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REPORT

Host mortality, predation and the evolution of parasite virulence

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Abstract

One of the most accepted views in the theoretical literature on virulence evolution is that a parasite's virulence will evolve to higher levels when its host's background mortality rate increases. Surprisingly, however, although many sources of background mortality involve predation, there has not yet been any theoretical research that explicitly considers how the dynamics of this important ecological interaction affects virulence evolution. Here, we consider how predation affects virulence evolution by explicitly introducing a predator into a classical susceptible–infected–susceptible epidemiological model. We find that, contrary to previous predictions, different sources of host mortality affect virulence evolution in different ways. Moreover, the way in which virulence evolution is affected depends on how tightly coupled the predator's dynamics are to the host population, and this can result in somewhat counterintuitive results. For example, indirect ecological effects can cause elevated host mortality to result in the evolution of *lower* parasite virulence, even if this elevated mortality arises from factors unrelated to predation.

Keywords

Epidemiology, food web, indirect effects, infection, pathogen, predator–prey.

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INTRODUCTION

The study of virulence evolution has become an extremely active area of research (Bull 1994; Ebert & Herre 1996). This is no doubt because of the fact that this phenomenon is inherently interesting in itself, while also potentially important to applied issues. However, in spite of this interest, it has not yet been possible to make general predictions about virulence evolution using the principles of evolutionary biology, in part, due to the lack of consensus between theoretical and empirical studies.

One set of predictions for which concerted attempts have been made to connect theory with data relates to how different aspects of the host's biology affect the evolution of virulence. One of the most accepted views in this field is that a parasite's virulence will evolve to higher levels when its host's background mortality rate increases (Anderson & May 1982; Kakehashi & Yoshinaga 1992; Lenski & May 1994; Ebert & Weisser 1997). However, although this prediction appears nearly universal in the theoretical literature, conflicting empirical results question its generality (e.g. Treat 1975; Ebert & Mangin 1997).

One explanation for this discrepancy might be inherent in the construction of the models from which this prediction is derived. Recent theoretical studies have attempted to explore this issue by including some more realistic attributes specific to the host–parasite interaction. For example, it has been shown that virulence can decrease with an increase in background mortality rate when virulence makes a host more susceptible to other sources of mortality (Williams & Day 2001), when there is within-host competition (Gandon *et al.* 2001), or when virulence is measured using different indices of host mortality (Day 2002a).

Another potentially relevant extension of standard theory is to reconsider the way that background mortality is usually modelled. Most epidemiological models treat background mortality as a constant parameter, despite the fact that many sources, such as predation, are often dynamic variables. In fact, theoretical investigations into virulence evolution that explicitly consider the ecological setting in which the host is found (i.e. its resources, predators, competitors, etc.) are virtually non-existent (but see Hochberg *et al.* 2000). As a result, it is unknown how these types of ecological interactions will affect the evolution of this important aspect of host–parasite interactions.

In this paper, we consider how predation affects virulence evolution by explicitly introducing a predator

into a standard susceptible–infected–susceptible (SIS) epidemiological model. To facilitate an understanding of the consequences of including an explicit predator in theory on virulence evolution, we consider two extreme predator–prey scenarios: (i) a static predator, where the predator's density is fixed and regulated by some external factor; and (ii) a dynamic predator, where the predator's density is set by its consumption of hosts and its per capita death rate (see Day *et al.* 2002 for further justification of these scenarios).

In our model, there are several different ways in which the host's background mortality might be varied and so affect virulence evolution. These include: increasing the predator's attack rate on either susceptible or infected hosts, decreasing the predator's death rate (which might increase predator density), and increasing host mortality unrelated to predation. Thus, host mortality can be separated into predator-related and -unrelated sources, and it is then possible to explore how these different mortality sources affect the evolution of parasite virulence.

