Notes and Comments

Predation Can Increase the Prevalence of Infectious Disease

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ABSTRACT: Many host-pathogen interactions are embedded in a web of other interspecific interactions. Recent theoretical studies have suggested that reductions in predator abundance can indirectly lead to upsurges in infectious diseases harbored by prey populations. In this note, we use simple models to show that in some circumstances, predation can actually increase the equilibrial prevalence of infection in a host, where prevalence is defined as the fraction of host population that is infected. Our results show that there is no complete generalization possible about how shifts in predation pressure translate into shifts in infection levels, without some understanding of host population regulation and the role of acquired immunity. Our results further highlight the importance of understanding the dynamics of nonregulatory pathogens in reservoir host populations and the understudied effects of demographic costs incurred by individuals that survive infection and develop acquired immunity.

Keywords: host-pathogen, predator-prey, regulation, immunity.

There is a growing recognition of the need to integrate the principles of community ecology with host-pathogen epidemiology (Collinge and Ray 2006). Even specialist host-pathogen interactions are embedded in webs of interactions with other species, generating potentially strong indirect interactions and complex dynamic feedbacks (Dwyer et al. 2004; de Castro and Bolker 2005; Keesing et al. 2006). Predation by generalist predators on infected hosts can alter the incidence of parasitism (Hudson et al. 1992; Arneberg et al. 1998; Ostfeld and Holt 2004; Hall et al. 2005). Packer et al. (2003) and Ostfeld and Holt (2004) have recently presented models suggesting that predator removal can indirectly harm prey populations as a result of pathogen outbreaks, and Holt and Dobson (2006) argue that such removal indirectly facilitates spillover infection to nonprimary hosts. Hall et al. (2005) show that selective predation on parasitized hosts can make it difficult for parasites to persist. Thus, one might conclude that a general result of ecological theory is that predators will reduce the prevalence of infection in host populations that are prey for those predators.

In this note, we demonstrate that in some quite reasonable scenarios, predators can instead lead to an increase in pathogen prevalence. We show that the pattern relating disease prevalence to predation pressure can be influenced by the mode of host population regulation and the presence of immune classes and that a hump-shaped relationship between the magnitude of generalist predation pressure and disease prevalence can readily arise.

Previous studies (Packer et al. 2003; Ostfeld and Holt 2004) have assumed that a focal host species is attacked by a generalist predator whose numbers are driven by factors other than abundance of the focal species. For simplicity, we make this same assumption. Packer et al. (2003) briefly mention the effect of incorporating acquired immunity but do not explore the topic in any detail. We consider three models, all with acquired immunity, and assume that newborn individuals are susceptible to infection. In the first model, the pathogen does not regulate the host, which instead is limited by other factors (e.g., availability of nest sites or food) to a given carrying capacity, K. In the second, we assume the host is entirely regulated by the pathogen. The third model assumes joint regulation of the host by both direct density dependence and the pathogen, and it also assumes frequency-dependent transmission. These models bracket a wide range of more complex assumptions about pathogen impacts on host numbers and transmission dynamics.

There are two complementary measures of disease load in a host population: the actual density of infected individuals, I, and prevalence, $p = I/N$, where N is total pop-

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ulation size. Prevalence scales the probability of disease transmission per encounter between an infected host and a random susceptible host. Prevalence is what is empirically estimated from a random sample of individuals in the population, and it can sometimes be the measure most useful for assessing infection risk (e.g., when a child is bitten by a dog, the parents' level of concern should increase with increasing prevalence of rabies in the local dog population). The abundance of the infected class I^*, by contrast, measures the actual disease load in the population. This may be of particular importance when considering the likelihood of spillover between the focal host species and a second, incidental host. For a given prevalence, a rare reservoir host is of much less concern than is an abundant reservoir host. Because prevalence can be estimated from a sample, it is in general much easier to assess empirically than the actual abundance of infected individuals (which for mobile animals usually requires analyses of mark-release-recapture data). We will show that these two measures of disease load can respond differently to shifts in predation pressure.

