One hand washes the other: cooperation and conflict in hygiene and immunity

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Perspective: Maximum of 7500 words and 10 figures, tables or text boxes.

Abstract

In humans and wild animals, pathogens impose costs on both the individual and the social group as a whole. To minimise these costs, group-living species have evolved many hygienic and immune traits that benefit from cooperation between individuals, thereby subjecting them to the laws of social evolution. Such social contracts include reciprocal grooming, altruistic self-isolation, spiteful treatment of infected individuals, and costly immune resistance responses. In highly social animals such as eusocial insects, these traits often present as complex "collective" or "social" immune systems. Even the expression of individual-level phenotypes such as sickness behaviours and immunological tolerance can depend heavily on social context, and understanding whether such responses present a benefit for the individual, the group, or both can be critical for understanding their functions and eco-evolutionary consequences. As yet, our consideration of these traits has mostly concerned individuals, or collective immunity in eusocial insect taxa. Consequently, their broader epidemiological consequences and implications for the evolution of sociality are relatively unclear. Here, I describe a wide number of socially evolved hygienic and immune traits in wild animals, both in social insects and in other taxa. I outline the problems that emerge when evolving and enforcing these anti-disease functions, discussing the conflicts that arise and their implications for evolutionary and cultural transitions in social complexity, and their potential analogues in human public health.

Keywords: Disease ecology, Ecoimmunology, Social evolution, Social behaviour, Public goods, Cooperation, Conflict

Introduction

Animal societies feature a delicate balance of cooperation and conflict. Pro-social ("cooperative") traits are generally costly to the focal individual but beneficial to others, which motivates individuals to "cheat" and minimise their contributions to public goods where possible (Hamilton 1963). Whether a given cooperative trait can evolve depends on the balance of cost to the individual and benefit to others, moderated by genetic relatedness to those individuals (Hamilton 1963, 1964b, a; West *et al.* 2001) and a series of intricate enforcement mechanisms (Ågren *et al.* 2019). Parasites are an important selective force in wild populations, and are often costly to infected individuals (Graham *et al.* 2011a; Tompkins *et al.* 2011) and to social groups as a whole (Kappeler *et al.* 2015; Ezenwa *et al.* 2016b; Schmid-Hempel 2021). As such, animals have evolved a wide range of behavioural and immunological methods for avoiding infection or reducing its impact. Because these traits themselves are often costly to the infected individual while benefitting its conspecifics (Graham *et al.* 2005, 2021; Buck *et al.* 2018; Hart & Hart 2018), investment in such traits (i.e., "**immune cooperation**") will depend on social context and patterns of relatedness. Reciprocally, failing to invest in such traits is analogous to "**immune cheating**", foisting the burden of infection on conspecifics while protecting one's own health. For example, Trinidadian guppies (*Poecilia reticulata*) become more sociable when infected with ectoparasites, which may function to reduce one's own burden by spreading the parasites to others (Reynolds *et al.* 2018). Due to this tension between individual and group priorities, cooperative immune traits are likely subject to the laws of social evolution – that is, immunity is a public good *sensu stricto*. Moreover, because infectious disease can be a major cost of sociality (Cote & Poulin 1995; Altizer *et al.* 2003), disease-related cooperation may grow in importance with increasing social complexity, potentially representing an important prerequisite to the evolution of higher sociality. Here, I outline the social contracts that exist around infection and immunity in wild animals. I provide examples for each, where they exist, and outline how they could be influenced by social evolution.

Notably, much interest in the social evolution of immunity has been focussed on (eu)social insects (bees, wasps, ants, and termites), rather than being generalised across animals, and without necessarily using social evolution terminology or kin selection frameworks. These insects have incredibly advanced, pervasive social immune systems that evolved in highly cooperative scenarios to decrease the risk of disease for the colony as a whole (Cremer *et al.* 2007; Cotter & Kilner 2010; Meunier 2015; Simone-Finstrom 2017; Schmid-Hempel 2021). Many such functions so closely mirror within-individual immune systems that they are thought to comprise an important component of transitions to greater social complexity (Pull & McMahon 2020). This focus on (eu)social insects is understandable, because in addition to their complexity, they are tractable and easy to study, and for decades they have been a foundational study system for social evolution (Hamilton 1964b; Ratnieks & Visscher 1989; Griffin & West 2003). Additionally, eusocial insects have long been important for the study of infectious disease in general: For example, work on ant pathogens has revealed how

behavioural traits and network structure determine transmission (e.g. (Heinze & Walter 2010; Stroeymeyt *et al.* 2018; Alciatore *et al.* 2021)). Similarly, much work has focussed on bees and their pathogens to help identify the cause of widespread bee decline (e.g. (Cox-Foster *et al.* 2007)). Given these foci, it is unsurprising that eusocial insects have been a prominent study system for social immunity; however, the concept is worthy of further generalisation, which will benefit from recognising social immunity's analogues and primitive forms in other taxa.

Importantly, it has previously been noted that "personal immunity" and "social immunity" exist on a continuum (Cotter & Kilner 2010), with many intermediate forms between individual and communal immune functions (e.g. with communal antimicrobials; (Baracchi *et al.* 2012b)). While other taxa may not have evolved such complex social immune systems as eusocial insects, each individual's disease burden is nevertheless heavily dependent on its social context and the disease burden of those around it, which create conflict and opportunities for cooperation *sensu stricto*. For example, each individual that avoids or resists disease for selfish reasons may ultimately help the group by extension, because they are less likely to transmit the infection to their compatriots (Frank 1998). Encouragingly, theoretical models have demonstrated that immune responses could respond to kin selection (Frank 1998; Best *et al.* 2011; Horns & Hood 2012), and the tension between individual and social immunity has been linked with the popular concept of "herd immunity", where population-level resistance is common enough that individual-level immunity becomes less necessary (Cotter & Kilner 2010). Similarly, many have discussed general benefits of sociality for disease (Loehle 1995; Kappeler *et al.* 2015; Ezenwa *et al.* 2016b; Hart & Hart 2021), but without delving into the conflict that arises between the individual and the group in deciding who provides these benefits. Thus, the social evolution of disease could benefit from a more general formulation. As well as informing the evolution of social systems and disease resistance in wildlife, understanding these fundamental phenomena could inform public health policies in human societies, which likewise rely on cooperation and shared hygiene goals.

