Management of Disease in Wild Mammals

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Editors

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Preface

In recent years nobody could have failed to notice the frequent and often sensationalist media headlines warning of the latest global disease threat to humankind. But behind all the hyperbole lie real challenges related to dealing with the increasing incidence of emerging zoonotic disease events, the majority of which are thought to originate in wildlife (Jones et al. 2008). There are also many important diseases of domestic livestock which also occur in wildlife (e.g. foot and mouth disease and classical swine fever in wild boar, bovine tuberculosis in deer, badgers or possums), some of which can have a devastating impact on the farming industry, the wider rural economy and ultimately the public purse. But we should also not forget that wildlife diseases may have serious implications for the conservation of biodiversity. For some of the rarest, most endangered species (such as the Ethiopian wolf) disease may pose the greatest threat to their survival. If we are to avoid or reduce these impacts then we must improve our ability to detect and manage the risks associated with disease in wildlife populations. This is a challenge that will require expertise from many different disciplines: veterinary, ecological, medical, economic, political and zoological. In such an interdisciplinary field it is difficult to stay up to date with contemporary ideas and with techniques that may be rapidly evolving. We hope that in some small way this book contributes to informing people from a range of disciplines on our current state of knowledge and potential future directions in the management of disease in wildlife.

Largely because of our personal interests and expertise we have focused in this book on disease in wild mammals, although much that is discussed will be relevant to other wild fauna. Our aim has been to present and discuss the main issues related to disease management in wild mammals, and in doing so we have inevitably drawn upon the opinions of experts in a range of fields. We have attempted to be as inclusive as possible, in the knowledge that this is a topic at the interface between several scientific disciplines. We also acknowledge the important role that scientific knowledge plays in underpinning policy, and have therefore produced a text that is hopefully also accessible to those without a scientific training, but who are nevertheless important players in the development and implementation of disease management plans.

The editors have worked in the field of wildlife diseases for many years and whilst we maintain interests in other fields we continue to have close links with

each other, particularly in the area of bovine tuberculosis in wildlife. We have seen at first hand how opinions change over time (albeit slowly in some cases), and how this process depends on the views and foresight of a wide diversity of experts. We have thus sought to include the opinions of many additional experts in different fields and would formally like to acknowledge their invaluable contributions. The co-authors not only gave generously of their time and expertise in helping to write individual chapters, but in many cases also improved the book by commenting on and correcting errors throughout the text. In addition we would like to thank Fred Landeg, Hamish McCallum, Menna Jones, Pete Robertson and Robbie McDonald for reviewing parts of the text and giving us additional perspectives. GCS and RJD would also like to thank Chris Cheeseman for his support and enthusiasm over the years. Many of the authors are involved in the Wildlife Disease Association and in particular with the European Section and we wish to collectively acknowledge the important contributions this organisation has made to promoting scientific endeavour in this field.

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Chapter 1
The Science of Wildlife Disease Management

Richard J. Delahay, Graham C. Smith, and Michael R. Hutchings

1.1 What is Disease?

In its widest sense disease can be regarded as any impairment of normal functions. However, for the purposes of this book we will mostly restrict our discussion to infectious diseases, the agents of which are often described as parasites or pathogens. For convenience, these organisms are often split into two categories that reflect their broad characteristics, and their relative size. The macroparasites are multi-cellular organisms that live in or on the host, such as helminths and arthropods, while microparasites include viruses, bacteria, fungi and protozoa. The main functional differences between the two relate to their generation times, with microparasites exhibiting relatively higher within-host reproductive rates and shorter generation times than macroparasites. As a result microparasites are frequently associated with acute disease, although they can induce long-lived immunity to re-infection in recovered hosts. Macroparasites by contrast are more likely to produce chronic infections often characterised by short-lived immunity in heavily infected hosts, and re-infection. Macroparasites may also have distinct life stages that can survive outside the host (e.g. eggs or larvae) and sometimes require other host species to complete their life cycle. Two important groups of pathogens fall outside this classification: rogue proteins (prions) implicated in transmissible spongiform encephalopathies (TSEs) and infectious cancers, of which Tasmanian devil facial tumour disease is a well known example. However, in broad respects these are most usefully considered as microparasites, often producing acute clinical signs without host immunity.

Disease can affect individual hosts by reducing growth rates or fecundity, increasing metabolic requirements, changing patterns of behaviour and ultimately may cause death. Sub-lethal effects of pathogens may also enhance mortality rates by for example, increasing the susceptibility of the infected host to predation. However,

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the intimate relationships between hosts and parasites have in many instances evolved over time into subtle and potentially complex interactions, such that infection does not in itself necessarily lead to disease. Many parasites have little detrimental effect on their hosts for most of the time, only causing pathological damage if this delicate balance is upset, for example when the parasites become too numerous or when the immunological capability of the host is impaired. This balance could be influenced by many factors including nutrition, concomitant infections and a variety of physiological stressors.

Parasites are natural components of ecosystems. They influence the structure of ecological communities (Wood et al. 2007) and are important agents of evolutionary change (Clayton and Moore 1997; Little 2002). Hosts and their parasites are locked in an evolutionary arms race, an endless game of 'hide and seek', which finds its ultimate expression in the complex immune systems of mammals. So fundamental is the role of parasitism in the development of biological systems that the imperative to avoid disease may have been an important driver for the evolution of sexual reproduction, which provides a means for recombination of genetic material and the inheritance of protective genes.

