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Review

Modification of host social networks by manipulative parasites

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Abstract
Social network models provide a powerful tool to estimate infection risk for individual hosts and track parasite transmission through host populations. Here, bringing together concepts from social network theory, animal personality, and parasite manipulation of host behaviour, I argue that not only are social networks shaping parasite transmission, but parasites in turn shape social networks through their effects on the behaviour of infected individuals. Firstly, I review five general categories of behaviour (mating behaviour, aggressiveness, activity levels, spatial distribution, and group formation) that are closely tied to social networks, and provide evidence that parasites can affect all of them. Secondly, I describe scenarios in which behaviour-altering parasites can modify either the role or position of individual hosts within their social network, or various structural properties (e.g., connectance, modularity) of the entire network. Experimental approaches allowing comparisons of social networks pre- versus post-infection are a promising avenue to explore the feedback loop between social networks and parasite infections.

Keywords
animal personality, connectance, host manipulation, modularity, spatial segregation.

1. Introduction

In recent years, social network models have provided a strong predictive framework to understand both infection risk at an individual host level and parasite transmission through host populations (Drewe, 2010; Godfrey et al., 2010; Fenner et al., 2011; MacIntosh et al., 2012; Godfrey, 2013; Grear et al., 2013; Springer et al., 2017; White et al., 2017). Social networks characterise

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the set of pairwise interactions among individuals in a group, accounting for the frequency, duration, intensity and directionality of interactions (Wey et al., 2008; Sih et al., 2009). They can be visualised as simple diagrams in which individual hosts are represented by nodes connected together by edges that capture the strength and/or direction of all pairwise interactions between individuals. Simple measures of the properties of each node can be obtained to quantify the role or position of each individual within the network. Similarly, various measures of whole-network structure can be derived and used for comparisons of different host populations, or the same population at different points in time. Several types of behavioural interactions can be measured among pairs of individuals and formalised as social networks, including those involving physical contact (grooming, fights, etc.) and spatial proximity (territory overlap, refuge sharing, etc.). These sorts of interactions determine opportunities for transmission by many parasites and pathogens with direct life cycles that are transmitted during physical contact or close proximity between two conspecific hosts. These 'contagious' parasites include many bacteria, viruses, and ectoparasitic metazoans (e.g., lice, fleas, mites, monogeneans). Many empirical studies (see review in Godfrey, 2013; White et al., 2017) have demonstrated that host social networks can reliably predict which individuals (the 'superspreaders') disproportionately transmit parasites and spread disease to others, and which are most susceptible to acquire parasites.

Here, I turn this statement on its head: instead of social networks shaping parasite transmission, I argue that parasites shape social networks through their effects on host behaviour. Although the influence of parasitism on social behaviour has long been recognised (Moore, 2002; Klein, 2003), here I examine it in light of social networks. Modification of individual host behaviour is likely to affect pairwise interactions, with inevitable repercussions on the structure of the whole social network. Changes to pairwise interactions or network structure may have little or no consequences for the transmission success of the parasite inducing them. For example, if a parasite with a complex life cycle somehow alters the social network of host species A, but must be transmitted to host species B to complete its development, its effect on host A's social network may be inconsequential for its own success. However, animal populations are typically co-infected by multiple parasite species (Pedersen & Fenton, 2007). The influence of host social structure on the transmission of one parasite can only be properly understood

in light of the effects of other parasites on host social structure. Thus, the alteration of host social network by a parasite can (i) re-direct the transmission routes of one or more other contagious parasite species also infecting the host population, and (ii) modify the dominance hierarchy or other aspects of the host's social structure. Both of these outcomes have huge implications for individual fitness.

