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Future UK land use policy and the risk of infectious disease in humans, livestock and wild animals☆

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ABSTRACT

We explore the degree to which the consideration of infectious diseases may be of importance in the formulation of land use policy. We look at the diseases of humans, domestic livestock and wild vertebrates, and distinguish two types of human disease: those which form epidemics involving transmission from person to person, and those which are contracted from non-human sources and spread very little through the population. Land use affects the density and distribution of people, and urbanisation typically increases the risk of an epidemic and the speed with which it spreads. More subtle effects may occur through changes in the network of contacts between individuals. Land use policy that affects the distribution of breeding sites for disease vectors (e.g. mosquitoes) or the passage of potential pathogenic microorganisms through the environment from farm animals to humans can also influence non-epidemic disease risk.

Livestock disease is critically affected by stocking density and the network of contacts between individuals and herds. Land use and agricultural policy can be very important in reducing the risks of disease outbreaks. We explore the complex relationship between intensification and disease risks. We suspect that land use policy may affect the viability of threatened species of vertebrates, though our relatively poor knowledge of disease epidemiology in wild animals makes policy formation difficult. Though climate change may act together with land use policy to determine disease risk, we consider this interaction to less important than that between land use and the socioeconomic drivers of global change. We conclude by assessing the importance of disease in the three types of host to land use policy. We suggest that the consideration of wildlife diseases is a low to medium priority for land use policy, though we attach high uncertainty to our conclusion; the consideration of human diseases we think a low priority (with medium uncertainty); while that of livestock diseases we argue is a high priority (with low uncertainty).

Introduction

In this article we explore how changing land use patterns in the 21st century may influence the types and prevalence of infectious diseases afflicting humans, livestock and wild animals, and investigate the implications for policy development. By infectious diseases we mean those caused by pathogenic agents such as bacteria, viruses or single- and multi-celled parasitic organisms. We do not discuss the fascinating topics of how changes in the built and natural environment may affect human wellbeing through exposure to pollutants or nanoparticles, opportunities for exercise, mental health, etc. To make our task manageable we deal only with domesticated and wild vertebrates, and do not consider how land use may affect the spread and severity of crop and forestry diseases, nor the topical issue of bee diseases. Our review concentrates on epidemiological questions. While we touch on economic and related social science topics, they are not our main concerns. We also focus on land use issues as they apply to disease in the UK.

Land use change has historically been driven by political, societal and economic forces which have led to the marked urban expansion and agricultural intensification seen in the UK in the past two centuries (Overton, 1996). In 1801 about three in ten people in Britain lived in a town or city (Brown, 1991): by 2001, this ratio had risen to nearly eight in ten (Pointer, 2005).

The same population and economic pressures that have shaped land use have had huge effects on the health of the human population, and on disease in domesticated animals and wildlife. We believe that the explicit consideration of disease has had only a minor influence on land use policy in the past, and that in the future it will remain subordinate to the main socioeconomic drivers. Nevertheless, our increasing understanding of pathogens, and how they interact with their hosts and the environment, allows us a better appreciation of how land use policy may affect the prevalence

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of infectious diseases. We argue that a consideration of disease risk should be part of an evidence-based appraisal of different land use policy options.

We have organised the rest of the paper as follows. First we introduce some basic epidemiological concepts in a section that can be skipped by readers with a disease background. We then look at human epidemic diseases and explore the health policy implications of the local density and distribution of people which in turn are determined by patterns of land use. We then ask how land use may affect the risk of human exposure to diseases established in other species for which humans are accidental hosts. In the next two sections we concentrate on diseases of livestock and then of wild animals. In the final two sections we look ahead to future challenges and assess the relative importance of land use policy for the different types of disease system considered here. This short article cannot be a comprehensive treatment of all aspects of disease in humans and other vertebrates. Instead we give illustrative examples, some (placed in boxes) at slightly greater length, and try to tease out general principles and identify the topics of greatest relevance.

Epidemiology background

The study of disease risk and spread requires an amalgam of biology and applied mathematics and there is a very large technical literature relevant to the topics in this review (Levin et al., 1997). It is not necessary for our purposes to delve into much of this detail, but a few basic epidemiological ideas are useful for the discussion that follows. In epidemiology the concept of the basic reproductive number or R_0 plays a central role. R_0 is the average number of secondary infections that arise when a new infection occurs in a population of susceptible individuals (Anderson and May, 1991). For an infectious disease to invade, the basic reproductive number must be greater than one ($R_0 > 1$). Understanding the conditions (which may involve land use patterns) where $R_0 > 1$ tells us whether an epidemic may occur. For many diseases R_0 is a function of the density of susceptible individuals and there is a threshold host density below which epidemics cannot occur.

The invention of agriculture in about 8000BC allowed local densities of humans and their domesticated animals to increase dramatically, rendering both able to sustain outbreaks of new diseases (McNeill, 1977). These events can be reconstructed from the analysis of contemporary DNA sequence data. Knowing the magnitude of R_0, even roughly, can be immensely valuable in determining health and disease control policy. There are some diseases where $R_0 = 0$. For example, humans contract rabies from diseased animals but seldom if ever infect other humans (Lumio et al., 1986). In other cases R_0 lies between 0 and 1. Thus avian influenza can infect humans and a few secondary cases have been recorded, but there is on average less than one for each primary case. Hence epidemics cannot occur, at least with current strains (World Health Organisation, 2006a). Pathogens that primarily attack animals but can also infect humans are known as zoonoses.

