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Focus

Phenotypic Variability Induced by Parasites: Extent and Evolutionary Implications

R. Poulin and F. Thomas

The diversity of ways in which parasites can modify the host genotypic signal has been documented in recent years. For example, parasites can shift the mean value and increase the variance of phenotypic traits in host populations, or alter the phenotypic sex ratio of host populations, with several evolutionary implications. Here, Robert Poulin and Frédéric Thomas review the types of host traits that are modified by parasites, then explore some of the evolutionary consequences of parasite-induced alterations in host phenotypes and suggest some avenues for future research.

Natural selection acts on phenotypes, favouring organisms with certain phenotypic characteristics over others. An organism's phenotype is the result of interactions between its genotype and various external factors that modulate the expression of the genotype. If phenotypes were perfect expressions of genotypes, then selection would also act directly on gene frequencies. Although it is not always the case, in general, organisms with the most extreme traits are also the ones with genotypes coding for maximum expression of the traits. There are, however, several environmental factors that generate noise in the expression of genotypes and weaken the link between selection on phenotypes and selection on genotypes. There have been numerous reports in recent years of parasites causing important shifts in the expression of host phenotypic traits. Parasites commonly act to scramble the host's genotypic signal, and the resulting host phenotype is a compromise between host and parasite genotypes. Parasite-induced phenotypic alterations are more than statistical noise, and can have important evolutionary consequences, given that parasites do not always neutralize host reproduction.

Parasites meet the conditions necessary to be direct agents of selection[1]. There has been some attention given to the role of parasites as driving forces of evolution, leading to long-term adaptive phenotypic responses by host populations[2–5]. However, most recent explorations of the evolutionary consequences of parasite-induced changes in host phenotype have stayed within the context of sexual selection, with much work focusing on the effects of parasites on secondary sexual characteristics[6,7] and levels of fluctuating asymmetry[8]. In this context, the evolution of hosts takes place because of the much lower reproductive potential of parasitized hosts. The evolutionary implications of parasite-induced changes in non-sexually selected traits when parasitized hosts still contribute offspring to the next generations are only now being considered.

Phenotypes influenced by parasites

Parasites modify a wide range of physiological, behavioural and morphological traits in their hosts (Table 1). Behavioural changes in particular have been well documented in a variety of host–parasite systems[9,10]. Often, the same parasite alters more than one distinct host trait, creating even more pronounced phenotypic differences between infected and uninfected hosts. For example, the cestode *Schistocephalus solidus* alters the vertical distribution[11], responses to large fish[12] and body colouration[13] of its stickleback intermediate host. From the stickleback perspective, these changes influence natural selection via predation by birds and fish, as well as sexual selection through mate choice. Crustaceans

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harbouring acanthocephalan cystacanths are also good examples of parasitized animals displaying alterations in both behaviour and body colouration[14].

It is important to note that in many reported examples of parasite-induced changes in host phenotypes, the evidence rests on comparisons using naturally infected hosts. Therefore, it is possible that some aberrant phenotypes are a cause of infection, rather than its consequence; only experimental infections can confirm absolutely that the differences between healthy and infected individuals are the result of parasitic infection.

Explanations for these changes fall into three categories, each difficult to distinguish from the others[10]. First, parasite-induced changes in host phenotype might represent adaptive responses by the host, aimed at eliminating or outlasting the parasite, or at minimizing its effects on host fitness. Second, changes in host phenotype might be the expression of parasite genes altering the host phenotype in ways that benefit the parasite, for example by increasing its probability of transmission. Third, parasite-induced alterations in the host phenotype might be mere pathological consequences of infection, not adaptive to either host or parasite. Each of the examples listed in Table 1 is the product of one or more of these three scenarios. Whatever their evolutionary origin, changes in host phenotypes can influence the immediate and long-term action of natural selection on hosts. The examples considered below assume that parasites alter host phenotypes without suppressing host reproduction completely (as is the case with many examples in Table 1).

