Parasitism and the tradeoffs of social grouping: The role of parasite transmission mode

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Abstract

Animals form social groups to gain benefits to numerous fitness-enhancing processes, such as foraging, defense, and energy expenditure. While social grouping can increase parasite exposure, it can also serve as a defensive mechanism against parasites (defined broadly here as organisms with obligate, persistent, and harmful consumer associations with a host). Here, we present a conceptual framework that explores how host sociability affects parasite infection risk in the context of parasite life history, arguing that the positive or negative impact of a social lifestyle on infection risk is strongly linked to the parasite's transmission mode. This framework focuses on common, non-mutually exclusive differences in parasite transmission: direct vs. indirect, density- vs. frequency-dependent, and simple vs. complex life cycles. We then use this framework to discuss the mechanisms for active parasite avoidance, passive effects of infection-induced phenotypes, and their impacts on host social networks, as well as the additional factors that can modulate these dynamics (e.g., parasite virulence, infection intensity, co-infection by multiple parasites, and environmental factors). The goal of this broad, comparative approach is to provide researchers from multiple disciplines with a unified framework to better understand the relationship between social grouping and host-parasite interactions across diverse systems.

1. INTRODUCTION

Despite growing evidence that links parasitism with host physiology, morphology, and cognition, the role of parasites in generating intra- and interspecific variation in their hosts is understudied (Nadler et al. 2023). While parasites can have these diverse impacts on their hosts, studies rarely account for the possibility of parasites' presence (Chretien et al. 2023). In particular, the reciprocal nature of parasitism and host behavior, and the mechanistic drivers of this bidirectional relationship, remains an exciting but often underappreciated research area (Hawley and Ezenwa 2022).

Many animals use social grouping as a behavioral strategy to enhance fitness, which we broadly define here as the tendency to live in groups for prolonged periods (Ward and Webster 2016). A social lifestyle exposes animals to parasites that are transmissible via direct contact between individuals ('directly transmitted parasites'). This risk could influence individuals' tendency to socialize, individual social preferences, and dynamic interactions within and among groups (e.g., group size, composition, assortment; Rifkin et al. 2012, Patterson and Ruckstuhl 2013). Parasites can also be transmitted through the environment or interactions with other species ('indirectly transmitted parasites') (May and Anderson 1979). For these parasites, social grouping could aid hosts in limiting infection risk, by reducing the per capita host attack rate by parasites (Mooring and Hart 1992), increasing detection of free-living parasites (Stumbo et al. 2012), and promoting cost-sharing of anti-parasite defensive strategies (Brutsch et al. 2017). Given the diversity of parasite transmission modes, disentangling the complex feedback loop between parasites and social grouping remains difficult (Ezenwa et al. 2016b).

Here, we look at the role of parasitism in host social grouping through the lens of parasite transmission mode, presenting a conceptual framework for how parasites alter the tradeoffs of

group living. We define parasite transmission mode through dichotomies commonly used by disease ecologists, eco-immunologists, and parasitologists (Figure 1). We discuss the mechanisms for active parasite avoidance, passive effects of infection-induced phenotypes, and their combined impacts on host social networks, as well as highlighting the importance of considering additional factors that modulate these dynamics (e.g., parasite virulence, infection intensity, co-infection, and the environment). This framework connects research at the intersection of behavioral ecology, physiology, immunology, parasitology, and disease ecology, and thereby reaches a more mechanistic understanding of the effects of parasite transmission mode on social grouping of animal hosts. Given the framework's multidisciplinary focus, we define a parasite broadly as any organism (e.g., viruses, bacteria, fungi, protozoa, and metazoa) with an obligate and persistent consumer association with an individual of another species to which it does harm (Anderson and May 1978, Lafferty and Kuris 2002).

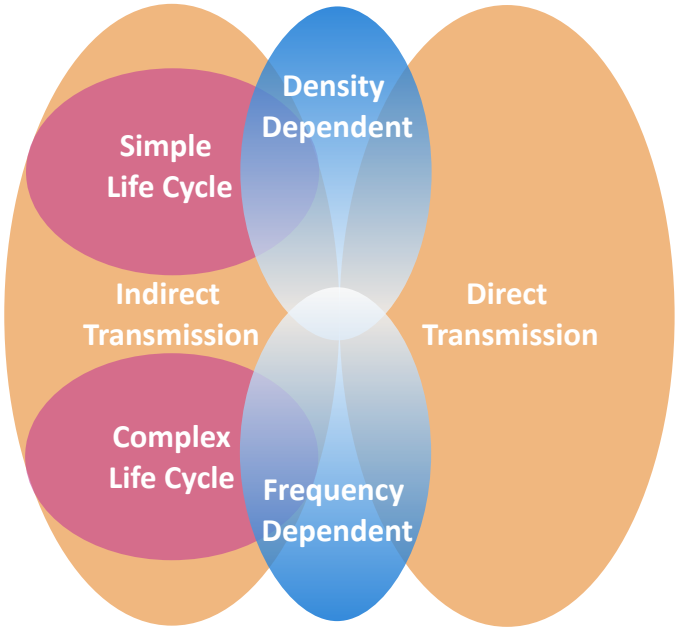

Figure 1. Venn diagram of parasite transmission modes that may impact the tradeoffs of host sociability. Transmission mode refers to the method that a parasite uses to infect a susceptible host. Overlap represents that certain routes of transmission may be found in multiple transmission mode categories.

2. PARASITE TRANSMISSION MODE

Parasite transmission mode refers to the method by which parasites move between susceptible hosts in a population (Antonovics 2017). Below, we describe common transmission dichotomies that influence the ecological and evolutionary processes that shape social grouping (Figure 1). These dichotomies include direct versus indirect transmission, density- versus frequency-dependent transmission, and simple versus complex lifecycle (presented and described here as a sub-category of indirect transmission). Note that while we present dichotomies as distinct categories for clarity, host-parasite systems may fall on a continuum between each category depending on the host and parasite's life history (Roche et al. 2011, Antonovics et al. 2017, Hopkins et al. 2020). Further, while the hierarchical ordering of these dichotomies varies (Antonovics et al. 2017), this classification provides a framework to hypothesize about the ecological implications of parasite infection risk on a host's social phenotype.

2.1. Direct versus indirect transmission

In direct transmission (DT), infection occurs through physical contact or close proximity between infectious and susceptible individuals (Anderson and May 1979). As group living promotes spatial overlap among individuals, it increases the transmission risk of DT parasites. Tactile transmission includes, for example, ectoparasite transfer (skin-skin; Hillgarth 1996),

ingestion of infectious agents (skin-oral; Hernandez and Sukhdeo 1995), and sexual transmission (genital-genital; Mansuy et al. 2016). If the parasite is droplet- or air-borne (Ghosh et al. 2015), transmission does not require tactile contact, as infectious propagules released by the infected host need only come into contact with the susceptible host's eyes, nose or mouth (McCallum et al. 2001). Given the connection between social interactions and infection risk by DT parasites, DT parasites present a substantial potential cost of social grouping, and often reduce individual social tendencies in the short term (Hawley et al. 2021, Stockmaier et al. 2023).

Parasites with an indirect transmission mode (IT) are transmitted either through free-living infectious stages present in the environment or interactions with heterospecific infected hosts (Begon et al. 2002, Antonovics et al. 2017). IT parasites undergo a developmental phase outside their final host before infecting a new susceptible host (Lafferty and Kuris 2002), in either a particular environmental medium or in intermediate host species (Chernin 2000), dictating the type of parasite life cycle (see 2.3. Simple versus complex life cycle). As transmission of IT parasites is a multi-step process, gregarious species can use social grouping as a mechanism to dilute their individual infection risk (Mooring and Hart 1992).

2.2. Density- versus frequency-dependent transmission

Density-dependent (DD) transmission increases directly with host population density, but requires a minimum population size to persist (Anderson and May 1981, McCallum et al. 2001). Hence, DD parasites likely present the greatest cost of social grouping as both larger groups and a higher group density will result in greater transmission, with reductions to group size being a potential strategy that leads to parasite extinction (Lloyd-Smith et al. 2005a).