The value of R_0, while critical, does not completely describe disease spread. In particular, details of the contact network, which describes the pattern of contacts between members of a population, can be important. There are many situations in which social contacts are thought to show ‘small-world’ properties, whereby the majority of connections are local but a few very well-connected individuals have contacts spanning different communities (Watts and Strogatz, 1998). These members can be ‘superspreaders’ of infection, and their role in the contact network is clearly fundamental to the spread and control of infection (Lloyd-Smith et al., 2005). So while reducing overall mixing between members of a hypothetical population would lower R_0 and slow the spread of infection, isolating and quarantining or vaccinating those most responsible for infection achieves the same aims through a more targeted means (May and Anderson, 1984).

It is also useful to distinguish the major routes of disease transmission. For many diseases the pathogen is relatively short-lived outside the host and transmission requires contact or close proximity between infectious and susceptible individuals. But some diseases may persist for long periods of time in the environment and, in the case of some bacterial gut pathogens for example, may even be able to reproduce outside the vertebrate host. How land use patterns affect the transport and relative longevity of pathogens in the environment is a significant issue for policy formulation (Chadwick et al., 2008; Oliver et al., 2007). Finally, pathogens may be moved from one host to another by a vector, often a blood-feeding insect or tick. In tropical countries, where malaria and other insect-vectored diseases are major causes of mortality and morbidity, reducing the habitat suitable for vector reproduction is a major goal of land use planning (World Health Organisation, 2006b). In the UK such considerations have been of less importance, though this may change with the increasing spread of vectors through human agency as well as the possible threats posed by climate change (Department of Health, 2008).

Epidemic diseases of humans

Here we look at diseases where $R_0 > 1$, that is where a disease can spread from human-to-human in an epidemic. We discuss first diseases associated with urbanisation, and then infections transmitted by arthropod vectors.

Infectious disease risks associated with urbanisation

Urban areas are characterised by large contiguous populations as well as by high population densities, both of which influence infectious disease. In many cases, this influence is purely a function of the numbers, distribution and contact networks of individuals, but it may also be a more complex consequence of the social and economic status of people attracted to urban centres.

Population density tends to increase R_0 in directly transmitted diseases by increasing the number of contacts between susceptible and infected hosts (Anderson and May, 1991). Measles provides a striking example of this. Epidemics in big cities affect a greater proportion of people than epidemics in small towns and villages, and occur more frequently (see Box 1).

Understanding the structure of the human contact network can help mitigate some of the effects of urbanisation. Schools can play a very important role in disease spread because they bring together large concentrations of susceptible individuals: children not yet exposed to diseases that cause subsequent immunity. This means that the vaccination of schoolchildren against influenza can have a strong effect on the spread of infection through the broader community (Cohen, 2004). The UK contains some of the largest secondary schools in Europe (Eurydice, 2005), particularly in its cities, and these are growing in size. There have been calls for a move towards a system of more distributed, smaller schools for educational reasons (Teach First, 2007), and such a trend would also have positive effects on infection control in the community. Similar issues would apply to the design of other institutions, for examples prisons and army camps.

An area of social science research that would help inform planning for disease risks is the effect of urban concentration and its opposite, urban sprawl, on human contact networks. Within a region and for the same total population density, is it better from the point of view of disease spread to build discrete cities

Box 1: Measles in the UK

Measles is a highly contagious viral disease spread chiefly by inhaling droplets of saliva (Health Protection Agency (e)). Natural immunity is lifelong and hence it is normally an infection of childhood. The MMR vaccine has been effective in reducing measles outbreak sizes but vaccination coverage is low for a number of reasons including alarmist media stories (Jansen et al., 2003). As this is a notifiable disease in the UK there is a rich source of data on measles cases. Before the advent of widespread immunisation, measles epidemics used to take place about every 2 years. The epidemic began when sufficient susceptible hosts were available and ceased when enough individuals were immune, having had the disease. Births in following years replenished the pool of susceptible hosts, eventually giving rise to the conditions ($R_0 > 1$) for a subsequent epidemic.

Over 60 years ago, the statistician Maurice Bartlett used measles data from towns in England and Wales to illustrate the relationship between community size and the timing of epidemics: bigger communities have more frequent epidemics (Fig. 1). Because of random processes, the infection is more likely to become lost in small towns. An epidemic requires both a sufficient density of susceptible individuals and the introduction of the infection (Bartlett, 1960). Similar results are found for other diseases though the pattern of infections such as the common cold and influenza is complicated by the existence of multiple viral strains with different degrees of cross-immunity. The transmission dynamics of measles is an example of how human population density and distribution can interact to influence disease spread at geographic scales. Measles epidemics exhibit complex dynamical patterns correlated with population distributions. They proceed in pronounced waves which begin in large urban centres, where disease extinction never occurs, before radiating to smaller towns (Grenfell et al., 2001).

within which most human-to-human contact occurs, or allow communities to merge, giving rise to a less clumped contact network? Alternatively, might there be little difference because of the homogenising effects of highly connected individuals, who are few in number but highly connected?