THE MODEL

We use a combined SIS/predator–prey model to describe the ecological dynamics of the host–parasite–predator system (Fig. 1). In this model, the cycle of infection, transmission, and clearance proceeds as follows: a host population is maintained by immigration at rate θ; horizontal transmission of the parasite occurs according to the law of mass action with rate parameter β; and the infection is cleared through host defence mechanisms at rate c without any long-term consequences. To incorporate predation, we partition host mortality into predator-induced mortality at per capita rates $a_S P$ and $a_I P$ and for susceptible and infected hosts, respectively, parasite-induced mortality rate (i.e. virulence), v, and mortality independent of either of these sources at per capita rate u. We also allow for the possibility that virulence affects the susceptibility of infected hosts to predation by allowing a_I to vary with v. In particular, many empirical studies suggest the rate of infected hosts lost to predation increases with virulence because having a virulent infection makes the host less able to evade predators effectively (Holmes & Zohar 1990; Milinski 1990). This results in the following system of differential equations:

$$dS/dt = \theta - \beta SI - uS + cI - a_S SP \quad (1)$$

$$dI/dt = \beta SI - (u + v + c)I - a_I IP \quad (2)$$

$$dP/dt = \varepsilon(a_S S + a_I I)P - \delta P \quad (3)$$

where S, I, and P denote the population densities of susceptible hosts, infected hosts, and predators respectively.

The model discussed above assumes that the predator and prey dynamics are coupled, and it is what we refer to as the dynamic predator scenario. The predator has a linear functional response, a conversion efficiency of ε, and a constant per capita death rate of δ. For the static predator case, we assume that the predator population density is constant and independent of the host's dynamics. Thus, under these assumptions the system reduces to eqns 1 and 2. Note that this scenario is conceptually equivalent to the standard epidemiological models, provided the attack rate on the infected class is independent of virulence, because predation risk is then simply an additional constant source of host mortality.

To consider the evolutionary changes in virulence, we assume virulence evolution is limited by trade-offs between life-history attributes. Most theoretical investigations of virulence assume a trade-off between virulence and parasite transmission (Bull 1994; Read 1994; Ebert & Herre 1996; Frank 1996; but see Day 2002b). As in most theories on virulence evolution, we assume the transmission coefficient, $\beta(v)$, is a saturating, non-decreasing function of virulence. Unlike most such theories, however, we use the results of Day & Proulx (in preparation; available upon request) to derive an equation giving the evolutionary dynamics of virulence within our predator–prey–parasite food web. This allows a better understanding of how the ecological interactions in the food web produce the selective regime that governs virulence evolution. To do so, we simply need to differentiate the *per capita* rate of change of infected hosts from eqn 2 with respect to virulence, and then multiply this by a constant parameter, g, representing the genetic variance in virulence (Day & Proulx, in preparation; available upon request). This approach is analogous to that of standard quantitative genetic models, in which the evolutionary dynamics of a trait are governed by an equation consisting of its genetic variance, multiplied by the selection gradient; i.e. the change in fitness that results from a unit change in

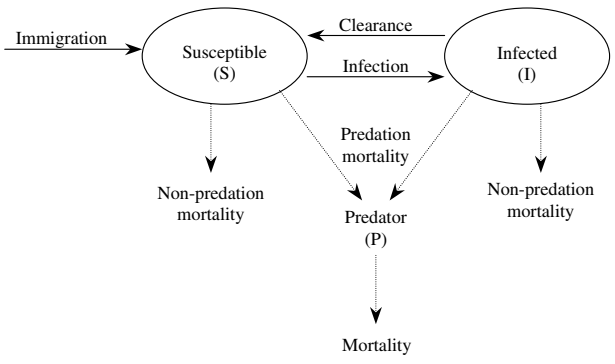

Figure 1 Box diagram of the susceptible–infected–susceptible (SIS) model with predation. Solid arrows indicate transitions between classes, while dotted arrows indicate mortality sources.

trait (Abrams 2001). The per capita rate of change of infected hosts is the appropriate index of parasite fitness, and therefore we obtain

$$\mathrm{d}v/\mathrm{d}t = g\left(S\frac{\mathrm{d}\beta}{\mathrm{d}v} - 1 - \frac{\mathrm{d}a_I}{\mathrm{d}v}P\right). \quad (4)$$

