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# Collective disruption: consequences of parasitism for collective animal behaviour

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Highly coordinated animal groups such as schooling fish, migrating birds and swarming insects are ubiquitous in nature, and these complex displays of collective behaviour emerge from local interactions between individuals. Although collective behaviours are known to confer benefits, they also come with the standard costs of group living, including increased risk of parasite infection. Notably, parasites can have profound effects on individual behaviour, which may in turn affect the inter-individual interactions that drive collective behaviour. Thus, given the commonness of parasites in animal populations and their widely appreciated effects on animal hosts, parasitism may be a key force shaping the ecology of collective behaviour. In this article, we use information transfer as a unifying theme to explore how the effects of parasites on individuals translate to the collective, focusing on four common collective behaviours: decision-making, collective movement, synchronization and construction. We also discuss the implications of parasite-altered collective behaviour for processes such as parasite transmission, wildlife conservation and animal culture.

## 1. Introduction

Highly coordinated animal groups like migrating birds, schooling fish, herding ungulates and marching locusts all represent collective animal behaviour. Surprisingly, these complex group-level patterns arise from relatively simple individual-level interactions [1]. For example, ant foraging trails are a by-product of individual ants depositing pheromones at regular intervals between a food source and nest. A single ant initiates the trail, subsequent ants reinforce it and positive feedback produces the shortest path to a newly discovered food source for the entire colony to follow [2]. In this way, collective behaviour enhances group performance.

Animals form groups for many reasons: to reduce predation, increase access to resources or mates and obtain help rearing young [3]. Once groups form, collective behaviour can reinforce these advantages; however, the benefits of group living also come with costs [3]. One cost that has generated considerable interest is parasitism. Theory suggests that parasite infection, defined broadly here as victimization by any infectious organism,

is an automatic cost of group living which acts to reduce group size or minimize the frequency of interactions between individuals that promote parasite transmission [3,4]. Consequently, parasitism should theoretically dampen collective behaviour via negative selection pressure on group size or interaction frequency. However, collective behaviour itself can act as a powerful defence against parasites. For example, social insects commonly respond to parasitism using coordinated actions that reduce the impact or spread of infection in colonies, a phenomenon called social immunity [5]. In fact, links between parasitism and collective behaviour have been largely studied from this collective defence perspective [6–8]. However, there is growing recognition that effects of parasitism on individual behaviour have implications for a much broader range of collective behaviours [9–11]. In this article, we examine how the effects of parasites on individual behaviour scale up to disrupt these other forms of collective behaviour.

The idea that parasitism has consequences for collective behaviour follows naturally from known individual-level impacts of parasite infection. Changes in individual behaviour resulting from host physiological responses to infection [12,13] or from parasite manipulation [14,15] are widely documented across taxa. For instance, lethargy and loss of appetite are hallmark sickness behaviours that manifest in response to a range of parasite infections [16,17], while manipulation of hosts by parasites can lead to both extreme and subtle changes in individual activity (e.g. [18,19]). Given such profound effects of parasites on individual behaviour, collective behaviour may be equally, if not more strongly affected [20–22]. Here, we propose that the transfer of information between individuals, a fundamental mechanism underlying almost all group-level behaviours, provides a valuable framework for understanding when and how parasites should affect collective behaviour. For example, imagine a group of ants forming a foraging trail, this time with parasite-infected individuals who deposit pheromone more slowly or do not accurately distinguish between the quality of different food sources, two changes that affect an ant's ability to share information with nestmates. A simple mathematical model predicts that as parasite prevalence in the group increases, these two parasite-induced changes in individual behaviour will have distinct implications for group-level resource exploitation (box 1). To extend this insight, we integrate principles of collective behaviour with literature on parasite-associated changes in individual behaviour to identify general pathways through which effects of parasites on individuals might translate to collectives and to generate hypotheses testable across a range of animal systems. Finally, we also consider the implications of parasite-altered collective behaviour for ecological and evolutionary processes such as parasite transmission, wildlife conservation and animal culture.

**Box 1.** Effects of parasitism on collective foraging decisions: a mathematical model.

Pheromone signalling between individual ants allows colonies to preferentially exploit the best food sources when confronted with multiple options. To examine how ant colonies collectively choose a superior resource, Sumpter & Beekman [2] used an experiment where two food sources were placed equidistant from an ant nest: feeder A (a high-quality resource), contained a 1 M sugar solution, and feeder B (a low-quality resource), contained a 0.1 M sugar solution. Ants were allowed to recruit colony members to each feeder over a 1-hour period, and a mathematical model was used to describe how variation in the rate of pheromone deposition between feeders shaped the collective decision to exploit the high-quality feeder:

$$dX_A/dt = (\alpha + \beta_A X_A)(N - X_A - X_B) - sX_A/(K + X_A)$$
$$dX_B/dt = (\alpha + \beta_B X_B)(N - X_A - X_B) - sX_B/(K + X_B)$$

In the model [2], $X_A$ and $X_B$ represent the number of ants foraging at each feeder, respectively. $N$ is the total number of ants and $(N - X_A - X_B)$ is the number of exploring ants that are looking for a food source. $\alpha$ is the rate at which an explorer randomly encounters a feeder. $\beta$ is the rate at which explorers are recruited to feeder A or feeder B. The recruitment rate varies with the quality of the food source because food quality determines the amount of pheromone deposited by ants that have already discovered a feeder. $sX_A / (K + X_A)$ is a saturating function determining the rate at which individual ants lose the pheromone trail.

