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Behavioural influences on disease risk: implications for conservation and management

Jason T. Hoverman [a, *], Catherine L. Searle [b]

[a] Department of Forestry and Natural Resources, Purdue University, West Lafayette, IN, U.S.A.
[b] Department of Biological Sciences, Purdue University, West Lafayette, IN, U.S.A.

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While parasites are fundamental components of ecological systems, emerging infectious diseases are a growing concern for conservation and management. Understanding the drivers and consequences of disease emergence in natural systems is complex because of the diverse array of factors associated with disease dynamics. Host behaviour plays an important role in disease dynamics across multiple scales (individuals to landscapes). Here, we synthesize our current understanding of the interplay between behaviour and disease in the context of conservation. We review the general importance of behaviour for determining the probability of exposure to parasites and the likelihood of infection once exposed. We also discuss the influence of infection on behaviours that affect disease transmission in populations and the potential trait-mediated indirect interactions that can influence disease risk within communities. Furthermore, we present several case studies demonstrating how the incorporation of behaviour into conservation and management strategies is critical for understanding emerging infectious diseases. Given the fundamental relationships between behaviour and infectious disease, there is a need for the development of practical methods for integrating this knowledge into conservation. Establishing a dialogue and forming collaborations between scientists and wildlife managers across multiple scales is an essential step. Ultimately, conservation practices that integrate knowledge of behaviour and infectious diseases will have a greater chance of success.

Host behaviour plays an important role in understanding disease dynamics at multiple scales. For individuals, host behaviour (e.g. activity level, habitat choice) affects the probability of encountering parasites and the risk of infection (Moore, 2002). Behaviour also plays a critical role in the transmission of parasites in populations and communities (Poulin, 2007b). Human modification of the landscape can influence the amount of contact among populations and the movement of populations, which alters both behaviour and disease risk (Dobson & Foufopoulos, 2001). Given that host behaviour is a fundamental component of disease dynamics, conservation and management efforts that account for these effects are necessary. Here, we synthesize our current understanding of the interplay between behaviour and disease in the context of conservation. We begin with a brief overview of the importance of parasites in ecology and evolution. Then, we summarize the significance of behaviour in disease dynamics at different scales (individuals to landscapes). Using a series of case studies, we then discuss how behaviour has or could play a role in conservation within the context of disease. We conclude with future challenges for integrating behaviour into conservation efforts seeking to manage disease risk.

THE IMPORTANCE OF PARASITES

Like free-living species, parasites are fundamental components of natural systems (Kuris et al., 2008; Lafferty et al., 2008). Research over the past several decades has revealed their importance within ecological and evolutionary frameworks (Lafferty et al., 2008; Poulin, 2007a). For instance, given that most free-living species have at least one parasite species, a large portion of biodiversity on the planet is represented by parasites (Dobson, Lafferty, Kuris, Hechinger, & Jetz, 2008). In some communities, parasites can represent a large fraction of the biomass and production (Kuris et al., 2008; Preston, Orlofske, Lambden, & Johnson, 2013). Because many parasites have complex life cycles, they also can play important roles in food webs (e.g. connectance, energy transfer; Lafferty et al., 2008). From an evolutionary perspective, parasites

* Correspondence: J. T. Hoverman, Department of Forestry and Natural Resources, Purdue University, West Lafayette, IN 47907, U.S.A.
E-mail address: jhoverm@purdue.edu (J. T. Hoverman).

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can shape the evolution of hosts by placing selection pressures on host populations and regulating host populations (Grenfell et al., 2004; May & Anderson, 1983).

While the importance of parasites in ecology and evolution is increasingly recognized, emerging infectious diseases are a growing concern for humans and wildlife (Daszak, Cunningham, & Hyatt, 2000; Dobson & Foufopoulos, 2001; Jones et al., 2008). Infectious diseases can be classified as emerging for several reasons, including an increase in prevalence or virulence in a host population, spread to a new population or species, or recent evolution of the parasite (e.g. evolved virulence, strain diversification). Emerging infectious diseases such as canine distemper in carnivores, chytridiomycosis in amphibians and white-nose syndrome in bats have received considerable attention due to their devastating effects on host populations (Daszak et al., 2000; Fisher et al., 2012). Many factors can contribute to the emergence of infectious diseases, which has been extensively reviewed (Daszak et al., 2000; Dobson & Foufopoulos, 2001). However, isolating these mechanisms is challenging because of the simultaneous and interacting effects of anthropogenic factors (e.g. climate change, pollution, eutrophication, species loss). Importantly, emerging infectious diseases are one contributor to what is becoming known as the sixth mass extinction (Wake & Vredenburg, 2008). It is imperative that conservation and management practices use a comprehensive approach to combat disease outbreaks, including the consideration of host behaviour in establishing policies for managing infectious diseases. In this review, we focus on host behaviour because very little is known about parasite behaviour, particularly in the context of conservation. However, we acknowledge that behaviour of both hosts and parasites can influence disease outcomes and may be necessary to consider when establishing conservation practices.

