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Behavioural ecology and infectious disease: implications for conservation of biodiversity

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Behaviour underpins interactions among conspecifics and between species, with consequences for the transmission of disease-causing parasites. Because many parasites lead to declines in population size and increased risk of extinction for threatened species, understanding the link between host behaviour and disease transmission is particularly important for conservation management. Here, we consider the intersection of behaviour, ecology and parasite transmission, broadly encompassing micro- and macroparasites. We focus on behaviours that have direct impacts on transmission, as well as the behaviours that result from infection. Given the important role of parasites in host survival and reproduction, the effects of behaviour on parasitism can scale up to population-level processes, thus affecting species conservation. Understanding how conservation and infectious disease control strategies actually affect transmission potential can therefore often only be understood through a behavioural lens. We highlight how behavioural perspectives of disease ecology apply to conservation by reviewing the different ways that behavioural ecology influences parasite transmission and conservation goals.

This article is part of the theme issue 'Linking behaviour to dynamics of populations and communities: application of novel approaches in behavioural ecology to conservation'.

1. Introduction

Parasites and their hosts coevolve, often in an arms race in which hosts exhibit behavioural strategies for clearing or avoiding parasites, and parasite traits facilitate transmission and evade host immunity [1,2]. Host behaviours include strategies to mitigate parasite exposure, such as foraging to avoid contaminated areas [3], and reduced contact with sick conspecifics [4,5]. Other behaviours, however, facilitate the transmission of parasites, including promiscuous mating [6], and feeding on infected prey [7]. The interplay of behaviour and parasite transmission has consequences that scale up to populations [8], as well as communities and ecosystems [9] (figure 1). It is, therefore, necessary to understand the many connections between host behaviour and parasitism to effectively model population and community dynamics.

In the modern age of anthropogenic impacts on biodiversity, multiple compounding factors are putting species at risk of extinction. Infectious diseases are among the top five causes of species extinctions and have been implicated in the extirpation or extinction of 4% of species [10,11]. Many more species are currently threatened by the combined effects of disease and anthropogenic factors such as habitat loss, fragmentation and harvesting [12,13]. Parasitism can negatively impact host population density [14,15], and behaviours related to parasite avoidance, such as monogamy, can reduce effective population size [16,17]. When parasites cause declines in host population sizes, the host becomes more vulnerable to extinction owing to other behavioural phenomena, such as Allee effects and demographic stochasticity [18,19]. Thus, the interactions between behaviour and disease are essential for understanding population viability.

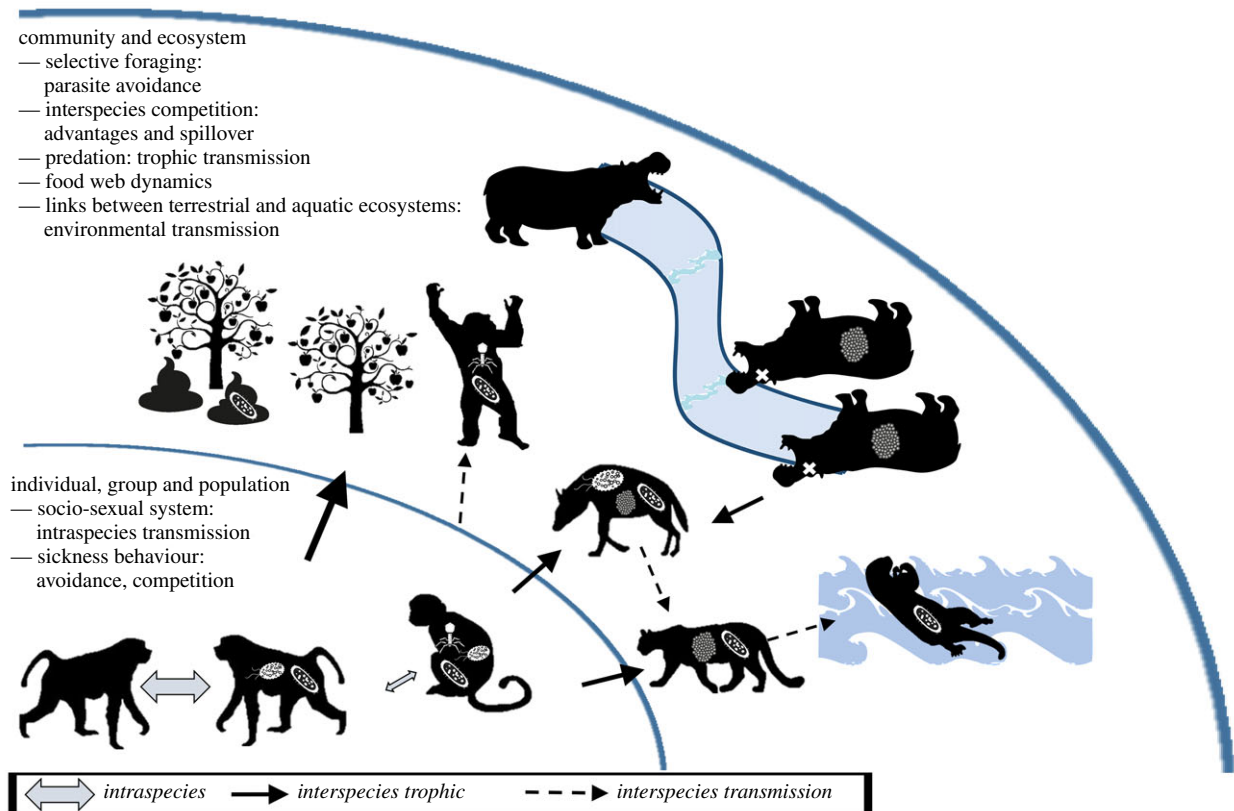


Figure 1. Schematic illustrating the links between behaviour and infectious disease transmission across levels of biological organization. From individual behaviours, such as avoiding sick conspecifics, to group- and population-levels, parasites can affect parasite dynamics within species. Parasitism affects communities and ecosystems via cross-species spillover and food webs dynamics.

Several definitions are important for the review that follows. Throughout, we use an ecological definition of a parasite as any organism that lives in or on a host at some cost to that host, including micro-parasites such as bacteria, protozoa and viruses, and macroparasites, such as helminths and arthropods [11]. Parasites are transmitted via direct contact among individuals—e.g. grooming, mating, fighting—and indirectly via shared space and exposure to infectious stages of parasites in the environment. Vector-borne parasites are transmitted when infected vectors, such as mosquitoes and arthropod ectoparasites, feed on hosts. Parasites can also have multiple hosts throughout their life cycles, and can be transmitted through intermediate hosts to definitive hosts, including trophic transmission when a predator eats infected prey [11,20,21]. We generally consider three measures of parasitism. Prevalence refers to the proportion of individuals that are infected. Intensity or parasite ‘load’ is a measure of how many parasites infect a host individual. Richness is the number of parasite species found in an individual host, a group, or a species.

Here, we review how behaviour influences parasite transmission, with impacts on individuals, populations, communities and ecosystems (figure 1). We especially focus on the nexus of disease, behaviour and ecological principles that can be leveraged for management and conservation (table 1). First, we examine the effects of behaviours on parasite transmission because behaviour affects how hosts interact with the environment and other individuals, thus influencing exposure to parasites. Second, we investigate the effects of parasite infection on behaviour because infection then influences the acquisition of resources, fecundity and predation risk, including through sickness behaviours, mating patterns and manipulation of host behaviour by parasites. Recognizing that these

concepts are not mutually exclusive, we illustrate how they are often involved in feedbacks of transmission.

2. Behaviours that influence parasite transmission

The behavioural interactions among individuals, with the environment, and vectors affect the transmission of parasites (figure 1). While some behaviours increase the risk of parasite transmission, others are counterstrategies to avoid parasites. The interactions among individuals, driven by their social organization, network dynamics and patterns of range use, influence the probability of contacts that can facilitate transmission. When parasites infect multiple hosts, the patterns of interactions among species in communities creates a complex system of transmission potential. In this section, we discuss each of these phenomena in turn, illustrating how behaviour affects transmission with consequences for species viability and conservation.

