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Evolutionary Parasitology

How can your parasites become your allies?

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Although parasitic infection is usually detrimental, it can be beneficial to the host in some situations. Parasites could help their host by providing a new function or modifying one of the host's life-history traits. We argue that the evolution towards a lasting mutualistic relationship would be more likely when parasites endow hosts with new abilities rather than alter a trait because hosts are less likely to evolve a new capability on their own than adjust their life history by microevolutionary steps. Furthermore, we underline how evolved dependence – the host's loss of ability to live alone owing to a long history of evolution in the presence of its parasites – has shaped contemporary mutualistic relationships.

Conditionally helpful parasites

Parasites are organisms that make a living by exploiting other species. Some authors have more restrictively defined them as organisms with durable and long-lasting interactions with their hosts [1]; for others, they have to feed on only one or very few hosts [2]. Unfortunately, such criteria are not universal, and exceptions exist for each of these definitions. What everybody agrees is that being parasitized is detrimental, and this criterion is used here to define parasites. In other words, parasite-free hosts have a higher fitness than their infected conspecifics. However, many studies have described cases in which notorious parasites are beneficial to their hosts (see Refs [3,4] for reviews). In these reported cases, the parasites are not always helpful because the benefits they provide are conditional and exist only in specific environments (depending on abiotic factors or biotic factors such as host status) – otherwise, they would generally be referred to as mutualists. For example, some trypanosomes have positive effects on their rodent hosts when the host food is deficient in pyridoxine (vitamin B6). In these conditions, young infected squirrels have a greater mass gain than uninfected controls. However, when the environment contains pyrodixine, uninfected squirrels perform better than infected ones [5]. Unlike mammals, trypanosomes are able to synthesize vitamin B6 [6], indicating that the parasite provides this vitamin to the host. We coined the term 'conditionally helpful parasites' to define those parasites that are beneficial to their host in specific environments but still have an overall negative impact in others.

Such conditionally helpful parasites, in addition to exemplifying the existence of a continuum of interspecific relationships between parasitism and mutualism [7,8], could have important consequences for the evolution of host–parasite systems. First, they reduce the overall cost of parasitism and, thus, the selective pressures for host resistance to infection (Box 1). Second, conditionally helpful parasites might evolve towards mainly mutualistic interactions. The probability of such an evolutionary shift, however, will depend on the mechanisms that underlie the benefits provided to the host. We thus briefly review how infection can be conditionally beneficial to the host, before exploring the evolutionary origin of and the conditions for the sustainability of the mutualistic interactions between hosts and former parasites. Furthermore, we highlight the role of another process leading to the apparent cooperation between hosts and parasites: evolved dependence, which occurs when hosts lose the ability to live without the parasites that frequently infect them.

How parasites can help their hosts

We propose to classify the mechanisms by which parasites can help their host into two broad categories. They can either provide the host with a function that it cannot perform alone or modify a host life-history trait, which consequently becomes beneficial in a particular environment. Note that throughout this article, we refer to life-history traits in a broad sense, as any trait that depends on the allocation of host resources. Other classifications might also be relevant [4], but we will focus on these two mechanisms because they could lead to different evolutionary consequences, as shown below.

The parasite provides a new function to the host

Parasitic infection can be beneficial because the parasite permits its host to do something that it was not able to do without the parasite. In the above example of rodents that benefit from trypanosome infection in the absence of pyridoxine [5], the parasite apparently provides the host with the vitamin or, at least, the ability to cope without it. Parasites can also help their host compete against conspecifics. For instance, bacteria of the genus *Cædibacter* make their hosts, the freshwater ciliates of the *Paramecium*

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Box 1. When resistance is useless

If infection is beneficial in some environments but costly in others, the selective pressure on the host for resistance to infection depends on the frequency of the particular environment. Because the overall fitness of a genotype over several generations is the geometric mean of its fitnesses in the different environments [39], resistance to infection might not be selected even if the environment in which the parasite is helpful is rare. Indeed, because geometric means are very sensitive to low values, an infrequent but great benefit of infection could counterbalance a frequent but slight cost of infection (Figure I).

