Parasitology

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Bidirectional interactions between host social behaviour and parasites arise through ecological and evolutionary processes

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Review

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Abstract

An animal's social behaviour both influences and changes in response to its parasites. Here we consider these bidirectional links between host social behaviours and parasite infection, both those that occur from ecological *vs* evolutionary processes. First, we review how social behaviours of individuals and groups influence ecological patterns of parasite transmission. We then discuss how parasite infection, in turn, can alter host social interactions by changing the behaviour of both infected and uninfected individuals. Together, these ecological feedbacks between social behaviour and parasite infection can result in important epidemiological consequences. Next, we consider the ways in which host social behaviours evolve in response to parasites, highlighting constraints that arise from the need for hosts to maintain benefits of sociality while minimizing fitness costs of parasites. Finally, we consider how host social behaviours shape the population genetic structure of parasites and the evolution of key parasite traits, such as virulence. Overall, these bidirectional relationships between host social behaviours and parasites are an important yet often underappreciated component of population-level disease dynamics and host–parasite coevolution.

Introduction

Social behaviours, which serve key roles in parasite transmission, can both influence and respond to parasite infection through ecological and evolutionary processes (Fig. 1; Ezenwa *et al.*, 2016*a*). While past work has documented diverse ways in which an animal's social behaviours influence parasite ecology (Fig. 1A), the ability of parasites to, in turn, alter host social behaviours *via* ecological (Fig. 1B) or evolutionary (Fig. 1C) processes has been understudied relative to predators, the other major class of natural enemy (Krause and Ruxton, 2002). Further, the role of host social behaviours in driving the evolution of parasite traits (Fig. 1D) such as virulence and host range has received surprisingly little attention (Schmid-Hempel, 2017). Given the importance of social behaviours for the transmission, and thus fitness, of diverse types of parasites, understanding the ways in which parasites and host social behaviours interact is critical for predicting both parasite evolution (Schmid-Hempel, 2017), and disease dynamics at population scales (Ezenwa *et al.*, 2016*a*).

Here we consider the key bidirectional interactions, both ecological and evolutionary, that occur between parasites and host social behaviours, which we define broadly as any direct behavioural interaction between conspecifics (Box 1). Work to date has shown that host social behaviours can be important yet complex drivers of parasite risk through ecological processes (Fig. 1; Arrow A; Altizer *et al.*, 2003; Schmid-Hempel, 2017). For example, social behaviours such as gregariousness (Box 1) can increase the probability or extent of parasitism by bringing hosts into close proximity (Rifkin *et al.*, 2012), but gregariousness can also augment the ability of hosts to resist or tolerate parasites and pathogens once exposed (Ezenwa *et al.*, 2016*b*). Parasite infection, in turn, can have reciprocal and far-reaching ecological effects on animal social behaviours (Arrow B), both by altering the social behaviours of infected hosts (e.g. Lopes *et al.*, 2016) and, in some cases, the uninfected conspecifics with which they interact (e.g. Behringer *et al.*, 2006). In addition to these ecological processes, parasites can influence animal social behaviours *via* evolutionary mechanisms (Arrow C) by driving selection on group size and avoidance behaviours that help to ameliorate the costs associated with a heightened risk of parasitism for highly social individuals (e.g. Loehle, 1995; Buck *et al.*, 2018). Finally, social behaviours of hosts are predicted to exert strong selection on traits of parasites (Arrow D) given the importance of these host behaviours for parasite fitness (i.e. spread and long-term persistence). Thus, we end by considering how host social behaviours might shape the genetic structure of parasite populations and the evolution of parasite traits (Arrow D).

Given the vast literature on host social behaviours and parasites, we do not attempt an exhaustive review, but instead selectively synthesize key concepts in the field and exciting

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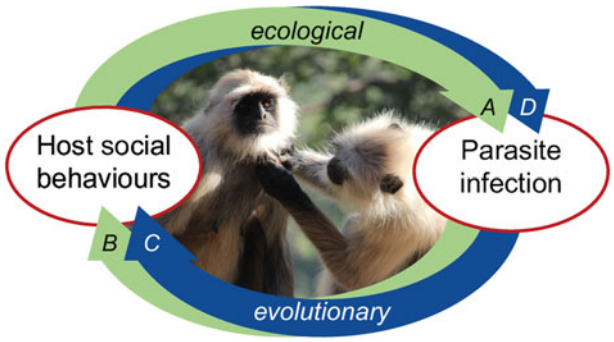


Fig. 1. Host social behaviours influence and respond to parasites *via* both ecological (light green arrows) and evolutionary (dark blue arrows) processes. In terms of *ecological processes*, social behaviours such as allogrooming can influence exposure and physiological responses to parasites (A). In turn, parasite infection can alter social behaviours of actively infected hosts and their uninfected conspecifics (e.g. allogrooming given or received) (B). In terms of *evolutionary processes*, parasites can shape the evolution of group size and relative investment in parasite avoidance behaviours such as allogrooming (C). Host social behaviours such as allogrooming can also exert selection on parasite traits like virulence by altering host connectedness (D). Inset picture: Gray langur (*Semnopithecus* sp.): https://commons.wikimedia.org/wiki/File:Monkeys_Grooming.jpg

new findings or perspectives. We structure our review by considering ecological and evolutionary processes independently, but we note that these processes will show considerable overlap and feedback. Importantly, ecological processes for hosts often occur on timescales relevant for parasite evolution. Thus, we end our review with a brief discussion of ecological-evolutionary feedbacks between host social behaviours and parasites. We limit the taxonomic scope of our review to animal hosts, but by defining social behaviours broadly, we discuss concepts and examples that apply to taxa exhibiting a wide degree of sociality (Box 1). Finally, although the COVID-19 pandemic underscores the importance of reciprocal interactions between social behaviours and parasites in humans (e.g. Block *et al.*, 2020), we focus our review on non-human animals for brevity, while recognizing that the concepts discussed here can be extended to all social taxa and their parasites (e.g. Townsend *et al.*, 2020).

Box 1. Glossary of terms commonly used throughout the paper (note that this list is not exhaustive but includes terms for which definitions sometimes vary across contexts).

Gregariousness/sociality: Used interchangeably to describe the tendency to associate with conspecifics in social groups. The temporal stability of group associations can be highly variable across taxa.

Infection intensity: The number of parasites of a certain type in a single infected host.

Modularity: The degree of substructuring or subdivisions within and among social groups in a given interaction network.

Parasite/pathogen: Used interchangeably to represent organisms that live on or within hosts, deriving benefit while reducing the fitness of their hosts.

Social behaviour: Defined here broadly as behavioural interactions that occur among conspecifics and vary in duration (Blumstein *et al.*, 2010). These interactions can be 'negative' (e.g. aggression, avoidance) or 'positive' (e.g. allogrooming, affiliation) in nature (Hofmann *et al.*, 2014), and can occur within or outside the context of discrete social groups. For brevity, we do not discuss mating behaviours in this paper, although they fall within the scope of our definition.

Socially transmitted parasite: Used here to encompass parasitic taxa that spread *via* close contact between host conspecifics over space or time. For our purposes, this includes several types of horizontal transmission (defined broadly as that occurring within a generation): direct contact (touching, biting, etc.), airborne (respiratory) and two indirect modes: fomite (spread *via* surfaces) and environmental, which includes fecal-oral spread (as per Antonovics *et al.*, 2017). For brevity, we do not discuss sexual horizontal transmission.

Susceptibility/Resistance: Used interchangeably to represent a host's physiological ability ('resistance') or lack thereof ('susceptibility') to prevent or eliminate infection by parasites or pathogens.

Tolerance: The ability of hosts to reduce the fitness costs of a given parasite load.

Virulence: The degree of harm that a parasite causes its host, typically measured as reductions in host fitness.

Ecology: social behaviours influence and respond to parasite infection

Social behaviours, which by definition bring conspecifics into close proximity, have long been recognized as particularly likely to influence and respond to parasite spread (e.g. Alexander, 1974; Loehle, 1995). In this section, we consider both how social behaviours alter parasite transmission (Arrow A; Fig. 1), and in turn, how parasite infection can dynamically alter host social behaviours (Arrow B). Although it has long been recognized that parasites can alter animal behaviour (reviewed in Moore, 2002), the extent to which parasites influence the social dynamics of hosts *via* ecological processes, and the degree of individual heterogeneity in infection-induced changes in sociality, are only beginning to be uncovered. We focus on this exciting growing area, highlighting potential sources of heterogeneity in parasite-mediated changes in host social behaviours (Fig. 2), and their consequences for epidemiological and coevolutionary feedbacks (Ezenwa *et al.*, 2016*a*).

