, for example, is known to have adverse effects on British mammals, including direct mortality and behavioural and reproductive effects<sup>65</sup>, and an increasing number of studies show that common environmental pollutants (eg pesticides, herbicides and metals) may impair the immune system of a wide range of animal taxa<sup>37,66</sup>. For

instance, while no causal link has been established to date between organochlorines and susceptibility to PDV<sup>26</sup>, organochlorines are known to decrease the efficiency of immunity in laboratory animals<sup>67</sup>, and harbour seals that died of PDV during the 1988 outbreak were found to have elevated levels of organochlorines in their blubber in some (but not all) localities<sup>68</sup>.

These considerations suggest that while the impact of a given epizootic on a given population of wildlife, all else being equal, may not result in population extinction, the increasing burdens of anthropogenic habitat loss and fragmentation, invasive species, climate change, and contamination of the environment with complex mixtures of metals and agro-chemicals, may result in a situation in which wildlife populations have an increased likelihood of disease-mediated disadvantage, or even extinction. This point aside, there remains the possibility of epizootic outbreaks so severe that it is sufficient of itself to cause the extinction of a species. One example of this exists: *Batrachochytrium dendrobatidis*, the bacterium which causes chytridiomycosis, an emerging infectious disease associated with multi-species declines in amphibians worldwide<sup>37,69</sup>, has been directly linked to the extinction in 1997 of the Australian sharp-snouted day frog (*Taudactylus acutirostris*)<sup>70</sup>. This represents the first case of extinction of a free-ranging wildlife species where disease is thought to have acted as both the proximate and ultimate cause of a species' extinction, in the absence of other factors<sup>70</sup>. One worrying conclusion from the authors of that study is that due to the logistical and technical, and other difficulties involved in assessing the role of pathogens in extinctions, infectious diseases are likely to have been a severely underestimated cause of historical and present biodiversity loss.

## 4. Disease in wildlife and domestic animals

---

The transmission of parasites and pathogens between wildlife populations and those of domestic animals - either livestock or pet/working animals - is governed by two parallel processes, known as *spill-over* and *spill-back*<sup>10</sup>; (Figure 1). *Spill-over* is the transmission of infectious agents from domestic animals to wildlife, and *spill-back* is the reverse process (Figure 1), and both can precipitate emergent infectious diseases in their respective host populations. *Spill-over* is particularly a threat for endangered species in which small populations can be infected from the much larger disease reservoirs in livestock (chapter 3). Conversely, *spill-back* from disease reservoirs in abundant populations of wild animals to UK livestock has (economic) implications for livestock health and productivity<sup>74-75</sup>, implications for the health of pets and companion animals<sup>74</sup> and ultimately implications for human health through potential zoonotic infection (chapter 7).

*Spill-over* and *spill-back* occur because many parasites and pathogens can infect multiple host species, but the impacts of all multi-host infections are not equal. Outbreaks of bovine tuberculosis and avian influenza, which can both *spill-back* from a number of wildlife hosts, have severe consequences for animal health, and the economy<sup>76</sup>, and would stimulate significant state intervention if detected (chapter 8). Other infections, such as toxoplasmosis or sarcoptic mange (which is found in 63% of urban red fox populations nationwide, 55% of semi-natural habitats and 37% of agricultural habitats<sup>43</sup>, and can *spill-back* to domestic dogs<sup>75</sup>), have more minor implications, both for livestock and for wildlife conservation. The probability of *spill-back* depends on many interacting factors, including how many of the host population there are, where they are, how mobile they are and the prevalence of the

disease among them, as well as the density and conditions in which livestock are maintained, the infection route (eg direct contact, faecal-oral transfer - particularly for grazing animals - and infection through insect vectors) and the factors that affect the likelihood of the transference of infectious agents (eg warm and damp weather may increase the transfer of any infectious disease that has a free-living life stage - such as helminth parasites - or which is transferred through an insect vector<sup>77</sup> (see also chapter 7).

Given the complexity of the potential interactions, it is unsurprising that in many cases the available information is still not sufficient to decide if a given 'disease-wildlife species-livestock' triangle is of concern for animal health authorities and wildlife managers, or, if it is a concern, how it should be treated<sup>75-76</sup>. For example, the 2001 foot-and-mouth disease outbreak in the UK showed that deer, at least at current UK densities, are not a true reservoir for this disease, because culling of infected livestock resolved the problem - if a reservoir had existed elsewhere, then livestock would have been reinfected from this source. The question remains, however, as to what would have occurred if other potential hosts (eg wild boar, *Sus scrofa*, which are free roaming across much of the south of England<sup>78</sup>) had been abundant<sup>76</sup>? Similarly, setting a threshold density above which a wildlife host becomes a 'problem' - in terms of being a competent disease reservoir - is fraught with complexity. Not all species are equally competent hosts for a disease and their densities will vary markedly. A study of the role of wild deer in the spread of bTB to cattle, for example, examined four UK species of deer (red, fallow, roe and muntjac) and concluded that even assuming virtually 100% bTB prevalence, population density would have to





## Diseases of deer

Diseases pass readily between wild and farmed deer herds in the UK as well as to other livestock species, thereby providing dual reservoirs for disease. Deer can be affected by tuberculosis, including bovine TB (bTB, *Mycobacterium bovis*)<sup>80</sup>, in addition to another species of *Mycobacterium*, *M. avium paratuberculosis*, or Johne's disease<sup>81</sup>. In both cases, these diseases result in significant economic impacts for deer farmers, with animals infected with bTB needing to be destroyed, and those suffering from Johne's disease exhibiting a loss of body condition<sup>75</sup> and, ultimately, death if untreated. Foot and Mouth Disease (FMD), caused by a virus transmitted via aerosol as well as fomites (a term for any inanimate object or substance capable of carrying infectious organisms, such as urine, faeces, skin, hair etc.), may also affect both farmed and wild deer<sup>82</sup>. Rarely fatal in itself, FMD is nonetheless highly infectious and requires, if reported, destruction of all infected herds of cloven-hoofed animals. Such control measures occur throughout the UK in 2001, with a smaller outbreak occurring in 2007, again resulting in substantial economic impacts.

Using midges as a vector for transmission, bluetongue, caused by the Bluetongue virus, infects both domestic and wild ruminants, including deer species, with infected animals exhibiting swelling, and haemorrhaging in and around the mouth and nose, as well as flu-like symptoms<sup>82</sup>.

exceed 91 per km<sup>2</sup> for red deer and 200 per km<sup>2</sup> for roe deer before maintenance host status would be achieved, whereas fallow deer may act as maintenance hosts at densities as low as 25 per km<sup>2</sup> when prevalence rates were approaching 100%, and at 75 per km<sup>2</sup> when only 30% were infected<sup>79</sup>. However, these figures are based upon assumptions about disease transmission rates that are uncorroborated, and density limits that were set according to maximum densities seen in the field at local densities rather than in the landscape as a whole<sup>79</sup>: even for a disease as well studied as bTB and species as well studied as UK deer, accurate figures that could set a threshold for an effective management response remain elusive.

In spite of the complexities, though, some trends are apparent. In particular welfare politics and consumer requirements are resulting in more extensive farming systems, in which animals are maintained in a more 'free-range' fashion. Meanwhile, wildlife populations are increasingly managed through feeding, translocations and even fencing, thus becoming more and more like extensively raised livestock. Both situations are likely to increase the exchange of pathogens or vectors<sup>76</sup>. Conversely, however, managing farms in a 'wildlife-friendly' fashion, with ungrazed wildlife strips, and a greater availability, width and continuity of hedgerow, has been shown to be associated with lower risk of bTB in cattle herds<sup>83</sup>. In this latter case it is likely that a nuance of the way in which badgers and cattle interact is modified by the presence of hedges - which provide long forage which in turn might allow cattle to avoid areas used by badgers - to reduce contact, and therefore the chance of disease transmission, between the species<sup>83</sup>.

Also, for any species to be a competent wildlife reservoir it must be abundant and likely through its distribution and

behaviour to interact, however indirectly, with livestock. For example, toxoplasmosis is one of the commonest causes of abortion, stillbirth and neonatal death in sheep in the UK<sup>84</sup> as well as infecting humans (chapter 7), and the principal source of infection for this disease is the domestic cat<sup>75</sup>. Cats themselves are primarily infected through hunting rodents<sup>75</sup> (see also Box 3), and so ultimately the wildlife reservoirs for this disease are extremely abundant UK rodent populations, even though the only true host is the cat. To ensure that cats are re-infected from their rodent prey, the *Toxoplasma* parasite modifies rodents' behaviour, apparently to make the rats easier to catch (Box 3). Abundant populations of rodents, in particular brown rats (*Rattus norvegicus*), are notably also a wildlife reservoir for leptospirosis, which can be transferred to domestic species. Whilst rarely causing mortality in cattle and pigs, leptospirosis may reduce fecundity of farm animals, for example leading to abortions, stillbirths, or the production of weaker and less viable offspring in cattle<sup>85</sup>, while in pigs infection can result in infertility<sup>86</sup>. Similarly rabbits, which are not rodents but which certainly are abundant, have become

increasingly recognised as reservoirs of disease for humans, such as *Escherichia coli* VTEC<sup>87</sup>, and for livestock, including paratuberculosis<sup>88</sup> and potentially sheep scab, a damaging mite infection of sheep<sup>75</sup>. In the latter case rabbits may be infected with populations of the mites (*Psoroptes* sp.) that may contain sub-populations of *P. ovis* which can infest sheep<sup>89</sup>, and so the question arises whether wild rabbits might be acting as a reservoir for sheep scab, especially since an initial eradication of sheep scab from the UK in 1953 coincided with the decimation of the UK's rabbit population following the introduction of myxomatosis<sup>75</sup>. Finally, the bacterium *Listeria monocytogenes*, may be commonly present in soil in molehills and is a causative agent for listeriosis. Where molehills have the potential to infect silage there is a concurrent increase in listeriosis<sup>90</sup>, especially in sheep which demonstrate symptoms including septicaemia, abortion and encephalitis<sup>91</sup>.

Meeting the criteria of abundance and likelihood, though distribution and behaviour, of transferring infectious agents, UK deer populations may be a reservoir for a large range of infectious diseases, both now and in the future<sup>74</sup>. All six species of deer resident in Britain have expanded in range over the last 30 years<sup>104</sup>, probably due to a combination of increased protection, re-establishments and introductions, land-use changes and an absence of natural predators<sup>105</sup>. Transmission of parasites and pathogens between deer and livestock probably does not result from direct contact, but rather through the distribution of parasites or pathogens, or their vectors, into the environment. Major transmission routes for

## White-nose syndrome in bats

White-nose syndrome (WNS) is the name for a group of symptoms associated with the deaths of millions of bats in North America. *Pseudogymnoascus destructans* (syn. *Geomyces destructans*), the causal agent of WNS, is a soil fungus that grows optimally at the temperatures found in winter hibernacula<sup>92</sup> and affects hibernating bats.

The fungus was most likely introduced to North America from Europe<sup>93</sup> where it was first confirmed from a bat in France in 2009<sup>94</sup>. A subsequent review found the fungus has a widespread distribution across continental Europe<sup>95-96</sup>, where it has been isolated from at least eight *Myotis* bat species<sup>95</sup>. The fungus was confirmed for the first time in the UK in July 2013. The positive cases were discovered in environmental samples collected at hibernation sites in South East England and from a live bat swabbed in hibernation<sup>97</sup>. Although *P. destructans* is present, however, WNS has not been found this side of the Atlantic. It seems likely that European bat species may have evolved immunity to the disease<sup>95</sup>. With the discovery of *P. destructans* at five sites in South East England<sup>97</sup> it is hoped that UK bats have the same immunity, but more surveillance work and research are required for this hope to be confirmed.

### BOX 3: Kamikaze rats

Microparasites and macroparasites may cause morbidity and death in their hosts through a variety of mechanisms, but all entail disrupting some function of the host's body to the parasite's advantage. An intriguing subset of diseases are those that have few adverse effects on the functioning of the host's body but instead modify the host's behaviour. *Toxoplasma gondii* (the parasite responsible for the disease toxoplasmosis) infects many vertebrate species but has only one definitive host: the cat<sup>109</sup>. Parasites in any other intermediate host (for example brown rats, *Rattus norvegicus*) need to return to the cat to complete their life cycle (although *Toxoplasma* can persist in wild rat populations in the absence of cats via congenital transmission<sup>98</sup>). Indeed, infected rats display abnormal behaviours that make them more susceptible to predation by cats, and also, incidentally, to poisoning<sup>110</sup>. *T. gondii* infected rats show higher levels of activity than uninfected rats, which may predispose them to be attractive to cats (which are attracted to moving objects, but show less interest in stationary ones)<sup>111</sup>. No such increase in activity was observed for rats infected with parasites that do not require a definitive host to complete their life

cycle (*Leptospira* spp., *Cryptosporidium parvum*, *Coxiella burnetti*, *Hymenolepis nana*, *Syphacia muris*) and for which predation would result in death of both host and parasite. Further, whereas uninfected rats show a (completely understandable) avoidance of cat-scented areas, infected rats not only failed to avoid these areas, but actually showed a significant (apparently suicidal) preference for them<sup>112</sup>. Rats infected with *T. gondii* are also less cautious about novel food-related items, more likely to be trapped and more likely to approach a mildly fearful object than are their uninfected counterparts<sup>98</sup>. In essence, the parasite alters the rats' behaviour in a way that increases the likelihood of the rats being predated by the definitive cat host, and therefore multiplying.

These effects are not limited to rats. Similar effects are known in humans: links have been demonstrated between *Toxoplasma* infection and personality, psychomotor performance (which may lead to increased risk of traffic accidents<sup>113-114</sup>), as well as a number of psychiatric disorders such as depression, anxiety and schizophrenia<sup>115</sup>, and the probability of committing suicide<sup>116</sup>.



## Diseases of rats

Despite the risk of disease to humans associated with commensal Norway rats (*Rattus norvegicus*), prior to the mid-1990s this area had received little attention. An examination of 510 wild rats across the UK, found that wild rat populations carried at least 13 zoonotic and 10 non-zoonotic parasitic species, few of which had been investigated in UK rats previously, and that individual rats simultaneously carried between two and nine potentially zoonotic parasites<sup>33, 98-100</sup>. All zoonotic parasites identified can cause serious disease in humans and/or domestic livestock. For example, listeriosis commonly causes encephalitis in ruminants, septicaemia and liver damage in other mammals, and in humans is particularly dangerous for pregnant women. The most prevalent parasite detected was *Cryptosporidium parvum* which can trigger enteritis and enterocolitis in mammals (including humans)<sup>101</sup>. Another protozoan parasite, *Toxoplasma gondii*, was also detected at high levels. Toxoplasmosis causes human congenital abnormalities<sup>102</sup> and is estimated to cost the UK sheep industry £12m-£24m due to a loss of 0.5m lambs per year<sup>103</sup>.

many macro- and microparasites are both faecal-oral and urinary-oral routes, especially where deer and livestock share access to agricultural pastures, in which ingestion or investigation of forage contaminated with faeces during grazing may lead to transmission opportunities<sup>74</sup>. Diseases of deer that have significance for livestock include bovine tuberculosis, bovine viral diarrhoea virus, Johne's disease, Louping ill and tick-borne fever (TBF), and a variety of helminth parasite infections<sup>74</sup>. As with foot-and-mouth, while it is almost certain that some transmission of these diseases has occurred between livestock and the deer population, it is unclear either

for these diseases, or any of the many other shared parasites and pathogens, whether the deer population acts as a sufficiently competent reservoir to permit an outbreak<sup>74</sup>. For example bTB in UK deer has been linked to its presence in other species, especially badgers<sup>106</sup>, but data on the role of deer and the epidemiology of bTB in livestock are lacking<sup>107</sup>. It is worth noting, however, that while deer may not be proven competent hosts for many diseases, they certainly *are* heavily implicated in providing a major wild reservoir for the tick vectors (*Ixodes* sp.)<sup>108</sup> of several infections, including Louping ill and TBF (which are both significant infections of sheep, respectively causing fatalities and sterility/abortion;<sup>74</sup> and Lyme disease (which infects humans; chapter 7).