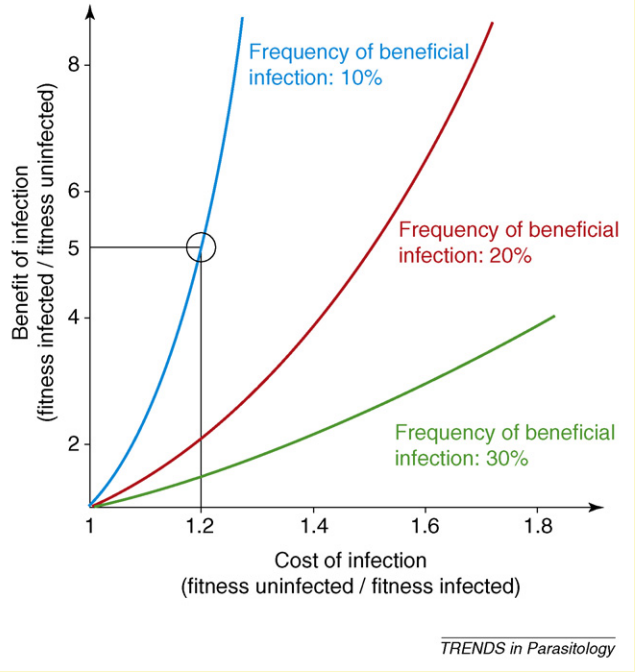

Figure I. Fitnesses of resistant and susceptible host genotypes in temporally variable environments. Fitnesses are calculated as geometric means. Fitness depends on three factors: cost of infection in environments in which infection is costly (*x* axis), benefit of infection in environments in which infection is beneficial (*y* axis) and frequency of environmental conditions in which infection is beneficial (lines). For example, when infection is only beneficial in 10% of cases (circle), the cost of infection is such that the fitness of resistant hosts is 1.2 times greater than the one of susceptible hosts, and the fivefold greater fitness of susceptible hosts when infection is beneficial could offset the frequent cost of parasitic infection. When the frequency of beneficial infection increases, the same benefits can outweigh even greater costs of infection.

genus, produce a toxic form of the bacteria, which kills uninfected neighbors [9]. Another type of new function is the ability to deal with adverse abiotic conditions. Heavy metals are toxic to most organisms, but in some fishes, infection by acanthocephalan parasites reduces the amount of lead found in host tissues: by acting as lead-diverting organs, these parasites might help their host tolerate high environmental levels of this heavy metal [10,11]. Similarly, freshwater clams (*Pisidium amnicum*) infected by digenean trematodes tolerate higher concentrations of pollutants than uninfected controls [12]. Fungal endophytes illustrate the range of new functions that parasites can provide. Endophytic fungi are found in most plants, from trees to grasses, where they live asymptomatically within plant tissues. In grasses, many have an overall negative impact on host growth and seed production [13], but others seem to provide protection to the plant against various biotic or abiotic stresses. For instance, they can produce alkaloids that protect the host from herbivores and other pathogens [14].

Infection affects a life-history trait of the host

Conditional benefit can also occur when the parasite causes a plastic change in a life-history trait of the host that happens to set the trait to a favorable level when the host is in a particular environment. For instance, when the freshwater ciliates *Paramecium caudatum* that are infected by the bacterial endoparasite *Holospora obtusa* are exposed to a quick increase in temperature from 25 °C to 35 °C, they have a greater survival rate than their uninfected conspecifics. This resistance seems driven by the increased expression of heat-shock proteins induced by infection [15]. At the time of the temperature increase, therefore, infected hosts already have large amounts of heat-shock proteins and are able to better tolerate the change. The upregulation of defense components owing to a parasite infection can also sometimes help the host to resist another parasite. Murine gammaherpesvirus 68 usually establishes lifelong latency in memory B cells, macrophages and dendritic cells after a brief lytic replication period. This chronic infection induces the sustained production of the antiviral cytokine interferon-c and systemic activation of macrophages. The upregulation of the basal innate immune system causes mice infected by the virus to be resistant to infection by the bacterial pathogen *Listeria monocytogene* [16]. Interestingly, parasites themselves can benefit from being infected by other parasites. For instance, the chestnut blight fungus, *Cryphonectria parasitica*, can be infected by double-stranded RNA virus hyperparasites that consequently reduce fungal virulence, preventing the pathogen from killing its tree host too quickly [17]. A theoretical simulation of this tripartite chestnut–fungus–virus association showed that the modification of this life-history trait of the fungus (i.e. virulence) can be beneficial when it moves the trait closer to its optimum, thus making infection by the hyperparasite beneficial to its fungal host [17].