Models and Results

Nonregulatory Pathogen

Consider the common cold in humans. It clearly has its own dynamics, but there is no evidence to suggest that it has more than a negligible effect on host population size. For such pathogens, the host population provides a template against which pathogen dynamics are played out, but with no noticeable effect on the host; the pathogen is in effect a commensal at the level of the population. In this case, in the phrase of Jaenike and Perlman (2002), pathogens are a kind of "trophic garnish" on their hosts. Understanding the dynamics of such near-commensal pathogens can be important, we suggest, because they provide the potential for emerging diseases arising from spillover onto novel host species, or in the original host if there is environmental change influencing pathogenicity.

A useful limiting case for analyzing the dynamics of a nonregulatory pathogen is to assume that the host population stays fixed at its carrying capacity. Total host numbers could be regulated by strong density dependence arising from a number of mechanisms (e.g., territoriality capping numbers), so that if there is a change in mortality, there is a rapid compensatory response in recruitment. Given strong intrinsic host regulation, a reasonable starting approximation is for host numbers to stay fixed at the carrying capacity K, so if S, I, and R are, respectively, the numbers of prey/host individuals in the susceptible, infected, and recovered (and immune) classes, then $K = S + I + R$. With density-dependent disease transmission, the total rate of new infections is βSI, where β is the disease transmission coefficient. After substitution for S, the equations describing pathogen dynamics are

$$\frac{dI}{dt} = \beta(K - I - R)I - [\gamma + m_{\mathrm{I}}(C)]I,$$

$$\frac{dR}{dt} = \gamma I - m_{\mathrm{R}}(C)R, \tag{1}$$

where γ is the rate of recovery from infection, m_{I} and m_{R} are per capita rates of mortality of the infected and recovered classes, respectively, and C denotes predator abundance. We assume that mortality in each host class increases with C.

The pathogen can establish in the prey population only if the basic reproduction number $R_0 > 1$, where

$$R_0(C) = \frac{\beta K}{\gamma + m_{\mathrm{I}}(C)}. \tag{2}$$

For the disease to be established, K must exceed a threshold K_{th}, given by

$$K_{\mathrm{th}}(C) = \frac{1}{\beta}[\gamma + m_{\mathrm{I}}(C)]. \tag{3}$$

Since mortality increases with C, R_0 decreases and K_{th} increases with increasing C, making it more difficult for the pathogen to establish at higher predation pressure.

By contrast, given that the pathogen is established ($K > K_{\mathrm{th}}$), its prevalence can increase with increasing predation, at least over some range of mortalities and for certain patterns of predation. The equilibrial prevalence $p^* = I^*/K$ is

$$p^* = \frac{1 - \dfrac{\gamma + m_{\mathrm{I}}(C)}{\beta K}}{1 + \dfrac{\gamma}{m_{\mathrm{R}}(C)}}. \tag{4}$$

Note that mortality enters in both the numerator and denominator of expression (4) and that the mortality of infected individuals has a different effect on p^* than does the mortality of recovered hosts. This potentially leads to a nonlinear relationship between predation levels and disease prevalence. This relationship depends strongly on host abundance and on the pattern of prey selectivity exhibited by the predator.

Consider first host abundance. As K increases, expression (4) approaches $p^* \simeq m_{\mathrm{R}}(C)/[\gamma + m_{\mathrm{R}}(C)]$. If the death rate of immune individuals increases with C, so