Host social behaviours alter parasite ecology (Arrow A)

Parasites spread *via* close contact between conspecifics over time or space (which we term 'socially transmitted parasites' hereafter for simplicity; Box 1) are hypothesized to pose a greater risk for host species that exhibit social behaviours such as group living (Krause and Ruxton, 2002). Classic mathematical models for socially transmitted parasites [e.g. susceptible-infectious-recovered (SIR) compartmental models] often assume that the rate of contact between susceptible and infectious individuals increases with host density (Begon *et al.*, 2002). On a local scale, this results in higher contact rates, and thus parasite transmission, for animals in larger social groups. Indeed, two meta-analyses support the hypothesis that larger social groups generally harbour higher prevalence and/or infection intensity (Box 1) of parasites spanning diverse transmission modes (Rifkin *et al.*, 2012; Patterson and Ruckstuhl, 2013). In contrast, however, there is some evidence that group living can dilute host risk of infection with highly mobile parasites by reducing *per capita* attack rates (the encounter-dilution effect; Côté and Poulin, 1995). The encounter-dilution effect primarily applies to parasites that actively seek hosts by flying or swimming; the likelihood of being singled out by these parasites can decrease with increasing group size (Côté and Poulin, 1995; Patterson and Ruckstuhl, 2013).

Recent work suggests that social group substructure may in some cases be equally or more important than group size in predicting parasite risk (Griffin and Nunn, 2012; Nunn *et al.*, 2015; Sah *et al.*, 2018). If the majority of close social interactions in large groups occur between subsets of individuals (e.g. 'cliques'), this modularity (Box 1) can act as a 'social bottleneck' that contains parasite spread within subgroups and reduces spread to the group at large (e.g. Nunn *et al.*, 2015). In support of this idea, the social networks of eusocial insect colonies can be highly structurally subdivided, and epidemiological models show that this

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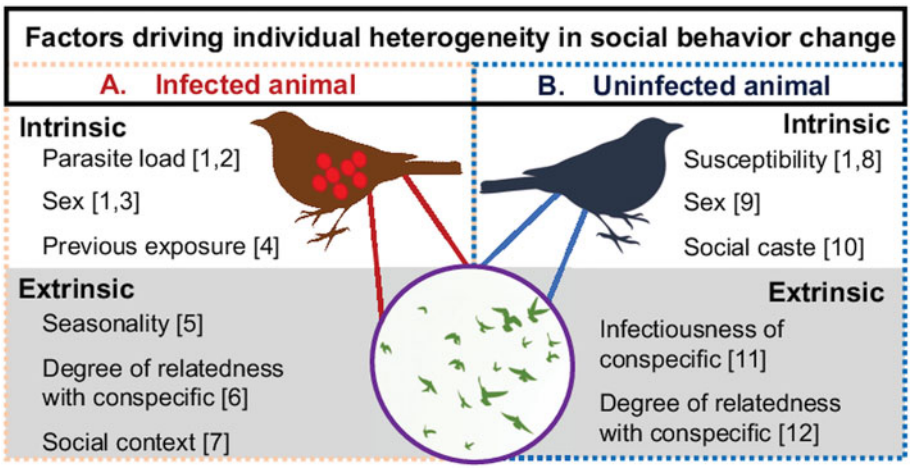


Fig. 2. Factors both intrinsic and extrinsic to individuals underlie heterogeneity in the extent to which hosts alter social behaviours in the face of infection. Here we list factors that have thus far been shown to influence the degree of parasite-induced social behaviour changes for infected (A) or uninfected (B) hosts, with representative references. While parasite manipulation can also alter social behaviours of infected hosts (A), here we focus solely on behavioural changes hypothesized to be host-mediated. [1] Stephenson (2019); [2] Houde and Torio (1992); [3] Siva-Jothy and Vale (2019); [4] Walker and Hughes (2009); [5] Owen-Ashley and Wingfield (2006); [6] Stockmaier *et al.* (2020b); [7] Lopes *et al.* (2012); [8] Zylberberg *et al.* (2012); [9] Bouwman and Hawley (2010); [10] Stroeymeyt *et al.* (2018); [11] Stephenson *et al.* (2018); [12] Poirotte and Charpentier (2020).

constitutive modularity dampens the transmission of an entomopathogenic fungus within colonies (Stroeymeyt *et al.*, 2018). Similarly, a comparative study of 19 non-human primate species found that higher levels of modularity may help ameliorate the heightened risk of parasite spread in large social groups, as higher modularity was associated with lower parasite richness (Griffin and Nunn, 2012). However, perhaps because of its protective function, social group modularity tends to increase with group size across taxa (Nunn *et al.*, 2015), making it challenging to tease apart whether resulting patterns of parasitism are a function of group size, modularity or both.

Individual variation in social behaviours can also have important effects on transmission risk. As shown through descriptive network approaches that quantify social connections among conspecifics using direct behavioural interactions or physical proximity, individuals that have ties to multiple social 'cliques' (VanderWaal *et al.*, 2016) or those highly connected to neighbouring conspecifics (e.g. Bull *et al.*, 2012) can have an increased likelihood of parasite infection (but see Drewe, 2010 for the importance of type and directionality of interactions). Similarly, bold or 'pro-active' personality traits, which correlate with social network centrality in some taxa (e.g. Aplin *et al.*, 2013), may influence social parasite transmission: two studies of mammalian species found that bolder individuals had a higher seroprevalence of viruses largely spread *via* aggressive interactions (Natoli *et al.*, 2005; Dizney and Dearing, 2013). While these correlational studies suggest the effects of variation in social behaviour on parasite risk, field studies generally cannot directly elucidate cause and effect (Arrow A *vs* B: does behaviour affect parasites or *vice versa*?). Further, it is challenging to disentangle the relative contributions of individual variation in exposure *vs* susceptibility to field patterns of transmission [VanderWaal and Ezenwa, 2016; see 'Synthesis: ecological feedbacks between social behaviours and parasite infection' section], particularly when traits relevant for both exposure and susceptibility can simultaneously be influenced by social context (e.g. Müller-Klein *et al.*, 2019). Experimental studies, while not possible for all host–parasite systems, can isolate the effects of host social behaviour *per se* on parasite transmission risk. For example, Keiser *et al.* (2016) used experimental epidemics to show that bolder female social spiders (*Stegodyphus dumicola*) had a higher risk of acquiring a cuticular microbe. Future studies could examine how individual differences in 'social personalities', which are seldom quantified in themselves (e.g. Kulahci *et al.*, 2018), influence the transmission dynamics of socially transmitted parasites.

Overall, the social behaviours of groups and individuals appear to strongly influence parasite transmission risk (Arrow A). However, in order to fully elucidate the effects of social behaviours on parasite transmission, it is critical to also consider how parasite infection affects host social behaviours (Arrow B), as both processes together will ultimately underlie the dynamics of socially transmitted parasites.

Parasite infection influences host social behaviours (Arrow B)

The way in which parasite infection alters the social behaviours of both infected hosts and their uninfected conspecifics (Arrow B) has received relatively less attention than the effects of social behaviours on parasite risk (Arrow A)]. This is somewhat surprising given that it has long been recognized that hosts often behave differently during infection (reviewed in Moore, 2002). Changes in social behaviours during infection can broadly result from parasite-mediated manipulation of host behaviours to promote transmission to new hosts (reviewed in Klein, 2003), or from host-mediated behavioural changes, which typically occur from one of three mechanisms: (1) as side-effects of tissue damage or energy needs associated with infection, (2) *via* expression of 'sickness behaviours' that are part of a host's broader, adaptive immunological responses to infection (Hart, 1988), or (3) as active self-isolation to prevent ongoing spread, a behaviour largely seen in eusocial insects (Shorter and Rueppell, 2012). All four possibilities, whether parasite- or host-mediated, can lead to notable changes in social behaviours of hosts, with important consequences for parasite transmission. For example, three-spined sticklebacks (*Gasterosteus aculeatus*) infected with the socially transmitted parasite *Glugea anomala* are more likely than their uninfected counterparts to be attracted to conspecifics, a behaviour predicted to augment transmission (Petkova *et al.*, 2018). Whether behavioural changes in that system are parasite- or host-mediated remains unclear, but in this section, we focus on changes in behaviour during infection that are likely host-mediated, and consider parasite-mediated behavioural changes in 'Host social behaviours influence parasite evolution (Arrow D)' section.