Evolution towards mutualism: why the mechanism of conditional benefit matters

As long as the conditional benefits provided to the host remain, on average, less important than the intrinsic cost of parasitic infection, these organisms can still be considered as parasites (Box 1). But they could become real mutualists if the average fitness of parasitized hosts – across all possible environments – became higher than that of unparasitized ones. This could happen because of an environmental change, because the benefits of infection in the favorable environment increased (or the costs decreased) or because the favorable environment became more frequent (Box 1). Furthermore, the parasitized host population might even shift its ecological niche to one whereby infection is more often advantageous [18]. If so, would these new mutualistic relationships between the host and former parasites evolve towards long-lasting mutually beneficial associations?

In any case, a newly evolved mutualistic interaction will last only as long as the host advantage over other symbiont-free hosts lasts (i.e. as long as parasitized hosts are

not outcompeted by unparasitized ones). Thus, for infected hosts to maintain their competitive superiority, the advantage provided by the parasite must not be obtained easily by a population of uninfected hosts exposed to similar conditions. Otherwise, any individual from such a population migrating to an infected population relying on parasite help could easily outcompete the residents and invade the population because it would not have to pay the extra cost of bearing the symbiont.

Whether uninfected hosts can evolve a phenotype equivalent to hosts helped by parasites will depend on the mechanism underlying the parasitic help, which is of crucial importance for the fate of emerging mutualistic associations. When the help is based on a modification of host life-history traits, it is likely that such a change could evolve in the absence of the parasite, provided that the hosts were under selection for this trait. However, new abilities are much less likely to appear in the absence of the parasite, making the high fitness of infected hosts in beneficial environments more difficult for uninfected hosts to obtain. Indeed, the microevolution of quantitative traits is more likely than the appearance of new abilities because they rely on different processes. Most mutations of the appropriate genes would affect the value of a life-history trait, creating the material for natural selection to operate (and, indeed, genetic variability is found for virtually any quantitative trait studied [19]), whereas the appearance of new functions would generally necessitate several unlikely mutations or genetic rearrangements [20,21]. Using the metaphor of the adaptive landscape – in which all possible phenotypes are on a multidimensional map, and hills and valleys represent zones of high and low fitness, respectively – a change in a host life-history trait driven by the parasite would be equivalent to helping the host to climb a high fitness hill present in the beneficial environment. Uninfected hosts, however, would already be at the foot of the same hill, and thus could climb it on their own by microevolutionary steps given enough time. A new parasite-influenced function would be equivalent to bringing the host to the top of a new, distant fitness peak and leaving the uninfected population far behind, evolutionarily speaking (Figure 1).

In short, your parasites will become your allies in the long run only if they provide you with something that you do not have the evolutionary potential to obtain on your own. This probably explains why contemporary mutualistic symbionts are sometimes classified as providers of resources and defenders against enemies [22] but never as 'modifiers' of traits.

