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Parasitism as a constraint on the rate of life-history evolution

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Abstract

There are a number of ways in which a host can respond in evolutionary time to reductions in survival and reproduction due to a virulent parasite. These include evolving physiological morphological, or behavioural mechanisms of resistance to infection (or to proliferation, once infection has occurred). But a more unexpected tactic is also possible. This is for hosts to reproduce (slightly) sooner when in the presence of a virulent parasite as compared to when the parasite is less virulent or absent. As such, hosts which reproduce younger may be at a selective advantage, since they can both evade parasitism in time and, even when parasitised, can reduce the likely impact of the parasite on survival and reproductive success. We employ a simple mathematical model to propose that parasites and pathogens can act as important agents in the evolution of the timing of reproduction and associated life-history characters (e.g. body size). Once established in a semelparous host population, evolutionary increases in parasite virulence should result in the evolution of shorter lived hosts; whereas the evolution of less virulent forms of the parasite should be accompanied by the evolution of longer lived hosts. We argue that in the presence of a sufficiently virulent parasite the evolution of longer pre-reproductive life-spans should require the previous or concomitant evolution of morphological, behavioural or physiological resistance to parasitic infection and proliferation.

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Introduction

The evolution of life-history characteristics is generally considered to be driven by age-specific mortality schedules (for review, see Promislow and Harvey 1990). These mortalities can result from environmental variables such as local climatic conditions, competition for limiting resources, or predation from higher trophic levels. The role of parasites in the life-history of their hosts remains largely unexplored (Minchella 1985).

In this paper, we explore how parasites may be of considerable importance in shaping the life-history evolution of their hosts and, in particular, in limiting the attainment of longer pre-reproductive life-spans (hereafter abbreviated PRLS). Such limits may come about because parasites may impose both age-independent reductions in survival throughout pre-reproductive development, and age-specific mortalities resulting from the accumulation of parasites within host individuals as they age. In the absence of a sufficient immune response, the accumulation of parasites with age may result from (1) longer exposure time to a (constant) population of infectious stages, (2) direct multiplication of the parasite within the host (for infections by microparasites, such as viruses), and (3) augmentation of the environmental parasite load between birth and reproduction (as in the case of viral diseases of discrete generation invertebrate hosts). Minchella (1985) suggested that parasite induced changes in life-history variables (e.g. fecundity, gigantism) may be a more cost-effective means of conserving fitness than the evolution of constitutive resistance to parasitic infection.

Specifically, we develop a mathematical model to consider how the growth pattern of a single parasite population influences the reproductive value of a semelparous host. We show that as long as the parasite has a negative effect on host survival as the host nears reproductive maturity, then there is the potential for the evolution of shorter times to reproduction, as compared with parasite-free populations. Under certain conditions, harbouring the parasite can result in the evolution of longer times to reproduction. We suggest that the coevolution of host-parasite associations should be reflected by a negative correlation between parasite severity (i.e. a combination of virulence and pathogenicity) and time to reproduction. We conclude that the evolution of longer PRLS should require the previous or concomitant evolution of sufficient defences against parasites, and that a slowing or reduction in maturation time constitutes a form of resistance to parasitism.

The model

We consider a simplified caricature of a semelparous host, in which there is a development period from birth (at time $t = 0$) to potential reproductive maturity ($t = t_o$), and a single point in time T beyond t_0 at which reproduction occurs.

During their pre-reproductive lives ($t < T$), hosts experience mortalities other than parasitism at a constant rate μ and mortalities due to a population of parasites (numbering $P\{t\}$) at a rate $vP\{t\}$. Instantaneous decreases in the per capita

probability of survival of the host, $Q\{t\}$, are given by the differential equation

$$\frac{dQ}{dt} = -\mu Q - vP\{t\}Q, \quad Q\{0\} = 1, \quad P\{t < t_p\} = 0, \quad P\{t_p\} = P_0, \quad (1)$$

where the negative effects of the parasite on host survival begin at time t_p (soon after the time of infection).