Clearly it is important to have a proper understanding of the role of wildlife as reservoirs of infection before embarking on costly disease-eradication programmes in domestic species<sup>75</sup>. With expanding deer populations another outbreak of foot-and-mouth disease could bring with it the possibility of spill-over into the deer population, and if this occurred controlling the disease may require not only destruction of infected livestock but also deer management to prevent spill-back from the new wildlife host. Similarly if wild boar populations become abundant then diseases such as classical swine fever - which was eradicated from Great Britain in 1966 but which has since made several comebacks including one serious outbreak in East Anglia in 2000, affecting 16 farms - may pass to a wildlife reservoir, severely complicating disease control measures. Equally clearly, sufficient data on the capacity for British mammals to act as reservoir species for livestock, or to suffer as a result of spill-over from livestock, is lacking in the vast majority of cases. This is perhaps unsurprising given the melting pot of farm types (intensive through to 'wildlife-friendly' and organic), stocking practices, stocking densities, distributions and densities of wild mammals and commensal species (particularly rats), and how each of these factors affects the behaviour, interactions and likelihood of disease persistence and transference between species.

## 5. Badgers and bovine tuberculosis

How best to control the scourge of bovine tuberculosis (bTB) in British cattle, and especially how to manage the role of badgers in infecting cattle, is amongst the most challenging wild mammalian disease problems for science and society. Although much remains to be discovered, more is known about the ecology of this disease than any other in the UK, and there are good summaries of the evidence. For example, in last year's SOBM, Macdonald and Burnham (2012) summarise the background up to the point when the 2013 badger culls (which they forecast to be 'unpromising') began. More recently Godfray *et al*<sup>117</sup> provided a restatement of the natural science evidence base relevant to the control of bovine tuberculosis in Great Britain, so here we can do no better than summarise their review.

Bovine tuberculosis is an infectious disease of cattle caused by the bacterium *Mycobacterium bovis*. In Great Britain it costs farmers and the taxpayer heavily, through testing and compensation for slaughtered animals. Both the incidence and geographical distribution of bTB in cattle have increased in England and Wales since the mid-1980s. Across much of Britain herds are tested annually: where infection is detected, infected animals are destroyed, cattle sales and movements are restricted and contacts of the infected herd traced. In 2012, for example, 37 068 cattle were destroyed after testing positive, and a further 943 close contacts were also slaughtered. Such breakdowns (abrupt collapses in disease status) of bTB in areas of low incidence tend to be associated with cattle movements from high incidence areas. It remains a puzzle, however, why some regions contain areas with high bTB incidence (many parts of Wales, the Midlands and the West Country) but others do not (east and north England, Scotland). Added to this, farms that have had a herd breakdown suffer a recurrence of the disease more often than expected by chance, while many farms in high incidence areas escape infection much more often than would be expected by chance. Similar recurrence is a

relatively rare event in low incidence regions.

This understanding of factors governing the geographical distribution and incidence of bTB, and what stimulates a breakdown, remains incomplete. Two further, and substantial, impediments to the control of bTB are the limited sensitivity of diagnostic tests, and the involvement of badgers.

### Testing and surveillance

Methods of diagnostic tests for bTB infection in cattle are neither 100% sensitive (100% sensitivity means that the test gives no false negatives, ie that no infected animals are missed), nor 100% specific (specificity being a measure of the percentage of uninfected animals that are incorrectly identified as infected, creating false positives). The single intradermal comparative cervical tuberculin test (SICCT or 'skin' test) has a herd-level *specificity* above 99% and herd-level *sensitivity* of 51% (meaning that it is unlikely to give false positives, but may miss infected animals). The skin test relies on a somewhat subjective interpretation of the relative size of two lumps generated by an immunological response in the skin. The gamma interferon (IFNg) test has lower specificity (96%) and higher herd-level sensitivity (67%). Cattle movements, especially from high-incidence areas, are associated with increased risk of bTB infection, and the skin test is a valuable tool in reducing this risk through pre-movement testing. A live test for bTB in badgers (Brock TB StatPak) has a sensitivity of about 50%.

### Badgers and bTB

Although both badgers and cows tend to do well in places with mixed pasture and woodland, there is little evidence of an association between high badger densities and elevated cattle TB incidence. Nonetheless similar genotypes of *M. bovis* are found, more often than would be expected by chance, in local cattle and badger populations. Much was learnt from the Randomised Badger Culling Trial (RBCT), which took place between

Time period	Proactive culling area		Area surrounding cull	
	Central estimate (%)	95% Confidence interval	Central estimate (%)	95% Confidence Interval
During trial	-23	-12 to -33	+25	-1 to +56
After trial	-28	-15 to -39	-4	-26 to +24
Entire period	-26	-19 to -32	+8	-14 to +35

Table 1. Averages and confidence intervals of percentage difference in new confirmed herd breakdowns between sites subjected to proactive culling, compared with no-cull areas: data for the during trial time period, the after trial period and for the entire time period; reproduced from Godfray *et al*<sup>117</sup>.\*

\* These figures are a comparison of cull and non-cull sites, and hence represent relative differences. As background incidence was rising throughout the monitoring period, absolute reductions in rates of new confirmed cattle herd breakdowns (compared with historical rates) would be smaller than the relative reductions shown here, and absolute increases would be larger than the relative increases shown here.



## Badgers, parasites and nutrition

Badgers are notoriously susceptible to bovine tuberculosis, but also suffer a variety of other infections. For example, the coccidial gut parasites *Isospora melis* and highly pathogenic *Eimeria melis* occur commonly among badgers<sup>119</sup>. *E. melis* causes infected cubs to lose fluid and suffer malabsorption and anorexia<sup>120</sup>. In our studies, we found an *E. melis* prevalence of 66.4% in cubs in their first year, compared with 8.5% in adults. When these infections coincide with shortages of food and water, cub mortality rates peak at over 90% , where mortality is due to malnutrition<sup>120-121</sup>, whereas in years of plenty, over 50% of the cohort will typically survive<sup>122</sup>. Cubs surviving infection typically suffer stunted growth: the most heavily infected (surviving) males attain adult body-lengths that are typically 5 cm shorter (7% of 70.5 cm), and females 3.5 cm (5% of 670 cm) shorter, than the least severely infected<sup>120</sup>. This is because male cubs grow faster than females, and thus suffer a greater impairment due to coccidiosis as a trade off between combating the infection and investing in skeletal development<sup>120</sup>. This example highlights the ability of diseases to have unexpected population-level impacts, particularly when combined with fluctuations in other environmental parameters. Dry spring weather and gut parasites may seem to be relatively innocuous but together can produce severe malnutrition resulting in widespread mortality amongst badger cubs and therefore a decline in the size of badger populations.

1998-2005 and which estimated that 50% of confirmed herd breakdowns in the year before culling began were due to badgers, but that the percentages varied widely between areas. Furthermore, cattle can infect badgers too, and it isn't known whether TB could persist in badgers without infection from cattle. In the proactive culling areas of the RBCT (where bTB incidence was high), post-mortem and culture examination of badgers revealed 2% to 38% (mean 14%) prevalence, though more than half the infections may escape detection. Exactly how *M. bovis* is transmitted between badgers and cattle is unknown.

### Culling badgers

The RBCT found that proactive culling resulted in a reduction in the rate of new confirmed cattle herd breakdowns inside culling areas (which diminished over the six years; Figure 2; Table 1), but a parallel increase in the incidence of confirmed herd breakdowns within 2 km of the culling areas (which waned after culling stopped; Figure 2; Table 1). Reactive culling appeared to make herd breakdowns significantly worse (the presence and extent of badger culling were associated with increased risk of a confirmed herd breakdown on nearby farms, and when compared to no-cull areas the breakdowns were more prolonged). The perturbation effect (in which the disruption to badger populations from culling alters the survivors' behavioural ecology, and perhaps immunology, resulting in increasing spread of the disease)<sup>118</sup>, may explain why culling consistently increased the prevalence of *M. bovis* infection in badgers, particularly in culling areas surrounded by weaker barriers to badger movement, on land close to culling area boundaries, and following proactive culls which were not conducted simultaneously across the entire area.



The reductions in cattle bTB achieved by the RBCT proactive culling may have arisen from a number of facets of the trial and the way in which it was conducted: the culling resulted in an approximately 70% reduction in badger density and badger immigration was limited by the use of geographical barriers; also the culls were conducted simultaneously across entire areas and repeated annually over at least four years with access to most (about 70%) of the necessary land, with inaccessible areas targeted. If culling, mimicking the RBCT, were extended over larger geographic areas this might be expected to move the balance between benefits (the reduction in herd breakdowns in the culling areas) and costs (the increased breakdowns in peripheral areas) towards a net benefit. An analysis assuming a circular 150km<sup>2</sup> area and proactive culling similar to that carried out in the RBCT predicted that over a 9.5 year period with proactive culling in the first five years there would be a relative reduction in confirmed herd breakdowns of 20-34% (central figure 27%) within the culled area. When the additional herd breakdowns in a peripheral 2 km area are included, the overall reduction falls to 3-22% (central figure 12%) or 8-24% (central figure 16%), depending on assumptions (Table 1 separates these figures for culled and peripheral areas). Such a prediction, however, requires a target 70% reduction in badger densities, a

figure that the two recent trials in 2013, in Somerset and Gloucestershire, fell sadly short of - even after extensions they killed only 65% and 39%, the latter of which, in Gloucestershire, approximates the situation known to deliver the worst possible outcome. Following this epic failure it is hard to see how continuing this approach could be justified.

### Vaccination

There are two targets for vaccination: the cattle and the badgers. BCG vaccine (a live attenuated strain of *M. bovis*) reduces the severity of disease in cattle (in one trial by 56%-68%), but also leads to false positives using the skin test (resolved by tests that Differentiate Infected from Vaccinated Animals (DIVA) (95% relative sensitivity, 96% specificity)). An injectable BCG vaccine reduces the risk of vaccinated badgers testing positive to a test of progressed infection (ie becoming diseased) by 74%, and of testing positive to live tests by 54% (when more than a third of the social group was vaccinated, the risk to unvaccinated cubs was reduced by 79%). Importantly, trapping and injecting does not lead to perturbation. Trials with oral vaccine suggest that they too can reduce the severity of the disease in vaccinated badgers.

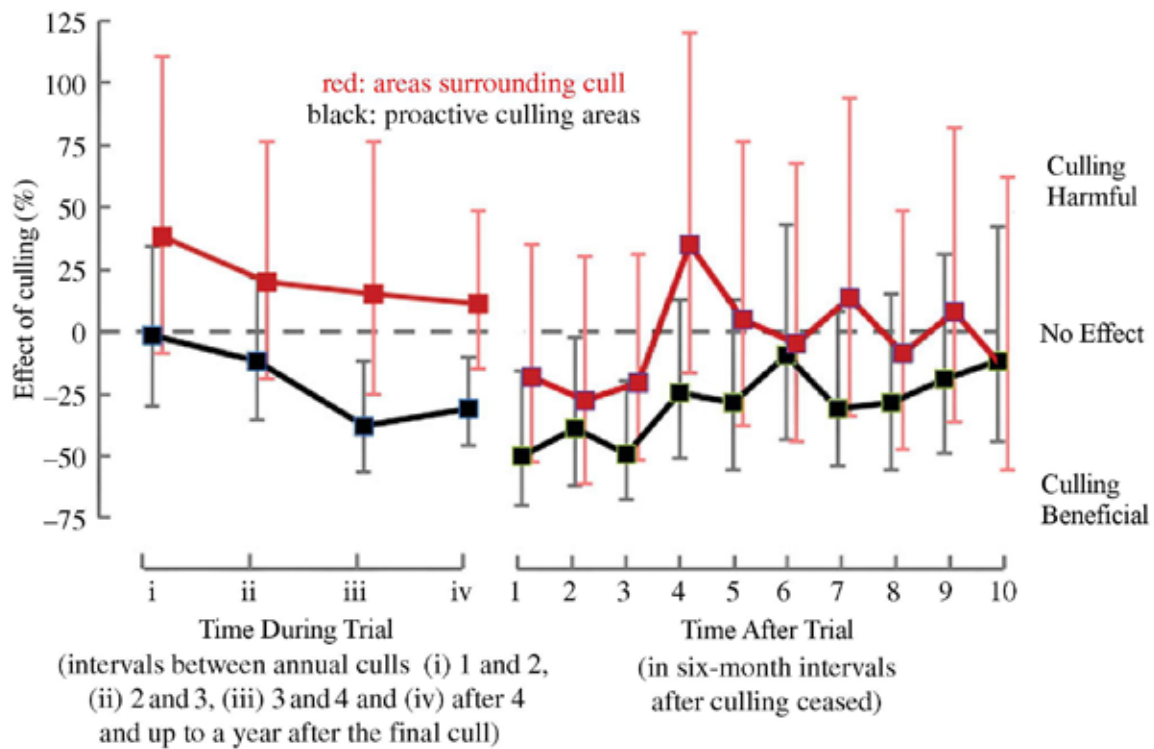


Fig. 2. Results from the RBCT proactive culling, reproduced from Godfray *et al*<sup>117</sup>. The black lines show the percentage difference (with 95% confidence limits) in new confirmed herd breakdowns between sites subjected to proactive culling compared to no-cull areas\*. The red lines show the same information for lands up to 2 km outside the proactive culling area compared to land up to 2 km outside the no-cull trial areas\*. Averages for each period are presented in Table 1.

## 6. Disease and animal movement

There is nothing new about humans moving other animals around the globe<sup>123</sup>. Rats and mice, for example, have been commensal with humans since Neolithic times<sup>124-125</sup> and as a result have been inadvertently naturalised on islands and archipelagos worldwide<sup>126</sup> (where they continue to impact heavily on endemic fauna<sup>127</sup>). What is novel, however, is the modern scale of global movements of both wild and domestic animals. In 2007 (the most recent year for which data are available), global international exports of cattle were nine million individuals and of sheep were 14.6 million individuals<sup>128</sup>. The UK's 2007 contribution to these movements was to import over 20 200 head of cattle, 68 200 sheep, 432 100 pigs and 5.9 million chickens, and to export over 71 600 pigs, 31 000 chickens, 20 600 cattle and 1 000 sheep. Similarly, although precise estimates are difficult because much wildlife trade occurs through informal or illegal networks, up to 40 000 live primates, four million live birds, 640 000 live reptiles and 350 million live tropical fish are traded globally each year<sup>129-130</sup>. Additionally wildlife may be moved for conservation reasons: for the reintroduction of endangered species, the translocation of animals, often for species protection in areas undergoing development work, or for bringing injured or orphaned individuals into captivity for treatment - in the UK hedgehogs<sup>131</sup> and bats<sup>132</sup> are frequently taken into captivity for rehabilitation.

