**Mechanisms and Consequences of Infection-induced Phenotypes**

ABSTRACT

1. Researchers studying animal phenotypes often overlook the potential influence of parasites hiding inside their study organisms. Yet, most wild animals host parasites, which can alter individual phenotypes (e.g., morphology, physiology, behaviour).
2. Infection-induced phenotypes stem from several non-mutually exclusive mechanisms (i.e., adaptive host defences, infection-induced debilitation, and host manipulation by parasites), driven by parasites and/or hosts.
3. Changes in host phenotype can impact all levels of biological organization, from cells to communities. The nature and magnitude of these effects can vary depending on the biotic and abiotic environmental factors experienced by hosts and parasites.
4. This special feature highlights recent insights into the ways parasites alter host phenotypes across a range of systems. Here, we contextualize how each contribution expands our knowledge of the role of parasites in driving individual variation in animal phenotypes.
5. Looking to the future, we need to better understand how infection-induced phenotypes fluctuate with natural variation in infection (e.g., infection intensity, co-infection) and whether studies in lab-based environments provide strong proxies for host-parasite interactions in the wild. The time is ripe to acknowledge, critique, and discuss the implications of infection on host phenotypes across taxonomic boundaries and biological levels of organization.
6. INTRODUCTION

Biologists often assume that their study organisms are healthy, performing to the best of their abilities given their environment, and acting to maximize their own fitness. In reality, most wild animals are host to multiple parasite species (Viney & Graham 2013), some of which can drive substantial changes to an individual’s morphological, physiological and behavioural phenotype (Moore 2002; Poulin 2007; Lopes 2023). Consequently, biologists studying almost any aspect of animal ecology need to be aware of the myriad of ways that infection may interact with the hosts’ traits being measured.

Here, we define a parasite as any organism (or virus) with a harmful, obligate and durable consumer relationship with an individual of another species (Parmentier & Michel 2013), including bacteria, fungi, protozoa and metazoa. Although the complex interactions among host phenotypes and parasite infection have been studied by researchers for decades, phenotypic variation driven by infection has often historically been overlooked rather than interpreted as ecologically relevant with important evolutionary consequences (Minchella 1985; Ezenwa *et al.* 2016). Still today, many researchers do not consider parasites as important sources of intra- and inter-individual variation in the phenotypic traits of their study organisms (Chretien *et al.* 2023).

This special feature highlights the various ways that parasites drive variation in host phenotypes. The contributed articles highlight diverse host-parasite systems across a broad range of taxa and include perspective and concept articles (Adamo 2023; Lopes 2023; Pavey & Vyas 2023; Poulin, Jorge & Salloum 2023; Stockmaier *et al.* 2023; Tillman & Adelman 2023), a systematic literature review (Chretien *et al.* 2023), a meta-analysis (Wu 2023) and observational and experimental research conducted in laboratory and field settings (Alves & Aubin‐Horth 2023; Ngo *et al.* 2023; Salerno, Buck & Kamel 2023; Seguel *et al.* 2023; Vindas *et al.* 2023; Will *et al.* 2023). In this Editorial, we contextualize each featured article’s contribution to our understanding of how parasites generate intra- and inter-individual differences in host phenotypes, how this trait variation reverberates across levels of biological organization from cells to communities, and the role of the host’s external environment in modulating these effects. We also provide our perspective on the future of research on infection-induced phenotypes in light of current and future global changes that may alter disease dynamics in both the short and long-term.

1. MECHANISMS FOR INFECTION-INDUCED PHENOTYPES

There is growing recognition for how widespread infection-induced phenotypic changes are in natural systems, and the mechanisms driving these changes in individuals remain an active area of research. The observed phenotype of an organism represents the combined net result of efforts by its parasites to colonize their hosts, extract resources, and evade host defences, melded with the host’s strategies to avoid infection itself and minimize the costs associated with infection once established (Sarabian, Curtis & McMullan 2018). These complex interactions between the host and its parasites make it challenging to determine whether an observed infection-induced phenotype represents one or more broad mechanisms, including adaptive host defences, parasite-induced host debilitation, and host manipulation by parasites.