The probability of survival between birth and reproduction, is found by integrating equation (1) over the period $t = 0,T$, giving

$$Q\{T\} = \exp\left(-\mu T - v\int_{t_p}^{T} P\{t\}dt\right), \quad t_p \leq T. \quad (2)$$

At any single time $T > t_0$, each surviving host produces $\eta\{T, P\{T\}\}$ offspring. In general, η should be an increasing function of time (until senescence occurs), and a decreasing function of parasite number. The quantity representing the average number of future newborn offspring produced per host, or the reproductive value, is

$$\Phi = \eta\{T, P\{T\}\}Q\{T\}. \quad (3)$$

The host population can only persist if its reproductive value (evaluated from birth to reproduction) is greater than or equal to unity. Over evolutionary time one or more of the parameters should change so as to increase Φ.

If the age-structure and numbers of the host population are stable, and the per capita effect of the parasite on the host remains constant from generation to generation (e.g. v is constant), then we can examine how T should evolve so as to maximise Φ. The optimal time of reproduction, or T_{opt}, can be found by differentiating (3) with respect to time, and searching for the critical point(s) resulting in a maximum for Φ. The optimum, if it exists, is found from the roots of the equation

$$\frac{1}{\eta\{T, P\{T\}\}}\frac{\partial\eta\{T, P\{T\}\}}{\partial T} - \mu - vP\{T\} = 0. \quad (4)$$

A necessary (but not sufficient) condition for the existence of at least a single finite maximum is

$$\frac{\partial\eta\{T, P\{T\}\}}{\partial T} > 0, \quad (5)$$

or, as would be expected for semelparous hosts, if the per capita number of offspring increases with development time.

If equation (4) has at least a single positive real root

$$\frac{\partial Q}{\partial T} > \frac{\partial\eta\{T, P\{T\}\}}{\partial T} \quad \text{for all } T, \quad (6)$$

then, in accord with general life-history theory (e.g. Charlesworth 1980), it can be shown that parasite-induced decreases in survival and birth of the host will result in the evolution of smaller values of T. In other words, increases in parasite virulence ($vP\{T\}$) will tend to select for shorter PRLS. Evolution of the parasite towards avirulence should be accompanied by the coevolution of longer PRLS.

An example

We present a hypothetical example of a host-parasite association to illustrate how the time at which a host reproduces could theoretically evolve in response to changes in parasite virulence.

Suppose that as the host nears reproductive maturity the parasite population changes as

$$\frac{dP}{dt} = rP, \quad P\{t < t_p\} = 0, \quad P\{t_p\} = P_0\,, \tag{7}$$

so that at any time t ($t_p \leqslant t \leqslant T$) the parasite population is given by

$$P\{t\} = P_0 \exp r(t - t_p)\,. \tag{8}$$

If $r > 0$, $r = 0$, or $r < 0$, then the parasite population is growing, remaining stationary or shrinking with time, respectively.

Exponential growth or decay, though an over-simplification of the dynamics of parasite populations, may be a reasonable first approximation for the intra-generation growth of some microparasitic infections of invertebrates (Anderson and May 1981; Hochberg 1991). An example of a microparasite exhibiting exponential growth within host individuals comes from an investigation of the spread of a nuclear polyhedrosis virus through larvae of *Trichoplusia ni* (van Beek et al. 1990). Their data show that the spread of the virus through its host is characterised by a ca 12 hour lag phase, followed by a ca 36 hour exponential growth phase, and finally the death of the host from the disease. Cases in which $r \leqslant 0$ could arise, for instance, if the host has evolved some form of age-related resistance to infection and proliferation of the parasite.

Further, let us assume that the number of offspring produced per surviving host is given by

$$\eta\{T, P\{T\}\} = \frac{\eta_0(T - t_0)}{1 + \beta P\{T\}}, \quad t_0 < T\,, \tag{9}$$

where η_0 gauges the importance of development time beyond the reproductive threshold t_0, and $1 + \beta P\{T\}$ reflects the negative effect of the parasite on the average number of offspring produced by those hosts which survive to reproduce.