(a) Social organization

Ecological and social factors lead many species to be solitary throughout most of the year, while others have a tendency to form aggregations of social groups. Parasitism is hypothesized to be a significant cost of group living [33–35]. Species that live in larger groups are predicted to have higher rates of parasitism than those that are solitary or form smaller groups owing to the higher local density of hosts [14], accumulation of infectious material in the environment [36], and migrants that carry new parasites into groups [37]. Indeed, larger flocks of birds

Table 1. Links between empirical examples of animal behaviour and parasite transmission with consequences and applications to conservation and management.

behaviour	effect of parasites on populations	conservation and management	example host–parasite system
behaviours that affect parasite transmission			
group size	group-living can increase parasitism with negative consequences for mortality	group-living species monitored for parasite, and larger colonies targeted for interventions	mange in wolves [22]
	benefits of group-living can offset costs of parasites		helminths in ungulates [23]
			Ebola in gorillas [24]
social network properties	variation among individuals in contact rates predict outbreak patterns	individuals with high centrality targeted for intervention (e.g. vaccination/removal)	hantavirus in rodents [25]
	individuals highly connected in networks can be super-spreaders	understanding how network subdivision affects parasite transmission	facial tumour disease in Tasmanian devils [26,27]
	overall network subdivision impacts outbreak dynamics		viruses in chimpanzees [28]
parasite infection affects behaviour			
sickness behaviour	sick individuals reduce activity and social interactions, decreasing potential for transmission	recognizing sickness behaviour important for culling/treating target individuals	finches and <i>Mycoplasma</i> [4]
	healthy individuals may preferentially interact with sick individuals because they are poor competitors, increasing transmission potential		
mate choice	females choose healthy males as mates, decreasing transmission from infected males	monitoring transmission of sexually transmitted diseases important for management	meta-analyses of birds, fishes, amphibians, lizards and endo- and ecto-parasites [29,30]
		mate choice affects breeding population size and variation in population size owing to demographic stochasticity	
parasite manipulation of hosts	parasites affect host behaviour to promote transmission	recognizing how trophic transmission affects predator–prey dynamics	<i>Toxoplasma</i> transmission, especially from small prey to carnivores [31]
		monitoring rabies, feral dogs and endangered carnivores	ethiopian wolves and rabies lyssavirus [32]

had higher parasite prevalence than solitary birds [38,39], and group-living gorillas had higher mortality rates from Ebola (97%) than solitary males (77%, [24]). It would, therefore, appear that living in groups can have significant costs owing to parasitism.

Despite the theory outlined above that predicts a positive relationship between group size and parasitism, empirical results have often found no relationship, or even a negative association, between group size and parasitism. In African bovids, for example, group size was not correlated with parasitism in 11 sympatric species, and only buffalo and hartebeest had a higher prevalence of helminth infection in larger groups than smaller ones [23]. In comparative studies of primates and rodents, as well as meta-analyses across multiple taxa, there were weak or nonsignificant relationships

between group size and parasite species richness [40–43]. These results suggest that other mechanisms are buffering individuals in larger groups from the higher risk of parasitism.

The benefits of group living, such as herd immunity, may offset the costs of parasitism in larger groups [44–47]. Other mechanisms by which the benefits of group living outweigh the costs of parasitism include increased vigilance for predators and access to resources. For example, although parasite infection risk was higher in larger groups of Grant's gazelles (*Nanger granti*) than in smaller groups, the cumulative benefit of vigilance allowed individuals to spend more time feeding in larger groups [48]. Solitary wolves (*Canis lupus*) were more likely to die from mange infection than those that lived in packs, most likely owing to the increased probability of prey capture in groups [22]. Thus, sociality can increase the

probability that individuals gain access to resources, and decrease the threat of predation and competition. Increased nutrient intake and decreased stress could offset deleterious impacts of parasites, such as decreased foraging owing to lethargy or nutrients lost to helminths. As habitats are lost and degraded, however, the availability of resources often declines, making it more difficult to sustain large social groups and impacting population health beyond expectations based on nutrients or parasites alone [49].

(b) Social networks and transmission

The patterns of behavioural interactions among individuals also affect the probability of parasite transmission over and above what would be expected based on random interactions among individuals (i.e. a mass action model). Heterogeneity in contact rates leads some individuals to be more connected in social networks than others. The highly connected, or more central, individuals probably play key roles in parasite transmission, potentially serving as super-spreaders [50]. Heterogeneities in contacts led to the '20/80 rule,' in which a small proportion of the population (20%) is predicted to account for a large majority of the contacts and transmission potential (80%) [51]. Variation in contact rates among individuals and through time is especially important for predicting characteristics of networks with impacts for disease outbreaks [28,52–56], and to clarify why the relationship between group size and parasitism is inconsistent and weak across studies [57,58].

In addition to the properties of individuals, variation in pairwise interactions can produce structure in the overall network. In primate social groups, for example, subgroups often emerge, within which interactions are more frequent relative to interactions between subgroups [59,60]. In social network theory, subgrouping is measured using metrics such as community modularity [61]. High modularity can decrease transmission because fragmentation of the network and cohesion within subgroups tend to 'trap' parasites within a subgroup, leading to lower outbreak size and delayed transmission dynamics [53,57,58,60,62]. These features of network structure and individual centrality can, therefore, have important applications to conservation management strategies.

Social network theory can be applied in designing intervention strategies such as vaccination or culling (table 1). Because only a subset of individuals accounts for the majority of disease transmission, targeting those individuals for treatment or removal would be more effective than random treatment [51]. In an epidemiological simulation on observed networks of chimpanzees (*Pan troglodytes*), vaccinating the most central individuals in social networks would reduce the proportion of the population that would have to be treated by 10%, compared to random treatment [28]. When parasites are highly contagious, however, all individuals become infected quickly and connectivity in the network does not affect outbreak dynamics.

Though centrality-based approaches have potential applications to disease interventions and population management, the effectiveness is dependent on properties of the whole network. One such case is the Tasmanian devil facial tumour disease, in which Tasmanian devils (*Sarcophilus harrisi*) transmit cancerous cells among individuals through biting [63] (table 1). The disease reduces fecundity and increases mortality, leading to a 60% decline in population size [64]. Although contact networks of Tasmanian devils exhibit

heterogeneities in potential disease transmission, their networks show low modularity [26]. Targeted vaccination programmes focused on central individuals would, therefore, have little effect on slowing the transmission of facial tumour disease [27]. Instead, isolating uninfected populations is an important priority. For conservation, it is important to understand how the pattern of interactions among hosts as well as overall network structure affect the process of parasite transmission, but also how parasite infections affect the dynamics of the network. In future research, dynamic network models could be applied to investigate spatio-temporal variation in contacts that are important for parasite transmission [54,55].

Owing to the difficulty of studying the social relationships of a whole population, social networks of subgroups can be used to identify individual traits linked to parasite transmission, such as sex, body size and relative location in foraging parties, which can be used in lieu of quantitative data on individual network centrality. For example, larger-bodied and more aggressive deer mice (*Peromyscus maniculatus*) have higher contact rates and probability of hantavirus infection than smaller individuals [25], and large male mice had a higher prevalence of *Leptospira* infection than small males and females [65]. Chimpanzees that foraged with the rest of the group had higher centrality than those that foraged alone, and this salient behaviour could be used to target individuals for vaccination [28]. Targeted intervention programmes that account for these transmission-related behavioural traits could improve the efficacy of management, and also allow managers to forecast the spread of diseases by predicting contact rates based on the frequency of individuals with specific behavioural traits in the population, thus improving recommendations for interventions.

(c) Range use, intergroup encounters and dispersal

Living in distinct, stable home ranges creates structure in population networks, and can lead to barriers that act to quarantine infectious diseases, thus potentially reducing disease risk at the population level [66,67]. In this context, it is important to consider the influence of territoriality, intergroup encounters and dispersal on parasite transmission. Territorial behaviour can act as a parasite-avoidance strategy to reduce contacts with neighbouring individuals or groups, but it may also promote environmental transmission through increased home range use intensity, i.e. a large day range relative to home range [20]. Territorial species must patrol and defend their range boundaries, and this tends to increase the intensity of range use compared to non-territorial species [68,69]. In a spatially explicit individual-based model of faecal-oral parasite transmission in a socially structured population, range-use intensity was a strong predictor of parasite prevalence [36]. Furthermore, greater use of core areas was positively related to parasite prevalence, while home-range overlap among groups was not [36]. Across primate species, range-use intensity and territoriality were positively associated with parasite species richness, including parasites with direct and indirect transmission modes [70]. The association between range-use intensity and the probability of exposure to infectious agents in the environment leads to increased threat of parasitism for species forced into small patches owing to habitat loss and fragmentation (e.g. [71]). Thus, parasitism can compound other effects of anthropogenic habitat loss, such as limited resources.