BEHAVIOUR AND DISEASE ACROSS SCALES

Individuals and Populations

For individual hosts, a broad range of factors can influence disease risk; age, sex, body size and genetics determine the probability of exposure to parasites or the likelihood of infection once exposed (Schmid-Hempel, 2011). Host behaviours also can influence disease risk (Fig. 1). For instance, parasite burdens tend to be higher in hosts with higher activity levels or with broader home ranges because encounter rates with parasites are greater (Craft, Volz, Packer, & Meyers, 2011). Similarly, parasite burdens can be higher in hosts with extensive contact networks (Craft et al., 2011). Alternatively, hosts can display defensive behaviours to reduce their chances of becoming infected. Antiparasite behaviours such as erratic movement, grooming, migration and self-medication can function to decrease parasite loads (Daly & Johnson, 2011; Hart, 1994, 1997; Taylor, Oseen, & Wassersug, 2004). For instance, ruminants infected with gastrointestinal nematodes will change their diets and selectively consume plants with anthelmintic properties (Villalba, Miller, Ungar, Landau, & Glendinning, 2014).

Once a host is infected, there is a diverse set of potential effects on the host, including changes in growth, development, reproduction, physiology, gene expression, morphology and behaviour (Hart, 1990). While we focus on behaviour, we underscore that these other effects can occur jointly with behaviour and interact to influence disease outcomes (e.g. morbidity, mortality). Sickness behaviours such as lethargy, reduced grooming, loss of appetite and sneezing commonly occur following infection (Hart, 1990; Loehle, 1995; Fig. 1). While many sickness behaviours are simple by-products of infection rather than adaptations for the host or parasite (Poulin, 1995), they can still be important within the context of populations and communities (see below). Some parasites with complex life cycles alter the behaviour of their hosts to facilitate transmission between host species (Poulin, 1995). For instance, parasite-induced vulnerability to predation has been documented in many systems where the parasite requires both the predator and the prey to complete its life cycle (discussed below).

Because individuals are embedded in populations, the behaviour of individuals can scale up to influence population-level processes in several ways. A regularly observed pattern in host populations is parasite aggregation, in which a small percentage of the population harbours the majority of the parasites (Poulin, 2007a). Such infection heterogeneity is driven by many factors, including variation in host traits such as behaviour (Wilson et al., 2001). For instance, experimental manipulations of behaviour, size and immunity in tadpoles demonstrated that individual-level variation in behaviour (i.e. lower activity levels) was a major driver of trematode aggregation in the population (Johnson & Hoverman, 2014). If infected hosts exhibit sickness behaviours or other trait changes that increase parasite loads, these effects can be amplified, leading to stronger patterns of parasite aggregation in the population (Johnson & Hoverman, 2014). The behaviour of individuals within a population also influences transmission rates, such as with superspreaders. In brief, superspreaders are hosts that are responsible for a disproportionately large number of the transmission events for a population (Lloyd-Smith, Schreiber, & Getz, 2005; Stein, 2011). Hosts that display risky behaviours or have large contact networks are ideal superspreaders in populations. In humans, classic examples of superspreaders include Mary Mallone ('Typhoid Mary') for typhoid fever and Gaetan Dugas for HIV (Hudson, Perkins, & Cattadori, 2008; Paull et al., 2012). As we will discuss below, highly infected individuals or superspreaders are frequently targeted in conservation and management strategies to prevent disease spread in populations.

Communities

Host–parasite interactions are embedded in complex ecological communities. One of the primary goals of disease ecology is to expand beyond single host–parasite interactions to address how other ecological interactions (e.g. competition, predation) influence disease dynamics. Indeed, competition and predation can play pivotal roles in disease dynamics via trait-mediated indirect interactions. Trait-mediated indirect interactions occur when the interaction between two species (e.g. two competitors, a predator and its prey) alters the traits of a species, which in turn change interactions with other species in the community (Werner & Peacor, 2003). From a disease perspective, shifts in the activity or habitat use of hosts induced by the presence of competitors and predators have the potential to influence infection risk (Orlofske, Jadin, Hoverman, & Johnson, 2014; Raffel, Martin, & Rohr, 2008; Szuroczki & Richardson, 2009; Thiemann & Wassersug, 2000; Fig. 1). For instance, research with amphibians and zooplankton (e.g. *Daphnia*) has found that predators induce lower activity levels or shifts in habitat, respectively, leading to increased risk of infection (Decaestecker, De Meester, & Ebert, 2002; Orlofske et al., 2014).