The basis for arguing that parasites can shape host social networks through their effects on host behaviour comes from two other conceptual frameworks that have guided research efforts in animal behaviour in recent years. First, the hypothesis that many parasites manipulate the behaviour of their host as an adaptive strategy to enhance their transmission success has now been proven beyond doubt (Moore, 2002; Thomas et al., 2005; Poulin, 2010). The phenomenon has been documented empirically in hundreds of host and parasite taxa spanning multiple phyla (Moore, 2002; Poulin & Maure, 2015). Host manipulation by parasites can result in the appearance of complex and novel behaviours in the host, such as jumping in water in hairworm-infected crickets (Thomas et al., 2002), fruit mimicry in nematode-infected ants (Yanoviak et al., 2008) or attraction to cat odours in *Toxoplasma gondii*-infected rats (Berdoy et al., 2000). More often, however, it involves small changes in how individuals respond to stimuli following infection, resulting for example in infected hosts ignoring approaching predators, or shifting to slightly different microhabitats. Parasite-induced changes in host behaviour are not always adaptive for the parasite. In many cases, they may be adaptive responses by the host to eliminate the parasite or compensate for its deleterious effects, or they may be of no benefit for either host or parasite (Moore, 2002; Poulin, 2010). Whether or not these behavioural changes are adaptive does not necessarily matter in the present context, as long as they affect host social networks.

Second, the idea that animals have personalities has emerged as the dominant framework to understand intraspecific behavioural variation and its ecological and evolutionary consequences (Réale et al., 2007). Animal personality refers to the behavioural differences among individuals in a population that are consistent over time and across different contexts (Réale et al., 2010; Stamps & Groothuis, 2010). In principle, animal personalities can be measured using any suite of behaviours. In practice, however, researchers have focused on five general behavioural axes (Réale et al., 2007), two of which are highly relevant to a population's social structure: (i) aggressiveness, i.e.,

an individual's agonistic reaction towards conspecifics; and (ii) sociability, or an individual's reaction to conspecifics other than aggression. Whether through adaptive manipulation, depletion of the host's energy stores, or mere pathology, parasites have been proposed as possible influential factors in the expression of personality traits (Barber & Dingemanse, 2010; Poulin, 2013). This has now been substantiated empirically in several host-parasite associations (e.g., Hammond-Tooke et al., 2012; Kekalainen et al., 2014; Pan et al., 2016).

Social networks, animal personality, and parasite manipulation of host behaviour are brought together in this short essay to argue that parasites can modify social networks via their effects on host behaviour. I begin by reviewing evidence that parasites can alter several behaviours relevant to host social structure, i.e., behaviours that shape the nature, frequency or strength of interactions among individuals. I then explore in turn the consequences of parasite-induced changes in host behaviour for the position and role of individual hosts within their social network, and for the architecture of the whole network. These are presented as various scenarios involving parasites with particular behavioural effects. Hopefully, they will serve to illustrate the potentially large but generally ignored impact of parasites on host social networks.

2. Parasite-induced changes to relevant behaviours

The kinds of behaviour known to be altered by parasitic infection and likely to impact social interaction networks fall into five main categories: mating behaviour, aggressiveness, activity levels, spatial distribution and group formation. These are discussed in turn below, and summarised with examples in Table 1. The list of behaviours covered here is not meant to be exhaustive, but instead illustrative of the potential effects of parasites on host social structure.

First, parasites have long been known to affect mate choice, reproductive success and sexual selection in animals (Hamilton & Zuk, 1982; Schall, 1983; Moore, 2002; Heins et al., 2004). One common outcome is that parasitised males mate less frequently and with fewer females, either because of their reduced attractiveness or their lower ability to compete for access to mates (e.g., Schall & Dearing, 1987; Forbes, 1991; Zohar & Holmes, 1998). Another possibility is that infection can lead to assortative pairing,

Table 1.
Selected examples of 5 general types of behavioural changes induced by parasitic infection.