As discussed above, group size alone did not explain variation in helminth infection in most African bovids. Instead,

nuances of sociality and territoriality explain variation in parasitism. Among Grant's gazelles, territorial males have higher infection intensity with directly transmitted strongyle worms than non-territorial males and nursery herds which float among territorial males [23]. Furthermore, gregarious and territorial ungulate species had higher parasite richness than solitary and non-territorial species [23]. These effects may be partially explained by higher exposure to more highly contaminated soils in territorial than non-territorial species [3,72]. Variation in physiology also plays a proximate role, with aggression linked to higher testosterone and therefore potentially compromising the immune system compared to less aggressive males [73]. The intrinsic and extrinsic traits that affect heterogeneity in exposure and susceptibility are important for future research to understand variation in parasitism.

Dispersal, including leaving the natal territory, can facilitate disease transmission, especially in group-living species. The effects of sex-biased dispersal may lead to different outcomes for parasite transmission. For example, theoretical models showed that dispersal of low-ranking males in a socially structured population had little effect on intergroup sexually transmitted disease (STD) transmission, given that these males were less likely to have access to mates and therefore had lower STD prevalence [74]. By contrast, an individual-based model revealed that parasite transmission was driven by the dispersal of infected females from groups when males died owing to the disease, which the authors termed 'parasite-mediated dispersal' [37]. Consistent with this theoretical model, empirical data on primate species showed that prevalence and richness of STDs are higher when females disperse [74].

To apply these behavioural perspectives in a conservation context, managers will need to account for the combined effects of intergroup encounters and dispersal patterns. For example, dispersing individuals may not always be the disease super-spreaders. In lions, dispersing bachelor males were not the most important individuals in the spread of canine distemper virus as they rarely come into contact with resident individuals; instead, intergroup encounters among female prides with resident males were more likely to transmit the virus [75]. Modelling approaches that can integrate data on behavioural ecology, including social networks, range-use intensity, intergroup encounters and dispersal patterns, allow for more realistic predictions of parasite transmission. Furthermore, these models can produce nuanced understanding of the most important factors driving transmission.

Similarly, the combined effects of territoriality and dispersal behaviours have important applications to strategies for mitigating the impacts of disease spread in wildlife management. Culling or harvesting involves removing a subset of the population to manage disease and overpopulation (table 1). Culling can be effective because host population density is a limiting factor for parasite transmission [13,76,77], and can alleviate both the threat of disease and the negative impacts of overpopulation (e.g. white-tailed deer, [78,79]). Such strategies may require extremely large proportions of the population to be removed, which is problematic for endangered wildlife [77]. However, when individuals are removed, territories may open, with individuals dispersing into those vacant areas and increasing parasite transmission [80,81]. Predicting the efficacy of culling for disease management must, therefore, incorporate aspects of behavioural ecology.

European badgers (*Meles meles*) in the UK, for example, are implicated in the spread of bovine tuberculosis to cattle, posing problems for livestock [82]. Culling was used to control badger populations but also destabilized their territories, which led to immigration into the culled area, greater overlap in ranges, and less stable range boundaries [82]. This destabilizing effect can increase disease prevalence if the rate, duration and spatial extent of culling are inadequate to properly control the population [82,83]. An area for future research is to model the effects of culling and vaccination regimes that apply knowledge of territorial behaviour to dynamic social networks. For example, male badgers are responsible for most between-group contacts that could facilitate parasite transmission among groups [84]. Using simulations, one approach would be to test the prediction that maintaining populations of territory-holding males via vaccination will decrease dispersal by stabilizing territories. Though badgers are not an endangered species, the system is important for understanding how behaviour affects disease transmission, and how strategies to mitigate spill-over to livestock can implement behavioural ecology theory. The findings from the badger system can then be used to inform disease management of endangered species.

(d) Shared use of limited resources

The spatial distribution and abundance of resources influence the probability of contacts and thus parasite transmission. Illustrating the feedbacks among behaviours, intergroup encounters often occur at shared resources, which can lead to the transmission of parasites, both within and between species (figure 1). In gorillas, for example, different groups overlapped in the fruit trees they visited and were exposed to potentially infectious agents that could lead to parasite transmission [85]. These observations are especially worrisome because Ebola is an important viral parasite that could be transmitted among groups, with devastating impacts on ape populations [24,85–87]. The patterns of shared space and resource use among groups of gorillas is mirrored in many species, and can be modelled to understand the general effects of space use and intergroup encounters for the transmission of parasites.

Behavioural choices associated with drinking water can also influence parasitism. For example, when water is limited, sharing waterholes can generate intense exposure to parasites. Empirical results illustrate how sharing limited water sources was related to bovine tuberculosis (*Mycobacterium tuberculosis*) prevalence in wild boars and red deer [88,89] and mass outbreaks of anthrax (*Bacillus anthracis*) in hippos (*Hippopotamus amphibius*) [90]. In theoretical models, seasonal waterhole sharing markedly increased disease prevalence, and seasonal migration increased disease risk at shared resources, but also enabled hosts to escape parasites that built up in their 'normal' home ranges [91]. Understanding the impacts of the distribution and use of limited resources on the probability of disease transmission can be important for managing diseases in wildlife and livestock, especially as anthropogenic pressures cause declines in the abundance of resources and as a result changes in ranging patterns.

The union of movement and landscape ecology with disease epidemiology has important applications to conservation given anthropogenic habitat degradation [92]. In simulations of intergroup encounters based on data from long-term studies of endangered primates, direct transmission of a parasite was

related to the abundance of resources in the landscape [93]. The highest probability of intergroup encounters and thus parasite transmission occurred at intermediate levels of resource density. By contrast, the probability of parasite transmission was low when resources were highly abundant or scarce. As natural habitats shrink and animals are forced into smaller, lower quality ranges, the effects of ranging behaviour on parasite transmission potential will be important for predicting future population dynamics.

(e) Cross-species transmission

Many behaviours affect parasite transmission among multiple hosts (figure 1). Cross-species transmission involves a source or reservoir host that transmits a parasite to a target species, with deleterious effects for the target species [50,94]. Reservoir species are susceptible to the parasite but live long enough to support transmission, use spaces that expose them to parasites and vectors, and are physiologically similar to other hosts such that the parasite can establish in multiple hosts [95]. The probability of transmission among species is predicted to be higher for species that have similar behavioural and ecological niches. Examples of cross-species transmission illustrate the negative impacts of reservoirs on the management and conservation of target species [94,96]. Reservoirs allow the parasite to persist, even if the population size of the target is small. Developing management plans to mitigate a disease outbreak in an endangered species must therefore also consider if it is a disease caused by a multispecies parasite and how to manage the potential reservoirs as well as the target threatened species.

Overlap in space and shared resources among species creates opportunities for transmission [85,89,97]. Canine distemper virus in lions most likely had multiple spill-over events from sympatric hyenas and jackals, which could transmit the virus at shared resources such as carcasses [98]. Therefore, in most complex communities with multiple, closely related species, the opportunities for parasite transmission are greater than would be predicted from a single-species model. Management strategies should incorporate these multi-host dynamics to accurately represent transmission risks to threatened species (e.g. [98]).

3. Effects of infection on behaviour and transmission

The review above illustrates how host behaviour affects parasite transmission patterns. While many of these behaviours are probably independent of parasite infection, infection itself can change the behaviour of hosts in many ways. Some behaviours, including sickness behaviours, are outcomes of infection that help to clear the parasite. Parasitic infection has long been hypothesized to drive mate choice decisions, with concomitant effects on fitness and the evolution of phenotypes. In other cases, the parasites themselves have evolved mechanisms to manipulate host behaviour to promote their own transmission. This section describes the feedback between parasitic infection and host behaviour that affects transmission dynamics. The effects of infection on behaviour can be especially important for the management of wildlife because infection often increases the chances of onward transmission and the risk of predation.

(a) Sickness behaviours

The most direct way that parasitic infection can affect the behaviour of an individual is through sickness behaviours. Sickness behaviours include a suite of behavioural changes related to infection, such as general inactivity or lethargy, increased sleep, reduced social interactions and feeding, and postures that help to reduce heat loss and mount a fever response [99]. Although these behaviours have benefits for conserving energy and mounting an immune response to clear the parasite, they also probably entail significant fitness costs, such as susceptibility to predation [100] or losing competitive interactions.