Host-mediated changes in behaviour during infection, such as self-isolation and sickness behaviours, often reduce the degree of interaction with conspecifics and thus the spread of socially transmitted parasites. While active self-isolation is rare outside of eusocial insects, sickness behaviours are a conserved component of vertebrate immune responses that include general reductions in activity levels and specific reductions in non-essential activities (Hart, 1988), such as many forms of social interaction (e.g. allogrooming). For example, Lopes *et al.* (2016) stimulated sickness behaviours in wild house mice (*Mus musculus domesticus*) by injecting individuals with bacterial endotoxin, and found that immune activation resulted in lower activity levels and fewer direct social interactions with conspecifics relative to controls. Similarly,

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work in two other mammalian systems found that infected individuals (or those expressing sickness behaviours) are less likely than control individuals to engage in affiliative allogrooming with conspecifics [banded mongooses (*Mungos mungo*), Fairbanks *et al.*, 2014; vampire bats (*Desmodus rotundus*), Stockmaier *et al.*, 2018]. In vampire bats, these changes in allogrooming during sickness behaviour expression, potentially in combination with reduced contact calling (Stockmaier *et al.*, 2020*a*), result in significant reductions in several measures of social connectedness relative to controls (Ripperger *et al.*, 2020). Overall, host-mediated reductions in social interactions during infection, particularly when they occur during the host's infectious period, likely reduce transmission of socially transmitted parasites.

The extent to which infected hosts alter their social behaviour is likely to depend on the energetic costs of a given parasite infection and the importance of that social behaviour for maintaining host fitness (Ezenwa *et al.*, 2016*b*). In some systems, social behaviours of hosts appear to be maintained during infection (Powell *et al.*, 2020), which may be common for infections by low-virulence parasites. In other cases, infected animals may maintain a subset of social interactions potentially most important to host recovery, including those with high inclusive fitness benefits. For example, vampire bats injected with endotoxin to induce sickness behaviours continued to groom close kin (mother or offspring) at levels similar to controls, but reduced the extent to which they groomed non-kin (Stockmaier *et al.*, 2020*b*). In some systems, social behaviours of hosts can even be augmented during infection. For example, male guppies (*Poecilia reticulata*) with high loads of a socially transmitted ectoparasite showed higher sociality relative to males with lower parasite loads (Stephenson, 2019), and rhesus monkeys (*Macaca mulatta*) given low-dose endotoxin injection show marked increases in social behaviours with conspecifics (Willette *et al.*, 2007). The ultimate mechanisms underlying these patterns remain unknown, but in some systems, the maintenance or even augmentation of sociality during infection may be a form of tolerance (Box 1), allowing hosts to minimize the fitness impacts of infection *via* group living (Ezenwa *et al.*, 2016*b*). For example, recent work in Grant's gazelle (*Nanger granti*) suggests that association with larger groups benefits gazelle infected with gastrointestinal parasites by allowing them to better ameliorate the costs associated with infection-induced anorexia (Ezenwa and Worsley-Tonks, 2018). Given that infected hosts experience anorexia (e.g. Adelman *et al.*, 2013) and higher predation risk (e.g. Alzaga *et al.*, 2008; Stephenson *et al.*, 2016) in many social taxa, future work should examine whether enhanced gregariousness during infection is a common mechanism of tolerance across taxa, with important consequences for ecological feedbacks between social behaviour and parasite transmission.

Parasite infection can also alter social interactions by changing the behaviour of uninfected hosts towards their infected conspecifics. Among taxa spanning fish, birds, crustaceans, social insects and mammals, infected or immune-activated individuals display visual cues of infection (e.g. lethargy: Zylberberg *et al.*, 2012) or release distinct chemical cues that conspecifics can use to avoid them (e.g. Arakawa *et al.*, 2009; Anderson and Behringer, 2013; Stephenson and Reynolds, 2016) or, in the case of honey bees (*Apis mellifera*), remove them from the colony (Baracchi *et al.*, 2012). Intriguingly, recent work in mice suggests that the scent of uninfected hosts themselves can change when they are housed with an infected conspecific (Gervasi *et al.*, 2018), suggesting the potential for complex downstream effects of infection status on social group dynamics and resulting transmission.

In some highly social animals, uninfected groupmates continue to engage in intimate interactions such as allogrooming with conspecifics that are infected or expressing sickness behaviours. At the extreme are some eusocial insects, where individuals care for infected conspecifics, likely because their high degree of relatedness favours the evolution of seemingly 'altruistic' behaviours *via* kin selection [see 'Parasites and the evolution of host social behaviour (Arrow C)' section]. But even in systems where groupmates are not as closely related, uninfected individuals often maintain intimate social interactions with infected conspecifics. For example, uninfected conspecifics in two social mammals groom visibly diseased groupmates or those expressing sickness behaviours at a similar intensity to controls, even when allogrooming reciprocity from these individuals is greatly reduced (e.g. mongooses: Fairbanks *et al.*, 2014; vampire bats: Stockmaier *et al.*, 2018); furthermore, uninfected vampire bats continue to share food with conspecifics expressing sickness behaviours (Stockmaier *et al.*, 2020*b*). In mandrills (*Mandrillus sphinx*), the degree to which uninfected individuals maintain social interactions with infected conspecifics appears to depend on kinship: mandrills reduce grooming towards parasitized partners that are non-kin, but maintain grooming if these potentially contagious partners are offspring or close maternal kin (Poirotte and Charpentier, 2020). Finally, in other systems, uninfected conspecifics are attracted to feed near (male house finches, *Haemorhous mexicanus*: Bouwman and Hawley, 2010) or socially explore (mice: Edwards, 1988) infected conspecifics. Understanding heterogeneity in the behaviour of uninfected hosts towards infected conspecifics (Fig. 2B), which can vary from avoidance to attraction, will help predict the conditions in which parasite-induced changes in sociality lead to positive or negative ecological feedbacks that ultimately maintain or dampen parasite epidemics (Fig. 1).

The effects of infection on social interactions between groups are also key to understanding pathogen transmission dynamics (Cross *et al.*, 2005), but have generally received less attention than within-group social interactions. Because infected individuals or those expressing sickness behaviours are less likely to explore their surroundings than uninfected individuals (e.g. Lopes *et al.*, 2016), they may be less likely to interact with other social groups, either temporarily or permanently (as occurs in banded mongooses; Fairbanks *et al.*, 2014). In other cases, infected individuals may be more likely to leave an existing group, as has been observed among European badgers (*Meles meles meles*) with bovine tuberculosis (Cheesman and Mallinson, 1981; Weber *et al.*, 2013). Whether infected individuals join new social groups, either temporarily or permanently, will also depend on whether infected individuals are 'accepted' by conspecifics in the new social group (Butler and Roper, 1996). Uninfected guppies appear to largely prevent the integration of experimental intruders with ectoparasite infections into existing shoals (Croft *et al.*, 2011). In contrast, honey bee colonies were more likely to accept entry by foreign bees infected with Israeli acute paralysis virus than foreign controls, which may represent a unique case of pathogen manipulation of chemical signals that mediate aggressive interactions in this species [Geffre *et al.*, 2020; see 'Host social behaviours influence parasite evolution (Arrow D)' section]. The movement or dispersal of uninfected individuals between groups can also be driven by conspecific infection or disease status, as occurs in western lowland gorillas (*Gorilla gorilla gorilla*), where adult females are more likely to emigrate from social groups with a higher prevalence of facial lesions associated with a contact-transmitted skin disease (Baudouin *et al.*, 2019). Overall, more studies are needed on how parasite infection influences among-group movements for both infected hosts and uninfected conspecifics, particularly for taxa where social group composition is relatively fluid, such as fission–fusion societies.

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Studies have only recently begun to address how changes in social behaviours of both infected and uninfected conspecifics scale up to influence host social networks and disease dynamics. Chapman *et al.* (2016), for example, used a deworming approach to examine how parasite infection in vervet monkeys (*Chlorocebus pygerythrus*) influenced social interactions in ways relevant to population-level spread. Dewormed individuals (particularly juveniles) had more frequent social interactions with more total conspecifics, suggesting that uninfected individuals may generally be more central in vervet monkey social networks, thereby attenuating parasite spread. Likewise, two recent studies combined experimental manipulations of infection status or sickness behaviour with network modelling to examine how parasite infection might influence the dynamics of socially transmitted pathogens (Lopes *et al.*, 2016; Stroeymeyt *et al.*, 2018). Lopes *et al.* (2016) used empirical contact data from mice induced to express sickness behaviours to simulate disease outbreaks across social networks, showing that changes in social interactions associated with sickness behaviours resulted in highly attenuated disease outbreaks. Although Lopes *et al.* (2016) did not find evidence of conspecific avoidance in their system, recent work in *Lasius niger* ants showed that responses of both parasite-contaminated ants and their uncontaminated nestmates contributed together to changes in group social networks that inhibited the spread of pathogens through colonies (Stroeymeyt *et al.*, 2018). Thus, understanding the behaviour of both infected hosts and the uninfected conspecifics they interact with is key for elucidating ecological feedbacks that dampen or augment disease spread within and among social groups.