Table 1. Some documented effects of parasites on host phenotypes[a]

Host[b]	Parasite	Phenotypic effect	Refs
Effects on motor activity			
Copepod (E)	Cestode	Increased swimming activity	16
Cockroach (E)	Acanthocephalan	Reduced running speed	37
Smelt and eel	Various helminths	Reduced swimming speed	38
Mouse (E)	Protozoan	Impaired motor performance	39
Mouse (E)	Nematode	Impaired motor performance	40
Effects on behaviour			
Snail	Trematode	Altered microhabitat choice	20, 41
Isopod	Acanthocephalan	Altered microhabitat choice	14, 15
Mayfly	Nematode	Female behaviour in males	30
Cockroach (E)	Acanthocephalan	Altered microhabitat choice	37
Beetle (E)	Cestode	Altered tendency to migrate	42
Sockeye salmon	Various helminths	Impaired orientation during migration	43
Bully (fish)	Trematode	Impaired predator evasion	44
Stickleback	Cestode	Altered vertical distribution	11
Killifish	Trematode	Decreased tendency to school	45
Mouse (E)	Nematode	Loss of dominance	46
Mouse (E)	Nematode	Altered microhabitat choice	40
Humans (E)	Nematode	Impaired memory	47
Effects on morphology			
Snail (E)	Trematode	Increased body size	48, 49
Isopod	Acanthocephalan	Altered body colouration	14
Midge	Nematode	Males resembling females	29
Mayfly	Nematode	Males resembling females	30
Stickleback	Cestode	Altered body colouration	13
Rat (E)	Cestode	Increased body size	50

[a] This list is not comprehensive.
[b] Studies using experimental manipulations of parasite levels are denoted by (E).

Parasites and the distribution of host phenotypes

Parasites affect many continuous phenotypic variables of hosts, such as body size or behavioural traits. Among uninfected hosts, the frequency distribution of trait values often follows a normal distribution that reflects genotypic differences combined with environmental noise. Parasitic infection can shift the mean value of a phenotypic trait one way or the other, and increase its variance in the overall host population (Fig. 1). Of course, the importance of this effect for the host population depends on the abundance of the parasite, or the mean number of parasites per host. When the host is a vertebrate, the influence of parasites is often, but not always, proportional to the severity of the infection. However, the presence of a single parasite is usually enough to induce an alteration in host phenotype when the host is an invertebrate (most cases in Table 1). Thus, prevalence, or the proportion of infected hosts, is often the key population parameter.

The distribution of the trait can be displaced but remain normal if the prevalence of infection is high; if prevalence is moderate the distribution of trait values is likely to become skewed (Fig. 1a). This is often seen in studies of behavioural modification of hosts by parasites: for most behavioural measures, the range of values obtained for parasitized hosts overlaps with that obtained for unparasitized hosts, but causes the overall mean value to shift towards one extreme[15,16]. Lauckner, studying shell sizes in marine gastropods, described the effects of larval trematodes as a 'grotesque distortion' of the frequency distribution of shell sizes[17]. The resulting overall population distribution was either highly skewed or multimodal, depending on locality, whereas that of the unparasitized component of the gastropod population was unimodal and normal.

If the shift in phenotype caused by parasites is large, and if prevalence is less than 100%, the distribution of trait values is likely to become bimodal, with parasitized and unparasitized individuals forming distinct groups (Fig. 1b). Whether the distribution of trait values becomes skewed or bimodal, it no longer reflects the distribution of trait values expected from host genotypes, and selection on that trait can be disrupted by parasites in many ways.

Strong environmental effects on phenotypes can render selection 'myopic', ie. capable of seeing and acting on only the phenotypes that happen to be present in given conditions[18]. Parasites are rarely considered as environmental effects on host phenotypes in an evolutionary context. However, changes in the frequency distribution of host traits caused by parasites are common, involving host traits such as body size[17,19], activity levels[16] or location in the habitat[20]. If natural selection is acting on the host trait, parasites can weaken the coupling between selection on phenotypes and selection on genotypes. For instance, if directional selection favours extreme values of a host trait, and if parasites cause