The reproductive value can be found by substituting equations (8) and (9) into (3) to give,

$$\Phi = \frac{\eta_0(T - t_0)}{1 + \beta P\{T\}} \exp(-\mu T - v(P\{T\} - P_0)/r) \quad \text{if } r \neq 0 \tag{10a}$$

$$\Phi = \frac{\eta_0(T - t_0)}{1 + \beta P\{T\}} \exp(-\mu T - vP_0(T - t_p)) \quad \text{if } r = 0 \tag{10b}$$

Further, substituting (8) and (9) into equation (4), and differentiating with respect to time results in

$$\frac{1}{(T_{opt} - t_0)} - \frac{r}{1 + (\beta P_0 \exp r(T_{opt} - t_p))^{-1}} - \mu - vP_0 \exp r(T_{opt} - t_p) = 0\,, \tag{11}$$

from which the optimal time of reproduction, T_{opt}, can be found numerically.

For the special case of $\beta = 0$ (i.e. the parasite has no effect on the reproductive output of surviving hosts), the relationship between parasite virulence (reflected in part by r) and optimum reproduction time is

$$r = \frac{\ln[(T_{opt} - t_0)^{-1} - \mu] - \ln[vP_0]}{T_{opt} - t_p}\,. \tag{12}$$

In other words, (assuming all other parameters remain constant) there is a negative relationship between virulence and optimum reproduction time.

Figure 1 illustrates how host survival and potential reproduction typically combine to determine the optimal time of reproduction in the face of a growing parasite population (i.e. $r > 0$). For the example shown in Fig. 1a, in the absence of the parasite, the reproductive value has a single maximum corresponding to an optimal pre-reproductive period of $T_{opt} = 70$ and an optimal reproductive value of $\Phi_{opt} = 12.5$. In the presence of the parasite, both pre-reproductive survival and the reproductive output of survivors are always reduced (Figs 1b, c). In the example shown in figure 1b the parasite is introduced 10 time units prior to potential reproductive maturity, the resulting optimal pre-reproductive period is reduced to $T_{opt} = 33$, and the reproductive value from Φ_{opt} 12.5 to $\Phi_{opt} = 4$. As expected, when the parasite is introduced later in the host's life (at the point of reproductive maturity), the reproductive value is greater ($\Phi = 6$) and the optimal reproductive time later ($T_{opt} = 38$) (Fig. 1c).

As predicted from equations (11) and (12) increasing the virulence of the parasite when the effect on reproduction is small always results in shorter times to optimal reproduction (Fig. 2). Interestingly, as the growth rate of the parasite population increases, there is a progressive decrease in the window during which the optimal reproductive point can occur (since the host can only persist if $\Phi \geqslant 1$, Fig. 2). This means that even if the evolution of the reproductive value of the host does not attain its optimum, Φ_{opt}, we would expect that more virulent parasites should limit the range of T which will result in the persistence of the host population.

The findings presented in Figs 1 and 2 assume that there are no genetic constraints involved in the evolution of PRLS. If we hypothesize that the propen-

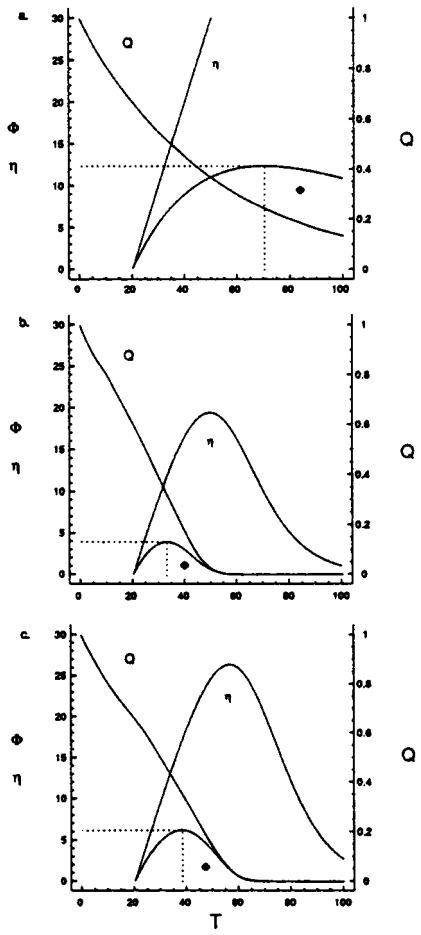

Fig. 1. Effect of time of reproduction on the survival (Q), reproductive output of survivors (η), and final reproductive value (Φ). The dotted line shows the optimal time of reproduction and corresponding reproductive value. a. No parasite present; b. Parasite is introduced at $t = 10$; c. Parasite is introduced at $t = 20$. Other parameters: $r = 0.1$, $\eta_0 = 1$, $P_0 = 1$, $\beta = 0.01$, $\mu = 0.02$, $\nu = 0.005$, and $t_0 = 20$.

sity of evolution of PRLS is directly related to the fitness gain per unit development time evolved, then we can explore how the model parameters affect the likelihood of the evolution of the PRLS (under the assumption that the parasite has been introduced for the first time into a host population).

