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Review

Host life history responses to parasitism

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ABSTRACT – Parasites and their infections can adversely effect a host's growth, reproduction and survival. These effects are often not immediate, but increase with time since infection. A general prediction from evolutionary biology is that hosts suffering from this type of infection should preferentially allocate resources towards reproduction, even if this is at the expense of their growth and survival. This review illustrates this argument with several empirical studies showing hosts behaving in this manner. These studies indicate that one way for hosts to reduce the costs of parasitism is by altering their life history traits to bring forward their schedule of reproduction. © 2000 Éditions scientifiques et médicales Elsevier SAS

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1. Introduction

How can hosts reduce the costs of parasitism? One possibility is for hosts to try and avoid contact with parasites. A second is to have defensive mechanisms capable of preventing the establishment of an infection. A third is an immune system that can suppress or clear infections that manage to become established. An extensive literature covers the latter two means by which hosts reduce the costs of parasitism. This review deals with a less well known option — that hosts can reduce the costs of parasitism by altering their life history traits.

2. Parasitism and life history theory

An organism's life history traits are those describing its age-specific growth, reproduction and survival [1, 2]. Life history theory aims to understand how these traits interact and how such interactions influence an organism's biological fitness. For example, if an organism grows to become a large, fecund adult that survives long enough to reproduce several times, it is likely to have a high biological fitness. However, interactions among traits are physiologically constrained by the amount of resources an individual has at its disposal; as more resources are devoted to reproduction, fewer will be available to maintain growth or survival. Thus, increased allocation to one trait will be traded-off against a reduced allocation to other traits. Individuals that differ in how they allocate resources to their life history traits are likely to have a different fitness. Natural selection will favour those individuals whose pattern of resource allocation yields the highest fitness.

Although physiological constraints generate negative correlations in how individuals can allocate resources between two traits, the same two traits can be positively correlated at the population level. This is particularly so if the population is dispersed over environments differing in resource availability. As individuals encounter more resources in their environment they can simultaneously increase their investment in more than one trait, e.g., in both growth and reproduction. Thus, an individual's environment can have an influential role in the expression of its life history traits and on its fitness [3].

Parasites can be an important component of the environment that hosts encounter. Not only can parasites reduce the host's resources, but they can also differentially affect particular traits, such as the host's longevity. Thus, parasitism can act as a factor that changes the host's environment, and what was the optimal pattern of resource allocation in the absence of parasitism may not be optimal in the presence of parasitism [4].

The impact of parasitism is often not immediate but increases with time since infection or from the beginning of the host-parasite interaction. This time lag can provide an opportunity for the host to alter its life history traits before the full costs of the parasite are experienced. This

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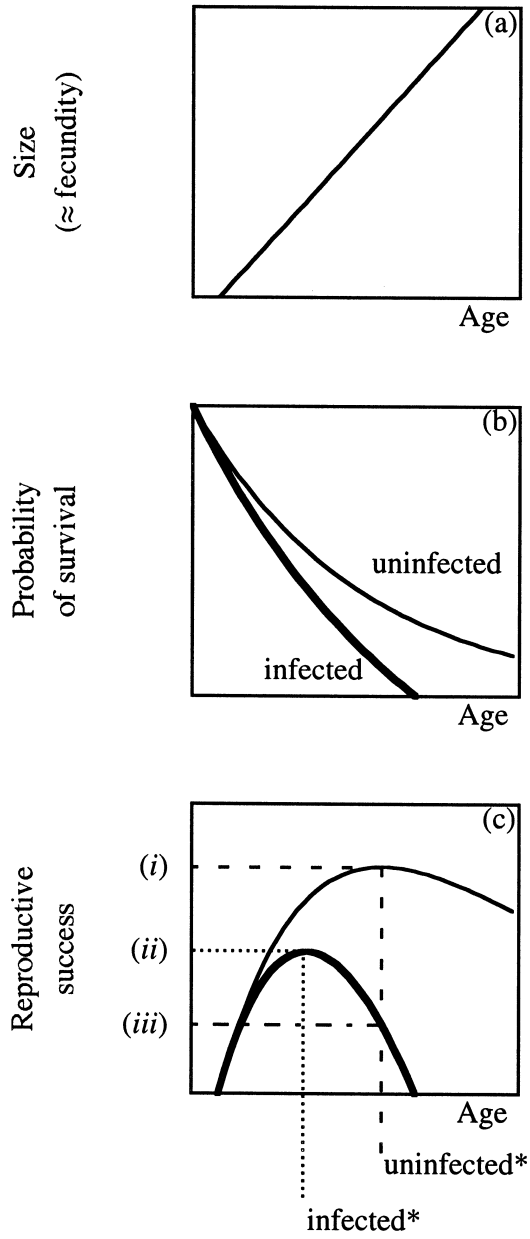