To examine how parasitism could disrupt collective foraging, we modified this model to explore the effect of infection on individual ants' ability to translate resource cues into signals for nestmates (see electronic supplementary material for model code). To do this, we included terms that allow an ant's recruitment rate $\beta$ (i.e. the strength of the pheromone trail an ant deposits) to differ when infected. In our modified model, $p$ is the proportion of the colony that is infected and $\beta_i$ is the recruitment rate of infected individuals:

$$dX_A/dt = (\alpha + (1 - p)\beta_A X_A + (p)\beta_{iA} X_A)(N - X_A - X_B) - sX_A/(K + X_A)$$
$$dX_B/dt = (\alpha + (1 - p)\beta_B X_B + (p)\beta_{iB} X_B)(N - X_A - X_B) - sX_B/(K + X_B)$$

We varied the rate at which infected ants recruit colony members to each feeder to predict the colony-level impacts of parasitism in response to two plausible scenarios. First, we examined the effect of parasite infection on the energy budget of infected individuals, which we inferred would affect the rate at which infected ants recruit colony members to feeders as described in fire ants [23]. To model this 'reduced energy' scenario, we reduced the infected ant recruitment rate for both feeders by a factor of 10 (based on a 7- to 15-fold reduction in recruitment efficiency reported in [23]) and examined three infection prevalence levels: 20%, 50% and 80%. Values for all other model terms were maintained as in Sumpter & Beekman [2] (table 1), such that the original model is equivalent to our modified model at 0% prevalence. We found

that parasitism disrupted colony preference for the high-quality food source (feeder A), and the magnitude of this effect increased with parasite prevalence (figure 1*a*). At low (20%) and intermediate (50%) prevalence, the collective decision to exploit the higher quality food source (feeder A) over the lower quality food source (feeder B) was robust to the presence of parasites. However, at high prevalence (80%), the number of ants visiting feeder A was nearly equal to the number of ants visiting feeder B by the end of the trial. Thus, collective resource exploitation can be quite resilient to parasite infection until high levels of parasite infection are reached.

In a second scenario, we modelled parasite disruption of the ants' ability to differentiate the high-quality resource from the low-quality resource, as may occur in other insects like bees [24]. To model this 'reduced discrimination' scenario, we set the infected ant recruitment rate for the high-quality feeder equal to the rate for the low-quality feeder. Here, we found that infection also reduced the colony preference for the high-quality feeder (figure 1*b*). However, collectively, the ants made the correct choice at each prevalence level, despite individual ants being more evenly distributed among the two feeders. This latter observation suggests that collective behaviour may be resilient to certain types of changes in individual behaviour.

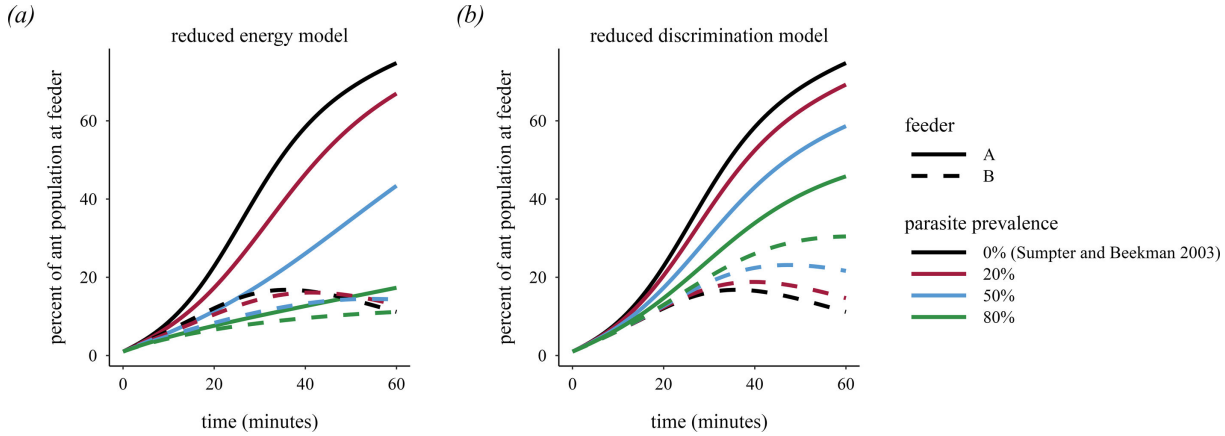


**Figure 1.** Predictions of an ant foraging model that accounts for parasitism under two different scenarios: (*a*) iInfected ants deposit less pheromones ('reduced energy' model) or (*b*) iInfected ants are unable to differentiate between high and low quality of food sources ('reduced discrimination' model). The number of ants visiting the high-quality food source (feeder A; solid lines) or low-quality food source (feeder B; dashed lines) was tracked for three levels of infection prevalence: 20% (red), 50% (blue), and 80% (green). The prevalence level of 0% (black) is equivalent to the original Sumpter & Beekman [2] model.

**Table 1** Parameter values for the Sumpter & Beekman model [2] and our modified model accounting for parasitism. The 'reduced energy' scenario models the effect of parasites on host energy budget, and the 'reduced discrimination' scenario models the effect of parasites on host resource discrimination.

| parameter | | reduced energy model | reduced discrimination model |
|---|---|---|---|
| **from Sumpter & Beekman [2]** | | | |
| total number of foraging ants | $N$ | 100 | 100 |
| rate at which ants lose pheromone trail | $S$ | 1 | 1 |
| saturation constant | $K$ | 10 | 10 |
| rate that ants randomly find a feeder | $a$ | 0.0052 | 0.0052 |
| strength of recruitment to feeder A by uninfected ants | $\beta_A$ | 0.0015 | 0.0015 |
| strength of recruitment to feeder B by uninfected ants | $\beta_B$ | 0.0010 | 0.0010 |
| **added in modified model** | | | |
| prevalence of infection (%) | $P$ | 20, 50, 80 | 20, 50, 80 |
| strength of recruitment to feeder A by infected ants | $\beta_{iA}$ | 0.00015 | 0.0010 |
| strength of recruitment to feeder B by infected ants | $\beta_{iB}$ | 0.00010 | 0.0010 |