As would intuitively be expected, we find that the selection pressure exerted by the parasite (measured in units of rate of increase of a mutant host per unit development time evolved) increases with parasite virulence (Fig. 3a–e). Differing values of other parameters also have straightforward effects. For example, as the pre-reproductive natural mortality rate increases, selection pressure decreases for

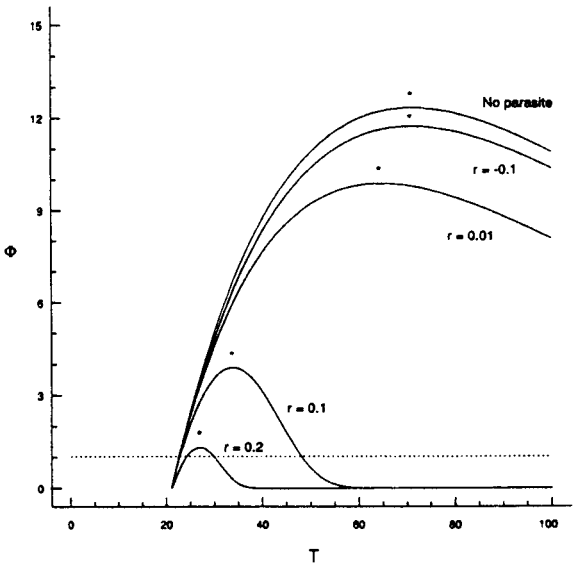

Fig. 2. Effect of time of reproduction on the reproductive value for different growth rates of the parasite population. Curves are obtained by solving equation (10a) for various values of r. Horizontal line at $\Phi = 1$ indicates the persistence threshold of the host population. Asterisks indicate the level of T resulting in a maximum Φ. Other parameters: $\eta_0 = 1$, $P_0 = 1$, $\beta = 0.01$, $\mu = 0.02$, $\nu = 0.005$, $t_0 = 20$, and $t_p = 10$.

any given level of r (Fig. 3a). In contrast, as pre-reproductive mortality due to the parasite increases, selection pressure increases (Fig. 3b). Similar relationships are obtained for increases in initial parasite number (Fig. 3c) and time of infection (Fig. 3d); but, as the potential pre-reproductive period decreases there is an increase in selection pressure (Fig. 3e). Thus, given a limited amount of genetic variation for reproduction time in the host population, we should expect that host populations subjected to parasites of high virulence should attain T_{opt} faster than those exposed to benign forms.

As long as condition (6) is satisfied, reducing the potential number of offspring produced by the host results in increased selection pressure for shorter PRLS (Figs 3a–e, $\beta = 0.1$ in Fig. 3f). But if β from equation (9) is sufficiently large and r sufficiently negative ($r < -\mu$), then condition (6) is violated and a qualitatively different pattern occurs ($\beta = 1$, $\beta = 10$, Fig. 3f). As r increases from negative values, selection pressure initially increases. This selection pressure, contrary to what occurs when (6) holds, is for *longer* optimal times to reproductive maturity than would be the case in the absence of the parasite. This happens because the partial fitness gained from $\eta\{T, P\{T\}\}$ exceeds the loss to $Q\{T, P\{T\}\}$. As r is increased, selection pressure decreases (even though the parasite is effectively more virulent!), since it is becoming less and less favourable to reproduce late. Finally, when the