Here, I present a framework for considering the social evolution of immunity and hygienic behaviour (Figure 1-2), using social evolution terms as defined in (Ågren *et al.* 2019). To expand on substantial prior work discussing disease-related benefits of sociality (Loehle 1995; Kappeler *et al.* 2015; Ezenwa *et al.* 2016b; Hart & Hart 2021) and collective immunity in eusocial insects (Cremer *et al.* 2007; Cotter & Kilner 2010; Meunier 2015; Simone-Finstrom 2017; Schmid-Hempel 2021), I focus less on the benefits of these actions to the group, and more on the costs to the individual – and therefore the potential conflict over *which* individual performs them. For a range of animal systems, I give examples of individual-level "cooperative" acts, and equivalent "cheating" acts, across all levels of social complexity (Table 1), and I discuss evidence for the roles of kin selection, reciprocity, and enforcing in evolving such traits. I discuss specific difficulties for evolving and enforcing cooperative anti-disease traits, and for researchers studying them, and I identify general similarities between these processes and those acting in human societies. Ultimately, I focus on developing a

generalisable framework for the social evolution of immunity, expanding the focus on taxa other than social insects. I argue that sophisticated social immune systems have primitive analogues and homologues spread across the tree of life, many of which deserve explicit consideration as (somewhat) socially evolved traits.

Figure 1: The social evolution of a costly anti-disease trait.

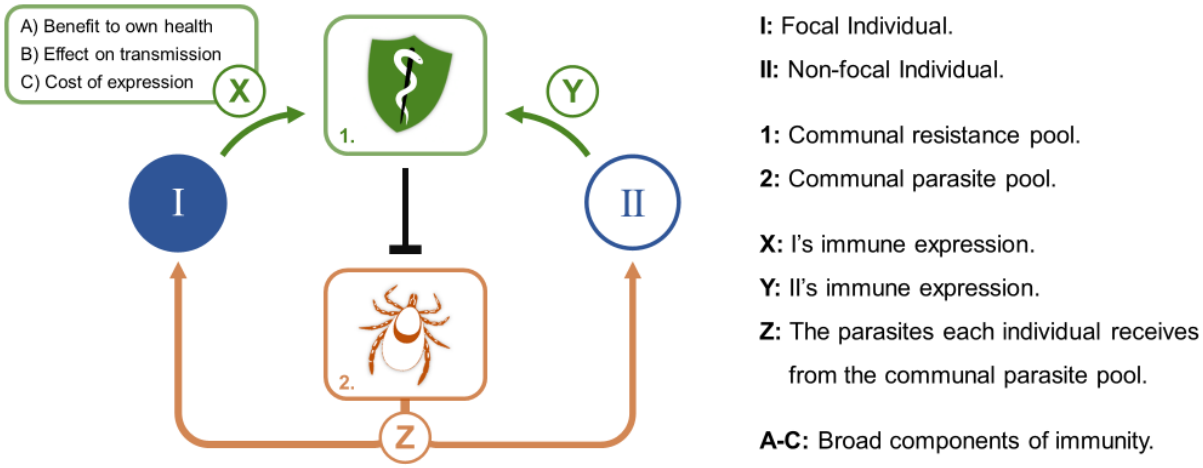

Figure 1: General framework for the social evolution of a costly anti-disease trait. The fitness of a focal individual (I) depends its own interactions with immunity (1) and parasites (2), and on those of another individual (II) who represents the average for the rest of the population. In this scenario, both individuals contribute separately to the communal resistance pool (X and Y), which acts to reduce the communal parasite pool (Z). Both individuals receive equal numbers of parasites from the communal parasite pool. Because the number of parasites in the population is dependent on the communal resistance pool (black blunt line), individuals could "cheat" by attempting to under-invest in resistance (X), but still benefitting from those of others (Y), or they could "cooperate" by investing in resistance proportionally. Immunity is also decomposed here into three traits: benefit to self (A), benefit to others (B), and cost of expression (C); the only immune trait expressed by individual (II) that is of relevance to the focal individual (I) is its effect on transmission. Although I refer to immune resistance here, the same might be true of investment in a hygienic behaviour such as grooming, or in any other trait that might reduce the parasites in the population. Below, I decompose this system to illustrate how individual (I) could maximise its inclusive fitness. In many immune responses, individuals will not support their own health (A) and contribute to the resistance pool (B) separately; instead, the two will be mechanistically linked in some ways, and the individual will have a degree of prioritisation when allocating resources between the two – i.e., a ratio A:B, where a large ratio represents a large investment in personal health (i.e., **self-serving immunity**) and a small ratio represents a large allocation towards decreasing parasite transmission in the population as a whole (i.e., **altruistic immunity**).

Figure 2: Maximising inclusive fitness with an anti-disease trait.