Synthesis: ecological feedbacks between social behaviours and parasite infection

The bidirectional feedbacks between host social behaviours and parasite infection make it challenging to determine whether ecological patterns such as group size–parasitism relationships ['Host social behaviours alter parasite ecology (Arrow A)' section] result from the effect of social interactions on parasite risk (Arrow A), the effect of infection on social behaviours (Arrow B) or both. Experimental manipulation of parasite infection allows direct elucidation of causality. For example, Ezenwa and Worsley-Tonks (2018) treated a subset of Grant's gazelles with anti-helminthic drugs and found that individuals in larger social groups re-acquired gastrointestinal parasites more rapidly, supporting the idea that larger group sizes augment the risk of acquiring parasites (Arrow A). Because they also found that parasitized gazelle benefit from larger group sizes where they can spend more time foraging [see 'Parasite infection influences host social behaviours (Arrow B)' section], parasitized Grant's gazelles may actively seek out larger social groups (Arrow B), further contributing to patterns of higher parasite prevalence in larger groups. Although such changes in sociality with parasitism have not yet been explicitly examined in this system, the ability of gregariousness to augment host tolerance of infection may produce positive feedbacks between infection and social behaviour, facilitating longer persistence of parasite loads in larger groups.

The strength of ecological feedbacks between social behaviour and infection will be influenced by the degree of heterogeneity in the behaviour of both infected and uninfected hosts (Fig. 2), as well as the way in which behavioural heterogeneity covaries with physiological resistance to parasites. Recent studies reveal that individual variation in social behaviour among uninfected individuals often covaries with their susceptibility to infection (Fig. 2B), a pattern with unknown causality but hypothesized to result from hosts balancing their investment in behavioural *vs* physiological immunity. Individual hosts with less effective physiological defences against parasites appear to avoid behaviours entailing high infection risk (Barber and Dingemanse, 2010): mice (Filiano *et al.*, 2016) and zebrafish (*Danio rerio*; Kirsten *et al.*, 2018) that express lower levels of interferon γ (and are therefore potentially more susceptible to intracellular parasites) are less social, and house finches with lower levels of circulating immune proteins more strongly avoid conspecifics expressing sickness behaviours (Zylberberg *et al.*, 2012). Stephenson (2019) built on these findings by demonstrating that the pattern is similar, with the most susceptible individuals showing strongest conspecific avoidance, when considering susceptibility to the most prevalent parasites in an animal's environment, rather than a general immune component. Intraspecific variation in parasite susceptibility can therefore covary with intraspecific variation in behaviour, leading to potential dampening of ecological feedbacks, and reduced epidemic potential, if individuals that are the most social are also least likely to acquire infection (Hawley *et al.*, 2011).

Once transmission occurs, behavioural changes of parasite-contaminated or actively infected hosts are also heterogeneous (Fig. 2A). Factors extrinsic to the host, such as social context (Lopes, 2014) and seasonality (Owen-Ashley and Wingfield, 2006), as well as factors intrinsic to the host, such as sex (Silk *et al.*, 2018; Stephenson, 2019), social caste (Stroeymeyt *et al.*, 2018) and previous exposure to the parasite (Walker and Hughes, 2009), can dramatically affect behavioural changes in response to infection. Additionally, behavioural changes of infected animals often positively covary with infection intensity (Edwards, 1988; Houde and Torio, 1992; Barber and Dingemanse, 2010), which is naturally highly variable in host populations (Shaw *et al.*, 1998). Thus, hosts that harbour the highest infection intensity (a potential proxy for infectiousness) are also typically the ones most likely to alter their social behaviours (and thus contact rates) in ways that result in ecological feedbacks relevant for parasite transmission. Hawley *et al.* (2011) used an SIR model to show that positive covariation among individuals between their infectiousness and contact rate, whereby the most heavily infected individuals are the most social, can lead to rapid epidemic spread. Recent work demonstrating that infected animals can benefit from living in groups (Almberg *et al.*, 2015; Ezenwa and Worsley-Tonks, 2018) suggests that this positive covariation may occur broadly in systems where animals use social behaviour to increase tolerance. Conversely, when the most infectious individuals elicit the strongest avoidance in uninfected conspecifics (e.g. in guppies: Stephenson *et al.*, 2018), this negative covariation can lead to rapid fade-out of a parasite from a host population. Experimental probing of individual-level relationships (e.g. Stephenson, 2019) will ultimately allow a better understanding of the potential ecological feedbacks that arise from bidirectional relationships between social behaviour and parasite infection, and the way in which these feedbacks are influenced by sources of heterogeneity both intrinsic and extrinsic to hosts (Fig. 2; Hawley *et al.*, 2011; VanderWaal and Ezenwa, 2016; White *et al.*, 2018).

Evolution: parasites drive, and evolve in response to, host social evolution

Parasites are considered key drivers of and constraints on the evolution of host social behaviour (Alexander, 1974; Hart, 1990; Loehle, 1995; Buck *et al.*, 2018; Fig. 1, Arrow C), but the effects of parasites on host social evolution have largely been inferred using comparative studies within and among taxa to elucidate signatures of the 'ghosts of parasites past' (cf Mooring *et al.*, 2006). In this section, we consider ways in which parasites likely influence the evolution of host social behaviours, and discuss some of the constraints on and opportunities for studying these effects.

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In addition, parasites themselves are likely to evolve in response to variation in host social behaviours (Hughes *et al.*, 2008; Schmid-Hempel, 2017), which provide key opportunities for parasite transmission and thus fitness (Fig. 1, Arrow D). We therefore consider how host social behaviours can shape parasite population genetics and their potential to respond to selection, as well as the ways in which host social behaviours impose selection on parasite traits such as virulence, transmission mode and host manipulation.

Parasites and the evolution of host social behaviour (Arrow C)

Akin to parasite-induced changes in social behaviour *via* ecological processes (Arrow B), the social behaviours of both infected and uninfected individuals can evolve in response to parasites (Townsend *et al.*, 2020). Here, we focus on evolutionary changes in the social behaviours of uninfected hosts that are likely to reduce the fitness costs imposed by their socially transmitted parasites. These include reductions in overall individual gregariousness (mechanism 1) that manifest as lower average group sizes for group-living taxa; reductions in social interactions with some but not all conspecifics (mechanism 2), which often manifest as increases in modularity; and reductions or augmentation in specific social behaviours that either increase or decrease parasite risk, respectively (mechanism 3). While these three mechanisms involve fixed phenotypic changes in social behaviours in response to parasite-mediated selection, the costs associated with reduced sociality for many taxa may favour the evolution of conspecific avoidance only in the presence of specific cues of infection (mechanism 4; Amoroso and Antonovics, 2020; Townsend *et al.*, 2020). We briefly explore each of these four mechanisms and discuss constraints associated with evolving phenotypic changes in social behaviours in the face of parasites.

Mechanism 1: evolutionary changes in overall gregariousness

Given the higher risk of parasite spread associated with larger group sizes for many systems (e.g. Nunn and Altizer, 2006; Woodroffe *et al.*, 2009; Rifkin *et al.*, 2012), socially transmitted parasites are predicted to exert selection against individual association with larger groups. For example, given the heritable variation in individual gregariousness (e.g. halictid bees: Kocher *et al.*, 2018; shoaling guppies: Kotrschal *et al.*, 2020), socially transmitted parasites may drive the evolution of reduced gregariousness and lower average host group sizes by causing higher parasite-mediated mortality in more gregarious individuals. Recent evidence suggests, for example, that attraction to conspecific chemical cues in social Caribbean spiny lobsters (*Panulirus argus*) has declined over time, potentially in response to the emergence of the lethal *PaV1* virus (although other factors might have contributed; Childress *et al.*, 2015). Overall, direct empirical evidence for parasite-mediated shifts in gregariousness resulting from evolutionary processes is scarce, potentially (at least in part) because these shifts are obscured by those driven by predators, which are often hypothesized to have opposing effects to those of parasites (Mikheev *et al.*, 2019). Larger groups can serve a protective function against predators, and empirical studies have documented heritable, positive associations between predation pressure and social tendencies of prey (e.g. Seghers, 1974; Jacquin *et al.*, 2016). While the immediate mortality associated with predation could exert stronger selection pressure than that associated with many parasites (e.g. Koprivnikar and Penalva, 2015; Daversa *et al.*, 2019), parasites and the 'landscape of disgust' that they elicit (i.e. the detection and avoidance of areas with high potential parasite risk; Weinstein *et al.*, 2018) are posited to have far-reaching evolutionary consequences, rivalling those of predators, for host behaviours. Nonetheless, determining the relative strength of selection by parasites *vs* predators on host social behaviours remains a considerable challenge.