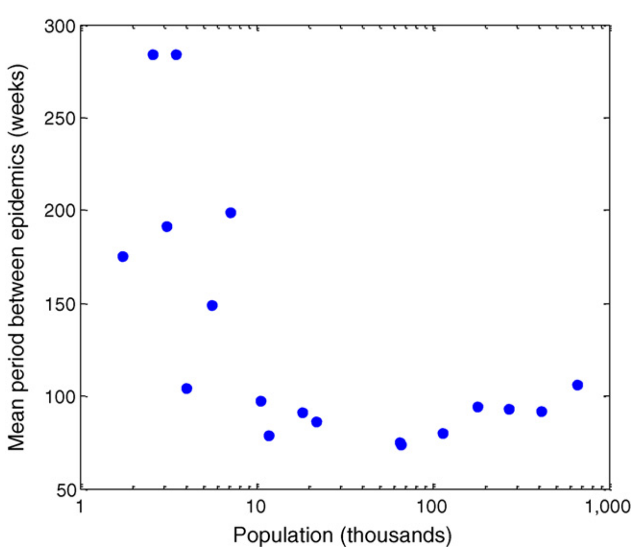

Fig. 1. The relationship between population size and period between measles epidemics. Data points represent towns and cities of different size.

The distribution and density of urban centres is also relevant to contingency planning for the spread of new "emerging" diseases, whether arising 'naturally', for example an influenza pandemic, or by deliberate release in an act of terrorism. In addition to the general issues of numbers and densities, the proximity of major urban areas to hubs of international travel, such as major airports, can be significant. It was through air travel that SARS cases were introduced into cities such as Toronto and Singapore in 2003 (McLean et al., 2005).

The challenges of containing the spread of disease in high density urban areas is amplified if cities attract individuals who are of greater than average susceptibility or who have a contact structure that facilitates the spread of disease. Understanding these issues has been critical in controlling the spread of HIV in urban centres (Centers for Disease Control and Prevention, 2009; Coyle et al., 1998) where major high-risk groups including intravenous drug abusers, sex workers, and at least until the 1980s, communities of gay people with high rates of partner turnover. All these groups tended to congregate in large cities. Unlike directly transmitted diseases such as measles or smallpox, epidemiological theory does not predict that the prevalence of sexually transmitted diseases will always rise with increased population densities. The concentration of HIV in UK cities, and more generally the higher prevalence of sexually transmitted infections in London compared with smaller cities and other areas (Health Protection Agency, 2006a), is most likely due to sociological processes.

Urban areas may also facilitate disease transmission by the structure of their environment, in particular by the quality of the housing stock. There are several health implications of living in poor-quality housing (Bonnefoy, 2007). They include poor respiratory health arising from mould growth, air pollution and poor ventilation. This can increase susceptibility to infectious diseases. Recent studies of tuberculosis cases from Canada and New Zealand highlighted links between housing conditions and the spread and outcome of disease (Canadian Tuberculosis Committee, 2007; Baker et al., 2008). In the UK, migrant communities face a disproportionate burden of disease from tuberculosis and HIV (Health Protection Agency, 2006b). Where these communities are concentrated in poor-quality housing, these poor living conditions increase their susceptibility to infection.

The most rapid phase of urbanisation in the UK is almost certainly over. But elsewhere in the world, urbanisation is one of the most significant social drivers and has huge consequences for the environment, food security and economics as well as health. "Megacities" with populations in excess of 10 million people are growing in number in Africa, South America and Asia (United Nations, 2007). Much of this urbanisation is endogenous and is not subject to urban planning. These cities are likely to be incubation grounds for novel emerging infections, and globalisation will spread these diseases throughout the world, including the UK (Ho and Su, 2004; Saker et al., 2004). Simple urban planning measures, for example not placing live animal markets in city centres, may help control some of these risks.

Vector-borne diseases

In the tropics there are major vector-borne diseases that are either restricted to man (e.g. malaria; Greenwood and Mutabingwa, 2002) or that can cause self-sustaining outbreaks in humans (e.g. yellow fever; Monath, 2001). These very important diseases are chiefly problems in the developing world where the most efficient vectors occur. Even with significant climate change they are unlikely to become threats to the UK (Department of Health, 2008), although malaria was present in the UK until the early 1900s (Dobson, 2003) and as recently as the late 1940s in Italy. However, novel emergent vector-borne diseases cannot be discounted. For example, the Chikungunya virus, which causes a debilitating disease in humans, used to be confined to the tropics and is spread primarily by the yellow fever mosquito (*Aedes aegyptii*). In the mid-2000s, however, it mutated so that it could be spread by the Asian tiger mosquito (*Aedes albopictus*) which can survive in warm but non-tropical regions (Schuffenecker et al., 2006). Capable of breeding in water trapped in tyres, *A. albopictus* has been spread round the globe, in particular through the extensive trade in used tyres (Knudsen, 1995). This vector was responsible for a contained outbreak in Italy in 2007 (Bonilauri et al., 2008), and the Health Protection Agency considers *A. albopictus* as a potential coloniser of the UK (Health Protection Agency (a)). There are several species of *Aedes* in the UK, some restricted to tree holes, others to freshwater bodies, and others still to salt marshes (Cranston et al., 1987). Were one of these species to become a vector of Chikungunya (or another pathogen) it would clearly influence land use policy decisions in ways that would be difficult to predict.

Box 2: TBE in Central and Eastern Europe

Tick-borne encephalitis (TBE) is a vector-borne viral disease that affects the central nervous system (NHS: TBE). The virus naturally infects rodents, particularly mice, and is chiefly transmitted by woodland-dwelling ticks. However, the virus also infects other vertebrate species including humans. It causes severe symptoms in humans, including inflammation of the brain (encephalitis). Although an effective vaccine exists, uptake has not been extensive in all countries where the disease is widespread.