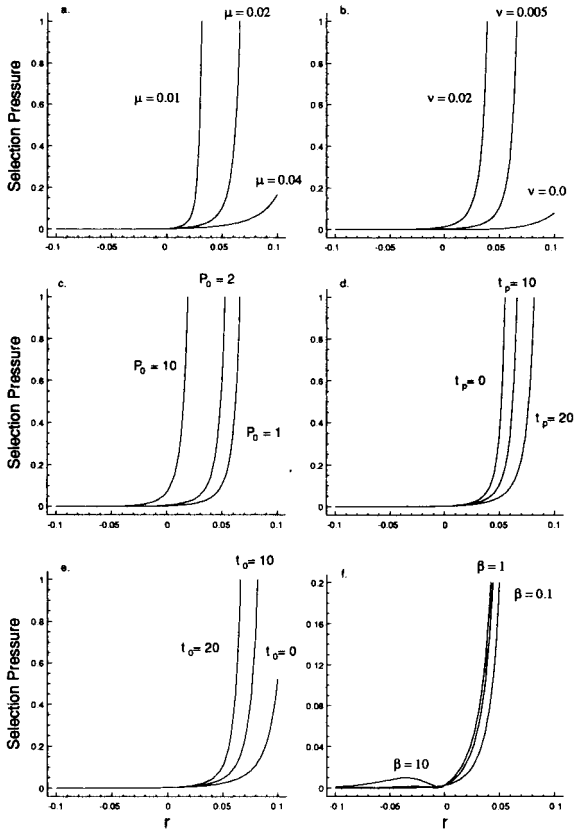

Fig. 3. The influence of parasite virulence (r) and other parameter values on the selection pressure acting on PRLS. This is measured as $(\Phi\{T_{opt}\} - \Phi\{T'_{opt}\})/|T_{opt} - T'_{opt}|\Phi\{T'_{opt}\}$ (where Φ is evaluated in the presence of the parasite, T_{opt} is the optimal time of reproduction in the presence of the parasite, and T'_{opt} is the optimal time of reproduction in the absence of the parasite), assuming that the rate at which a mutant with T_{opt} increases in frequency in a population of hosts with T'_{opt} is $(\Phi\{T_{opt}\} - \Phi\{T'_{opt}\})/\Phi\{T'_{opt}\}$. Note differing vertical scale for Fig. 3f. Unless otherwise specified, parameters as for Fig. 2.

parasite population is stationary or growing, further increases in r are always associated with an increase in selection pressure (Fig. 3f). Similar patterns (not shown) can be generated for the other model parameters as a function of r if β is sufficiently high such that condition (6) does not hold.

When the net fitness accrued per unit maturation time evolved is examined, we find that (assuming condition (6) holds) the maximum fitness benefit occurs for parasites of intermediate levels of virulence (Fig. 4). This can be intuitively understood as follows. In the presence of a benign parasite (i.e. $r < 0$), there is little fitness lost, so that its partial recovery is small relative to the necessary change in reproductive time. As the parasite becomes increasingly virulent, ever more fitness

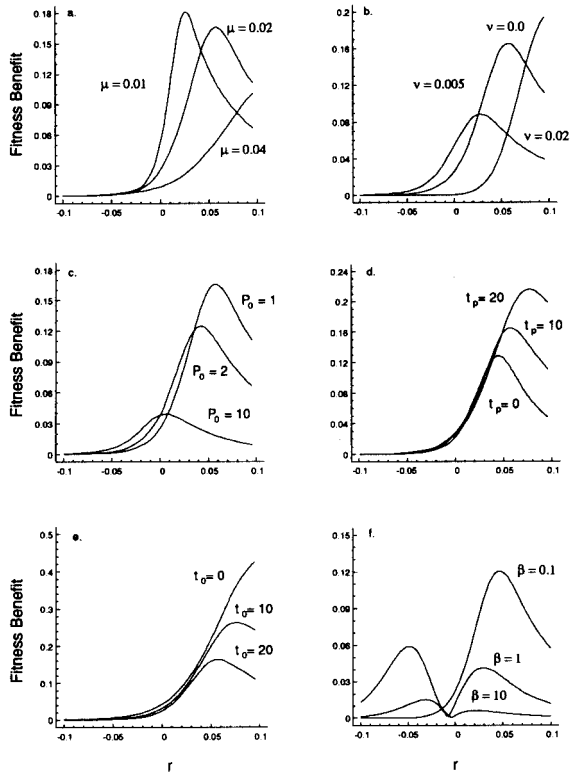

Fig. 4. The influence of parasite virulence (r) and other parameter values on the amount of fitness gained per unit time evolved $((\Phi\{T_{opt}\} - \Phi\{T'_{opt}\})/|T_{opt} - T'_{opt}|)$. See Fig. 3 and the text for further description of parameters.