Common garden and experimental evolution studies that rely on variation in parasite presence (either naturally, for common garden studies, or experimentally) provide promising approaches for directly characterizing evolutionary effects of parasites on host gregariousness. However, even these studies can be challenging to interpret, as results will depend on the virulence of the parasite considered, as well as the competing fitness benefits generated by particular social behaviours. One common-garden study in Trinidadian guppies, for example, found consistent evidence for a heritable, positive effect of predatory pressure on shoal size, but a relatively weak and non-heritable negative effect of parasite pressure on shoal size (Jacquin *et al.*, 2016). However, populations were characterized as having been under selection by parasites based on one observation of the presence or absence of a single species of ectoparasite. In general, strong selection against sociality is most likely imposed by highly virulent parasites with epidemic rather than endemic dynamics (Kessler *et al.*, 2017), as may be the case for many emerging pathogens (Bolker *et al.*, 2010). Further, opposing selection pressures from predation and the many other benefits of group living [e.g. access to mates (Adamo *et al.*, 2015), foraging efficiency (Krause and Ruxton, 2002), transfer of protective microbes (Ezenwa *et al.*, 2016*b*), opportunities for social learning and information transfer (McCabe *et al.*, 2015; Romano *et al.*, 2020) and social support (Snyder-Mackler *et al.*, 2020)] likely limit the ability of many hosts to evolve lower levels of gregariousness in response to parasite pressure (Townsend *et al.*, 2020).

The evolution of lower gregariousness in response to socially transmitted parasites will also be constrained by the conflicting selection pressure that other parasites can place on host social behaviours (Townsend *et al.*, 2020). For example, while socially transmitted parasites should generally select against gregariousness and association with large groups (Anderson and May, 1982; Schmid-Hempel, 2017), some mobile and vector-borne parasites may select for higher gregariousness in systems where *per capita* attack rate declines with group size [Mooring and Hart, 1992; see 'Host social behaviours alter parasite ecology (Arrow A)' section]. Given that all hosts are likely affected by communities of parasites with distinct transmission modes (e.g. Townsend *et al.*, 2018), opposing selection pressures across parasite taxa could obscure parasite-mediated selection on gregariousness. Further, even parasites that are socially transmitted might not always select against sociality if group living ameliorates the fitness costs of a given parasite infection, as appears to be common across taxa (Almberg *et al.*, 2015; Ezenwa *et al.*, 2016*b*; Ezenwa and Worsley-Tonks, 2018; Snyder-Mackler *et al.*, 2020). For example, the food-finding benefits or enhanced predator protection provided by social groups might be sufficiently important for parasitized individuals (e.g. Adelman *et al.*, 2017) that the same parasite can exert opposing selection pressures on its host: selection against overall gregariousness to reduce infection risk, but selection for gregariousness to reduce fitness costs once infected. Thus, the degree to which specific social behaviours are favoured will depend on the parasites that are prevalent and most virulent in a given environment, and the extent to which a given social behaviour leads to infection or reduces fitness costs for each parasite.

Mechanism 2: evolutionary reductions in social interactions with some but not all conspecifics

Given the diverse benefits of group living, parasite-mediated selection may favour reductions in particular social interactions within or among host social groups, rather than reductions in

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overall gregariousness (and thus group size). Reductions in interactions with certain conspecifics can, in some cases, manifest as higher modularity either within or among groups. Nunn *et al.* (2015) found that diverse social taxa show higher levels of modularity in larger social groups, and that this within-group substructuring protected larger groups from socially transmitted parasites in network-based models [see 'Host social behaviours alter parasite ecology (Arrow A)' section]. However, it remains unknown whether this higher modularity in larger social groups represents an evolved response to limit parasite spread (as likely occurs in eusocial insects; Stroeymeyt *et al.*, 2018), or simply a side-effect of the need for individuals to limit social interactions within larger groups (Nunn *et al.*, 2015). Further, while colony-level selection from parasites could generate the within-colony modularity (Stroeymeyt *et al.*, 2018) and even the age-structured division of labour (Udiani and Fefferman, 2020) seen in many eusocial insects, the behavioural traits on which individual-level selection would act to generate emergent differences in within-group modularity for social taxa outside of eusocial insects remain unclear.

Reducing interactions with other groups or colonies (often termed 'outgroup' interactions) may have protective effects for individuals by reducing the input of parasites from outside groups (Freeland, 1976). While there is indirect support in humans for the idea that heightened parasite stress promotes in-group interactions (e.g. Fincher and Thornhill, 2012), it remains unknown whether there is heritable, individual-level variation in the degree of ingroup *vs* outgroup interactions in non-human animals, and whether such behaviour responds to selection from socially transmitted parasites. Finally, as with overall gregariousness, there are likely numerous constraints on the ability of taxa to evolve their social structure in ways that minimize the spread of all socially transmitted parasites. For example, Sah *et al.* (2018) found that no single social network organization had the lowest epidemic probability or duration when the transmission potential of a hypothetical parasite was varied in network simulations. Thus, behavioural traits that underlie social network structure such as modularity may be unlikely to respond to selection if they do not provide protection against a wide range of socially transmitted parasites infecting a given host taxa.

Mechanism 3: evolutionary changes in specific social behaviours

Parasite-mediated selection may be most likely to favour reductions in specific high-risk social behaviours such as agonistic interactions, allowing hosts to reduce transmission risk without concomitant loss of the broader benefits of sociality. For example, in banded mongooses, within-troop aggression facilitates wound invasion by *Mycobacterium mungi* (Flint *et al.*, 2016). Thus, given the heritable variation in aggression in this species, this emerging pathogen could favour reductions in the degree of aggression in which banded mongooses engage. Tasmanian devil facial tumour disease (DFTD), a disease caused by contagious cancer cells that are transmitted largely *via* biting (Hamede *et al.*, 2013), may represent an example of this process: Hubert *et al.* (2018) document that some of the genes under selection in devils (*Sarcophilus harrisii*) since the emergence of DFTD have homologues associated with human social behavioural disorders.

Similarly, selection pressure from parasites could favour a higher frequency of specific social behaviours that reduce parasite spread, such as social grooming or hygienic behaviours (i.e. removal of dead or infected individuals from the colony, as occurs in many eusocial insects; Cremer *et al.*, 2018). Indeed, in eusocial insect colonies, hygienic behaviours are known to be heritable (Spivak and Reuter, 2001), with candidate genes that show evidence for positive selection (Harpur *et al.*, 2019). Increases in allogrooming frequency may similarly evolve in response to parasite-mediated selection from ectoparasites when such behaviours effectively reduce ectoparasite load (e.g. de Brooke, 1985). However, allogrooming can simultaneously expose the groomer to socially transmitted endoparasites such as those spread *via* fecal–oral routes (Biganski *et al.*, 2018). Thus, hosts may be under simultaneous selection pressure to avoid grooming individuals with endoparasitic infections, as occurs in mandrills (Poirotte *et al.*, 2017).

Mechanism 4: evolution of avoidance of infected conspecifics

Parasite-mediated selection on social behaviours is likely to favour the ability of hosts to specifically avoid individuals that pose a high infection risk. This would allow social interactions with uninfected individuals, and their associated benefits, to be maintained, while reducing interactions most likely to facilitate pathogen transmission (Amoroso and Antonovics, 2020). Thus, it is no surprise that diverse social taxa have evolved the ability to detect and avoid conspecifics that likely pose infection risk [see 'Parasite infection influences host social behaviours (Arrow B)' section]. The degree of heritability of these avoidance behaviours in natural systems, and thus their ability to respond to selection, is not well understood, but the genetic basis of the detection and avoidance of conspecifics has been demonstrated in mice (Kavaliers *et al.*, 2005), whereas imprinting during development appears to be key in guppies (Stephenson and Reynolds, 2016). Future work should examine the extent to which the detection and avoidance of infected conspecifics is heritable, which may require the use of study systems amenable to captive breeding.

