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Exploiting host compensatory responses: the 'must' of manipulation?

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Parasite-induced alterations of the host phenotype have been reported in many systems. These changes are traditionally categorized into three kinds of phenomena: secondary outcomes of infection with no adaptive value, host adaptations that reduce the detrimental consequences of infection and parasitic adaptations that facilitate transmission. However, this categorization is a simple view, and host modifications should be considered as co-evolved traits, rather than a total takeover. Here, we present a novel scenario of manipulation, which has considerable potential to resolve issues that are specific to the evolution of behavioural alterations induced by parasites. It is proposed that certain parasites affect fitness-related traits in their hosts to trigger host compensatory responses because these responses can meet the transmission objectives of parasites.

Host changes induced by parasites: total takeover or compromise?

Parasite-induced alterations of host behaviour have been reported in a wide range of protozoan and metazoan parasites [1,2]. Host changes that follow infection are traditionally categorized into three kinds of mutually exclusive phenomena. One hypothesis states that they are secondary outcomes of resource exploitation by the parasite and have no adaptive value for the parasite or the host. A second hypothesis states that they could be host adaptations that aim to reduce or compensate for the detrimental fitness consequences of infection. Finally, the 'manipulation hypothesis' states that host behavioural changes that follow infection constitute parasitic adaptations that facilitate transmission. The evolutionary capacity of parasites is often greater than that of the host. For this reason, natural selection often favours parasite transmission traits. There are many examples of host behavioural changes that indeed increase the chances of infective stages reaching their next host or being released in a suitable habitat.

With few exceptions, parasitic manipulation dramatically reduces host fitness. Therefore, the 'manipulation hypothesis' *sensu stricto* is commonly seen as a 'zero-sum game' (i.e. the gain or loss of one antagonist is exactly balanced by the loss or gain of the other) with clear winners (parasites) and losers (hosts) [3]. However, this need not necessarily be so. Host behavioural modifications should be considered as co-evolved traits (i.e. a phenotype shared by the host and its parasite) rather than a total parasite takeover [3,4]. From an evolutionary viewpoint, behavioural changes in infected hosts, even when they result in clear fitness benefits for the parasite, are not necessarily an illustration of the extended phenotype of the parasite alone (i.e. parasite genes expressed in host phenotypes) [5]. They are also direct products of natural selection acting on the host genome. Presently, very few studies of manipulative changes have explored the degree to which parasite-manipulated behaviours could be a compromise between host and parasite strategies. The most extreme illustration of the interactive nature of the relationship between parasites and hosts in determining host behaviour is the 'mafia-like' strategy of manipulation [6,7] in which parasites select for collaborative behaviour in their hosts by imposing extra fitness costs (e.g. fecundity reduction) in the absence of compliance. This hypothesis, which was proposed initially to explain why certain bird species accept cuckoo eggs in their nests rather than risk complete clutch destruction [7,8], has been recently discussed in a broader parasitological context [9]. Although conceptually appealing, the 'mafia-like' hypothesis faces criticism regarding how the strategy originates and is maintained in natural populations [9]. Apart from two studies on avian brood parasites [7,8], no experimental evidence currently supports this hypothesis, even after specific tests in other systems [10]. Here, a novel scenario of interactive manipulation, which has the potential to resolve issues that are specific to the evolution of behavioural alterations induced by parasites, is presented.

Compensatory responses in the living world

The genotype of an individual determines its developmental limits, but the resulting phenotype is also shaped by numerous environmental influences [11]. In response to adverse environmental conditions, many species have evolved phenotypically plastic life-history traits that are altered in a state-dependent manner to alleviate fitness costs incurred during life [12]. These compensatory

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responses can be short-term alterations in behaviour and/or life-history traits that affect either exposure to environmental conditions or their fitness consequences. For instance, plants display pronounced developmental plasticity in the face of resource limitations that enables them to balance their resource acquisition to maximize fitness [13]. Another potentially important plastic response to poor environmental conditions is a shift towards greater reproductive efforts earlier in life (i.e. fecundity compensation). For instance, cladoceran crustaceans can produce larger clutches of smaller offspring earlier in the presence of predatory fish [14].