is recovered per unit change in optimal reproductive time. But as we consider still more virulent parasites, a point is reached in which there is little fitness left to recover, and any increase in virulence is met by an increasingly small return in fitness per unit time evolved. As was expected, in no case did the final maximum level of fitness of the host in the presence of the parasite exceed the maximum level in the parasite's absence.

In cases where condition (6) is violated, two local maxima may arise (Fig. 4f): one associated with avirulent parasites and the evolution of longer PRLS, and the other with virulent parasites and the associated evolution of shorter PRLS. Thus, under the restricted conditions in which (6) does not hold, there can exist a local fitness maximum for each of the two qualitatively different parasite growth patterns ($r < 0$ or $r > 0$). Similar patterns (not shown) are obtained for the other model parameters when condition (6) is violated.

Figure 4 also illustrates how changes in other parameters affect the fitness benefit accorded per unit of evolved time to maturity. For example, as the pre-reproductive

natural mortality rate increases, the fitness benefit decreases for relatively benign parasites, but increases for relatively pathogenic ones (Fig. 4a). So, hosts at a high risk of natural mortalities (i.e. of short PRLS) receive only a small benefit from evolving life-history resistance, and the benefit only becomes substantial when infected by highly pathogenic parasites. In contrast, as pre-reproductive mortality due to the parasite increases, the fitness benefit increases for shrinking or slowly growing parasite populations and decreases for highly virulent parasites (Fig. 4b). Similar relationships are found for increases in initial parasite number (Fig. 4c) and time of infection (Fig. 4d); whereas, as the potential pre-reproductive period decreases there is always an increase in the fitness benefit (Fig. 4e). Finally, as suggested by the patterns of selection pressure in Fig. 3f, increasing the impact of the parasite on the production of offspring generally decreases the fitness benefit if β is sufficiently small, but increases it for a range of $r < -\mu$ if β is sufficiently large.

Discussion

A host's evolutionary response to the invasion of a parasite, or to the evolution of parasitic virulence and pathogenicity once present in the host population, need not be limited to morphological, behavioural, and physiological mechanisms – the host's response may also include changes in the age of reproduction and its life-history correlates (e.g. body size). This is because by simply living longer the host is prone to new infections and increases in the severity of the disease caused by earlier infections. We argue, therefore, that parasites may constitute a cost to the evolution of longer PRLS, and may even impose selection pressures for the evolution of shorter PRLS. On the other hand, evolution towards shorter PRLS due to other selection pressures should confer with it added resistance to parasites.

Relevance to life-history theory

In agreement with life-history theory for iteroparous taxa (e.g. Williams 1957; Charlesworth and Williamson 1975; Stearns 1976; Charlesworth 1980; Calder 1984), we found that the mortality schedule imposed by the parasite should determine how the host responds. We distinguish three scenarios, based on the simulations of our simple model.

(1) Benign parasites (e.g. $r < 0$) will have little or no selective impact on host life-history (because little fitness is lost), and what little impact does occur may not be met by rapid life-history evolution, since relatively little fitness is gained per reproductive time unit evolved (Fig. 4). In situations where the reproduction function $\eta\{T, P\{T\}\}$ increases with time at a faster rate than the survival function $Q\{T\}$ decreases (i.e. when condition (6) is violated and the parasite population is shrinking), there is the potential for parasites to select for longer PRLS. A further interesting special case involves parasites which are benign prior to and during the initial phases of reproduction, but become adverse towards the end of reproduction

and post-reproduction. In such cases the parasite acts as a sort of transmissible senescence gene; the host has no evolutionary response to parasitism and its associated disease (Michalakis, et al. in press).

(2) Parasites of intermediate adversity (e.g. $r > 0$) should be met with a concerted life-history response by the host. Evolution of the parasite to more virulent (and pathogenic) forms should induce a shortening in PRLS; evolution towards avirulence should be answered by longer PRLS.