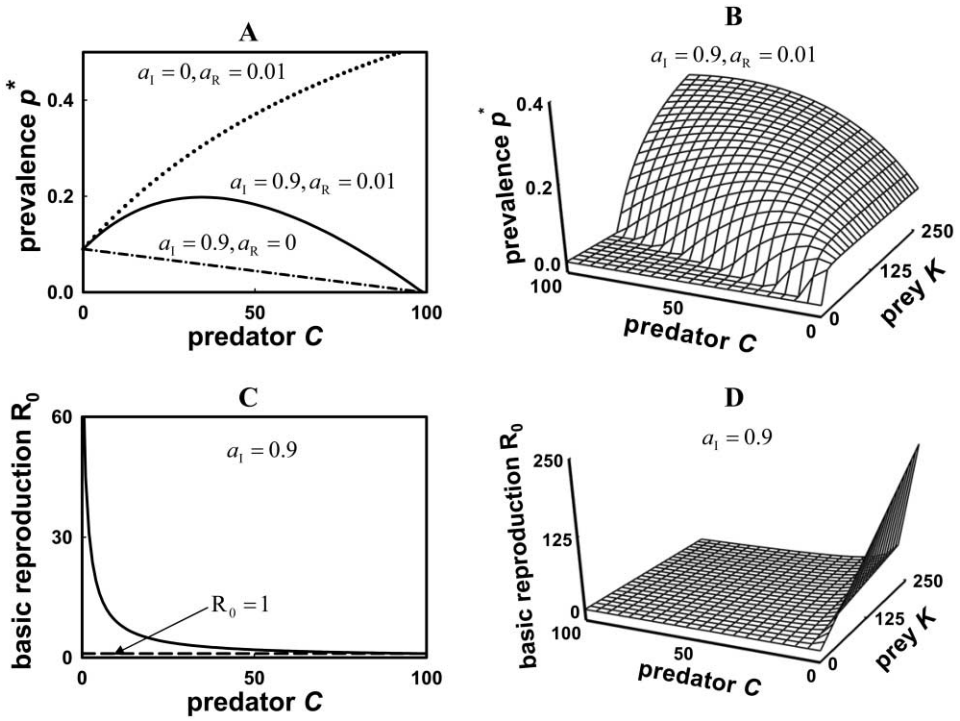

Figure 1: Theoretical example of how predation can increase disease prevalence for a nonregulatory pathogen. In the examples shown, equilibrial prevalence p^* and the basic reproduction number R_0 are plotted against predator abundance C. For simplicity, we assume that mortalities are linear functions of C. *A*, Equilibrium prevalence p^* (eq. [4]) is plotted against C for three examples of foraging choices with host carrying capacity $K = 90$. *B*, Three-dimensional plot showing dependence of p^* on both prey and predator abundances K and C, with predation on both prey classes. As noted in the text, often prey are more abundant than predators, and we have scaled the axes to reflect this fact of natural history. *C*, Basic reproduction number R_0 (eq. [2]) is plotted against C for $K = 90$, corresponding to two of the attack patterns shown in *A*. *D*, R_0 plotted against C and K. Other model parameters are $\beta = \gamma = 1$ and $m_{I0} = m_{R0} = 0.1$.

does p^*. By contrast, when K gets low (so the R_0 of the infection approaches 1), dp^*/dC becomes proportional to $-dm_I(C)/dC < 0$ (details not shown), so in this limit, an increase in predation will typically depress disease prevalence.

This leads to qualitative predictions about how shifts in predation should influence patterns of disease prevalence in prey communities for diseases with density-dependent transmission. Typically, predators are less abundant than their prey taken as a whole (Carbone and Gittleman 2002; Marquet 2002). Nonetheless, in the diet of generalist predators, it is likely that there will be some abundant prey species as well as some scarce prey species. In abundant host populations, an increase in predator abundance could increase the prevalence of infectious host-specific diseases. By contrast, host-specific diseases in scarce prey should decline when predator numbers rise.

The expected relationship between predator abundance and disease prevalence is strongly influenced by the pattern of prey selectivity exhibited by the predator, as modulated by prey behavior. In some cases, only infected prey individuals can be caught by the predator. In others, infected prey may go into hiding to aid recovery, so the predator encounters only healthy prey. Finally, the infection may have only marginal effects on prey vulnerability to predation.