Behaviour	Host	Parasite	Consequence of infection	References
Mating behaviour	Lizard, *Sceloporus occidentalis*	Malaria, *Plasmodium mexicanum*	Lower mating frequency	Schall & Dearing (1987)
	Damselflies, *Enallagma ebrium*	Mites, *Arrenurus* spp.	Lower mating frequency	Forbes (1991)
	Amphipod, *Gammarus insensibilis*	Trematode, *Microphallus papillorobustus*	Assortative pairing based on infection	Thomas et al. (1996)
Aggressiveness	Rainbow trout, *Oncorhynchus mykiss*	Trematode, *Diplostomum spathaceum*	Increased aggressiveness	Mikheev et al. (2010)
	Salamander, *Plethodon angusticlavius*	Mite, *Hannemania eltoni*	Reduced aggressiveness	Maksimowich & Mathis (2000)
	Grouse, *Lagopus lagopus*	Nematode, *Trichostrongylus tenuis*	Reduced aggressiveness	Fox & Hudson (2001)
	Mouse, *Mus musculus*	Nematode, *Trichinella spiralis*	Loss of dominance	Rau (1984)
Activity levels	Lizard, *Sceloporus occidentalis*	Malaria, *Plasmodium mexicanum*	Reduced stamina	Schall (1982)
	Eel, *Anguilla anguilla*	Nematode, *Anguillicola crassus*	Reduced swimming speed	Sprengel & Lüchtenberg (1991)
	Lemming, *Dicrostonyx richardsoni*	Protozoan, *Sarcocystis rauschorum*	Increased exploratory activity	Quinn et al. (1987)
	Rat, *Rattus norvegicus*	Protozoan, *Toxoplasma gondii*	Increased running activity	Webster (1994)

Table 1.
(Continued.)

Behaviour	Host	Parasite	Consequence of infection	References
Spatial distribution	Snail, *Ilyanassa obsoleta*	Trematode, *Gynaecotyla adunca*	Infected individuals move to the upper shore	Curtis (1987)
	Roach, *Rutilus rutilus*	Cestode, *Ligula intestinalis*	Infected individuals move to the littoral zone	Loot et al. (2001)
	Amphipod, *Gammarus insensibilis*	Trematode, *Microphallus papillorobustus*	Infected individuals move to the water surface	Helluy (1983)
Group formation	Bumblebee, *Bombus lucorum*	Parasitoid fly, Conopidae	Infected individuals leave their colony	Schmid-Hempel & Müller (1991)
	Stickleback, *Gasterosteus aculeatus*	Cestode, *Schistocephalus solidus*	Infected individuals do not join shoals	Barber et al. (1995)
	Brine shrimps, *Artemia* spp.	Cestodes (3 species)	Infected individuals are more likely to swarm	Rode et al. (2013)

with infected males mating mostly with infected females, and uninfected males pairing preferentially with uninfected females (Thomas et al., 1996, 1999). For example, this can result from the combined effect of reduced attractiveness of infected males and reduced choosiness of infected females, or from the spatial segregation of infected and uninfected individuals (see below). Therefore, parasites can structure sexual interactions by determining how often an individual mates, and who mates with whom.

Second, parasitism can modulate host aggression levels. For example, an association between rabies and overt signs of aggression in dogs and other canids has long been recognised (see Wang et al., 2010). Although other parasites also cause increased aggressiveness (e.g., Mikheev et al., 2010), most parasite infections tend to make the host less aggressive and more likely to lose intraspecific disputes for territories or other resources (e.g., Maksimowich & Mathis, 2000; Fox & Hudson, 2001). Parasites may even cause a previously dominant host to lose its position in a social hierarchy, or prevent hosts to rise in the hierarchy (Rau, 1984; Gourbal et al., 2002). The upshot of these various impacts is that the frequency and intensity of aggressive interactions initiated or received by an individual, as well as their outcome, may be altered by parasite infection.