Kin selection may play a role in the degree to which infected animals evolve to express sickness behaviours, thus altering the ability of uninfected animals to detect and avoid them in ways that promote inclusive fitness. Shakhar and Shakhar (2015), for example, proposed that kin selection would most likely favour social withdrawal after infection in species that live in close contact with kin, leading to the prediction that sickness behaviours and social withdrawal would be more pronounced in these species. Although this prediction has not been tested with respect to sickness behaviours in particular, active self-isolation of infected individuals (e.g. Bos *et al.*, 2012) is present almost exclusively within eusocial insects, for which high within-colony relatedness facilitates the evolution of several seemingly altruistic collective defence behaviours (i.e. 'social immunity' or 'behavioural immunity') *via* kin selection (reviewed in Schmid-Hempel, 2017; Cremer *et al.*, 2018). While these patterns support the existence of an 'inclusive behavioural immune system' (Shakhar and Shakhar, 2015), studies outside the eusocial insects are sorely needed.

Kin selection will also alter the extent to which uninfected individuals evolve to avoid or care for infected individuals. In terms of avoidance, the degree to which a reduction in affiliative social behaviours is favoured after infection may vary with the inclusive fitness benefits that these behaviours confer (Shakhar and Shakhar, 2015), as occurs in mandrills [see 'Parasite infection influences host social behaviours (Arrow B)' section]. Certain parasites could even favour the evolution of care-giving, as seen in eusocial insects that preferentially allogroom pathogen-contaminated individuals (Cremer *et al.*, 2018) if the care of infected kin contributes to inclusive fitness by enhancing host recovery and subsequent reproduction. The degree to which such care is favoured is also likely to depend on the potential costs of infection. For example, a simulation-based analysis of human societies (Kessler *et al.*, 2017) suggested that parasites with intermediate virulence (e.g. measles) could select for substantial care-giving behaviour towards kin; in contrast, pathogens with high fatality and transmission rates (e.g. Ebola) selected for

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the avoidance of all infected individuals, while low-virulence, widespread pathogens (e.g. scabies) were relatively neutral, as care-giving and avoidance had little effect on either recovery or transmission. Other parasites might favour care-giving even if highly virulent. For example, parasites that have strong, negative impacts on fecundity (e.g. that cause host castration) but are not easily transmitted among group members might promote helping behaviour by infected individuals, essentially creating a sterile caste of helpers within their family groups (O'Donnell, 1997). Thus, traits of parasites such as virulence and transmission mode, which can themselves evolve in response to host social behaviours, are critical to consider.

Host social behaviours influence parasite evolution (Arrow D)

For socially transmitted parasites, host social behaviours shape transmission opportunities (Arrow A), which in turn determine a parasite's population structure and evolutionary dynamics. The relatively short generation time of parasites means that host social behaviours may lead to genetic changes in parasite populations within just one or a few host generations. Here, we consider the influence of host social behaviours on (1) fundamental population genetic processes and (2) adaptive evolution of parasites. Our scope of social behaviours includes a diversity of host interactions (Box 1) that may have distinct effects on parasite evolution (Schmid-Hempel, 2017). We focus on social behaviours that change the size and connectivity of host groups, with a brief consideration of behaviours that might change host relatedness.

We first consider the role of host behaviour in shaping the population genetics of parasites and thereby their potential to respond to selection. Increases in the size and connectivity of host social groups can decrease parasite population structure, increase gene flow and promote genetic diversity, leading to overall increases in the effective size of parasite populations. This prediction applies particularly when parasite prevalence increases with host group size, and when transmission opportunities increase with host connectivity. Because larger host groups often maintain larger parasite populations [see Host social behaviours alter parasite ecology (Arrow A) section; Rifkin *et al*., 2012; Patterson and Ruckstuhl, 2013], host social grouping can contribute to the maintenance of parasite genetic diversity at neutral loci and loci under selection by limiting the probability of stochastic extinction of parasite populations (Barrett *et al*., 2008). In addition, connectivity of social groups can increase connectivity of groups of parasites (i.e. demes) if parasite transmission increases alongside direct contacts of hosts. Increased connectivity means increased gene flow and reduced genetic differentiation between parasite groups, both at the level of host individual and population (e.g. Nadler *et al*., 1990). In a test of these predictions, Van Schaik *et al*. (2014) compared the parasites of greater mouse-eared bats (*Myotis myotis*) and Bechstein's bats (*M. bechsteinii*), congeners which differ in their social system: maternal colonies of *M. myotis* mix readily, and individuals hibernate in large clusters, mate in harems and migrate relatively long distances, while maternal colonies of *M. bechsteinii* never mix, and individuals hibernate alone, meet briefly during mating and migrate relatively short distances. Their respective *Spinturnix* wing mite species differ accordingly in their population genetic structure: nuclear genetic diversity of *S. myoti* is very high, with little genetic differentiation between mites in different bat colonies, while nuclear genetic diversity of *S. bechsteini* is lower, with marked differentiation between colonies, suggesting strong genetic drift in small, isolated mite populations. This work demonstrates that larger, more connected social groups host parasite populations that are more genetically diverse.

Increasing host connectivity can also reduce parasite aggregation, with parasites more uniformly distributed rather than clumped on a subset of hosts. Reducing parasite aggregation lowers within-host competition and variance in reproductive success, increasing effective population size for parasites (Whitlock and Barton, 1997; Poulin, 2007). Empirical data support reduced aggregation for ectoparasites with increased host sociality: comparative studies show reduced aggregation of lice in colonial bird species relative to territorial species (Rózsa *et al*., 1996; Rékási *et al*., 1997) and in large *vs* small social groups of Galapagos hawks for amblyceran lice (*Buteo galapagoensis*; Whiteman and Parker, 2004). Taking these processes of parasite connectivity and aggregation together, we generally expect increases in the size and connectivity of host social groups to decrease the effects of genetic drift and promote responses to selection in parasite populations (reviewed in Nadler, 1995; Barrett *et al*., 2008). However, in both bat and avian systems, the sensitivity to host social system varied among parasite taxa, with the structure of some parasites (bat flies and avian ischnoceran lice) unresponsive to differences in group size and connectivity of the same bat (*M. bechsteinii*) and bird (*B. galapagoensis*) hosts (Whiteman and Parker, 2004; Reckardt and Kerth, 2009; van Schaik *et al*., 2015) that produced notable changes in the population structure of wing mites and amblyceran lice, respectively. This contrast between parasite taxa highlights the fact that host social behaviour is but one of many factors that can shape parasite population genetics, and it would be valuable to weigh its relative importance across a broader diversity of host–parasite systems.

In addition to shaping the population genetic structure of their parasites, host group size and connectivity may impose direct selection on virulence, a key parasite trait (Box 1). The common assumption of a trade-off between transmission and virulence predicts that reduced connectivity, or increased modularity, of host groups selects against virulence. The ecological structure of host groups means that parasites with high transmission and virulence should end up with low effective transmission rates because they rapidly deplete the local density of susceptible hosts. This process of 'self-shading' favours mutants with low transmission and low virulence, which maintain a higher average density of susceptible hosts and lower probability of extinction (Boots and Sasaki, 1999). Genetic structure could also lead to 'kin shading': within host groups, nearby parasites are likely kin, such that reduced transmission also confers an inclusive fitness benefit (Wild *et al*., 2009; Lion and Boots, 2010). Moreover, Lipsitch *et al*. (1995) proposed a 'law of diminishing returns': repeated contact between hosts selects for lower virulence because the increased opportunities for transmission between individuals make the benefits of increasing transmission rate too small to offset the cost of increased virulence. By these arguments, the clustering associated with the modularity of social groups should select for parasites with low virulence.