Conversely, parasites can have large effects on ecological processes. In addition to free-living species, parasites can initiate trait-mediated indirect interactions (Fig. 1). Green frog tadpoles, *Lithobates clamitans*, exposed to free-living stages of trematode parasites increased their activity levels as an avoidance response (Marino & Werner, 2013). As a result, tadpoles experienced greater predation rates by larval dragonflies, which target highly active prey. Parasite-induced behavioural changes can have broader implications for the structure and function of communities, particularly when infections significantly alter the behaviour of keystone species or ecosystem engineers. In several systems, parasites alter

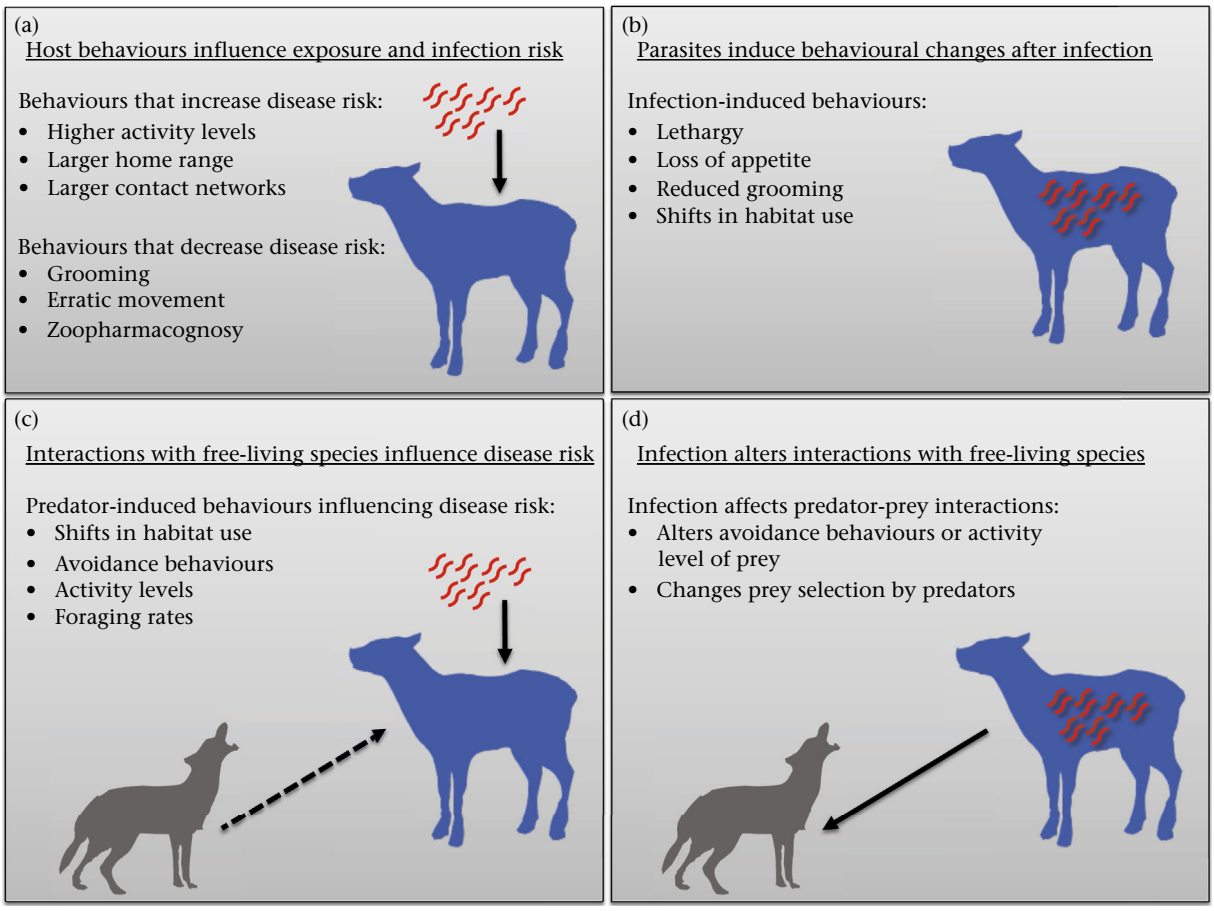

Figure 1. Schematic of the interplay between host behaviour and disease risk. (a) Host behaviours can influence the probability of exposure to parasites or the risk of infection once exposed. (b) Following infection, host behaviours can be altered. These behavioural changes can be by-products of the infection or adaptions for the parasite that increase transmission rates. (c) Within communities, interactions with other free-living species (e.g. competitors, predators) can influence the exposure and infection risk via changes in host behaviour. In the illustrated example, the presence of predators (i.e. wolves) induces behavioural changes in the prey (i.e. ungulates) that increase the risk of infection. (d) Parasites can also influence the interactions between infected hosts and free-living species. Here, infected prey display behaviours that influence predation rates.

the behaviour of key herbivores, causing a cascade of effects on communities. For instance, periwinkles are dominant grazers in intertidal zones. However, trematode infections in periwinkles can dramatically reduce their feeding rates, which ultimately shifts the composition of the algal community towards more edible species (Wood et al., 2007). In intertidal communities in New Zealand, cockles serve as ecosystem engineers by causing bioturbation associated with their burrowing activity and by providing surfaces for the attachment of epibionts. Parasite infection leads to reduced activity and burrowing in cockles, which increases the risk of predation by whelk and fish. In addition, there are a series of knock-on effects including changes in community structure, primary productivity and secondary consumer biomass (Poulin, 2007b). Thus, trait-mediated indirect interactions can be driven by parasite infection or by other members of the community, with a variety of effects on disease dynamics.