Disease is a ubiquitous characteristic of ecosystems. In humans (the most comprehensively studied mammal) over 1,400 diseases have been identified, in our livestock we know of over 600 and in domestic carnivores nearly 400 have been recorded (Cleaveland et al. 2001). Over 60% of human diseases are zoonotic, and for those considered to be of emerging importance, the figure rises to 75% (Taylor et al. 2001). By inference alone there are likely to be many thousands of diseases affecting the 5,400 or so mammal species in the world. Nevertheless, despite the clear implication that they are likely to play an important role in the epidemiology of some diseases of importance to human health and livestock, information on the pathogens of wild mammals is relatively poor.

1.2 The Significance of Wildlife Diseases

There is no doubt that recent years have seen a growing recognition of the potential importance of wild mammals in the epidemiology of diseases that impact on global human health, agriculture and biodiversity. In terms of public health, this has been manifest in high profile reports of hanta virus, Lyme disease and SARS-associated coronavirus in humans, and their links to wild mammals. In some countries, wild mammals are implicated in the persistence of bovine tuberculosis and brucellosis infection in cattle, which have impacted severely on the welfare and productivity of domestic animals and imposed high costs on stakeholders. Some such diseases are the subject of eradication programmes as their potential impact on human activities is so acute. But wildlife populations themselves may also be threatened by disease, particularly if they are already fragmented, and vulnerable to extinction from stochastic events. This is illustrated by examples such as the impact of rabies on populations of the endangered African wild dog (*Lycaon pictus*) and Ethiopian

wolf (*Canis simensis*), and of facial tumour disease in Tasmanian devils (*Sarcophilus harrisii)*.

In this book we focus on the management of disease in wild mammals, although many of the issues and approaches discussed here will apply to other wildlife. Wild mammals are of particular interest because they share so many common pathogens with domestic livestock and humans, and consequently play a prominent role in the dynamics of diseases of public health and agricultural concern. Most known zoonotic diseases infect carnivores, livestock and commensal rodents, probably as a result of the historical and evolutionary associations with humans. Mammals are also of particular value as sensitive barometers of ecosystem health, sitting as they do at, or near the top of, trophic food chains. For this reason they have often served as key species for conservation initiatives, under the, often unstated, assumption that their protection will safeguard the habitats that they and many other species inhabit.

The growing importance of diseases in wild mammals to a range of human activities has occurred against the background of a rapidly changing world, in which the interface between human and wildlife populations has been profoundly modified by urbanisation, agricultural intensification, climate change and habitat degradation. Some wild mammals have proven extremely adaptable in the face of anthropogenic changes to the environment. The most adaptive species tend to be those with generalist diets and opportunistic habits. Some have increased in abundance and distribution, as they have become habituated to agricultural and urban environments. Examples include red foxes (*Vulpes vulpes*) and Eurasian badgers (*Meles meles*) in the UK, both of which have successfully adapted to life in highly urbanised environments. Furthermore, the high densities of badgers observed in some rural areas of the UK are in no small part due to the abundance of food afforded them by the modern pastoral farming landscape. In several instances the direct management of wild mammals for hunting or game farming has resulted in localised concentrations of unsustainably high density. Wild boar (*Sus scrofa*) and red deer (*Cervus elaphus*) in parts of Central and Western Europe, and white-tailed deer (*Odocoileus virginianus*) in some regions of the North-Eastern USA are notable examples. But the wild mammals with which we have the longest standing and most intimate relationships are undoubtedly the commensal rodents with whom we share our homes and farmland across the globe. Within modified environments, these adaptive species may frequently live in close proximity to humans and our domesticated animals, thus enhancing opportunities for inter-specific transmission of pathogens. For most wild mammals however, human activities have had a devastating impact, largely through the destruction and degradation of their habitat, but also through direct exploitation and pollution. The result is that many species of wild mammal survive in diminished and fragmented populations that are vulnerable to the effects of disease (Chapter 11). Out of 5,416 species of wild mammal, 1,094 were regarded as 'threatened' (i.e. vulnerable, endangered or critically endangered) with extinction by the International Union for Conservation of Nature (IUCN 2007).

In recent decades there has been an unprecedented increase in the global transport of people, animals and animal-derived products. International air travel now

provides the opportunities for a disease that would once have taken many months or years to traverse a single continent, to be carried to the far corners of the globe within a matter of hours. Live wild animals are translocated in the interests of the pet trade, game management and conservation, and their products are distributed in the form of, often illegal, bushmeat, 'medicines', trophies and other merchandise. The associated risks of introducing new diseases to previously isolated and naïve populations can have potentially catastrophic consequences. Nearly 38 million live wild vertebrates were legally imported into the USA between 2000 and 2004 (Marano et al. 2007), including 23,000 mammals and at least 263 non-native species. One widely reported consequence of these imports was the 2003 outbreak of the zoonotic monkeypox virus which was initiated by infection in exotic African rodents imported for the pet trade (Guarner et al. 2004). Such events emphasise the need to develop contingency plans to ensure some level of preparedness to deal with disease introductions that could establish in endemic wildlife populations (Chapter 9).