Equations 1–4 give the joint epidemiological and evolutionary dynamics. The first term in the parentheses of eqn 4 is the parasite's transmission benefit due to a unit increase in virulence; note that it is proportional to the density of susceptible hosts, meaning that the transmission benefit of virulence increases with increasing density of susceptible hosts. The second term of eqn 4 (i.e. –1) is simply the parasite's direct mortality cost due to a unit increase in virulence. The final term is the parasite's mortality cost, effected through predation, due to a unit increase in virulence; note that a change in the parasite's virulence will not affect its mortality cost via predation unless the attack rate of the predator on infected prey is a function of virulence (i.e. $\mathrm{d}a_I/\mathrm{d}v \neq 0$).

To analyse virulence evolution, we assume that evolutionary dynamics occur on a much slower time scale than the epidemiological dynamics. This allows us to substitute the (stable) endemic equilibrium values of S and P (denoted S^* and P^*, respectively), obtained by setting eqns 1–3 equal to zero and solving for S and P (and I), into eqn 4. Equation 4 can then be set equal to zero, giving

$$S^*\frac{\mathrm{d}\beta}{\mathrm{d}v} - \frac{\mathrm{d}a_I}{\mathrm{d}v}P^* = 1 \quad (5)$$

as the expression that determines the equilibrium level of virulence (it can also be shown that, for the assumptions used here, this equilibrium is stable under the dynamics given by eqn 4). We can then determine how a parameter, x, affects the equilibrium level of virulence by implicitly differentiating eqn 5 with respect to x. This gives (see appendix),

$$\frac{\mathrm{d}v^*}{\mathrm{d}x} \propto \frac{\partial S^*}{\partial x}\frac{\mathrm{d}\beta}{\mathrm{d}v} - \frac{\mathrm{d}a_I}{\mathrm{d}v}\frac{\partial P^*}{\partial x}. \quad (6)$$

The sign of expression 6 indicates the direction in which the equilibrium level of virulence changes in response to an increase in parameter x.

RESULTS

Some insight into the results that follow is afforded by noting a useful analogy between epidemiological models and other exploiter–victim models, such as predator–prey models. In this case, the susceptible class can be viewed as the victim and the infected class as the exploiter. This perspective is helpful for understanding the indirect effects of changes in the various parameters below.

Static predator

For the scenario where the predator density is constant, the endemic equilibrium is given by

$$S^* = \frac{c + u + v + a_I P}{\beta} \quad (7a)$$

$$I^* = \frac{\theta - S^*(u + a_S P)}{u + v + a_I P}. \quad (7b)$$

An interesting observation to note from these equations is that the population's productivity (as measured by immigration rate) does not affect the density of susceptible hosts in the population. Rather, the influx of any new susceptible hosts is converted into a higher density of infected hosts at equilibrium. As a result, expression 6 shows that immigration rate does not influence virulence evolution.

How does host mortality rate affect virulence evolution? As mentioned before, there are several ways host mortality can be elevated. These include: (i) increasing the attack rate on susceptible hosts, a_S; (ii) increasing the attack rate on infected hosts, a_I; (iii) increasing mortality unrelated to predation or parasitism, u; or (iv) increasing the predator's density, P. We can use eqn 7a in expression 6 to determine how each of these will affect the equilibrium level of virulence.

To begin, suppose that the susceptibility of infected hosts to predation is not affected by the parasite's virulence (i.e. $\mathrm{d}a_I/\mathrm{d}v = 0$). Increasing the attack rate on susceptible hosts, a_S, has no effect on the equilibrium level of virulence because it leaves the equilibrium density of susceptible hosts, S^*, unchanged. This result is somewhat counterintuitive, and arises through indirect effects. An increase in a_S would initially decrease the density of susceptible hosts, but as in many exploiter–victim models, at equilibrium the density of the victim (i.e. susceptible hosts) is unchanged because of a compensatory decrease in the density of the exploiter (i.e. infected hosts), which thereby reduces the loss rate of the victim.