Landscapes

Movement of host species across landscapes can dramatically affect disease dynamics (Altizer, Bartel, & Han, 2011). Understanding how host dispersal and migration influence disease risk is increasingly important as human activities lead to more fragmented landscapes, limiting movement of organisms. Changes to landscapes can influence disease and behaviour through altered migration and dispersal and increased interactions between domestic and wild species.

Host migration can spread parasites across landscapes or allow hosts to reduce infection prevalence. For example, the spread of West Nile virus (WNV) has been partially attributed to migratory birds (Malkinson et al., 2002; Owen et al., 2006; Rappole, Derrickson, & Hubalek, 2000), although evidence that migratory birds are a primary driver of WNV spread remains equivocal (Rappole & Hubalek, 2003). In contrast, migration could reduce disease risk in host populations through two mechanisms: migratory escape and migratory culling. When host species inhabit a single location, parasites may build up in the environment through time. Thus, migratory species may benefit from seasonally leaving contaminated areas if parasites are unable to survive until their hosts return. This process, termed 'migratory escape' (Loehle, 1995), will allow hosts to return to habitats with a reduced risk of infection. Additionally, migratory species may experience reduced infection prevalence through 'migratory culling' (Bradley & Altizer, 2005). Infection can create physiological stress for hosts, making them unable to complete energetically costly migrations. Thus, migration events can cull infected individuals and reduce infection prevalence in host populations. These processes of migratory escape and migratory culling can act simultaneously (as outlined in our example below), but may also act independently. Combined, both processes will lower rates of infection in migrating species.

Dispersal behaviours can also have large impacts on infectious disease. At the metapopulation level, dispersal may spread parasites across landscapes. The size and configuration of individual populations can influence the likelihood of disease outbreaks and subsequent persistence (Hess, 1996; Stapp, Antolin, & Ball, 2004). For example, populations of black-tailed prairie dogs, *Cynomys ludovicianus*, are more likely to go extinct during plague outbreaks if they are very large or small. This is likely because large populations are more likely to acquire infections and support larger vector populations, while small populations are more likely to go extinct due to stochasticity (Stapp et al., 2004).

Synthesis

From the above overview, it is clear that behaviour is important in disease dynamics across multiple scales by influencing exposure rates, susceptibility to infection, pathogen loads and transmission within populations and across species. However, a significant challenge in disease ecology is linking the effects observed at multiple spatial scales into a single framework to understand disease dynamics (Johnson, De Roode, & Fenton, 2015). Indeed, most disease-centred ecological research tends to focus on a single scale due to the complexities associated with understanding cross-scale interactions and the need for both observational and experimental studies to assess the relative importance of different processes (Johnson et al., 2015). Future research should focus on key behavioural determinants of disease dynamics at different scales.

INFECTIOUS DISEASES AND CONSERVATION

Given the diverse linkages between behaviour and disease, an understanding of behaviour can improve our ability to forecast disease emergence and potentially manage disease outbreaks. However, relatively few studies have explicitly incorporated behaviour into conservation and management strategies targeting diseases. In this section, we provide several examples illustrating the linkages between behaviour and disease in conservation.

Social Behaviour and Disease Risk

From a conservation perspective, the emergence of an infectious disease in threatened and endangered species has been the focal point of many research programmes. These species have been reduced to low populations sizes and typically possess lower levels of genetic diversity. For species already pushed to the brink of extinction, the added insult of an infectious disease could be devastating. Tasmanian devils, *Sarcophilus harrisii*, the world's largest remaining carnivorous marsupial, are a prime example of an endangered species threatened by an emerging disease (Fig. 2a). Historically, the species was widespread throughout Tasmania, but in recent years, their populations have declined precipitously; they were listed as endangered by the International Union for the Conservation of Nature and Natural Resources (IUCN) in 2008. A major player in the population decline of Tasmanian devils is devil facial tumor disease, which is caused by a transmissible cancer with a high fatality rate (Hamede, McCallum, & Jones, 2013; Hawkins et al., 2006; McCallum et al., 2009). In brief, live tumor cells are directly transmitted between hosts during intimate contact (Hamede et al., 2013). The tumor cells replicate clonally in the host but appear to be genetically distinct from the host's cells (Pearse & Swift, 2006).

In Tasmanian devils, spread of facial tumor disease is linked to genetic diversity and behaviour (McCallum et al., 2009; Siddle et al., 2007). First, this species has very low genetic diversity in the major histocompatibility complex (MHC), which plays a key role in mounting immune responses (Siddle et al., 2007). When infected with facial tumor disease, the tumor cells are not recognized as foreign bodies and thus are not attacked by the immune system. Host behaviours also are critical in this system because they aid in transmitting the cancer (McCallum et al., 2009). During the mating season, Tasmanian devils display aggressive interactions through biting with their sharp canine teeth. Bites can occur during aggressive encounters between males and when females are mate-guarded. This biting behaviour is ideally suited for spreading facial tumor disease because the tumor cells must reach below the epidermal tissue to initiate the infection (tumor cells do not persist outside a host). Because these aggressive interactions are largely restricted to the breeding season when individuals are gathered together, disease transmission is best described as frequency dependent (McCallum et al., 2009). Diseases with frequency-dependent transmission are problematic because they do not require a threshold host population size for parasite establishment, so they can persist in small populations and potentially drive host populations to extinction (McCallum, Barlow, & Hone, 2001).