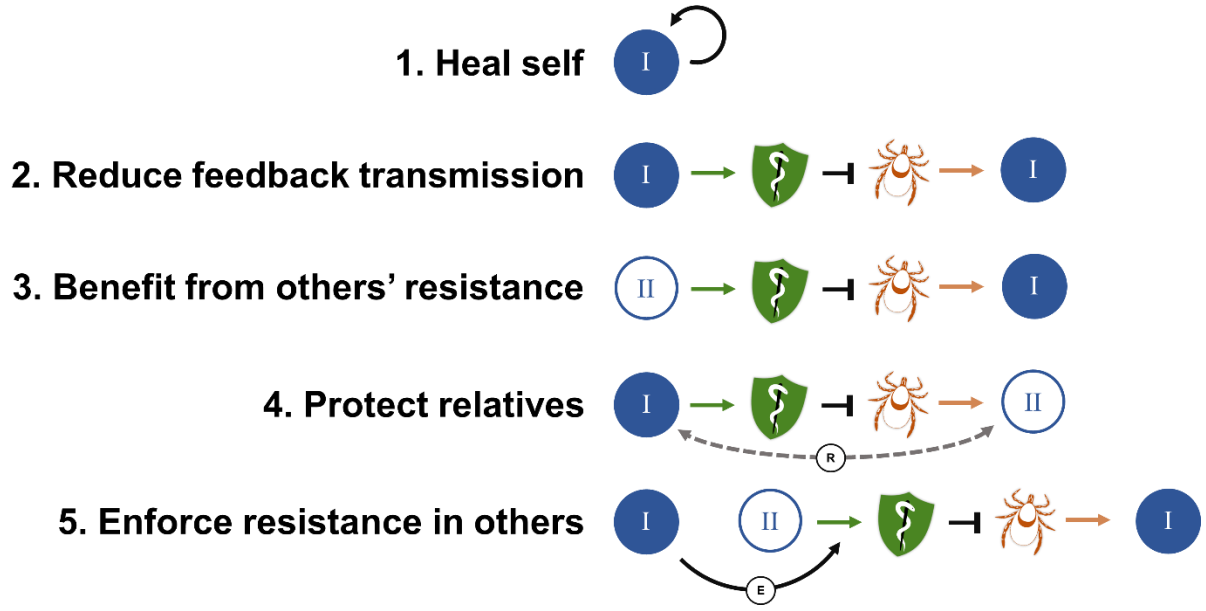

Figure 2: Framework for maximising inclusive fitness given the presence of a parasite and an anti-parasite trait. Given the complex linkages between the immunity, parasites, and enforcing of multiple individuals, there are a number of pathways through which a focal individual (I) can maximise its inclusive fitness relative to the rest of the population (II). 1) Invest in healing itself in a way that could be self-serving, accounting for the costs of parasitism but without reducing transmission to the population. 2) Invest in reducing its transmission to the pool of parasites, which will reduce its future exposure through feedback mechanisms. 3) Benefit from other individuals reducing their transmission to the parasite pool, reducing its exposure and therefore its burden. 4) Invest in reducing its transmission to the pool of parasites, benefitting its close relatives (who share some high proportion of its genes), and therefore itself indirectly. 5) invest in ensuring that individual (II) allocates appropriate resources to the resistance pool (i.e., enforcement). For example, this might involve ostracising or behaving aggressively towards individuals that are infected or failing to signal their disease status. Assuming that such enforcement is effective at reducing transmission relative to its resource costs, allocation of resources to it could increase fitness.

Table 1: Contingency table of socially evolved anti-parasite traits.

Trait	Effect on actor	Effect on recipient	Comment
Immune resistance	+/-	+	Depends on benefit of resistance *versus* tolerance, role in reducing parasite count, and correlation with parasite shedding.
Honest Signals	-	+	Constrained by necessarily honest signalling paradigms.
Self-isolation	+/-	+	Could be altruistic (avoid infecting conspecifics) or selfish (avoid persecution/outcompetition).
Avoidance	+/-	-/+	Avoids becoming infected, but expends energy and potentially results in suboptimal habitat use and loss of social benefits.
Ostracism	-	-	Involves avoiding or removing infected individuals, losing social benefits and potentially risking adverse interactions.
Persecution of immune cheaters	-	-	Involves actively removing cheating individuals, thereby expending energy and potentially risking own exposure to cheating individuals' pathogens.
Grooming	-/+	+	Expends energy and risks exposure to endoparasites, but reduces another's ectoparasite burden.
Caring for sick	-	+	Inherently involves directing energy or resources towards infected individuals.
Protective microbe sharing	~+	~+	Potentially mutually beneficial, but risks (over-)exposure to one another's pathogens.
Protective molecule sharing	-/+	+	Antimicrobial molecules benefit the group as a whole, but are costly to produce. Can serve personal immune functions if not shared with the group.
Collective physiology	+	+	Likely selfish herd effect serving to protect against heat-sensitive pathogens.

Table 1: Generalised prosocial (cooperative) anti-disease functions, each of which involves a degree of cooperation or conflict. The examples are arranged in the other of the text. Includes the cost and benefit to the actor and recipient, accompanied by notes about what factors modulate the balance of cost *versus* benefit. The top three entries generally involve an infected actor; in the rest, the actor is generally uninfected.

Social contracts in immunity and hygiene

Immune responses: resistance versus tolerance

Strong resistance responses, which generally seek to inhibit parasite replication and reduce burden, can be extremely costly (Sheldon & Verhulst 1996; Viney *et al.* 2005; Graham *et al.* 2011a, 2021). If resistance reduces the number of parasites shed to the rest of the population, investing in this cost can be construed as an altruistic act. Supporting this interpretation, (Frank 1998) demonstrated that inducible immune responses may become more likely with increasing relatedness. These results were corroborated in a dynamic, spatially explicit modelling framework by (Best *et al.* 2011), who showed that kin structuring and local transmission provoked greater expression of resistance responses. As such, resistance can be conceived as a "public good", where individuals contribute to a shared resistance pool, and individuals that contribute less to this pool nevertheless benefit from the immunity offered by others (Figure 1).

There is some empirical evidence supporting the idea that resistance is a socially evolved trait. For example, the immune response to phytohaemagglutinin (PHA) injection is substantially higher in cooperatively breeding birds than in their non-cooperatively-breeding relatives (Spottiswoode 2008). Similarly, antimicrobial compounds in burying beetles are the source of conflict over who invests in the social immune response and how much (Cotter *et al.* 2010). The greater expression of immunity in social scenarios may be facultative and individual-level, as with density-dependent prophylaxis (Wilson & Cotter 2009), or it may be obligate and between-species, as with the cooperatively breeding birds (Spottiswoode 2008). Between- and within-species variation in social structure could therefore be an important determinant of the balance between constitutive and inducible immunity.

Reciprocally, individuals that can maintain their own health while continuing to host and transmit pathogens (i.e., "tolerant" individuals (Råberg *et al.* 2009)) could represent a cost for those around them, opening themselves up to punishment and suffering indirect fitness costs. Although resistance has been widely considered in the evolution of the immune response (Frank 1998; Best *et al.* 2011; Débarre *et al.* 2012), tolerance has been less frequently examined. Tolerance has enjoyed a relatively recent increase in popularity in the literature (Raberg *et al.* 2009; Graham *et al.* 2011b), and is also less likely to evolve with spatially kin-structured models, implicating it as a socially evolved trait (Horns & Hood 2012). The relative expression of resistance *versus* tolerance will depend on the balance of costs and benefits for the individual *versus* the group, and is equivalent to the ratio of A:B in Figure 1. Providing evidence for this tradeoff will require more studies of kin structure alongside immune resistance, alongside fitness consequences to quantify tolerance (e.g. (Hayward *et al.* 2014)).

The immune system is inordinately complex, with many cells and effectors that contribute to a range of hard-to-predict consequences for each individual infection. Quantifying and comparing how investment in immunity varies across kin-selected contexts may be very difficult for these reasons,

but there are broad categories of responses that we might expect to see across such contexts. For example, individuals housed among close kin might be expected to invest more in strong inflammatory responses than in regulatory responses, compared to their conspecifics housed among non-kin. These different arms of immunity could be measured based on the expression of inflammatory cells *versus* those associated with healing and immune regulation. Quantifying the change in fitness associated with these responses in an experimental context could further support these mechanisms: for example, if those in a kin-heavy treatment exhibit more inflammatory immune responses when infected, and if those are associated with reduced fitness and reduced parasite shedding, this would provide strong evidence for the evolution of altruistic immunopathology. This could be further supplemented with models of the evolution of immunopathology, building on prior work (Cressler *et al.* 2015) but incorporating social structure in addition.