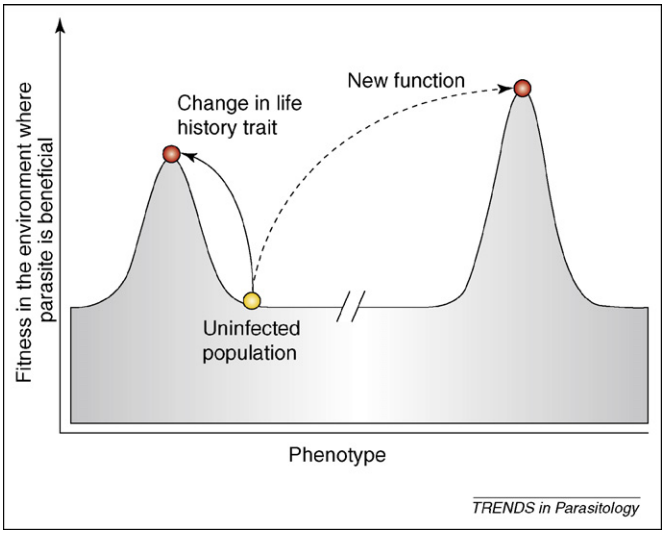

Figure 1. Hypothetical fitness landscape of a host species. Parasitic infection (red dots) modifies the phenotype of the host, which then becomes advantageous in some environments. New functions provided by parasites might enable hosts to access fitness peaks unlikely to be reached by mutation (dotted arrow), whereas a favorable parasite-induced change in a life-history trait (solid arrow) would bring the host to a fitness peak that was already accessible by microevolutionary steps.

Evolved dependence: apparent help from parasites

Conditional help is not the only possible origin of mutualism between hosts and their former parasites. In mutualistic associations, as they are usually defined, both partners experience a greater fitness together than on their own. Yet, this definition also applies to couples of species that have lost the ability to live apart, even when they would have done much better alone if they had evolved separately. This can happen when hosts have faced frequent infection in the past and have evolved a dependence on their parasite – for example, because they lost a function that the parasite performed redundantly or even because they have never evolved in an environment deprived of parasites and adaptations that are useful in the presence of the parasites become deleterious in their absence [23]. This process does not require the parasite to have ever had any prior beneficial effect on the host and, thus, constitutes a separate case from conditionally helpful parasites. An extreme example of evolved dependence to a parasite has been demonstrated in wasps of the genus *Asobara* that are infected by the endosymbiontic bacteria *Wolbachia*. Indeed, *Asobara tabida* wasps artificially deprived of their parasite by antibiotics are simply unable to produce their own eggs [24]. Pannebaker and colleagues [25] further demonstrated that the parasite controls the cell-death regulation of the wasp for its own protection, which results in a disruption of oocyte development when *Wolbachia* is absent. Evolved dependence to helminth intestinal parasites could also be the origin of some human immune diseases. The long history between humans and their intestinal parasites that constantly evolved ways to evade and/or attenuate the immune system might well have predisposed us to immune malfunctions when parasites are removed suddenly, as is the case in developed countries with high hygiene standards [26,27]. For some of these diseases, such as Crohn's disease, the ingestion of benign *Trichuris* worm eggs even seems to be a promising method of treatment [28].

Although the above-cited parasites could be considered allies, the benefits of infection do not originate in the services they provide. Instead, they are due to the long coevolutionary history of the host and the parasites. To account for these cases, De Mazancourt and colleagues [29] have suggested distinguishing between 'ultimate' mutualisms, in which both partners gain a real advantage from the association, and 'proximal' mutualism owing to evolved dependence, in which both partners are better living together only because they have lost the ability to live separately (e.g. *A. tabida* wasps and their *Wolbachia*

Box 2. Future directions and tests for long-lasting mutualisms

Evolutionary parasitology research usually has been focused on parasites with large deleterious impacts on their hosts. However, parasites with small negative effects are more likely to perform conditional help, and possibly new mutualisms, because even small benefits could outweigh their cost. Using conditionally helpful parasites to investigate the evolution towards mutualism or parasitism would, thus, require a focus on mild parasites, such as benign intestinal worms, mild plant pathogens [23] or insect endosymbionts [40].