An increase in the attack rate on infected hosts, a_I, leads to a higher equilibrium density of susceptible hosts because it decreases the density of the infected hosts (i.e. the exploiters), and thus the loss rate of susceptible hosts through infection is decreased. As a result, the transmission benefit of a unit increase in virulence goes up while the mortality cost of virulence remains the same. This selects for the evolution of higher virulence. Similarly, increasing the background host mortality rate, u, or the (constant) predator density, P, also leads to a higher equilibrium density of susceptible hosts and therefore selects for higher virulence. Table 1 summarizes the effect that an increase in each of the mortality parameters has on the equilibrium level of virulence.

Table 1 Effect of increased mortality parameters on the direction of virulence evolution for both static and dynamic predator scenarios. ↑ indicates that an increase in a particular parameter causes an increase in the equilibrium level of virulence, while ↓ indicates a decrease in virulence is predicted in response to an increase in a particular parameter. Note that all entries assume that the attack rate on infecteds is independent of virulence (i.e. $da_I/dv = 0$)

Parameter	State Predator	Dynamic Predator
u	↑	↑ if $a_I < a_S$ ↓ if $a_I > a_S$
P	↑	N/A
a_S	No effect	↑
a_I	↑	↑
θ	No effect	↑
δ	N/A	↓

Although the above results illustrate that all mortality parameters do not have equivalent effects on the evolution of virulence, it is relatively straightforward to show that these results are, in fact, in complete agreement with many standard theoretical results of virulence evolution. In such models it is typically the change in the *total mortality of infecteds not induced by the parasite* that governs the direction of evolution in virulence. In the present case, total external mortality has been partitioned into predation mortality, a_IP, and other sources, u, giving a total rate of $\mu = u + a_IP$. Noting that eqn 7a can be written as $S^* = (\mu + c + v)/\beta$, expression 6 implies that $dv^*/dx \propto \partial\mu/\partial x$, where x is any external mortality source (i.e. $x = \delta, a_S, a_I, u$), and the standard result is recovered.

When the attack rate on the infected class varies with virulence (i.e. $da_I/dv \neq 0$), the predictions become more diverse, as expression 6 now depends on the density of susceptible hosts *and* the density of the predator. The predictions for a_S and u are not affected by this change because they still only affect virulence evolution through their effects on the density of susceptible hosts. However, increasing the predator density, P, now affects the benefit of a unit increase in virulence through a change in the density of susceptible hosts (as it did before) as well as the cost of a unit increase in virulence through predation. For example, suppose a_I increases linearly with virulence from a baseline attack rate equal to a_S, giving $a_I(v) = a_S + av$. The overall effect on virulence is proportional to $a_S - a(c + u)$, and therefore depends on the parameter values of the model. In particular, virulence *decreases* with higher predator densities provided this value is negative; that is, when parasite virulence sufficiently increases host susceptibility to predation, a result that recapitulates the findings of Williams & Day (2001).

Dynamic predator

For the case where the predator's density is dynamically coupled to the prey's density, the endemic equilibrium is given by

$$S^* = \frac{c\delta + a_I\varepsilon\theta}{\beta\delta + \varepsilon(a_Iu - a_S(u + v))} \tag{8a}$$

$$I^* = \frac{\delta - a_S\varepsilon S^*}{a_I\varepsilon} \tag{8b}$$

$$P^* = \frac{\beta S^* - (c + u + v)}{a_I}. \tag{8c}$$

In this scenario, it is interesting to note that increased immigration rates now elevate the equilibrium number of susceptibles, although the total host density remains the same (provided that $a_I = a_S$). As a result, the equilibrium level of virulence increases as the transmission benefit of a unit increase in virulence goes up.