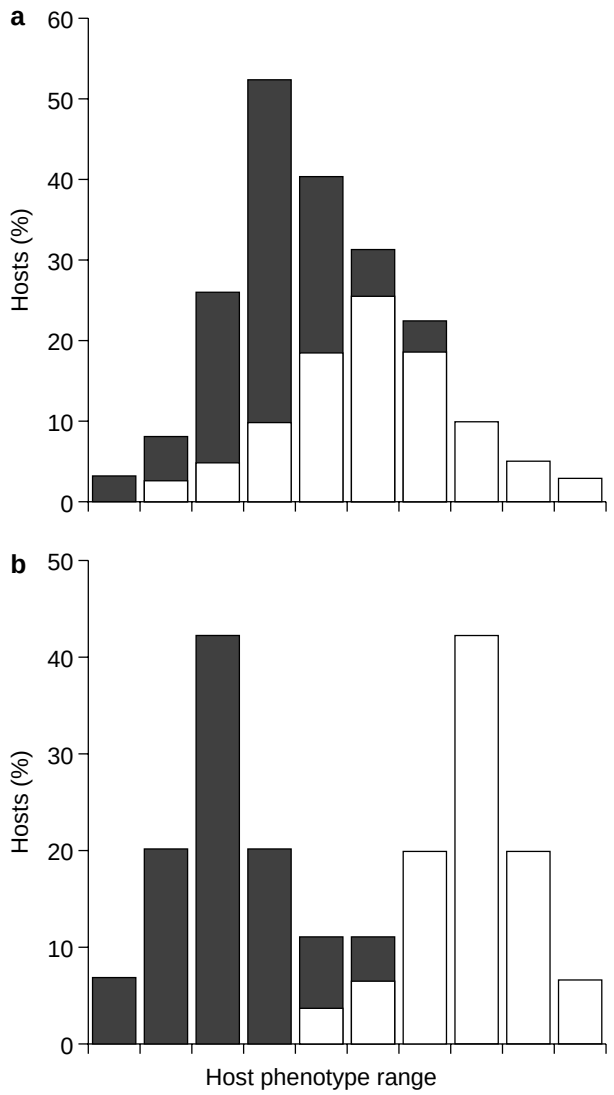


Fig. 1. Graphical illustration of the effect of parasitism on the frequency distribution of phenotypic traits of hosts. In these model examples, the prevalence of parasitism is 50%, and parasitism is assumed to decrease the mean trait value in both examples. The host trait could be any continuous, phenotypic character, such as body size, and the host phenotype range represents the range of trait values from the smallest one observed to the largest. In (a), trait values are normally distributed among both unparasitized hosts (open bars) and parasitized hosts (closed bars), and the two distributions overlap extensively; however, because parasitized hosts display lower trait values on average, the overall distribution is skewed to the left and the majority of hosts in the population have values smaller than the mean for unparasitized hosts. In (b), trait values are normally distributed among both unparasitized hosts and parasitized hosts, but the two distributions are almost disjunct; therefore, the overall distribution of trait values in the host population is bimodal, with unparasitized and parasitized hosts representing distinct morphs.

decreases in the trait, regardless of host genotype, then changes in gene frequencies for that trait might be reduced across host generations. In other words, individual hosts with genotypes coding for high values of the trait will not necessarily achieve the corresponding phenotype because they become infected, and will incur reductions in reproductive success as a consequence. Thus, one effect of parasitism might be to slow down the evolutionary change in specific host traits when parasitized hosts still pass on their genes to the next generation. Estimating quantitatively the impact of parasite-induced alterations of host phenotype on host evolution is likely to prove difficult. For any given system, one will require data on several parameters, including the abundance of parasites, their precise effects on host phenotype, the relative reproductive success of parasitized hosts versus unparasitized hosts and the intensity of selection acting on host phenotype.

In many systems, parasites cause such large alterations in host phenotypes that they actually split the host population into discrete subunits (Fig. 1b). Some helminths alter the colouration of their hosts to such a large extent that human observers can tell precisely which host individuals are parasitized and which are not[13,14,21]. Other parasites cause a clear spatial segregation between parasitized and unparasitized hosts. For example, the trematode *Microphallus papillorobustus* causes its intermediate host, the amphipod *Gammarus insensibilis*, to move upwards in the water column, whereas unparasitized amphipods stay near the bottom[22]. One of the consequences of a marked parasite-induced segregation of host phenotypes would be to accentuate the uncoupling between host genotype and host phenotype.