Frequency-dependent transmission (FD) occurs when the rate of contact between susceptible and infectious individuals is independent of population density (Best et al. 2011,

127 Antonovics 2017), if, for example, the overall population is subdivided into smaller groups of
128 varying sizes as in human villages (Sauvage et al. 2007) and territorial pack animals (White et al.
129 2017). Sexually transmitted diseases are also FD, as the host's mating system dictates the
130 number of partners that a susceptible individual encounters (Thrall et al. 1993). Given the nature
131 of FD transmission, the role of FD parasites on social grouping varies depending on the social
132 trait examined and the life history of each specific parasite and host. While DD and FD
133 transmission were traditionally considered distinct categories, principles associated with both
134 transmission modes have been identified within host-parasite systems, varying depending on the
135 patterns of social behavior through time (e.g., seasonal variation and across contexts) (Smith et
136 al. 2009). Thus, rather than a true dichotomy, host-parasite systems lie on the spectrum from
137 pure DD to pure FD (Hopkins et al. 2020).
138
139 *2.3. Simple versus complex life cycle*
140 In parasites with a simple life cycle (SLC), parasites pass from one host to another through
141 infectious propagules in the environment. After a developmental phase, which can vary from
142 days to months (Brooker et al. 2006), these propagules can be transmitted to a susceptible host
143 via a specific medium, such as water or feces, and/or via shared habitat (Sih et al. 2018). Some
144 SLC parasites are mobile, overcoming physical processes in their environment to seek new hosts.
145 For sedentary SLC parasites, the encounter rate with susceptible hosts is proportional to the
146 propagules' density and distribution in the environment (Boldin and Kisdi 2012). If transmission
147 relies on spatial overlap, the timing must occur in parallel with a sedentary SLC parasite's
148 infectious period following its developmental phase (Sih et al. 2018). Thus, while social
149 individuals may be at higher risk of encountering infectious SLC parasites that have a short

developmental phase (Rifkin et al. 2012), long developmental timing can decouple links between social grouping and SLC infection risk (Sih et al. 2018).

In contrast, complex life cycle (CLC) parasites require one or more intermediate hosts for growth and development prior to reaching the final host, where the parasite reproduces (Parker et al. 2015). At each developmental stage, parasites may specialize in infecting one host species (i.e., host specialist) or be able to infect multiple species (i.e., host generalist). Various factors can alter host specificity, such as the temporal and spatial overlap of hosts and parasites, sensory mechanisms for the parasite to detect a suitable host, and strategies for the parasite to evade host anti-parasite defenses (Poulin et al. 2011, Doherty et al. 2022). Transmission of CLC parasites between host species can occur via multiple routes, including passive transport, active movement, vector-borne, and trophic interactions (Grear et al. 2013, Hobart et al. 2022). With passive transport, the infectious propagules are transmitted through the environment via physical processes, such as wind and water currents (Akullian et al. 2012, Behringer et al. 2018). Many CLC parasites have a mobile phase that can actively seek out a subsequent host, allowing them to overcome environmental obstacles to transmission (e.g., air or water turbulence) (Fingerut et al. 2003a, Buck and Lutterschmidt 2016). Vectors (often haematophagous arthropods like mosquitos and ticks) carry disease-causing infectious agents from infected to susceptible individuals (Wilson et al. 2017). In trophically-transmitted parasites, infectious propagules are transmitted through predation events (Grear et al. 2013). For each CLC transmission route, group living can dilute an individual's per capita infection risk (Mooring and Hart 1992).

3. SOCIAL CONSEQUENCES OF PARASITES

As parasitism is ubiquitous, most social animals harbor one or more parasite species (McCabe et al. 2015). To understand the role of parasite transmission mode on host sociability, the mechanisms for active parasite avoidance, passive effects of infection-induced phenotypes, and its role in social networks need to be considered (Table 1; Hawley et al. 2021).

Table 1. Examples of the impact of parasite transmission mode on social grouping, based on a subset of the studies compiled in this review, grouped based on the mechanism of social impact.

Mechanism	Host	Parasite	Transmission Mode	Impact on social grouping	References
Active avoidance	Mandrill *Mandrillus sphinx*	Varied gut protozoans (protozoa)	Simple Life Cycle (stationary, rapid development)	Uninfected individuals avoid grooming infected groupmates.	Poirotte et al. 2017
Active avoidance	Caribbean spiny lobster *Panulirus argus*	*Panulirus argus* virus 1 (virus)	Density-Dependent	Individuals avoid den-sharing with infected conspecifics.	Behringer et al. 2006; Behringer and Butler 2010
Active avoidance	Fathead minnows *Pimephales promelas*	*Ornithodiplostomum ptychocheilus* (metazoa)	Complex Life Cycle (mobile)	Individuals increase shoal cohesion by up to 15 times in response to free-living parasites (i.e. cercariae).	Stumbo et al. 2012
Passive effects	Rainbow trout *Oncorhynchus mykiss*	*Diplostomum spathaceum* (metazoa)	Complex Life Cycle (mobile)	Reduced vision following infection of the eye lens reduces the social group's ability to maintain cohesion.	Seppälä et al. 2008
Passive effects	Milkweed leaf beetles *Labidomera clivicollis*	*Chrysomelobia labidomerae* (metazoa)	Frequency-Dependent	Infected males interact with other males, and steal females from established mating pairs, more than uninfected males.	Abbott & Dill 2001
Passive effects	Three-spined stickleback *Gasterosteus aculeatus*	*Schistocephalus solidus* (metazoa)	Complex Life Cycle (trophically transmitted)	Mixed-infection groups take greater risks and reduce cohesion.	Demandt et al. 2018, 2020; Jolles et al. 2020b
Social network	Guppy *Poecilia reticulata*	*Gyrodactylus* spp. (metazoa)	Density-Dependent	Introduction of infected newcomers increases the frequency of fission-fusion events.	Croft et al. 2011; Stephenson et al. 2018
Social network	Giraffe *Giraffa camelopardalis*	*Trichostrongylus* spp., *Trichuris* spp. (metazoa)	Frequency Dependent, Simple Life Cycle (stationary, 5-10 day development)	Sub-grouping within populations relies on weak ties among cliques for transmission.	VanderWaal and Ezenwa 2016
Social network	Sleepy lizard *Tiliqua rugosa*	Tick *Amblyomma limbatum* (metazoan)	Simple Life Cycle (mobile, 8 day development)	Highly connected lizards with consistent refuge sharing had higher tick loads than less connected individuals.	Leu et al. 2010

3.1. Active parasite avoidance

Once cues of parasites or infection are detected, individuals may use active avoidance to minimize risk of parasite exposure (Lopes et al. 2022), including avoidance of certain individuals, substances, and habitats where parasites or infected group-mates are found (Curtis 2014). Similar to the non-consumptive effects that predators impose on their prey (i.e., landscape of fear; Clinchy et al. 2013), parasites can impact hosts over various spatial and temporal scales (i.e., landscape of disgust; Weinstein et al. 2018), with wide-ranging consequences for the dynamics of animal groups (Doherty and Ruehle 2020).

3.1.1. Sensory basis for parasite and infection detection

Detecting transmissible infections (DT and some SLC parasites) involves visual, olfactory, auditory, and mechanical cues (Behringer et al. 2018). Visual cues of infections can be physical changes in host appearance (Rahn et al. 2015) or behaviors associated with "sickness" (Hart and Hart 2021). Sickness behaviors, such as reduced overall activity and exploration (Lopes et al. 2021), may cause avoidance of infectious individuals (Hart and Hart 2021). Infection can also be detected through an individual's odor (Stephenson et al. 2018), as demonstrated in mice (Kavaliers and Colwell 1995), bullfrogs (Kiesecker et al. 1999), guppies (Stephenson et al. 2018), mongooses (Mitchell et al. 2017), and mandrill monkeys (Poirotte et al. 2017). These changes in chemical profile can stem from fluctuations in an individual's commensal microbial community, release of metabolic by-products associated with the immune response, or suppression of androgenic hormones (Penn and Potts 1998). Infection status may also be transmitted through auditory cues, such as vocal signals (Lopes et al. 2022). For example, when yellow-bellied marmots are infected with *Eimeria* (SLC parasite with a 1-2 day environmental

development), they exhibit noisier and less structured vocal signals (Nouri and Blumstein 2019). These sensory cues of infection may not be isolated to a single modality, as sensory redundancy can ensure communication of infection risk (Reichert et al. 2023).

3.1.2. Avoidance of infected group-mates by uninfected individuals

Once sensory cues of infection are detected, uninfected individuals may avoid socializing with infected individuals to avoid transmission (Figure 3; Barber et al. 2000, Kavaliers and Choleris 2018). Such aversive behavioral responses can arise before hosts become visibly symptomatic, as seen in social Caribbean spiny lobsters (*Panulirus argus*) that avoid asymptomatic individuals infected with the DD *Panulirus argus* virus 1 (PaV1) (Behringer et al. 2006, Behringer and Butler 2010). Individuals can also modulate the strength of avoidance behaviors depending on a group-mate's infection stage (Stephenson et al. 2018). In mice (*Mus muscululus domesticus*), avoidance of individuals infected by the stationary SLC parasite *Eimeria vermiformis* (2-7 day environmental development; Fayer 1980) is lower prior to the infection being transmissible, even if infected individuals exhibit detectable cues of infection (Kavaliers et al. 1997). Notably, effective infection avoidance behaviors can be associated with improved fitness outcomes (Newey and Thirgood 2004).