## 2. Information transfer: a general link between parasitism, individual behaviour and collective performance

To understand how parasites influence group-level animal behaviour, it is useful to consider the mechanisms by which individual interactions scale up to collective behaviour. The sharing of information gives individuals access to knowledge about the environment they would not otherwise have, enabling them to coordinate activity and facilitating the emergence of collective behaviour [1]. In many groups, individuals process personal and shared information to arrive at collective decisions (e.g. choosing a nesting site [25]), and group decisions often determine collective movement patterns (e.g. foraging routes [26]). Transferred information is also used to synchronize collective actions (e.g. predator evasion [27]). Coordinated behaviours also result in the construction of physical structures that no one member could produce alone (e.g. nests [28]). Notably, when parasites change the behaviour of individuals, information transfer is vulnerable to disruption, with potentially cascading

effects on collective outcomes (figure 2). Of course, parasites can also have lethal effects on hosts that eliminate the participation of infected individuals in collective behaviours. However, here we focus on more subtle non-lethal effects of parasites on behaviour.

Successful information transfer requires that signals or cues transmitted by one individual are detected by and modify the behaviour of other individuals. Parasites can affect this process by disrupting an individual's ability to transmit or detect relevant signals (figure 2). For example, coordinated swarming is a hallmark of locust behaviour and shifts from solitary to gregarious swarming occur in response to the production and detection of aggregation pheromones by individual locusts [29]. However, in the oriental migratory locust (*Locusta migratoria manilensis*), infection by a microsporidian parasite (*Paranosema locustae*) downregulates the ability of infected locusts to produce aggregation pheromones, which in turn reduces the biosynthesis of serotonin and dopamine [30], two neurotransmitters that initiate and maintain gregarious behaviour [30,31]. Mechanistically, these changes occur because *P. locustae* alters the physiology of the locust hindgut in ways that suppress growth of gut bacteria that produce aggregation pheromones [30,32]. Consequently, the parasite disrupts not only the ability of infected locusts to transmit the swarming signal but also the capacity of infected individuals who receive the signal to translate it into changes in aggregation behaviour. Uninfected locusts exposed to infected individuals also show reductions in serotonin production and aggregation propensity [30], suggesting that the impact of infection on information transfer is both direct (i.e. manifests in infected individuals) and indirect (i.e. cascades to uninfected individuals). Given these dual effects, variation in parasite prevalence among locust groups could translate into substantial differences in the occurrence of group swarming behaviour across populations.

Parasites can also influence information transfer by affecting the content of information being transmitted (figure 2). For instance, some viruses may affect the quality of information about food sources transmitted by honeybees (*Apis mellifera*) via effects on host sensory perception and learning. Specifically, honeybee foragers infected with either deformed wing virus (DWV) or Israeli acute paralysis virus (IAPV) have impaired sensory perception and learning abilities, leading to a proboscis extension response that occurs at lower sucrose concentrations [24,33]. This behavioural change could affect the quality of information about resources that infected foragers share with conspecifics. DWV also disturbs memory formation in foragers [33], while IAPV depresses their ability to navigate back to the hive [34]. These two effects might influence the nature of information that infected individuals share with conspecifics, as well as the ability of foragers to retain and transfer this information.

By disrupting the transmission and quality of information shared between conspecifics, parasites could have profound effects on collective performance. In bees, because positive feedback in the information transfer process facilitates the spread of information throughout a colony [35], parasitism may amplify suboptimal information about resource quality and location, generating intergroup heterogeneities in resource acquisition between infected and uninfected groups. In locusts, the effects of parasitism on aggregation signalling could generate substantial variation in the occurrence of group swarming across populations driven by differences in infection prevalence. More generally, parasite-associated alterations in information transfer may lead to predictable changes in collective behaviour that manifest across host and parasite taxa. Below, we explore links between parasitism, information transfer and four common collective behaviours (decision-making, collective movement, synchronization and construction; figure 2), using variation in host and parasite traits to predict when parasitism should have the strongest consequences for group-level performance, which determines the average fitness of individuals within the group.

## 3. Consequences of parasitism for common collective behaviours

### (a) Decision-making

Collective decision-making is the process by which social animals reach key decisions, such as where to forage or where to move using cues or signals from group members. Information sharing allows groups to make faster, more accurate decisions that enhance group cohesion and individual fitness [36,37]. Although collective decision-making typically dampens individual errors and reduces the risk of mistakes [37], the transfer of parasite-altered information might compromise collective decisions depending on how this information flows through the group. For example, control over collective decisions can range from (i) highly centralized where a single leader determines group behaviour (e.g. grey wolves [*Canis lupus*] [38]) to (ii) fully distributed where each individual has influence over group decisions (e.g. olive baboons [*Papio anubis*] [26]). By structuring how parasite-altered information flows through a group, the degree to which decision-making is skewed from centralized to distributed may determine the vulnerability of collective decisions to the effects of parasitism.

When decision-making is centralized, a suboptimal choice made by a single leader is highly influential and spreads through the entire group. Thus, infection of a leader could result in a sharp transition from correct to incorrect group decisions (figure 3*a*, orange line). Models of homing pigeon navigation serve as an analogy—the navigational accuracy of a flock typically improves when movement decisions are driven by a single, highly accurate leader. However, if the leader is inaccurate, these errors propagate and reduce the navigational accuracy of the entire group [39]. As such, a leader's susceptibility to infection should determine the extent to which parasites affect collective decisions. When the leader is highly susceptible to infection, the probability that decision-making will be disrupted by parasites may be high. Interestingly, dominance status, which can reflect leadership in some species (e.g. yellow baboons [*P. ursinus*] [40]), is frequently associated with increased parasite risk in vertebrates [41]. Thus, centralized forms of collective decision-making may be especially vulnerable to parasitism in certain contexts. However, groups with leaders that are more resistant to infection and groups that show flexibility in their centralized decision-making structures [42] may be buffered against mistakes made by infected leaders.