Figure 1. Imagine a host that grows up until a certain age, reproduces once, and then dies. Its size is positively correlated with its fecundity, and both increase with time spent in growth (a). The probability that uninfected hosts will survive until a particular age declines with age (b). The product of these two functions describes the host's potential reproductive success as a function of its age (c, thin line). The highest point in this curve (*i*) defines the optimal age for uninfected hosts to reproduce (uninfected*). Now imagine that the same individual is infected by a parasite that progressively reduces the host's probability of survival (b). This additional mortality changes the profile of an infected host's potential reproductive success (c, thick line). The maximum reproductive success that infected hosts can gain (*ii*) is less than that for uninfected hosts (*i*) and is optimised by reproducing at an earlier age (infected*). Note also that had infected hosts continued to reproduce at the same age as uninfected hosts, their potential reproductive success would be even lower (*iii*). Each axis is on an arbitrary scale. Modified from reference [6].

will particularly be the case for examples where the host cannot effectively stop an infection from becoming established or suppress it immunologically. Several authors have predicted that, when faced with this situation, hosts that bring forward their schedule of investment into reproduction will be favoured by natural selection [5–10]. Essentially, the effect of parasitism is to make the host's prognosis for future reproductive success much bleaker. Consequently, infected hosts should try and gain as much reproductive success as they can before the accumulating costs of their infection become prohibitive (for a graphical representation, see *figure 1*). The next section outlines some experimental studies in which hosts have responded to parasitism in this manner.

3. Empirical evidence

When freshwater snails reach reproductive maturity, they start diverting resources into egg production at the expense of further growth and longevity. In laboratory conditions, the rate of egg production by the snail *Biomphalaria glabrata* rises to a peak several weeks after maturity and then tails off to zero over the following months. This is likely to be the optimal pattern (i.e., the one favoured by evolution) for unparasitised individuals. However, if snails that are close to, or have recently reached, maturity are infected by the trematode *Schistosoma mansoni,* they show a higher rate of egg production than uninfected control snails in the first few weeks postin-

fection [11, 12]. This increased reproductive effort is only maintained for a few weeks, after which infected snails produce fewer eggs than control snails. Ultimately, infected snails have a lower lifetime reproductive output than control snails [11]. The faster decline of egg production by infected individuals can be partly attributed to the accumulating costs of *S. mansoni* as it replicates within the hosts' reproductive tissues; this process eventually castrates the host and renders it sterile.

The faster decline in later egg production by infected individuals is, however, also partly due to a cost of their early increase in reproductive effort. Evidence for this comes from snails that remain uninfected although they have been exposed to *S. mansoni*. These exposed-but-uninfected individuals show a similar pattern of egg production as the infected individuals and they produce a lower total number of eggs than control snails [11]. The response by these uninfected individuals tells us three things; (i) the threat of infection by this castrating parasite is enough to induce the host's schedule of reproduction to be brought forward, (ii) an increase in reproductive output in early adult life is traded-off against a reduced reproductive output later in life, and (iii) the control snail's allocation of resources to reproduction gives the highest fitness in the absence of infection.

The latter two points indicate that uninfected hosts suffer a cost to their overall fitness, if they trade-off an increase in early reproductive output against a reduced fecundity later in life. This situation is different for infected hosts, as their fecundity is progressively decreased by the castrating effect of *S. mansoni*. Thus, they will not experience the costs of trading-off early vs. late reproductive success. Subsequently, the alteration of life history traits towards an earlier investment into reproduction helps reduce the costs of parasitism for these infected hosts [11].

An increase in the rate of egg laying following infection has also been reported for the cricket *Acheta domesticus* after having been challenged by the bacterium *Serratia marcescens* [13]. In this case, an infection is potentially lethal, killing the host within 12 to 36 hours of inoculation into its haemocoel. The number of eggs laid by infected females in the 24-hour period following infection is higher than for control females given a saline injection. Furthermore, female crickets injected with components of the bacterial cell wall also increase the number of eggs they lay. Thus, as is the case for *B. glabrata*, a cue indicating a potential infection is enough to induce the host to increase its reproductive effort [13]. The experiment did not run for long enough to demonstrate whether an increase in egg laying behaviour is traded-off against a reduced reproductive success later in life. Such trade-offs are generally expected from life history theory, and are often observed [1, 2]. However, as female crickets first produce their eggs, and then enrich them with nutritional reserves, an increase in the rate of egg laying may be traded-off against less reserves being allocated to each egg [13]. This, in turn, may decrease the quality of the offspring and their probability of survival. Thus, a cost of increasing the rate of egg laying may be to decrease a female's potential contribution to the next generation of crickets. Such a trade-off would also help to explain why uninfected females lay eggs at a slower rate than infected females.