Parasites can also alter the tendency for groups to split (fission) or merge (fusion). For DT parasites, individuals that would typically maintain fidelity to a single group may leave groups when infection risk becomes high (Freeland 1976) or following the introduction of a potentially infected newcomer (Croft et al. 2011). This wariness of newcomers may be advantageous, as newcomers can introduce a novel DT parasite to the group (Woodroffe et al. 2009). Reducing group size may also lessen the infection rate of a DT parasite (Figure 2). Ritchie

et al. (2021) showed, for example, that the infection rate of the DT-transmitted fungus *Batrachochytrium dendrobatidis* is faster in larger groups of the California slender salamander (*Batrachoseps attenuatus*). While parasites that are transmitted through social interactions or spatial overlap (e.g., DT) may increase the frequency of fission events, we know little about how environmentally transmitted parasites (e.g., IT) alter fission-fusion dynamics.

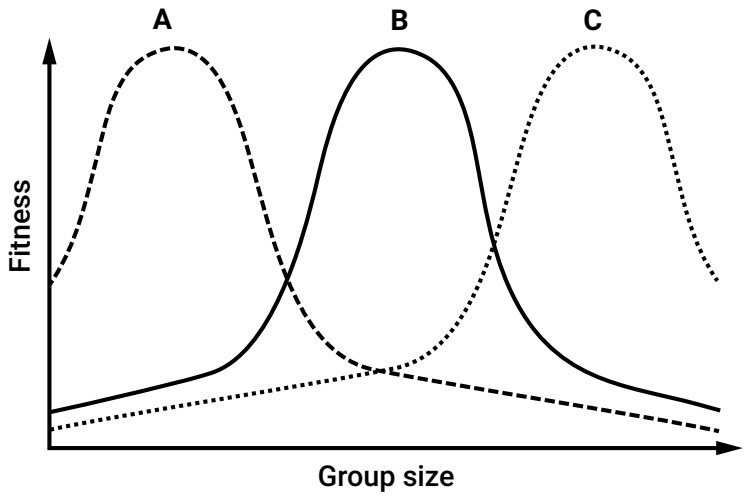

Figure 2. A potential theoretical relationship between host group size and fitness under varied parasite risk, assuming all other factors kept constant (e.g., foraging efficiency, competition for resources, predator defense, and parasite avoidance). Curve A represents social contexts in which reducing social contacts through lower group sizes would be the most effective strategy to reduce transmission, such as directly transmitted parasites. Curve C illustrates environments in which larger group sizes dilute the risk of parasite infection, including indirectly transmitted parasites with a simple (mobile or have a long environmental development phase) or complex (mobile, vector, or trophically transmitted route of infection) life cycle. Curve B represents environments in which risk of any parasite infection is low. Note that curve shape is for illustrative purposes only.

3.1.3. Avoidance initiated by infected group-mates

Infected individuals may also spend less time with uninfected group mates or disperse to a new area, particularly when species live in societies with high kin structure (Iritani and Iwasa 2014). Some social species have evolved 'warning' signals of infection. In familial dampwood termite (*Zootermopsis angusticollis*) colonies, individuals infected by the DT fungus *Metarhizium anisopliae* use a mechanical alarm signal to warn nest-mates to stay away (Rosengaus et al. 1999). However, this self-induced social isolation can lead to higher post-infection mortality (Kohlmeier et al. 2016). Whether infected individuals initiate avoidance will likely depend on factors related to parasite transmissibility, severity of infection costs, and inclusive fitness benefits of leaving the group (Iritani and Iwasa 2014).

3.1.4. Use of social grouping to reduce infection severity

Individuals in a cooperative social group can mount stronger anti-parasite defenses to some parasites when working together (i.e., social immunity; Cremer et al. 2018). For example, ants indiscriminately partake in allogrooming regardless of infection status to achieve frequent, low-level exposure to DT fungal parasites, increasing both individual and colony-level survival following outbreaks (Konrad et al. 2012). When risk of mobile CLC parasites is high, individuals may stay with a group that they would otherwise leave, as even brief isolation while moving between groups could increase their risk of exposure to free-living parasites (Figure 3). In fathead minnows *Pimephales promelas*, Stumbo et al. (2012) showed that individual infection risk by mobile, free-living *Ornithodiplostomum ptychocheilus* parasites (i.e., cercaria) was three times higher for solitary versus shoaling individuals. Similar to the dilution effect of group

268 behavior on predation risk (Foster and Treherne 1981), infection intensity by mobile parasites
269 decreases as group size increases (Patterson and Ruckstuhl 2013). Individual risk of parasite
270 attack is diluted if the parasite cannot attack a greater number of individuals in a larger group
271 (Hart 1994, Patterson and Ruckstuhl 2013), suggesting that group size preferences could evolve
272 (in part) in response to mobile parasite prevalence (Figure 2). Individuals may also reduce
273 infection risk through social learning, with mice learning anti-parasite defensive behaviors after
274 only a single observation of another mouse being attacked by a blood-feeding fly (Kavaliers et
275 al. 2001, 2003). In theory, an individual's parasite exposure history could drive behavioral
276 plasticity in social preferences through time but this topic remains an unexplored area.
277 Parasite risk can also alter tradeoffs associated with different positions in a social group,
278 resulting in altered position shuffling. Individuals at the front of a mobile group generally benefit
279 from having first access to any food encountered (Krause et al. 1998), but often face higher
280 predation and mobile parasite risk (Newsom et al. 1973, Morrell and Romey 2008). Newsom et
281 al. (1973) showed that leaders in herds of East African Zebu cattle (*Bos indicus*) had a higher
282 infection intensity of the mobile SLC brown ear tick (*Rhipicephalus appendiculatus*) than
283 followers. As leader positions provide individuals with first access to food, these positions may
284 also come with a higher risk of acquiring trophically-transmitted CLC parasites, but this idea is
285 yet to be studied. Conversely, central positions in groups may provide safety from mobile
286 parasites in a similar way as has been documented in groups attacked by predators with certain
287 hunting strategies (Krause and Ruxton 2002, Ward and Webster 2016).
288 Group cohesion varies in response to parasite exposure and risk, but the directionality of
289 the change depends on the parasite's transmission mode and mobility. When mobile parasite
290 stages are attacking (e.g. CLC parasite), the cohesion of social groups increases in a similar way

to when predators attack (Stumbo et al. 2012). When encountering DT or SLC parasites with limited mobility and short developmental periods, individuals can reduce their infection risk through reduced group cohesion (Friant et al. 2016). Taken together, the available evidence suggests that mobile CLC parasites may impact group dynamics similarly to predators, while parasites that can be transmitted among group members reduce group cohesion and coordination as group members try to limit infection risk.

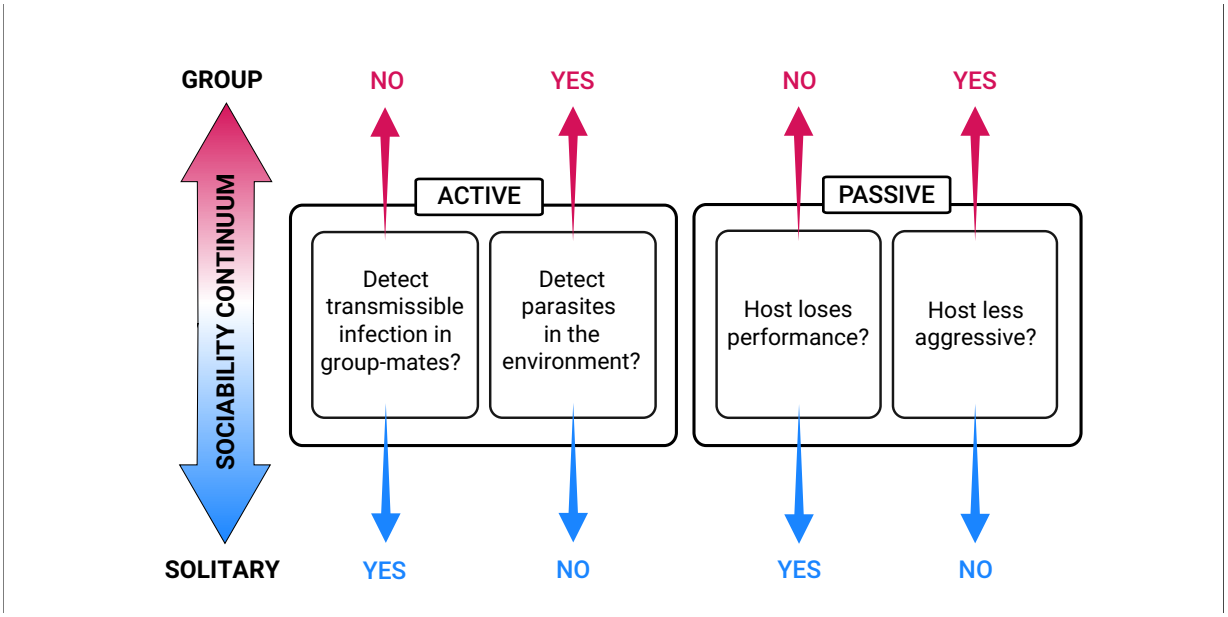

Figure 3. Predictions for how parasites with varying transmission modes could influence host behavior along the sociability continuum from a solitary to a group lifestyle because of active parasite avoidance ('active') and passive effects of infection-induced phenotypes ('passive'). Arrows marked 'yes' indicate the directionality that the mechanism pushes the individual along the sociability continuum.