(3) Severe parasites (e.g. $r \gg 0$), leading to $\Phi < 1$, can potentially drive a host population extinct (assuming that the parasite also exploits host species other than the one considered here). Due to the finite amount of genetic variation for shorter pre-reproductive development to be found in a given host population (and the high costs involved in its evolution), the evolutionary solution of the host could be expected to include changes in both PRLS and in other, more direct, forms of resistance (e.g. physiological), both of which act to reduce the effective virulence of the parasite.

Mechanisms for the evolution of longer PRLS

Our results lead to two non-exclusive mechanisms for the evolution of PRLS (and its life-history covariates).

First, given a host-parasite association in which the parasite is sufficiently virulent and host reproduction occurs at T_{opt}, any tendency on the part of the parasite to evolve towards less virulent forms should result in the host evolving towards longer PRLS. If we assume that other parameters do not evolve, the maximum attainable PRLS will be just that existing in the absence of the parasite.

Second, to pass PRLS limits achieved in the absence of the parasite, the host must previously or simultaneously evolve some degree of behavioural, morphological, or physiological resistance so as to effectively reduce P_0, r, β, v and/or increase t_p. In cases where resistance is sufficient to reduce the parasite population growth rate to negative levels, but the negative effects on host reproduction remain high (i.e. condition (6) is violated), then longer PRLS may evolve without changes to μ and t_0. Otherwise, once sufficient resistance to parasitism has evolved, the costs incurred from parasitism in evolving smaller μ and larger t_0 may be small enough to permit the latter's evolution. This mechanism is consistent with the finding of more sophisticated immune systems in larger animal taxa (Harvell 1990a).

Figure 5 illustrates how the evolution of PRLS might hypothetically proceed in the presence of a parasite population. The necessary requirement of the evolution of longer PRLS is the previous or concomitant evolution of resistance to parasite severity (Fig. 5a). Hosts which would otherwise evolve shorter PRLS in the absence of the parasite, can be expected to approach these optima faster when the parasite is present (Fig. 5b). Similar evolutionary pathways, involving increases in the sophistication of immune systems with accompanying costs in terms of life-history variables (e.g. growth rate and the timing of reproduction) have been suggested by Lively (1986) and Harvell (1986, 1990b).

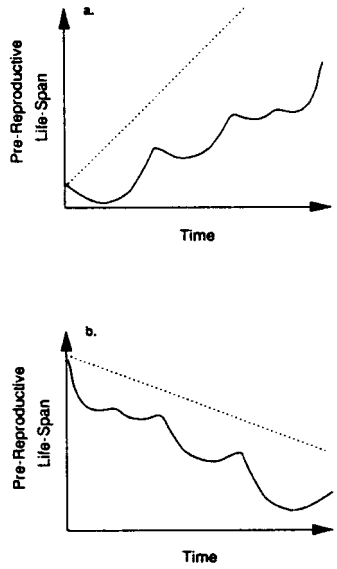

Fig. 5. Examples of hypothetical trajectories for the evolution of pre-reproductive life-span (PRLS) in the absence (broken line) and presence (solid line) of the parasite. a. Scenario in which selection pressures in the absence of the parasite are for longer PRLS. b. Case in which selection pressures in the absence of the parasite are for smaller PRLS. See text for further discussion.

Predictions, limitations and future directions

Our results lead to the expectation that co-evolved host-parasite associations should be characterised by an inverse relationship between parasite severity (combining virulence and pathogenicity) and PRLS (and its life-history covariates), across species of host. As yet, we have no empirical evidence to test this prediction. Nevertheless, an inference from the work of Lively (1986) and Harvell (1986; 1990b) that increased immuno-sophistication is positively associated with species of longer life-spans is consistent with our model that parasitism constitutes an important evolutionary constraint to the evolution of longer times to reproduction.

The results of our theoretical study should be interpreted with some caution since we did not consider, for example, costs involved in the evolution of resistance, explicitly genetic systems, complex growth patterns of the parasite population, the coevolution of the parasite, complex life-histories of the host (e.g. more than one reproductive period), or the evolution of other forms of resistance. Nevertheless, our model serves as a starting point from which more realistic models of life-history evolution can be developed and analysed.

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