Evidence for host life history responses to parasitism is not limited to studies involving egg production. Males of the fruit fly *Drosophila nigrospiracula* experimentally infested with the ectoparasitic mite *Marocheles subbadius* increase their sexual activity by investing more time and energy in courtship behaviour [14]. They are also quicker to copulate with females than uninfected males and so are likely to gain reproductive success sooner. Data from a related species (*D. melanogaster*) shows a physiological cost of elevated sexual activity, in that the life span of more active males is reduced [15]. However, mites substantially reduce the longevity of their hosts. Hence, a male's survival is more adversely affected by parasitism than by an increase in its sexual activity, and less fitness will be lost by increasing investment into reproduction. Interestingly, the response shown by males is also dose-dependent: as more mites infest a male, and its longevity is further reduced, the greater the increase in sexual activity [14]. This reveals that males are scaling the decrease in their longevity due to their own activity (reproductive effort) relative to that imposed by the environment (the mites).

For many species, the investment into reproduction does not stop at the production of offspring but also includes a period rearing them. The effort expended by parents is also a life history trait that has been shown to alter in response to parasitism. For example, if the nest of a great tit (*Parus major*) is experimentally infested with the blood-sucking hen flea *Ceratophyllus gallinae*, the male responds by increasing the rate at which he provisions food to the chicks [16]. This increased parental effort helps the chicks offset the costs of blood lost to the fleas. In doing so, males increase the chick's chances of fledging and going on to reproduce. The increase in male provisioning behaviour is associated with its own cost: it is positively correlated with the probability of contracting malaria and perishing during the following winter [17]. This pattern could arise because increased foraging behaviour brings the males into greater contact with mosquitoes. Alternatively, it could demonstrate that resources invested into parental care are traded-off against those directed towards the male's immunocompetence. Either way, the male's behaviour indicates there is a fitness dividend to increasing investment into the investment already made (albeit at the risk of becoming ill) rather than abandoning their current brood and risking the chances of them not successfully reproducing in the future. Thus, this represents a trade-off between current and future reproductive success, and male great tits responded to parasitism by hen fleas with an increase in their current reproductive effort. Similar responses to ectoparasites have also been reported from the blue tit *P. caeruleus* [18–20].

In the examples outlined above, the hosts had already reached reproductive maturity by the time they were challenged by parasitism. Consequently, these studies could not investigate the effect of parasitism on a host's age at maturity; this is a trait directly related to an individual's schedule of reproduction. For many organisms, an individual's age at maturity also defines the point at which it stops growing and, hence, the size it will be as an adult.

This is an important trait because adult size is frequently positively correlated with an individual's survival and its fecundity, and, therefore, its fitness. One example of parasitism inducing a response in a host's age at maturity is that females of the mosquito *Culex pipiens* bring forward their age at pupation if they have been infected by the microsporidian parasite *Vavraia culicis* [21]. The costs of an infection increase with time as *V. culicis* proliferates and produces spores within host cells. The damage caused to host tissues as spores accumulate is correlated with a reduction in the reproductive success of female mosquitoes [22]. By shortening their larval period and pupating earlier, infected females tend to emerge as smaller, lighter adults [22]. This indicates that they were trading-off their potential adult size, and therefore fecundity, for an earlier age at pupation. This is a further example of a host altering its life history traits to bring forward its schedule of reproduction in response to parasitism.

4. Host responses in an evolutionary context

The examples outlined above described how individual hosts alter their life history traits to reduce the costs of being parasitised. In each case, the infected individuals have a lower fitness than if they had not been infected, but a higher fitness than if they had not altered their life history traits. Consequently, the ability to facultatively alter investment among life history traits in response to parasitism is likely to be favoured by natural selection. However, the scope for hosts to alter their life history traits may be limited, e.g., the benefits of increased parental provisioning can be constrained by low environmental food availability [23], or a high intensity of parasitism [24]. Equally, there might not be enough time for hosts to change resource allocation during the course of infection. In such cases, and if successive generations of a host are at a high risk of infection, there will be selection pressure for a different response to reduce the costs of parasitism. Such a response could be the evolution of defensive mechanisms to block infection, or alternatively, the life history traits of hosts could evolve towards earlier schedules of reproduction. In other words, if the risk of being infected is sufficiently high, one could expect that all individuals show early reproduction, irrespective of infection status, rather than just those individuals that are infected.