Honest signals of infection status

Truthful signalling is central to classical social evolution theory. Signalling about disease status and immune investment is likely to play an important role in social disease dynamics, by allowing conspecifics to make informed decisions about how to respond and behave in the signalling individual's company. For example, because sickness behaviours often involve highly costly processes like anorexia, sickness has been suggested as an altruistic signal to conspecifics (Shakhar & Shakhar 2015). Accordingly, there are many examples of honest disease signalling: for example, house mice (*Mus musculus*) produce less attractive sexual signals when treated with an immune agonist (Lopes & König 2016).

The equivalent non-cooperative act would be to hide one's disease status, which has also been demonstrated: for example, male zebra finches treated with an immune agonist become notably sick, but their sickness behaviours are reduced when they are in close proximity to a female; that is, they did not communicate their infection status honestly, choosing instead to hide it and to prioritise mating (Lopes *et al.* 2013). Disease has long been known to be linked to honest signalling, and many classical theories for the evolution of ostentatious signals (e.g. peacocks' elaborate tails) revolved around the difficulty of generating these costly traits while being heavily infected with parasites (Hamilton & Zuk 1982; Balenger & Zuk 2014). Relatedly, many ornaments are symmetrical and difficult to grow where parasites drive fluctuating asymmetry during development (Møller & Pomiankowski 1993; Watson & Thornhill 1994). Certain behaviours may also be harder to perform when infected: grooming, for example, is highly energetically expensive (Giorgi *et al.* 2001), and reduced grooming in sick individuals produces a dirty coat that might honestly signal infection status (Hart & Hart 2021). These facts likely constrain the evolution of signals around disease and reduce the possibility of cheating.

While these phenomena represent clear honest indicators of (lack of) infection, it is unclear whether there are signals that represent immune investment itself in the absence of infection. They may not be biologically realistic because host-parasite systems are highly unstable and often locked in a tight

coevolutionary cycle, where hosts evolve resistant phenotypes, which parasites quickly evolve the ability to infect, and so on (King *et al.* 2009). In these scenarios, purely-immune-based phenotypes may never be stable or reliably indicative of a host's resistance: that is, the only proof of effective immunity is lack of infection. All signals must work within the sensory limits of the detector (Dawkins & Guilford 1991), and thus there may be relatively few reliable indicators. Many systems use chemical cues produced by infection: for example, Caribbean spiny lobsters (*Panulirus argus*) detect chemicals that indicate infection with a socially transmitted virus (Behringer *et al.* 2006); honey bees' (*Apis mellifera*) cuticular hydrocarbons identify those with deformed wing virus (Baracchi *et al.* 2012a); and mandrills (*Mandrillus sphinx*) can smell individuals carrying gastrointestinal parasites (Poirotte *et al.* 2017). Importantly, parasites have often evolved sophisticated mechanisms for manipulating host behaviour and signalling to facilitate their own transmission (Poulin 2010); as such, it may be difficult to extricate the host's selfish signalling from its altruistic signalling and the signals produced by the parasite itself.

Honest signals of infection may have complex socioecological consequences when they affect signals that are used for other purposes. For example, it has been suggested that *Mycoplasma* infection in house finches (*Carpodacus mexicanus*) presents an "evolutionary trap" acting through signalling and competition (Bouwman & Hawley 2010). In this system, male house finches preferred to feed closer to infected individuals because sick individuals were less likely to be aggressive; as such, the finches were using non-aggressive behaviour as a positive signal that, while honestly signalling that the individual was sick, nevertheless resulted in its conspecifics becoming more easily infected (Bouwman & Hawley 2010). This also serves to illustrate the more general point that in many circumstances, when calculating the cost/benefit of a given action, animals have to rank disease avoidance alongside a range of other priorities like resource acquisition (Hutchings *et al.* 2006); in many such cases, disease may be the lesser of two evils.

Self-isolation, sickness, and suicide

An infected individual may altruistically reduce its contact with other individuals to reduce the risk of transmitting the infection to them, thereby benefitting the conspecifics. This act will have two main costs: the energy involved in the movement or separation behaviour itself, and the loss of the benefits of sociality – i.e., of other cooperative phenomena like group foraging and defence.

Disease-associated reductions in sociality are an important component of sickness behaviours, which could be construed as an altruistic trait (Shakhar & Shakhar 2015). However, sick individuals are also more generally lethargic; because sociality is closely related to movement and space use (Webber *et al.* 2022), sickness-associated reductions in sociality could be merely a byproduct of a selfish desire to conserve energy. Infection-driven changes in movement are known to alter the emergent social network, e.g. in sticklebacks (*Gasterosteus aculeatus*; (Jolles *et al.* 2020)). Similarly, although house mice exhibit strong sickness behaviours (Lopes *et al.* 2016), such behaviours are not modulated by the presence of kin, which is what we would expect if they had an altruistic function (Lopes *et al.*

2018). In vampire bats (*Desmodus rotundus*) closer kin relationships *reduced* the expression of sickness behaviours rather than *increasing* them, although this was likely heavily influenced by other benefits of social interactions (Stockmaier *et al.* 2020). Nevertheless, the expression of sickness behaviours does depend on social context, e.g. in zebra finches (Lopes *et al.* 2012). Assessing whether sickness behaviour is cooperative or self-serving will require wider testing of social modulation and fitness consequences in a range of species.

Self-ostracism (i.e., movement to an entirely different remote area) is a more obviously costly behavioural response to infection. For example, sick termites altruistically exit the nest and die, thereby reducing the probability that they will infect others (Rueppell *et al.* 2010). However, leaving could also benefit the infected individual, e.g. by avoiding competition or aggression from healthier individuals (McFarland *et al.* 2021). Sick ants likewise become unsociable, although it is less clear whether this act is to the individual costly (Bos *et al.* 2012). Separating altruistic self-removal from more self-serving reasons to leave will involve identifying whether a sick animal's prospects truly suffer when they leave the group and whether they would have been better had they stayed (i.e., whether the act has fitness costs), particularly if persecution of sick individuals is possible.