The perpetual movement of people, animals and products around the world is not the only anthropogenic process that creates opportunities for enhanced disease transmission. Environmental degradation in a wide variety of guises may also be a driving factor in the emergence of wildlife diseases. Airborne pollution, habitat fragmentation and the eutrophication of aquatic ecosystems have for example all been linked to disease outbreaks in wildlife (Dobson and Foufopoulos 2001). But, the most pervasive and potentially damaging environmental impact to arise from human activity is undoubtedly global climate change. The consequences for global ecosystems will clearly have significant implications for the ecology of wild mammals and their pathogens (Epstein 2001), as well as presenting major challenges to human activities. Changes in global weather patterns are likely to be accompanied by an increasing tendency for the emergence (and re-emergence) of pathogens and their vectors in new geographic areas and in novel hosts. The development of methods to predict such events and of co-ordinated systems to provide appropriate responses, are major challenges for the international community.

1.3 Managing Disease in Wild Mammals

It is important to consider the question of when disease in a wildlife population requires management intervention. After all, diseases are natural components of ecosystems, although it is often a moot point as to whether a particular pathogen would have existed in a wild population in the absence of its purported introduction by humans or livestock. Human modification of the environment has been so substantial and widespread that the question often arises as to what constitutes a natural ecosystem and, perhaps more importantly, what we can consider to be a natural disease event. The question of when and when not to manage, essentially rests on the extent to which the disease endangers human health, wealth, welfare or conservation aspirations, and the likelihood that intervention will have a beneficial

effect. Opinions on the point at which a line is crossed and management becomes necessary, may vary widely between stakeholders of differing perspectives, and the search for 'common ground' is a continuing challenge for policy makers and politicians. However, even when a problem is identified as sufficient to warrant management, this may not necessarily mean that intervention is best directed at the wildlife population or the pathogen. In many cases changes to other components of the system (e.g. human behaviour) may be more effective. This may be particularly true when such approaches are targeted at the more tractable elements of the system (e.g. livestock husbandry), which can be managed using the existing socio-economic and legislative framework.

Once the decision to intervene has been reached then the objective of management will need to be determined. This may be prevention or control of disease, or even local or global eradication of the pathogen. The appropriate approach will depend on the characteristics of the problem and in particular on the correct identification of reservoirs of infection (see below). Inevitably, prevention and control are generally more easily achieved than eradication, not least because the latter requires the accurate identification of all reservoirs of infection. The appropriate target of disease management may be the pathogen itself (Chapter 6), one or more host populations (Chapter 7), or some element of the environment that influences transmission (Chapter 8). In this book we will discuss each in turn, although in practice a combination of approaches may be most successful.

Despite the clear requirement to develop effective means of dealing with wildlife disease issues, advances in practical management have lagged far behind the development of disease ecology theory. In particular, managers have been slow to respond to the need to understand and accommodate the ecological complexities of wild mammal populations in intervention plans. And yet, understanding wildlife disease problems is invariably as much an ecological as it is a veterinary challenge. This is elegantly illustrated by an example from the UK where in 1997 the Government convened an Independent Scientific Group (ISG) of experts in veterinary science, ecology, epidemiology, statistics and economics, to investigate the effects of badger culling on bovine tuberculosis in cattle. The results of the large scale field experiment and related research they initiated, showed that attempts to reduce disease in cattle by culling badgers caused changes in the behaviour of the wild host that under certain circumstances were counter-productive for disease control (Independent Scientific Group 2007). Their findings illustrate the fundamental importance of understanding host ecology and social behaviour (Chapter 2) for the development of disease control strategies, and the clear need to identify, characterise and quantify the key ecological processes that drive disease transmission and persistence (Chapter 3) in wildlife populations. Hence we need to look critically at existing assumptions of disease control and management, particularly where they are underpinned by experience in dealing with disease in domestic animals. The development of successful approaches to the management of disease in wild populations will require careful consideration of the entire host community, of the economic dimensions, and of the practical challenges of successfully implementing any intervention. Where management of disease involving wildlife was once the almost exclusive domain of veterinarians, it is now

increasingly recognised that it requires a multi-disciplinary approach involving ecologists, epidemiologists, experts in public health, mathematical modellers, geographic information specialists, statisticians and economists. Such an approach is essential if we are to further our understanding of the dynamics of disease in wildlife and to develop sustainable strategies for their management.

The key to developing effective tools for the management of disease involving wildlife is a sufficient understanding of the conditions required for the persistence of pathogens. Many important diseases infect multiple hosts, some of which will constitute persistent sources of infection for other species, whilst others will not. Unfortunately, many past attempts to manage disease in wildlife populations have failed to recognise this distinction and have instead been rooted in a poor or even misguided understanding of the host community and the likely impact of intervention on disease dynamics. Central to our understanding of any disease system is the concept of the reservoir host. An over-abundance of definitions of disease reservoirs can be found in the literature, each emphasising different aspects, and together leading to no small amount of confusion. A clearer conceptual framework may be achieved by taking an ecological community-based approach which defines a reservoir as "*one or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained, and from which infection is transmitted to the defined target population*" (Haydon et al. 2002b). Past attempts to manage disease involving wildlife have all too often been aimed at 'suspected' reservoirs with little hard evidence that they represented the most important source of infection. That said, it can be difficult to unequivocally identify a reservoir host population. Although correlative and risk-based associations can provide strong circumstantial evidence, only interventions that can isolate target populations can produce experimental evidence, and these are rarely possible.