If parasite virulence affects the susceptibility of an infected host to predation (i.e. $da_I/dv \neq 0$); however, then the mortality cost of a unit increase in virulence also increases because the predator's density is also elevated by S^*. Under these circumstances, virulence will still increase with immigration rate only if the benefits outweigh the costs. It is also worth noting that these predictions hold true only for immigration rates below a certain threshold (note that this is the case regardless of whether or not a_I depends on v). As the immigration rate increases, compensatory increases in the predator density continue until, eventually, the loss of infected hosts through predation exceeds the generation of new infected hosts, and the parasite then goes extinct.

Again, we can also ask how increases in the different components of host mortality affect virulence evolution. Now, host mortality can be elevated by: (i) decreasing the predator's death rate, δ; (ii) increasing the attack rate on susceptible hosts, a_S; (iii) increasing the attack rate on infected hosts, a_I; or (iv) increasing mortality unrelated to predation or the parasite, u (see Table 1 for summary of effects).

For this scenario, we only consider the case where the attack rate on the infected class is independent of virulence (i.e. $da_I/dv = 0$). A decrease in δ results in an increase in predator density, and this reduces the density of susceptible and infected hosts. However, the susceptible hosts are still victims of the infected hosts, whose decreased density thereby results in a *net increase* in the density of susceptible hosts at equilibrium. This selects for (by eqn 6) a higher level of virulence.

Unlike the static predator case, an increase in the attack rate on susceptible hosts, a_S, now selects for a higher level of virulence. The reason is that this increases the predator density, which thereby reduces the density of infected hosts, and this allows the equilibrium density of susceptible hosts

to be larger. Similarly, an increase in the attack rate on infected hosts, a_I, also results in a higher equilibrium level of virulence.

Perhaps the most surprising result occurs when we consider an increase in the mortality rate that is unrelated to predation (i.e. u). Where this resulted in an increased level of virulence in the static predator case, it now results in a *decreased* level of virulence provided the attack rate on infected hosts is greater than that on susceptible hosts. More specifically, the equilibrium level of virulence changes in a direction given by the sign of $-(a_I - a_S)$. This occurs because, as opposed to the static predator case, predator density now decreases because of the decrease in prey density cause by the increase in u. This, in turn, reduces the rates at which both infected and susceptible hosts are removed through predation. Moreover, this reduction will be greater for infected hosts whenever $a_I > a_S$. Thus, the relative abundance of infected hosts will go up. Because they are exploiters of the susceptible hosts, the equilibrium density of susceptible hosts will then decline, selecting for a reduced level of virulence. Notice, however, that this prediction depends on the predator–prey interaction. If the predator preferentially preys on the susceptible hosts, then the equilibrium level of virulence will increase. And if the predator fails to differentiate between susceptible and infected hosts, then host mortality no longer has any affect on the parasite's virulence. These distinctions emphasize the importance of the host's environment in determining the outcome of virulence evolution.

As in the static predator case, we can attempt to identify the connection between these results and those of standard theory. In this case, the total external mortality of infecteds not induced by the parasite is given by $\mu = u + a_I P^*$, and we again have (by eqn 8c) that $S^* = (\mu + c + v)/\beta$. Thus, for any mortality term, x, $dv^*/dx \propto \partial\mu/\partial x$, and we again recover the standard result. However, as opposed to the static predator case, changes in certain non-mortality parameters can induce *opposite* changes in the total natural mortality rate of infecteds (μ) and the equilibrium level virulence (v^*) via indirect effects on predator density. For example, by eqns 6 and 8a, increased clearance (c) causes virulence to increase (by increasing the equilibrium density of susceptibles), while the direction of change in total natural mortality rate is given by the sign of $\partial\mu/\partial c = \beta(\partial S^*/\partial c) - 1$. When this quantity is negative, virulence evolves in the opposite direction to the one predicted by the change in total natural mortality rate of infected hosts. By eqn 8a, this occurs when the inequality $a_I u > a_S(u + v^*)$ is satisfied.