Adaptive host defences include changes in host morphology, behaviour, or physiology that reduce parasite exposure, load, and pathology (Poulin 1995; Curtis 2014; Lopes 2023). Hosts can limit parasite exposure using avoidance-type behaviours (e.g., avoiding environments that harbour high densities of infectious propagules) (Stockmaier *et al.* 2023) and pre-exposure shifts in physiology (e.g., altering host phenotype in response to cues of infection from conspecifics) (Lopes 2023). Following infection, parasite defence strategies can include resistance-type (limit parasite load; e.g., grooming) and tolerance-type (reduce the infection’s pathology without altering infection intensity; e.g., food-sharing among social individuals) behaviours (Stockmaier *et al.* 2023). In the review by Tillman and Adelman (2023), the authors connect parasite exposure and infection with host foraging behaviour, describing, for example, self-medication, where hosts preferentially ingest foods containing compounds that fight infection. Lethargy, a common sickness behaviour, can also help hosts to reduce energy investment in non-essential activities so that energy can be reallocated to processes that enhance fitness (e.g., immunity, predator vigilance) (Lopes *et al.* 2021; Tillman & Adelman 2023). Behavioural alterations can occur even in the absence of any detectable infection; Vindas *et al.* (2023) show that parasite-exposed (but uninfected) medaka *Oryzias latipes* exhibit greater activity, aversion to risk, and sociality than individuals that were not exposed to parasites.

While many of the above phenotypic changes represent adaptive host responses, they were historically attributed to parasite-induced host debilitation. Wu (2023) presents a thorough meta-analysis of the various functional disruptions induced in amphibian hosts by the debilitating chytrid fungus (*Batrachochytrium dendrobatidis*). The analysis indicated that across host species, this infection leads to a higher resting metabolic rate, lower body condition, impaired osmoregulation, and altered behaviour in hosts. However, for over 30 years, researchers have appreciated that infection-induced phenotypes are not always maladaptive, but instead may represent a coordinated evolutionary strategy to fight attacking parasites (Hart 1988).

Alternatively, some parasites may actively manipulate their host’s phenotype to promote transmission to the next susceptible host. For example, trophically transmitted parasites that infect the Arctic charr *Salvelinus alpinus* enhance the host’s red coloration to make them more conspicuous to predators that act as the parasites’ final host (Johansen *et al.* 2019). Parasites manipulate host phenotype through a variety of mechanisms, such as altering biochemical signalling pathways. Alves and Aubin‐Horth (2023) used pharmacological treatments to investigate the biochemical mechanisms for reduced anti-predator behaviour (e.g., increased surface swimming) in three-spine stickleback *Gasterosteus aculeatus* hosts infected by the trophically-transmitted tapeworm *Schistocephalus solidus.* Infected sticklebacks have increased expression of a host gene that is important for myo-inositol synthesis, a molecule that is a precursor for important second messengers such as 1,4,5, triphosphate (IP3) (Alves and Aubin-Horth, 2023). The authors’ findings demonstrate the complex, multifactorial nature of parasitic manipulation: although reducing myo-inositol concentrations in infected fish partly reversed the effects of parasitism, infected fish did not, in fact, have increased myo-inositol levels. Pavey and Vyas (2023) provide a perspective in this special feature suggesting that parasite manipulation of hosts is an example of Richard Dawkins’ extended phenotype concept (Dawkins 2016) and may be accomplished through epigenetic changes in the host. Ngo *et al.* (2023) presents empirical evidence supporting a parasite-induced extended epiphenotype, as *Toxoplasma gondii-*infected, wild-caught mice exhibit the same DNA hypomethylation as lab-reared mice that were experimentally infected with *T. gondii*.

While researchers often label host phenotypic changes in response to parasite risk or infection into one of these three distinct categories (i.e., adaptive host defences, parasite-induced host debilitation, and host manipulation by parasites), infection-induced phenotypes likely result from some combination of these mechanisms. The contribution of each mechanism to the infection-induced phenotype can vary depending on factors such as host infection tolerance, host immunological resistance, parasite developmental stage, parasite load, and/or co-infection by multiple parasites. For example, Seguel et al. (2023) found that co-infection by blood-sucking helminths and coccidia parasites altered both the adaptive host response as well as the debilitating effects of infection. The blood sucking helminths stimulated a systemic immune phenotype that resulted in higher levels of shedding of co-infecting coccidia parasites. This enhanced coccidia shedding was associated with reduced host condition and reproductive success through the debilitating effects of that infection. Moving forward, further mechanistic studies at the molecular and biochemical level will help us understand how parasitic mechanisms and host responses integrate to produce the infection-induced phenotype.