Because there is no treatment or vaccine, Hamede et al. (2013) suggested that behaviour could be used in management and conservation strategies. For instance, they suggested that removing highly aggressive animals identified during trapping events could be a strategy for reducing transmission. While there have been no attempts to apply this strategy to manage the disease, animal

Figure 2. Host–parasite interactions that are strongly influenced by host behaviours. (a) Transmission of facial tumor disease in Tasmanian devils, *Sarcophilus harrisii*, is driven by biting and aggressive interactions between individuals. Photo credit: Andrew Storfer. (b) Migratory populations of monarch butterflies, *Danaus plexippus*, support lower parasite loads due to migratory culling and migratory escape. Photo credit: Sonia Altizer.

behaviourists would be ideally suited to explore the implications of such an approach. For instance, the viability of this strategy may be limited if populations of Tasmanian devils continue to decline. Ultimately, facial tumor disease provides an important conservation lesson: that diseases with frequency-dependent transmission can threaten species of concern with extinction (McCallum, 2008). Given the strong influence of behaviour on disease dynamics in this system, interactions between animal behaviourists and disease ecologists will be vital for Tasmanian devil conservation.

Migration and Disease Risk

The impacts of migration behaviours on infectious disease have been well studied in monarch butterflies, *Danaus plexippus*, which are commonly infected with the protozoan parasite *Ophryocystis elektroscirrha* (Altizer & Oberhauser, 1999; Fig. 2b). In North America, populations of monarchs exhibit several patterns of migration; eastern populations migrate up to 5200 km to their overwintering grounds, while populations in southern Florida are nonmigratory (Brower & Malcolm, 1991). Populations also exhibit variation in parasite prevalence; nonmigratory populations exhibit much higher parasite prevalence than migratory populations (Altizer, Oberhauser, & Brower, 2000). These empirical observations are supported by theoretical models, which suggest that populations with more extreme migrations (in terms of time spent away from breeding sites and distance migrated) have lower rates of infection (Hall, Altizer, & Bartel, 2014). Additionally, migratory populations are expected to be less vulnerable to population declines driven by infectious disease (Hall et al., 2014).

Migratory culling and migratory escape likely drives these patterns. Infection with *O. elektroscirrha* in monarchs is known to shorten flight distances, reduce body size and increase mortality (Altizer & Oberhauser, 1999; Bradley & Altizer, 2005). Thus, the chances of a monarch successfully reaching overwintering grounds are greatly reduce when infected (supporting migratory culling; Bradley & Altizer, 2005). Parasite prevalence increases throughout the breeding season (Bartel, Oberhauser, de Roode, & Altizer, 2011), indicating that habitats may become contaminated through time (supporting migratory escape; Loehle, 1995). Thus, both migratory culling and migratory escape may act together to reduce infection in migratory populations. There is also evidence that *O. elektroscirrha* is less virulent in populations with long migratory distances compared to populations with shorter migratory distances (de Roode & Altizer, 2010). This indicates that migration can also influence long-term, evolutionary patterns of host–parasite interactions.

Migrating species such as monarchs are of particular conservation concern (Wilcove & Wikelski, 2008). A number of human activities disproportionately affect migratory species, such as habitat destruction, erection of barriers and encounters with humans and domestic species. Monarch migrations are considered an endangered phenomenon, primarily due to habitat loss (Brower & Malcolm, 1991). However, climate change is also threatening migratory monarchs by shifting breeding habitats northward (Batalden, Oberhauser, & Peterson, 2007). If monarchs and their food sources are unable to make these distributional shifts, migrating populations of monarchs may be particularly at risk, leaving only populations that do not migrate, increasing the risk of parasite-induced population declines. Additionally, introduction of exotic milkweed, which provides year-round food for monarchs in the southern United States, has increased the number of nonmigratory populations, leading to increased infection prevalence (Satterfield, Maerz, & Altizer, 2015). Thus, human activities that reduce migration of monarchs may indirectly increase parasite prevalence in this host species. Conservation efforts should therefore focus on preserving habitat for migrating populations and controlling the distribution and spread of exotic milkweed. Future studies should focus on identifying ways to help maintain migratory populations and to reduce rates of disease in nonmigrating populations.