Though they do not directly consider social behaviour, theoretical models support the evolution of reduced virulence with increased modularity of host populations (e.g. Claessen and de Roos, 1995; Rand *et al*., 1995; Boots and Sasaki, 1999). In models that explicitly incorporate spatial structure, transmission ranges from global to local, either by modifying transmission of the parasite (e.g. Boots and Sasaki, 1999) or by varying host contact structure from random interactions between hosts to clustered, regular interactions, modelling modularity within social groups (e.g. Van Baalen, 2002). Generally, as transmission becomes increasingly local, or host contacts become more clustered, the evolutionary optima for transmission rate and correlated virulence shift lower (though see Read and Keeling, 2003). Consistent with theory, Boots and Mealor (2007) found that, in experimental populations of the host *Plodia interpunctella*, a granulosis virus (PiGV)

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evolved reduced infectivity when host mobility was reduced (for further experimental support from other systems, see Kerr *et al.*, 2006; Dennehy *et al.*, 2007; Berngruber *et al.*, 2015). In contrast to modularity, other characteristics of social groups – such as size – may select for increased virulence. Indeed, increasing the size of host modules in spatial models brings the evolutionary dynamics closer to that of well-mixed host populations (Van Baalen, 2002). With transmission and/or host interactions less clustered and regular, the cost of self-shading falls, boosting the evolutionary optima for transmission and virulence. While these models generally assume that host mobility or contact networks (and by extension, modularity) do not vary with parasite status, it is important to also consider infection-induced changes in behaviour and their inherent heterogeneity ['Synthesis: ecological feedbacks between social behaviours and parasite infection ' section; Fig. 2]. These dynamic behavioural feedbacks in response to infection (Arrow B) may alter predictions for virulence evolution (e.g. see Pharaon and Bauch, 2018 on human social behaviour).

Virulence may also evolve indirectly in response to selection that host social behaviour imposes on parasite transmission mode. For parasites with genetic variation in transmission mode, frequent transmission opportunities in host social groups are expected to select for an increased rate of horizontal transmission, whereas among solitary or territorial hosts, reduced transmission opportunities should favour vertical transmission, which ensures transmission from parent to offspring (Antonovics *et al.*, 2017). Selection on transmission mode may in turn impose selection on virulence: experimental studies show that parasite lineages evolve higher virulence with increased opportunities for horizontal transmission (Bull *et al.*, 1991; Turner *et al.*, 1998; Messenger *et al.*, 1999; Stewart *et al.*, 2005), whereas a recent comparative study suggests that vertical transmission favours the evolution of obligate mutualisms (Fisher *et al.*, 2017). Thus, assuming a trade-off between transmission modes, social grouping may indirectly select for increased virulence *via* evolutionary shifts in transmission mode. It is not clear, however, how many host–parasite systems have a significant genetic variation in transmission mode (Antonovics *et al.*, 2017). Moreover, in a key proof of principle study, Turner *et al.* (1998) did not find that transmission mode evolved in response to host density, a potential proxy for host social behaviour.

A further indirect mechanism through which host social behaviour may affect parasite virulence evolution is through its effects on the likelihood of coinfection, which is hypothesized to alter the costs and benefits of virulence for parasites (Bremermann and Pickering, 1983; Alizon *et al.*, 2013). Several studies have found that larger, more connected host groups support richer, more genetically diverse parasite communities (Ranta, 1992; Griffin and Nunn, 2012) and populations (e.g. van Schaik *et al.*, 2014). These studies suggest that hosts in such groups are more likely to be co-infected with multiple species or strains of parasites (though see Bordes *et al.*, 2007). Coinfection could select for increased virulence if virulence stems from the depletion of host resources: in this case, within-host competition favours more virulent parasites that draw more aggressively on host resources (Bremermann and Pickering, 1983; Frank, 1992; de Roode *et al.*, 2005). Alternatively, coinfection could lead to reduced virulence if virulence stems from collective action, like the production of public goods: in this case, competition between unrelated strains favours cheaters, limiting the growth of the parasite population and suppressing virulence (Turner and Chao, 1999; Chao *et al.*, 2000; Brown *et al.*, 2002). As of yet, these predictions are untested in the context of host sociality.

Overall, there is a substantial body of theory and data indicating that host social behaviours likely drive virulence evolution through several interacting pathways: host group size and modularity affect parasite population genetics, and impose both direct and indirect selection on virulence. In contrast, there is surprisingly little research investigating the effect of host social behaviours on the evolution of other parasite traits (Schmid-Hempel, 2017). Here we highlight two topics – host specialization and manipulation – that have received some attention, in hopes of stimulating more research in these areas. First, behaviours that dictate how social groups or modules assemble may determine parasite prevalence and selection for specialization. In many systems, individual hosts preferentially interact with kin due to active choice or physical proximity (e.g. Grosberg and Quinn, 1986; Archie *et al.*, 2006; Davis, 2012). Parasitism may even enhance kin grouping if, for example, individuals actively avoid parasitized non-kin but continue to associate with parasitized kin [see 'Parasite infection influences host social behaviours (Arrow B)' section]. Kin association boosts the mean relatedness of hosts encountered by a parasite lineage, above that predicted if hosts met at random. Taken to its extreme, socializing with kin could create conditions for a parasite akin to host monoculture (King and Lively, 2012; Lively, 2016): on average, increased relatedness, or decreased genetic diversity, of host groups promotes parasite transmission (i.e. the monoculture effect as in Baer and Schmid-Hempel, 1999; Altermatt and Ebert, 2008; Ekroth *et al.*, 2019). Moreover, host relatedness can mimic the selection parasites face under serial passage (Ebert, 1998): generations of transmission within relatively homogeneous host groups may lead to the evolution of host specialization (Bono *et al.*, 2017), either due to trade-offs or relaxed selection for performance on alternate hosts (Kassen, 2002). In systems where hosts do not associate with kin (e.g. Russell *et al.*, 2004; Riehl, 2011; Godfrey *et al.*, 2014), we expect the opposite: increased genetic diversity of interacting hosts should limit parasite spread and maintain parasite populations with relatively broad host ranges. This argument makes the interesting prediction that parasites that jump to novel host populations or species may preferentially derive from diverse host groups. We emphasize that there are few tests of these ideas – our predictions for the impact of group assembly on parasite evolution are based on studies of non-social systems and a few social insect systems (Sherman *et al.*, 1988; Schmid-Hempel, 2017).

Finally, behavioural manipulation of hosts, which includes any parasite-induced change in host behaviour that promotes parasite transmission (Poulin, 2010), is a trait that may experience selection in the context of social behaviour. Parasites transmitted socially could increase their probability of transmission by increasing the rate at which infected hosts interact with susceptible hosts. By this argument, selection of parasite manipulation would intensify host social behaviour. Nonetheless, there is little evidence in support of this hypothesis. Although there is strong evidence of host manipulation in parasites with other transmission modes such as trophic (e.g. trematodes – Carney, 1969) or vector-borne transmission (e.g. *Leishmania* – Rogers and Bates, 2007), there are few accounts of socially transmitted parasites manipulating host contact rates (Poulin, 2010). Some socially transmitted viruses, including rabies, can increase aggression and thereby physical contact, but whether this constitutes adaptive manipulation remains under review due to the variable manifestation of symptoms (Lefevre *et al.*, 2009; Poulin, 2010). In fact, across parasites, it is far more common that parasitism leads to reduced activity and social isolation (Poulin, 2019). An exception are the microsporidia and cestode parasites of brine shrimp (*Artemia franciscana* and *A. parthenogenetica*): these parasites increase swarming of brine shrimp near the water surface, which may increase trophic transmission of the cestode to its avian host and direct transmission of microsporidia to nearby

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Artemia (Rode *et al.*, 2013). Poulin (2010) hypothesizes that evidence for host manipulation in socially-transmitted parasites is limited because the benefits of manipulation are smaller than the costs: for host taxa with high degrees of sociality, many factors already promote interactions with conspecifics, so parasites may gain relatively little in the way of additional transmission opportunities by augmenting contact within groups. Recent work, however, suggests that parasites may induce behavioural changes that increase an infected host's probability of acceptance into new social groups. Geffre *et al.* (2020) found that honey bees infected with Israeli acute paralysis virus (IAPV) are accepted into foreign colonies at higher rates than control bees, even though bees can detect and avoid IAPV-infected nestmates. In comparison, colonies did not show higher acceptance of foreign bees that were immune-stimulated but not infected, suggesting a specific manipulation by IAPV to increase between-colony transmission. The authors speculate that these results point to a coevolutionary battle between parasite manipulation of host social behaviour and hosts' own social defences.