At one extreme, if the predator selectively attacks only the infected prey, increasing predator abundance should monotonically decrease the equilibrium prevalence of the disease, as shown in the susceptible-infected (SI) model by Packer et al. (2003). At the other extreme, if predators focus only on immune individuals, p^* increases monotonically with increasing C. Finally, if the predator indiscriminately attacks all prey individuals, the equilibrium prevalence p^* can initially increase with predator abundance C but eventually will start decreasing at large values of C, leading to the hump-shaped dependence between p^* and C shown in figure 1. For simplicity, in the examples shown in the figure, we assume that per capita prey mortality rates are linear functions of predator abundance, $m_i(C) = m_{0,i} + a_iC$, where the subscript i denotes S, I,

and R classes, $m_{0,i}$ denotes background mortality, and a_i is the predator attack rate on prey class i.

If we assume that the infectious agent does not affect mortality rates (as with the common cold) and, moreover, that predation is inflicted equally on all host classes (so $m_I = m_R \equiv m$), then after a little manipulation we can show that a small increase in predator abundance will increase disease prevalence if the following condition holds:

$$\frac{\gamma}{m(C)^2}\{\beta K - [\gamma + m(C)]\} > 1 + \frac{\gamma}{m(C)}. \tag{5}$$

This inequality is likely to hold (and hence, predation to increase disease prevalence) if (1) prey carrying capacity K is high, (2) disease transmission rate β is high, and (3) prey mortality m is low. If the recovery rate γ is low, by contrast, the above expression is not likely to hold.

One interesting pattern emerges with asymmetric predation on different prey classes. Figure 1 shows an example in which predation is inflicted mainly on infected individuals but there is a small amount of predation on recovered individuals (solid line in fig. 1*A*; surface in fig. 1*B*). In the absence of predation on immune individuals, as noted above, prevalence simply declines with increasing predation. But with a small amount of predation on the immune class, there is a very broad pattern of increasing prevalence with predation, particularly when prey numbers are greater than predator numbers.

To explore this effect in more detail, in appendix B in the online edition of the *American Naturalist*, we examine systematically the impact of differential predation on the infected and immune prey classes. In figure B1, we plot the range of predator abundance (denoted C_+) over which $dp^*/dC > 0$, scaled against the entire range (denoted C_{all}) for which $p^* > 0$, for different values of the predator attack rates a_I and a_R. The plot reveals two features: (1) $C_+ > 0$ as long as both $a_I > 0$ and $a_R > 0$, implying that the behavior is not restricted to a small corner of the attack parameter space; and (2) C_+ is largest (up to almost 50% of the entire range) when a_I is high and a_R is low; that is, the expectation that disease prevalence will increase with increasing predator numbers is particularly likely to hold if the predator predominantly attacks the infected individuals but also occasionally captures some recovered individuals. This assumption certainly applies to some natural systems. For instance, red grouse that are parasitized are more vulnerable to predation, but unparasitized grouse can still be caught by predators (Hudson et al. 1992). We suspect that it will be frequently the case that predators will focus attacks on infected prey but also capture some healthy prey as well. This highly plausible pattern of attacks is likely to lead to a counterintuitive increase in prevalence with increasing predation, particularly when prey are more abundant than predators (see fig. 1*B*).

The effect of increasing disease prevalence with increasing predation arises because of feedbacks arising from host regulation. Because we have assumed rapid recruitment of newborns into the population, leading to a population regulated tightly by K, predation that reduces abundance of the recovered class is compensated for by higher recruitment into the susceptible class. This increases the supply rate of susceptible hosts, which in turn increases disease transmission, and, hence, prevalence. An indirect effect of predation inflicted on immune prey individuals may thus be an increase in the supply of hosts available for infection, thereby facilitating the spread of the pathogen in the host population. Note that this effect does not influence the negative impact of predation on the basic reproductive number of the pathogen because this number is evaluated when the pathogen is very rare and immune hosts are vanishingly rare (see fig. 1*C*, 1*D*).

In model (1), the actual number of infected individuals is, of course, $I^* = p^*K$, so the pattern in abundance of infecteds directly tracks the pattern in prevalence. In the next model that we consider, p^* and I^* can respond differently to predation.