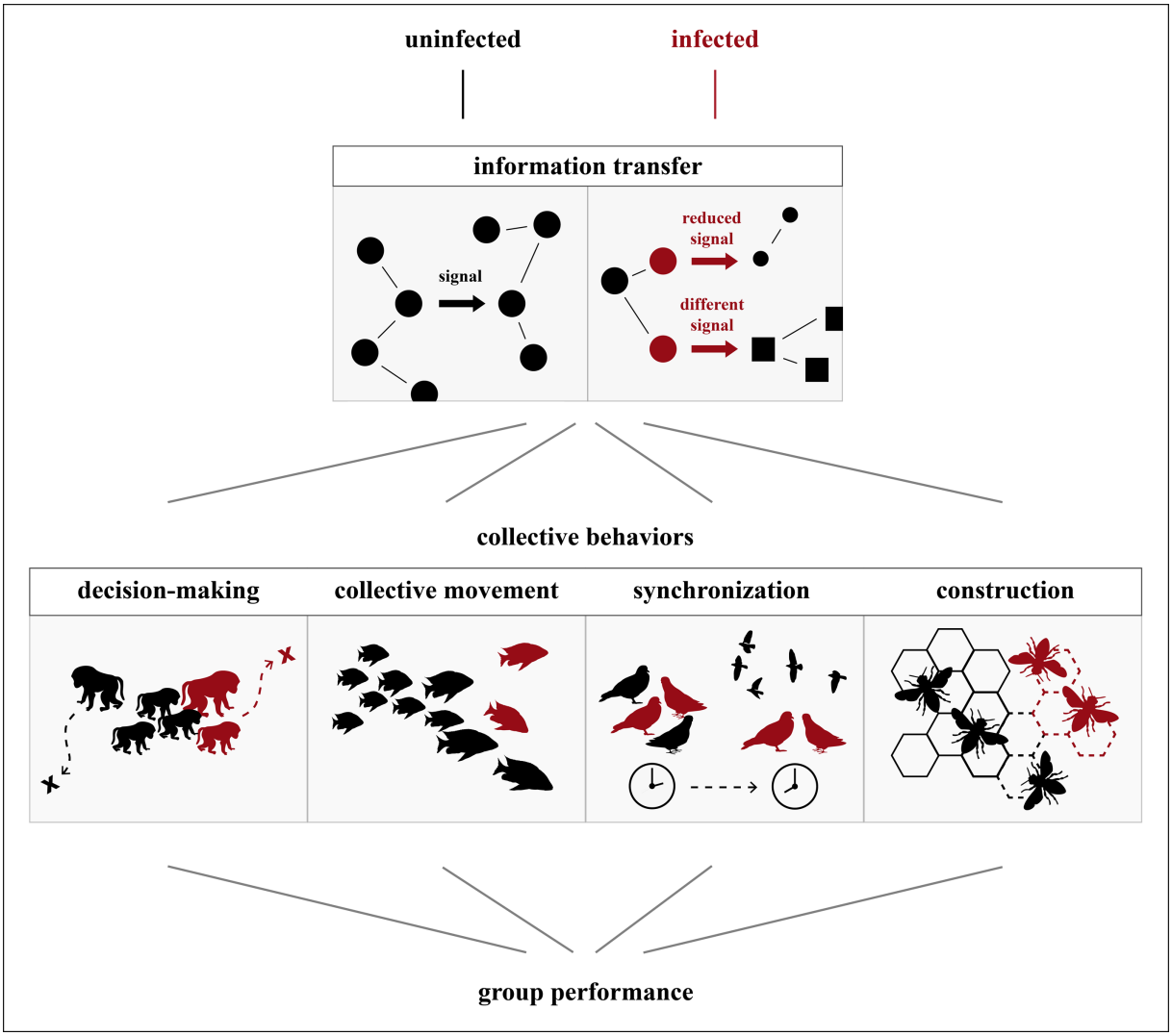


**Figure 2.** The effects of parasites on individual animal behaviour can shape how information is transferred among group members. Infected (red) and uninfected (black) individuals might differ in the quality and/or quantity of information they transfer to conspecifics. Therefore, the presence of infected individuals could compromise a group's ability to make decisions, move, synchronize activities or build physical structures, with implications for group performance, which determines the average fitness of individuals within the group.

For distributed decision-making, a suboptimal choice made by any one individual propagates more slowly through the group, thus key features of the decision-making process probably affect the precise shape of the relationship between parasite infection rate in the group and the likelihood of incorrect choices (figure 3*a*, blue line). For example, decisions based on compromise (e.g. olive baboons deciding to move in a direction that compromises between the choices of initiators with discordant views [26]) may mute incorrect choices induced by parasitism. Conversely, quorum responses, where a threshold number of individuals committing to a choice sharply increases the adoption of the behaviour by the rest of the group [36], may amplify incorrect decisions. As more individuals adopt a suboptimal behaviour (as they might when infected), other individuals are disproportionately biassed to conform to the common behaviour [43]. This process could result in a sigmoidal relationship between the number of infected individuals and the probability of suboptimal decision-making by the group, with some threshold number of infected individuals acting as an inflection point for the rapid adoption of the suboptimal decision (figure 3*a*, blue line). For instance, stickleback fish infected with the cestode *Schistocephalus solidus* spend less time shoaling and more time feeding in open water [44,45], and in experimental settings when infected fish compose the majority of a shoal, uninfected fish also adopt this more risky behaviour [46]. Finally, if infected individuals have discordant views, it is possible that as the prevalence of infection in a group increases, conflicts of interest resulting from excess heterogeneity among group members will also increase [47], hindering some forms of distributed decision-making.