An important consideration when moving wildlife - and livestock, although these are subject to stringent veterinary examination for a number of notifiable diseases (chapter 8) - is that any individual animal actually represents an entire 'biological package', comprising the host animal and a plethora of passenger organisms, including viruses, bacteria, fungi and a range of additional parasites and pathogens<sup>130, 133</sup>. Given that an unknown, but substantial proportion of international wildlife trade is illegal<sup>130, 134-135</sup> - representing, by some measures, the second largest illegitimate global business after narcotics<sup>136</sup> - this trade represents a severe risk of 'pathogen pollution', the human-induced movement of infectious agents to new regions<sup>137</sup>. In essence any movement of animals from a given geographical location to another may increase the risk of disease transmission to and from both wildlife and livestock, potentially rendering all individuals in the community at risk of contracting a novel disease<sup>138</sup>. An obvious example is the devastating effect of crayfish plague carried by signal crayfish on UK populations of white-clawed crayfish (chapter 3). Similarly the movement of amphibians both for the pet trade and for reintroduction for wildlife conservation is known to have facilitated the spread of chytridiomycosis on a global scale<sup>130</sup>. This disease was caused by the aquatic fungal pathogen *Batrachochytrium dendrobatidis* (*Bd*) and initially spread via international trade in African clawed frogs (*Xenopus laevis*) resulting in a 30% decline in amphibian species worldwide<sup>139</sup>. Currently there is no obligation for amphibians to be screened for the pathogen, either for entry into the UK or more widely throughout Europe<sup>140</sup>.

Such health concerns are not limited only to native species that are closely related to those introduced; rather the introduction of any species can have unforeseen knock-on consequences for entirely unrelated species. The discovery of an opisthorchid fluke parasite (*Pseudoamphistomum truncatum*) in English otters (*Lutra lutra*) has been linked to the introduction of two intermediate fish host species, the sunbleak (*Leucaspius delineatus*) and the topmouth gudgeon (*Pseudorasbora parva*)<sup>129</sup>, both of which have become established in a number of river systems in southern England after escaping from an ornamental fish supplier in the mid-1980s<sup>141</sup>. Also the risk of emerging zoonosis from wildlife movements is large (chapter 7) and any wildlife movement, even for conservation purposes, carries the potential to negatively impact human health. Bavarian beavers (*Castor fiber*) can carry a parasitic tapeworm, *Echinococcus multilocularis*, which is the causative agent of the highly lethal human disease alveolar echinococcosis<sup>142</sup>. This parasite has been identified in Great Britain<sup>143</sup> and if any of the illegally released Tayside feral beavers are infected - which in 2012 were thought to number 146 individuals in the wild, living in at least 38 separate family groups<sup>144</sup> - the parasite could become established in Scottish wildlife. The parasite does not occur in mainland Norway<sup>145</sup>, and so Norwegian beavers (the source population for the experimental reintroduction trial in Knapdale in Scotland) are not carriers. Faced with such potential for the spread of novel and deleterious diseases to a range of unintended hosts it is perhaps only slightly comforting that not all wildlife introductions result in increased disease risk: in Ireland, introduced bank voles were found to be responsible for a decline in *Bartonella* haemoparasites, transmitted via fleas to native wood mice, believed to be due to the role of bank voles in providing an increase in the number of alternative hosts for infected fleas<sup>146</sup>.

Reintroductions for wildlife conservation represent a disease risk not only to established wildlife<sup>153</sup> but also to the released animals themselves. Common environmental parasites and pathogens can have significant negative effects on reproduction and survival, thereby reducing the likelihood of establishment for the new population, and insufficient disease risk management has caused several reintroduction programmes to fail<sup>154</sup>. Captive reared animals, which are relatively free of common environmental pathogens may be particularly at risk<sup>155</sup>, lacking the immunity to combat otherwise benign infections. For example, 43% of reintroduced water voles and their offspring were found to be infectious for leptospirosis four months post-reintroduction, compared with a typical incidence of 6.2% in wild water voles<sup>156</sup>, suggesting that they may have been more susceptible to acquiring leptospires. An additional effect of captivity occurs if individuals are kept under inappropriate conditions leading them to become stressed and immunocompromised. Prior to reintroduction the water voles in the above study were housed by the breeder



## Coronaviruses and bats

There is increasing evidence that European bats carry a range of viruses. While these are not known to be zoonotic (ie can be transmitted to humans), in many cases they belong to families that do include viruses that are able to cross the species barrier. Coronaviridae is one such family and has received a lot of attention since the emergence of severe acute respiratory syndrome (SARS-CoV) a decade ago<sup>147</sup> and more recently Middle-Eastern respiratory syndrome (MERS-CoV)<sup>148</sup>. Coronaviruses cause a range of problems in humans including respiratory and gastrointestinal diseases<sup>147, 149</sup> (human coronavirus is one cause of the common cold) and can be divided into three groups: alpha (Group 1), beta (Group 2) and gamma (Group 3). Both SARS-CoV and MERS-CoV belong to betacoronavirus, Group 2b. Bats have been identified as reservoirs of SARS-like coronaviruses in China<sup>147,150</sup>. A recent study has also found betacoronaviruses in European bats, including isolates from common pipistrelles (*Pipistrellus pipistrellus*) in the Netherlands, and alphacoronaviruses present in common pipistrelles, and three other species (Daubenton's bat, *Myotis daubentonii*; noctule, *Nyctalus noctula*; and pond bat, *M. dasycneme*)<sup>151</sup>. A UK study recently isolated alphacoronaviruses from a Daubenton's bat along with a Natterer's bat (*M. nattereri*)<sup>152</sup>. It is not known if any of these viruses are zoonotic but these discoveries highlight the need for surveillance programmes for this family of viruses in wildlife.

in laboratory cages containing between one and eight individuals. The water voles' ability to mount an immune response was inversely related to the number in the cage; given that water voles are normally territorial and solitary, the high density cages were likely to have been stressful<sup>157</sup>, and even for individually housed captive water voles both too-confining housing and the attachment of radio-collars for monitoring purposes are known to negatively impact on water voles<sup>158</sup>. With a weakened ability to combat infections such individuals are substantially more at risk of illness and death, particularly because reintroduced individuals may move around more and contact many more individuals when becoming familiar with their new environment and establishing territories<sup>159</sup>, all of which increases the potential for disease transmission.

The above arguments also apply to rehabilitated wildlife. There are a large number of wildlife rescuers and rehabilitators in the UK, both individuals and institutions, who take in injured or abandoned wildlife with a view to eventually releasing these animals in better condition. These animals are typically kept in unnaturally close confinement, both with conspecifics and with their human carers, and so risk obtaining a disease which is then released into the wild with the individual<sup>160</sup>. Co-housing amphibians, for example, has been shown to amplify the population prevalence and intensity of infection with *Batrachochytrium dendrobatidis*<sup>140</sup>. This is not to imply that such ventures do no good - UK hedgehogs maintained in captivity for at least one month post-rescue have a greater chance of longer survival when re-released than those maintained in captivity for less than one month<sup>131</sup> - but rather that such activities carry an inherent disease risk that is difficult to detect and mitigate.

Disease screening appears to be sensible not only for livestock but also for any human-mediated wildlife movement. Worldwide, however, 24% of translocations for conservation have no disease screening, and only 25% of mortality cases post-translocation are investigated<sup>10</sup>. While a 'zero-risk tolerance' philosophy for wildlife movement with respect to disease may be desirable, this has unfortunately proved unattainable for nearly all wildlife-conservation programmes<sup>161</sup>. The term 'disease screening' is often used to describe the examination of animals to detect disease problems but screening for all known parasites and pathogens is practically impossible (Box 4). There are hundreds of parasites and pathogens that could infect a single species<sup>133</sup> and the vast majority of any such diseases identified are likely to be benign under most circumstances. Nevertheless prevention remains the most cost-effective method of disease management<sup>162</sup>, and the current strategy for livestock movements is to screen for selected diseases known to have severe impacts (chapter 8). It is clear that similar approaches to prevention are equally desirable for wildlife movements.

## 7. Zoonotics

---

Transmission of pathogens to humans from other species is a natural feature of ecosystems and our engagement with them<sup>11</sup>, and such zoonotic infections form the majority of human diseases<sup>12</sup>. Of 1415 human diseases identified in a worldwide review in 2001, 868 (61% of all diseases) were zoonotic<sup>12</sup>. In the UK, zoonoses can be divided into three broad areas of concern. The first are those zoonoses which currently exist in wildlife reservoirs in the UK, and which therefore can be contracted by people coming into contact with infective material passed on from the wild host. The second are those which exist overseas in wildlife and which may be transported into the UK either through natural processes (eg via migratory species or those able to traverse the English channel) or importation of infected animals. The third are emerging infectious diseases, transmitted to individuals within the global human population as a novel infection from animal hosts, which may or may not have the potential to precipitate an epidemic, with infection spreading around the world through normal channels of human movements<sup>24</sup>.

### Endemic UK zoonoses

Endemic UK zoonoses are those which have existed in the UK for a number of years and, while potentially serious for individuals if contracted, are unlikely to result in mass mortality. There are currently approximately 40 potential zoonoses in the UK<sup>163</sup>, the most common of which are listed in Table 1. Human risk groups inevitably comprise people whose occupations involve working with animals. The Health and Safety Executive identifies approximately 300 000 people in a variety of occupations who are potentially exposed<sup>163</sup>, with farm workers

being particularly at risk, due to their close contact with livestock<sup>164</sup> (Table 1).

The most prevalent zoonotic diseases in the UK in 2011 (Table 1) were campylobacteriosis (72 150 confirmed human cases), salmonellosis (9 455 cases), cryptosporidiosis (3 655 cases), vero cytotoxin-producing *Escherichia coli* (1 407 cases) and Lyme disease (1 201 cases)<sup>165</sup>. It is worth noting, however, that many instances of zoonotic infection remain unreported. For example the ratio of unreported to reported human *Campylobacter* infection is estimated as 9.3 to 1, suggesting that in 2011, there were approximately 740 000 *Campylobacter* cases<sup>166-167</sup>. Similarly, the actual number of cases of cryptosporidiosis in the UK in 2011 is likely to have been ~ 34 000<sup>166-167</sup>. Of these five zoonoses, the four most common are infections of the digestive tract, which are hosted primarily in livestock populations and secondarily in domestic pets and wildlife, and which are most frequently passed to humans through consumption of contaminated food products or through direct contact (principally farm workers) (Table 1). The fifth, Lyme disease, differs in that the majority of cases are acquired by members of the general public when pursuing outdoor recreational activities, through the bite of infected ticks (*Ixodes* species) which act as vectors for the causal bacteria, all species within the genus *Borrelia*<sup>165</sup>. Again unlike the top three zoonoses, the four species of *Borrelia* that occur in Britain are maintained largely in wild populations of animals. *B. garinii* and *B. valaisiana* are maintained in birds, ranging from guillemots (*Uria aalge*) to blackbirds (*Turdus merula*)<sup>168</sup>, and the principal hosts for the other species *B. afzelii* and *B. burgdorferi*, are grey





## Bats and European Bat Lyssavirus

The majority of work on disease in UK bats has focused on European Bat Lyssavirus, of which there are two strains EBLV1 and EBLV2. Over 11 500 bats have been tested for EBLV since surveillance began in 1987<sup>170</sup>, and a total of 10 bats, all Daubenton's (*Myotis daubentonii*), have been found with live EBLV2 virus. Of these, the first was captured in 1996 but it was suspected the individual had originally come from the continent<sup>170</sup>. However, in 2002 a juvenile captured in Lancashire and raised in captivity tested positive, providing definitive evidence for rabies in island Britain. In a separate incident in 2002, a Scottish bat worker who had not been vaccinated against rabies died of EBLV2a<sup>171</sup>, and so the disease is now known to be able to spill-over to humans, as well as to domestic livestock and other wildlife<sup>172-173</sup>. Elsewhere in Europe there have been four other human cases of EBLV in humans as well as spill-over of EBLV1 into one stone marten and two sheep<sup>170-171, 173</sup>. Active surveillance work has identified a small proportion of Daubenton's bats with antibodies to EBLV2, indicating previous exposure to the disease<sup>174</sup>. Additionally antibodies to EBLV1 have been found in a serotine (*Eptesicus serotinus*) and two Natterer's bats (*Myotis nattereri*) but the live virus has not been found in the UK<sup>175</sup>.

squirrels and rodents like mice and voles<sup>168-169</sup>. Wildlife is a sufficiently effective reservoir for this zoonosis that a recent study suggests that oral immunisation of wildlife to Lyme disease may be a long-term strategy to reduce human Lyme disease risk<sup>169</sup>.

Wildlife plays key roles in the maintenance and transmission of other notable zoonotic diseases.

Toxoplasmosis (the 7<sup>th</sup> most reported zoonosis in 2011, with 364 human cases) is principally passed to humans through contact with water, food or soil contaminated with the faeces of infected cats<sup>165</sup>; but rats, mice and voles represent a significant reservoir, from which cats receive the infection, for the intermediate stage of the parasite<sup>176</sup>. Brown rats (*Rattus norvegicus*) are further implicated in acting as a reservoir for a number of diseases including Leptospirosis, Cryptosporidium, Pasteurella, Listeria, Yersinia, Coxiella and Hantavirus<sup>32</sup>. Of these rat-borne diseases several have the capacity to cause serious illness and even fatalities in humans. Leptospirosis (52 reported cases in 2011) can give rise to Weil's Disease (which develops in the small proportion of extreme cases where complications result in multiple organ failure<sup>177</sup>). *Coxiella* (which causes Q fever; 112 cases in 2011) and Hantavirus (one reported case in 2011), can both cause non-specific, influenza-like symptoms in humans, which can be fatal if misdiagnosed, or left untreated<sup>178</sup>. While rats are almost certainly not the sole reservoir for any of these diseases - for example water voles have also been shown to be wildlife hosts for Leptospirosis<sup>21</sup> and *Coxiella* infects a great range of animal species and can be maintained solely in livestock populations<sup>75</sup> - they are commensal with humans and extraordinarily abundant, and therefore likely to provide a substantial interface for the transfer of disease to both humans and livestock<sup>179</sup>.