Regulatory Pathogen

Packer et al. (2003) briefly touch on a susceptible-infected-recovered (SIR) model for hosts regulated solely by the pathogen but do not discuss it in any detail. Because density dependence in this model emerges solely and indirectly from infection dynamics, there is no reason to expect compensatory increases in host reproduction following an increase in predation, which drove the effect we explored in the previous section. It is interesting that nonetheless, in some circumstances, a hump-shaped relationship between prevalence and predation pressure can occur. This suggests that several distinct processes can act to generate a counterintuitive facilitative effect of predation on pathogen prevalence. The details of the model and analysis are presented in appendix A.

In contrast to the nonregulatory model explored above, equilibrial prevalence and the density of infecteds can show different and opposite responses to changes in predation. The details are laid out in appendix A, and an example is shown in figure A1. As in the simple SI model discussed by Packer et al. (2003) and Ostfeld and Holt (2004), and for the same reasons, the equilibrial abundance I^* of infected prey always decreases with increasing predation, regardless of the pattern of mortality inflicted by predation. An increase in the death rate of infected hosts reduces the number of secondary infections generated per infected host, thus reducing the number of infected individuals.

An increase in the death rate of either susceptible or immune hosts in effect reduces the productivity of these hosts, which indirectly depresses the abundance of infected individuals that can be sustained in the population. However, as shown in appendix A, equilibrial prevalence p^* can in some circumstances nonetheless increase with an increase in predation. Two conditions necessary for this to occur are that immune individuals be vulnerable to predation and, moreover, that there be a long-term demographic cost of the infection, so that immune individuals have a lower birth rate than do susceptible hosts who have never been infected. The reason that prevalence can go up with increasing predation, even as the abundance of infecteds declines, is that total host population size also responds to shifts in predation, and it can decline even faster.

Other Models

We have explicitly considered two limiting cases of host regulation, one in which the pathogen does not regulate the host at all and the other in which the pathogen is the sole factor regulating host growth. The real world is doubtless bracketed between these two extremes. Moreover, we have assumed density-dependent disease transmission, while the kinetics of transmission is often more complex than this. We have examined a range of models including immunity with alternative assumptions about host population regulation and different functional forms for disease transmission, and we frequently observe similar results (M. Roy and R. D. Holt, unpublished manuscript). Here we present just one example. We will assume that there is density dependence in births, which regulates the host when it is free from the pathogen. Moreover, we will assume that disease transmission is frequency dependent.

The equations are

$$\begin{aligned}\frac{dS}{dt} &= (b_S S + b_I I + b_R R)(1 - dN) - m_S S - \beta\frac{SI}{N},\\ \frac{dI}{dt} &= \beta\frac{SI}{N} - (\gamma + m_I)I,\\ \frac{dR}{dt} &= \gamma I - m_R R\end{aligned} \tag{6}$$

(where we choose parameters such that $N < 1/d$). After considerable grinding of algebraic gears, it can be shown that the equilibrium prevalence is

$$p^* = \frac{\beta - \gamma - m_I}{\beta(1 + \gamma/m_R)}. \tag{7}$$

As in our first model, the mortality rates of infected and immune hosts enter in quite different ways. An increase in predation on infected individuals reduces prevalence. But increased predation on immune individuals always increases disease prevalence. When there is indiscriminate predation on all classes, a hump-shaped relationship between predation and disease prevalence can occur (see fig. 2 for an example; note that the increase occurs over a substantial fraction of the range of predator densities that permit the pathogen to persist). Interestingly, host birth rates, density dependence, and attack rates on susceptibles drop out of expression (7) for prevalence, but they do enter into the expression for infected host abundance (details not shown). This implies that p^* and I^* can respond in qualitatively different ways to changes in predation.