The latter scenario has been proposed to explain why populations of the snail *Potamopyrgus antipodarum* tend to reproduce at a smaller size (indicating at an earlier age) in populations regularly exposed to a high risk of being infected by castrating trematode parasites [25]. This observed correlation is suggestive that parasites can influence evolutionary change in their hosts' life history traits. Stronger evidence for this capacity comes from a study of the marine snail *Cerithidea californica*. As for *P. antipodarum*, reproducing snails in populations of *C. californica* exposed to a high risk of infection by castrating parasites tend to be smaller than snails from populations less exposed to infection [26]. However, in this study, immature snails were reciprocally transplanted between populations regularly experiencing a high or low risk of infection. Individuals originating from 'high-risk' populations continued to reproduce at a relatively small size, despite being in a 'low-risk' environment; the reverse was true for 'low-risk' snails transplanted to a 'high-risk' environment [26]. These experimental results support the hypothesis that the life history traits of the different host populations are different at the genetic level and that the populations have evolved differently in response to the risk of being infected by a castrating parasite, rather than because of some unknown environmental parameter.

The ability of parasitism to drive evolutionary change in host life history traits is potentially relevant to the application of parasites in biological control programs. For example, the microsporidian parasite *Edhazardia aedis* is being considered as a potential biological control agent against the yellow fever mosquito *Aedes aegypti* [27]. Laboratory experiments suggest this parasite can have a severe impact on a female mosquito's reproductive success [28], but that this effect is lesser for females with early ages at pupation [29, 30]. Mosquitoes have no known immune response capable of suppressing the replication of these intracellular parasites. Consequently, one possible outcome of repeatedly exposing generations of this host to this parasite is that the host will evolve towards earlier ages at pupation [31]. An experiment demonstrated that *Ae. aegypti* has the potential to alter its life history traits in response to a few generations of selection for earlier or later ages at pupation [32]. Furthermore, when challenged by infection with *E. aedis*, the 'fast' selected lines suffer a lower cost to their fitness than 'slow' selected lines [32]. Thus, if the application of *E. aedis* does have an impact on populations of *Ae. aegypti,* it is possible that the host will respond by altering its life history traits towards earlier ages at maturity. How other life history traits interact with such a response could influence the success of a control program. For example, earlier ages at pupation are likely to be correlated with the emergence of smaller adult mosquitoes. Small female mosquitoes generally require protein from more than one blood meal to mature a clutch of eggs [33]; so adult size influences a female's biting rate and her probability of becoming infected. However, an increased biting rate is traded-off against the risk of mortality while blood-feeding [33]; this influences a female's longevity and her ability to transmit infection. Whether the effect of parasitism on these trade-offs would lead to an overall increase or decrease in the capacity of this host to vector disease is unknown, but worth testing before the launch of a control program.

A similar rationale can be applied to other instances where attempts to control pests of a medical or economic importance involve parasites that require time to develop and have an impact on host condition, e.g., nematodes and entomopathogenic fungi.

5. Examples of host life history traits not responding to parasitism

Some insights into the conditions where a change in host life history traits will be observed can be taken from

studies where hosts have not altered their traits in response to parasitism. For example, the increase in egg laying by *B. glabrata* [11, 12], depends on the age at which the host is infected by *S. mansoni* [34]; when infected as young juveniles, the host is already castrated by the time it reaches reproductive maturity, and so cannot alter its egg laying behaviour. This, and other molluscan studies [35, 36], indicate that the relative development of host and parasite at the time of infection can be important, as can the subsequent interaction of their growth dynamics. Furthermore, the manner in which different species of trematode castrate their host may also influence the host's ability to alter its reproductive output [37].

Male fruit flies illustrated that individuals can grade their response to parasitism in relation to the threat to their longevity and reproductive future [14]. Other studies have noted that males and females have responded differentially to the same parasite treatment: only male great tits were reported to increase the provisioning of chicks in response to ectoparasites [16], and only female *C. pipiens* larvae pupated earlier when infected by *V. culicis* [21]. In both cases, the authors argued that the effect of parasitism was more severe on the potential reproductive success of the host sex that responded to attack.

Whether hosts can alter different life history trade-offs in response to different types of parasite remains unclear. The increase in egg laying shown by crickets to a bacterial challenge does not occur in response to oviposition by the parasitoid fly *Ormia ochracea* [13]; this is despite the development of the fly's larvae being invariably fatal to its host. This suggests that crickets can alter their reproductive behaviour in response to some, but not all, forms of parasitic attack. Identifying the factors determining who wins the battle between host and parasite for control of the host's resources are likely to be informative in this respect [38].

6. Summary

The aim of this review was to introduce and present the hypothesis that hosts can, and do, reduce the costs of parasitism by altering their life history traits. In particular, empirical evidence was chosen to illustrate the advantages to hosts of bringing forward their schedule of reproduction in response to parasites that do not have an immediate impact on their host's fitness. These responses included; maturing earlier, raising sexual activity, increasing investment into early reproductive output, and expending more effort on current offspring. Further evidence was cited to support the idea that host life history traits may evolve in response to the type of selection pressures imposed by this type of parasite. These studies suggest that a host's investment in its life history traits can also constitute a form of response to parasitism, alongside those that avoid, block or suppress parasitic attack.

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