### Zoonoses within dispersal distance

The UK has remained free of many diseases that are routinely transmitted by wildlife between countries that share land borders. Migratory wildlife species able to cross the channel, however, represent a risk in terms of the introduction of novel zoonoses. For example avian influenza (H5N1), which originated in water bird species in the Far East, was detected in the UK in 2006 when an infected Whooper swan (*Cygnus cygnus*) was found in Scotland, and again in 2007 in Suffolk in a domestic poultry unit, probably transmitted via contact with wild birds<sup>180</sup>. In 2002 European Bat Lyssavirus 2 (EBLV2) was identified as potentially being carried by UK bats after a captive Daubenton's bat (*Myotis daubentonii*) developed abnormal behaviour. Subsequent research suggested this

**TABLE 1** Laboratory confirmed cases of zoonotic disease in humans and animals in the UK 2011. From Defra 2011 Zoonoses Report

Disease/causative agent	Humans			Animals (livestock, domestic and wild)		Notifiable?*
	Notifiable?†	Reported cases in 2011	Principal sources of infection	Typical route of infection	Reported cases in 2011	
<i>Campylobacter</i>	Yes†	72,150	Livestock > Domestic animals > Wildlife	Contaminated food or water (faecal-oral route)	407	No
<i>Salmonella</i>	Yes†	9,455	Livestock > Wildlife	Consumption of contaminated food	2,671	No
<i>Cryptosporidium</i>	Yes†	3,655	Livestock > Wildlife	Contaminated food or water Contact with animals or faeces Direct person-to-person contact	1,381	No
VTEC O157 ( <i>E. coli</i> )	Yes†	1,407	Livestock	Contaminated food and water Direct contact with livestock Person-to-person contact	No data	No
<i>Lyme Disease</i>	Yes	1,201	Wildlife > Livestock	Tick bite	NA	No
<i>Pasteurella</i>	No	668	Domestic animals	Contact with domestic pets	316	No
<i>Toxoplasma</i>	No	364	Domestic animals	Food or water contaminated with cat faeces Consuming or handling raw meat.	146	No
Listeriosis	Yes†	164	Ubiquitous in environment	Unwashed / uncooked food	145	No
Q Fever	Yes	112	Livestock > Domestic animals	Consumption of unpasturised milk. Direct contact with infected animals	7	No
<i>Taenia spp.</i>	No	94	Livestock > Wildlife	Consumption of undercooked meat	No data	No
Yersiniosis	No	55	Livestock > Wildlife	Ingestion of food contaminated with faeces of infected animals.	22	No
Leptospirosis	Yes	52	Livestock > Wildlife	Contact with urine from farm animals and commensal wildlife (particularly rodents)	3	No
Psittacosis ( <i>C. psittaci</i> )	Yes	41	Pets and Wildlife	Contact with pet and wild bird species	8	No
<i>Mycobacterium bovis</i> ***	Yes	31	Livestock > Wildlife	Consumption of unpasturised dairy products	6528 (144)	Yes
<i>Brucella sp</i> (Brucellosis)	Yes	25	Livestock	Consumption of unpasturised dairy products, particularly outside of the UK	71	Yes
<i>Hydatid disease</i>	No	15	Livestock	Contact with dog faeces - farm dogs and sheep perpetuate cycle	0	No
<i>BSE58 / vCJD53</i>	No	5	Livestock	Linked to the ingestion of BSE infected beef	7	Yes
<i>Toxocara</i>	No	4	Domestic animals	Direct contact with infected dogs and cats	No data	No
Hanta virus	Yes	1	Wildlife	Wild / commensal rodents		No
Orf	No	1	Livestock	Direct contact with infected livestock	35	No
<i>Streptococcus suis</i>	No	1	Livestock	Contact with infected pig meat	119	
Anthrax	Yes	0	Varied	Varied, but e.g. contaminated recreational drugs, imported animal products	0	Yes
Avian influenza	Yes	0	Livestock > Wildlife	Poultry	0	Yes
Chlamydiosis	No	0	Livestock	Inhalation of aerosols in proximity to infected animals	451	No
Rabies 'Classical'	Yes	0	Livestock > Wildlife	Direct contact with livestock and wildlife	0	Yes
Rabies EBLV	Yes	0	Wildlife	Direct contact with infected bats	0	NA
<i>Trichinella</i>	No	0	Livestock	Consumption of raw or undercooked meat	0	No
West Nile Virus	NA	0	Wildlife	Mosquito vectored from wild birds	NA	Yes
Swine Influenza	Yes	NA	NA	NA	35	No

\* List of human notifiable diseases at <http://www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/NotificationsOfInfectiousDiseases/ListOfCausativeAgents/>

†Notifiable as "Food poisoning"

\*\* List of animal notifiable diseases at <http://www.defra.gov.uk/ahvla-en/disease-control/notifiable/#measure>. See XREF 8.

\*\*\* Numbers in brackets represent non-bovine sources, excluding badgers

---

form of EBLV2 was closely related to that found in bats in the Netherlands, indicating that mixing between bat populations from both countries occurred<sup>174</sup>. Zoonoses and wildlife diseases may also be carried across the English channel by insects. Schmallenberg virus (SBV - associated with fever, reduced milk yields, still births and birth defects in livestock) has been confirmed in UK livestock samples in the UK since 2012 but only in areas at risk of midge incursion from northern mainland Europe during the summer/autumn 2011. This finding suggests that livestock were probably infected by midges blown across the English Channel<sup>181</sup>. The implications for UK human health if malaria-bearing mosquitoes spread into Northern Europe are that we may be unlikely to escape the spread of this disease.

### Emerging infectious diseases

Many zoonotic diseases are extremely prevalent in the UK human population, but those that can cause serious illness and/or death (eg avian influenza, rabies, anthrax) currently have very low rates of occurrence in the UK (Table 1). However, there remains a consistent threat from emerging infectious disease to the global human population<sup>11-12, 182</sup>, including to the UK. A recent study reported the emergence of 335 infectious diseases (EIDs) in the global human population between 1940 and 2004 of which the majority (60.3%) of EID events were caused by zoonotic pathogens and 71.8% of these zoonotic EID events were caused by pathogens with a wildlife origin<sup>182</sup>. Recently emerged zoonoses include, for example, Nipah virus (passed from fruit bats to pigs and thence to humans and dogs;<sup>10</sup>), hantavirus pulmonary syndrome (acquired from wild rodents, particularly deer mice *Peromyscus* sp. in the USA), monkeypox (which, despite the name, is typically held in a rodent reservoir in West Africa, and was introduced to the USA by an exotic animal dealer whose shipment contained infected rope squirrels, *Funisciurus* sp., Gambian rats, *Cricetomys* sp., and dormice, *Graphiurus* sp.<sup>183</sup>. The resultant outbreak infected 71 people in the USA in 2003), severe acute respiratory syndrome (SARS, which emerged as a result of the handling and butchering of wildlife for meat: the original infection emerged in wildlife market and restaurant

workers in southern China<sup>184</sup>), Ebola (outbreaks of Ebola amongst humans occur through the handling of wild animal carcasses - most often for bushmeat, and typically of gorillas and chimpanzees, but also deer species - in the forest zone between Gabon and Republic of Congo. Human outbreaks are typically preceded by an outbreak in wildlife<sup>185</sup>) and simian immunodeficiency virus (the animal precursor to HIV)<sup>11</sup>. Some of these zoonoses have become established as human pathogens that do not require repeated animal-to-person transmission (eg HIV<sup>11</sup>), and others, such as SARS could have established but were contained by rapid global response to their emergence<sup>186</sup>. All of these diseases can be fatal. To give just one example, the Nipah virus, which has fruit bat reservoir hosts in Malaysia, became established in domestic pig populations leading to an outbreak in humans, mainly those involved with pig farming or abattoir working<sup>187</sup>, in 1998-99 which led to the deaths of more than 100 people in peninsula Malaysia and Singapore and the destruction of one million pigs<sup>11</sup>.

It is unlikely that an EID will arise from UK wildlife, because the risk factors for zoonotic disease emergence require novelty, expansion of human populations and significant changes in landuse, which more usually occur in southern latitudes<sup>182, 188</sup>. The possibility of a UK epidemic resulting from emerging diseases originating in wildlife populations and passed into the UK through migrating wildlife, livestock movements or (in the case of a global pandemic) human to human transmission, aided by international air travel<sup>189</sup>, however, remains a worrying possibility. Indeed, a principal conclusion from a multidisciplinary meeting to discuss lessons learned about SARS was that 'humankind has had a lucky escape'. Only 1000 people died, in part due to the timing of infectiousness in humans (that coincides with the first symptoms, rather than people becoming infectious before exhibiting symptoms, making spread less likely) and because the virus flew from Hong Kong to Toronto, rather than to a city with a poorer health infrastructure or, for example, higher incidence of HIV in the population, which could have led to that country becoming endemic for SARS<sup>186</sup>.

## 8. Monitoring and regulation of disease in British mammals

UK policy concerning disease in populations of British mammals is targeted at preventing and controlling those diseases that have potentially severe implications for human health and livelihoods. Such implications comprise two potential impacts: direct impacts on human health through zoonotic infection, and indirect impacts through infection of livestock or work animals, which may have detrimental financial implications for anyone whose income relies on the production of animal products. Indeed in the latter case the costs of a given disease outbreak range from £2 million (minor) to over £3 billion (major outbreak)<sup>190</sup>.

Some specific infectious diseases in humans (such as cholera, measles and malaria; Table 1) when diagnosed must be reported by a doctor to the Local Authority or local Health Protection Unit<sup>191</sup>. Similarly, those diseases in animals that represent substantial risks to human health and livelihoods are also deemed to be 'notifiable'. Notifiable diseases are listed under the Animal Health Act, 1981, and a number of Orders made under the act, including the Infectious Diseases of Horses Order 1987, the Specified Diseases (Notification and Slaughter) Order 1992 (as amended) and the Specified Diseases (Notification) Order 1996 (as amended). Under the Animal Health Act 1981, "any person having in their possession or under their charge an animal affected or suspected of having one of these diseases must, with all practicable speed, notify that fact to a police constable"<sup>190</sup>. In actuality, the body responsible for investigating incidents of suspected notifiable diseases is the Animal Health and Veterinary Laboratories Agency (AHVLA), an executive agency working on behalf of Defra, and the Scottish and Welsh Governments<sup>190</sup>. If a notifiable disease is confirmed or suspected the law provides for animals to be culled, and gives an inspector powers to declare an infected place where disease is suspected; to carry out a veterinary inquiry, prohibit the movement of animals, carcasses and other potentially infected materials and equipment onto or off the premises and require the proper cleansing and disinfection of premises and equipment<sup>192</sup>.

### Of mice and nematodes: how does infection affect movement?

Infections may impact not only on individuals' survival and reproduction but also their behaviour. For example a study on infection with the intestinal nematode parasite *Heligmosomoides polygyrus* on the movements of wild male wood mice (*Apodemus sylvaticus*) during the breeding season<sup>193</sup> demonstrated that infected mice were found to move significantly further and faster than uninfected mice, covering larger territorial areas. Possible reasons include that infected mice require a larger food intake, necessitating greater movement to locate sufficient food within a patchy landscape, or that competition between males for females may force infected (subordinate) male mice to move further than uninfected (dominant) ones for mating opportunities. Both could result from *H. polygyrus* manipulating its host to range further to increase the distribution of its larvae (similar to toxoplasmosis in rats), or alternatively the behaviour of host mice may influence their susceptibility to infection (those with larger ranges are more likely to acquire infection). Regardless of cause, an association between behavioural differences and infection with a parasite is not confined to mice, but is repeated across numerous other host/parasite relationships. In this particular case, agricultural practices including harvest might reduce transmission rate (by destroying burrows in which contaminated faeces may accumulate)<sup>194</sup>.

Notifiable diseases can be either exotic (normally not present in Great Britain) or endemic (normally present), and either type may also be potentially zoonotic (chapter 7). While there have been over 14 exotic disease outbreaks in the last 10 years in UK animal populations, including foot and mouth disease, bird flu

#### Box 4: Disease screening

A common misapprehension is that wild animals can be effectively 'health screened' for every disease of interest. Specific diseases can be tested for, particularly post-mortem or in individuals that already exhibit symptoms of illness, but 'screening' animals in a bid to identify a large range of potential emerging infectious diseases is costly and time-consuming and raises a number of logistical issues. Given the vast range of parasites and pathogens where on earth would one begin? Should such screening be conducted by conservationists or vets, each of whom will have different priorities? What proportion of a population needs to be

sampled, for which pathogens and on what timescale? How often should repeat monitoring of these dynamic systems be conducted? Such are the considerations necessary for disease monitoring in wildlife, and the answers to these questions, and the many more similar questions, are neither easy, nor do they come cheap. Nevertheless, given the impact on wildlife and livestock that a spill-over epidemic could have, an effective system for identifying such diseases prior to an epidemic would certainly be beneficial.





## Rabbits and haemorrhagic disease virus

Thousands of wild and domestic European rabbits *Oryctolagus cuniculus* have died throughout Europe, Asia, Australia and New Zealand from rabbit haemorrhagic disease virus (RHDV)<sup>197</sup>. First recognised in China in 1984 after a major epidemic, RHDV was subsequently discovered on the British mainland in 1992<sup>198-199</sup> and has since been widespread. The disease is characterised by haemorrhagic lesions, particularly affecting the liver and lungs, with up to 90% mortality occurring within 48 hours of infection<sup>197</sup>. Research into the origins of RHDV in the UK suggests that an innocuous form of RDHV, the non-pathogenic rabbit calicivirus (RCV), was present in Britain at least 30 years before the initial outbreak of RHDV in China<sup>197</sup>. Transport of domestic rabbits between Europe and China is likely to have played a significant role in disease transmission. Despite both RHDV and the myxoma virus having significant impacts upon the wild rabbit population throughout Britain, the UK population of rabbits remains substantial and widespread.

and bluetongue, the majority of exotic diseases currently listed as notifiable have not, or have only very rarely, been recorded in the UK<sup>195</sup>. The presence of these diseases on the list of notifiable diseases, therefore, reflects a concern that exotic diseases present a significant threat to British livestock, and should accordingly be limited from entering and becoming established in the UK. As part of its remit Defra monitors for new disease incidents in EU Member States, countries on the borders of the EU and the UK's third country trading partners<sup>196</sup>, and in doing so works with veterinary organisations in these countries as well as with the World Organisation for Animal Health (Formerly Office International des Epizooties)<sup>196</sup>.

The obvious intention of the above approach is to limit the transport of infected livestock and animal produce and in so doing limit the ingress of exotic notifiable diseases. However, a separate route for ingress of exotic notifiable disease is through wildlife vectors (chapter 4). By definition wildlife movements do not follow any easily regulated pathways, and any wild animal that regularly moves between countries represents a potential vector for an exotic notifiable disease to enter the UK. The GB Wildlife Disease Surveillance Partnership was created to address this issue. The partnership comprises a number of institutions - including the AHVLA, Scottish Agricultural College (SAC), Institute of Zoology (IoZ), the Food and Environment Research Agency (FERA), the Centre for Environment, Fisheries and Aquaculture (CEFAS), the Wildfowl and Wetlands Trust (WWT), Natural England (NE) and the Forestry Commission England (FCE) - and its remit is to produce a quarterly report on instances of notable, and notifiable, disease events in the UK. For example the January-June

2012 report included (amongst many other reports) a pathology analysis of a stranding of a female white-beaked dolphin (*Lagenorhynchus albirostris*) which revealed the individual had a *Streptococcus spp.* infection, examination of a mass mortality of mute swans (*Cygnus olor*) which excluded avian influenza virus (a notifiable disease which is potentially zoonotic) as the cause, and noted a 7% annual decline in the national greenfinch population since a trichomonosis epidemic started in 2005<sup>200</sup>. The cumulative effect of the activities of the participating institutions is to examine the causes of mortality and sickness in a broad range of wildlife species to ensure that none of the cases represent the emergence of a notifiable disease which could cause health problems either for the human or for the domestic livestock populations.

## 9. The Future

Despite the geographical quirk which makes Britain an island, in terms of our collective susceptibility to disease we are very much part of the global community. The continuous movement - both legal and illegal - of people, livestock, pets, wildlife, meat and animal products between countries means that almost nowhere on the planet is exempt from global disease trends. In the same way that movements of British and American military personnel were largely responsible for spreading the lethal 1918-20 influenza epidemic, every person and animal product travelling internationally is a potential carrier of infectious disease.

Similarly, whereas other conservation concerns - such as the effects of habitat loss, climate change, establishment of invasive species and loss of biodiversity - are often (generally wrongly) perceived only to have indirect relevance to human wellbeing, the emergence of a new and virulent disease of wildlife is of direct relevance to human health and livelihoods, because any such disease can be transmitted to livestock and/or to us. In short, when it comes to preventing the emergence and spread of new diseases, human interests align with those of our livestock and sympatric wildlife, and UK interests align firmly with those of the rest of the world.

A number of current global trends have the potential to affect disease in UK mammals: urbanisation, climate change, environmental contaminants and emerging infectious diseases. We discuss each of these briefly in the context of global disease risk.

### Urbanisation

Urbanisation is increasing globally and its ecological impacts extend beyond urban areas<sup>201</sup>. The term 'urban' as applied to wildlife incorporates small towns, neighbourhoods and back yards, cities and/or urban centres<sup>202</sup>. The numbers and diversity of wildlife, particularly of mammals, generally increase with distance from urban centres, through suburbs and into the countryside<sup>203</sup>. The exceptions to this rule are species that are adapted to urban living which can actually occur at much higher densities in these places<sup>204</sup>. The low diversity but high densities of species in urban areas has several disease implications. Firstly high densities increase contact rates between individuals - especially around food sources eg domestic rubbish or feeders for garden birds and mammals - and so favour the transmission of diseases spread by direct contact or oral-faecal routes<sup>201</sup>. Second, wildlife beyond urban areas can be affected by diseases that are maintained in urban-adapted hosts. For example, rates of toxoplasmosis in southern sea otters (*Enhydra lutris nereis*) off the coast of California are three times higher near urban areas, probably due to run-off water that is contaminated with cat faeces<sup>205</sup>. Third, transport and trade routes meet in cities, and so cities are first points of entry for many inbound novel diseases and hubs for potential cross-species transmission<sup>201</sup>. West Nile Virus (a strain thought to originate in Israel) was

initially introduced into New York City in 1999 - probably through human activities<sup>206</sup> - and spread rapidly across North America causing over 2800 human cases and tens of thousands of wild bird deaths<sup>207-208</sup>. Lastly, urban centres are sources of environmental contamination which may adversely affect immunocompetence in wildlife species (chapter 3; see below).