Discussion

It appears that the interplay of host regulation, immune responses, and the pattern of predator selectivity jointly determine whether predators reduce or paradoxically increase the abundance and prevalence of infected individuals. If pathogens and predators are nonregulatory and the host is strongly regulated by other factors, increased host births compensate for increased deaths. Because births provide fresh, susceptible hosts, an increase in predation on recovered individuals can thus indirectly lead to an increase in the fraction of the population that is infected. By contrast, when a host is regulated solely by a pathogen, an increase in predation will usually depress the

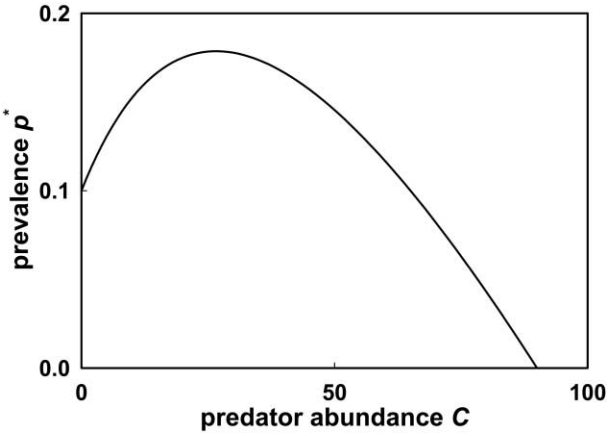

Figure 2: Equilibrium prevalence p^* (eq. [7]) plotted against predator abundance C, assuming logistic density dependence in the host and frequency-dependent disease transmission. The parameters used are $\beta = 15$, $\gamma = 5$, $m_{I0} = m_{R0} = 1$, and $a_I = a_R = 0.1$. Prevalence increases with predator abundance over approximately one-third of the range of predator density permitting pathogen persistence.

abundance of infected hosts (as suggested by Packer et al. [2003]). But in some cases, if past infection or acquired immunity has sustained demographic costs, disease prevalence can actually increase with increased predation. Surveys of disease prevalence along environmental gradients of predation pressure may thus provide an incomplete and indeed misleading picture of how absolute infection levels shift along these gradients. Our brief treatment of a third model with logistic host growth and frequency-dependent transmission illustrates that a nonmonotonic relationship between prevalence and predation might crop up in a wide range of alternative model structures. The theoretical finding that increases in predation can sometimes increase disease prevalence thus appears to be robust. We should stress that a necessary condition for the effect is that hosts have an immune response to infection. Moreover, if host mortality rates are sufficiently high that very few individuals are found in the immune class, one would not expect the effect to be quantitatively important.

We suggest that there are compelling reasons to consider the dynamics of pathogens that are nonregulatory to their primary hosts. Many zoonotic infections are believed to be maintained by reservoir hosts in which they appear to be benign (Ostfeld and Holt 2004; G. Glass, personal communication). For instance, hantavirus in wild Norway rat populations in inner-city Baltimore has no measurable demographic impacts on its rodent hosts (Child et al. 1989), but it is of public health concern because of potential spillover to human hosts (Glass et al. 1993). Shifts in the ecology of these hosts, such as alterations in predation pressure, can lead to changes in the likelihood of transmission across species, and in novel hosts the pathogen can at times wreak considerable demographic damage. Our results show that there is no complete generalization possible about how shifts in predation pressure on reservoir hosts translate to shifts in risk of infection to novel hosts, without some understanding of host population regulation, the pattern of predator selectivity, and the likelihood of acquired immunity in the reservoir host.

Note that the patterns expected depend strongly on the diet choices of the predator. If a predator completely ignores recovered prey, then predation will always depress pathogen abundance. The complications we have demonstrated depend on predators being able and willing to attack hosts that have recovered from the disease. However, some of the strongest effects can occur when the rate of predation on immune hosts is quite low (as in fig. 1). If predators avoid infected prey, then predation will often enhance prevalence. The whole issue of how relative attack rates vary among host classes is poorly understood empirically. A variety of scenarios seem plausible. For instance, on average, recovered prey will be older than either susceptible or infected prey. If there are behavioral changes during the life history such that older individuals are more vulnerable to predation (e.g., because they engage in mating behaviors or territorial squabbles, or simply because of aging), then on average there could be greater predation inflicted on recovered individuals, relative to younger classes of infected individuals, simply because the former are older.