3.2. Passive effects of infection-induced phenotypes

306 Parasite infection may also indirectly affect social grouping by inducing phenotypic change.
307 Even minor changes in host physiology or movement behavior can have substantial social
308 repercussions by altering the tradeoffs of grouping (Jolles et al. 2020a). Parasite-induced
309 phenotypes can, for example, reduce investment in defense in favor of food acquisition or
310 enhance the need to use social grouping to minimize energy expenditure (Krause and Ruxton
311 2002, Ward and Webster 2016).

312 Parasite infection can alter host behavioral phenotypes and responses to sensory cues
313 through impairments imposed by the infection or parasite manipulation to enhance transmission.
314 In several DT host-parasite systems (e.g., Bouwman and Hawley 2010), infection reduces host
315 aggression, resulting in enhanced cooperative foraging and reduced competitive interactions
316 (Figure 3). Some parasite infections, particularly endoparasites typical of IT infections, can alter
317 individual sensory abilities (e.g., visual acuity), thereby impairing their host's ability to maintain
318 social cohesion (Seppälä et al. 2008). In some cases, parasites may hijack their host's phenotype
319 to enhance their own fitness (Godfrey and Poulin 2022), with impacts on host social grouping.
320 For example, male milkweed leaf beetles *Labidomera clivicollis* infected by FD-transmitted
321 mites (*Chrysomelobia labidomerae*) interact more with other males and steal females from
322 established mate pairs, likely enhancing transmission to both males and females (Abbott and Dill
323 2001). In trophically-transmitted CLC parasites, infection may alter host behaviors that increase
324 detectability by predators (e.g., increasing activity or conspicuous behaviors) and reduce anti-
325 predator responses (Godfrey and Poulin 2022). Conversely, as parasites often require close
326 proximity between group-mates for transmission, particularly DT, DD, and FD parasites, the
327 parasite may increase their host's social tendencies to promote transmission (Godfrey and Poulin

2022). All these social impacts result from the host's altered behavioral phenotype, rather than active parasite avoidance by the host or its group-mates.

Parasite infection may also come with energetic consequences for the host. Maintenance metabolic rate could increase as parasites extract resources from their host or maximum metabolic rate and aerobic capacity may drop due to damaged tissues involved in metabolically-costly processes (Robar et al. 2011, Binning et al. 2017). Any increase in maintenance metabolic rate can shift the tradeoff from prioritizing defense to acquiring food despite risk. For example, the CLC tapeworm *S. solidus* consumes a large portion of the food intake of their three-spined stickleback intermediate host (Giles 1983), leading to increased individual and group risk-taking behavior (Demandt et al. 2018, Demandt et al. 2020).

Locomotor performance could also be altered due to host impairments, responses to the parasite and parasite manipulation of hosts (Binning et al. 2017). Both endo- and ectoparasites may alter their host's ability to stay with a social group through impaired locomotor performance (Allan et al. 2020) and increased energetic costs of movement (Binning et al. 2013; Figure 3). These changes can result in individuals either moving to positions at the back of the group or even becoming incapable of staying with their group (Jolles et al. 2020b), which may lead to assortment of individuals based on preferred locomotor pace (Killen et al. 2017). Alternatively, when infected and uninfected individuals stay together, these groups may be slower, less cohesive, and less aligned as a result (Jolles et al. 2020b). While many such social effects could be hypothesized across the broad variety of host-parasite systems, the consequences of parasite infection for collective movement remain understudied.

3.3. Role of parasite transmission mode in social networks

Host social networks (i.e., social interaction structure in a population or group) and parasite transmission are closely interlinked (Mistrick et al. 2022). Not only does social network structure shape epidemiological dynamics of many parasites, but parasite spread also influences network structure (Ezenwa et al. 2016a). This bidirectional relationship not only impacts ongoing parasite transmission, but also other eco-evolutionary processes, such as behavioral contagions (Evans et al. 2020).

The direct role of social networks is clearest for DT (including DD and FD) and some SLC parasites, as specific types of social interactions offer primary transmission routes (White et al. 2017). Variation in network connectivity among individuals can generate more explosive outbreaks (Figure 3), with onward infection dominated by particular highly-connected individuals (Lloyd-Smith et al. 2005b). Simultaneously, sub-divisions in the social network that cause a more modular structure can limit parasite spread, by trapping infections within subsets of the overall group or population (Sah et al. 2017). Consequently, individual network position is correlated with infection status (Briard and Ezenwa 2021). Well-connected individuals face greater infection risk due to more frequent social encounters, but less socially integrated individuals can be more susceptible when infected, if social integration is associated with poor condition and/or immune performance (Balasubramaniam et al. 2016, Ezenwa et al. 2016b). For DT parasites, sickness behavior of infected individuals can reduce network connectivity in some host-parasite systems (Lopes et al. 2016), but could also increase connectivity in others, such as in dogs with the furious form of rabies (Brookes et al. 2019).

While social networks play a less direct role in IT parasite transmission, they often shape which individuals share food and water sources, meaning that network connections may still be correlated with infection routes. Often networks that incorporate temporal lags are best

correlated with SLC parasite transmission (Leu et al. 2010, Grear et al. 2013), as direct contact is not required and there may be an environmental development phase (Sih et al. 2018). Because of this difference between DT and IT parasites, network approaches can help tease apart the contribution of each transmission mode for parasites with varied transmission (e.g., Blyton et al. 2014). In contrast, the one study to have investigated if social network centrality was associated with CLC parasite infection risk found no effect, although this study focused on a trophically-transmitted parasite (Grear et al. 2013). No studies (to our knowledge) have examined how social networks impact transmission of mobile stages of CLC parasites (e.g., trematode cercariae; Figure 3).

The passive and active social responses to parasites described previously can alter social network dynamics. For example, infection avoidance behaviors against DT parasites can lead to predictable changes to network structure, such as uninfected individuals having a higher and more variable number of social interactions than infected individuals (Shaw and Schwartz 2008) and being clustered together within the network (Gross et al. 2006), with these changes promoting parasite re-emergence or persistence (Gross et al. 2006). In non-human animals, an experimental manipulation of *Gyrodactyalus* sp. (DT ectoparasite) load in guppies *Poecilia reticulata* showed that avoidance of infected individuals resulted in less clustered social networks (Croft et al. 2011). In the ant *Lasius niger,* parasite exposure caused behavioral changes that increased modularity, clustering and assortativity in colony networks in a manner that reduced parasite transmission (Stroeymeyt et al. 2018). The latter study is important, as it shows individual behavior in response to infection can lead to adaptive group-level changes in network structure that match theoretical models in network science.

How active avoidance behaviors influence network structure may differ across the DT-IT and SLC-CLC transmission mode dichotomies due to variation in the landscape of disgust according to transmission mode (see "Active parasite avoidance"). For IT parasites, any active infection avoidance will be tied more to the ecological (e.g., habitat) than social environment (see previous sections). For some CLC parasites, infection exerts few costs to hosts, so minimal pressures would exist to promote avoidance (e.g., trophically transmitted CLC parasites; Øverli and Johansen 2019). However, assuming there are cues for parasite presence in the environment (e.g., fecal matter for SLC parasites or chemical alarm cues for mobile cercariae parasites; Poulin et al. 1999, Hutchings et al. 2000), avoidance of risky or contaminated areas could influence network structure in different ways depending on the spatial distribution of these risky areas. Similarly, if infection risk with CLC parasites varies with time across daily, tidal or seasonal cycles (e.g., Fingerut et al. 2003b), then this could drive predictable changes to network structure if contact rates are altered by changes to spatial behavior. However, studies examining how host social networks studies may be shaped by infection avoidance have focused on DT parasites, with impacts of IT parasites requiring much greater theoretical and empirical attention.