### Environmental contamination

Pollutants, pathogens and environment interact, and in complex ways<sup>209</sup>. These interactions, and their relevance to conserving British mammals, are poorly understood, but more and more studies are linking anthropogenic contaminants to wildlife disease. Industrial, agricultural and urban centres all create high concentrations of pollutants such as heavy metals and pesticides<sup>210-212</sup>, and these can negatively affect immune response in wildlife species. The rate of infection with avian malaria (*Plasmodium relictum*) of house sparrows (*Passer domesticus*) is higher where the environment is more contaminated with lead, typically in heavily urbanised habitats due to its previous use as a petrol additive<sup>209</sup>. A five-year field study of two amphibian species, the marine toad (*Bufo marinus*) and whistling frog (*Eleutherodactylus johnstonei*), found high levels of metals and pesticides in tissues samples to be associated with a weakening of the immune response and an increase in helminth (a type of macroparasite) infections<sup>213</sup>. Similarly, evidence is accumulating that man-made environmental pollutants are associated with increased cancer rates - in particular through lowered resistance to viral oncogenesis (cancer caused by a viral agent) - in a range of wildlife, including turtles, beluga whales and benthic fish<sup>214</sup>. One speculation is that components of plastics such as bisphenol A, a compound known to cause or contribute to cancers in humans and rodents, especially in marine ecosystems, may be responsible<sup>215</sup>. Increasingly, it looks as if environmental contamination with pesticides, herbicides, trace metals and plastics is negatively impacting the ability of various wildlife (and plausibly humans) to resist infectious disease. It seems likely that these threats will worsen as the human population increases with concomitant industrialisation and urbanisation.

### Climate change

Climate change, particularly unprecedented extremes and extents of weather variability, add substantially to the threats facing UK mammals<sup>216</sup>. Even where climate change is perhaps not the primary threat to, or stressor of, a species' population dynamics, climatic effects can nevertheless interact with other factors, such as habitat loss, disease, or competition with invasives, to exacerbate the pressures on wildlife<sup>216</sup>. Climate change will affect the risks posed by infectious diseases doubtless in complicated ways that are difficult to predict<sup>217</sup>. For British terrestrial mammals climate change is most likely to affect any diseases that have a free-living stage (eg eggs laid outside of the host) or which are transferred by vectors (ie ticks, mosquitoes and midges). Many such



### Foxes and sarcoptic mange

Sarcoptic mange, caused by the burrowing mite *Sarcoptes scabiei*, is a disease of widespread importance<sup>42</sup>, having the potential to 'spill-over' between wild and domestic mammals<sup>10, 76</sup>. Sarcoptic mange is responsible for epizootic disease in wild canids in North America, Europe and Australia, wild cats in Europe and Africa, wild ungulates and wild boars in Europe, wombats and koalas in Australia, and great apes and various wild Bovids in Africa<sup>42</sup>. The disease is now widespread in Britain, particularly amongst populations of red foxes (*Vulpes vulpes*)<sup>43</sup>. Transmission of mites between hosts is believed to be through both direct contact, through allogrooming, sucking and aggressive interactions, and indirect contact through fomites. Mites consume tissue fluid and living cells<sup>220</sup>. Once in the skin, mites release a secretion that causes hypersensitivity and itching in the host<sup>42</sup>. In foxes, hyperkeratosis (the crusty skin characteristic of mange) is noticeable one to two months after initial infection, with the average time from diagnosis to death being 3.7 months<sup>221</sup>. Although mange itself is not always fatal, death is frequently caused by secondary symptoms, including starvation, hypothermia and bacterial infections<sup>222</sup>. Frequency-transmitted pathogens can pose a significant risk to compromised populations - mange has caused significant declines in isolated hairy-nosed wombat *Lasiorchinus latifrons* populations<sup>223</sup>, and is a major cause of mortality for cheetah populations in the Masai Mara<sup>224</sup>. Understanding its population dynamics and epidemiology is therefore essential for successful wildlife disease management<sup>225-226</sup>.

diseases are currently limited to certain climates because outside these climates the ticks, mosquitoes and midges die before the disease can develop fully<sup>77</sup>. Human diseases such as malaria, *African trypanosomiasis*, Lyme disease, tick-borne encephalitis, yellow fever, plague, and dengue have all increased in incidence or geographic range in recent decades<sup>218-219</sup>. Whether these spreads resulted directly from climate change remains unclear, but several lines of evidence link warmer and wetter conditions with increasing viability, population sizes and biting rates of the disease vectors, and with increased rates of development and lengths of time over which the disease parasites are infective<sup>77</sup>. In short the mechanism by which warmer climates might lead to diseases expanding their range is proven, even if evidence for a direct relationship is lacking, due to the complexities involved in obtaining appropriate data.

In general climate change has three important implications for diseases of terrestrial mammals: (i) increasing the spread of diseases into the temperate zone; (ii) increasing the elevation to which diseases can spread in mountainous regions; and (iii) increasing the length of the season over which a given pathogen is infective<sup>77</sup>. In marine ecosystems, although there is evidence for temperature and climate-related links in some marine diseases, separating the effects of climate change from other anthropogenic disturbances is complicated by a lack of reliable data<sup>77</sup>.

---

Bluetongue - a viral disease of sheep, cattle and deer - has recently expanded into Northern Europe due to increased survival of the midges that carry it<sup>227</sup>. Similarly the mites that cause sarcoptic mange, which in the UK infects foxes (chapter 3) persist for longer in the environment in warmer and wetter conditions - which increases the likelihood of the mites being transmitted without the need for direct contact between hosts<sup>220</sup>. A warming climate may increase the prevalence and intensity of sarcoptic mange in higher latitudes, including in the UK. The chytrid disease of amphibians requires cool, moist, high-altitude conditions: while this may mean that global warming could limit the spread of the disease in some areas<sup>77</sup>, higher elevations may become suitable and so mountain populations of amphibians could be threatened<sup>228</sup>.

The potential for climate change to permit diseases to spread to UK habitats, and therefore impact on wildlife and livestock communities is clear. Although direct evidence of any such effects in the UK is lacking at present, the potential effects of climate change on the incidence of liver fluke, West Nile Virus and bluetongue in the UK are of particular concern<sup>74</sup>.

### Emerging infectious diseases, human population density, land use change and movements of animals

The effort and effectiveness of reporting emergent infectious diseases may be improving, but that cannot alone explain their recorded increase since 1940; the result is escalating risk to both livestock and human health<sup>182</sup>. Disease emergence can be characterised as a three step process<sup>188</sup>:

Stage 1 (pre-emergence) occurs when the disease is still in its natural wildlife reservoir and ecological, social, or socioeconomic changes (eg change in land use) allow it to expand within its host population, spread to a new region, or be transmitted to another non-human population or species. Typically this occurs due to large-scale

environmental, agricultural, or demographic shifts such as the movement of livestock to a region for the first time, or transportation of wildlife from a region for food<sup>188</sup>.

Stage 2 (localised emergence) is the initial spill-over of a wildlife or livestock disease to people. Causes range from handling of butchered wildlife to exposure to any infective material in wildlife markets or livestock farms, or in the wild (eg SARS). Outcomes vary widely, from small clusters of human cases to large outbreaks, some with limited person-to-person transmission.

Stage 3 (full pandemic emergence) is sustained person-to-person transmission and large-scale spread, often aided by global air travel (eg HIV/AIDS, SARS) or the international movement of reservoir hosts or vectors through trade (eg West Nile virus)<sup>188</sup>.

An array of factors contribute to conditions suitable for the emergence of a zoonotic pandemic. These factors include increasing human densities<sup>182</sup>, land use changes<sup>182, 188</sup>, the prevalence of bushmeat markets<sup>229-231</sup> - in which urbanised humans come into contact with a large variety of wild animal pathogens (to which they are unlikely to have resistance) through consuming meat provided by bushmeat hunters (who may have some immunity to these diseases)<sup>232</sup> - the large-scale global transport of animals<sup>123</sup> - both wildlife (eg for the pet-trade<sup>233</sup>) and livestock<sup>129, 234</sup> - and the huge number (over a billion) of international human travellers every year<sup>11</sup>. Arrayed against this threat are increasing abilities to predict emergence 'hotspots'<sup>182, 188</sup>, and positive political will for countries to act together to strengthen global networks against pandemic emergence<sup>188</sup>. Zoonotic diseases, by definition, are a key concern of human-health agencies, agricultural authorities and natural resource managers, all of whom should work cooperatively to address the challenge of how researchers can intervene before a pathogen reaches the human population, and to develop appropriate responses if an outbreak is suspected/possible<sup>188</sup>.