Changes to interactions among individuals in relation to infection status affects the probability of parasite transmission. Infected mice used fewer nest sites than uninfected mice, leading to reduced interactions with other individuals [101]. This change in behaviour would probably decrease the probability of transmission owing to the decreased interactions between infected and susceptible individuals. Sickness behaviours are generally expected to reduce contact rates and social transmission of parasites; however, in captive house finches (*Carpodacus mexicanus*), healthy males increased their time spent feeding in proximity to males experimentally infected with *Mycoplasma gallisepticum*, and won aggressive interactions more often than with uninfected males [4]. This change in behavioural interactions owing to parasitic infection probably increases the probability of transmission. Individual-based models revealed that the decreased activity of infected individuals can result in increased contact rates and transmission when resources are limited and individuals are crowded in space [102]. Therefore, sickness behaviours can have opposing outcomes for the probability of onward transmission. This highlights that quantitative assessments of sickness behaviours in wildlife are important for understanding how the recovery of infected animals affects population dynamics via effects on disease transmission and the potential for increased predation risk. Conservation strategies may aim at using sickness behaviours to reduce transmission rates by isolating infected individuals.

(b) Mate choice

For many sexually reproducing animals, direct physical contact is needed for fertilization. As we discussed previously, contacts among individuals are fundamental to the social network, and provide opportunities for the exchange of infectious agents. Mate choice behaviour, therefore, has important impacts on individuals' reproductive success and the probability of infection with a parasite, effects which may scale up to population-level phenomena such as host population growth rates and parasite prevalence. Understanding the causes and consequences of mate choice behaviour can clarify the roles of coevolved parasites in driving host phenotypes.

Parasitism is hypothesized to affect mate choice, with the exaggeration of male advertisement characteristics commonly posited to be driven by female choice, including in relation to parasitism. Females may choose males with exaggerated sexual traits because males have heritable genetic resistance to infectious disease [103]. In meta-analyses of inter- and intraspecific studies with ecto- and endoparasites, half of the studies found positive associations between exaggerated male sexual traits and parasite intensity, but the overall relationship was not significant [29]. Rather than choosing

males based on indirect signals of genetic resistance to parasites, females seem to use direct signals of infection to avoid sick males [6,30]. Experimental evidence across studies of more than 15 host species has shown that females chose healthy males over infected males in the absence of exaggerated sexual traits [30,104,105]. Therefore, the cues of infection status may directly influence female mating preferences, affecting male reproductive success as well as the possibility of parasite transmission.

A counter-example is found in Tasmanian devil facial tumour disease described above. Animals with the highest reproductive success are the most socially dominant and most frequent biters—behaviours that result in a high probability of infecting others with facial tumour disease [106]. Thus, Tasmanian devils that have the highest fitness are also most exposed to the parasite. Management options available to conserve the population include large-scale removal of all potentially infected individuals (those greater than 2 years of age) and isolation of uninfected populations [27].

Regardless of the proximate and ultimate mechanisms of sexual selection, the subsequent influence of infection on mating behaviours and reproductive success has important implications for population growth. The links between individual behaviours—especially mate selection—and parasite transmission are important to consider in assessing population viability and wildlife management [16]. Selective mating affects an individual's probability of finding a mate and thereby influences effective population size, variance in fecundity, and the probability of extinction [19,107]. Thus, information on mate choice and reproductive success increases the accuracy of population viability models. Furthermore, high reproductive skew may increase the frequency of beneficial genes, but may also eliminate genetic variation that is important for adapting to future change and coevolving parasites [108]. Awareness of these consequences is important for population management and breeding programs. These behaviours scale up to population-level phenomena, with impacts on social organization, networks, territoriality, and range use all of which influence the transmission of parasites.

(c) Mating system

The mating system of organisms may be a selective response to infected individuals and the risk of parasite transmission. While mate choice refers to which individuals choose to mate with one another, mating systems refer to the ways in which individuals associate for reproduction, including short- and long-term associations. Lifetime monogamy is a potential behavioural counter-strategy to STDs because mating with only a single partner would reduce exposure to STDs [67]. In support of this hypothesis, promiscuity was positively related to the number of basal white blood cells, a measure of investment in immune defence, in a comparative study across species of primates and carnivores [109,110]. While monogamy may reduce parasite transmission, it is also a mating strategy that reduces the effective population size by decreasing the proportion of mated females because of the difficulty of finding bachelor males, and thus increases demographic stochasticity and extinction probability [16]. Therefore, different optimal strategies exist between avoiding infectious disease and maximizing reproductive success.

Monogamy can be a safe strategy in the context of STDs, but variation in other factors, especially STD prevalence, is

correlated with changes in the probability of infection that can outweigh the protective effects of monogamy [111]. Based on simulations of a single host species with multiple mating strategies and a theoretical STD that was sterilizing but not deadly, when STD transmissibility and prevalence were low, monogamy reduced the probability of individual infection. When transmissibility and prevalence were high, however, the chances of mating with an infected male were high whether females were monogamous or promiscuous, and females maximized their chances of conception by having multiple mates [111,112]. These results highlight that while monogamy should generally reduce STD transmission, variation in STD prevalence and other factors also affect the optimal mating strategy.

(d) Parasite manipulation of host behaviour

Selection can act on parasite traits to manipulate host behaviour when the traits benefit transmission. Parasites often exhibit exquisite adaptations to manipulate host behaviours in ways that increase the probability of onward transmission, often at substantial costs to the host [113]. These 'parasite manipulation' behaviours are distinct from sickness behaviours because they increase parasite transmission, while sickness behaviours are aimed at clearing infection and thus reducing transmission. Parasites manipulate the behaviour of hosts in a variety of ways, including within- and among-species transmission. Many parasites have complex life cycles including multiple hosts, such as those nematodes and trematodes that encyst in the flesh of intermediate hosts that will be preyed upon by the definitive host. These parasites benefit from increased predation on the intermediate host, as increasing predation rates increase transmission.

Several parasites exploit their hosts' central nervous systems to promote their own transmission [114]. Rabies, a lyssavirus that is transmitted through saliva, causes behavioural changes in the host that increase aggression and decrease fearful responses, both of which promote transmission by increasing biting [114,115]. Rabies has important impacts on wildlife management, such as when it was introduced to threatened Ethiopian wolves by a feral dog, causing increased aggression and salivation, and killed 10% of the known population (table 1, [32]). Effective rabies management will necessarily include multiple perspectives that combine population ecology, epidemiology, behavioural modelling [116], as well as treatment and population control of feral dogs [115].

STDs generally benefit from increased sexual behaviour of infected hosts [117]. One way to achieve this, at least in female mammals, is through sterility: by eliminating gestation and lactational amenorrhea, a sterilizing STD would increase the frequency of mating and thus transmission. STDs can have major impacts on individual host fitness, causing severe damage to reproductive organs [118] and infertility [119] that have negative consequences for population growth and are important for wildlife management and captive breeding [119,120].

Parasites with complex life cycles can manipulate host behaviour to promote trophic transmission by affecting the likelihood of predation (table 1). *Toxoplasma*, for example, is a protozoan parasite with a complex life cycle. Infectious stages reside in multiple hosts, especially rodents and carnivores, such as cats [121]. Rodents, the intermediate hosts, become infected by exposure to oocytes in the faeces of cats or in the environment, and the parasite transmits back to the cat,

the definitive host, through predation. Consistent with the manipulation hypothesis, infected rodents exhibited increased exploratory behaviour and became less fearful when confronted with cat odour's [122]. Similarly, killifish infected with trematodes which encyst their brains were preyed on significantly more frequently by herons, the definitive host, than were uninfected fish [7]. By affecting host population growth and the behaviour of hosts that make them more vulnerable to predation, parasites have important impacts on food webs [15,122–126], with potentially destabilizing effects for ecosystems. Addressing how behavioural changes owing to parasitic infections at lower trophic levels may affect higher levels of the food chain is therefore important to assess the resilience of an ecosystem to environmental change.

4. Conclusions

We reviewed the connections between parasitism and the behaviour of animals, and how those behaviours affect ecology and conservation. Our review reveals how feedbacks between parasitism and behaviour affect population dynamics via survival and fecundity are linked across scales, from individuals to ecosystems. Transmission begins with individual-level behavioural interactions with conspecifics, with the environment, and with other species. These processes affect population-level phenomena, and ultimately they can impact ecosystem dynamics. This review illustrates the ways in which host behaviour affects the dynamics of parasite transmission, via ecological and social mechanisms, and how those dynamics affect host population viability. The interconnections among mechanisms lead to a complex system in which many factors may simultaneously facilitate and impede parasite transmission (figure 2). To understand the outcome of behavioural effects on parasite transmission, research needs to account for the trade-offs of ecological and social processes for parasite transmission.