While human cases of TBE have been gradually going up across Europe, there has been a marked increase in Central and Eastern Europe since 1993 (Randolph, 2001). Climate change may play a role, but does not fully explain these trends (Randolph, 2004). Indeed, recent work indicates that changes in agricultural and human behavioural patterns resulting from societal disruption after the ending of Soviet rule may be responsible (Sumilo et al., 2008). Changes in land cover played an important role in several countries, with agriculture shifting from large-scale, state owned enterprises to small privately owned farms, allowing the growth of habitats favourable for ticks. In addition, socioeconomic conditions played a role. Increased unemployment and poverty drove people into forests to seek wild food sources, increasing their exposure to infection. While economic conditions are clearly very different in the UK, land use policies that increased tick habitat, combined with policies that encouraged more people to visit the countryside, could contribute to similar changes in the risk of the disease. For TBE the risks can be controlled by vaccination but this might not be true for other emerging diseases.

Non-epidemic diseases of humans

Changes in land use policy may bring people into contact with known but previously rare pathogens, or even with disease organisms hitherto not recorded in humans. Here we have in mind infectious diseases that do not cause epidemics ($R_0 < 1$) and where policy decisions chiefly revolve around reducing the risk of exposure. We structure our discussion in terms of the major environmental reservoirs for the disease and their modes of transmission to humans.

Vector-borne diseases with animal reservoirs

The risk of exposure to vector-borne diseases is influenced by the numbers and distribution of the animal species that make up the environmental reservoir of the disease; the numbers and distribution of the vector (here always an insect or tick) and the behaviour and activities of humans that influence the probability of being bitten by the vector. Changes in land use potentially affect all three of these.

The radical land use changes that occurred after the break up of the Soviet Union provide excellent examples of how these factors can combine to affect disease risk. The case of tick-borne encephalitis in the Baltic States is described in more detail in Box 2. The major tick-borne disease in the UK is Lyme disease (borreliosis) transmitted chiefly by hard-bodied ticks in the genus *Ixodes* (Health Protection Agency (b)). The pathogen occurs in many wild animals but infections in deer transmitted by deer ticks are the chief risk to man (Simpson, 2002). Land use policy that leads to increased deer populations, directly through greater forestation or less intensive agriculture, or indirectly through removal of human and non-human deer predators, tends to increase prevalence (Böhm et al., 2007). Similarly, increased recreational use of the countryside will lead to more disease cases. The combination of high levels of tourism and abundant deer makes the New Forest a hot spot for this disease (Smith et al., 2000). Future policy should consider the possibility of novel routes of infection, perhaps through ticks feeding on rodents in urban settings (Matuschka et al., 1997). Better GP education might be needed to help doctors recognise Lyme disease, as well as rarer tick-borne diseases.

There are currently no major human diseases vectored by insects in the UK. Nevertheless, recent experience abroad cautions against ignoring potential risks. West Nile Virus (WNV) was a poorly known mosquito-borne virus until it appeared in North America in 1999 and caused extensive deaths in wild birds (Lanciotti et al., 1999). It is now established and kills over 100 people each year in the US (Hayes et al., 2005). All larval mosquitoes are aquatic. Land use change that results in more freshwater breeding sites (for example flood storage areas) can increase their abundance. The complex biology of mosquitoes and the large number of poorly characterised mosquito-borne viruses circulating in wild vertebrates argue for high levels of vigilance to detect emerging problems such as occurred in the US after the introduction of WNV.

Diseases contracted by direct contact with animals

Humans in the UK rarely contract diseases directly from wild animals. A rare example is a naturalist who died in 2003 after being bitten by a rabid bat, the only reported human rabies infection contracted in the UK since the early 1900s (Fooks et al., 2003). Humans may contact helminth (worm) infections from pets which in turn were infected through contact with wild or farm animals. This can be serious for certain rare infections (e.g. cystic echinococcosis) or for immunocompromised patients (Budke, 2006). Farm workers occasionally contract animal diseases such as bovine tuberculosis, brucellosis, avian flu, Q-fever or even, in exceptional circumstances, foot-and-mouth (Defra, 2007). The pathogen *E. coli* O157 (see also below) has been caught by children at petting zoos (Heuvelink et al., 2002) and during farm visits (Stirling et al., 2008). Were land use changes to increase the numbers of people living in close proximity to or working with farm or wild animals, these risks might increase. This might happen if more land was used as smallholdings for local food production. Probably the most relevant policy responses are improved risk assessment procedures for activities involving animals, and the education of primary health workers to recognise uncommon infections.

Diseases contracted indirectly from animals

Some pathogens derived from wild or farm animals infect humans through long-lived stages, such as spores that are transported through the environment. Here the policy issues include reducing the prevalence of the pathogen in the definitive host, managing contamination of the environment by the pathogen, controlling movement of the pathogen though the environment (for example into watercourses), and reducing human exposure to the pathogen (Chadwick et al., 2008; Oliver et al., 2007).

A major category of pathogens of this type are those that inhabit the gut of farm and wild animals and that can cause diarrhoeal and gastrointestinal diseases in man. These include bacteria, particularly *Campylobacter*, *Salmonella* and verocytotoxic *E. coli* strains such as O157; protozoa including *Cryptosporodia* and *Giardia*; and viruses such as rotavirus (Health Protection Agency (c)). Although it has a slightly different natural history, Leptospirosis, which causes Weil's disease, presents similar issues (Health Protection Agency (d)).

The three main routes of transmission to humans are, in decreasing order of importance: through contaminated food, through the environment, and through direct contact with animals (see above) (Young et al., 2007; Rangel et al., 2005). Contamination via the environment occurs primarily through drinking contaminated water, eating shellfish, and bathing in rivers, lakes and the sea (Mourato et al., 2003). Except where obvious clusters of cases occur, it is often difficult to quantify the risks and distinguish infection through the environment from that through food.