On a larger scale, parasites that greatly modify host phenotypes could play a role in host speciation[23,24]. The conditions necessary for sympatric host speciation driven by parasitism are unlikely to be met in Nature, even when parasites segregate the host population into two distinct groups. For example, minimal gene flow between parasitized and unparasitized hosts would be enough to disrupt it. Parasite-mediated allopatric divergence, however, is entirely possible when different populations of the same host species differ greatly in levels of parasitism. Phenotypic traits would show different distributions among these host populations, providing different raw materials for selection. In unparasitized populations, the coupling between genotype and phenotype would be much stronger than in heavily parasitized populations. Given identical selective pressures among host populations, the result could be a more rapid evolution of certain traits in unparasitized populations than in parasitized ones. Over evolutionary time, this divergence would not be sufficient for allopatric speciation, as the evolution of pre- or post-zygotic isolation would be necessary, but it could facilitate it. Perhaps the best candidates among parasites for a role in host speciation, either allopatric or sympatric, are bacteria of the *Wolbachia* group. These widespread bacteria live in the reproductive tissues of arthropods and cause cytoplasmic incompatibility between eggs and sperm of different individuals[25]. Thus, their effect on host phenotype is not visible externally but, nonetheless, it contributes to the reproductive isolation of host strains and species.

Parasites and the host sex ratio

Parasites also affect discrete phenotypic traits that define groupings within the host population. The effect of parasites can be to change the ratio of one type of host to another. The best-documented examples involve parasites that distort the phenotypic sex ratio of host populations[24,26,27]. These include many vertically transmitted bacteria and protozoans parasitic in crustaceans that can be transmitted only from infected mother to

offspring. When present in a male offspring, they feminize their host – they convert genotypic males into fully functional phenotypic females[27,28].

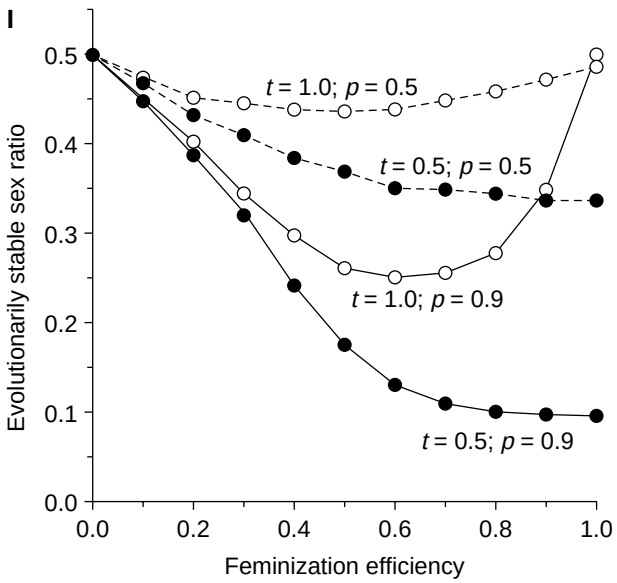
Similarly, many mermithid nematodes parasitic in insects[29,30] and rhizocephalan crustaceans parasitic in other crustaceans[31] can feminize male hosts, producing phenotypic males that resemble and behave like females. The completion of the life cycle in these parasites is entirely dependent on a female-specific behaviour. However, mermithids and rhizocephalans sterilize their hosts and, thus, both female hosts and feminized male hosts are removed from the reproductively active host population. Therefore, the effect of these parasites on the operational sex ratio of the host population is likely to be minimal compared with that of the sex ratio distorters mentioned above.