## (b) Collective movement

Some of the most striking collective behaviours in nature involve animal groups moving together. These cohesive movements help relieve the costs of energetically demanding tasks, helping group members avoid predators, locate resources and access mating opportunities [48–50]. To coordinate movement, individuals transfer information about their direction, speed and/or position, while following simple rules of attraction and repulsion to optimize interindividual distances [1,51]. However, since parasites can generate heterogeneity in the ability of individuals to (i) move and (ii) perceive the movement of others [52],

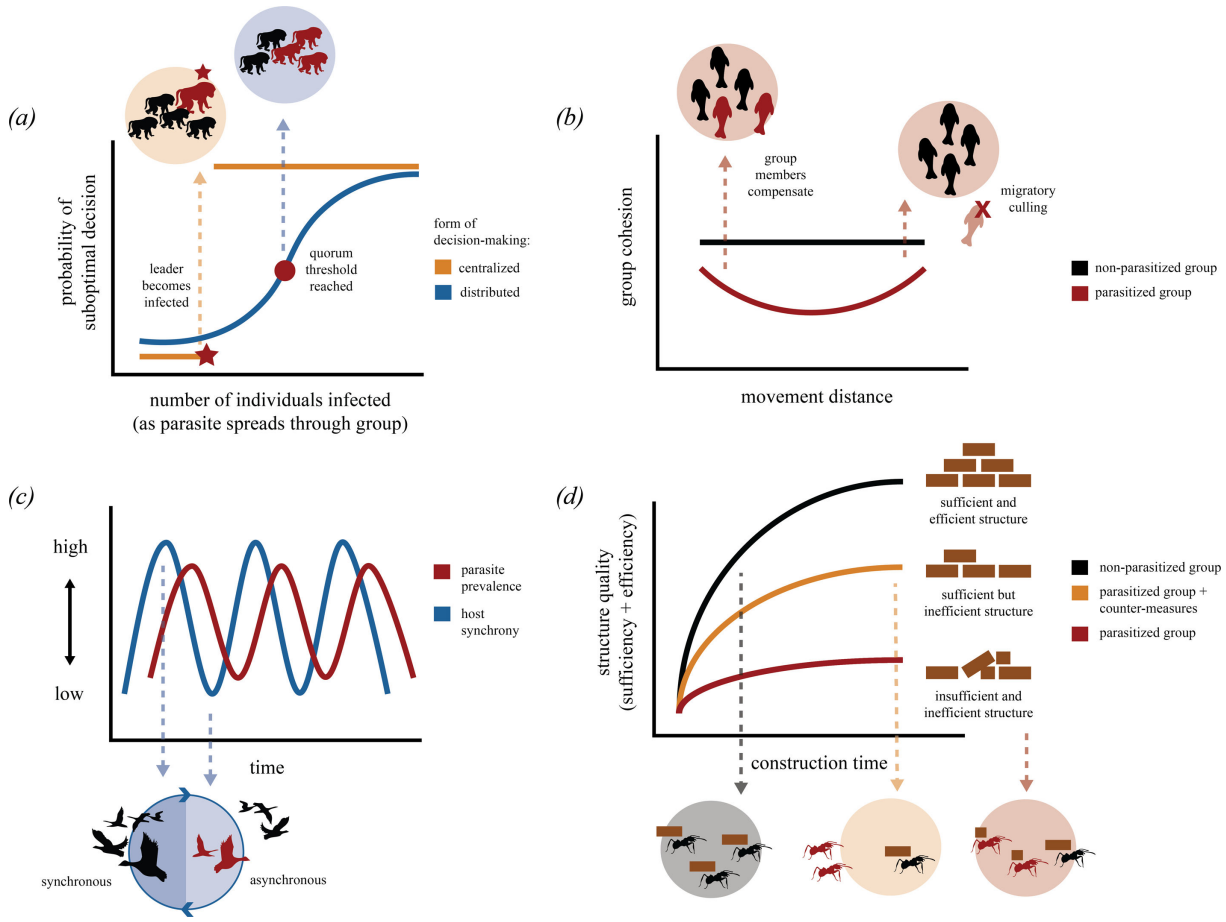


**Figure 3.** Hypothesized relationships between parasitism and common collective behaviours. (*a*) The consequences of parasitism for group decision-making and the likelihood of suboptimal decisions being made may depend on whether decision-making is a centralized or distributed process. (*b*) Movement distance may influence the degree to which parasites compromise group movements, with intermediate movement distances potentially being the least resilient to disruption. (*c*) If parasites desynchronize the activity of infected and uninfected group members, out-of-phase cycles of behavioural synchrony and parasite prevalence could emerge. (*d*) Parasite effects on group members could lead to the construction of either insufficient and inefficient physical structures (if infected individuals become cognitively impaired) or sufficient but inefficient physical structures (if infected individuals do not participate in construction activities and uninfected group members attempt to compensate).

parasitism may compromise collective movement, with variation in parasite traits helping to predict how these disruptions emerge.

Parasites that impose physical constraints on individual mobility should have strong effects on collective movement. For example, parasites like *S. solidus* take up a large proportion of the stickleback host body cavity, and infected fish show load-dependent declines in speed, acceleration and manoeuvrability [9]. Pairs of fish composed of either one or two infected individuals moved slower and were less aligned and responsive when startled compared to uninfected pairs [9]; and among slightly larger stickleback groups, both swimming speed and group cohesion differed between infected and uninfected groups [11]. Likewise, the trematode *Crassiphiala bulboglossa* can encyst in muscle tissue and disrupt mobility of killifish hosts [53,54]. These movement deficiencies impair the coordination of entire groups, which may explain the observation that killifish shoals with a high prevalence of *C. bulboglossa* infection adopt a broader, more phalanx-shaped formation compared to the narrow, elliptical shape of shoals with low infection prevalence [53]. Even when groups containing parasitized members maintain effective coordination, there may be subtle costs to group members. For example, baboons of different body sizes with different locomotor capacities adjust their movement patterns (e.g. stride frequency) to maintain spatial group cohesion during group movements [55]. Such adjustments could result in energetic costs for some individuals (smaller animals) and opportunity costs for others (larger animals), and increasing disparities in these costs could ultimately impose constraints on group movement [55]. A similar phenomenon could apply to infected and uninfected individuals.