# References

- Lyles, A.M. and A.P. Dobson (1993) Infectious disease and intensive management: population dynamics, threatened hosts, and their parasites. *Journal of Zoo and Wildlife Medicine*, 24, 315-326.
- Collinge, S.K. and C. Ray (2006) Community epidemiology. *Disease Ecology* (eds S.K. Collinge and C. Ray), pp. 1-5. Oxford University Press, Oxford
- Scott, M.E. (1988) The impact of infection and disease on animal populations; implications for conservation biology. *Conservation Biology*, 2, 40-56.
- Anderson, R.M. (1978) The regulation of host populations growth by parasite species. *Parasitology*, 76.
- Munson, L., K.A. Terio, M. Ryser, E. Lane, and F. Courchamp (2010) Wild Felid Diseases: Conservation Implications and Management Strategies. *Biology and Conservation of Wild Felids* (eds D.W. Macdonald and A. Loveridge), pp. 237-262. Oxford University Press, Oxford
- Anderson, R.M., R.M. May, K. Joysey, D. Mollison, G.R. Conway, R. Cartwell, H.V. Thompson, and B. Dixon (1986) The Invasion, Persistence and Spread of Infectious Diseases within Animal and Plant Communities [and Discussion]. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences*, 314, 533-570.
- Taubenberger, J.K. and D.M. Morens (2006) 1918 Influenza: the Mother of All Pandemics. *Emerging Infectious Diseases*, 12, 15-22.
- Potter, C.W. (2001) A history of influenza. *Journal of Applied Microbiology*, 91, 572-579.
- May, R.M. (1988) *Conservation and Disease*. *Conservation Biology*, 2, 28-30.
- Daszak, P., A.A. Cunningham, and A.D. Hyatt (2000) Emerging Infectious Diseases of Wildlife- Threats to Biodiversity and Human Health. *Science*, 287, 443-449.
- Karesh, W.B., A. Dobson, J.O. Lloyd-Smith, J. Lubroth, M.A. Dixon, M. Bennett, S. Aldrich, T. Harrington, P. Formenti, E.H. Loh, C.C. Machalaba, M.J. Thomas, and D.L. Heymann (2012) Ecology of zoonoses: natural and unnatural histories. *The Lancet*, 380, 1936-1945.
- Taylor, L.H., S.M. Latham, and M.E.J. Woolhouse (2001) Risk factors for human disease emergence. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 356, 983-989.
- Cunningham, A.A., A.P. Dobson, and P.J. Hudson (2012) Disease invasion: impacts on biodiversity and human health. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 367, 2804.
- Cleaveland, S., M.K. Laurenson, and L.H. Taylor (2001) Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 356, 991-999.
- Pimentel, D., L. Lach, R. Zuniga, and D. Morrison (2000) Environmental and economic costs of nonindigenous species in the United States. *Bioscience*, 50, 53-65.
- Pimentel, D., R. Zuniga, and D. Morrison (2005) Update on the environmental and economic costs associated with alien-invasive species in the United States. *Ecological Economics*, 52, 273-288.
- Anderson, R. (1995) Evolutionary pressures in the spread and persistence of infectious agents in vertebrate populations. *Parasitology*, 111, S15-S31.
- Riordan, P., P. Hudson, and S. Albon (2006) Do parasites matter? Infectious diseases and the conservation of host populations. *Key Topics in Conservation Biology*, 156-172.
- Harrington, L.A., M. Gelling, V. Simpson, A. Harrington, and D.W. Macdonald (2012) Notes on the health status of free-living, non-native American mink, *Neovison vison*, in southern England. *European Journal of Wildlife Research*, 58, 875-880.
- Strachan, R., T.P. Moorhouse, and M.G. Gelling (2011) *Water vole conservation handbook*, third edition. *Wildlife Conservation Research Unit*.
- Gelling, M., D. Macdonald, S. Telfer, T. Jones, K. Boum, R. Birtles, and F. Mathews (2012) Parasites and pathogens in wild populations of water voles (*Arvicola amphibius*) in the UK. *European Journal of Wildlife Research*, 58, 615-619.
- Craft, M.E. (2010) Ecology of infectious diseases in Serengeti lions. *Biology and Conservation of Wild Felids*. (eds D.W. Macdonald and A. Loveridge), pp. 263-281. Oxford University Press, Oxford
- Craft, M.E. (2010) Ecology of infectious diseases in Serengeti lions. *Biology and Conservation of Wild Felids* (eds D.W. Macdonald and A. Loveridge), pp. 263-282. Oxford University Press, Oxford
- Macdonald, D.W. and M.K. Laurenson (2006) Infectious disease: Inextricable linkages between human and ecosystem health. *Biological Conservation*, 131, 143-150.
- Macdonald, D.W. and F. Tattersall (2003) *State of Britain's Mammals 2003*. PTES.
- Härkönen, T., R. Dietz, P. Reijnders, J. Teilmann, K. Harding, A. Hall, S. Brasseur, U. Siebert, S.J. Goodman, and P.D. Jepson (2006) The 1988 and 2002 phocine distemper virus epidemics in European harbour seals. *Diseases of aquatic organisms*, 68, 115-130.
- McCallum, H. and A. Dobson (1995) Detecting disease and parasite threats to endangered species and ecosystems. *Trends in Ecology & Evolution*, 10, 190-194.
- Gascoyne, S., M. Laurenson, S. Lelo, and M. Borner (1993) Rabies in African wild dogs (*Lycaon pictus*) in the Serengeti region, Tanzania. *Journal of Wildlife Diseases*, 29, 396-402.
- Sillero-Zubiri, C., A. King, and D. Macdonald (1996) Rabies and mortality in Ethiopian wolves (*Canis simensis*). *Journal of Wildlife Diseases*, 32, 80-86.
- Faine, S. (1998) *Leptospirosis Bacterial infections of people: Epidemiology and control* (eds A.S. Evans and P.S. Brachman), pp. 395-420. Springer.
- Waitkins, S.A. (1991) Rats as a source of leptospirosis - Weil's disease. *Sorex Technical Publication No. 4*. Cheshire: Sorex Ltd.
- Webster, J.P., W.A. Ellis, and D.W. Macdonald (1995) Prevalence of *Leptospira* and other zoonoses in wild brown rats on UK farms. *Mammalia*, 59, 615-622.
- Webster, J.P., W.A. Ellis, and D.W. Macdonald (1995) Prevalence of *Leptospira* spp. in wild brown rats (*Rattus norvegicus*) on UK farms. *Epidemiology and Infection*, 114, 195-201.
- Gelling, M., W. Zochowski, D.W. Macdonald, A. Johnson, M. Palmer, and F. Mathews (In review) *Leptospira* acquisition following the reintroduction of wildlife. *European Journal of Wildlife Research*.
- Gelling, M., P.J. Johnson, T.P. Moorhouse, and D.W. Macdonald (2012) Measuring Animal Welfare within a Reintroduction: An Assessment of Different Indices of Stress in Water Voles *Arvicola amphibius*. *Plos One*, 7.
- Deem, S.L., W.B. Karesh, and W. Weisman (2001) Putting Theory into Practice: Wildlife Health in Conservation. *Conservation Biology*, 15, 1224-1233.
- Smith, K.F., K. Acevedo-Whitehouse, and A.B. Pedersen (2009) The role of infectious diseases in biological conservation. *Animal Conservation*, 12, 1-12.
- Macdonald, D.W. and K. Laurenson (2006) *Infectious Disease and Mammalian Conservation*. *Biological Conservation*, 143-347.
- Hudson, J.R., H.V. Thompson, and W. Mansi (1955) Myxoma virus in Britain. *Nature*, 176, 783-783.
- Macdonald, D.W. and F. Tattersall (2001) *Britain's mammals: the challenge for conservation*. *Mammals Trust*, UK, London.
- Forrester, N.L., R.C. Trout, S.L. Turner, D. Kelly, B. Boag, S. Moss, and E.A. Gould (2006) Unravelling the paradox of rabbit haemorrhagic disease virus emergence, using phylogenetic analysis; possible implications for rabbit conservation strategies. *Biological Conservation*, 131, 296-306.
- Pence, D.B. and E. Ueckermann (2002) Sarcocystis mange in wildlife. *Revue scientifique et technique (International Office of Epizootics)*, 21, 385-398.
- Soulsbury, C.D., G. Iossa, P.J. Baker, N.C. Cole, S.M. Funk, and S. Harris (2007) The impact of sarcoptic mange *Sarcoptes scabiei* on the British fox *Vulpes vulpes* population. *Mammal Review*, 37, 278-296.
- Woodroffe, R., J.R. Ginsberg, and D.W. Macdonald (1997) *The African wild dog: status survey and conservation action plan*. IUCN.
- Creel, S. and N.M. Dreef (2002) *The African wild dog: behavior, ecology, and conservation*. Princeton University Press.
- Alexander, K. and M. Appel (1994) African wild dogs (*Lycaon pictus*) endangered by a canine distemper epizootic among domestic dogs near the Masai Mara National Reserve, Kenya. *Journal of Wildlife Diseases*, 30, 481-485.
- Macdonald, D.W. (1992) Cause of Wild Dog deaths. *Nature*, 360, 633.
- Holdich, D.M. and W.D. Rogers (1997) The white-clawed crayfish, *Austropotamobius pallipes*, in Great Britain and Ireland with particular reference to its conservation in Great Britain. *Bulletin Francais de la Peche et de la Pisciculture*, 347, 597-616.
- Lozan, J.L. (2000) On the threat to the European crayfish: A contribution with the study of the activity behaviour of four crayfish species (Decapoda: Astacidae)\*. *Limnologia*, 30, 156-161.
- Bubb, D.H., T.J. Thom, and M.C. Lucas (2005) The within catchment invasion of the non-indigenous signal crayfish (*Dana*) in upland rivers. *Bulletin Francais de Peche et de Pisciculture*, 376-377, 665-673.
- Bubb, D.H., T.J. Thom, and M.J. Lucas (2006) Movement, dispersal and refuge use of co-occurring, introduced and native crayfish. *Freshwater Biology* 51, 1359-1368.
- The UK Biodiversity Action Plan (UK BAP) ([www.jncc.defra.gov.uk](http://www.jncc.defra.gov.uk))
- Tompkins, D., A. White, and M. Boots (2003) Ecological replacement of native red squirrels by invasive greys driven by disease. *Ecology Letters*, 6, 189-196.
- Macdonald, D.W. and D. Burnham (2007) State of Britain's Mammals 2007. PTES.
- Brummer, C.M. and e. al (2010) Epidemiology of squirrelpox virus in grey squirrels in the UK. *Epidemiology and Infection*, 138, 941.
- Rushton, S.P., P.W.W. Lurz, J. Gurnell, and R. Fuller (2000) Modelling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the UK? *Journal of Applied Ecology*, 37, 997-1012.
- Rushton, S.P., P.W.W. Lurz, J. Gurnell, P. Nettleton, C. Brummer, M.D.F. Shirley, and A.W. Sainsbury (2006) Disease threats posed by alien species: the role of a poxvirus in the decline of the native red squirrel in Britain. *Epidemiology and Infection*, 134, 521-533.
- Sainsbury, A., R. Deaville, B. Lawson, W. Cooley, S.J. Farelly, M. Stack, P. Duff, C. McInnes, J. Gurnell, P. Russell, S. Rushton, D. Pfeiffer, P. Nettleton, and P.W. Lurz (2008) Poxviral Disease in Red Squirrels *Sciurus vulgaris* in the UK: Spatial and Temporal Trends of an Emerging Threat. *EcoHealth*, 5, 305-316.
- Sainsbury, A. and J. Gurnell (1995) An investigation into the health and welfare of red squirrels, *Sciurus vulgaris*, involved in reintroduction studies. *Veterinary Record*, 137, 367-370.
- Sainsbury, A.W., P. Nettleton, J. Gilray, and J. Gurnell (2000) Grey squirrels have high seroprevalence to a parapoxvirus associated with deaths in red squirrels. *Animal Conservation*, 3, 229-233.
- Tompkins, D.M., A.W. Sainsbury, P. Nettleton, D. Buxton, and J. Gurnell (2002) Parapoxvirus causes a deleterious disease in red squirrels associated with UK population declines. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 269, 529-533.
- Thomas, K., D.M. Tompkins, A.W. Sainsbury, A.R. Wood, R. Dalziel, P.F. Nettleton, and C.J. McInnes (2003) A novel poxvirus lethal to red squirrels (*Sciurus vulgaris*). *Journal of General Virology*, 84, 3337-3341.
- McInnes, C., L. Coulter, M. Dagleish, C. Fiegna, J. Gilray, K. Willoughby, M. Cole, E. Milne, A. Meredith, and D. Everest (2009) First cases of squirrelpox in red squirrels (*Sciurus vulgaris*) in Scotland. *Veterinary Record*, 164, 528-531.
- McInnes, C.J., A.W. Wood, K. Thomas, A.W. Sainsbury, J. Gurnell, F.J. Dein, and P.F. Nettleton (2006) Genomic characterization of a novel poxvirus contributing to the decline of the red squirrel (*Sciurus vulgaris*) in the UK. *Journal of General Virology*, 87, 2115-2125.
- Harrington, L.A. and D.W. Macdonald (2002) A review of the effects of pesticides on wild terrestrial mammals in Britain. *Wildlife Conservation Research Unit*, Oxford.
- Selgrade, M.K. (2007) Immunotoxicity—The Risk is Real. *Toxicological Sciences*, 100, 328-332.
- Ansar Ahmed, S. (2000) The immune system as a potential target for environmental estrogens (endocrine disruptors): a new emerging field. *Toxicology*, 150, 191-206.
- Hall, A.J., R.J. Law, D.E. Wells, J. Harwood, H.M. Ross, S. Kennedy, C.R. Allchin, L.A. Campbell, and P.P. Pomeroy (1992) Organochlorine levels in common seals (*Phoca vitulina*) which were victims and survivors of the 1988 phocine distemper epizootic. *Science of The Total Environment*, 115, 145-162.
69. Kilpatrick, A.M., C.J. Briggs, and P. Daszak (2010) The ecology and impact of chytridiomycosis: an emerging disease of amphibians. *Trends in Ecology & Evolution*, 25, 109-118.
- Schloegel, L., J.-M. Hero, L. Berger, R. Speare, K. McDonald, and P. Daszak (2006) The Decline of the Sharp-Shinned Day Frog (*Taudactylus acutirostris*): The First Documented Case of Extinction by Infection in a Free-Ranging Wildlife Species? *EcoHealth*, 3, 35-40.
- Foresight (2006) *Infectious Diseases: Preparing for the Future*. Executive Summary. Office of Science and Innovation, London.
- Anderson, R.M. and R.M. May (1978) Regulation and stability of host-parasite population interactions: I. Regulatory processes. *The Journal of Animal Ecology*, 219-247.
- Cleaveland, S., G. Hess, A. Dobson, M. Laurenson, H. McCallum, M. Roberts, and R. Woodroffe (2002) The role of pathogens in biological conservation. *The Ecology of Wildlife Diseases* (eds P.J. Hudson, A. Rizzoli, B.T. Grenfell, H. Heesterbeek, and A.P. Dobson). Oxford University Press.
- Bohm, M., P.C. White, J. Chambers, L. Smith, and M. Hutchings (2007) Wild deer as a source of infection for livestock and humans in the UK. *The Veterinary Journal*, 174, 260-276.
- Simpson, V. (2002) Wild animals as reservoirs of infectious diseases in the UK. *The Veterinary Journal*, 163, 128-146.
- Gortázar, C., E. Ferroglio, U. Höfle, K. Frölich, and J. Vicente (2007) Diseases shared between wildlife and livestock: a European perspective. *European Journal of Wildlife Research*, 53, 241-256.
- Harvell, C.D., C.E. Mitchell, J.R. Ward, S. Altizer, A.P. Dobson, R.S. Ostfeld, and M.D. Samuel (2002) Climate warming and disease risks for terrestrial and marine biota. *Science*, 296, 2158-2162.
- <http://www.britishtwildboar.org.uk/index.htm?map2.html>.
- Ward, A.I., T.R. Etherington, and G.C. Smith (2008) Exposure of Cattle to *Mycobacterium bovis* Excreted by Deer in Southwest England: a Quantitative Risk Assessment. Consultancy report to TB Programme, Food and Farming Group, Department of Environment, Food and Rural Affairs, London, UK.
- Ward, A.I. and G.C. Smith (2012) Predicting the status of wild deer as hosts of