Avoidance and ostracism

If infected or immune-cheating individuals can be reliably identified, it may benefit their conspecifics to avoid or ostracise them to avoid infection (Buck *et al.* 2018; Weinstein *et al.* 2018; Townsend *et al.* 2020). For example, Caribbean spiny lobsters (*Panulirus argus*) avoid individuals infected with a socially transmitted virus (Behringer *et al.* 2006), even vacating valuable sheltering spots when an infected individual enters (Butler *et al.* 2015). Such avoidance is clearly costly, and often trades off with other valuable processes like resource acquisition (Hutchings *et al.* 2006; Buck *et al.* 2018; Weinstein *et al.* 2018), but because it heavily benefits the focal individual by allowing it to avoid infection, it is unclear whether it might be cooperative. Where groups engage in avoidance collectively, such behaviours may most closely resemble a "selfish herd" dynamic, where an apparently costly social trait emerges not as a result of altruism but because each individual involved actually receives a direct fitness benefit. As a parallel, selfish herd dynamics facilitate dilution of micropredator attacks (Mooring & Hart 1992), which may be comparable on some scales to parasite dilution effects. Mandrills will avoid directing grooming behaviour towards protozoan-infected individuals to avoid infection (Poirotte *et al.* 2017), but only when grooming non-kin (Poirotte & Charpentier 2020). This kin-dependent modulation may support grooming's role as a kin-selected hygienic trait.

Forcing cheaters or infected conspecifics to keep their distance may reduce the threat of parasites, depending on the parasite's transmission range. For example, Honey bees evict infected individuals (Conroy & Holman 2022) and ants will kill infected brood (Pull *et al.* 2018). Similarly, if parasitised Trinidadian guppies become more sociable specifically to spread their own burden around (Reynolds *et al.* 2018), it may be adaptive for the other members of the group to exclude parasitised members

from joining. Such active ostracism of immune cheaters is likely to be more costly for the enforcers than avoidance, because ostracising may require a costly investment in aggressive interactions or signals to motivate the cheater individual to leave. If these interactions involve a risk of direct contact (as is likely), they could present a further cost by risking exposure to the parasite. As such, the balance of avoidance *versus* ostracism may depend on the cheater's perceived infectiousness and the relative risk of exposure given an aggressive ostracism-provoking interaction. This choice could be further weighted by the value of the territory being inhabited, where low-quality habitats are more likely to be left (i.e., avoidance) and higher-quality ones are more likely to be defended (i.e., ostracism).

Persecution and punishment

Uninfected individuals may also enforce immune cooperation using generalised aggression, without necessarily using such aggression to motivate the cheaters to maintain their distance. For example, in vervets (*Chlorocebus pygerythrus*), individuals direct aggression towards experimentally generated sick conspecifics (McFarland *et al.* 2021). As with active ostracism (although probably more so), miscellaneous direct persecution is likely to expose the enforcing individual to parasites by forcing them to have risky aggressive interactions which are likely to involve substantial proximity, direct contact, and fluid and gas exchange. Even if the recipient individual is not infected, such fights are likely to involve a lot of exertion and potentially personal harm.

In some cases, punishment might extend to killing infected individuals to remove them from the population. For example, honey bees (*Apis mellifera*) detect and remove infected conspecifics to prevent them from infecting others in the colony (Baracchi *et al.* 2012a). This violence might be aimed at certain (classes of) individuals: for example, because young individuals are often heavily infected (Ashby & Bruns 2018), (Hart & Hart 2021) suggest that mothers may commit infanticide to pre-emptively dispose of them and prevent infection from spreading.

Animals suspected of being cheaters (or of unknown cooperation status) may undergo immune trials at the hands of the social group, making them prove their status as cooperators or suffer punishment. For example, (Hart & Hart 2021) discuss the example of dwarf mongooses (*Helogale parvula*) and their tendency to undergo prolonged trial periods before eventual acceptance into a new social group (Rasa 1987). They suggest that the extended period of stress and ostracism (which may last more than 30 days) could make an intruder sick if they are harbouring a latent infection, allowing the new group to identify and reject them as non-cooperators and save themselves from infection (Freeland 1976). The ability to survive this trial period without succumbing to infection may effectively represent a costly honest signal of immune cooperation that is robust to cheating (see above).

Grooming

Allogrooming (i.e. the selective removal of ectoparasites from conspecifics' skin) inherently combats ectoparasite infection, with important ecological and evolutionary consequences (Johnson *et al.* 2010; Wilson *et al.* 2020). For the grooming individual, the behaviour comes with obvious costs: for example, time spent grooming detracts from other important behaviours like vigilance (Mooring & Hart 1995). Similarly, although the grooming animal often consumes the parasites, the energy gained could be minor relative to the cost of performing the action (Giorgi *et al.* 2001; Lafferty *et al.* 2008; Johnson *et al.* 2010). Grooming is therefore a prime candidate for a cooperative hygienic trait, and there is plenty of evidence spread across a wide range of taxa that social contracts exist around allogrooming. For example, pairs of horses often groom one another simultaneously and break away at a sign of stopping (Shimada & Suzuki 2020); rats are more likely to groom those who have previously groomed them (Schweinfurth *et al.* 2017); chimpanzee grooming is elongated by reciprocity (Machanda *et al.* 2014); and immune-challenged ants are preferentially groomed and cared for by their nestmates (Alciatore *et al.* 2021).

Although nobody has yet directly investigated the disease-related consequences of socially evolved grooming contracts, evolutionary modelling has shown that the balance of infection and allogrooming can influence the evolution of social systems (Wilson *et al.* 2020). To date, the evolution of grooming has been investigated most intensely in humans and closely related primates (Dunbar 1991; Jaeggi *et al.* 2017; Takano & Ichinose 2018) and less across animals in general. Sampling bias could further influence the observed trends because social grooming is an obviously reciprocal behaviour that is easy to observe. In many cases, the costs and benefits of grooming are difficult to examine because grooming serves a lot of purely social functions unrelated to disease, like assessing mate quality (Stopka & Graciasová 2001) and quelling antagonism (Madden & Clutton-Brock 2009). Quantifying the consequences of allogrooming for individual disease status, and extricating them from their many other miscellaneous social effects, will be an important challenge for future empirical investigations.

Notably, grooming can risk exposure to some types of parasites while reducing conspecific burdens of ectoparasites. For example, mandrills are less likely to groom ectoparasites from an individual with endoparasite infection, implying that they risk contracting the infection by grooming said individual (Poirotte *et al.* 2017). In ants, nestmates contact individuals that have been exposed to fungal parasites and remove infectious material with their mouth by allogrooming (Konrad *et al.* 2012), which could likewise expose them to the parasite that they are removing – depending on transmission mode. This elevated exposure risk to certain parasites could represent another complex cost of grooming behaviour, whether grooming primarily serves a social function or reduces ectoparasite infection.