Parasites that do not affect hosts physically can still disrupt collective movement by interfering with an individual's perceptual abilities, which are necessary for maintaining group cohesion during movement [51,56]. European starlings sense the activity of a consistent number of neighbours in their flocks to optimally balance individual opportunity costs with group cohesion [56]. However, passerine birds challenged with lipopolysaccharide, an immune stimulant that does not cause infection, often show sickness behaviours such as hypothermia and decreased locomotion [57], both of which may affect the number of neighbours sick individuals can perceive during flight. Since models predict that observing fewer than the optimal number of neighbours disrupts the trade-off between individual effort and group consensus [56], the movement efficiency of parasitized flocks could be impaired.

The consequences of parasitism for collective movement may vary not only across parasite types but also host movement distance. During short-distance collective movements, such as daily movements within a home range, both infected and

uninfected individuals may be able to adjust their movements, thereby maintaining group cohesion [55]. However, during long-distance movements such as migration, the higher energetic costs of movement may make adjustments impossible for infected individuals, ultimately resulting in their selective removal at the longest migration distances (e.g. migratory culling [58]). These differences could generate a nonlinear relationship between parasite disruption of group cohesion and movement distance, with negative effects of parasitism manifesting most prominently at intermediate distances (figure 3*b*).

## (c) Synchronization

When individuals adjust the timing of their activities to conform to that of neighbours, synchronization emerges [1]. Whereas solitary animals independently determine how to allocate their time among behavioural tasks, information sharing allows group members to coordinate their behaviours, thereby optimizing group activity [59]. Behavioural synchrony is facilitated by phenotypic similarity among group members [60,61] and can provide benefits such as reduced individual predation risk and increased group productivity [62,63]. However, parasitism may disrupt behavioural synchronization by introducing excess heterogeneity in individual behaviour. Infection-induced changes in the ability of group members to (i) send and (ii) receive social information may represent distinct mechanisms by which parasitism breaks down collective synchrony.

First, disrupted information transfer from infected to uninfected individuals may interrupt the flow of information individuals use to synchronize behaviour. To maintain synchrony, group members can use information from conspecifics to determine when to switch between behavioural states in a coordinated fashion. This information often propagates through the group as a wave, with group members responding to and simultaneously passing on signals to those closest to them [59,64]. Parasitism might disrupt synchrony if infected individuals cannot contribute to the information wave. For example, when *S. solidus*-infected sticklebacks were positioned in the middle of a shoal, group anti-predator responses were out of sync when compared to uninfected groups. Parasitized groups differed because the lack of an escape response by infected fish inhibited the responses of uninfected fish positioned further from the source of the attack [64]. Notably, the extent of such desynchronization probably depends on the position of infected individuals within the group relative to the stimulus inducing the change in behaviour [64]. In the stickleback study, infected individuals were placed in central positions within groups; however, in free-ranging fish shoals, infected individuals also occupy peripheral positions (e.g. killifish [53]). If these peripheral positions represent the closest or farthest points from environmental stimuli, parasitism could have stronger or weaker effects on synchrony, respectively.

Another way that parasites might interfere with collective synchrony is by preventing infected individuals from responding to information received from group members. For example, parasites that infect host sensory organs can directly interfere with the perception of conspecifics [65,66]. Changes in physiological state could also lead parasitized individuals to modify their activity patterns in ways that deprioritize responding to signals from group members, thereby desynchronizing the behaviour of infected and uninfected group members. Indeed, a meta-analysis of animal migration studies found that across birds, fish and insects, infection was associated with a significant negative effect on arrival and departure dates, stopover site arrival and staging time [67]. For example, yellow-rumped warblers (*Setophaga coronata*) infected with haematozoan parasites tended to reach stopover sites later than their uninfected counterparts [68], while other bird species spent significantly more time at stopover sites when infected [69]. This is likely because infected birds need to spend more time refuelling to support the combined energetic costs of migration, infection and immune activation. Thus, differing behavioural priorities of infected versus uninfected individuals can introduce noise into the behavioural signals that facilitate synchrony.

Interestingly, the breakdown of synchronization due to infection could act to reduce parasite transmission, conferring some benefits to hosts. A mathematical model by Bauer *et al.* [70] predicted that bird flocks migrating asynchronously should have lower infection prevalence than synchronous migrants due to reduced contacts between infected and uninfected hosts. Over long timescales, this phenomenon might generate cyclical feedback between collective synchrony and parasite transmission (figure 3*c*). Asynchronous migration of parasitized groups may decrease parasite transmission due to temporary separation between susceptible individuals that move and infected individuals that are left behind. If reduced transmission and lower energy expenditure due to delayed migration allow parasitized birds to recover and eventually resynchronize their behaviour, groups could regain collective synchrony. However, this would once again facilitate transmission, increasing parasite prevalence. Such feedback could lead to offset oscillations in behavioural synchrony and parasite prevalence (figure 3*c*).

## (d) Construction

Some animal groups construct impressive physical structures resulting from interactions between group members and the environment. Collective behaviours are responsible for the construction of bee hives, termite mounds and the hunting webs of social spiders [71], and these structures are often central to group survival and reproduction. Construction arises from simple rules based on pheromones, cues from existing structures and available resources [28,72], allowing groups to produce building blocks of sufficient integrity and then assemble these building blocks efficiently (figure 3*d*, black line). However, parasite-related changes in information transfer might disrupt collective construction by (i) leading to structures of insufficient integrity (figure 3*d*, red line) or (ii) generating a trade-off between sufficiency and efficiency, leading to structures that are well-constructed but of suboptimal size (figure 3*d*, orange line).