1. BIOLOGICAL LEVELS OF VARIATION

Work on a range of host taxa, including insects (Adamo 2023), fish (Alves & Aubin‐Horth 2023), and mammals (Ngo *et al.* 2023), suggests that infection-induced phenotypes stem from immunological, neurophysiological, gene regulatory, and other cellular-level mechanisms (Adamo 2013). Epigenetic changes in hosts likely drive much of the infection-induced variation among individuals (Pavey & Vyas 2023). As one example of how epigenetic changes in the brain lead to increased behavioural variation within a population, male mice (*Mus musculus*) infected with *T. gondii* showed higher levels of hypomethylation of the arginine-vasopressin (AVP) promoter in the posterodorsal medial amygdala than uninfected controls (Ngo *et al.* 2023). Therefore, a population of mice with both infected and uninfected individuals would show a greater range in methylation of the AVP promoter than in uninfected populations. Research on laboratory mice demonstrates that changes in AVP-expressing neurons in this brain region are involved in reducing host avoidance of felid predators (e.g., Hari Dass & Vyas 2014). Therefore, infection increases the spectrum of anti-predator behaviours across individuals in the population via epigenetic changes in the brain.

These cellular-level effects result in a broadening of the range of phenotypes observed among individuals in a population, which inevitably alters conspecific interactions. Cues of infection risk from conspecifics can stimulate complementary behavioural avoidance mechanisms of the infected individual and physiological readiness for infection resistance, both of which represent anticipatory measures of parasite risk (Lopes 2023). While these changes in both behaviour and physiology should impact an individual’s social tendencies, the way in which sociality shifts likely depends on the animals’ typical social structure. Stockmaier *et al.* (2023) reviews how behavioural mechanisms of parasite defence shift in animals that range along the sociality spectrum from solitary to colonial. For example, social insects (i.e., colonial social structure) may use exposure to low, harmless parasite loads to enhance social immunity, while division of labour reduces risk of harmful parasite exposure to key individuals in the social hierarchy (e.g., the reproductive queen) (Stroeymeyt *et al.* 2018). On the opposite end of the spectrum, asocial animals (i.e., solitary lifestyle) have limited exposure to infectious conspecifics but may avoid aggressive encounters when they are themselves battling an immune challenge. Locatello *et al.* (2013) illustrate this effect in the solitary common octopus *Octopus vulgaris*, as immune-stimulated (with lipopolysaccharides) individuals interact less with a conspecific challenger than sham-treated individuals, likely to avoid competitive interactions when their ability to defend their resources is impaired.

Population-level effects likely reverberate through communities and ecosystems when such changes impact hosts’ heterospecific interactions, such as predator-prey encounters and heterospecific communication. Salerno, Buck and Kamel (2023) found that the presence of predator cues and infected conspecifics together drive phenotypic outcomes, such as personality, in snails. An understanding of the complex spatial interactions between predators and parasites in a given habitat (the overlaid “landscape of fear” and “landscape of disgust”; (Buck, Weinstein & Young 2018)) will be critical for predicting variation within and among hosts, especially in variable environments. Cooperative heterospecific interactions may also drive phenotypic changes related to parasite risk and infection. Lopes (2023) reinforces the importance of conspecific communication of infection cues for parasite readiness. Whether cues of infection from heterospecifics can also induce phentoypic changes in uninfected hosts, as occurs for heterospecific alarm calls signalling predation risk (Magrath *et al.* 2015) and brood parasitism (Lawson *et al.* 2020), is an exciting area for future work (Sarabian, Curtis & McMullan 2018; Friesen & Detwiler 2021).

1. ROLE OF ENVIRONMENT IN INFECTION-INDUCED PHENOTYPIC CHANGES

The way in which parasites alter host phenotypes can be strongly influenced by abiotic and biotic interactions with the environment, as highlighted by several papers in this special feature. Incorporating environmental context is inherently challenging, but key for understanding the fitness outcomes of infection for both hosts and parasites. In some systems, infection-induced changes vary with environmental context in ways potentially adaptive for parasites. Will *et al.* (2023) monitored hundreds of ant cadavers over more than a year to interrogate the role of biotic and abiotic factors in fungal manipulation of ant summiting behaviour. Several environmental factors, from vegetative type and canopy openness to humidity and precipitation, proved important in predicting cadaver presence. The authors highlight the value of such field-based and natural-history focused studies for uncovering the context-dependence of host-parasite interactions, and their potential adaptive drivers.