Third, perhaps the least surprising effect of parasites on their host is a reduction in activity, measured as either the frequency, duration or speed of movements, or as the proportion of time spent performing a particular activity instead of remaining inactive (e.g., Lester, 1971; Schall, 1982; Sprengel & Lüchtenberg, 1991). Infected hosts are sometimes characterised as lethargic or listless (Moore, 2002). This results either from adaptive manipulation of host physiology by the parasite to enhance its own transmission, or from simple pathology. In contrast, in other cases parasites caused an increase in host activity levels post-infection (Quinn et al., 1987; Webster, 1994). For example, lemmings, *Dicrostonyx richardsoni*, show a higher frequency of exploratory activity when infected by the coccidian *Sarcocystis rauschorum* compared to uninfected conspecifics (Quinn et al., 1987). The consequences for host social networks is that depending on whether parasite infections depress or boost activity levels, infected hosts may show lower or higher probabilities of encountering and interacting with conspecifics.

Fourth, some manipulative parasites can induce their hosts to occupy different microhabitats than those used by healthy individuals. There are multiple examples of this phenomenon, ranging from trematode-infected snails

migrating to the upper intertidal zone (Curtis, 1987) to cestode-infected fish moving into the shallow waters of the littoral zone (Loot et al., 2001). These microhabitat shifts bring the parasite's current host closer to where the parasite's next host is actively feeding, probably improving the chances of the parasite being trophically transmitted. Sometimes, microhabitat shifts occur as a consequence of parasite-induced alterations to how the host responds to stimuli. For instance, amphipods infected with certain trematodes or acanthocephalans become attracted to light and gather at the water surface, in contrast to healthy individuals which are photophobic (Bethel & Holmes, 1973; Helluy, 1983). The net effect of these microhabitat shifts is a split of the host population into two, spatially segregated groups: infected individuals in one place and healthy ones in a different place. The set of possible interaction partners available to any individual is thus limited, and the structure of the whole social network is changed by the actions of these parasites.

Fifth, social interactions in general, and group formation in particular, are common targets for parasite manipulation of host behaviour. Many parasites can alter the average spacing between individuals (e.g., Shaner et al., 2017), cause the splintering of host groups by inducing infected individuals to leave the group, or prevent infected individuals from keeping up with a moving group. In eusocial hymenopteran insects, colony members can either abandon the colony or spend a large proportion of their time away from the colony following infection by trematodes (Carney, 1969), nematodes (Yanoviak et al., 2008) or parasitoids (Schmid-Hempel & Müller, 1991). Badgers infected with bovine tuberculosis also tend to leave their group (Cheeseman & Mallinson, 1981). Fish infected with helminth parasites often struggle to remain within their school and spend much time swimming on their own (Krause & Godin, 1994; Barber et al., 1995). In all these cases, infected individuals become isolated, temporarily or completely, from other group members. In other cases, however, parasites may have the opposite effect, and induce their hosts to form groups (e.g., Rode et al., 2013). Therefore, the precise impact of parasite infections on the structure of host social networks depends on the particular situation, but is probably always far from negligible.

In addition to behavioural changes induced directly by the parasite (Table 1), hosts may respond behaviourally to the presence of parasites, with consequences for either their position in the social network or the structure

of the network as a whole. For example, healthy individuals may avoid contact with, or even avoid coming close to, visibly infected individuals, with consequences for all individuals in terms of connectivity or network architecture. For example, Croft et al. (2011) found that guppies, *Poecilia reticulata*, avoided shoaling with individuals harbouring contagiously-transmitted, ectoparasitic monogeneans, leading to changes in their social network. Alternatively, animals may form larger groups or reduce the distance that separates them in an effort to protect themselves against mobile ectoparasites. For example, Poulin & FitzGerald (1989) demonstrated that juvenile sticklebacks, *Gasterosteus* spp., formed larger shoals when free-swimming crustacean ectoparasites were introduced into their experimental pools, presumably to reduce their individual infection risk through a dilution effect. Similarly, fathead minnows, *Pimephales promelas*, also form tighter shoals when exposed to the free-swimming infective stages of trematodes (Stumbo et al., 2012). Wild horses and monkeys also form larger and tighter groups in seasons with greater attacks by blood-sucking dipterans, also apparently to gain protection through a dilution effect (Freeland, 1977; Duncan & Vigne, 1979). Here and in the following sections, I focus exclusively on changes in host behaviour directly caused by the parasite, via adaptive manipulation or non-adaptive patho-physiological mechanisms, and not on host responses to mitigate infection risk.