Where do environmental contaminants originate? Stringent sewage treatment regulations have reduced the importance of man as a source of pathogens, while wild animals are seldom abundant enough to cause major contamination. Farm animals and their waste products are the main source of pathogens, especially where animals are reared intensively (Chadwick et al., 2008). Policy for agricultural land should consider the implications of contamination with this type of pathogen, especially where intensification is planned.

Pathogens present in agricultural waste can persist in the environment, particularly in the soil and in watercourses. In some cases it is possible to reduce pathogen prevalence in the farm animal. But often the organisms concerned do not cause disease in livestock, while the increased use of antibiotics on farms is to be strongly discouraged because it increases the risk of drug resistance (Gold and Moellering, 1996). Managing farm waste through the optimal design of slurry storage and preventing farm animals from contact with watercourses reduces the probability of pathogens moving off the farm. A major potential source of contamination is the application of slurry as manure (Chadwick et al., 2008). The greatest risks occur if slurry application is followed by a storm. This applies especially if the ground is waterlogged, increasing surface flow, or is very dry and cracked, increasing sub-surface flow and leaching. There has been considerable research on these risks, and best practice advice for farmers is available (Defra, 2001). On a landscape scale, the creation of non-agricultural buffer areas along rivers and streams can reduce the transport of pathogens. It can also reduce erosion and promote biodiversity (Marshall and Moonen, 2002). Increasingly sophisticated catchment-scale hydrological models can also be valuable in predicting contamination, especially following storms (Haydon and Deletic, 2006; Dorner et al., 2006). Finally, human exposure can be reduced by educating high-risk groups, for example hill walkers and surfers, about contamination routes, and by increased surveillance (Philipp et al., 1992). Recent advances in molecular techniques raise the prospect of continuous, real-time sampling of sea water off bathing beaches, with automatic alerts in the event of contamination by specific pathogens (Fong et al., 2005).

Many of the issues concerning gut pathogen contamination have important social science aspects. The extent of livestock farming and its intensification, and the degree to which slurry is used as a fertiliser, have complex economic and social drivers. The take-up of best practice for manure and slurry management requires careful design of incentives for the farmer. Finally, the financial consequences of contamination events may be disproportionate to the number of people infected. The infection in 2000 of 18 scouts camping in Aberdeenshire by *E. coli* O157 severely damaged the tourism reputation of the whole region (Howie et al., 2003).

Land use and livestock disease

In recent years three livestock diseases, FMD, HPAI and BSE,[1] have caused massive economic, social and animal-welfare problems in the UK. In addition to these high profile infections, several other infectious diseases are of potential interest with regard to UK livestock and animals (Defra (a)). Trade in animals, trade in food, contact between livestock and wildlife and intensification of farming practices have all been implicated in outbreaks of livestock infections and in the ongoing transmission of endemic infections. Since each of these factors is intimately connected with the way land is used, land use policy is an important determinant of the risk of transmission of livestock infections.

The FMD outbreak of 2001 illustrated the role of trade in live animals in spreading infection. Foot-and-mouth is a highly infectious disease of sheep and cattle. The outbreak of 2001 was nationwide, and the structure of the livestock industry is thought to have played a significant role in its spread (Food and Rural Affairs report, 2001). The outbreak coincided with a time of year at which there was a particularly high level of sheep trading and livestock movements. As a result, the virus had spread widely across the country before cases were first recognised. The volume of animal movement was due partly to the distribution of livestock markets. Sheep and cattle markets had become fewer, but larger, than at any time in the past (Defra (b), Roberts, 2001). Overall the outbreak cost over £9 billion (Defra/DCMS, 2002), including indirect damage to the non-agricultural rural economy including tourism.

HPAI (Highly Pathogenic Avian Influenza) is a disease of birds that can also be zoonotic. Poultry are highly susceptible to HPAI and the highly virulent subtype H5N1 is now well-established in poultry in south-east Asia. In the UK to date, September 2009, there have been three outbreaks of HPAI in poultry farms (Defra (c)). We do not have a definite explanation for the source of infection in any of these outbreaks, but imported turkey meat and possible contact with waterfowl are strongly suspected (Mayor, 2007). Biosecurity measures have ensured that none of these outbreaks have caused human infection. However, land use management for poultry farms is essential in ensuring that avian influenza in UK farms does not reach endemic levels as in South-East Asia. Important factors include limiting exposure to wild birds, particularly through shared watercourses; maintaining biosecurity to limit opportunities for the spread of infection; and production management to reduce transmission (thus lowering R_0) within flocks (Bouma et al., 2009).

Other livestock diseases are also derived from wildlife, and the risk of cross-infection is influenced by the structure of the rural environment. Bovine tuberculosis is an important disease of cattle and in the UK has a natural reservoir in the wild badger population.

[1] Foot-and-mouth disease; highly pathogenic avian influenza and bovine spongiform encephalopathy.

Box 3: Bovine tuberculosis—badgers and cattle

Bovine tuberculosis (bovine TB) is a disease of cattle caused by the bacterium *Mycobacterium bovis*. Prior to the introduction of milk pasteurisation, *M. bovis* was a significant human disease but today its prime importance is as a disease of cattle. Bovine TB has been eliminated or is rare in many dairy and cattle-raising areas where there are no major wildlife reservoirs of the disease. However, bovine TB continues to be a problem in the British Isles, parts of North America and New Zealand where badgers, deer and possums, respectively, are important hosts for *M. bovis* (Krebs, 1997).