Like other environmental factors, parasites can affect the optimal strategies of their hosts and have important roles in the evolution of compensatory responses. Hosts that are unable to resist infection by other means (e.g. immunity) are favoured by selection if they partly compensate for the parasite-induced losses by adjusting their life-history traits. This prediction is supported by several theoretical and empirical studies that show that infected hosts can adjust their reproductive effort or growth in a way that maximises fitness. For example, parasitized hosts compensate fitness costs owing to infection by using mechanisms such as an increased rate of egg laying [15,16], enhanced courtship behaviour [17,18], increased offspring number and/or size (see, for example, Refs [19,20]) and/or greater parental effort [21–23]. In other cases, hosts compensate by diminishing their reproductive effort, presumably to enhance survival, which, in return, could increase the probability of outliving the parasite [24–26]. Thus, compensation appears as an extremely widespread strategy among organisms that are exposed to adverse conditions and, clearly, parasitism can be a decisive environmental factor that triggers host compensatory responses.

Should parasites exploit host compensatory responses?

Compensatory responses that are displayed by free-living organisms often seem to be compatible with parasite objectives. Parasites can directly exploit compensatory responses that have been selected in other ecological contexts by mimicking the proximal cues that induce them. Alternatively, because of their considerable effects on host fitness, parasites might be the direct causal agents of compensatory responses displayed by the host. In these situations, natural selection should favour parasites that are able to initiate the host compensatory responses that benefit parasite transmission and host fitness. In what follows, we present examples of parasite-induced host behavioural changes that deserve consideration in the light of the scenario presented here; Box 1 provides additional examples.

Foraging activity

Predation risk

In an article unrelated to parasitism, Gotthard [27] mentioned that caterpillars that hatch late in the season accelerate their growth to pupate before winter, but the rate of predation was significantly higher on these fast-growing larvae than on the slow-growing larvae. The mechanistic link between growth compensation and enhanced predation risk is a greater exposure to predators in late-hatching individuals when foraging. An increased predation risk could potentially benefit trophically transmitted parasites because this type of parasite requires a predation event to complete their life cycle. Parasitized hosts often have increased energy requirements and forage more to compensate for the negative effect of infection. However, the subsequent increased predation risk has been traditionally viewed as a by-product of infection that is coincidentally beneficial for the parasite. This seems to be the most parsimonious explanation. Parasites live at the expense of their hosts and, consequently, they have reasons other than transmission to divert energy away from the host (e.g. growth, and maturation of gonads). In the present evolutionary context, parsimony can be viewed differently. Host exploitation by parasites can affect many fitness-related traits in hosts, such as survival, fecundity or sexual attractiveness (parasite virulence in Figure 1). These phenomena should favour the evolution of compensatory responses in the host, such as increased foraging or reproductive activity (host 1 compensatory response in Figure 1). When host compensatory responses both mitigate the costs of infection for the host and meet the transmission objectives of the parasite, natural selection should favour all genes involved in this interaction. In the parasite, selection will favour genes that are responsible for the induction of the pathological effects that are optimal

Box 1. Other examples of parasite-induced host behavioural changes

Throughout the Hawaiian Islands, corals from the genus *Porites* are susceptible to infection by the trematode *Podocotyloides stenometra* [42,43]. Infected *Porites* are characterized by the appearance of swollen pink nodules. Because these parasitized polyps are costly to the coral (i.e. they cause reduced growth), it should be adaptive for the coral to eliminate and replace them, in the same way that this is done for any polyps in which function is impaired. A radical way to physically remove an impaired polyp is to 'offer' it to predators. Interestingly, butterfly fish (the definitive host) indeed feed preferentially on parasitized polyps, and this phenomenon enables the regeneration of a healthy polyp [42]. Although the higher susceptibility of infected polyps to fish looks like a case of manipulation *sensu stricto*, it is also in accordance with the idea that the parasite relies on host compensatory responses to favour its transmission.