If infected individuals are unable to process information from the environment and nestmates, they may be unable to retain information about construction rules or produce construction materials (e.g. spider silk, beeswax). This could lead to the production of subpar building blocks that reduce the integrity of built structures. For example, worker honeybees use a

wax produced in their abdomen to build honeycombs [73]. Combs have stereotypical hexagonal cells that individual workers create following cues from existing structures [74], and workers must also accommodate cells of different sizes and shapes and irregular comb configurations [75]. Individual cognitive ability is critical for these building tasks since workers must learn elements of the building technique such as the angle and size of cells [76] and solve design challenges imposed by the nest environment [75]. Since parasites negatively affect cognition and memory in honeybees [24,33,34], colonies with infected workers may construct less optimal combs, and these errors could be amplified over time via positive feedback. Computer simulations show that even small changes in behavioural construction rules lead to dramatic differences in the arrangement of the resulting structure [74]. Thus, parasite-compromised construction might provide less space for brood items or stored resources, alter ventilation and/or increase exposure to environmental stressors [77].

Parasitism might also affect construction outcomes if infected individuals do not participate in construction activities. A lack of participation could arise as a host defence against infection (e.g. self-isolation) or due to sickness behaviour (e.g. lethargy). While this phenomenon could help reduce building errors, the shift in behaviour could result in fewer workers being available for construction efforts, leading to well-constructed (i.e. sufficient) but smaller (i.e. inefficient) structures. For example, in termite colonies infested with mites, both infected and uninfected individuals spend more time resting and less time transporting building materials for nest construction [78]. Similarly, in social wasps, colonies with a high prevalence of gregarine infection foraged less for nest materials, had fewer competent workers and constructed smaller nests per capita. In turn, these smaller nests resulted in smaller brood sizes [79].

## 4. Ecological and evolutionary implications of parasite-altered collective behaviour

Our exploration of the effects of parasites on common collective behaviours reveals a variety of ways that parasites, via the disruption of information transfer between individuals, may change the collective performance of groups. Testing these predictions across a broad range of host and parasite taxa will add to our understanding of when, how and why the environment generates variation in animal collective behaviour and the specific role parasites play in generating among-group variation. Collective behaviour influences almost all aspects of how social animals navigate their environment; therefore, we expect parasite-associated alterations in collective behaviour to have broad ecological and evolutionary relevance. Here, we use parasite population dynamics, wildlife conservation and animal culture as examples to illustrate some of these potentially far-reaching consequences.

Many of the same social rules that allow individuals to transfer information and function as a collective also affect the transmission of parasites, resulting in an infection–information trade-off [80]. Animals may manage this trade-off by modulating either their social interactions [80] or the relative composition of behavioural phenotypes within groups [22]. Such adaptive modulation is supported by work on social immunity in ants, where exposure to a fungal parasite altered the social interactions of foragers, dampening colony-wide parasite transmission [81]. This example emphasizes the inherently bidirectional nature of the connections between parasitism and collective behaviour, where parasite infection triggers some change in collective behaviour that then feeds back on parasite dynamics [22]. In the case of social immunity, collective behaviour is deployed as a defence against infection, and the consequence is a reduction in parasite transmission. In other contexts, parasite-associated changes in collective behaviour may increase transmission within or between groups instead. For example, by influencing individual social behaviour, high predation risk often promotes more cohesive and coordinated group structures that reduce an individual's risk of attack [82]. However, parasitism can disrupt group cohesion and coordination, as shown in sticklebacks infected with the tapeworm *S. solidus* [11]. Sticklebacks serve as intermediate hosts for *S. solidus*, while piscivorous birds are the definitive hosts. Thus, successful bird predation on fish facilitates parasite transmission to definitive hosts. *S. solidus* is thought to manipulate individual fish behaviour to increase host vulnerability to predation and promote its own transmission [83], and observed changes in group-level behaviour may be an extension of this manipulation [11,21]. If so, in the stickleback-*S. solidus* system, parasite-induced changes in fish collective behaviour could result in increased fish predation, thereby promoting parasite transmission from fish to birds and ultimately back to fish. Indeed, the manipulation of host collective behaviour could be a general mechanism by which some parasites promote their own transmission. Interestingly, parasite-induced changes in host group characteristics such as cohesion should not always benefit parasites [21], and this may depend in part on parasite transmission mode. For parasites transmitted through the food chain like *S. solidus*, disrupting group cohesion could be advantageous if these changes increase predation on infected hosts. In contrast, for directly transmitted parasites that require close contact between hosts, disrupting group cohesion could increase dispersion among potential hosts (e.g. increased nearest neighbour distances [11]), reducing within-group transmission.

The effects of parasites on individual behaviour have relevance for wildlife conservation [84]. Likewise, parasite-induced changes in the collective behaviour could have implications for conservation, for example, by accelerating host population declines. When populations fall below a critical size threshold, concomitant declines in individual fitness and population growth rates can lead to a free fall towards extinction, a phenomenon called the Allee effect or positive density-dependence [85]. Interestingly, collective behaviours often generate positive feedbacks between group dynamics and individual payoffs, prompting the idea that collective behaviour could be one mechanism underlying Allee effects [86,87]. If so, parasite-associated disruptions of collective behaviour could contribute to host population extinction. A possible example is the rapid effect of colony collapse disorder (CCD) on honeybee population declines [88], which may reflect not only the virulence of the parasites contributing to CCD but also positive feedback between colony size, collective behaviour and colony performance. First, rapid mortality of individual bees (a parasite virulence effect) reduces population size to a critical level. Next, parasite-induced declines in colony size disrupt collective behaviour, preventing efficient completion of core tasks (e.g. finding food, rearing