3. Parasitism and the social roles of individual hosts

In a social network, the role and position of each individual, i.e., each node in the network, can be characterised in many ways. Most basic metrics that can be derived quantify some aspect of the connectivity of an individual to others in the network, or their centrality in chains along which key resources (e.g., food, information) flow through the network (Newman et al., 2006; Scott, 2013). Some of the most basic node-level metrics include its degree (number of direct connections, or edges, to other nodes) and strength (sum of edge weights to and from that node, with the weight of an edge indicating the interaction's frequency or intensity), but also the distribution of edge weights (from even to highly different weights) and the directionality of edges (whether the node is a donor or recipient of interactions).

There are many possible ways in which parasites can modify these properties of individual nodes. In one scenario, a social network in which males

and females have roughly equal probabilities of mating with any member of the other sex could be altered by a parasite capable of inducing assortative pairing (Figure 1A). Each individual would see its number of potential

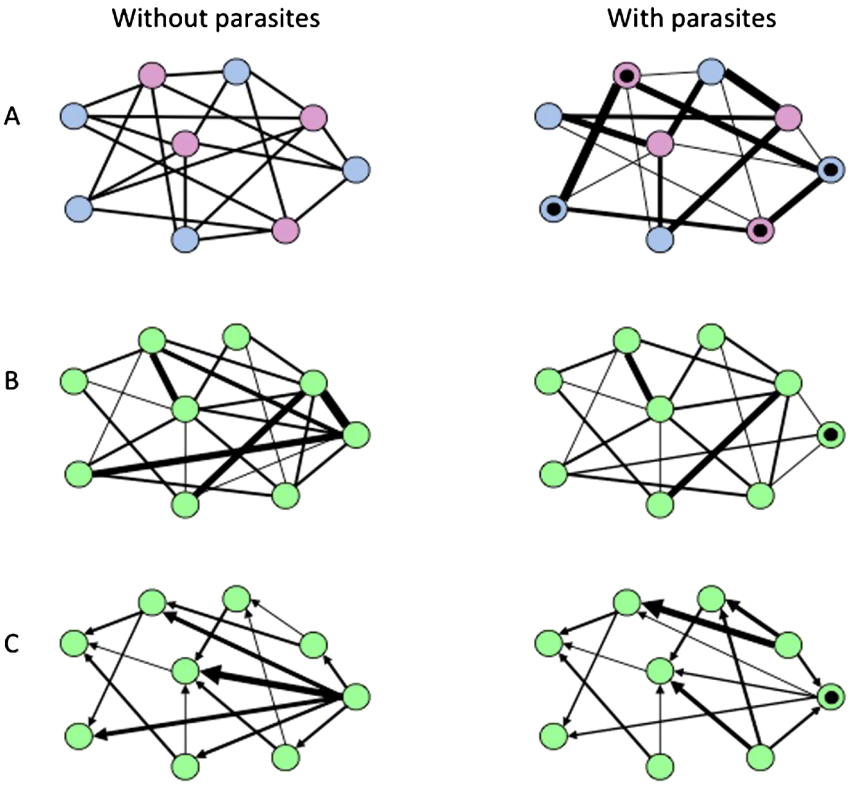

Figure 1. Hypothetical social networks illustrating the potential impact of parasites on the role of infected individuals. Individuals are represented by nodes (circles) and their interactions are represented by edges (lines) connecting them. Edge weight (line thickness) is proportional to interaction strength, which captures the probability, frequency, duration and/or intensity of interactions. Parasite infection is indicated by a black dot inside a node. (A) Without parasites, males (blue nodes) have an equal probability of mating with any female (pink nodes), and vice versa; with some individuals parasitized, mating probabilities become biased such that infected males are more likely to mate with infected females, and uninfected males more likely to mate with uninfected females. (B) Parasite infection makes an individual less likely to interact with others, reducing both the node's degree (number of edges to other nodes) and strength (sum of edge weights to and from that node). (C) Once parasitized, a previously dominant individual has reduced aggressiveness and loses its position in the hierarchy; instead of directing more aggression towards others than it received (arrows indicate net outcome), it now receives as much as it gives.

partners reduced to only a subset of those previously available, and/or its probability of mating with any particular member of the other sex strongly modified.