Biodiversity Loss: Imperiled Predators and Healthy Herds

While biodiversity loss has been documented in nearly all systems and across all trophic levels, larger-bodied animals have been disproportionately reduced due to human activities (Estes et al., 2011). Indeed, the loss of top predators is a ubiquitous pattern in terrestrial, freshwater and marine systems (Estes et al., 2011). Numerous theoretical studies have explored the implications of predator removal on disease dynamics (reviewed in Hatcher & Dunn, 2011). In general, these studies predict that predator removal will tend to increase infection in prey populations across a broad range of conditions (Holt & Roy, 2007; Packer, Holt, Hudson, Lafferty, & Dobson, 2003). Predators are predicted to have the greatest effect on prey infection levels when predators selectively consume infected prey (i.e. behavioural preference). In many systems, predators preferentially select inactive or behaviourally abnormal prey because these individuals are more easily captured (reviewed in Moore, 2002). By targeting infected prey, predators remove parasites from the system (assuming no trophic transmission) while simultaneously increasing the abundance of healthy individuals (Fig. 3). Empirical support for the predicted effects of predators on disease dynamics has been found in several systems (Hudson, Dobson, & Newborn, 1992; Murray, Cary, & Keith, 1997; but see Duffy, Housley, Penczykowski, Caceres, & Hall, 2011). For instance, Hudson et al. (1992) demonstrated that red grouse, *Lagopus lagopus scotica*, populations have lower nematode burdens in the presence of predators compared to populations without predators, suggesting that predators selectively prey on heavily infected prey. Moreover, they followed the field study with an experimental study providing anthelminthic treatment to red

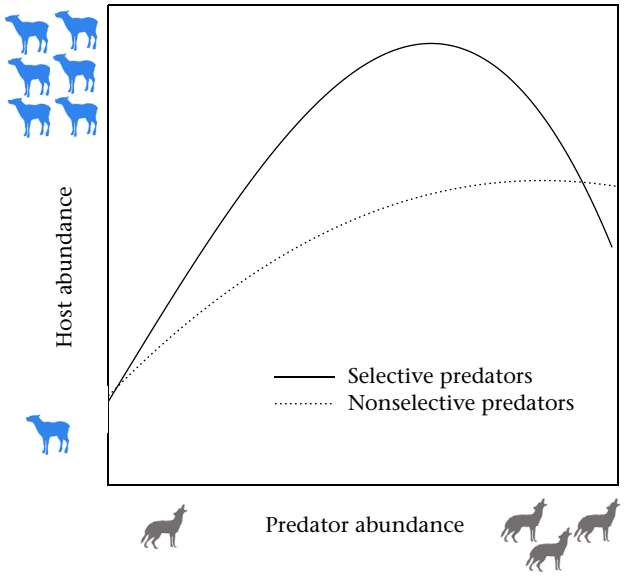

Figure 3. Modelling results of Packer et al. (2003) demonstrating the release of a host population from regulation by a macroparasite with increased predation. Increases in the abundance of a nonselective predator (dotted line) lead to an increase in host abundance. However, with selective predators (i.e. predators that target infected prey; solid line), growth of the host population increases at a faster rate because a large fraction of the parasites are removed. Redrawn from Packer et al. (2003).

grouse populations and demonstrated that reduced parasite burdens reduced the risk of predation.

These results have led to the development of the healthy herds hypothesis, which posits that selective predators can reduce infection levels and disease risk in prey by acting as parasite sinks (Packer et al., 2003). The healthy herds hypothesis has important implications for conservation strategies. The maintenance or restoration of predator communities may be critical for reducing disease occurrence and outbreaks in species of concern. By restoring historic trophic interactions that have been broken by human activities, conservation efforts may have a greater likelihood of success if disease is a contributing factor to species loss. However, the application of this theory to wildlife disease management is lacking (Joseph et al., 2013). Interestingly, many management programmes focusing on the recovery of prey populations call for predator reduction or removal, which could inadvertently increase disease risk. Thus, it will be important for future conservation and management plans to integrate modelling approaches that help predict population dynamics when predators and parasites co-occur in communities.

CHALLENGES IN WILDLIFE DISEASE MANAGEMENT

Disease management has historically been applied to human and livestock populations (Wobeser, 2002). However, recent efforts have been directed towards managing diseases in wildlife populations. While disease is a natural component of wildlife populations, with important ecological and evolutionary implications, human activities such as climate change, global trade and habitat alteration have inadvertently altered the interaction between wildlife and their parasites (Langwig et al., 2015). As a result, emerging wildlife diseases are an increasing concern. The management of disease within wildlife populations is extremely challenging because wildlife populations are relatively large and difficult to monitor (Wobeser, 2007). In addition, these populations are embedded within communities where the other species present may have numerous effects on the focal species (e.g. by acting as a reservoir host, predator or food resource). Unlike many human and livestock systems, there are numerous environmental factors that influence disease dynamics in wild populations that complicate modelling approaches. Collectively, these issues limit the efficacy of traditional management strategies such as vaccination, quarantine, habitat modification and relocation for wildlife populations (Wobeser, 2002). Here, we address some of the future challenges associated with wildlife disease management within the context of behaviour.