OTHER PARASITE CHARACTERISTICS

While transmission mode plays a pivotal role in driving the relationship between infection and host sociability, other factors may shift this dynamic and should be considered. Below, we highlight some examples of potential ways that these factors can shape the links between parasite transmission mode and social grouping in hosts.

Parasite virulence

Parasites exhibit intra- and inter-specific variation in virulence (i.e., infection-induced reduction in host fitness) (Cressler et al. 2016), varying from subclinical (i.e. not severe enough to present measurable effects) to lethal effects (Bull and Lauring 2014). The transmission-virulence trade-off theory predicts that parasites evolve a virulence level that maximizes their transmission (Alizon et al. 2009). Thus, virulence should be higher in IT (particularly mobile stages) versus DT parasites, since these parasites are not dependent on host movement or social interactions for transmission. However, host-parasite systems exhibit plasticity in virulence depending on individual-to-individual transmission rates, host population density, and contact rates among social groups (Frank & Schmid-Hempel, 2008; Cressler et al., 2016). Heterogenous contact rates among potential hosts (e.g., through sub-grouping) in the population can also drive reduced virulence, since transmission opportunities to new groups are rare and host mortality is costly for the parasite's fitness (Cressler et al. 2016).

Infection intensity

While infection is often treated as binary (uninfected vs infected), in reality, individuals vary in the number of parasites found on or in a single host (i.e., infection intensity; Rózsa et al. 2000). Heterogeneity in infection intensity can occur due to diverse factors, including differences in individual resistance to parasites, acquired immunity following previous infections, and host behavior modulating parasite encounter rate (Lopes et al. 2022). Higher parasite intensities are typically associated with more dramatic host effects (e.g., Moretti et al. 2017), such as individual social standing can also be impacted. In the treefrog *Hypsiboas prasinus*, male calling performance declines with rising SLC-helminth parasite intensity, which decreases males' ability to attract high quality female mates (Madelaire et al. 2013).

442

443 *Co-infection by diverse parasites*

444 Animals are often co-infected by several parasite species (Pedersen and Fenton 2007, Viney and
445 Graham 2013), which may have conflicting strategies to maximize their fitness. For instance,
446 conflict between parasite species can arise when a host is infected by both DT and IT parasites
447 (Hafer 2016). DT parasites benefit from increased host sociality to maximize encounters with
448 susceptible individuals, while transmission routes for IT parasites (e.g., trophic transmission,
449 active migration) are often limited by social mechanisms that can dilute individual's risk of
450 infection (Rahn et al. 2015, Buck and Lutterschmidt 2016). The degree to which these co-
451 infections interact to shift the host's social phenotype remains largely unexplored.

452

453 *Environmental factors*

454 Hosts and their parasites experience environmental conditions that can alter their spatial-
455 temporal distribution and transmission rates (Cable et al. 2017). Variation in climate likely
456 impacts parasites with an environmentally transmitted stage, common in IT parasites. For
457 instance, parasite development slows during dry and cold seasons (Turner and Getz 2010, Kutz
458 et al. 2014) and the emergence patterns of free-living, aquatic parasites are dictated by
459 temperature, light and water depth (Fingerut et al. 2003b). For hosts, activity and exploration
460 generally increase at warmer temperatures, leading to higher host encounter rates with IT
461 parasites and greater social interactions that facilitate DT parasite transmission (Barber et al.
462 2016). Seasonal and anthropogenic changes in water flow can also impact parasite transmission.
463 Reynolds et al. (2019) show that interrupted water flow leads to higher transmission of the DD
464 ectoparasite *Gyrodactylus turnbulli* and reduced shoal cohesion in guppy hosts. This

unpredictable cocktail of environmental factors enhances the complexity of parasite transmission and its interaction with host sociability.

CONCLUSIONS

Parasites contribute to host intra- and interspecific variability in the social phenotypes. Although it has been 50 years since Alexander (1974) first proposed a link between social grouping and parasitism, we have much to learn about the complex interactions between gregarious hosts and their parasites. We know little about how parasite exposure history could influence social phenotypes. Even when the host successfully fights off a parasite's invasion, parasite exposure can alter the host's behavior (Parker et al. 2023, Vindas et al. 2023), highlighting that the parasites found in study animals may represent only a fraction of the host's experience. Further, we still have much to learn about the bidirectional relationship between social dynamics and infection risk, including factors like group positioning, composition, and social network structure, and represent exciting opportunities for new research in this area.

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488 REFERENCES

489 Abbott, P., and L. M. Dill. 2001. Sexually transmitted parasites and sexual selection in the
490 milkweek leave beetle, *Labidomera clivicollis*. Oikos **92**:91-100.
491 Akullian, A. N., D. Lu, J. Z. McDowell, G. M. Davis, R. C. Spear, and J. V. Remais. 2012.
492 Modeling the combined influence of host dispersal and waterborne fate and transport on
493 pathogen spread in complex landscapes. Water Quality, Exposure and Health **4**:159-168.
494 Alexander, R. D. 1974. The evolution of social behavior. Annual Review of Ecology and
495 Systematics. **5**:325-383.
496 Alizon, S., A. Hurford, N. Mideo, and M. Van Baalen. 2009. Virulence evolution and the trade-
497 off hypothesis: history, current state of affairs and the future. Journal of Evolutionary Biology
498 **22**:245-259.
499 Allan, B. J. M., B. Illing, E. P. Fakan, P. Narvaez, A. S. Grutter, P. C. Sikkel, E. C. McClure, J.
500 L. Rummer, and M. I. McCormick. 2020. Parasite infection directly impacts escape response
501 and stress levels in fish. Journal of Experimental Biology **223**.
502 Anderson, R. M., and R. M. May. 1978. Regulation and stability of host-parasite interactions. I.
503 Regulatory processes. Journal of Animal Ecology **47**:219-247.
504 Anderson, R. M., and R. M. May. 1979. Population biology of infectious diseases: Part I. Nature
505 **280**:361-367.
506 Anderson, R. M., and R. M. May. 1981. The population dynamics of microparasites and their
507 invertebrate hosts. Philosophical Transactions of the Royal Society B **291**:451-524.
508 Antonovics, J. 2017. Transmission dynamics: critical questions and challenges. Philosophical
509 Transactions of the Royal Society B **372**.

510 Antonovics, J., A. J. Wilson, M. R. Forbes, H. C. Hauffe, E. R. Kallio, H. C. Leggett, B.
511 Longdon, B. Okamura, S. M. Sait, and J. P. Webster. 2017. The evolution of transmission
512 mode. Philosophical Transactions of the Royal Society B **372**.
513 Balasubramaniam, K., B. Beisner, J. Vandeleest, E. Atwill, and B. McCowan. 2016. Social
514 buffering and contact transmission: network connections have beneficial and detrimental
515 effects on *Shigella* infection risk among captive rhesus macaques. PeerJ **4**:e2630.
516 Barber, I., B. W. Berkhout, and Z. Ismail. 2016. Thermal change and the dynamics of multi-Host
517 parasite life cycles in aquatic ecosystems. Integrative and Comparative Biology **56**:561-572.
518 Barber, I., D. Hoare, and J. Krause. 2000. Effect of parasites on fish behaviour: a review and
519 evolutionary perspective. Reviews in Fish Biology and Fisheries **10**:131-165.
520 Begon, M., M. Bennett, R. G. Bowers, R. P. French, S. M. Hazel, and J. Turner. 2002. A
521 clarification of transmission in terms of host-microparasite models: numbers, densities and
522 areas. Epidemiology & Infection **129**:147-153.
523 Behringer, D. C., and M. J. Butler. 2010. Disease avoidance influences shelter use and predation
524 in Caribbean spiny lobster. Behavioral Ecology and Sociobiology **64**:747-755.
525 Behringer, D. C., M. J. Butler, and J. D. Shields. 2006. Avoidance of disease by social lobsters.
526 Nature **441**:421.
527 Behringer, D. C., A. Karvonen, and J. Bojko. 2018. Parasite avoidance behaviours in aquatic
528 environments. Philosophical Transactions of the Royal Society B **373**.
529 Best, A., S. Webb, J. Antonovics, and M. Boots. 2011. Local transmission processes and disease-
530 driven host extinctions. Theoretical Ecology **5**:211-217.
531 Binning, S. A., D. G. Roche, and C. Layton. 2013. Ectoparasites increase swimming costs in a
532 coral reef fish. Biology Letters **9**:20120927.