Basic epidemiological models can be made significantly more realistic when incorporating information on behaviour. Disease-driven population declines or extinctions have led to wildlife management approaches that apply principles of population ecology, epidemiology and network theory (table 1). In the future, with habitat loss, degradation and climate change, the important interplay between parasitism, behaviour and population dynamics will be perturbed, with consequences for the conservation of wildlife. Incorporating information on behavioural ecology can improve the efficacy of wildlife management decisions in light of future threats to biodiversity.

Areas of future research in this realm include modelling approaches that incorporate heterogeneities in contacts among individuals, social groups, and species through time [55,75,98]. Furthermore, combined intervention strategies including culling, vaccination and contraception may elicit the desired effects of decreased transmission without adversely perturbing the social organization of populations. Realistic models that incorporate these nuances can give

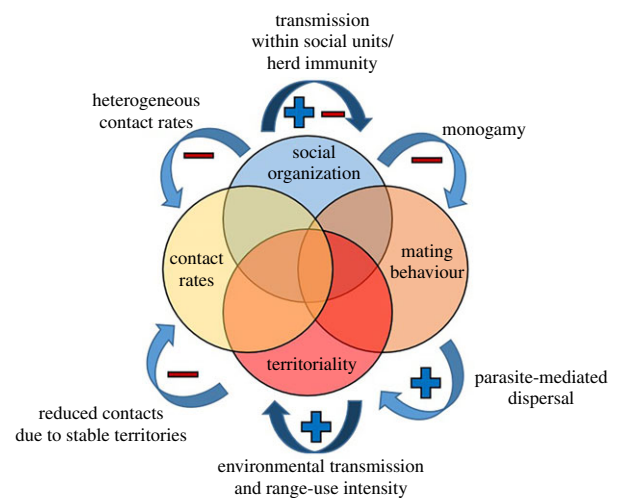


Figure 2. Venn diagram illustrating the overlap in processes of behavioural ecology that affect parasite transmission dynamics. Plus and minus signs indicate whether the behavioural mechanism facilitates or impedes parasite transmission. For example, social behaviours can have both positive (increased transmission within social units) and negative effects on parasite transmission (heterogeneous contact rates among individuals, modularity in social units/populations). The relationships between behavioural concepts demonstrate how variation in social and mating behaviour (e.g. monogamy), can decrease parasite transmission, while mate-seeking behaviours can increase transmission (e.g. via parasite-mediated dispersal).

finer insights into the use of different intervention strategies (e.g. [78]), such as in African carnivores suffering from canine distemper virus [75,98]. Another area requiring further research includes comparing multiple measures of infection together. In fishes, for example, parasite species richness was higher in migratory species than non-migratory, but prevalence and intensity did not differ [127]. Therefore, it is important to make clear predictions and interpretations in the light of the variables measured to understand how behavioural mechanisms might affect richness, prevalence and intensity. A significant gap remains to understand how natural host–parasite interactions should be integrated into conservation and management practices [125,128]. In closing, the links between parasites and host behaviour are important for theories of population, community and ecosystem ecology. An integrated theory of disease ecology and behaviour will ultimately help us forecast and manage the consequences of anthropogenic effects on ecosystem health.

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References

1. Betts A, Gray C, Zelek M, MacLean R, King K. 2018 High parasite diversity accelerates host adaptation and diversification. *Science* **360**, 907–911. (doi:10.1126/science.aam9974)
2. Ezenwa VO, Archie EA, Craft ME, Hawley DM, Martin LB, Moore J, White L. 2016 Host