Before management strategies can be implemented, it is vital to know how parasites spread through a population (Craft, 2015). The transmission rate of infection can be calculated by multiplying the number of contacts within a network by the probability that a contact results in transmission (Fig. 4). While this calculation is theoretically straightforward, obtaining estimates of these parameters is especially difficult for wildlife. One reason for this difficulty is that behaviour strongly influences the contact structure or network for a population. Because factors such as migration, dispersal, sociality and territoriality influence contact rates, there can be substantial heterogeneity in contact rates among individuals. Superspreaders are a prime example of such heterogeneity in contact networks. Consequently, conventional disease models that assume populations are well mixed with homogenous contact structures can fail to capture the complexities of disease transmission in wild populations. To account for heterogeneity in contact networks, there has been a recent shift to using network modelling. In brief, network models incorporate individual-level heterogeneity into contact patterns (reviewed in Craft, 2015). Technological advances such as radiotelemetry, radio frequency identification, global positioning system tracking and video monitoring have aided in the application of network modelling by generating detailed information on contacts in populations. While assumptions regarding the probability of transmission given contact between individuals are still necessary, network modelling can improve our ability to understand disease dynamics in wildlife when host behaviour generates heterogeneity in contact rates. Network modelling has mainly been applied to human and livestock systems, yet studies focused on wildlife on are the rise (Craft, 2015). Importantly, these studies can provide insights into key individuals or groups for future management strategies. Moreover, animal behaviourists can play a critical role in the development of network models because of their detailed understanding of factors (e.g. migration, dispersal, sociality, territoriality) that potentially contribute to disease transmission.

Current management of infectious diseases in natural systems generally involves one of a few approaches. One approach is to cull infected populations, either by targeting infected individuals to reduce infection prevalence, or by culling a set number of individuals to reduce host population densities (Bolzoni, Tessoni, Groppi, & De Leo, 2014; Donnelly et al., 2003; Lachish, McCallum, Mann, Pukk, & Jones, 2010). While disease models that include network modelling can provide potential targets for culling, this approach has a number of limitations. In general, this strategy cannot be used for species with small populations, because culling could drive the species to extinction. This method will also be

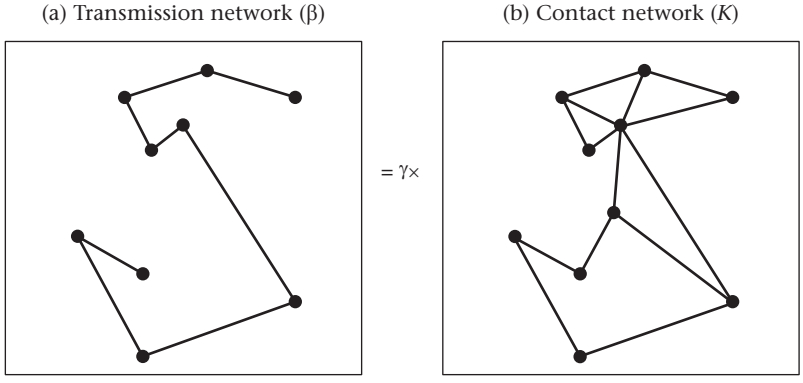

Figure 4. Visual conceptualization demonstrating that the transmission rate of an infection within a network (β) is related to the number of contacts within the network (*K*) multiplied by the probability that a contact results in transmission (γ). Closed circles represent individuals in the population. Lines connecting individuals represent (a) transmission events or (b) contacts within the population. Figure adapted from Craft (2015).

ineffective if reservoir species or alternative hosts are present in the community, because they can maintain the parasite even if the target species reaches very low densities. These nontarget species can be the focus of culling to reduce disease risk (Donnelly et al., 2003), but this approach could lead to unintended indirect effects on the species of interest. Moreover, culling can lead to behavioural changes in the focal host, which may alter disease risk.

The culling of European badgers, *Meles meles*, in the United Kingdom is a classic example of how behaviour influences disease management (Woodroffe et al., 2006). To reduce the risk of disease caused by bovine tuberculosis (TB) in cattle, the British government implemented culling of badgers to reduce spillover. Over the course of 30 years of culling, the incidence of TB in the cattle generally increased. Moreover, experimental studies have demonstrated both increases and decreases in TB incidence in areas were badgers were culled, indicating that this management approach may not be working (Donnelly et al., 2003). One of the driving forces in increased TB incidence in the cattle in culled areas was behavioural changes in the remaining badgers; the remaining badgers increased movement rates, leading to higher contact rates with cattle and increased likelihood of transmission. This system demonstrates the important role of behaviour in determining the success of disease management. When culling reduces population size, the behaviour of the remaining individuals can be altered, which can directly or indirectly influence disease transmission. In general, culling may be a useful strategy for disease management, but unintended effects on the behaviour of target species and communities may reduce the effectiveness of these programmes. Again, animal behaviourists can play a role in predicting these effects by collaborating with disease ecologists and managers to forecast behavioural changes in response to management practices.