Caring for sick

Caring for infirm individuals is an advanced prosocial anti-disease trait that has been relatively rarely investigated in wild animals (Hart & Hart 2021), with examples largely restricted to eusocial insects.

For example, (Alciatore *et al.* 2021) showed that immune-challenged ants were "cared for" by their nestmates; *Lasius neglectus* ants exhibit care behaviours that are moderated by the carer's own susceptibility to infection (Konrad *et al.* 2018). Sick vampire bats (*Desmodus rotundus*) are groomed by their conspecifics despite reducing their grooming of others (Stockmaier *et al.* 2018) – although this represents a continuation of normal routines rather than a specific "caring" response designed to help them recover. In many animal species, caring for sick will often involve feeding the infected individual and altering movement to accommodate its lethargy, giving it the best chance to recover. Identifying these changes empirically will likely require a sophisticated understanding of the study subject's energy budgets. Food sharing is a common phenomenon in social animals (e.g. in chimpanzees (Silk 1978) and ants (Modlmeier *et al.* 2019)), and given its importance in vampire bats (Wilkinson 1984; Carter & Wilkinson 2013) this behaviour could become a model for cooperative anti-disease behaviour. This possibility is particularly attractive given that vampire bats are also fast becoming a model system for sickness behaviours and kin selection (Stockmaier *et al.* 2018). Conversely, dwarf mongoose move less to accommodate the energetic capacities of sick individuals (Rasa 1983; Hart & Hart 2021).

Sharing protective microbes and molecules

Because the microbiota play an important role in immune defence, social contracts could emerge where individuals share microbes. If microbiota can be acquired through social contact (Dill-McFarland *et al.* 2019; Raulo *et al.* 2021) and if those microbiota help protect against colonising parasites (Sassone-Corsi & Raffatellu 2015), individuals may seek contact to confer (and receive) increased resistance to infection through microbe sharing (Kappeler *et al.* 2015). If administered carefully, slow microbiome seeding could also drive exposure to low levels of pathogens that confer resistance without being as dangerous as a full exposure – i.e., "variolation" (Kappeler *et al.* 2015; Gandhi & Rutherford 2020). This process is doubtless important in eusocial insects – for example, *Lasius* ants actively immunise themselves with *Metarhizium* fungi from infected nestmates in a way that safely provides resistance (Konrad *et al.* 2012); however, it is unclear whether the same happens in less sophisticated social immune systems. In their discussion of dwarf mongooses attempting to join an unfamiliar group, (Hart & Hart 2021) suggest that extended periods of minimal (but greater-than-zero) contact rates could allow the new group to sample their microbiota under a relatively low-risk exposure scenario (and *vice versa*), allowing gradual acquisition of immunity for both parties (Hart & Hart 2021). Although intuitively reciprocal, this process could be very difficult to quantify for the individuals involved, and it is easy to imagine that the correct beneficial dose of pathogens is hard to measure, or to administer. Even in humans, although variolation is commonly suggested, it is often pointed out that guessing the correct (safe) infective dose is prohibitively difficult and risks full infection (Gandhi & Rutherford 2020). Purposeful microbe seeding may be difficult to evolve and enforce in animals for the same reasons that intentional exposure and variolation are rarely recommended in humans, and its relevance as a cooperative immune trait could be minimal for many societies.

On the other hand, social contracts do emerge in sharing antimicrobial *molecules*. For example, wasp venom plays an important antimicrobial role in social immunity in social *Polistes* wasps (Turillazzi *et al.* 2006); however, in primitively social Stenogastrine wasps the venom is only found on individuals' cuticles rather than on shared nest material, implicating venom more as a personal antimicrobial than as part of the shared immune system (Baracchi *et al.* 2012b). Venom may be costly for individuals to produce, such that in less-social Stenogastrines there would be irreconcilable conflict over who produced the communal resource for the nest, preventing its use in social immunity. In burying beetles (*Nicrophorus vespilloides*), mothers secrete an antimicrobial that protects their offspring, but the production of which has substantial associated fitness costs for the mother – which suggests that this molecule represents a public good, and a source of sexual conflict between males and females (Cotter *et al.* 2010).

Although these externally secreted antimicrobial examples all come from invertebrates (and arthropods specifically), this phenomenon is generalisable to vertebrates in the form of transgenerational immune molecule transfer (Roth *et al.* 2018). In mammals, milk contains a high concentration of antibodies and other nutrients and immune molecules, which are widely accepted to help prime the naïve animal's immune system to help resist infection (Roth *et al.* 2018). Importantly, it has been suggested that allonursing (i.e., providing milk to another individual's young) could serve a similar function in mammals including deer, house mice, and cooperatively breeding mongooses, allowing non-maternal adults in the social group to help bolster the young animal's immune system (Roulin & Heeb 1999). Because milk and antibody production are both costly, there are clear conflicts over who might invest in providing this immune help to young sickly offspring (and to which other individuals' offspring). Nevertheless, as yet it is unknown whether disease-related social contracts in nursing exist, whether they might be moderated by genetic interrelationships and immune status, and whether they will serve a social function or an anti-disease function – and if the latter, through which mechanism (Figure 1).

Physiology

Cooperative physiological regulation could influence disease resistance. Specifically, hibernating bats synchronise their arousals from torpor (Park *et al.* 2000) which may serve an immunological purpose, helping them to combat psychrophilic (cold-loving) parasites such as white nose fungus (Field *et al.* 2018; Fritze *et al.* 2019). This could lead to "selfish herd" dynamics, where all individuals benefit from cooperative thermoregulation because arousing simultaneously is easier than individually. Such dynamics could be generalisable to other species that undergo social thermoregulation like emperor penguins (Gilbert *et al.* 2006) and ladybirds (Szejner-Sigal & Williams 2022). Modelling the energetic costs (and immune benefits) of arousal, and the cost/benefit of cooperation with surrounding conspecifics, will likely help to address whether there is notable conflict around this behaviour. For example, what motivates the first individuals to arouse given that it would be easier if they waited for

others to do so first? Is the order of arousal correlated with disease status or immune resistance, and if so in what direction?