- behaviour—parasite feedback: an essential link between animal behaviour and disease ecology. *Proc. R. Soc. B* **283**, 20153078. (doi:10.1098/rspb.2015.3078)
3. Ezenwa VO. 2004 Selective defecation and selective foraging: antiparasite behavior in wild ungulates? *Ethology* **110**, 851–862. (doi:10.1111/j.1439-0310.2004.01013.x)
 4. Bouwman KM, Hawley DM. 2010 Sickness behaviour acting as an evolutionary trap? Male house finches preferentially feed near diseased conspecifics. *Biol. Lett.* **6**, 462. (doi:10.1098/rsbl.2010.0020)
 5. Hawley DM, Altizer SM. 2011 Disease ecology meets ecological immunology: understanding the links between organismal immunity and infection dynamics in natural populations. *Funct. Ecol.* **25**, 48–60. (doi:10.1111/j.1365-2435.2010.01753.x)
 6. Loehle C. 1997 The pathogen transmission avoidance theory of sexual selection. *Ecol. Modell.* **103**, 231–250. (doi:10.1016/S0304-3800(97)00106-3)
 7. Lafferty KD, Morris AK. 1996 Altered behavior of parasitized killifish increases susceptibility to predation by bird final hosts. *Ecology* **77**, 1390–1397. (doi:10.2307/2265536)
 8. Sutherland WJ. 1996 *From individual behaviour to population ecology*. Oxford, UK: Oxford University Press.
 9. Wong B, Candolin U. 2015 Behavioral responses to changing environments. *Behav. Ecol.* **26**, 665–673. (doi:10.1093/beheco/aru183)
 10. Smith KF, Sax DF, Lafferty KD. 2006 Evidence for the role of infectious disease in species extinction and endangerment. *Conserv. Biol.* **20**, 1349–1357. (doi:10.1111/j.1523-1739.2006.00524.x)
 11. Pedersen AB, Jones KE, Nunn CL, Altizer S. 2007 Infectious diseases and extinction risk in wild mammals. *Conserv. Biol.* **21**, 1269–1279. (doi:10.1111/j.1523-1739.2007.00776.x)
 12. de Castro F, Bolker B. 2005 Mechanisms of disease-induced extinction. *Ecol. Lett.* **8**, 117–126. (doi:10.1111/j.1461-0248.2004.00693.x)
 13. Daszak P, Cunningham AA, Hyatt AD. 2000 Emerging infectious diseases of wildlife: threats to biodiversity and human health. *Science* **287**, 443–449. (doi:10.1126/science.287.5452.443)
 14. Anderson RM, May RM. 1978 Regulation and stability of host-parasite population interactions. I. Regulatory processes. *J. Anim. Ecol.* **47**, 219–247. (doi:10.2307/3933)
 15. Dobson AP, Hudson PJ. 1992 Regulation and stability of a free-living host-parasite system: *Trichostrongylus tenuis* in red grouse. II. Population models. *J. Anim. Ecol.* **61**, 487–498. (doi:10.2307/5339)
 16. Legendre S, Clobert J, Møller AP, Sorci G. 1999 Demographic stochasticity and social mating system in the process of extinction of small populations: the case of passerines introduced to New Zealand. *Am. Nat.* **153**, 449–463. (doi:10.1086/303195)
 17. Lande R, Engen S, Sæther B-E. 2003 *Stochastic population dynamics in ecology and conservation*. Oxford, UK: Oxford University Press.
 18. Sæther B-E, Engen S. 2019 Towards a predictive conservation biology: the devil is in the behaviour. *Phil. Trans. R. Soc. B* **374**, 20190013. (doi:10.1098/rstb.2019.0013)
 19. Engen S, Lande R, Sæther B-E. 2003 Demographic stochasticity and Allee effects in populations with two sexes. *Ecology* **84**, 2378–2386. (doi:10.1890/02-0123)
 20. Nunn CL, Altizer SM. 2006 *Infectious diseases in primates: behavior, ecology and evolution*. Oxford, UK: Oxford University Press.
 21. Begon M, Bennett M, Bowers RG, French NP, Hazel S, Turner J. 2002 A clarification of transmission terms in host-microparasite models: numbers, densities and areas. *Epidemiol. Infect.* **129**, 147–153. (doi:10.1017/S0950268802007148)
 22. Almberg ES, Cross PC, Dobson AP, Smith DW, Metz MC, Stahler DR, Hudson PJ. 2015 Social living mitigates the costs of a chronic illness in a cooperative carnivore. *Ecol. Lett.* **18**, 660–667. (doi:10.1111/ele.12444)
 23. Ezenwa VO. 2004 Host social behavior and parasitic infection: a multifactorial approach. *Behav. Ecol.* **15**, 446–454. (doi:10.1093/beheco/aru028)
 24. Caillaud D, Levêro F, Cristescu R, Gatti S, Dewas M, Douadi M, Gautier-Hion A, Raymond M, Ménard N. 2006 Gorilla susceptibility to Ebola virus: the cost of sociality. *Curr. Biol.* **16**, R489–R491. (doi:10.1016/j.cub.2006.06.017)
 25. Clay C, Lehmer EM, Stephen JM, Dearing MD. 2009 Sin Nombre virus and rodent species diversity: a test of the dilution and amplification hypotheses. *PLoS ONE* **4**, e6467. (doi:10.1371/journal.pone.0006467)
 26. Hamede R, Bashford J, Jones M, McCallum H. 2012 Simulating devil facial tumour disease outbreaks across empirically derived contact networks. *J. Appl. Ecol.* **49**, 447–456. (doi:10.1111/j.1365-2664.2011.02103.x)
 27. Hamede RK, Bashford J, McCallum H, Jones M. 2009 Contact networks in a wild Tasmanian devil (*Sarcophilus harrisii*) population: using social network analysis to reveal seasonal variability in social behaviour and its implications for transmission of devil facial tumour disease. *Ecol. Lett.* **12**, 1147–1157. (doi:10.1111/j.1461-0248.2009.01370.x)
 28. Rushmore J, Caillaud D, Hall RJ, Stumpf RM, Meyers LA, Altizer S. 2014 Network-based vaccination improves prospects for disease control in wild chimpanzees. *J. R. Soc. Interface* **11**, 20140349. (doi:10.1098/rsif.2014.0349)
 29. Hamilton WJ, Poulin R. 1997 The Hamilton and Zuk hypothesis revisited: a meta-analytical approach. *Behaviour* **134**, 299–320. (doi:10.1163/156853997X00485)
 30. Able DJ. 1996 The contagion indicator hypothesis for parasite-mediated sexual selection. *Proc. Natl Acad. Sci. USA* **93**, 2229–2233. (doi:10.1073/pnas.93.5.2229)
 31. Smith A, Clark P, Averis S, Lymbery A, Wayne A, Morris K, Thompson R. 2008 Trypanosomes in a declining species of threatened Australian marsupial, the brush-tailed bettong *Bettongia penicillata* (Marsupialia: Potoroidae). *Parasitology* **135**, 1329–1335. (doi:10.1017/S0031182008004824)
 32. Randall DA *et al.* 2004 Rabies in endangered Ethiopian wolves. *Emerg. Infect. Dis.* **10**, 2214. (doi:10.3201/eid1012.040080)
 33. Alexander RD. 1974 The evolution of social behavior. *Annu. Rev. Ecol. Syst.* **5**, 325–383. (doi:10.1146/annurev.es.05.110174.001545)
 34. Krause J, Ruxton GD. 2002 *Living in groups*. Oxford, UK: Oxford University Press.
 35. Freeland W. 1976 Pathogens and the evolution of primate sociality. *Biotropica* **8**, 12–24. (doi:10.2307/2387816)
 36. Nunn CL, Thrall PH, Leendertz FH, Boesch C. 2011 The spread of fecally transmitted parasites in socially-structured populations. *PLoS ONE* **6**, e21677. (doi:10.1371/journal.pone.0021677)
 37. Nunn CL, Lindenfors P, Pursall ER, Rolff J. 2008 On sexual dimorphism in immune function. *Phil. Trans. R. Soc. B* **364**, 20080148. (doi:10.1098/rstb.2008.0148)
 38. Lutz HL, Hochachka WM, Engel JL, Bell JA, Tkach VV, Bates JM, Hackett SJ, Weckstein JD. 2015 Parasite prevalence corresponds to host life history in a diverse assemblage of Afrotropical birds and haemosporidian parasites. *PLoS ONE* **10**, e0121254. (doi:10.1371/journal.pone.0121254)
 39. Gonzalez AD, Matta NE, Ellis VA, Miller ET, Rickles RE, Gutierrez HR. 2014 Mixed species flock, nest height, and elevation partially explain avian haemoparasite prevalence in Colombia. *PLoS ONE* **9**, e100695. (doi:10.1371/journal.pone.0100695)
 40. Nunn CL, Altizer S, Jones KE, Sechrest W. 2003 Comparative tests of parasite species richness in primates. *Am. Nat.* **162**, 597–614. (doi:10.1086/378721)
 41. Bordes F, Blumstein DT, Morand S. 2007 Rodent sociality and parasite diversity. *Biol. Lett.* **3**, 692–694. (doi:10.1098/rsbl.2007.0393)
 42. Rifkin JL, Nunn CL, Garamszegi LZ. 2012 Do animals living in larger groups experience greater parasitism? A meta-analysis. *Am. Nat.* **180**, 70–82. (doi:10.1086/666081)
 43. Patterson JEH, Ruckstuhl KE. 2013 Parasite infection and host group size: a meta-analytical review. *Parasitology* **140**, 803–813. (doi:10.1017/S0031182012002259)
 44. Ezenwa VO, Ghai RR, McKay AF, Williams AE. 2016 Group living and pathogen infection revisited. *Curr. Opin. Behav. Sci.* **12**, 66–72. (doi:10.1016/j.cobeha.2016.09.006)
 45. Kappeler PM, Cremer S, Nunn CL. 2015 Sociality and health: impacts of sociality on disease susceptibility and transmission in animal and human societies. *Phil. Trans. R. Soc. B* **370**, 20140116. (doi:10.1098/rstb.2014.0116)
 46. Uchino BN. 2006 Social support and health: a review of physiological processes potentially underlying links to disease outcomes. *J. Behav. Med.* **29**, 377–387. (doi:10.1007/s10865-006-9056-5)
 47. Holt-Lunstad J, Smith TB, Layton JB. 2010 Social relationships and mortality risk: a meta-analytic review. *PLoS Med.* **7**, e1000316. (doi:10.1371/journal.pmed.1000316)