533 Binning, S. A., A. K. Shaw, and D. G. Roche. 2017. Parasites and host performance:
534 Incorporating infection into our understanding of animal movement. Integrative and
535 Comparative Biology. **57**:267-280.
536 Blyton, M. D., S. C. Banks, R. Peakall, D. B. Lindenmayer, and D. M. Gordon. 2014. Not all
537 types of host contacts are equal when it comes to *E. coli* transmission. Ecology Letters **17**:970-
538 978.
539 Boldin, B., and E. Kisdi. 2012. On the evolutionary dynamics of pathogens with direct and
540 environmental transmission. Evolution **66**:2514-2527.
541 Bouwman, K. M., and D. M. Hawley. 2010. Sickness behaviour acting as an evolutionary trap?
542 Male house finches preferentially feed near diseased conspecifics. Biology Letters **6**:462-465.
543 Briard, L., and V. O. Ezenwa. 2021. Parasitism and host social behaviour: a meta-analysis of
544 insights derived from social network analysis. Animal Behaviour **172**:171-182.
545 Brooker, S., A. C. Clements, and D. A. Bundy. 2006. Global epidemiology, ecology and control
546 of soil-transmitted helminth infections. Advances in Parasitology **62**:221-261.
547 Brookes, V. J., S. Durr, and M. P. Ward. 2019. Rabies-induced behavioural changes are key to
548 rabies persistence in dog populations: Investigation using a network-based model. PLoS
549 Neglected Tropical Diseases **13**:e0007739.
550 Brutsch, T., G. Jaffuel, A. Vallat, T. C. Turlings, and M. Chapuisat. 2017. Wood ants produce a
551 potent antimicrobial agent by applying formic acid on tree-collected resin. Ecology &
552 Evolution **7**:2249-2254.
553 Buck, J. C., and W. I. Lutterschmidt. 2016. Parasite abundance decreases with host density:
554 evidence of the encounter-dilution effect for a parasite with a complex life cycle.
555 Hydrobiologia **784**:201-210.

Bull, J. J., and A. S. Lauring. 2014. Theory and empiricism in virulence evolution. PLoS Pathogens **10**:e1004387.

Cable, J., I. Barber, B. Boag, A. R. Ellison, E. R. Morgan, K. Murray, E. L. Pascoe, S. M. Sait, A. J. Wilson, and M. Booth. 2017. Global change, parasite transmission and disease control: lessons from ecology. Philosophical Transactions of the Royal Society B **372**.

Chernin, J. 2000. Parasite examples grouped according to life-cycle. Parasitology. Taylor & Francis, Inc., New York.

Chretien, E., J. De Bonville, J. Guitard, S. A. Binning, E. Melis, A. Kack, A. Cote, M. Gradito, A. Papillon, V. Thelamon, M. Levet, and M. Barou-Dagues. 2023. Few studies of wild animal performance account for parasite infections: A systematic review. Journal of Animal Ecology. **92**:794-806.

Clinchy, M., M. J. Sheriff, L. Y. Zanette, and R. Boonstra. 2013. Predator-induced stress and the ecology of fear. Functional Ecology **27**:56-65.

Cremer, S., D. Pull, and M. A. Furst. 2018. Social immunity: Emergence and evolution of colony-level disease protection. Annual Review of Entomology **63**:105-123.

Cressler, C. E., L. D. Mc, C. Rozins, V. D. H. J, and T. Day. 2016. The adaptive evolution of virulence: a review of theoretical predictions and empirical tests. Parasitology **143**:915-930.

Croft, D. P., M. Edenbrow, S. K. Darden, I. W. Ramnarine, C. van Oosterhout, and J. Cable. 2011. Effect of gyrodactylid ectoparasites on host behaviour and social network structure in guppies Poecilia reticulata. Behavioral Ecology and Sociobiology **65**:2219-2227.

Curtis, V. A. 2014. Infection-avoidance behaviour in humans and other animals. Trends in Immunology **35**:457-464.

578 Demandt, N., M. Praetz, R. Kurvers, J. Krause, J. Kurtz, and J. P. Scharsack. 2020. Parasite
579 infection disrupts escape behaviours in fish shoals. Proceedings of the Royal Society B:
580 Biological Sciences **287**:20201158.
581 Demandt, N., B. Saus, R. Kurvers, J. Krause, J. Kurtz, and J. P. Scharsack. 2018. Parasite-
582 infected sticklebacks increase the risk-taking behaviour of uninfected group members.
583 Proceedings of the Royal Society B: Biological Sciences **285**.
584 Doherty, J.-F., and B. Ruehle. 2020. An integrated landscape of fear and disgust: The evolution
585 of avoidance behaviors amidst a myriad of natural enemies. Frontiers in Ecology and Evolution
586 **8**.
587 Doherty, J. F., M. Milotic, A. Filion, and A. Eriksson. 2022. Host specificity and the
588 reproductive strategies of parasites. Parasitology **149**:534-541.
589 Evans, J. C., M. J. Silk, N. J. Boogert, and D. J. Hodgson. 2020. Infected or informed? Social
590 structure and the simultaneous transmission of information and infectious disease. Oikos
591 **129**:1271-1288.
592 Ezenwa, V. O., E. A. Archie, M. E. Craft, D. M. Hawley, L. B. Martin, J. Moore, and L. White.
593 2016a. Host behaviour-parasite feedback: An essential link between animal behaviour and
594 disease ecology. Proceedings of the Royal Society B: Biological Sciences **283**.
595 Ezenwa, V. O., R. R. Ghai, A. F. McKay, and A. E. Williams. 2016b. Group living and pathogen
596 infection revisited. Current Opinion in Behavioral Sciences **12**:66-72.
597 Fayer, R. 1980. Epidemiology of protozoan infections: The coccidia. Veterinary Parasitology
598 **6**:75-103.
599 Fingerut, J. T., C. A. Zimmer, and R. K. Zimmer. 2003a. Larval swimming overpowers turbulent
600 mixing and facilitiates transmission of a marine parasite. Ecology **84**:2502-2515.

Fingerut, J. T., C. A. Zimmer, and R. K. Zimmer. 2003b. Patterns and processes of larval emergence in a estuarine parasite system. Biological Bulletin **205**:110-120.

Foster, W. A., and J. E. Treherne. 1981. Evidence for the dilution effect in the selfish herd from fish predation on a marine insect. Nature **293**:466-467.

Freeland, W. J. 1976. Pathogens and the evolution of primate sociality. Biotropica **8**:12-24.

Friant, S., T. E. Ziegler, and T. L. Goldberg. 2016. Changes in physiological stress and behaviour in semi-free-ranging red-capped mangabeys (*Cercocebus torquatus*) following antiparasitic treatment. Proceedings of the Royal Society B: Biological Sciences **283**.

Ghosh, B., H. Lal, and A. Srivastava. 2015. Review of bioaerosols in indoor environment with special reference to sampling, analysis and control mechanisms. Environment International **85**:254-272.

Giles, N. 1983. Behavioural effects of the parasite *Schistocephalus solidus* (Cestoda) on an intermediate host, the three-spined stickleback, *Gasterosteus aculeatus* L. Animal Behaviour **31**:1192-1194.

Godfrey, S. S., and R. Poulin. 2022. Host manipulation by parasites: From individual to collective behavior. Page 207-222 *in* V. Ezenwa, S. M. Altizer, and R. Hall, editors. Animal Behavior and Parasitism. Oxford University Press.

Grear, D. A., L. T. Luong, and P. J. Hudson. 2013. Network transmission inference: Host behavior and parasite life cycle make social networks meaningful in disease ecology. Ecological Applications **23**:1906-1914.

Gross, T., C. J. D'Lima, and B. Blasius. 2006. Epidemic dynamics on an adaptive network. Physical Review Letters **96**:208701.

623 Hafer, N. 2016. Conflicts over host manipulation between different parasites and pathogens:
624 Hart, B. L. 1994. Behavioural defense against parasites: interaction with parasite invasiveness.
625 Parasitology **109**:S139-S151.
626 Hart, L. A., and B. L. Hart. 2021. How Does the Social Grouping of Animals in Nature Protect
627 Against Sickness? A Perspective. Frontiers in Behavioral Neuroscience **15**:672097.
628 Hawley, D. M., and V. O. Ezenwa. 2022. Parasites, host behavior, and their feedbacks. Pages 15-
629 32 *in* V. Ezenwa, S. M. Altizer, and R. Hall, editors. Animal Behavior and Parasitism. Oxford
630 University Press.
631 Hernandez, A. D., and M. V. K. Sukhdeo. 1995. Host grooming and the transmission strategy of
632 *Heligmosomoides polygyrus*. Journal of Parasitology **81**:865-869.
633 Hillgarth, N. 1996. Ectoparasite transfer during mating in ring-necked pheasants *Phasianus*
634 *colchicus*. Journal of Avian Biology **27**:260-262.
635 Hobart, B. K., W. E. Moss, T. McDevitt-Galles, T. E. Stewart Merrill, and P. T. J. Johnson.
636 2022. It's a worm-eat-worm world: Consumption of parasite free-living stages protects hosts
637 and benefits predators. Journal of Animal Ecology **91**:35-45.
638 Hopkins, S. R., A. E. Fleming-Davies, L. K. Belden, J. M. Wojdak, and N. Golding. 2020.
639 Systematic review of modelling assumptions and empirical evidence: Does parasite
640 transmission increase nonlinearly with host density? Methods in Ecology and Evolution
641 **11**:476-486.
642 Hutchings, M. R., I. Kyriazakis, T. G. Papachristou, I. J. Gordon, and F. Jackson. 2000. The
643 herbivores´ dilemma: Trade-offs between nutrition and parasitism in foraging decisions.
644 Oecologia **124**:242-251.