Distinguishing between mutualism and evolved dependence might not be easy because we, obviously, do not have access to the ecology of the host's ancestor. In particular, when a host depends on its symbiont or parasite for the processing of some function, it might be difficult to assess whether this particular function has been brought by the symbiont or whether the host lost it because of evolved dependence. The use of host phylogenies could be one way to differentiate these two situations [41]. If the host lost this trait because it was redundantly performed by both itself and its symbiont, some of the host-related taxa should have kept the ability to perform it alone. The presence or absence of the ability to perform the function in the different taxa of the host clade should, thus, inform whether this function is linked to the symbiont (i.e. is a new function) or is an ancestral trait in this clade (i.e. is a case of evolved dependence).

Experimental evolution would be an ideal test for our prediction that parasites providing a beneficial change in life-history traits would not form sustainable mutualistic associations. *Holospora obtusa* parasites, which increase the expression of heat-shock proteins in their *Paramecia* hosts [15], could be used for such a test. We would expect that a parasite-infected population maintained in the favorable environment (a *Holospora*-infected population of *Paramecia* kept at high temperature) would initially have a fitness advantage (such as a higher growth rate) over uninfected populations in the same conditions. This advantage should then decrease over time because uninfected *Paramecia* populations would be selected towards better performances at high temperature until they eventually overtook the *Holospora*-infected ones. The sustainability of associations whereby parasites provide new functions could be similarly tested by experimental evolution on adequate microorganism systems. In this case, the infected populations would be expected to always keep the highest fitness. Bacterial plasmids, which can be considered replication parasites that sometimes also encode useful proteins, might be good candidates for these experiments [42].

parasites). Thus, the cases of evolved dependence should not be considered as equivalent to the ultimate mutualisms arising from conditionally helpful parasites discussed above. The processes leading to evolved dependence, however, could be intertwined with other processes involved in the evolution of conditionally helpful parasites. Indeed, it would promote the fidelity of the interspecific association and, in turn, favor mutualism maintenance [30].

Concluding remarks

Parasites and mutualists form a continuum of interactions between purely costly and purely beneficial effects on the host, yet they traditionally have been studied separately. Many studies have focused on the evolution of parasite virulence [31,32], whereas others have investigated the maintenance of mutualism [30,33–35]. However, these are two approaches to the same question: what drives the evolution of interacting species towards antagonism or cooperation? Merging the methodologies and knowledge of parasitism and mutualism studies can be very fruitful [36,37] and could become a major development in these fields.

Because they are close to the intersection between these two opposite relationships, conditionally helpful parasites would be useful models for a better understanding of the role of parasitism in the emergence of mutualism. More information on the frequency and generality of conditional help by parasites would, thus, be needed greatly. In addition, attention should be given to finding the mechanisms underlying the service provided by the parasite because they shed light on the evolutionary future of the relationship (Box 2).

In conclusion, conditional outcomes of parasite infections emphasize the crucial role of environmental variability, through space and time, in parasitic and mutualistic associations and illustrate the geographic mosaic concept (i.e. that the coevolution of interacting species changes with the particular characteristics of their habitat) [38]. Parasites with drastically different effects across environments could, thus, also be useful for investigating the impact of this geographic mosaic of habitats on the evolution of interspecies interactions.

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Evolutionary Parasitology and Darwin's 200th Anniversary

Because 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. Evolutionary considerations are of great importance for our understanding of parasitology and could ultimately lead to treatments.

The first of these articles is the Opinion article on how parasites can aid host fitness by Simon Fellous and Lucie Salvaudon, entitled 'How can your parasites become your allies?', in this issue of *Trends in Parasitology*. In the next issue (March 2009 issue), Derek McKay will have an Opinion article entitled 'The therapeutic helminth?' on how the immune system evolved in response to helminth infection and how the immune response caused by certain helminths could have the potential to be used to treat inflammatory and autoimmune diseases. Other upcoming topics will include how competition contributes to parasite evolution, evolution of drug and vaccine resistance, host and parasite co-evolution, and host choice evolution by arthropod disease vectors.

Trends in Microbiology will also be running a series of articles on Evolutionary Microbiology, including an Opinion article by Snyder *et al.* entitled 'Bacterial flagellar diversity and evolution: seek simplicity and distrust it?' in the January 2009 issue (volume 17, issue 1).

Be on the look out for these and other exciting articles in 2009!