48. Ezenwa VO, Worsley-Tonks KE. 2018 Social living simultaneously increases infection risk and decreases the cost of infection. *Proc. R. Soc. B* **285**, 20182142. (doi:10.1098/rspb.2018.2142)
49. Chapman CA, Saj TL, Snaith TV. 2007 Temporal dynamics of nutrition, parasitism, and stress in colobus monkeys: implications for population regulation and conservation. *Am. J. Phys. Anthropol.* **134**, 240–250. (doi:10.1002/ajpa.20664)
50. White LA, Forester JD, Craft ME. 2017 Using contact networks to explore mechanisms of parasite transmission in wildlife. *Biol. Rev.* **92**, 389–409. (doi:10.1111/brv.12236)
51. Woolhouse ME *et al.* 1997 Heterogeneities in the transmission of infectious agents: implications for the design of control programs. *Proc. Natl Acad. Sci. USA* **94**, 338–342. (doi:10.1073/pnas.94.1.338)
52. Silk MJ, Hodgson DJ, Rozins C, Croft DP, Delahay RJ, Boots M, McDonald RA. 2019 Integrating social behaviour, demography and disease dynamics in network models: applications to disease management in declining wildlife populations. *Phil. Trans. R. Soc. B* **374**, 20180211. (doi:10.1098/rstb.2018.0211)
53. Sah P, Leu ST, Cross PC, Hudson PJ, Bansal S. 2017 Unraveling the disease consequences and mechanisms of modular structure in animal social networks. *Proc. Natl Acad. Sci. USA* **114**, 4165–4170. (doi:10.1073/pnas.1613616114)
54. Farine DR. 2018 When to choose dynamic vs. static social network analysis. *J. Anim. Ecol.* **87**, 128–138. (doi:10.1111/1365-2656.12764)
55. Manlove K, Aiello C, Sah P, Cummins B, Hudson PJ, Cross PC. 2018 The ecology of movement and behaviour: a saturated tripartite network for describing animal contacts. *Proc. R. Soc. B* **285**, 20180670. (doi:10.1098/rspb.2018.0670)
56. Pinter-Wollman N *et al.* 2013 The dynamics of animal social networks: analytical, conceptual, and theoretical advances. *Behav. Ecol.* **25**, 242–255. (doi:10.1093/beheco/art047)
57. Griffin R, Nunn C. 2011 Community structure and the spread of infectious disease in primate social networks. *Evol. Ecol.* **26**, 1–22. (doi:10.1007/s10682-011-9526-2)
58. Nunn CL, Jordán F, McCabe CM, Verdolin JL, Fewell JH. 2015 Infectious disease and group size: more than just a numbers game. *Phil. Trans. R. Soc. B* **370**, 20140111. (doi:10.1098/rstb.2014.0111)
59. Rimbach R, Bisanzio D, Galvis N, Link A, Di Fiore A, Gillespie TR. 2015 Brown spider monkeys (*Ateles hybridus*): a model for differentiating the role of social networks and physical contact on parasite transmission dynamics. *Phil. Trans. R. Soc. B* **370**, 20140110. (doi:10.1098/rstb.2014.0110)
60. Sumner KM, McCabe CM, Nunn CL. 2018 Network size, structure, and pathogen transmission: a simulation study comparing different community detection algorithms. *Behaviour* **155**, 639–670. (doi:10.1163/1568539X-00003508)
61. Newman ME. 2006 Modularity and community structure in networks. *Proc. Natl Acad. Sci. USA* **103**, 8577–8582. (doi:10.1073/pnas.0601602103)
62. Salathé M, Jones JH. 2010 Dynamics and control of diseases in networks with community structure. *PLoS Comput. Biol.* **6**, e1000736. (doi:10.1371/journal.pcbi.1000736)
63. Pearce A-M, Swift K. 2006 Allograft theory: transmission of devil facial-tumour disease. *Nature* **439**, 549. (doi:10.1038/439549a)
64. McCallum H. 2008 Tasmanian devil facial tumour disease: lessons for conservation biology. *Trends Ecol. Evol.* **23**, 631–637. (doi:10.1016/j.tree.2008.07.001)
65. Collares-Pereira M, Mathias M, Santos-Reis M, Ramalhinho M, Duarte-Rodrigues P. 2000 Rodents and *Leptospira* transmission risk in Terceira island (Azores). *Eur. J. Epidemiol.* **16**, 1151–1157. (doi:10.1023/A:1010916132497)
66. Freeland WJ. 1979 Primate social-groups as biological islands. *Ecology* **60**, 719–728. (doi:10.2307/1936609)
67. Loehle C. 1995 Social barriers to pathogen transmission in wild animal populations. *Ecology* **76**, 326–335. (doi:10.2307/1941192)
68. Mitani JC, Rodman PS. 1979 Territoriality: the relation of ranging pattern and home range size to defendability, with an analysis of territoriality among primate species. *Behav. Ecol. Sociobiol.* **5**, 241–251. (doi:10.1007/BF00293673)
69. Lowen C, Dunbar R. 1994 Territory size and defendability in primates. *Behav. Ecol. Sociobiol.* **35**, 347–354. (doi:10.1007/BF00184423)
70. Nunn CL, Dokey AT-W. 2006 Ranging patterns and parasitism in primates. *Biol. Lett.* **2**, 351–354. (doi:10.1098/rsbl.2006.0485)
71. Gillespie TR, Chapman CA. 2006 Prediction of parasite infection dynamics in primate metapopulations based on attributes of forest fragmentation. *Conserv. Biol.* **20**, 441–448. (doi:10.1111/j.1523-1739.2006.00290.x)
72. Ezenwa VO, Price SA, Altizer S, Vitone ND, Cook KC. 2006 Host traits and parasite species richness in even and odd-toed hoofed mammals, Artiodactyla and Perissodactyla. *Oikos* **115**, 526–536. (doi:10.1111/j.2006.0030-1299.15186.x)
73. VanderWaal KL, Ezenwa VO. 2016 Heterogeneity in pathogen transmission: mechanisms and methodology. *Funct. Ecol.* **30**, 1606–1622. (doi:10.1111/1365-2435.12645)
74. Nunn CL, Scully EJ, Kutsukake N, Ostner J, Schülke O, Thrall PH. 2014 Mating competition, promiscuity, and life history traits as predictors of sexually transmitted disease risk in primates. *Int. J. Primatol.* **35**, 764–786. (doi:10.1007/s10764-014-9781-5)
75. Craft ME, Volz E, Packer C, Meyers LA. 2011 Disease transmission in territorial populations: the small-world network of Serengeti lions. *J. R. Soc. Interface* **8**, 776–786. (doi:10.1098/rsif.2010.0511)
76. Anderson RM, May RM. 1979 Population biology of infectious diseases. Part I. *Nature* **280**, 361–367. (doi:10.1038/280361a0)
77. Dobson A, Meagher M. 1996 The population dynamics of brucellosis in the Yellowstone National Park. *Ecology* **77**, 1026–1036. (doi:10.2307/2265573)
78. Wasserberg G, Osnas EE, Rolley RE, Samuel MD. 2009 Host culling as an adaptive management tool for chronic wasting disease in white-tailed deer: a modelling study. *J. Appl. Ecol.* **46**, 457–466. (doi:10.1111/j.1365-2664.2008.01576.x)
79. Carstensen M, DonCarlos MW. 2011 Preventing the establishment of a wildlife disease reservoir: a case study of bovine tuberculosis in wild deer in Minnesota, USA. *Vet. Med. Int.* **2011**, 1–10. (doi:10.4061/2011/413240)
80. Donnelly CA *et al.* 2006 Positive and negative effects of widespread badger culling on tuberculosis in cattle. *Nature* **439**, 843–846. (doi:10.1038/nature04454)
81. McDonald RA, Delahay RJ, Carter SP, Smith GC, Cheeseman CL. 2008 Perturbing implications of wildlife ecology for disease control. *Trends Ecol. Evol.* **23**, 53–56. (doi:10.1016/j.tree.2007.10.011)
82. Carter SP, Delahay RJ, Smith GC, Macdonald DW, Riordan P, Etherington TR, Pimley ER, Walker NJ, Cheeseman CL. 2007 Culling-induced social perturbation in Eurasian badgers *Meles meles* and the management of TB in cattle: an analysis of a critical problem in applied ecology. *Proc. R. Soc. B* **274**, 2769–2777. (doi:10.1098/rspb.2007.0998)
83. Prentice JC, Fox NJ, Hutchings MR, White PC, Davidson RS, Marion G. 2019 When to kill a cull: factors affecting the success of culling wildlife for disease control. *J. R. Soc. Interface* **16**, 20180901. (doi:10.1098/rsif.2018.0901)
84. Silk MJ, Weber NL, Steward LC, Hodgson DJ, Boots M, Croft DP, Delahay RJ, McDonald RA. 2018 Contact networks structured by sex underpin sex-specific epidemiology of infection. *Ecol. Lett.* **21**, 309–318. (doi:10.1111/ele.12898)
85. Walsh PD, Breuer T, Sanz C, Morgan D, Doran-Sheehy D. 2007 Potential for Ebola transmission between gorilla and chimpanzee social groups. *Am. Nat.* **169**, 684–689. (doi:10.1086/513494)
86. Leendertz SAJ, Wich SA, Ancrenaz M, Bergl RA, Gonder MK, Humle T, Leendertz FH. 2017 Ebola in great apes: current knowledge, possibilities for vaccination, and implications for conservation and human health. *Mamm. Rev.* **47**, 98–111. (doi:10.1111/mam.12082)
87. Leendertz FH, Pauli G, Maetz-Rensing K, Boardman W, Nunn C, Ellerbrok H, Jensen SA, Junglen S, Christophe B. 2006 Pathogens as drivers of population declines: the importance of systematic monitoring in great apes and other threatened mammals. *Biol. Conserv.* **131**, 325–337. (doi:10.1016/j.biocon.2006.05.002)
88. Vicente J, Höfle U, Garrido JM, Acevedo P, Juste R, Barral M, Gortázar C. 2007 Risk factors associated with the prevalence of tuberculosis-like lesions in fenced wild boar and red deer in south central Spain. *Vet. Res.* **38**, 451–464. (doi:10.1051/vetres:2007002)
89. Barasona J, Vicente J, Díez-Delgado I, Aznar J, Gortázar C, Torres M. 2017 Environmental presence of *Mycobacterium tuberculosis* complex in aggregation points at the wildlife/livestock interface. *Transboundary Emerging Dis.* **64**, 1148–1158. (doi:10.1111/tbed.12480)