Interestingly, a major factor in the need for culling programmes is the human-induced disruption of trophic interactions. Substantial reductions in biodiversity across the globe have eliminated or altered ecological interactions that regulate populations. By reestablishing historic trophic interactions (e.g. apex predators), we can substitute ecological interactions for human-mediated culling to manage disease. This also speaks to the broader issue of managing for biodiversity rather than focusing on individual species. This is not a new concept (Kuusipalo & Kangas, 1994; Vanewright, Humphries, & Williams, 1991; Westman, 1990), and yet most conservation policies still target single species. As we describe above, both infectious disease risk and behaviour of a species can be influenced by community context. In some systems, loss of biodiversity can increase disease risk (Hall et al., 2009; Johnson, Preston, Hoverman, & LaFonte, 2013; LoGiudice, Ostfeld, Schmidt, & Keesing, 2003; Searle, Biga, Spatafora, & Blaustein, 2011). This negative relationship between biodiversity and disease risk is termed the 'dilution effect' (Keesing, Holt, & Ostfeld, 2006) and can be caused by multiple factors. From a conservation perspective, this means that the loss of biodiversity from human actions can lead to increased disease risk for the remaining species. While the dilution effect is not found in all systems (in fact, the opposite pattern can also be observed; Keesing et al., 2006), any changes to biodiversity and community structure can alter disease dynamics. Thus, the impacts of losing or adding a species to the community must be scrutinized for effects on disease risk and, subsequently, behaviour.

The relationship between host behaviour and infectious disease is also essential for effective reserve design. In particular, knowledge of how species migrate and disperse is necessary to balance the potential benefits and risks of connectivity. Hess (1996) demonstrated that connectivity can allow highly contagious diseases with moderate severity to increase the likelihood of metapopulation extinction. However, other theoretical models suggest that the benefits of habitat corridors outweigh risks (Gog, Woodroffe, & Swinton, 2002; McCallum & Dobson, 2002). For example, establishing marine protected areas can increase disease risk when fish frequently move between high-density reserves and unprotected areas. However, the presence of marine protected areas still has an overall positive effect on fisheries by reducing the likelihood of fishery collapse (McCallum, Gerber, & Jani, 2005). Thus, the relative costs and benefits of corridors and dispersal will vary with each host–parasite system, underscoring the continuing need for model development in this field.

Close proximity to human activities can also limit the effectiveness of wildlife reserves. For example, several infectious diseases of conservation concern can spill over from domestic to wild populations including pneumonia in bighorn sheep, *Ovis canadensis* (Foreyt & Jessup, 1982), toxoplasmosis in southern sea otters, *Enhydra lutris nereis* (Miller et al., 2002), and tuberculosis in African buffalo, *Syncerus caffer* (Michel et al., 2009). Bolder species or individuals that more readily interact with humans and domestic species will be at greater risk of acquiring these parasites. Additionally, human activities that encourage 'tameness' of hosts (e.g. feeding) can increase host densities near humans, altering disease risk (Hegglin, Bontadina, & Deplazes, 2015). In general, limiting overlap between human activities and wildlife can reduce the likelihood of parasite spillover into wild populations.

In addition to informing reserve design, knowledge of dispersal and migration behaviours can help predict how infectious diseases will be influenced by global changes. Climate change can alter the timing of migrations (Crick & Sparks, 1999; Mills, 2005), with a number of possible effects on disease. If parasites rely on hosts arriving to a location at a particular time, climate change may decrease disease risk by shifting this timing. However, changes in migratory phenology could also lead to novel host–parasite interactions with an increased disease risk for naïve hosts. Additionally, as climate change shifts optimal environmental conditions for organisms, hosts may disperse to new locations (Walther et al., 2002), also leading to novel host–parasite interactions or movement of reservoir species. In all, altered migration or dispersal patterns in response to climate change will shift relative disease risk across landscapes. Thus, modelling approaches that incorporate changes in range shifts and phenology for both hosts and parasites are necessary to predict future hotspots for disease outbreaks.

Finally, throughout this review, we have focused on host behaviour because virtually nothing is known about parasite behaviour in the context of conservation. However, parasite behaviour can vary with a number of biotic and abiotic conditions (Haas, Korner, Hutterer, Wegner, & Haberl, 1995; Koprivnikar & Poulin, 2009; Pasternak, Blasius, & Abelson, 2004) such that understanding their behaviour may be important for conservation efforts. For example, activity levels of the transmission stage of the amphibian pathogen *Batrachochytrium dendrobatidis* can be influenced by temperature (Voyles et al., 2012), and a major route of transmission of this parasite is through water, where it uses chemotaxis to locate its hosts (Moss, Reddy, Dorta, & Francisco, 2008). Thus, changes to the abiotic environment (e.g. through climate change) could influence the behaviour of this pathogen, and therefore its rate of transmission. Future work on this parasite and others should consider the role of anthropogenic factors on parasite behaviour. Understanding how parasite behaviour can be incorporated into wildlife management and conservation is an open area of research that merits greater consideration.

As outlined in this review, host behaviour plays a critical role in understanding disease dynamics at multiple scales. Human activities can alter both behaviour and disease dynamics of target species. Thus, understanding the relationships between behaviour and disease in the context of conservation is essential for developing

conservation policies for species, communities and ecosystems. Conservation practices that integrate knowledge of behaviour and infectious diseases will have a greater chance of success. For animal behaviourists, disease ecology is a ripe field for applying their expertise. The inclusion of behavioural data can dramatically improve our understanding of disease risk across multiple scales (individuals to ecosystems). Given the recent emphasis on incorporating ecological principles into disease ecology (Johnson et al., 2015), knowledge of animal behaviour will increasingly serve as a basis for exploring conservation in systems faced with emerging infectious diseases.

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