There is a general consensus that radical reductions in badger numbers (or their extermination) would reduce the incidence of bovine TB in the British Isles (Bourne, 2005). There is no consensus about whether this scale of culling is desirable, nor about whether the level of culling that realistically could be achieved in different parts of the UK would make a significant difference. Past land use policy in the west of England, a hotspot for bovine TB, has resulted in a patchwork of agricultural, forestry, recreational and residential areas. This pattern, combined with laws protecting wild animals, has led to a sustained increase in badger numbers (Bourne, 2005). Moreover, much land is owned by non-farmers who frequently refuse permission for badger culling on their land, in contrast to Ireland where the predominantly agricultural land-owning community seldom object to badger culling. Thus the environmental and socio-political consequences of land use change have affected disease prevalence and control options.

Current government expenditure on bovine TB surveillance, compensation and research is about £80 million per year. In July 2008, the government announced that it would not pursue badger culling as a control policy in England. Should future land use policy in affected regions be driven by efforts to reduce cow-badger contacts, or to offer farmers inducements and incentives to diversify out of dairy and beef?

There has been a long-running controversy about whether badgers should be culled to protect cattle, and whether there are alternative means of reducing badger-cattle contact (see Box 3).

BSE (bovine spongiform encephalopathy) was first confirmed in cattle in 1986 and by the end of 2008 there had been over 180,000 cases in the United Kingdom (Defra (d)). There were only 37 cases in 2008 and the number of cases per year is now halving each year, so the epidemic in cattle is nearing its end. Although the initial origin of infection remains obscure (Horn et al., 2001) it is clear that it was spread between cattle through the use of ruminant-derived meat and bone meal in animal feed. This feed was used to supplement the protein intake – particularly of dairy cows – as part of the effort to increase milk yields. BSE is thus an example of a livestock infectious disease that became a problem because of intensive farming practices.

The relationship between intensification and disease risk is not straightforward. Pigs are amongst the most intensively farmed animals in the UK, but the industry adopts strict 'vertical integration' practices to minimise potential infectious contacts between animals (Ribbens et al., 2008). Similarly, the poultry industry implements very strict biosecurity measures in its intensive rearing sheds. Being indoors these have the added advantage of effectively eliminating the possibility of direct contact with infectious wild birds (Defra (e)). So land use policy that demands more intensive livestock rearing does not necessarily increase the risk of disease, provided excellent biosecurity measures are put in place. But if these fail and infections become established, disease in intensive systems can cause massive losses, both economic and in animal lives.

Land use and diseases of wildlife

Disease in wild animals is often a natural part of community ecology and is not automatically to be avoided. Indeed, one could argue that infectious disease organisms that pose no health or economic risk to man are as deserving of protection as charismatic birds and mammals. Yet today, the vast majority of wild animals live in environments that are highly altered by man, and land use decisions may threaten the viability of populations through increased exposure to disease in ways that would not happen in unaltered landscapes (Smith et al., 2009). Although we should be happy to let infectious disease play its long-established role in regulating wildlife populations (Hudson et al., 2006), there is more urgency when infections threaten the extinction of an endangered species, particularly where human activity has been a contributing factor (Randall et al., 2006).

The UK, in common with most other countries, draws up 'Red Lists' of endangered and potentially endangered species (IUCN, 2008). Biodiversity Action Plans are then devised to determine policy to prevent their decline and extinction (UK BAP Website). Such plans often include the setting aside of protected areas, and as resources are always limited there is usually a trade-off between protecting *Single Large Or Several Small* areas, a dilemma so well-studied that it is referred to by the acronym SLOSS. There is a large conservation biology literature on SLOSS (Simberloff, 1988). It emphasises the balance between sustaining large populations which are less likely to become inbred or go extinct through chance and the risk of a catastrophe wiping out this single population. The risk of major disease has been considered in the SLOSS literature (Hudson et al., 2002), where the aim is to prevent disease reducing a population to so low a level that the risk of extinction is high. Such considerations tend to argue for multiple populations that reduce the spread of diseases, for the same reason that reducing the contact network is helpful for limiting disease in man and farm animals. In practice, the decisions made by conservation managers are nearly always highly constrained by the availability of land and resources. But although simple SLOSS trade-offs seldom arise, the underlying ideas may still be useful in distinguishing between actual planning options.

A second way that land use may influence wildlife disease is through increased exposure to pathogens. Where the same pathogen affects both a domesticated animal and a wild animal, land use changes that increase the density of the former may reduce populations of the latter. Box 4 describes how increases in pheasant numbers negatively affect wild partridges with which they share a gut parasite. The increase in aquaculture (fish farming), especially if managed poorly, can lead to huge increases in the density of fish parasites and pathogens that negatively affect free-ranging fish (Ford and Myers, 2008; Krkosek et al., 2007). In the case of salmon, where both farmed fish and wild-caught fish have significant economic value, though to different stakeholder groups, there are conflicts that are hard to resolve. The issue of cattle contracting bovine TB from badgers (see Box 3) can be inverted so that it is thought of as a conservation policy issue where a wild animal is harmed by agriculture. Similarly in parts of South Africa, lions are endangered by bovine TB caught from buffalo that in turn were infected by cattle (De Vos et al., 2001). Finally gorilla populations have suffered from diseases caught from human tourists (Butynski and Kalina, 1998). Though the precise policy implications vary with the species concerned, a common thread is the need to separate in space wild and domesticated animals that share the same disease.