Many parasites reduce the activity levels of their host, making them less likely to encounter or interact in any way with other group members. Other parasites can alter the average spacing between individuals, reduce the time individuals spend as part of the group, or even induce them to abandon group living altogether (see previous section). Therefore, a likely scenario is that parasite infection would cause reductions in the degree and strength of a node, i.e., the number of interacting partners and the frequency or intensity of interactions (Figure 1B). This could have cascading effects through part of the networks, as the properties of uninfected nodes that interact with the now parasitized individual would also be altered. Reduced interactiveness following parasitic infection due to lower activity levels or lethargy may be the most widespread side-effect of parasites, and thus probably plays an important but undetected role in structuring interactions within social networks.

Finally, consider the impact on social networks of a parasite capable of severely depressing aggression levels in its host. If the dominant individual in a group becomes infected, it may lose its top position in the hierarchy: instead of directing more aggression towards others and winning most disputes, after infection it may not fare so well during aggressive encounters and lose its dominant status (Figure 1C). Inevitably, this would have repercussions for the rest of the group, with previously subordinate individuals now allowed to rise through the ranks. The net strength (difference between outgoing and incoming weighted edges representing directed aggression) would be changed not only for the infected individual's node, but also for the nodes of its immediate interacting partners.

These are just some of the possible scenarios. In all cases, the consequences of infection by a behaviour-modifying parasite will include changed social or sexual interactions. In additions, the modified interactions of infected individuals may expose them to higher or lower risks of acquiring contagious parasites, whose transmission relies on physical contact or proximity between individual hosts. Thus, one parasite's effect on social networks could modulate the transmission of another parasite.

4. Parasitism and whole-network structure

It is also possible to characterise the architecture of an entire social network by measuring some of its properties (Newman et al., 2006; Scott, 2013). Beyond network size (number of individuals, or nodes, in the network), other standard metrics include average path length (minimum number of edges connecting any two nodes in the network, averaged across all pairs of nodes in the network), connectance (the proportion of realised edges out of all possible ones) and modularity (number of modules within the network). Modules represent partitioning within a network; they consist of distinct subsets of nodes having more interactions, i.e., edges, among themselves than with nodes from other modules.

There are at least two ways (see below) in which parasites can modify whole-network properties through their effects on individual behaviour. The extent and magnitude of this impact will obviously depend on the prevalence (proportion of host individuals that are infected) and/or the intensity of infection (the mean number of parasites per infected host). Here, I assume that these are high enough that a non-trivial proportion of the host population is infected with a parasite load sufficient to experience behavioural changes.

The first way in which whole-network structure could be modified involves parasites that induce their hosts to occupy different microhabitats than those used by healthy individuals. This can create spatially segregated modules within the network (Figure 2A). Similarly, parasites that modify the activity periods of their hosts could produce temporally segregated modules. In such cases, infected individuals are more likely to interact with other infected individuals, because they occupy the same microhabitat or are active at the same times, than with uninfected individuals, and vice versa. Interactions between members of the two subgroups can persist but with lower frequency or duration. Although the degree and strength of nodes may be roughly similar to what they were pre-infection, it is the realised edges that are now determined by infection status since some interactions become impossible because the two subsets of individuals no longer overlap in time or space.