The theory of sex allocation[32] predicts that, for most organisms, selection will favour individuals that invest equally in the production of male and female offspring, with the resulting sex ratio in the population being 1:1. The net effect of parasite-induced feminization for the host population is that the phenotypic sex ratio is more female biased than the primary or genotypic sex ratio. If the rate of transmission of the sex ratio distorter from mother to offspring and its efficiency of feminization are high, the parasite can quickly achieve fixation in the host population and drive it to extinction, owing to a lack of males. Such extreme situations are unlikely, and mathematical models have been developed to explore the possible evolutionary consequences of parasite-induced biases in host sex ratio[33–35]. The models agree that selection should favour host genes that code for production of more offspring of the rarer sex, ie. males, to compensate for the effect of the parasite. Thus, the evolutionarily stable primary or genotypic sex ratio of the host population should evolve to become male biased. The magnitude of the shift away from a 1:1 primary sex ratio would be modulated by many factors (Box 1), but it is expected to be influenced greatly by the prevalence of the parasitic sex ratio distorter.

The evolutionary impact of sex ratio distorters can potentially be severe. Some possible end results include monogeny, in which uninfected hosts produce only males or the non-transmitting sex[27,34,35], and the emergence of novel mechanisms of reproduction, such as parthenogenesis, or of sex determination in the host[35,36]. Whatever the outcome, the models illustrate the often important, although subtle influence of parasites on host evolution via their alteration of host phenotype.

Box 1. Parasitic Sex Ratio Distorters and the Evolution of the Host Sex Ratio

Mathematical models can be used to predict the evolutionarily stable genotypic sex ratio in host populations affected by parasitic sex ratio distorters. Hatcher and Dunn[35] have considered a system in which a parasite transmitted only from mothers to offspring is capable of feminizing genotypic male offspring. The transmission rate of the parasite can vary, however, as well as its feminizing efficiency. Thus, only a proportion of an infected female's offspring acquire the parasite and, of these infected offspring, only a proportion of the males are feminized by the parasites. The figure (Fig. I, below) illustrates the host primary sex ratio favoured by evolution, ie. the probability of an offspring becoming a female if unaffected by parasites, as a function of the feminizing efficiency of the parasite.



Feminization efficiency is defined as the proportion of infected offspring that are successfully feminized by the parasite. Two transmission rates, $t = 0.5$ and 1.0, and two prevalences of infection among adult host females, $p = 0.5$ and 0.9, are illustrated. For most situations, a male-biased genotypic sex ratio is favoured by selection, ie. the probability of an uninfected offspring becoming a female is less than 0.5. When the transmission rate and the efficiency of feminization are both maximal (1.0), infected and uninfected hosts form two distinct subpopulations with no gene flow between them, and there is thus no selection for a compensatory primary sex ratio.

Future directions

A full understanding of the evolutionary consequences of parasite-induced phenotypic alterations requires a better knowledge of these alterations themselves. Studies of phenotypic plasticity and evolution have illustrated how a single phenotypic change, induced by a minor genetic mutation, can result secondarily in more important phenotypic changes, owing to a series of compensatory responses via a plastic shift of related traits[18]. It seems likely that the ability of infected hosts to undergo a large phenotypic alteration, such as a change of microhabitat, depends also on the capacity for some other traits to accommodate the novelty. In other words, whether an initial change is induced by a mutation in the host genotype itself, or by new genes brought by the genotype of a parasite and expressed in the host phenotype, pressure on other traits will follow. For example, if parasitic infection pushes hosts into a new microhabitat that is much hotter and drier than the normal microhabitat of uninfected hosts, and if parasites are abundant, genes for resistance to desiccation and heat could be favoured in the host population. In this context, altered phenotypes of infected hosts are probably more complex than viewed traditionally, and parasites could act as a developmental switch channelling several associated traits in particular directions. It will be interesting to determine whether host genotypes evolve to accommodate parasite-induced phenotypic alterations.

From a quantitative genetics perspective, it should be possible to quantify the contribution of parasites to the phenotypic variance in host populations, and to model its long-term effects on the evolution of host phenotypes. The parasite component of host phenotypic

variance can be obtained easily by comparisons of unparasitized and experimentally parasitized hosts maintained under identical conditions; such data should be available but have not yet been used for this purpose. Natural systems can also prove useful for studies of the evolutionary influence of parasite-induced changes in host phenotype. In particular, fragmented host populations in which the abundance of parasites varies will prove ideal systems to investigate these phenomena.

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