Effective management of wildlife diseases needs to be based on sound science and developed on the basis of the objective review of previous evidence. This evidence-based approach has led to a radical change in the way human medicine is influenced by previous experience. Systematic review of the effectiveness of previous practices is now widely accepted as standard practice in public health and has been advocated for conservation management (Sutherland et al. 2004). There is a clear need to develop and maintain systems to support evidence-based practice in wildlife disease management. This implies a fundamental change from what has been common practice in the past, such that in the future the outcomes of disease management interventions should be systematically monitored, collated and made available to others. Inevitably however, even with unfettered access to evidence from past experiences of dealing with disease in wildlife, many unanswered questions regarding the potential impact of management interventions will remain. Some important areas of data shortfall may be addressed through systematic scientific investigations and experimentation, although in some cases this may be practically difficult, prohibitively expensive, or there may be insufficient time given the magnitude of the problem. As a consequence, the reality is that we will often be required to make decisions in the face of substantial uncertainty. In such

circumstances mathematical modelling can provide a powerful tool, both for increasing our understanding and for generating predictions of the likely outcome of interventions (Chapter 4). Mathematical simulations provide the opportunity to play out various scenarios under different conditions and to incorporate the known uncertainties of the system under investigation. If the modelled outcome of management decisions is robust to different underlying assumptions, then we can be more confident of its utility. If management decisions rely heavily on assumptions, then we have to make a decision based on the relative risk, and cost of each potential outcome. With sufficient understanding of the underlying assumptions, the limitations and levels of uncertainty associated with outputs, then the results of mathematical models of disease dynamics and management interventions can make valuable contributions to the decision-making process.

Modelling can therefore be used to help define interventions that are likely to give a positive benefit, in terms of reducing disease prevalence. However, the most effective techniques to reduce the burden of disease will likely require the most effort, and so be more costly. As resources are always limited, a balance needs to be struck between desired outcomes and their financial costs. This is where the application of economic analyses can help (Chapter 5). The costs and benefits of each potential strategy can be compared in terms of cost-effectiveness or the cost–benefit ratio, and so help to identify an 'optimum' strategy.

In the world of commerce it is widely recognised that you cannot manage what you do not measure. This is equally relevant to disease management. Unless we are able to identify changes in disease occurrence in wildlife populations through monitoring and surveillance (Chapter 10), we will not be able to identify situations that require action, and if we cannot monitor the impact of interventions, then we will not know whether they are working. This seems obvious enough, but in practice surveillance for diseases of wildlife is poorly developed in most countries. Also, past endeavours to control disease in wildlife have often been characterised by a failure to adequately monitor progress, describe the baseline pre-intervention situation against which to measure progress, or indeed to clearly state the objectives of the intervention. An appropriate programme of monitoring should therefore always accompany any wildlife disease management intervention, and should be designed so as to assess its effectiveness in achieving the stated objectives. Further development of methods for the surveillance and monitoring of pathogens and hosts is intrinsic to the future successful management of diseases in wildlife.

1.4 Conclusions

Management of disease in wild mammals should be sustainable, based on sound epidemiological and ecological knowledge, and must balance the requirements for preserving biodiversity, and protecting human health and economic well-being. Striking the appropriate balance between these interests will be a major challenge for the development of future national and international policies. The magnitude of

this task grows as the unrelenting processes of globalisation gradually move us in the direction of a free mixing population in which the opportunities for disease transmission and persistence are profoundly enhanced. At the same time, environmental degradation and habitat loss continue to reduce global biodiversity, and themselves contribute to the emergence of pathogens in wildlife. In the face of this growing threat to the health of humans, domestic animals and wildlife, there is an increasing awareness amongst many researchers, managers and stakeholders of the need to change the way we deal with these problems. All too often the management of wildlife diseases has in the past been characterised by reactive, unsustainable and ill-informed interventions that have ignored the fundamental importance of the ecology of hosts, pathogens and vectors, and have been out of step with the global imperative to conserve biodiversity. The conservation of species and preservation of healthy ecosystems are inextricably linked to sustained human well-being. Consequently the retention of biodiversity and the potential for adverse ecological impacts must become material considerations when choosing how we manage disease in wildlife. We need to start treating wildlife diseases as wildlife management issues, and to develop a greater capacity to predict and prepare for potential problems. To these ends we must ensure that we employ the appropriate contemporary tools such as mathematical modelling, risk assessment, economic analysis and GIS. And perhaps most importantly, we need to recognise the role that human activities play in perpetuating disease in wildlife, and the potential for changes in human attitudes and behaviour to reduce opportunities for disease emergence. The world has changed immeasurably in recent decades and so our approaches to managing disease in wildlife must change too.