- Mycobacterium bovis infection in Britain. *European Journal of Wildlife Research*, 58, 127-135.
81. Eubank, R., F. Kim-Madslie, and C.B. Hart (1999) Management and Welfare of Farm Animals. Universities Federation for Animal Welfare (UFAW). Fourth edition.
  82. Hartley, M., F. Voller, T. Murray, and H. Roberts (2013) Qualitative veterinary risk assessment of the role of wild deer in the likelihood of incursion and the impact on effective disease control of selected exotic notifiable diseases in England. *European Journal of Wildlife Research*, 59, 257-270.
  83. Mathews, F., L. Lovett, S. Rushton, and D.W. Macdonald (2006) Bovine tuberculosis in cattle: reduced risk on wildlife-friendly farms. *Biology Letters*, 2, 271-274.
  84. Blewett, D. and W. Watson (1984) The epidemiology of ovine toxoplasmosis. III. Observations on outbreaks of clinical toxoplasmosis in relation to possible mechanisms of transmission. *British Veterinary Journal*, 140, 54-63.
  85. Alonso-Andicoberry, C., F.J. Garcia-Pena, J. Pereira-Bueno, E. Costas, and L.M. Ortega-Mora (2001) Herd-level risk factors associated with *Leptospira* spp. seroprevalence in dairy and beef cattle in Spain. *Preventive Veterinary Medicine*, 52, 109-117.
  86. Milkovic, V., Stankov, A. Loncarevic, G. Jermolenko, R. Radovanovic, D. Nestic, and S. Veselinovic (1994) Reproduction disorders in sows caused by viral infections (Parvo, Smeđi, Aujeszky, SIRS). *Veterinarski Glasnik*, 48, 399-405.
  87. Scaife, H.R. (2006) *Veterinary Record*, 159, 175-178.
  88. Judge, J., R.S. Davidson, G. Marion, P.C.L. White, and M.R. Hutchings (2007) Persistence of *Mycobacterium avium* subspecies *paratuberculosis* in rabbits: the interplay between horizontal and vertical transmission. *Journal of Applied Ecology*, 44, 302-311.
  89. Bates, P. (1999) Inter- and intra-specific variation within the genus *Psoroptes* (Acari: Psoroptidae). *Veterinary Parasitology*, 83, 201-217.
  90. Atkinson, R.P.D., D.W. Macdonald, and P.J. Johnson (1994) The Status of the European Mole (*Talpa europaea* L.) as an Agricultural Pest and Its Management. *Mammal Review*, 24, 73-90.
  91. Barlow, R. and B. McGorum (1985) Ovine listerial encephalitis: analysis, hypothesis and synthesis. *Veterinary Record*, 116, 233-236.
  92. Blehert, D.S., A.C. Hicks, M. Behr, C.U. Meteyer, B.M. Berlouski-Zier, E.L. Buckles, J.T. Coleman, S.R. Darling, A. Gargas, and R. Niver (2009) Bat white-nose syndrome: an emerging fungal pathogen? *Science*, 323, 227-227.
  93. Warnecke, L., J.M. Turner, T.K. Bollinger, J.M. Lorch, V. Misra, P.M. Cryan, G. Wibbelt, D.S. Blehert, and C.K. Willis (2012) Inoculation of bats with European *Geomyces destructans* supports the novel pathogen hypothesis for the origin of white-nose syndrome. *Proceedings of the National Academy of Sciences*, 109, 6999-7003.
  94. Puechmaille, S.J., P. Verdeyroux, H. Fuller, M.A. Gouilh, M. Bekaert, and E.C. Teeling (2010) White-nose syndrome fungus (*Geomyces destructans*) in bat, France. *Emerging Infectious Diseases*, 16, 290.
  95. Puechmaille, S.J., G. Wibbelt, V. Korn, H. Fuller, F. Forget, K. Mühldorfer, A. Kurth, W. Bogdanowicz, C. Borel, and T. Bosch (2011) Pan-European distribution of white-nose syndrome fungus (*Geomyces destructans*) not associated with mass mortality. *Plos One*, 6, e19167.
  96. Wibbelt, G., A. Kurth, D. Hellmann, M. Weishaar, A. Barlow, M. Veith, J. Prüger, T. Gänfö, L. Grosche, and F. Bontadina (2010) White-nose syndrome fungus (*Geomyces destructans*) in bats, Europe. *Emerging Infectious Diseases*, 16, 1237.
  97. Bat Conservation Trust (2013) *Pseudogymnoascus destructans* discovered in the UK.
  98. Webster, J.P., C.F.A. Brunton, and D.W. Macdonald (1994) Effect of *Toxoplasma gondii* upon neophobic behaviour in wild brown rats, *Rattus norvegicus*. *Parasitology*, 109, 37-43.
  99. Webster, J.P. and D.W. Macdonald (1995) Cryptosporidiosis reservoir in wild brown rats (*Rattus norvegicus*): first report in the U.K. *Epidemiology and Infection*, 115, 207-209.
  100. Webster, J.P. and D.W. Macdonald (1995) Parasites of wild brown rats, *Rattus norvegicus*, on UK farms. *Parasitology*, 111, 247-255.
  101. Perryman, L. (1990) Cryptosporidiosis in rodents. *Cryptosporidiosis of man and animals*, 125-131.
  102. Schmidt, G.D. and L.S. Roberts (1989) *Foundations of Parasitology*, 4th edn. Times Mirror/Mosby, St Louis.
  103. Hutchinson, J., A. Wear, S. Lambton, R. Smith, and G. Pritchard (2011) Survey to determine the seroprevalence of *Toxoplasma gondii* infection in British sheep flocks. *Veterinary Record*, 169, 582-582.
  104. Ward, A.I. (2005) Expanding ranges of wild and feral deer in Great Britain. *Mammal Review*, 35, 165-173.
  105. Côté, S.D., T.P. Rooney, J.-P. Tremblay, C. Dussault, and D.M. Waller (2004) Ecological impacts of deer overabundance. *Annual Review of Ecology, Evolution, and Systematics*, 113-147.
  106. Krebs, J. (1997) Bovine tuberculosis in cattle and badgers. MAFF publications, London.
  107. Delahay, R., A. De Leeuw, A. Barlow, R. Clifton-Hadley, and C. Cheeseman (2002) The Status of *Mycobacterium bovis* Infection in UK Wild Mammals: A Review. *The Veterinary Journal*, 164, 90-105.
  108. Brown, R.N. and E.C. Burgess (2001) Lyme borreliosis. *Infectious Diseases of Wild Mammals* (eds E.S. Williams and I.K. Barker), pp. 435-454. Manson Publishing, London.
  109. Webster, J.P. (2001) Rats, cats, people and parasites: the impact of latent toxoplasmosis on behaviour. *Microbes and Infection*, 3, 1037-1045.
  110. Berdoy, M., J. Webster, and D. Macdonald (1995) Parasite-altered behaviour: is the effect of *Toxoplasma gondii* on *Rattus norvegicus* specific? *Parasitology*, 111, 403-409.
  111. Webster, J.P. (1994) The effect of *Toxoplasma gondii* and other parasites on activity levels in wild and hybrid *Rattus norvegicus*. *Parasitology-Cambridge*, 109, 583-583.
  112. Berdoy, M.L., J.P. Webster, and D.W. Macdonald (2000) Fatal attraction in rats infected with *Toxoplasma gondii*. *Proceedings of the Royal Society of London Series B-Biological Sciences*, 267, 1591-1594.
  113. Flegel, J. (2007) Effects of *Toxoplasma* on human behavior. *Schizophrenia bulletin*, 33, 757-760.
  114. Flegel, J., J. Klose, M. Novotná, M. Berenreiterová, and J. Havlíček (2009) Increased incidence of traffic accidents in *Toxoplasma*-infected military drivers and protective effect RhD molecule revealed by a large-scale prospective cohort study. *BMC infectious diseases*, 9, 72.
  115. Torrey, E.F., J.J. Bartko, and R.H. Yolken (2012) *Toxoplasma gondii* and other risk factors for schizophrenia: an update. *Schizophrenia bulletin*, 38, 642-647.
  116. Ling, V.J., D. Lester, P.B. Mortensen, P.W. Langenberg, and T.T. Postolache (2011) *Toxoplasma gondii* seropositivity and suicide rates in women. *The Journal of nervous and mental disease*, 199, 440.
  117. Godfray, H.C.J., C.A. Donnelly, R.R. Kao, D.W. Macdonald, J.A. McDonald, G. Petrokofsky, J.L.N. Wood, R. Woodroffe, D.B. Young, and A.R. McLean (2013) A restatement of the natural science evidence base relevant to the control of bovine tuberculosis in Great Britain†. *Proceedings of the Royal Society B: Biological Sciences*, 280.
  118. Macdonald, D.W., P. Riordan, and F. Mathews (2006) Biological hurdles to the control of TB in cattle: A test of two hypotheses concerning wildlife to explain the failure of control. *Biological Conservation*, 131, 268-286.
  119. Anwar, M., C. Newman, D. Macdonald, M. Woolhouse, and D. Kelly (2000) Coccidiosis in the European badger (*Meles meles*) from England, an epidemiological study. *Parasitology*, 120, 255-260.
  120. Newman, C., D.W. Macdonald, and M.A. Anwar (2001) Coccidiosis in the European badger, *Meles meles* in Wytham Woods: infection and consequences for growth and survival. *Parasitology*, 123, 133-142.
  121. Nouvellet, P., C. Newman, C. Buesching, and D.W. Macdonald (2013) A multi-metric approach to investigate the effects of weather conditions on the demographic of a terrestrial mammal, the European badger (*Meles meles*). *Plos One*, 8, e68116.
  122. Macdonald, D.W. and C. Newman (2002) Population dynamics of badgers (*Meles meles*) in Oxfordshire, UK: numbers, density and cohort life histories, and a possible role of climate change in population growth. *Journal of Zoology*, 256, 121-138.
  123. Dutton, A., B. Gratwicke, C. Hepburn, E.A. Herrera, and D.W. Macdonald (2013) Tackling unsustainable wildlife trade. *Key Topics in Conservation Biology 2* (eds D.W. Macdonald and K.J. Willis), pp. 74-91. Wiley-Blackwell, Malaysia.
  124. Auffray, J.C., E. Tchermou, and E. Nevo (1988) Origine du commensalisme de la souris domestique (*Mus musculus domesticus*) vis-à-vis de l'homme. *Comptes rendus de l'Académie des sciences. Série 3, Sciences de la vie*, 307, 517-522.
  125. Auffray, J.C., F. Vanlerberghe, and J. Britton-Davidian (1990) The house mouse progression in Eurasia: a palaeontological and archaeozoological approach. *Biological Journal of the Linnean Society*, 41, 13-25.
  126. Cucchi, T., J.-D. Vigne, J.C. Auffray, P. Croft, and E. Peltenburg (2002) Introduction involontaire de la souris domestique (*Mus musculus domesticus*) à Chypre dès le Néolithique pré-céramique ancien (fin IXe et VIIIe millénaires av. J.-C.). *Comptes Rendus Palevol*, 1, 235-241.
  127. Rodda, G.H., T.H. Fritts, and D. Chiszar (1997) The disappearance of Guam's wildlife. *Bioscience*, 47, 565-574.
  128. <http://kids.fao.org/glipha/>.
  129. Fevre, E.M., B.M.d.C. Bronsvoort, K.A. Hamilton, and S. Cleaveland (2006) Animal movements and the spread of infectious diseases. *Trends in microbiology*, 14, 125-131.
  130. Karesh, W.B., R.A. Cook, E.L. Bennett, and J. Newcomb (2005) Wildlife trade and global disease emergence. *Emerging Infectious Diseases*, 11, 1000.
  131. Molony, S.E., C.V. Dowding, P.J. Baker, I.C. Cuthill, and S. Harris (2006) The effect of translocation and temporary captivity on wildlife rehabilitation success: An experimental study using European hedgehogs (*Erinaceus europaeus*). *Biological Conservation*, 130, 530-537.
  132. Kelly, A., S. Goodwin, A. Grogan, and F. Mathews (2008) Post-release survival of hand-reared pipistrelle bats (*Pipistrellus* spp.). *Animal Welfare*, 17, 375-382.
  133. Davidson, W.R. and V.F. Nettles (1992) Relocation Of Wildlife - Identifying And Evaluating Disease Risks. *Transactions Of The Fifty-Seventh North American Wildlife And Natural Resources Conference*, 466-473.
  134. Gómez, A. and A.A. Aguirre (2008) Infectious diseases and the illegal wildlife trade. *Annals of the New York Academy of Sciences*, 1149, 16-19.
  135. Rosen, G.E. and K.F. Smith (2010) Summarizing the evidence on the international trade in illegal wildlife. *EcoHealth*, 7, 24-32.
  136. Wyler, L.S. and P.A. Sheikh (2008) International illegal trade in wildlife: Threats and US policy. DTIC Document.
  137. Smith, K.F., M. Behrens, L.M. Schloegel, N. Marano, S. Burgiel, and P. Daszak (2009) Reducing the risks of the wildlife trade. *Science*, 324, 594.
  138. Holt, R.D. and A.P. Dobson (2006) Extending the principles of community ecology to address the epidemiology of host-pathogen systems. *Disease Ecology* (eds S.K. Collinge and C. Ray), pp. 6 - 27. Oxford University Press, Oxford.
  139. Weldon, C., L.H. du Preez, A.D. Hyatt, R. Müller, and R. Speare (2004) Origin of the amphibian chytrid fungus. *Emerging Infectious Diseases*, 10, 2100.
  140. Fisher, M.C. and T.W. Garner (2007) The relationship between the emergence of *Batrachochytrium dendrobatidis*, the international trade in amphibians and introduced amphibian species. *Fungal Biology Reviews*, 21, 2-9.
  141. Pinder, A.C. and R.E. Gozlan (2003) Sunbleak and topmouth gudgeon: two new additions to Britain's freshwater fishes. *British Wildlife*, 15, 77-83.
  142. Kosmider, R., A. Paterson, A. Voas, and H. Roberts (2013) *Echinococcus multilocularis* introduction and establishment in wildlife via imported beavers. *Veterinary Record*, 172, 606-606.
  143. Barlow, A., B. Gottstein, and N. Mueller (2011) *Echinococcus multilocularis* in an imported captive European beaver (*Castor fiber*) in Great Britain. *Veterinary Record*, 169, 339-339.
  144. Campbell, R.D., A. Harrington, A. Ross, and L. Harrington (2012) Distribution, population assessment and activities of beavers in Tayside. Scottish Natural Heritage Commissioned Report No.540.
  145. Davidson, R. and L. Robertson (2012) European pet travel: Misleading information from veterinarians and government agencies. *Zoonoses and Public Health*, 59, 575-583.
  146. Telfer, S., M. Bennett, K. Boun, D. Carslake, R. Cavanagh, S. Hazel, T. Jones, and M. Begon (2005) Infection with cowpox virus decreases female maturation rates in wild populations of woodland rodents. *Oikos*, 109, 317-322.
  147. Smith, C., H. Field, and L.F. Wang (2011) Bat Coronaviruses. Investigating the Role of Bats in Emerging Zoonoses. pp. 102-122. Food and Agricultural Organisation of the United Nations, Rome, Italy.
  148. Cotten, M., T.T. Lam, S.J. Watson, A.L. Palser, V. Petrova, P. Grant, O.G. Pybus, A. Rambaut, Y. Guan, and D. Pillay (2013) Full-genome deep sequencing and phylogenetic analysis of novel human betacoronavirus. *Emerging Infectious Diseases*, 19, 736.
  149. Graham, R.L. and R.S. Baric (2010) Recombination, reservoirs, and the modular spike: mechanisms of coronavirus cross-species transmission. *Journal of virology*, 84, 3134-3146.
  150. Li, W., Z. Shi, M. Yu, W. Ren, C. Smith, J.H. Epstein, H. Wang, G. Cramer, Z. Hu, and H. Zhang (2005) Bats are natural reservoirs of SARS-like coronaviruses. *Science*, 310, 676-679.
  151. Reusken, C.B., P.H. Lina, A. Pielaat, A. de Vries, C. Dam-Deisz, J. Adema, J.F. Drexler, C. Drosten, and E.A. Kooi (2010) Circulation of group 2 coronaviruses in a bat species common to urban areas in Western Europe. *Vector-Borne and Zoonotic Diseases*, 10, 785-791.
  152. August, T.A., F. Mathews, and M.A. Nunn (2012) Alphacoronavirus detected in bats in the United Kingdom. *Vector-Borne and Zoonotic Diseases*, 12, 530-533.
  153. Rushton, S.P., P.W.W. Lurz, J. Gurnell, P. Nettleton, C. Brummer, M.D.F. Shirley, and A.W. Sainsbury (2006) Disease threats posed by alien species: the role of a poxvirus in the decline of the native red squirrel in Britain. *Epidemiology and Infection*, 134, 521-533.
  154. Woodford, M.H. (1993) International disease implications for wildlife translocation. *Journal of Zoo and Wildlife Medicine*, 24, 265-270.
  155. Viggers, K.L., D.B. Lindenmayer, and D.M. Spratt (1993) The importance of disease in reintroduction programs. *Wildlife Research*, 20, 687-698.
  156. Gelling, M. (2010) Health and welfare in reintroductions: lessons from small mammals. Department of Zoology, University of Oxford, Oxford.
  157. Gelling, M., I. Montes, T.P. Moorhouse, and D.W. Macdonald (2010) Captive Housing