90. Dudley JP, Hang'ombe BM, Leendertz FH, Dorward LJ, De Castro J, Subalaky AL, Clauss M. 2016 Carnivory in the common hippopotamus *Hippopotamus amphibius*: implications for the ecology and epidemiology of anthrax in African landscapes. *Mamm. Rev.* **46**, 191–203. (doi:10.1111/mam.12056)
91. Nunn CL, Thrall PH, Kappeler PM. 2014 Shared resources and disease dynamics in spatially structured populations. *Ecol. Modell.* **272**, 198–207. (doi:10.1016/j.ecolmodel.2013.10.004)
92. Dougherty ER, Seidel DP, Carlson CJ, Spiegel O, Getz WM. 2018 Going through the motions: incorporating movement analyses into disease research. *Ecol. Lett.* **21**, 588–604. (doi:10.1111/ele.12917)
93. Bonnell TR, Sengupta RR, Chapman CA, Goldberg TL. 2010 An agent-based model of red colobus resources and disease dynamics implicates key resource sites as hot spots of disease transmission. *Ecol. Modell.* **221**, 2491–2500. (doi:10.1016/j.ecolmodel.2010.07.020)
94. Hatcher MJ, Dick JT, Dunn AM. 2006 How parasites affect interactions between competitors and predators. *Ecol. Lett.* **9**, 1253–1271. (doi:10.1111/j.1461-0248.2006.00964.x)
95. Maia C, Dantas-Torres F, Campino L. 2018 Parasite biology: the reservoir hosts. In *The leishmaniasis: old neglected tropical diseases* (eds F Bruschi, L Gradoni), pp. 79–106. Cham, Switzerland: Springer International Publishing.
96. Power AG, Mitchell CE. 2004 Pathogen spillover in disease epidemics. *Am. Nat.* **164**, S79–S89. (doi:10.1086/424610)
97. Beyer W, Turnbull P. 2009 Anthrax in animals. *Mol. Aspects Med.* **30**, 481–489. (doi:10.1016/j.mam.2009.08.004)
98. Craft M, Hawthorne P, Packer C, Dobson A. 2008 Dynamics of a multihost pathogen in a carnivore community. *J. Anim. Ecol.* **77**, 1257–1264. (doi:10.1111/j.1365-2656.2008.01410.x)
99. Hart BL. 1990 Behavioral adaptations to pathogens and parasites: five strategies. *Neurosci. Biobehav. Rev.* **14**, 273–294. (doi:10.1016/S0149-7634(05)80038-7)
100. Adelman JS, Mayer C, Hawley DM. 2017 Infection reduces anti-predator behaviors in house finches. *J. Avian Biol.* **48**, 519–528. (doi:10.1111/jav.01058)
101. Lopes PC, Block P, König B. 2016 Infection-induced behavioural changes reduce connectivity and the potential for disease spread in wild mice contact networks. *Sci. Rep.* **6**, 31790. (doi:10.1038/srep31790)
102. Franz M, Kramer-Schadt S, Greenwood AD, Courtiol A. 2018 Sickness-induced lethargy can increase host contact rates and pathogen spread in water-limited landscapes. *Funct. Ecol.* **32**, 2194–2204. (doi:10.1111/1365-2435.13149)
103. Hamilton WD, Zuk M. 1982 Heritable true fitness and bright birds: a role for parasites? *Science* **218**, 384–387. (doi:10.1126/science.7123238)
104. Ehman K, Scott M. 2002 Female mice mate preferentially with non-parasitized males. *Parasitology* **125**, 461–466. (doi:10.1017/S003118200200224X)
105. Clayton DH. 1990 Mate choice in experimentally parasitized rock doves: lousy males lose. *Am. Zool.* **30**, 251–262. (doi:10.1093/icb/30.2.251)
106. Wells K, Hamede RK, Kerlin DH, Storfer A, Hohenlohe PA, Jones ME, McCallum HI. 2017 Infection of the fittest: devil facial tumour disease has greatest effect on individuals with highest reproductive output. *Ecol. Lett.* **20**, 770–778. (doi:10.1111/ele.12776)
107. Lee AM, Sæther B-E, Engen S. 2011 Demographic stochasticity, Allee effects, and extinction: the influence of mating system and sex ratio. *Am. Nat.* **177**, 301–313. (doi:10.1086/658344)
108. Wedekind C. 2002 Sexual selection and life-history decisions: implications for supportive breeding and the management of captive populations. *Conserv. Biol.* **16**, 1204–1211. (doi:10.1046/j.1523-1739.2002.01217.x)
109. Nunn CL, Gittleman JL, Antonovics J. 2000 Promiscuity and the primate immune system. *Science* **290**, 1168–1170. (doi:10.1126/science.290.5494.1168)
110. Altizer S *et al.* 2003 Social organization and parasite risk in mammals: integrating theory and empirical studies. *Annu. Rev. Ecol. Evol. Syst.* **34**, 517–547. (doi:10.1146/annurev.ecolsys.34.030102.151725)
111. Thrall PH, Antonovics J, Bever JD. 1997 Sexual transmission of disease and host mating systems: within-season reproductive success. *Am. Nat.* **149**, 485–506. (doi:10.1086/286001)
112. Kokko H, Ranta E, Ruxton G, Lundberg P. 2002 Sexually transmitted disease and the evolution of mating systems. *Evolution* **56**, 1091–1100. (doi:10.1111/j.0014-3820.2002.tb01423.x)
113. Moore J. 2002 *Parasites and the behavior of animals*. Oxford, UK: Oxford University Press.
114. Klein SL. 2003 Parasite manipulation of the proximate mechanisms that mediate social behavior in vertebrates. *Physiol. Behav.* **79**, 441–449. (doi:10.1016/S0031-9384(03)00163-X)
115. Fooks AR, Banyard AC, Horton DL, Johnson N, McElhinney LM, Jackson AC. 2014 Current status of rabies and prospects for elimination. *Lancet* **384**, 1389–1399. (doi:10.1016/S0140-6736(13)62707-5)
116. Sterner RT, Smith GC. 2006 Modelling wildlife rabies: transmission, economics, and conservation. *Biol. Conserv.* **131**, 163–179. (doi:10.1016/j.biocon.2006.05.004)
117. Lockhart AB, Thrall PH, Antonovics J. 1996 Sexually transmitted diseases in animals: ecological and evolutionary implications. *Biol. Rev.* **71**, 415–471. (doi:10.1111/j.1469-185X.1996.tb01281.x)
118. Knauf S *et al.* 2012 *Treponema* infection associated with genital ulceration in wild baboons. *Vet. Pathol.* **49**, 292–303. (doi:10.1177/0300985811402839)
119. Polkinghorne A, Hanger J, Timms P. 2013 Recent advances in understanding the biology, epidemiology and control of chlamydial infections in koalas. *Vet. Microbiol.* **165**, 214–223. (doi:10.1016/j.vetmic.2013.02.026)
120. McColl K. 1984 *Chlamydia* infection and infertility in the female koala (*Phascogale cinereus*). *Vet. Rec.* **115**, 655. (doi:10.1136/vr.115.25-26.655)
121. Tenter AM, Heckeroth AR, Weiss LM. 2000 *Toxoplasma gondii*: from animals to humans. *Int. J. Parasitol.* **30**, 1217–1258. (doi:10.1016/S0020-7519(00)00124-7)
122. Berdoy M, Webster JP, Macdonald DW. 2000 Fatal attraction in rats infected with *Toxoplasma gondii*. *Proc. R. Soc. Lond. B* **267**, 1591–1594. (doi:10.1098/rspb.2000.1182)
123. Hudson PJ, Dobson AP, Cattadori IM, Newborn D, Haydon DT, Shaw DJ, Benton TG, Grenfell BT. 2002 Trophic interactions and population growth rates: describing patterns and identifying mechanisms. *Phil. Trans. R. Soc. Lond. B* **357**, 1259–1271. (doi:10.1098/rstb.2002.1126)
124. Lafferty KD *et al.* 2008 Parasites in food webs: the ultimate missing links. *Ecol. Lett.* **11**, 533–546. (doi:10.1111/j.1461-0248.2008.01174.x)
125. Dougherty ER, Carlson CJ, Bueno VM, Burgio KR, Cizauskas CA, Clements CF, Seidel DP, Harris NC. 2016 Paradigms for parasite conservation. *Conserv. Biol.* **30**, 724–733. (doi:10.1111/cobi.12634)
126. Michalska-Smith MJ, Sander EL, Pascual M, Allesina S. 2018 Understanding the role of parasites in food webs using the group model. *J. Anim. Ecol.* **87**, 790–800. (doi:10.1111/1365-2656.12782)
127. Shaw AK, Sherman J, Barker FK, Zuk M. 2018 Metrics matter: the effect of parasite richness, intensity and prevalence on the evolution of host migration. *Proc. R. Soc. B* **285**, 20182147. (doi:10.1098/rspb.2018.2147)
128. Lyles AM, Dobson AP. 1993 Infectious disease and intensive management: population dynamics, threatened hosts, and their parasites. *J. Zoo Wildl. Med.* **24**, 315–326.