Emergent diseases are also a threat to wild animals. Two highly virulent diseases that attack a broad range of amphibians, chytridiomycosis and to a lesser extent ranavirus, have been associated with significant amphibian population declines around the world

Box 4: Interactions between gamebird populations

Changes in land use can affect the viability of wild species of animals. For example, the grey partridge (*Perdix perdix*) is a native UK bird that was once very common but has declined steadily in recent decades and is now classified as a threatened, "Red List" species. There are several reasons for the decline, in particular the loss of nesting sites brought about by agricultural intensification. But another factor, which is thought to have played a subtle but important role, is increased infection by parasites shared with an introduced semi-wild gamebird, the ring-necked pheasant (*Phasianus colchicus*) (Tompkins et al., 2001).

Every year millions of pheasants raised in captivity are released to be shot as game. Both pheasants and grey partridges are susceptible to infection by the nematode *Heterakis gallinarum*. While this parasite causes only limited symptoms in pheasants, it causes much more harmful effects in grey partridges, such as reduced food consumption and slowed weight gain (Tompkins et al., 2001). Moreover, nematode infection in the grey partridge population is sustained only through contact with pheasants. Infection is easily maintained in the pheasant population, while partridges transmit the infection only poorly. Harmful effects between species mediated by parasites are what ecologists call an "indirect effect" and may be responsible for structuring some natural communities of animals. The issue here is that the indirect effect is greatly amplified by man. Recommended land use practices in support of grey partridge conservation include the provision of nesting cover at field boundaries (UK BAP Species Action Plan). Consideration of the shared disease suggests that partridge conservation would be helped by land use policies that reduced pheasant rearing near the remaining strongholds of the bird (Tompkins et al., 2000).

(Stuart et al., 2004). It is suspected that the spread of both diseases has been at least partly due to man. In the UK, chytridiomycosis was first detected in wild frogs (*Rana temporis*) in 2005 (Cunningham et al., 2005). Several episodes of frog die-offs have heightened concern about UK amphibian populations. Where suitable breeding sites are available frogs (and other amphibians) survive well in suburban areas and simple changes in land use that make gardens more suitable for amphibians can make a major contribution to population resilience in the face of disease.

Looking ahead

In this section we look ahead to future disease challenges that may influence land use policy. In particular we explore the connection between land use, disease, and socioeconomic and environmental change. We also highlight areas where more research is needed.

Whether climate change will affect disease risk, particularly vector-borne infections, is a difficult and contentious question. Insect vectors depend sensitively on environmental conditions, including temperature, humidity and the availability of breeding sites that are often dependent on rainfall. Changes in climate may thus affect the range of vectors and possible vector-borne disease (Githeko et al., 2000). There have been concerns that warming of the UK could precipitate a return of malaria (Department of Health, 2002). However, it is not clear whether the effects of climate change will be significant in comparison with other drivers such as human movement, land use and trade (Tatem et al., 2006; Randolph, 2009). As we have seen, it appears that globalisation and the trade in used tyres have played a more significant role in the spread of Chikungunya virus than climate change (Knudsen, 1995).

The effects of climate change that are likely to be experienced first are those involving extreme events, in particular heavy rainfall. Rainstorms can be a major factor in spreading human pathogens in animal slurry through the environment. However, the consequences can be complex. Though such events can mobilise slurry, it is often delivered into a high-energy environment such as a fast-flowing river. It may be transported rapidly away from the source, making it difficult to attribute, and away from centres of population. But it may be discharged to the sea and affect swimmers and surfers. Coping with storms and flooding is an important goal of programmes exploring adaptation to climate change, and reducing pathogen spread should be included in this planning process.

Globalisation has increased the flows of people and trade across international borders and most economic scenarios assume this trend will continue. This makes it likely that humans and animals in the UK will be more frequently exposed to diseases with reservoirs abroad, including some flu strains and foot-and-mouth, and perhaps to novel emerging infections. Increasing globalisation seems to us to argue for an increased emphasis on disease control in land use policy formulation.

As other studies for this Foresight project have demonstrated, there is likely to be increasing competition for land in the UK, driven by higher population densities (Office for National Statistics, 2008) and novel requirements for land such as for biofuel production (Potocnik, 2007). This means increasing pressure on agricultural land at a time when global increases in population and consumption of meat and dairy products are all increasing the demand for food (Schmidhuber and Tubiello, 2007). To meet such pressures we shall almost certainly need to produce more food from the same or even less acreage, and to do this in a way that harms the environment the least. In other words we need sustainable intensification (Pretty, 1997).

A sustainable intensification agenda will have several ramifications for land use and disease. Currently a sizeable fraction of global agricultural productivity is lost to pest and diseases. Reducing the burden of disease, in part by implementing livestock disease management interventions at source such as vaccination and reductions in animal movements, will be important in making the best and most efficient use of land already in agriculture. Intensification in livestock also means greater densities of animals, and hence for the reasons discussed in early sections, greater risks of disease. Some of these risks may be mitigated by biosecurity measures, and successful intensification in future will require advanced disease risk reduction strategies.

An increased uptake of herd vaccination could play a role in the mitigation of infection risk. One problem with this approach is that at present, for a few diseases, vaccinated animals are indistinguishable from those that have undergone infection without symptoms or that have since recovered from the specific disease. In dealing with certain infections, such as FMD, previously infected animals are routinely destroyed, and so any widespread vaccination programme for such diseases will need to be able to identify those animals that have already received vaccination (Mackay et al., 2004). Doel et al. (1994) also raised the question of whether FMD vaccination stops infectiousness, or merely symptomatic disease. Nonetheless, for many diseases vaccination is a key control measure in minimising the transmission and/or severity of disease, such as its use in the control of Salmonella (Mastroeni et al., 2001).