A change in feeding preferences is reported frequently in parasitized organisms [1]. Because this change can help to fight the infection, it is frequently interpreted as a case of self-medication. For instance, when infected with the tachinid parasitoid *Thelaira americana*, the caterpillar host *Platyprepia virginalis* changes its feeding preference from lupine to hemlock [44]. This change apparently mitigates the costs of the infection for the host because infected caterpillars feeding on hemlock survived the emergence of the parasite and even metamorphosed into sexually mature adults without a loss of fecundity [45,46]. However, this host compensatory response seems to also benefit the parasite. The average pupal mass of flies (which is a good correlate of fecundity) that emerged from caterpillars reared on hemlock was, indeed, greater than the mass of flies emerging from lupine-fed caterpillars [44].

Females of the amphipod *Corophium volutator* compensate for the negative effect of the trophically transmitted trematode *Gynaecotyla adunca* on survival by increasing their reproductive activity [18,47]. Interestingly, it is known that pairing behaviour in amphipods is associated with an increased predation rate [48,49]. This example, however, must be considered carefully because, apparently, the increased sexual activity of parasitized gammarids occurs before the trematode is infective to vertebrate predators.

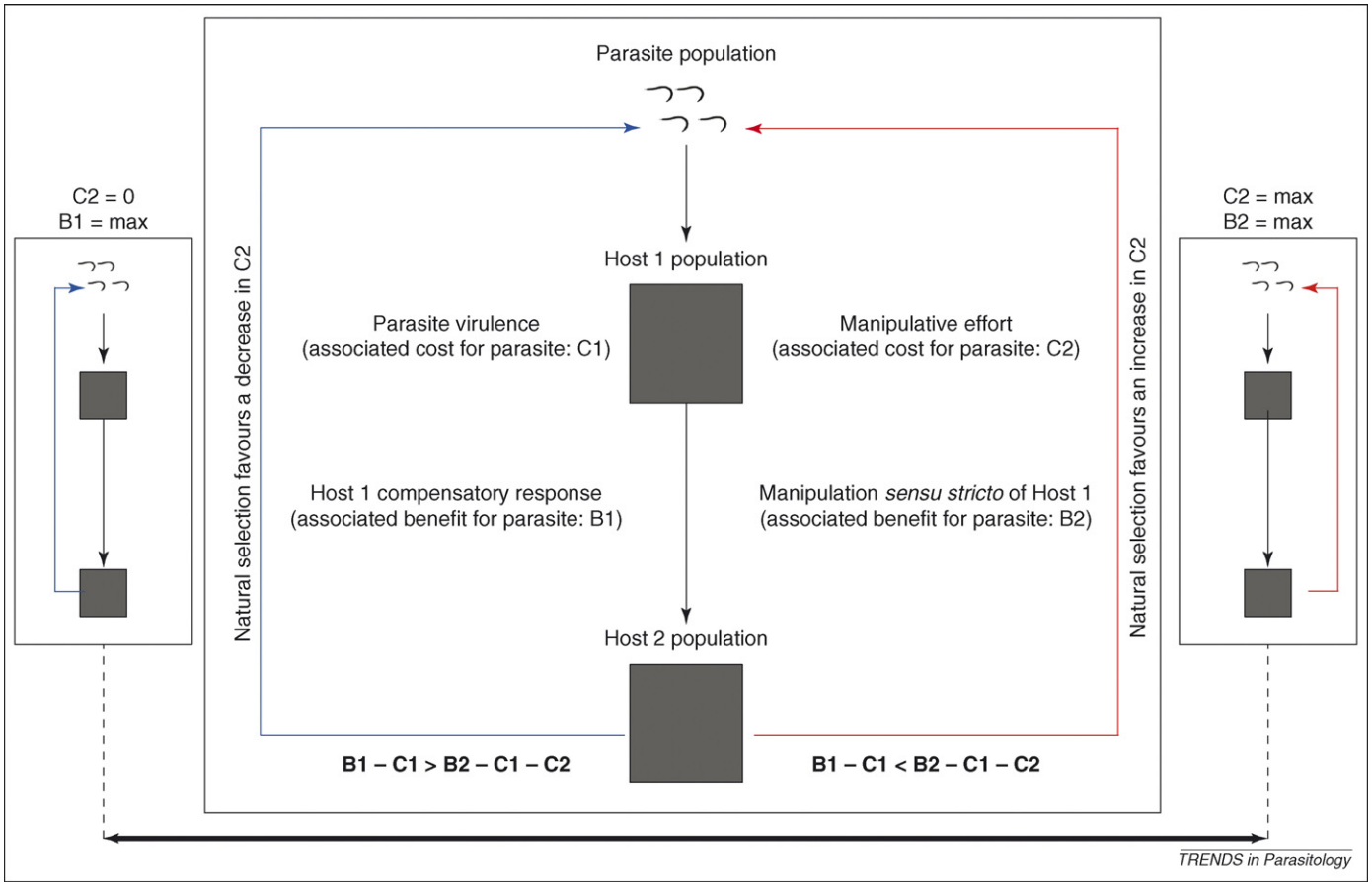

Figure 1. A schematic representation of the evolutionary dynamics of two manipulative strategies: (i) the manipulation *sensu stricto* and (ii) the exploitation of host compensatory responses. The two strategies can be represented by a continuum (which is shown by an arrow in bold) along which a parasite can both induce a host compensatory response (via its virulence, e.g. fecundity reduction) and invest energy in manipulation *sensu stricto* (i.e. a mixed strategy). Such a parasite has fitness costs associated with the manipulative effort of manipulation *sensu stricto* (C2) and with the virulence incurred to the hosts (C1), and gains transmission benefits from the *sensu stricto* manipulation (B2) and from the host compensatory responses (B1). When B1 – C1 > B2 – C1 – C2, natural selection favours a decrease in manipulative effort (i.e. a decrease in C2) and, thus, favours the strategy based on the exploitation of host compensatory response. Inversely, when B1 – C1 < B2 – C1 – C2, natural selection favours a higher investment in the manipulation *sensu stricto* strategy. At the extreme left is shown the case in which the parasite induces a host compensatory response that matches totally the objectives of the parasite. At the extreme right, no compensatory response exists in the host phenotypic repertoire; hence, making the hosts behave in a way that benefits the parasites can be achieved only by manipulation *sensu stricto*. This simplistic view has the advantage of emphasizing the fact that when the compatibility between the type of host compensatory response and the objective of the parasite is strong enough, the exploitation of host compensatory response is the best strategy of manipulation.