Chapter 2
Wildlife Population Structure and Parasite Transmission: Implications for Disease Management

Paul C. Cross, Julian Drewe, Victoria Patrek, Gareth Pearce, Michael D. Samuel, and Richard J. Delahay

2.1 Introduction

Emerging infectious diseases have become an important challenge for wildlife ecologists and managers. Management actions to control these diseases are usually directed at the parasite, the host population, or a key component of the environment, with the goal of reducing disease exposure and transmission. Control methods directed at the host population, however, remain limited in approach (e.g. vaccination, population reduction, test-and-remove) and scope, by financial, logistical, ethical and political constraints. Furthermore, these control methods have often been implemented without due consideration of how host ecology and behaviour may influence disease dynamics. This chapter highlights how host population structure and social organisation affect parasite transmission and prevalence.

Traditionally, variation in disease prevalence among species, genders, and ages may have been explained by immunological differences in susceptibility. However,

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ecological and behavioural factors can also affect the rates and routes of parasite transmission and potential control options. Using this information, future control efforts may be improved by focusing on subsets of individuals, areas, environmental factors, or times of year that are most important in the propagation and persistence of a pathogen.

The social systems of mammal populations exhibit structure at several levels. Individuals vary by age, sex, reproductive status, genetic relatedness, position in a dominance hierarchy, social interactions and patterns of space-use. Group sizes can vary within and among species, from solitary individuals that only interact during mating, to socially complex groups or aggregations of over a million individuals. Within a group, the sex, age and social status of an individual, as well as the season, will often affect the number and type of intra-specific contacts experienced, thus affecting exposure and transmission of parasites. Meanwhile, the transmission of a parasite among groups may depend on group size, composition, territoriality and levels of inter-group movement or contact. This chapter explores how the characteristics of host social systems may interact with parasite life-history characteristics to affect parasite transmission, prevalence and dynamics, and hence the effectiveness of disease management strategies.

2.2 Intra-Group Factors

The gender, age, dominance and reproductive status of hosts are some of the characteristics that affect parasite prevalence and transmission within a group of individuals. Most studies of these host characteristics have focused on differences in prevalence, while only a few have compared incidence rates (Begon et al. 1999; Caley and Hone 2002; Heisey et al. 2006). Disease prevalence depends on the transmission rate, disease-induced mortality, duration of infection (or duration of antibodies for seroprevalence), and the length of time a disease has been present in the population. On the other hand, incidence measures the rate of infection per unit time. The distinction between prevalence and incidence is important because differences in prevalence are often assumed to correspond to differences in incidence. In some cases, however, differences in prevalence may instead be driven by disease-induced mortality or infectious periods that vary by sex, age and dominance.

2.2.1 Sex

Several studies suggest male-biased infection for bovine tuberculosis (*Mycobacterium bovis* infection; bTB) and chronic wasting disease (CWD) in deer (see Box 2.1) (Shang et al. 2002; Miller and Corner 2005; Grear et al. 2006), cowpox in rodents (Burthe et al. 2006), and nematode infections in chamois (*Rupicapra r. rupicapra*) (Citterio et al. 2006). Analyses using data collated from studies on a range of mammal species also report male-biased prevalence and intensity of parasitism (Poulin

Box 2.1 Chronic wasting disease (CWD) in deer

CWD belongs to a family of diseases known as transmissible spongiform encephalopathies (TSEs) which affect a wide range of mammals including humans (Williams et al. 2002b). The causative agent of TSEs is most likely an abnormal prion protein that is consistently associated with the disease (Prusiner 1991). CWD is the only TSE that affects free-ranging cervids (Miller et al. 2000). The origins of the disease are unknown, but in North America it was first recognised in the 1960s in captive cervids, and since 1981 in free-ranging deer. Clinical signs of illness develop about 1.5 years after infection, and no captive or wild cervid has subsequently recovered (Williams et al. 2002b).

Studies of CWD in captive deer indicate that direct contact (Miller and Williams 2003), and contact with prion contamination of the environment (Miller MW et al. 2004) are potential routes of transmission, although their relative importance in wild populations is poorly understood. The route of transmission, the role of social groups, and the spatial scale over which transmission occurs are factors that will affect whether CWD behaves like a frequency or density-dependent disease (Gross and Miller 2001; Schauber and Woolf 2003). For example, groups of female deer may overlap spatially, but have limited direct contact with other groups (Schauber et al. 2007) and risks of infection with bTB and CWD increase with the level of genetic relatedness (Blanchong et al. 2007; Grear 2006). These social boundaries may limit the rate of direct transmission between groups because contact is reduced. Thus, indirect transmission of CWD may be an important route of between group infections, but direct contact and indirect transmission may be important routes within social groups.

Adult male mule deer (*Odocoileus hemionus*) and white-tailed deer (*Odocoileus virginianus*) tend to have a higher prevalence of CWD than adult females and this increases with age (Miller and Conner 2005; Grear et al. 2006). Because there are no indications that adult males are more susceptible or harbour the disease for longer, this suggests that differences in social structure and behaviour of males and females may influence disease transmission. Several hypotheses have been suggested to explain the increased risk of CWD infection in males compared to females. First, males are typically more social than females, especially outside the breeding season when they form single sex groups, within which unrelated males readily groom one another. In contrast, female grooming usually takes place between mother-daughter pairs or among individuals from the same matrilineal group. Second, transmission to susceptible males may increase during the breeding season when they contact infected females or visit scent stations used by infected males. These behaviours may expose breeding males to prions, which are shed through the alimentary tract. In addition, males may be at greater risk of contact with prions in the environment than females owing to their larger home range size and breeding season movements.