Finally, sustainable intensification implies optimal recycling of nutrients and waste products. This involves disease risk. If more animal slurry is used as fertiliser, a careful assessment of pathogen movement in the environment and potential human exposure will become increasingly important. The BSE epidemic in the UK shows the dangers that can arise when animal material is recycled without proper disease risk management.

Future research questions

All science reviews call for more research and we shall not be an exception. Throughout this paper we have stressed the importance of understanding the network of contact between individuals that determines how directly transmitted diseases spread. For human diseases, characterising contact patterns in different environments is an interdisciplinary area of research involving both the social and the natural sciences. Current techniques for surveying human contacts include diary-based methods in a peer group (Read et al., 2008) while new methods involving GIS and mobile phone positions are increasingly becoming feasible (Rogers and Randolph, 2003; Onnela et al., 2007). Future research, on a larger scale, could explore how different urban and rural environments shape human contact networks. An important question is also how these contacts might change during the course of an epidemic, for instance as the public reacts to the spread of a life-threatening infection (Smith, 2006). The patterns of these connections are also important. Certain individuals or localities may act as infection 'hubs' and particular transmission routes can be especially significant. A better understanding of this network structure could become central for targeted control of infection. Moreover, the ways in which contact networks scale as a population grows have not yet been characterised. Progress in this area of research could help to inform urban planning policy. A better understanding of the transport through the environment of zoonotic pathogens that are potentially harmful to man would also help develop rural land use policy.

The epidemiology of wildlife diseases is still relatively poorly understood, though substantial progress has been made in the past two decades (Daszak et al., 2000; Stallknecht, 2007). The increasing ease with which high-throughput molecular tools can be used to assess and monitor disease will be of major importance in this field. Technological advances in this area will also help explore the contact network through which disease spreads. An example is the Tasmanian Devil, a rare Australian marsupial whose survival has recently been threatened by a contagious facial cancer (McCallum and Jones, 2006). Devils are particularly susceptible to such an unusual disease because they are highly irascible and fights frequently break out at which transmission occurs. Understanding the contact network is critical for reserve design and disease management, and sophisticated radio telemetry is being used to assess it (McCallum et al., 2008). Similar techniques could be used to explore bovine TB and badger interactions.

Finally, we stress the need for continuing disease surveillance and emergency planning (Health Protection Agency, 2008), and for horizon-scanning exercises that seek to determine known risks (diseases abroad that have not yet entered the UK) as well as the unknown risks from future emergent diseases. These are inherently much harder to assess.

Conclusions for land use policy

Of all the types of infectious diseases discussed here, which will be most relevant for land use policy? Such relevance is determined by three factors: the likelihood or regularity of the disease occurring; the impact of the disease; and the degree to which land use policy can influence the occurrence and impact of infection. Here we explore how each of these factors relates to diseases of wildlife, humans and livestock, and provide our assessment (with its associated uncertainty) of their overall importance for land use policy.

Consider wildlife diseases first. Wherever populations of wild animals are closely examined, diseases are discovered. They frequently have significant effects on their hosts' population dynamics (Daszak et al., 2000). Though the study of wildlife diseases is still in its relative infancy, and most systems require further research, it is safe to say that they are common and regular. Assessing their societal impact is harder, because disease is part of the functioning of natural ecosystems. But disease may threaten the survival of some endangered species. This means that the UK is obliged to mitigate such diseases to fulfil its national and international commitments for biodiversity preservation. Policy levers to influence wildlife disease are not extensive. They chiefly revolve around nature reserve design and limiting cross-infection from domestic animals that share the same diseases. We see wildlife diseases as a low to medium priority consideration for land use policy, but say this with relatively high uncertainty due to our current poor understanding of wildlife disease epidemiology.

Turning to humans, infectious diseases such as the common cold, norovirus and seasonal influenza occur frequently and regularly. In addition, there are rare and unpredictable disease episodes, in particular those caused by pandemic influenza. There are also novel emergent infections that are even harder to predict, such as HIV, which became established, and SARS, which did not. The impact of the most common diseases is chiefly measured by the economic costs of people taking time off work, as well as some reduction in the lifespan of the sick and elderly (Molinari et al., 2007). The impact of the rarer and unpredictable diseases can be much greater and even catastrophic (Nguyen-Van-Tam and Hampson, 2003). Land use policy can influence human disease risk but the major risk factors such as population growth, urbanisation and globalisation are probably too strong and pervasive to be addressed by land use policy. Effective urban planning may have a role in mitigating some risks of infectious disease, while rural land use policy can help reduce the risk of humans contracting zoonoses. We thus see human diseases as a relatively low priority for land use policy, and we attach medium uncertainty to this conclusion largely because of the potential huge impact of a future pandemic. As we write, the full consequences of the current H1N1 "swine flu" pandemic are far from certain.

Finally we turn to the diseases of livestock. These are common and are likely to become more frequent if food security concerns lead to greater agricultural intensification. Even extensive systems can be at risk where transmission from wildlife is significant. Nearly all livestock diseases cause immediate economic harm to farmers. For the worst diseases, such as foot-and-mouth, there are serious consequences for the individual farmer whose herd is destroyed, for the local economy through reductions in tourism, and for the general economy through restrictions on international trade. We argue that land use and agricultural policy can have a major role in reducing the risks and consequences of these diseases, chiefly by modifying exposure to risk and managing the contact network that determines the spread of the disease. We view the control of livestock disease as a high priority for land use policy, and associate a low level of uncertainty to this assessment.

Finally we note that though it is outside the brief of this review, many of the same issues that affect livestock disease may also influence diseases of crops and forestry. Such considerations should also influence land use policy.

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