in the way they induce a compensatory response that enhances transmission (Figure 1: arrow in blue). However, there is a systematic tendency to consider host changes that result from energy depletion as 'non-adaptive' (see, for example, Ref. [28]). It is not an exaggeration to say that if things continue in this direction, it will soon be accepted as part of the definition of manipulation itself that host changes resulting from energy depletion cannot be adaptive for parasite transmission. To conclude that a given change is not adaptive, it would be necessary to demonstrate that it cannot be adaptive. This would undoubtedly require the reconsideration of the status of several changes that are currently considered as by-products of infection.

Biting behaviour in haematophagous insects

Haematophagous insects, when feeding on their hosts, can transmit numerous pathogens. Vector-borne parasites manipulate the feeding behaviour of their vectors in ways that render parasite transmission more probable [29,30]. An increased probing and feeding rate has often been reported in infected vectors such as tsetse flies, sandflies, fleas and mosquitoes that transmit *Trypanosoma*, *Leishmania*, plague and *Plasmodium*, respectively [30]. Where the underlying mechanism is known, increased biting usually results from mechanical blockage of mouthparts or associated apparatus [31–33] or, in the case of mosquitoes, decreased production of apyrase by salivary glands [34]. These pathologies cause difficulties during blood uptake that necessitate further probing and, therefore, more parasite transmission. In addition, parasite-induced fecundity reduction has been reported in several insect-vector–parasite associations such as mosquitoes–malaria, sandfly–*Leishmania*, filarial nematodes–mosquitoes and blackflies [35,36]. We do not argue that host compensatory responses can always explain the increased biting rate, but consider the following observation: when *Aedes ageypti* parasitized with *Plasmodium gallinaceum* are free to bite more and became fully engorged, they recover their normal fecundity [37]. Following the proposed hypothesis, the increased biting rate, at least in this system, might represent a host compensatory response to parasite-

induced fecundity reduction (however, see Ref. [38]). Increased biting rate and fecundity reduction are often reported in infected insect vectors, but the link between them remains largely unexplored. We strongly encourage further investigations on a broad variety of vector–parasite systems.