Reproduction as a complex form of immune cheating

Reproduction could be interpreted as immune cheating, for two reasons: first, because reproduction often comes with a substantial resource cost, reproductive individuals often have weaker immune resistance (Sheldon & Verhulst 1996; Knowles *et al.* 2009), which will likely result in greater pathogen transmission to the surrounding population. Simultaneously, reproduction produces young individuals that are often immunologically naïve and susceptible (Ashby & Bruns 2018), thereby presenting a resource for pathogens. Both these processes could therefore represent a cost to the population in the form of greater parasite abundance; reproductive effects like these have a substantial effect on epidemiology, often even being responsible for seasonal fluctuations in parasite prevalence (Altizer *et al.* 2006; Martin *et al.* 2008). Consequently, conspecifics could be motivated to punish those that reproduce, mediated by their relatedness to the reproducing individual (and therefore their offspring). For example, it has been suggested that male European badgers might kill unrelated offspring to prevent them from transmitting *Eimeria* parasites (Albery 2015). It has also been suggested that a mother might resort to infanticide when offspring represent an infection risk to her (Hart & Hart 2021).

Group-level costs of reproduction could select for a number of measures to avoid punishment, many of which might have led to the evolution of certain group structures. First, reproductive individuals with offspring might form temporary subgroups composed of closely related individuals. These groups could encourage greater cooperation in disease resistance, particularly in high-risk periods like youth and reproduction. For example, juvenile Gouldian finches (*Erythrura gouldiae*) form kin subgroups when integrating into a novel flock (Kohn *et al.* 2021). Additionally, reproductive individuals might disperse with their offspring to avoid persecution from conspecifics: for example, red deer (*Cervus elaphus*) with young often spend the first few months of their offspring's life in isolation (Clutton-Brock *et al.* 1982), resulting in reduced social connectedness (Albery *et al.* 2021), and badgers (*Meles meles*) often dig satellite setts away from their main social group when they are ready to give birth (Macdonald & Newman 2022). Notably this is not the only reason for maternal dispersal: (Hart & Hart 2021) suggest that isolation around parturition could protect the young from infection from other conspecifics. As such, reproductive isolation could be a mutually beneficial way to minimise the cost of a naïve immune system. Alternatively, conspecifics could reduce the cost of others' reproduction by improving the young individuals' immune systems through antibody transfer (Roth *et al.* 2018) – i.e., through allosuckling (Roulin & Heeb 1999).

General themes in disease-related social contracts

Hygiene and immunity therefore adhere to many relevant themes similar to other social contracts. Anti-disease traits can involve honest signals, enforcement, trials, reciprocity, and kin modulation, all

of which are staples of socially evolved traits. However, there are also specific elements to disease-related social contracts, including a variety of difficulties evolving and enforcing them. These difficulties include: nonspecific signalling associated with restrictions in immune resistance; difficulties safely punishing immune cheaters; contrasting effects of genetic relatedness; and complex feedbacks between individual, group, and parasite.

Nonspecific signalling of immune cooperation status

Many mechanisms of effective resistance might not lend themselves to accurate enforcement. Most importantly, generalised strong immune effector responses are likely to make an individual very sick, through a combination of immunopathology and direct resource costs (Graham *et al.* 2005; Viney *et al.* 2005; Cressler *et al.* 2014). Therefore, despite actually representing extreme commitment to transmission-reducing immunity, these individuals could be identified as "sick" and punished by conspecifics. In contrast, tolerant individuals who are continuing to transmit will be rewarded for seeming healthy, while in fact presenting a substantial infection risk. If immune enforcement and punishment of sick individuals is common and has strong fitness consequences (as it seems to; e.g. (McFarland *et al.* 2021)), the ability to selfishly evade such punishment could contribute to the widespread nature of tolerance responses in ecology. The social evolution of resistance-sickness-infection interactions is bound to continue being a complex but extremely fruitful area of research in coming years (Hart & Hart 2021).

The infection risk of effective immune enforcement

If it involves space sharing or direct contact, the act of enforcing anti-disease measures could risk exposing oneself to infection. Enforcement is critical to the stability of social contracts (Ågren *et al.* 2019), and this additional cost is likely a substantial obstacle to their evolution. This is a difficulty with positive enforcement – e.g., in mandrills, where grooming exposes individuals to endoparasites (Poirotte *et al.* 2017) – but may be even more so with negative or aggressive punishments. In many cases, immune cheats will be heavily infected and an important infection risk – particularly if their infection is easily detected. Avoidance (and ostracism, to a lesser extent) may be relatively more common with disease-related contracts because they reduce exposure risk. Hygienic behaviour should be risk-weighted by an individual's immunity: for example, grey mouse lemurs (*Microcebus murinus*) exhibit repeatable hygienic personalities when choosing between contaminated food items, which vary between sexes in potentially immune-mediated ways (Poirotte & Kappeler 2019). *Lasius neglectus* ants with a low-level infection alter their hygienic care to include more antimicrobial disinfection and thereby avoid hyperinfection (Konrad *et al.* 2018). Future studies could ask whether immune enforcement is generally carried out by specific (healthy) groups of individuals or certain personality types. For example, do eusocial insect societies support specific highly resistant castes of sanitary workers?

Tension between genetic relatedness, inbreeding, and cooperation

Genetic relatedness is a common underpinning factor encouraging cooperation in general (Griffin *et al.* 2004), and the same is likely to be true of cooperation around hygiene and immunity. For example, closer genetic relatedness favours faster social immune responses in *Cardiocondyla obscurior* ants (Ugelvig *et al.* 2010). However, genetic homogeneity is also commonly thought to exacerbate disease risk because genetic variation is important for effective resistance. As such, animals featuring close-knit kin groups may be more able to cooperate in hygiene and immunity, but with a reduced genetic pool that predisposes them to infection. There is relatively good broad evidence for diversity-infection relationships (King & Lively 2012; Ekroth *et al.* 2019; Gibson & Nguyen 2021) and mixed limited evidence for kin modulation of hygiene and immunity (Spottiswoode 2008; Lopes *et al.* 2018; Stockmaier *et al.* 2020), so the balance of these two forces is uncertain. Although not focussed on disease-related cooperation as I am here, a recent meta-analysis found that low diversity likely encourages greater cooperation and therefore benefits fitness, which counteracts its detrimental effects acting via infection (Bensch *et al.* 2021). Given that diversity effects on infection seem less common than might be expected in observational systems (Gibson & Nguyen 2021), cooperation in hygiene and immunity could represent an important factor introducing variation into diversity-infection relationships.