Iritani, R., and Y. Iwasa. 2014. Parasite infection drives the evolution of state-dependent dispersal of the host. Theoretical Population Biology **92**:1-13.

Jolles, J. W., A. J. King, and S. S. Killen. 2020a. The role of individual heterogeneity in collective animal behaviour. Trends in Ecology & Evolution **35**:278-291.

Jolles, J. W., G. P. F. Mazue, J. Davidson, J. Behrmann-Godel, and I. D. Couzin. 2020b. *Schistocephalus* parasite infection alters sticklebacks' movement ability and thereby shapes social interactions. Scientific Reports **10**:12282.

Kavaliers, M., and E. Choleris. 2018. The role of social cognition in parasite and pathogen avoidance. Philosophical Transactions of the Royal Society B **373**.

Kavaliers, M., E. Choleris, and D. D. Colwell. 2001. Learning from others to cope with biting flies: Social learning of fear-induced conditioned analgesia and active avoidance. Behavioral Neuroscience **115**:661-674.

Kavaliers, M., and D. D. Colwell. 1995. Discrimination by female mice between the odours of parasitized and non-parasitized males. Proceedings of the Royal Society B: Biological Sciences **261**:31-35.

Kavaliers, M., D. D. Colwell, and E. Choleris. 2003. Learning to fear and cope with a natural stressor: Individually and socially acquired corticosterone and avoidance responses to biting flies. Hormones and Behavior **43**:99-107.

Kavaliers, M., D. D. Colwell, K. P. Ossenkopp, and T. S. Perrot-Sinal. 1997. Altered responses to female odors in parasitized male mice: neuromodulatory mechanisms and relations to female choice. Behavioral Ecology and Sociobiology **40**:373-384.

Kiesecker, J. M., D. K. Skelly, K. H. Bear, and E. Preisser. 1999. Behavioral reduction of infection risk. Proceedings of the National Academy of Sciences **96**:9165-9168.

668 Killen, S. S., S. Marras, L. Nadler, and P. Domenici. 2017. The role of physiological traits in
669 assortment among and within fish shoals. Philosophical Transactions of the Royal Society B
670 **372**.
671 Kohlmeier, P., K. Hollander, and J. Meunier. 2016. Survival after pathogen exposure in group-
672 living insects: don't forget the stress of social isolation! Journal of Evolutionary Biology
673 **29**:1867-1872.
674 Konrad, M., M. L. Vyleta, F. J. Theis, M. Stock, S. Tragust, M. Klatt, V. Drescher, C. Marr, L.
675 V. Ugelvig, and S. Cremer. 2012. Social transfer of pathogenic fungus promotes active
676 immunisation in ant colonies. PLoS Biology **10**:e1001300.
677 Krause, J., P. Reeves, and D. Hoare. 1998. Positioning Behaviour in Roach Shoals: The Role of
678 Body Length and Nutritional State. Behaviour **135**:1031-1039.
679 Krause, J., and G. D. Ruxton. 2002. Living in Groups. Oxford University Press, Oxford.
680 Kutz, S. J., E. P. Hoberg, P. K. Molnar, A. Dobson, and G. G. Verocai. 2014. A walk on the
681 tundra: Host-parasite interactions in an extreme environment. International Journal for
682 Parasitology: Parasites and Wildlife **3**:198-208.
683 Lafferty, K., and A. Kuris. 2002. Trophic strategies, animal diversity and body size. Trends in
684 Ecology & Evolution **17**:507-513.
685 Leu, S. T., P. M. Kappeler, and C. M. Bull. 2010. Refuge sharing network predicts ectoparasite
686 load in a lizard. Behav Ecol Sociobiol **64**:1495-1503.
687 Lloyd-Smith, J. O., P. C. Cross, C. J. Briggs, M. Daugherty, W. M. Getz, J. Latto, M. S.
688 Sanchez, A. B. Smith, and A. Swei. 2005a. Should we expect population thresholds for wildlife
689 disease? Trends in Ecology & Evolution **20**:511-519.

690 Lloyd-Smith, J. O., S. J. Schreiber, P. E. Kopp, and W. M. Getz. 2005b. Superspreading and the
691 effect of individual variation on disease emergence. Nature **438**:355-359.
692 Lopes, P. C., P. Block, and B. Konig. 2016. Infection-induced behavioural changes reduce
693 connectivity and the potential for disease spread in wild mice contact networks. Scientific
694 Reports **6**:31790.
695 Lopes, P. C., S. S. French, D. C. Woodhams, and S. A. Binning. 2021. Sickness behaviors across
696 vertebrate taxa: proximate and ultimate mechanisms. Journal of Experimental Biology **224**.
697 Lopes, P. C., S. S. French, D. C. Woodhams, and S. A. Binning. 2022. Infection avoidance
698 behaviors across vertebrate taxa: Patterns, processes, and future directions. Page 237-256 *in* V.
699 Ezenwa, S. M. Altizer, and R. Hall, editors. Animal Behavior and Parasitism. Oxford
700 University Press.
701 Madelaire, C. B., R. José da Silva, and F. Ribeiro Gomes. 2013. Calling behavior and parasite
702 intensity in treefrogs*, Hypsiboas prasinus*. Journal of Herpetology **47**:450-455.
703 Mansuy, J. M., M. Dutertre, C. Mengelle, C. Fourcade, B. Marchou, P. Delobel, J. Izopet, and G.
704 Martin-Blondel. 2016. Zika virus: High infectious viral load in semen, a new sexually
705 transmitted pathogen? The Lancet Infectious Diseases **16**.
706 May, R. M., and R. M. Anderson. 1979. Population biology of infectious diseases: Part II. Nature
707 **280**:455-461.
708 McCabe, C. M., S. M. Reader, and C. L. Nunn. 2015. Infectious disease, behavioural flexibility
709 and the evolution of culture in primates. Proceedings of the Royal Society B: Biological
710 Sciences **282**:20140862.
711 McCallum, H., N. Barlow, and J. Hone. 2001. How should pathogen transmission be modelled?
712 Trends in Ecology & Evolution **16**:295-300.

Mistrick, J., M. L. J. Gilbertson, L. A. White, and M. E. Craft. 2022. Constructing animal networks for parasite transmission inference. Pages 53-70 *in* V. Ezenwa, S. M. Altizer, and R. Hall, editors. Animal Behavior and Parasitism. Oxford University Press.

Mitchell, J., M. A. Cant, E. I. K. Vitikainen, and H. J. Nichols. 2017. Smelling fit: scent marking exposes parasitic infection status in the banded mongoose. Current Zoology **63**:237-247.

Mooring, M. S., and B. L. Hart. 1992. Animal grouping for protection from parasites: Selfish herd and encounter-dilution effects. Behaviour **123**:173-193.

Moretti, E. H., B. Titon, Jr., C. B. Madelaire, R. de Arruda, T. Alvarez, and F. R. Gomes. 2017. Behavioral, physiological and morphological correlates of parasite intensity in the wild Cururu toad (Rhinella icterica). International Journal for Parasitology: Parasites and Wildlife **6**:146-154.

Morrell, L. J., and W. L. Romey. 2008. Optimal individual positions within animal groups. Behavioral Ecology **19**:909-919.

Nadler, L. E., S. A. Adamo, D. M. Hawley, and S. A. Binning. 2023. Mechanisms and consequences of infection-induced phenotypes. Functional Ecology **37**:796-800.

Newey, S., and S. Thirgood. 2004. Parasite-mediated reduction in fecundity of mountain hares. Proceedings of the Royal Society B: Biological Sciences **271 Suppl 6**:S413-415.

Newsom, R. M., P. N. P. Mella, and T. E. Franklin. 1973. Observations on the numbers of the tick *Rhipicephalus appendiculatus* on the ears of zebu cattle in relation to hierarchical status in the herd. Tropical Animal Health and Production **5**:281-283.