This last example appears to suggest that, rather than the change in the total natural mortality rate of infecteds, it is the change in *the total natural loss rate of infected hosts* that governs the direction of virulence evolution, where total natural loss rate is defined as $\hat{\mu} = \mu + c$. However, it can be shown that this, too, is an insufficient predictor of the direction of virulence evolution for all possible parameter manipulations. For example, with $\beta = \alpha v/(b + v)$, parameter combinations can be found such that increasing b decreases the total natural loss rate of infected hosts while increasing the equilibrium level of virulence (unpublished result). In general, our results show that, while simple descriptions of virulence evolution are available when parameters that cause direct mortality are varied (i.e. all parameters affecting $\hat{\mu}$), this is not the case for parameters that have indirect mortality effects. This illustrates that when ecological interactions are explicitly accounted for, the question of how changes in 'mortality' parameters affect virulence evolution is subtler, as changes in certain parameters that appear unrelated to mortality (e.g. parameters affecting β) can nevertheless induce mortality changes through indirect ecological effects.

As a final note, we emphasize that the above results all assume that a linear functional response governs predator dynamics. As non-linear functional responses often lead to non-equilibrium dynamics, we have not attempted to explore the consequences of this for virulence evolution. Nevertheless, it should be noted that even the simple results, summarized by the relationship $dv^*/dx \propto \partial\hat{\mu}/\partial x$, where x is any direct mortality term, are unlikely to hold when predator dynamics are determined by a non-linear functional response.

CONCLUSIONS

Our results show that the community in which the host is embedded (and the ecological interactions that this entails) can have important effects on virulence evolution, and that the inclusion of a predator can result in a variety of new predictions. It is well known that indirect ecological effects arising from compensatory changes in the density of various species in a food web can often result in counterintuitive predictions regarding changes in a focal species. The same effects arise in the more realistic ecological scenarios for virulence evolution considered here. It is only through the explicit inclusion of ecological factors that are thought to be important, such as predation, that we can accurately predict evolutionary responses in virulence.

By focusing on the two extreme scenarios of static and dynamics predators, our results highlight the importance of the extent to which the elements in the food web are coupled (also see Day *et al.* 2002). Additionally, these results illustrate that interactions between the effects of predation and parasitism on the host can generate novel and unanticipated predictions. This finding reinforces those of Williams & Day (2001), who argued that differential susceptibility to external mortality sources (such as

predation), as a result of different levels of host exploitation, can reverse widely believed predictions about expected evolutionary changes in parasite virulence.

Finally, another interesting result is that an increase in the host's productivity (as measured by immigration) can actually push the parasite towards extinction. With high immigration rates, the influx of susceptible hosts gets translated into a higher predator density, and this can drive the host population density below the threshold required for the parasite to remain extant. Such enhanced productivity would only increase a parasite's ability to persist in standard models (i.e. the simple static predator scenario).

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APPENDIX

In this appendix, we present the derivation of expression 6. Defining

$$f(v, x) = S^* \frac{\partial \beta}{\partial v} - \frac{\partial a_I}{\partial v} P^*,$$

eqn 5 can be written as

$$f(v^*, x) = 1. \tag{A1}$$

We can implicitly differentiate A1 with respect to x, treating v^* as a function of x, to get

$$\mathrm{d}v^*/\mathrm{d}x = \frac{-\partial f/\partial x}{\partial f/\partial v}. \tag{A2}$$

Now, if v^* is a stable equilibrium of eqn 4, then we must have $\mathrm{d}f/\mathrm{d}v < 0$. This gives

$$\mathrm{d}v^*/\mathrm{d}x \propto \frac{\partial S^*}{\partial x}\frac{\partial \beta}{\partial v} - \frac{\partial a_I}{\partial v}\frac{\partial P^*}{\partial x} + S^* \frac{\partial^2 \beta}{\partial x \partial v} - \frac{\partial^2 a_I}{\partial x \partial v} P^*. \tag{A3}$$

Assuming both a_I and β are independent of the parameter x yields expression 6.