Synthesis: evolutionary feedbacks between host social behaviour and parasite traits

The evolution of host social behaviours in response to parasites (Arrow C) and parasites in response to hosts (Arrow D) support the potential for coevolutionary feedback between social behaviour and parasite traits. Although direct examination is challenging, theoretical models have begun to explore reciprocal adaptation between host social behaviour and parasite traits, and the impact of the behavioural environment on coevolutionary trajectories. For example, Bonds *et al.* (2005) examined feedback between virulence and social behaviour, measured as variation in host contact rate. They made the key assumption that more gregarious hosts live longer, so increased contact carries both a fitness benefit and cost (parasite transmission). As a result, increasing contact rates select against virulence: the lower death rate of more gregarious hosts prolongs the window for parasite transmission, reducing the advantage of parasites with high transmission rates and, by correlation, high virulence. Decreasing virulence reduces the cost of social behaviour, thereby selecting for host contact. These changes in virulence and contact rate increase parasite prevalence, which, at its highest level, further selects for host contact: hosts may as well reap the benefits of socializing when there is no hope of avoiding infection. Prado *et al.* (2009) extended this work to incorporate spatial structure, showing that sociality selects for high parasite virulence and that high virulence, in turn, selects against sociality. Though their results differ somewhat, both models suggest that coevolutionary feedbacks between social behaviour, parasite prevalence and virulence could generate either positive or negative correlations between parasitism and social traits, such as group size, depending upon the life history and coevolutionary history of the study populations.

Other studies suggest that social behaviour is a contextual variable that alters the trajectory of coevolution between host resistance and parasite traits. Best *et al.* (2011) explored the evolution of host resistance and parasite virulence in a coevolutionary model with spatial structure. As in the above models of virulence evolution, Best *et al.* (2011) did not explicitly consider social behaviour, but drew parallels between social grouping of hosts and the treatment of host reproduction and parasite transmission as local (i.e. host offspring or new infections are placed in neighbouring sites, forming clusters) or global (i.e. placed randomly across the network). They found that local host reproduction and transmission select for increased host resistance and reduced parasite virulence. Similar to prior evolutionary models, the explanation for these coevolutionary patterns lies in the spatial distribution of susceptible and infected hosts (ecological structure) and the clustering of kin (genetic structure). A key result from Best *et al.* (2011) is that reproduction and transmission within local (e.g. social) groups could lead to heavily defended hosts with parasites that have low transmission rates and low virulence. Interestingly, this theoretical result matches Hughes *et al.* (2008)'s verbal prediction for social insects and their parasites. Given the importance of the scale of host interactions and transmission for these predictions, further understanding of the among-group movements of infected hosts [see 'Parasite infection influences host social behaviours (Arrow B)' section, Geffre *et al.*, 2020] would facilitate prediction of coevolutionary outcomes.

Host social behaviour may further alter coevolutionary trajectories if behavioural defences negatively covary with physiological defences against parasites . Physiological defences may decline in the presence of behavioural defences if there are trade-offs between defence components (Sheldon and Verhulst, 1996; Parker *et al.*, 2011) or if physiological defences prove redundant and thus experience relaxed selection (Evans *et al.*, 2006; Amoroso and Antonovics, 2020). There is some support for negative covariance of behavioural and physiological defences in social insect systems (Evans *et al.*, 2006; Viljakainen *et al.*, 2009; Harpur and Zayed, 2013; López-Uribe *et al.*, 2016) and more broadly [Klemme *et al.*, 2020; see 'Synthesis: ecological feedbacks between social behaviours and parasite infection ' section]. A key implication of covariance between defence traits is that host social behaviours could fundamentally alter the host defences against which parasites battle and thereby change the traits predicted to be under coevolutionary selection. Given the potential for behavioural defences to alter not only host evolution but also the strength and nature of reciprocal adaptation, it would be valuable to use an experimental evolution approach to directly test the trade-offs between behavioural and physiological defences.

Finally, host social behaviour may structure coevolutionary dynamics *via* its effect on parasite population genetics. Specifically, data from natural host–parasite interactions suggest that the size and connectivity of host social groups contribute to determining genetic diversity and gene flow in their associated parasite populations [see 'Host social behaviours influence parasite evolution (Arrow D)' section]. Coevolutionary models show that gene flow and genetic variation define the capacity for parasite populations to adapt to their evolving host populations and thereby drive coevolution (Lively, 1999; Gandon, 2002; Gandon and Michalakis, 2002). In particular, experimental evolution studies (Forde *et al.*, 2004; Morgan *et al.*, 2005) and meta-analyses of tests with natural host–parasite populations (Greischar and Koskella, 2007; Hoeksema and Forde, 2008) show that relatively low rates of gene flow can prevent parasites from adapting to their local host populations. While social behaviour entails its own complexities, the parallels we highlight suggest that the extensive body of work on the geography and spatial structure of host–parasite coevolution may prove valuable in formulating hypotheses and experiments on the evolution and coevolution of host sociality and parasites (Thompson, 2005).

Conclusions

The fundamental interactions between a host's social behaviours and its parasites have long been of interest, but we still have much to learn about the reciprocity of these interactions, and how these relationships play out for both ecological and evolutionary dynamics (Ezenwa *et al.*, 2016*a*). The bidirectional relationships between host social behaviour and parasites, which we visualize as four distinct arrows (Fig. 1), have largely been studied

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independently, although some have begun to connect these arrows. For example, Stephenson (2019) examined the full ecological feedback loop between behaviour and parasitism (i.e. Arrows A and B) by quantifying social behaviours of guppies both before and during infection, and illustrated that susceptibility-behaviour correlations can change dramatically in the presence of infection. While male guppies most susceptible to parasite infection were most likely to avoid social groups that may pose parasite risk, these highly susceptible guppies became most attracted to social groups once infected (Stephenson, 2019). Because these correlations between host susceptibility and social behaviour likely have important implications for both epidemiological and coevolutionary dynamics , these feedback loops should be examined using systems amenable to experimental infections and, ideally, experimental evolution. Such a system would enable, for example, artificially imposing selection on host social behaviour and testing whether parasite susceptibility evolves in tandem, or *vice versa*; exploring how parasites evolve in response to such artificially selected host lines; and testing how host social behaviours evolve in response to endemic parasitism.

While we largely considered ecological and evolutionary processes separately here, they are likely to interact in important ways (Ezenwa *et al*., 2016*a*). For example, our discussion of ecological interactions suggests that more gregarious host populations maintain larger, more genetically diverse parasite populations. This increase in the size and diversity of parasite populations may apply strong selection on host traits, including social behaviours such as gregariousness (Arrow A affects C). Further, their large effective population size means that parasite populations of gregarious hosts could respond more readily to selection imposed by their host populations, resulting in more rapid evolutionary changes in virulence, stronger local adaptation (Arrow A affects D), and ultimately more intense coevolution. These evolutionary changes in host social behaviours and parasite traits could feed back to alter the ecological interactions of host and parasite: for example, evolutionary changes in host sociality (Arrow C affects A) and parasite virulence (Arrow D affects A) would both affect parasite prevalence and hence parasite population size. While there are informative models investigating some of these ideas (e.g. Bonds *et al*., 2005; Pharaon and Bauch, 2018), experimental studies explicitly addressing these eco-evolutionary feedbacks between host social behaviour and parasite evolution would be welcome additions to this field.

Individual host heterogeneity is one factor that needs more explicit consideration from an eco-evolutionary perspective. Here we discuss one potential source of such heterogeneity as an example, though there are many others (Fig. 2). In many systems, host sex affects both an individual's social behaviour in the presence and absence of infection (Stephenson, 2019), and individual susceptibility (Klein, 2000; Duneau and Ebert, 2012). As a result, male and female hosts support parasite communities differing in size and composition, and provide their parasites with different transmission opportunities (e.g. Christe *et al*., 2007; Stephenson *et al*., 2015; Gipson *et al*., 2019). Parasite fitness therefore depends on the sex of the host, so selection should favour parasite preference for or specialization on one host sex (Duneau and Ebert, 2012), which a growing body of evidence supports (Christe *et al*., 2007; Duneau *et al*., 2012; Campbell and Luong, 2016). Whether such host specialization by parasites contributes to sex-specific evolution of physiological or behavioural parasite resistance (such as sex-specific social behavioural evolution) is an exciting and as yet untested idea. Overall, an explicit theoretical examination of the eco-evolutionary implications of heterogeneity between hosts, such as that due to sex, for behaviour-infection feedbacks is sorely needed.

The recent large-scale social distancing by humans in response to COVID-19 is arguably one of the most dramatic illustrations of the way in which host social behaviour can both influence and respond to parasite spread (Block *et al*., 2020). Perhaps one small positive outcome of this otherwise devastating pandemic will be renewed interest in the dynamic interactions between a host's social behaviours and the ecology and evolution of its parasites. Understanding these interactions not only sheds important light on basic scientific questions such as the costs and benefits of animal sociality, but also addresses critical public health questions about the way in which the behaviours of ourselves and our domesticated animals (via imposed housing conditions) may facilitate pathogen emergence, spread and evolution.

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