(continued)

Box 2.1 (continued)

Differences in movement and dispersal between male and female white-tailed deer may also be a significant component of CWD distribution across the landscape, especially in areas where animals do not show seasonal migration. Between 50% to 80% of yearling males disperse distances of 10 to 30 km, depending on habitat characteristics (Long et al. 2005), whereas less than 20% of females disperse (Rosenberry et al. 1999). Infected yearling males are therefore more likely to spread CWD into new areas. Prevalence of CWD in yearling males and females is similar and considerably lower than in adult males. If environmental transmission is an important route of infection in free-ranging deer, then adult males have the potential to contaminate a wider area than females because of their larger home ranges and increased movements during breeding.

To a limited extent, movement and dispersal information have been used to establish CWD surveillance zones and assess local disease prevalence. In addition, movement distances and spatial scales for disease transmission have been used to identify areas for intensive culling or disease detection around new CWD positive deer or in areas of high infection risk (e.g., infected game farms). However, culling strategies to reduce numbers of adult males (which have higher rates of infection) or yearling males (which have higher rates of dispersal) may deserve further consideration. Whether strategies that focus on these higher risk components of the deer population could reduce transmission or spread of CWD is currently unknown, as is the geographical scale over which control should be implemented. In many cases, implementation of such male-biased culling strategies to control CWD will conflict with goals for trophy deer management and make public support for this approach challenging. Because of the long-term chronic nature and slow transmission of CWD in deer, epizootics are likely to last for decades making control a long-term problem, and emphasising the need for prevention or early detection and eradication.

1996; Schalk and Forbes 1997; Moore and Wilson 2002). Several studies have identified positive correlations between host body weight and the intensity of parasite infection (Poulin 1995; Arneberg et al. 1998; Ezenwa 2004; Burthe et al. 2006). These findings have produced a variety of hypotheses to explain male-biased parasitism. Larger hosts may provide more space or a greater diversity of niches for parasites. They may also present a larger target for vectors, and the greater nutritional requirements of larger hosts could increase their exposure to parasites that can be transmitted by ingestion. In many species, males have larger home ranges, which may also lead to increased exposure. Sex-related differences in physiology and behaviour may also produce differences in exposure and susceptibility to pathogens. In male mammals, increased stress levels during the breeding season and the physiological effects of testosterone may be linked to immunosuppression and increased susceptibility to disease (Zuk and McKean 1996).

Mating behaviour is also likely to have important implications for parasite exposure, particularly when considering sexually transmitted diseases (STDs). In this case transmission rates are likely to depend more on the prevalence, or frequency, of the infectious individuals rather than their overall density, because the number of sexual contacts experienced by each individual is likely to be constant across a wide range of population densities. Mammal mating systems range from monogamy (one male mates with one female) to polygynandry (both sexes mate with multiple partners). Polygamy (one male mates with several females) is the most common mating system among mammals. This strategy tends to increase the variance in mating success amongst males; such that some males mate with many females whilst others fail to mate with any. Theoretical investigations suggest that this reproductive variation may increase the prevalence of disease amongst females and reduce prevalence in males, as the few reproductive males are more likely to acquire and transmit infection to their partners, while non-reproductive males remain uninfected (Thrall et al. 2000). Although few empirical studies have been conducted, the prevalence of STDs was significantly higher amongst adult females in studies of STDs in primates (Nunn and Altizer 2004) and koalas (*Phascolarctos cinereus*) (Jackson et al. 1999).

2.2.2 Age

The relationship between age and prevalence is related to host characteristics and disease or parasite life histories. Assuming that hosts do not recover from infection and disease-induced mortality is low, prevalence often increases with age because older individuals have been exposed for longer (Fig. 2.1, Heisey et al. 2006). This has been demonstrated for bTB in bison (*Bison bison*) (Joly and Messier 2004) and African buffalo (*Syncerus caffer*) (Jolles et al. 2005), and for CWD in mule deer (*Odocoileus hemionus*) (Miller and Corner 2005; Grear et al. 2006; see Box 2.1). When antibody titres persist, seroprevalence (i.e. prevalence based on serological test results) is also likely to increase with age. In these cases, seroprevalence reflects past exposure rather than current infection. The form of the relationship between age and prevalence is also influenced by changes in immunity, age-dependent exposure, and both host and parasite mortality (Heisey et al. 2006). For example, if parasite-induced mortality increases with time since infection then prevalence may be lower in older age categories than in juveniles because older individuals are likely to have had the disease for longer and as a result older infected individuals die at a faster rate (Fig. 2.1).