- during Water Vole (*Arvicola terrestris*) Reintroduction: Does Short-Term Social Stress Impact on Animal Welfare? *Plos One*, 5.
158. Moorhouse, T.P., M. Gelling, G.W. McLaren, R. Mian, and D.W. Macdonald (2007) Physiological consequences of captive conditions in water voles (*Arvicola terrestris*). *Journal of Zoology*, 271, 19-26.
159. Cunningham, A.A. (1996) Disease risks of wildlife translocations. *Conservation Biology*, 10, 349-353.
160. McCallum, H. and B.A. Hocking (2005) Reflecting on ethical and legal issues in wildlife disease. *Bioethics*, 19, 336-347.
161. Miller, P. (2007) Tools and techniques for disease risk assessment in threatened wildlife conservation programmes. *International Zoo Yearbook*, 41, 38-51.
162. Wobeser, G. (2002) Disease management strategies for wildlife. *Revue Scientifique Et Technique-Office International Des Epizooties*, 21, 159-178.
163. <http://www.hse.gov.uk/biosafety/diseases/zoonoses.htm>.
164. <http://www.hse.gov.uk/pubns/ais2.pdf>.
165. <http://www.defra.gov.uk/publications/files/pb13851-zoonoses2011.pdf>.
166. Tam, C., L. Viviani, B. Adak, E. Bolton, J. Dodds, J. Coven, M. Evans, J. Gray, P. Hunter, K. Jackson, L. Letley, K. Neal, G. Rait, G. Smith, B. Smyth, D. Tompkins, M. van der Es, L. Rodrigues, and S. O'Brien (2011) The Second Study of Infectious Intestinal Disease in the Community (IID2 Study). University of Manchester.
167. Tam, C.C., L.C. Rodrigues, L. Viviani, J.P. Dodds, M.R. Evans, P.R. Hunter, J.J. Gray, L.H. Letley, G. Rait, and D.S. Tompkins (2012) Longitudinal study of infectious intestinal disease in the UK (IID2 study): incidence in the community and presenting to general practice. *Gut*, 61, 69-77.
168. Kurtenbach, K., S. De Michelis, and H.S. Sewell (1999) The role of wildlife in the epidemiology of Lyme disease. *Zoonotic Diseases of UK Wildlife*. Proceedings of the British Veterinary Association Congress. Bath.
169. Voordonu, M.J., H. Tupper, Ö. Önder, G. Devevey, C.J. Graves, B.D. Kemps, and D. Brisson (2013) Reductions in Human Lyme Disease Risk Due to the Effects of Oral Vaccination on Tick-to-Mouse and Mouse-to-Tick Transmission. *Vector-Borne and Zoonotic Diseases*.
170. Racey, P., A. Hutson, and P. Lina (2013) Bat Rabies, Public Health and European Bat Conservation. *Zoonoses and Public Health*, 60, 58-68.
171. Fooks, A.R., L.M. McElhinney, D.J. Pounder, C.J. Finnegan, K. Mansfield, N. Johnson, S.M. Brookes, G. Parsons, K. White, and P.G. McIntyre (2003) Case report: isolation of a European bat lyssavirus type 2a from a fatal human case of rabies encephalitis. *Journal of medical virology*, 71, 281-289.
172. Fooks, A., S. Brookes, N. Johnson, L. McElhinney, and A. Hutson (2003) European bat lyssaviruses: an emerging zoonosis. *Epidemiology and Infection*, 131, 1029-1039.
173. Harris, S.L., S.M. Brookes, G. Jones, A.M. Hutson, P.A. Racey, J. Aegerter, G.C. Smith, L.M. McElhinney, and A.R. Fooks (2006) European bat lyssaviruses: Distribution, prevalence and implications for conservation. *Biological Conservation*, 131, 193-210.
174. Johnson, N., D. Selden, G. Parsons, D. Healy, S. Brookes, L. McElhinney, A. Hutson, and A. Fooks (2003) Isolation of a European bat lyssavirus type 2 from a Daubenton's bat in the United Kingdom. *Veterinary Record*, 152, 383-387.
175. Smith, G., S. Brookes, S. Harris, J. Aegerter, G. Jones, and A. Fooks (2006) EBLV2 prevalence in the United Kingdom as determined by surveillance testing. *Developments in biologicals*, 125, 265-271.
176. Dreesen, D.W. (1990) *Toxoplasma-Gondii* Infections in Wildlife. *Journal of the American Veterinary Medical Association*, 196, 274-276.
177. Vijayachari, P., A.P. Sugunan, and A.N. Shrivani (2008) Leptospirosis: an emerging global public health problem. *Journal of Biosciences*, 33, 557-569.
178. Webster, J.P., G. Lloyd, and D.W. Macdonald (1995) Q fever (*Coxiella burnetii*) reservoir in wild brown rat (*Rattus norvegicus*) populations in the U.K. *Parasitology*, 110, 31-35.
179. Webster, J.P. and D.W. Macdonald (1995) Diseases in farm rats: practical and theoretical implications. Review monograph: Rodent borne diseases (eds A.D. Mehan and D.I.G. Bateman).
180. Nerlich, B., B. Brown, and N. Wright (2009) The ins and outs of biosecurity: bird 'flu in East Anglia and the spatial representation of risk. *Sociologia Ruralis*, 49, 344-359.
181. [http://www.hpa.org.uk/web/HPAweb/HPAwebStandard/HPAweb\\_C/131713323654](http://www.hpa.org.uk/web/HPAweb/HPAwebStandard/HPAweb_C/131713323654).
182. Jones, K.E., N.G. Patel, M.A. Levy, A. Storeygard, D. Balk, J.L. Gittleman, and P. Daszak (2008) Global trends in emerging infectious diseases. *Nature*, 451, 990-993.
183. <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5227a5.htm>.
184. Guan, Y., B. Zheng, Y. He, X. Liu, Z. Zhuang, C. Cheung, S. Luo, P. Li, L. Zhang, and Y. Guan (2003) Isolation and characterization of viruses related to the SARS coronavirus from animals in southern China. *Science*, 302, 276-278.
185. Rouquet, P., J.-M. Froment, M. Bermejo, A. Kilbourn, W. Karesh, P. Reed, B. Kumulungui, P. Yaba, A. Delicat, and P.E. Rollin (2005) Wild animal mortality monitoring and human Ebola outbreaks, Gabon and Republic of Congo, 2001-2003. *Emerging Infectious Diseases*, 11, 283.
186. Weiss, R.A. and A.R. McLean (2004) What have we learnt from SARS? Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 359, 1137-1140.
187. Pulliam, J.R.C., J.H. Epstein, J. Dushoff, S.A. Rahman, M. Bunning, A.A. Jamaluddin, A.D. Hyatt, H.E. Field, A.P. Dobson, and P. Daszak (2012) Agricultural intensification, priming for persistence and the emergence of Nipah virus: a lethal bat-borne zoonosis. *Journal of The Royal Society Interface*, 9, 89-101.
188. Morse, S.S., J.A. Mazet, M. Woolhouse, C.R. Parrish, D. Carroll, W.B. Karesh, C. Zambrana-Toriel, W.I. Lipkin, and P. Daszak (2012) Prediction and prevention of the next pandemic zoonosis. *The Lancet*, 380, 1956-1965.
189. <http://www.ebi.ac.uk/zcan/disease/genes13.html>.
190. <http://www.defra.gov.uk/ahvla-en/disease-control/notifiable/>.
191. <http://www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/NotificationsOfInfectiousDiseases/ReportingProcedures/>.
192. <http://www.defra.gov.uk/ahvla-en/disease-control/notifiable/#measure>.
193. Brown, E.D., D.W. Macdonald, T.E. Tew, and I.A. Todd (1994) *Apodemus sylvaticus* infected with *Heligmosomoides polygyrus* (Nematoda) in an arable ecosystem: Epidemiology and effects of infection on the movements of male mice. *Journal of Zoology*, 234, 623-640.
194. Tew, T.E. and D.W. Macdonald (1993) The effects of harvest on arable wood mice, *Apodemus sylvaticus*. *Biological Conservation*, 65, 279-283.
195. <http://www.defra.gov.uk/animal-diseases/notifiable/>.
196. <http://www.defra.gov.uk/animal-diseases/monitoring/>.
197. Moss, S., S. Turner, R. Trout, P. White, P. Hudson, A. Desai, M. Armesto, N. Forrester, and E. Gould (2002) Molecular epidemiology of rabbit haemorrhagic disease virus. *Journal of General Virology*, 83, 2461-2467.
198. Fuller, H., D. Chaisey, M. Lucas, and J. Gibbins (1993) Rabbit haemorrhagic disease in the United Kingdom. *Veterinary Record*, 133, 611-613.
199. Trout, R., D. Chaisey, and G. Sharp (1997) Seroepidemiology of rabbit haemorrhagic disease (RHD) in wild rabbits (*Oryctolagus cuniculus*) in the United Kingdom. *Journal of Zoology*, 243, 846-853.
200. <http://www.defra.gov.uk/ahvla-en/files/pub-survrep-w0212.pdf>.
201. Bradley, C.A. and S. Altizer (2007) Urbanization and the ecology of wildlife diseases. *Trends in Ecology and Evolution*, 22, 95-102.
202. DeStefano, S. and R.M. DeGraaf (2003) Exploring the ecology of suburban wildlife. *Frontiers in Ecology and the Environment*, 1, 95-101.
203. Baker, P.J. and S. Harris (2007) Urban mammals: what does the future hold? An analysis of the factors affecting patterns of use of residential gardens in Great Britain. *Mammal Review*, 37, 297-315.
204. Marzluff, J.M. (2001) Worldwide urbanization and its effects on birds. *Avian ecology and conservation in an urbanizing world*, pp. 19-47. Springer.
205. Miller, M., I. Gardner, C. Kreuder, D. Paradies, K. Worcester, D. Jessup, E. Dodd, M. Harris, J. Ames, and A. Packham (2002) Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). *International journal for parasitology*, 32, 997-1006.
206. Lanciotti, R.S., G.D. Ebel, V. Deubel, A.J. Kerst, S. Murri, R. Meyer, M. Bowen, N. McKinney, W.E. Morrill, and M.B. Crabtree (2002) Complete genome sequences and phylogenetic analysis of West Nile virus strains isolated from the United States, Europe, and the Middle East. *Virology*, 298, 96-105.
207. Marra, P.P., S. Griffing, C. Caffrey, A.M. Kilpatrick, R. McLEAN, C. Brand, E. Saito, A.P. Dupuis, L. Kramer, and R. Novak (2004) West Nile virus and wildlife. *Bioscience*, 54, 393-402.
208. Petersen, L.R. and E.B. Hayes (2004) Westward ho?-The spread of West Nile virus. *The New England journal of medicine*, 351, 2257.
209. Bichet, C., R. Scheifler, M. Coeurdassier, R. Julliard, G. Sorci, and C. Loiseau (2013) Urbanization, Trace Metal Pollution, and Malaria Prevalence in the House Sparrow. *Plos One*, 8, e53866.
210. Orlioussi, G., Z. Kasprzykowski, W. Dobicki, P. Pokorny, and R. Polecholski (2010) Geographical and habitat differences in concentrations of copper, zinc and arsenic in eggshells of the Rook *Corvus frugilegus* in Poland. *Journal of Ornithology*, 151, 279-286.
211. Roux, K. and P. Marra (2007) The Presence and Impact of Environmental Lead in Passerine Birds Along an Urban to Rural Land Use Gradient. *Archives of Environmental Contamination and Toxicology*, 53, 261-268.
212. Scheifler, R., M. Coeurdassier, C. Morilhat, N. Bernard, B. Faivre, P. Flicoteaux, P. Giraudoux, M. Noël, P. Piotte, and D. Rieffel (2006) Lead concentrations in feathers and blood of common blackbirds (*Turdus merula*) and in earthworms inhabiting unpolluted and moderately polluted urban areas. *Science of The Total Environment*, 371, 197-205.
213. Linzey, D., J. Burroughs, L. Hudson, M. Marini, J. Robertson, J. Bacon, M. Nagarkatti, and P. Nagarkatti (2003) Role of environmental pollutants on immune functions, parasitic infections and limb malformations in marine toads and whistling frogs from Bermuda. *International Journal of Environmental Health Research*, 13, 125-148.
214. McAloose, D. and A.L. Newton (2009) Wildlife cancer: a conservation perspective. *Nature reviews cancer*, 9, 517-526.
215. Erren, T., D. Zeuß, F. Steffany, and B. Meyer-Rochow (2009) Increase of wildlife cancer: an echo of plastic pollution? *Nature reviews cancer*, 9, 842-842.
216. Newman, C. and D.W. Macdonald (2013) 2. The implications of climate change for terrestrial UK mammals. *Terrestrial biodiversity Climate change impacts report card Technical paper*.
217. Pascual, M. and M.J. Bouma (2009) Do rising temperatures matter. *Ecology*, 90, 906-912.
218. Gratz, N.G. (1999) Emerging and resurging vector-borne diseases. *Annual Review of Entomology*, 44, 51-75.
219. Lindgren, E., L. Tälleklint, and T. Polfeldt (2000) Impact of climatic change on the northern latitude limit and population density of the disease-transmitting European tick *Ixodes ricinus*. *Environmental health perspectives*, 108, 119.
220. Arlian, L., D. Vyszynski-Moher, and M. Pole (1989) Survival of adults and developmental stages of *Sarcoptes scabiei* var. *canis* when off the host. *Experimental & applied acarology*, 6, 181-187.
221. Newman, T.J., P.J. Baker, and S. Harris (2002) Nutritional condition and survival of red foxes with sarcoptic mange. *Canadian journal of zoology*, 80, 154-161.
222. Bornstein, S., T. Mörmel, and W.M. Samuel (2001) *Sarcoptes scabiei* and sarcoptic mange. *Parasitic Diseases of Wild Mammals*, Second Edition, 107-119.
223. Ruylys, L., D.A. Taggart, W.G. Breed, and D. Schult (2009) Sarcoptic mange in southern hairy-nosed wombats (*Lasiornis latifrons*): distribution and prevalence in the Murraylands of South Australia. *Australian Journal of Zoology*, 57, 129-138.
224. Gakuja, F., J. Ombui, N. Maingi, G. Muchemi, W. Ogara, R.C. Sorriguer, and S. Alasaad (2012) Sarcoptic mange and cheetah conservation in Masai Mara (Kenya): Epidemiological study in a wildlife/livestock system. *Parasitology-Cambridge*, 139, 1587.
225. Woodroffe, R. (1999) Managing disease threats to wild mammals. *Animal Conservation*, 2, 185-193.
226. Breed, A.C., R.K. Plowright, D.T. Hayman, D.L. Knobel, F.M. Molenaar, D. Gardner-Roberts, S. Cleaveland, D.T. Haydon, R.A. Kock, and A.A. Cunningham (2009) Disease management in endangered mammals. *Management of Disease in Wild Mammals* pp. 215-239. Springer.
227. Purse, B.V., P.S. Mellor, D.J. Rogers, A.R. Samuel, P.P. Mertens, and M. Baylis (2005) Climate change and the recent emergence of bluetongue in Europe. *Nature Reviews Microbiology*, 3, 171-181.
228. Alan Pounds, J., M.R. Bustamante, L.A. Coloma, J.A. Consuegra, M.P.L. Fogden, P.N. Foster, E. La Marca, K.L. Masters, A. Merino-Viteri, R. Puschendorf, S.R. Ron, G.A. Sanchez-Azofeifa, C.J. Still, and B.E. Young (2006) Widespread amphibian extinctions from epidemic disease driven by global warming. *Nature*, 439, 161-167.
229. Albrechtsen, L., J.E. Fa, B. Barry, and D.W. Macdonald (2005) Contrasts in availability and consumption of animal protein in Bioko Island, West Africa: the role of bushmeat. *Environmental Conservation*, 32, 340-348.
230. Fa, J.E., S. Seymour, J. Dupain, R. Amin, L. Albrechtsen, and D.W. Macdonald (2006) Getting to grips with the magnitude of exploitation: bushmeat in the Cross-Sanaga rivers region, Nigeria and Cameroon. *Biological Conservation*, 129, 497-510.
231. Macdonald, D.W., P.J. Johnson, L. Albrechtsen, S. Seymour, J. Dupain, A. Hall, and J.E. Fa (2012) Bushmeat trade in the Cross-Sanaga rivers region: Evidence for the importance of protected areas. *Biological Conservation*, 147, 107-114.
232. Swift, L., P.R. Hunter, A.C. Lees, and D.J. Bell (2007) Wildlife trade and the emergence of infectious diseases. *EcoHealth*, 4, 25-30.
233. Baker, S.E., R. Cain, F. van Kesteren, Z.A. Zommers, N. D'Crucze, and D.W. Macdonald (2013) Rough Trade: Animal Welfare in the Global Wildlife Trade. *Bioscience*, 63, 928-938.
234. Karesh, W.B., R.A. Cook, M. Gilbert, and J. Newcomb (2007) Implications of wildlife trade on the movement of avian influenza and other infectious diseases. *Journal Of Wildlife Diseases*, 43, S55-S59.

# People's Trust for Endangered Species

Our wildlife is disappearing. Our delicately balanced ecosystem is under great threat. One in ten species in the UK faces extinction and thousands more worldwide. People's Trust for Endangered Species saves endangered species and their habitats by involving people in practical conservation.

There is good reason to be alarmed if our own countryside is failing to sustain the 60 or so resident, wild mammals in the UK or around our shores. 39 of these species are on the UK conservation priority list. We have raised over a million pounds for work on British native mammals over the last decade for work on water voles, wildcats, dormice, hedgehogs, harvest mice, red squirrels, pygmy shrews, otters, polecats, pine martens, brown hares, water shrews, Cuvier's beaked whale and seven types of bat.

Each year we refocus and build our mammal programme. By gathering evidence through our own regular mammal monitoring our priorities adapt to where help is most needed. We support our partner organisations to conserve all priority mammal species but concentrate our in-house expertise currently on dormice, hedgehogs and water voles.

Almost all of our income comes from generous donations by people who share our passion for wildlife. Please help us today by donating to our work at [www.ptes.org](http://www.ptes.org).

Thank you.

People's Trust for Endangered Species 15 Cloisters House 8 Battersea Park Road London SW8 4BG  
020 7498 4533 [enquiries@ptes.org](mailto:enquiries@ptes.org) [www.ptes.org](http://www.ptes.org)  
Registered Charity Number 274206



## WildCRU

The Wildlife Conservation Research Unit's mission is to undertake original research on aspects of fundamental biology relevant to solving practical problems of wildlife conservation and environmental management.

WildCRU Zoology University of Oxford The Reanati-Kaplan Centre Tubney House Abingdon Road Tubney  
Abingdon OX13 5QL  
01865 611100 [wcru@zoo.ox.ac.uk](mailto:wcru@zoo.ox.ac.uk) [www.wildcru.org](http://www.wildcru.org)



### Copyright People's Trust for Endangered Species 2014

All rights reserved. Extracts of this publication may be made for non-commercial, in-house use, subject to the source being acknowledged. The views expressed in this document are not necessarily those of People's Trust for Endangered Species. Its officers accept no liability whatsoever for loss or damage arising from the interpretation or use of the information, or reliance upon the views contained therein.

*The State of Britain's Mammals a focus on disease* is the tenth of the annual updates following the publication of *Britain's Mammals: The Challenge for Conservation*. Copies of all publications can be obtained by contacting the People's Trust for Endangered Species at [www.ptes.org](http://www.ptes.org)

Photo credits: red squirrel (cover), ©iStockphoto.com/blackbravo; water vole, Ben Andrews; red squirrel, ©iStockphoto.com/rickochet; wild boar, ©iStockphoto.com/Byrdyak; red deer, Ben Andrews; brown rat, Laurie Campbell; badger, Ben Andrews; pippistrelle bat, Laurie Campbell; grey squirrel, ©iStockphoto.com/MichaelStubblefield; Daubenton's bat, Hugh Clark, BCT; rabbit, Ben Andrews; red fox, Ben Andrews.