Complexity, dynamic feedbacks, and multidimensionality

The problem of disease-related social evolution may have endured partly because both disease and social systems are highly complex and dynamic. Fundamentally, sociality alters many elements of exposure and susceptibility, with complex feedbacks that can be difficult to account for (Ezenwa *et al.* 2016a; Townsend *et al.* 2020; Hawley *et al.* 2021; Poulin & Filion 2021) and with both costs and benefits for disease (Loehle 1995; Kappeler *et al.* 2015; Ezenwa *et al.* 2016b; Hart & Hart 2021), which can create difficulties identifying how sociality alters disease in general. Moreover, individual- and group-level benefits of anti-disease traits are heavily intertwined. Because an individual's disease burden is so dependent on the burden of those around it, there is no such thing as a purely social benefit for disease: any action that reduces the population's burden will have some feedback benefit for the individual. For example, grooming is deemed a purely social benefit because it purely reduces the parasite burden of the recipient (Cotter & Kilner 2010), but for the grooming individual, removing naïve parasites from a close associate might reduce the possibility that said conspecific will transmit parasites back to the individual in the future. This is particularly true if grooming is preferentially directed towards individuals that are in close proximity, as in bats (Stockmaier *et al.* 2018), horses (Shimada & Suzuki 2020), and macaques (Sade *et al.* 1988). These processes are similar to by-product mutualisms that drive the evolution of some apparently-cooperative traits: for example, both house sparrows and meerkats benefit individually by being vigilant while feeding, but in both cases this behaviour also benefits the group (Elgar 1986; Clutton-Brock *et al.* 1999).

Conversely, each individual that avoids or resists disease for selfish reasons may ultimately help the group by extension, because they are less likely to transmit the infection to their compatriots (Frank 1998). Most pressingly, as outlined above, only certain elements of immunity are likely to reduce transmission, so an individual's cooperativeness will depend on its proportional investment in A) immunity that maintains its own health versus B) immunity that reduces transmission (the A:B Ratio depicted in Figure 1). This investment in individual health is directly reflected in the popular concept of "herd immunity", where population-level resistance is common enough that individual-level resistance becomes less necessary – but each individual can (generally) still stand to benefit from expressing their own resistance (Cotter & Kilner 2010). Even in scenarios where these feedbacks are unimportant, there may be nonlinearities and highly stochastic dynamics that necessitate a large dataset over a long timeline to understand. For example, infected bee colonies are worse at defending themselves from infection (Baracchi *et al.* 2012a). Due to these and other complexities, the social evolution of immunity may have required a specific framework to understand and untangle.

Future directions and open questions

Examining a wider diversity of systems and approaches, with an explicit view to testing conflict and cooperation in disease, will help to further test the social evolution of immunity and infection. Natural history will help to identify potential model systems like vampire bats (Carter & Leffer 2015). *Desmodus rotundus* is an established model for cooperative feeding behaviour (Wilkinson 1984; Carter & Wilkinson 2013) and sickness (Stockmaier *et al.* 2018); future analyses could look further at whether kin, reciprocity, illness, and care interact, testing the conditions under which social contracts evolve to prevent disease. Similarly, European badgers (*Meles meles*) have interesting spatial and social dimensions of pathogen transmission (Woodroffe *et al.* 2009, 2016; Albery *et al.* 2020) and are a good model system for kin effects in disease (Benton *et al.* 2016). Moving forward, a focus on animals other than eusocial insects – and particularly on those with less sophisticated social systems – will be integral to filling in the spectrum of cooperation.

Beyond empirical systems, mathematical models of sociality, movement, immunity, and disease could help to develop and test verbal arguments. Modern dynamic eco-evolutionary models of social evolution could attempt to replicate early social evolutions like (Frank 1998), incorporating more sophisticated pathogen transmission processes and to account for spatially explicit confounders. This marriage is already occurring (e.g. (Best *et al.* 2011; Débarre *et al.* 2012; Horns & Hood 2012)), but with a relatively restricted suite of immune processes, and no research has yet examined how socio-spatial behaviour itself could evolve to limit disease.

Specific experimental approaches could help to address social evolution of immunity – for example, variable kin structure treatments and observations have been used in mice (Lopes *et al.* 2018) and in vampire bats (Stockmaier *et al.* 2020). Comparative analyses across a range of systems could be employed to ask e.g. whether different relatedness structures affect the expression of resistance

responses or cooperation. This approach has shown that cooperatively breeding birds have stronger immune responses (Spottiswoode 2008), and similar approaches could be applied across cooperative breeders in other clades like mammals (e.g. (Lukas & Clutton-Brock 2017)), or across other groups that have been used for comparative analyses of social structure like ungulates (e.g. (Ezenwa 2004)). Similarly, researchers could look across a range of wasp species or populations with different kinship structures (e.g. (Foster & Ratnieks 2001; Jandt *et al.* 2014)) to investigate which conditions favour the production of costly prosocial antimicrobials (e.g. (Baracchi *et al.* 2012b)). More broadly understanding how individual-level responses lead to group-level consequences in disease will help to identify where conflicts manifest between individuals.

Finally, this area will become considerably more complex when parasite motivations are factored in. Many parasites actively manipulate their hosts to facilitate transmission (Poulin 2010; Poulin & Maure 2015), with complex impacts on host behaviour and emergent socio-spatial structure. For example, (Beros *et al.* 2021) found that the tapeworm *Anomotaenia brevis* elongates the lifespan of individual infected *Temnothorax nylanderi* ants, but endangers the life of their colony as a whole. Similarly, rather than representing an actively selfish act, infected guppies' tendency to become more social (Reynolds *et al.* 2018) might be due to a parasite phenotype acting to facilitate transmission – or it may represent a sort of "mutualistic" combination of selfish host trait and manipulative parasite trait. Depending on the transmission mode, the parasite's investment in shedding *versus* within-host replication, and their relationship to host pathology, it may become very difficult for either infected or uninfected hosts to judge the cost and benefits of a given cooperative immune trait, particularly where parasites create asymptomatic infections. Furthermore, for interested researchers, reliably identifying these terms is likely to become very difficult, particularly where parasite fitness is unquantifiable.

Concluding remarks

The social evolution of behaviour is a longstanding topic of interest in ecology and evolution. In this review, I have given an overview of socially evolved behaviours and immune traits that specifically serve to combat the spread of parasites, identifying the complexities evolving, enforcing, and investigating such traits. Fundamentally, both disease and cheating are viewed as a cost for the major transitions to sociality; examining their intersection may provide much-needed information concerning the nexus of processes that drive and inhibit the evolution of social complexity. Moreover, further interrogating this intersection empirically might likewise help with resolving public goods dilemmas in human health.

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