Nouri, K., and D. T. Blumstein. 2019. Parasites are associated with noisy alarm calls. Frontiers in Ecology and Evolution **7**.

735 Øverli, Ø., and I. B. Johansen. 2019. Kindness to the final host and vice versa: A trend for
736 parasites providing easy prey? Frontiers in Ecology and Evolution **7**.
737 Parker, B., J. R. Britton, I. D. Green, F. Amat-Trigo, and D. Andreou. 2023. Parasite infection
738 but not chronic microplastic exposure reduces the feeding rate in a freshwater fish.
739 Environmental Pollution **320**:121120.
740 Parker, G. A., M. A. Ball, and J. C. Chubb. 2015. Evolution of complex life cycles in trophically
741 transmitted helminths. I. Host incorporation and trophic ascent. Journal of Evolutionary
742 Biology **28**:267-291.
743 Patterson, J. E., and K. E. Ruckstuhl. 2013. Parasite infection and host group size: a meta-
744 analytical review. Parasitology **140**:803-813.
745 Pedersen, A. B., and A. Fenton. 2007. Emphasizing the ecology in parasite community ecology.
746 Trends in Ecology & Evolution **22**:133-139.
747 Penn, D., and W. K. Potts. 1998. Chemical signals and parasite-mediated sexual selection.
748 Trends in Ecology & Evolution **13**:391-396.
749 Poirotte, C., F. Massol, A. Herbert, E. Willaume, P. M. Bomo, P. M. Kappeler, and M. J. E.
750 Charpentier. 2017. Mandrills use olfaction to socially avoid parasitized conspecifics. Science
751 Advances **3**:e1601721.
752 Poulin, R., B. R. Krasnov, and D. Mouillot. 2011. Host specificity in phylogenetic and
753 geographic space. Trends Parasitol **27**:355-361.
754 Poulin, R., D. J. Marcogliese, and J. D. McLaughlin. 1999. Skin-penetrating parasites and the
755 release of alarm substances in juvenile rainbow trout. Journal of Fish Biology **55**:47-53.

Rahn, A. K., D. A. Hammer, and T. C. M. Bakker. 2015. Experimental infection with the directly transmitted parasite *Gyrodactylus* influences shoaling behaviour in sticklebacks. Animal Behaviour **107**:253-261.

Reichert, M. S., M. G. Bolek, and E. A. McCullagh. 2023. Parasite effects on receivers in animal communication: Hidden impacts on behavior, ecology, and evolution. Proceedings of the National Academy of Sciences **120**:e2300186120.

Reynolds, M., F. A. Hockley, C. A. M. E. Wilson, and J. Cable. 2019. Assessing the effects of water flow rate on parasite transmission amongst a social host. Hydrobiologia **830**:201-212.

Rifkin, J. L., C. L. Nunn, and L. Z. Garamszegi. 2012. Do animals living in larger groups experience greater parasitism? A meta-analysis. American Naturalist **180**:70-82.

Ritchie, K. L., V. T. Vredenburg, S. Chaukulkar, H. M. Butler, and A. G. Zink. 2021. Social group size influences pathogen transmission in salamanders. Behavioral Ecology and Sociobiology **75**:136.

Robar, N., D. L. Murray, and G. Burness. 2011. Effects of parasites on host energy expenditure: the resting metabolic rate stalemate. Canadian Journal of Zoology **89**:1146-1155.

Roche, B., J. M. Drake, and P. Rohani. 2011. The curse of the Pharaoh revisited: Evolutionary bi-stability in environmentally transmitted pathogens. Ecol Lett **14**:569-575.

Rosengaus, R. B., C. Jordan, M. L. Lefebvre, and J. F. Traniello. 1999. Pathogen alarm behavior in a termite: A new form of communication in social insects. Naturwissenschaften **86**:544-548.

Rózsa, L., J. Reiczigel, and G. Majoros. 2000. Quantifying parasites in samples of hosts. Journal of Parasitology **86**:228-232, 225.

Sah, P., S. T. Leu, P. C. Cross, P. J. Hudson, and S. Bansal. 2017. Unraveling the disease consequences and mechanisms of modular structure in animal social networks. Proceedings of the National Academy of Sciences **114**:4165-4170.

Sauvage, F., M. Langlais, and D. Pontier. 2007. Predicting the emergence of human hantavirus disease using a combination of viral dynamics and rodent demographic patterns. Epidemiology & Infection **135**:46-56.

Seppälä, O., A. Karvonen, and E. T. Valtonen. 2008. Shoaling behaviour of fish under parasitism and predation risk. Animal Behaviour **75**:145-150.

Shaw, L. B., and I. B. Schwartz. 2008. Fluctuating epidemics on adaptive networks. Physical Review E **77**:066101.

Sih, A., O. Spiegel, S. Godfrey, S. Leu, and C. M. Bull. 2018. Integrating social networks, animal personalities, movement ecology and parasites: A framework with examples from a lizard. Animal Behaviour **136**:195-205.

Smith, M. J., S. Telfer, E. R. Kallio, S. Burthe, A. R. Cook, X. Lambin, and M. Begon. 2009. Host-pathogen time series data in wildlife support a transmission function between density and frequency dependence. Proceedings of the National Academy of Sciences **106**:7905-7909.

Stephenson, J. F., S. E. Perkins, and J. Cable. 2018. Transmission risk predicts avoidance of infected conspecifics in Trinidadian guppies. Journal of Animal Ecology **87**:1525-1533.

Stockmaier, S., Y. Ulrich, G. F. Albery, S. Cremer, and P. C. Lopes. 2023. Behavioural defences against parasites across host social structures. Functional Ecology **37**:809-820.

Stroeymeyt, N., A. V. Grasse, A. Crespi, D. P. Mersch, S. Cremer, and L. Keller. 2018. Social network plasticity decreases disease transmission in a eusocial insect. Science **362**:941-945.

799 Stumbo, A. D., C. T. James, C. P. Goater, B. D. Wisenden, and S. Cotter. 2012. Shoaling as an
800 antiparasite defence in minnows (*Pimephales promelas*) exposed to trematode cercariae.
801 Journal of Animal Ecology **81**:1319-1326.
802 Thrall, P. H., J. Antonovics, and D. W. Hall. 1993. Host and pathogen coexistence in sexuallhy
803 transmitted and vector-borne diseases characterized by frequency-dependent disease
804 transmission. American Naturalist **142**:543-552.
805 Turner, W. C., and W. M. Getz. 2010. Seasonal and demographic factors influencing
806 gastrointestinal parasitism in ungulates of etosha national park. Journal of Wildlife Diseases
807 **46**:1108-1119.
808 VanderWaal, K. L., and V. O. Ezenwa. 2016. Heterogeneity in pathogen transmission:
809 Mechanisms and methodology. Functional Ecology **30**:1606-1622.
810 Vindas, M. A., H. L. E. Midttun, L. E. Nadler, R. Fontaine, F. A. Weltzien, Ø. Øverli, and I. B.
811 Johansen. 2023. Brain-infecting parasites leave lasting effects on behaviour even in resistant
812 hosts. Functional Ecology **37**:852-859.
813 Viney, M. E., and A. L. Graham. 2013. Patterns and processes in parasite co-infection. Advances
814 in Parasitology **82**:321-369.
815 Ward, A., and M. Webster. 2016. Sociality: The Behaviour of Group-Living Animals. Springer,
816 Switzerland.
817 Weinstein, S. B., J. C. Buck, and H. S. Young. 2018. A landscape of disgust. Science **359**:1213-
818 1214.
819 White, L. A., J. D. Forester, and M. E. Craft. 2017. Using contact networks to explore
820 mechanisms of parasite transmission in wildlife. Biological Reviews of the Cambridge
821 Philosophical Society **92**:389-409.

822 Wilson, A. J., E. R. Morgan, M. Booth, R. Norman, S. E. Perkins, H. C. Hauffe, N. Mideo, J.
823 Antonovics, H. McCallum, and A. Fenton. 2017. What is a vector? Philosophical Transactions
824 of the Royal Society B **372**.
825 Woodroffe, R., C. A. Donnelly, G. Wei, D. R. Cox, F. J. Bourne, T. Burke, R. K. Butlin, C. L.
826 Cheeseman, G. Gettinby, P. Gilks, S. Hedges, H. E. Jenkins, W. T. Johnston, J. P. McInerney,
827 W. I. Morrison, and L. C. Pope. 2009. Social group size affects *Mycobacterium bovis* infection
828 in European badgers (*Meles meles*). Journal of Animal Ecology **78**:818-827.