When hosts can recover from infection and become immune, juveniles may have a higher prevalence than adults because many adults may have already been exposed and recovered (Cattadori et al. 2005). Age-dependent changes in immunity may also influence host susceptibility to disease. Infants may initially be protected by maternal antibodies, but once passive immunity wanes they may become susceptible, as recorded for rabbit haemorrhagic disease (Cooke 2002) and tapeworm infestation in mice (Theis and Schwab 1992). Furthermore, senescent individuals

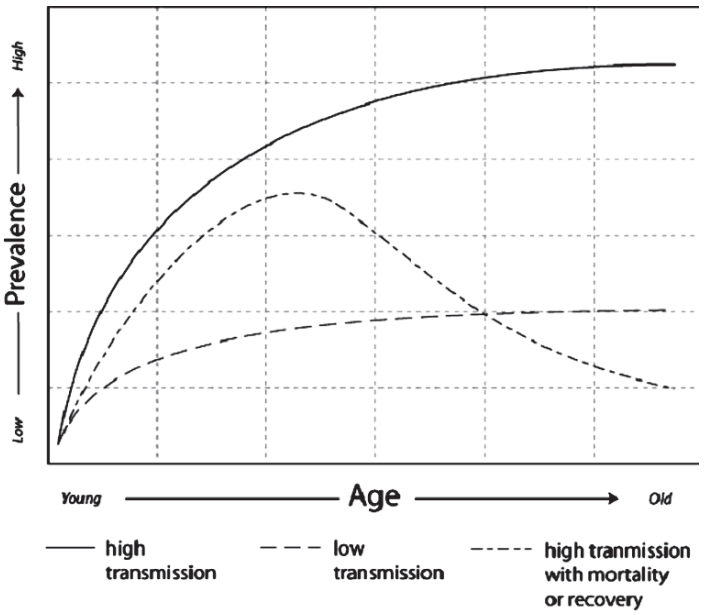

Fig. 2.1 Prevalence generally increases with age for many pathogens when individuals are born susceptible and do not recover. Higher transmission rates correspond to higher prevalence (compare solid and dashed lines). Disease recovery or disease-induced mortality may reduce prevalence in older ages (dot-dashed line)

may be more susceptible to disease due to declining immune function. Parasite-induced immunity may also affect age-prevalence patterns by either suppressing the immune system or priming the host for a stronger response to subsequent exposure. The latter seems to be the case for *Nematodirus gazellae* infections in saiga antelope (*Saiga tatarica tatarica*), in which parasite intensity peaked in 2–3 year olds but declined thereafter (Morgan et al. 2005).

2.2.3 Dominance

The influence of social dominance on parasitism is complicated by breeding behaviour, rank stability, and coping mechanisms for subordinates. Dominance is likely to affect exposure rates as well as stress. In general, mild and transient stressors enhance immunity, particularly innate immunity. Chronic stress, however, can suppress the immune system, but it remains unclear whether these changes are sufficient to increase the risk of infection (Dhabhar and McEwen 1999; Sapolsky 2005). Furthermore, those individuals that experience the most stress may be at either the top or the bottom of the dominance hierarchy, depending on the stability of the hierarchy and potential coping mechanisms (Sapolsky 2005). A study of captive cynomolgus monkeys (*Macaca fascicularis*) showed that low ranking individuals had higher rates of adenovirus infection (Cohen et al. 1997), whereas subordinate

males in a koala population had lower levels of STDs than dominant individuals (Jackson et al. 1999). At this point it is difficult to determine whether these differences are driven by contact patterns, routes of transmission, stress, susceptibility or combinations of these factors. Further research is necessary before information on dominance hierarchies can be used by managers to help control disease.

2.2.4 Superspreaders

Researchers, managers and disease modellers have in the past often assumed that all hosts are equally susceptible and infectious for microparasites. However, studies of some human diseases have shown that the distribution of the number of infections caused by an individual is also strongly skewed, whereby most individuals do not infect anyone, whilst a few infect many. As a result, it has been estimated that focusing half of all control effort on the most infectious 20% of cases may be up to threefold more effective than random control (Lloyd-Smith et al. 2005a). Such heterogeneities are also likely to apply to wildlife populations (Cross et al. 2007b) and so offer the potential for more effective management strategies if these so-called 'superspreaders' can be targeted. It is not yet clear to what extent this heterogeneity is due to differences in the immunological status of hosts, or to variations in contact rates arising from behaviour, or both. Therefore, there are significant logistical and diagnostic difficulties in identifying superspreaders in wildlife populations, which will require the development of new theoretical and diagnostic tools. In addition, it is not clear whether managers could focus control efforts on 'superspreader groups' and achieve similar improvements in effectiveness of control. Intuitively however, it seems reasonable to focus attention on individuals (or classes of animals) that have long infectious periods or high rates of contact with susceptibles, as they are likely to be significant in the spread of disease.

2.3 Inter-Group Factors

2.3.1 Territoriality

Territorial defence often involves aggressive encounters that may increase exposure to parasites. Defensive behaviours are energetically costly and may increase stress and testosterone levels, which can then suppress immune function (Zuk and McKean 1996). Acts of aggression may also enhance transmission by biting or scratching. Territorial species may also encounter a high rate of contact with infectious pathogens within their territory through environmental contamination with parasite-laden faeces. A study of strongyle nematodes in African bovids found higher levels of infection in territorial than in non-territorial species, most likely as a result of environmental contamination with faeces (Ezenwa 2004). On the other hand, territoriality may also serve to reduce parasite transmission by reducing the overall level

of direct contact between individuals or groups. This may be particularly pronounced amongst species that use indirect communication (e.g. scent marking and vocalisations) to minimise the need for direct contact. Individuals that occupy territories may also have access to more desirable resources making them less susceptible to parasitism. However, not all individuals within a single population will necessarily display territorial behaviour. Those that are unable to control a territory may "float" from one occupied territory to another, increasing their own exposure rates and facilitating the spread of disease across territories.