Sexual behaviour

Life-history theory predicts a shift towards greater reproduction early in life when the expectation of future reproduction decreases. Parasites with direct transmission would benefit from somehow decreasing the long-term reproductive outlook of their host because this should promote compensatory sexual activities that increase social interactions and, hence, short-term parasite transmission. For example, the sexually transmitted ectoparasite, *Chrysomelobia labidomera,* reduces the survival of its beetle host. In response, infected males compensate by developing a greater sexual drive before dying [39]. This behavioural modification clearly benefits the sexually transmitted parasite because enhanced sexual contact increases opportunities for transmission [39,40].

Gigantism

Many parasites reduce host fecundity, either partially or via full castration, by channelling energy away from host reproduction and towards their own growth [2]. A frequent consequence of fecundity reduction is host gigantism, especially in molluscs serving as first intermediate hosts of trematodes [41]. Although the adaptiveness of this phenomenon is unclear, gigantism fits well with the idea that phenotypic changes after infection can be co-evolved traits. If infection is not permanent, parasitized hosts might benefit from investing energy in growth because size and fecundity are positively correlated and, thus, fecundity compensation can theoretically occur later, after the death of the parasite. However, the first beneficiary of this strategy is the parasite because larger host size enables the parasite to produce thousands of infective larvae.

Concluding remarks

Most studies on parasitic manipulation consider that host phenotypic changes that benefit the parasites are compelling illustrations of the extended phenotype [5]. By contrast, the perspective presented here suggests that host behavioural changes after infection, even when they are beneficial for parasite transmission, can in fact be true compromises between host and parasite strategies (i.e. a shared phenotype). To our knowledge, it is novel to consider that parasites achieve transmission by triggering host compensatory responses, when these host compensatory responses fit (totally or in part) with the transmission route of the parasite. Is this strategy common? Further studies are clearly needed to answer this. For several reasons, this way of manipulating the host seems parsimonious compared with the hypothesis of manipulation *sensu stricto*, in which the parasite must exert a certain manipulative effort with putative fitness costs (Figure 1). Indeed, if among the arsenal of host compensatory responses, some are beneficial for transmission, then selection should favour the parasites that exploit these responses, not only because this meets their objectives but also because this requires no manipulative effort: the host does the work. The main task of the parasite is to induce a fitness-related cost in the host, which is something that parasites normally do. Another reason to believe that exploiting host compensatory responses is a likely scenario from an evolutionary perspective is that it also benefits the host: once infected, it is better for the host to behave in a way that alleviates the costs of infection, even when this also helps the parasite. Under these conditions, resistance is less likely to evolve than when there is no compensation from the host.

One might predict that manipulation *sensu stricto* should have evolved mainly in systems where there is nothing within the repertoire of host compensatory responses that can meet the transmission objectives of the parasite (Figure 1). The well-known example involving the small liver fluke (*Dicrocoelium dendriticum*) is a possible illustration of this situation. It is, indeed, difficult to imagine what kind of compensatory responses could make the ant climb to the tip of grass blades (Figure 1, extreme right).

Apart from the relevance of considering host compensatory responses in the context of transmission strategies, it could also shed new light on many other aspects of host–parasite relationships. For reasons mentioned previously, natural selection should favour parasites that impose specific costs to their host each time there is subsequently a compensatory response that is beneficial for them. In our opinion, these ideas might, thus, impinge on the ultimate basis of parasitic virulence and pathogenicity.

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Darwin's 200th Anniversary

Evolutionary parasitology is an intriguing part of parasitological research and there are many aspects of this topic, see for example the Opinion article 'Exploiting host compensatory responses: the 'must' of manipulation?' by LefÒvre et al. in this issue of *Trends in Parasitology* on parasitic manipulation of host compensatory responses.

As Charles Darwin is widely considered to be the 'father' of Evolution, and to commemorate the 200th anniversary of Charles Darwin's birthday (12 February, 1809), *Trends in Parasitology* will be featuring several articles with evolutionary themes in the course of 2009. This will include, among others, reviews on how parasites can become your allies, how competition contributes to evolution, the evolution of virulence, and host choice evolution by arthropod disease vectors.

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