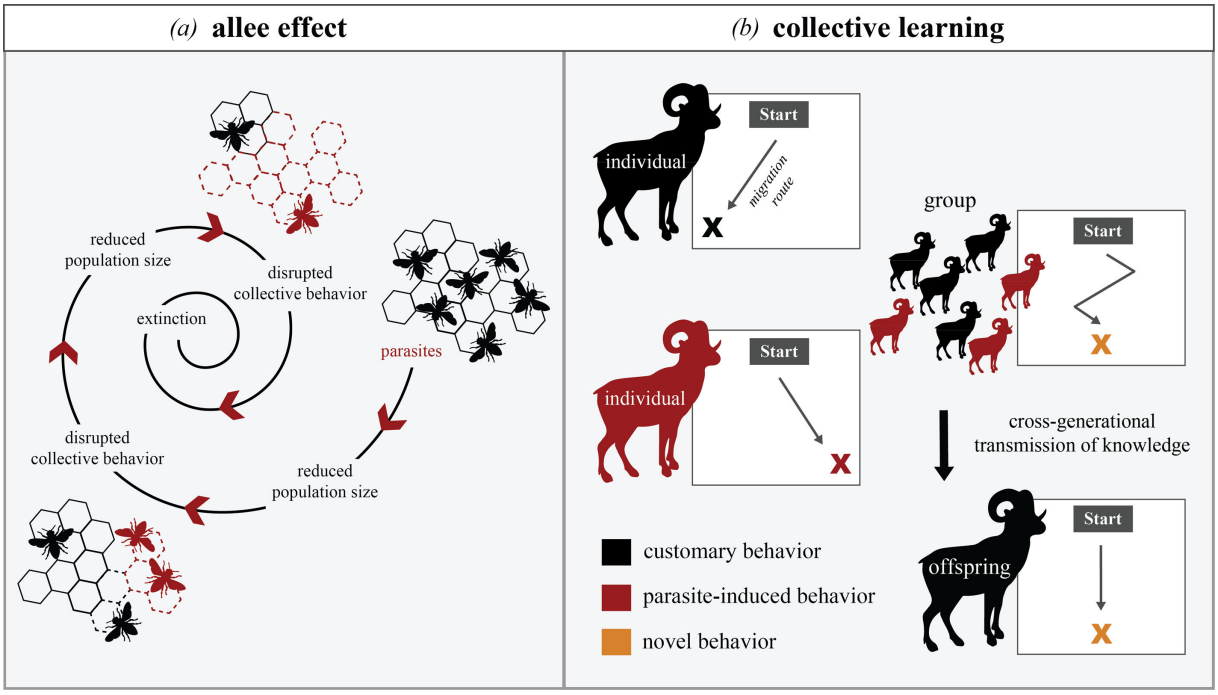


**Figure 4.** Parasite effects on collective behaviour have relevance for ecological and evolutionary processes. (*a*) Parasite-associated changes in the collective behaviour could accelerate host population declines via Allee effects. (*b*) Parasite-associated changes in how infected animals process social information could skew cross-generational transmission of cultural knowledge.

brood), thereby reducing colony performance and fitness. Hypothetically, this chain of events could trigger a positive feedback process where small colony size impairs group performance, further reducing colony size, ultimately leading to extinction (figure 4*a*). Models of CCD dynamics suggest that both initial colony population size and severity of infection help predict whether a colony will survive infection [89,90]. The influence of population size is likely due to Allee effects since populations above a critical size may be more robust to the loss of infected individuals [90] and one mechanism by which this Allee effect could operate is collective behaviour.

Culture, or behaviour that is learnt and transmitted between individuals, is widespread among animal species [91]. Cultural variation among populations typically arises as a result of local information transfer and culture spreads through social and collective learning processes [86,92]. As such, culture is likely sensitive to the effects of parasites, with potentially broad ecological and evolutionary ramifications [93]. For example, foraging innovations, such as self-medication, which are often transmitted vertically or horizontally between individuals [94], are observed more frequently in populations with high risks of infection [95]. More nuanced effects of parasites on animal culture may also emerge through impaired information transfer. For example, migration cultures of fish, birds and ungulates are thought to arise in part from collective learning, with migratory routes being shaped over time by group knowledge [86,96]. However, infected individuals may be less able to discern migratory paths with higher resource quality or may have different resource preferences than uninfected individuals, introducing variance in movement patterns (figure 4*b*). Moreover, since the environmental experiences of individuals in collective groups are influenced by the experiences of others via collective learning [97], parasites may affect what uninfected group members learn about the environment, especially if uninfected individuals take cues from infected individuals (figure 4*b*). This might ultimately alter collective memory for migratory paths, fundamentally changing the group knowledge that is passed down intergenerationally (figure 4*b*).

## 5. Conclusions

Collective behaviour emerges from interindividual interactions. Individual differences in behaviour can modulate collective outcomes, but information is lacking on relevant sources of these individual differences and how they translate to changes in group behaviour [47,98]. Parasitism is a potent source of heterogeneity in individual behaviour [99], thus our conceptual framework proposes that parasites may be an overlooked source of variation in collective behaviour in animal groups. Empirical studies testing the hypotheses outlined in this article can help advance our understanding of this phenomenon. Future research should pay particular attention to key components of the ecology, life history and behaviour of both hosts and parasites (e.g. host leadership type or parasite infection site) which may determine the magnitude of parasite effects on collective performance. Given widespread evidence that infectious diseases modulate social interactions across the animal kingdom [7] and a growing understanding of the role of collective behaviour in epidemiological dynamics in humans [100] and other animals [81], the time is ripe for a deeper integration of the fields of disease ecology and collective behaviour.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. Code supporting this article has been uploaded as part of the supplementary material [101].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. K.A.S.: conceptualization, investigation, project administration, visualization, writing—original draft, writing—review and editing; S.P.K.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; A.A.L.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; R.L.R.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; M.S.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; C.L.S.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; E.M.W.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; C.L.W.: conceptualization, investigation, visualization, writing—original draft, writing—review and editing; T.S.: conceptualization, investigation, supervision, visualization, writing—original draft, writing—review and editing; V.O.E.: conceptualization, investigation, project administration, supervision, visualization, writing—original draft, writing—review and editing.

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