2.3.2 Group Size and Population Density

Hosts living in large aggregations are likely to have more direct contacts than those in small groups. When parasite transmission is a function of direct contacts, then prevalence is likely to increase with group size or population density (McCallum et al. 2001). The relationship between transmission rate and host density has profound implications for disease management. If transmission rates increase with density then reducing population size or density may be an effective management option. The distinction between population size and density is important (De Jong et al. 1995). In many cases, host population size may be strongly correlated with the extent of area occupied, such that as population size increases so too does the area occupied, resulting in minimal changes to density and contact rates (Begon et al. 2002). Although it is logical to assume that contact and transmission rates increase with density, the relationship may be confounded by host behaviour (e.g. territoriality or hosts seeking contacts at low densities). Also, it is seldom clear how to estimate the area occupied (i.e. the denominator), as even in the simple case of a fenced park, not all habitats may be accessible or usable by a given host species. For group-living species, contact rates are more likely to be related to local group size than overall population density.

The aggregation of animals at experimental feeding sites has been associated with significant increases in the prevalence of endoparasites in raccoons (*Procyon lotor*) (Wright and Gompper 2005), *M. bovis* in white-tailed deer (*Odocoileus virginianus*) (Chaddock 1998), and brucellosis (*Brucella abortus*) in elk (*Cervus elaphus*) (Cross et al. 2007c). A population size of 200 susceptible animals in an area of 220 km^2 has been suggested as the threshold density necessary for the maintenance of classical swine fever virus in populations of free-living wild boar (*Sus scrofa*) (Artois et al. 2002). However, population size rather than density was important in determining whether cowpox would invade and persist in a field study of wood mice (*Apodemus sylvaticus*) and bank voles (*Clethrionomys glareolus*) (Begon et al. 2003). Meta-analyses have shown nematode parasite richness, abundance and prevalence to be positively associated with population density in mammals (Arneberg 2002). Group size has also been implicated in promoting parasitism. A meta-analysis covering diverse taxa showed a positive association between group size, prevalence and intensity of contagious parasites (Côté and Poulin

1995). The relationship between parasite species richness and group size however appears highly variable, with studies showing positive, negative and absence of association between the two factors.

For directly-transmitted parasites in a single-host system, the relationship between population density and parasite transmission may be complicated by several factors. One theoretical study showed that the probability of a pandemic occurring depended on rates of host movement among groups, group size and the duration of infectiousness (Cross et al. 2005). Chronic infections with long infectious periods (e.g. bTB) required less movement among groups to create a pandemic (i.e. an epidemic that propagates across a large region and hence many groups) than those causing acute conditions, because they were able to persist for longer within the local group. Longer persistence within a group increases the likelihood that an infectious individual moves to another group. Larger group sizes and higher movement rates amongst groups facilitated the invasion of acute infections (e.g. rabies and rinderpest). This suggests that group sizes and movement rates are likely to affect the spread of acute diseases, such as rabies, more than chronic infections, such as tuberculosis. However, parasites causing acute disease often persist in other ways, such as in the environment or alternative hosts, or by causing latent infections in some individuals.

Transmission rates that vary seasonally or annually are also likely to affect the relationship between host population size and parasite prevalence. Seasonal variation in host social behaviour, such as breeding or wintering aggregations of deer and migrations of wildebeest in East Africa, may introduce temporal patterns in disease transmission. For example, brucellosis induces abortions in elk and bison prior to and during the calving season (Cheville et al. 1998). Other individuals become infected by licking or consuming the contaminated foetus. In northwestern Wyoming, USA, brucellosis seroprevalence was higher at sites where elk were provided with supplementary feed later into spring, because the timing and duration of host aggregation coincided with peak transmission (Cross et al. 2007c). This sort of complexity in the relationship between host population size or density and parasite transmission may be common to many wild mammal disease systems.

The effects of group size and population density appear to vary widely for indirectly transmitted parasites. Studies of malaria in primates have shown a higher prevalence of infection in larger groups, possibly because more hosts increase the strength of olfactory cue to mosquito vectors (Davies et al. 1991; Nunn and Heymann 2005). In contrast, other studies have provided evidence that the prevalence of parasitised individuals can be negatively associated with host group size when the parasite has a mobile vector. In feral horses (*Equus cabalus*) this phenomenon probably arises as a result of their tendency to aggregate when biting flies are most abundant (Côté and Poulin 1995).

Many parasites are neither specific to one host species nor directly transmitted amongst individuals. In primates, 68% of recorded parasites infected more than one host species and 43% were transmitted indirectly (e.g. via fomites, contaminated soil or water), 32% by arthropod vectors, 15% by intermediate hosts and 34% could be transmitted by multiple routes (Pedersen et al. 2005). Parasites that are transmitted