Another possibility, probably much more common, involves infection by a prevalent parasite that causes its hosts to become lethargic, to the extent that they are less interactive within their social group. The outcome would be a network with lower connectance, i.e., a lower proportion of realised

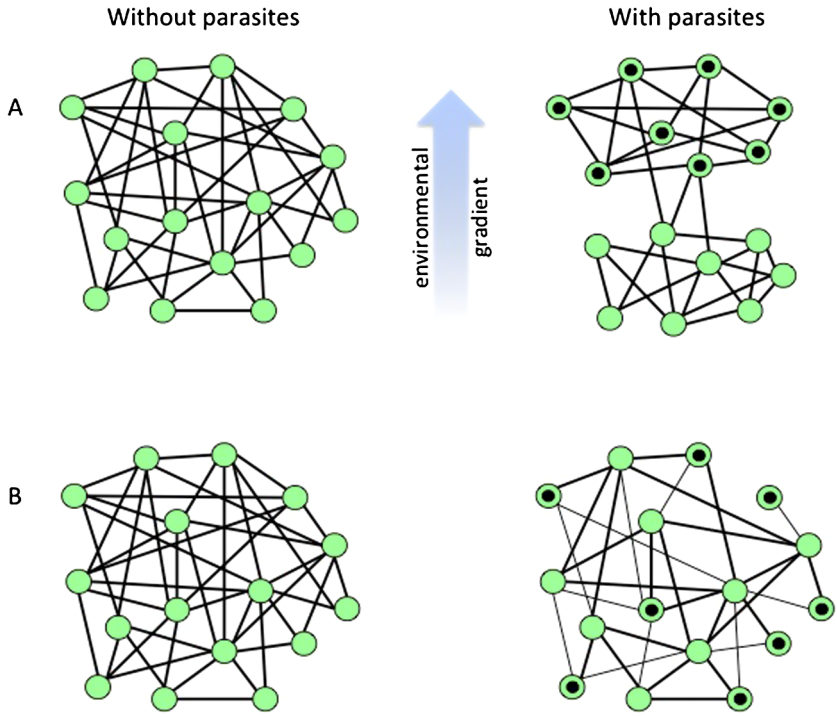

Figure 2. Hypothetical social networks illustrating the potential impact of parasites on whole network structure. See Figure 1 for explanation of network components. (A) Following infection of half of its members, a once well-connected group becomes split into two modules along an environmental gradient, such as water depth; infected and uninfected individuals occupy different microhabitats, and interact mostly with other members of their module. (B) When infected by parasites, individuals become less likely to interact with others, and as a result the connectance of the entire network is reduced.

edges compared to the same network in the absence of infections (Figure 2B). The average degree and strength of individual nodes would be lower, and so would the overall number of interactions keeping them connected. As in the case of parasite-induced changes to the role of individual group members, alterations to whole-network structure, whether measured as modularity or connectance, can have serious implications for the flow of information or other resources through the network, as well as for the transmission dynamics of other, contagiously-transmitted parasites (e.g., Sah et al., 2017).

5. Looking ahead

Here, I have argued that parasites shape social networks through their effects on individual host behaviour. The effects of parasites can be manifested by changes in the position or role of infected hosts in the network, or they can add up and lead to alterations to the structure of the whole network. Although it is possible that in certain situations, natural selection has favoured the evolution of parasites with the ability to modify host social networks, it is much more likely that changes to social networks result as collateral effects from either adaptive manipulation of particular host behaviours, or from pathological side-effects of infection. As plausible as the scenarios described here may be, there is as yet very little direct evidence (Croft et al., 2011) that parasites affect host social networks. The potential influence of particular parasite species may be difficult to demonstrate in natural situations. Animal populations, especially those of vertebrate species, are simultaneously infected by multiple species of parasites and pathogens, with various types of life cycles and different effects on host behaviour (Pedersen & Fenton, 2007). Therefore, an experimental approach would be necessary to demonstrate the causal effect of parasites on social networks. A comparison of replicated networks pre- and post-infection would allow this to be tested rigorously; subsequent experimental disinfection would determine whether the network can revert to its original configuration once parasites are eliminated. I suspect that evidence from such studies will start accumulating in coming years, strengthening the links between social behaviour and epidemiology. This evidence will also allow us to close the feedback loop: are social networks not only driving parasite transmission, but also reciprocally being shaped by parasites?

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