In other cases, environmental context can alter the costs and benefits of a given infection-induced phenotype for hosts. For instance, Tillman and Adelman (2023) discuss the potential for environmental factors, such as climate change and habitat fragmentation, to influence the interactions between infected host foraging decisions and disease dynamics. The authors argue that such complexities are critical for understanding the consequences of global environmental changes, both for a given host-parasite interaction, but also for the surrounding biological communities impacted by altered host behaviour (Tillman & Adelman 2023).

Cues from hosts and their symbionts can even interact in some systems. Poulin, Jorge and Salloum (2023) propose that parasites themselves have key species interactions that may influence the way in which they interact with their hosts. Parasites can alter the microbiome composition of their host, which could drive changes in host behaviour that are adaptive for the parasite (Poulin, Jorge & Salloum 2023). Similarly, parasites themselves differ in their microbiomes in ways that may enhance their own transmission. For example, Martinson *et al.* (2020) found that transmission of the *Howardula aoronymphium* nematode to its *Drosophila* host is greatly reduced if the nematodes are treated with an antibiotic that kills its intracellular bacterial symbiont, *Symbiopectobacterium*. Given the key role that environment plays in dictating a host’s microbiome (and likely that of its parasites) (Peixoto, Harkins & Nelson 2021), the role of the host and parasite’s microbiome in dictating the host’s phenotype remains an exciting area for future research.

1. FUTURE DIRECTIONS

Recognition of the pivotal role that parasites play in the variability of animal phenotypes in nature is surprisingly low (Chretien *et al.* 2023). Several contributions to this special feature present practical guidance for researchers working in a range of disciplines to assess the role of infection in their study system, including behaviour (Salerno, Buck & Kamel 2023; Stockmaier *et al.* 2023; Tillman & Adelman 2023; Vindas *et al.* 2023), performance (Chretien *et al.* 2023), physiology (Alves & Aubin‐Horth 2023; Lopes 2023; Seguel *et al.* 2023; Wu 2023), and epigenetics (Ngo *et al.* 2023). Yet, there are still many unanswered questions.

First, many studies refer to infection as a binary trait: uninfected versus infected. In reality, host infection may vary in intensity, with the role of infection varying drastically if the host has a low versus high parasite burden. Wu (2023) highlights the intensity-dependent effects of infection in amphibious hosts, finding that chytrid fungal infection only impacts reproduction at the highest infection loads. This paper underscores the importance of ecologically relevant metrics for understanding infection-related costs in hosts.

Further, researchers often study these ideas in hosts infected by a single parasite species. However, hosts may be co-infected by multiple parasite species with different transmission modes that compete for limited resources (Seguel *et al.* 2023). Similarly, a host may harbour more than one ontogenetic stage of a given parasite, which may also have diverging priorities in terms of how host phenotypes are impacted (Adamo 2023). Predicting the outcomes in terms of host phenotype when conflicts between parasitic species and life stages arise remains difficult (Ramsay & Rohr 2022).

To gain a better understanding of the effects of parasite-induced phenotypic change at the population, community and ecosystem level, we need further empirical evidence for how host-parasite interactions vary in real-world scenarios compared to controlled laboratory environments. Will *et al.* (2023) provide an impressive example for how this kind of study can be accomplished, by following the incidence of *Ophiocordyceps* fungi infected Florida zombie ant cadavers (*Camponotus floridanus*) for over 400 days at multiple sites to track how ecological conditions alter outcomes in this host-parasite system. Moving forward, more ecologically complex studies that incorporate these types of relevant factors (infection intensity, co-infection, environmental factors) will greatly advance our ability to tease apart the mechanisms behind infection-induced phenotypes in natural systems.

While biologists often work under the assumption that their study animals lack infections that drive major changes in their morphology, behaviour and physiology (Chretien *et al.* 2023), most animals are (co-)infected by parasitic species at different intensities that may vary in their impacts on hosts (Seguel *et al.* 2023; Wu 2023). Animal phenotypes are likely shaped and selected in response to both the risk of infection as well as infection itself (Alves & Aubin‐Horth 2023; Lopes 2023; Vindas *et al.* 2023). The recognition of parasites’ critical role in generating phenotypic variation among individuals is needed more than ever as we grapple with predicting how anthropogenic stressors, such as pollutants, climate change, and physical habitat destruction, will impact disease dynamics in the short and long-term (Tabachnick 2010; Ortega *et al.* 2021). The time is ripe to acknowledge, critique, and discuss the implications of infection on host phenotypes across taxonomic boundaries and biological levels of organization.

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CONFLICT OF INTEREST

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AUTHORS’ CONTRIBUTIONS

All authors contributed to writing and editing this editorial and gave approval for its submission.

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