Another interesting issue raised by our results for the regulatory SIR model is that there can be substantial effects of costs of acquired immunity, in our case as measured by the qualitative pattern in changes in disease prevalence as a function of changes in predation. There are two ways one can conceptualize "costs" of entering a recovered or immune host class. First, there can be lingering effects of having fought off a disease. For instance, sexually transmitted diseases can lead to partial or complete sterility, and individuals who recover from polio may have lifelong impaired muscular function. Second, there may be recurrent costs of the immune response itself. This is one area that needs much more empirical attention. Our results suggest that in addition to the intrinsic interest of understanding the costs of immunity, there may be important ecological effects of such costs.

There are, of course, many ways in which simple SI and SIR models of the sort considered here and elsewhere (Packer et al. 2003; Ostfeld and Holt 2004) greatly simplify host and pathogen dynamics. For instance, these models ignore variation among individuals in demography (e.g., age/stage classes, sex, or body condition), pathogen transmission, and predation risk. The life-history details of density dependence could strongly affect the availability of susceptible hosts for infection. Given acquired immunity, density dependence in births means that at high densities there is a reduced per capita rate of production of new, susceptible recruits entering the population. But if density dependence exists solely in deaths, there is no such reduction. Variation in mortality due to shifts in predation could thus have substantially different effects on disease dynamics, depending on the nature of density-dependent regulation in the host. Moreover, because of the additional time lags implicit in having an explicit life history with distinct stages, there is the potential for sustained oscillations; unstable host-pathogen dynamics can also emerge because of saturating functional responses even if predator numbers are fixed (Hall et al. 2005; M. Roy and R. D. Holt, unpublished data), and this effect is amplified when predators have strong numerical responses (e.g., Hochberg et al. 1990; Hethcote et al. 2004). In the models we have considered here, the equilibria appear to be stable if the predators act simply as a fixed mortality factor (M. Roy and R. D. Holt, unpublished manuscript). Predators could also cause shifts in prey behavior, leading to a variety of potential indirect effects on disease dynamics (Keesing et

al. 2006). For instance, if prey move less in order to reduce mortality risk when predators are abundant, then their contact rates may decline; conversely, if prey hide in limited numbers of refuges at high predator numbers, they may experience more contacts with a greater potential for infection. Finally, resource limitation can have a variety of effects on disease dynamics (Smith and Holt 1996). If changes in predation alter prey numbers, this can indirectly alter resource availability, which in turn can influence the ability of prey individuals to avoid or fight off infection.

Considering many of these theoretical extensions will be important for closely linking this set of ideas to empirical systems. For instance, in the rat-virus system mentioned above, it is likely that predation is size specific, and moreover, transmission appears to occur more readily in some age classes than in others (G. Glass, personal communication). So working out the effect on viral prevalence of changes in predation pressure (e.g., due to feral cat control programs) will require developing stage- and age-structured models tailored to this system. In broad comparative studies, what is desired at the very least is an assessment of prevalence as a joint function of host and predator abundance. The model for a nonregulatory pathogen presented above predicts that counterintuitive increases in disease prevalence with increasing predator abundance are more likely when the host is abundant than when it is rare. We are unaware of any data sets at present that would permit an assessment of this prediction.

Discerning the impact of changes in predation on disease prevalence in natural populations will require one to grapple with many of these real-world complexities. Given the growing evidence for disruption of top predators in terrestrial and marine ecosystems worldwide (Turner 1996; Terborgh et al. 2001; Baum and Myers 2004), we think this challenge warrants serious attention both theoretically and empirically.

Acknowledgments

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APPENDIX A

SIR Model without Direct Density Dependence

The SIR model is as follows:

$$\begin{aligned}\frac{dS}{dt} &= [b_S - m_S(C)]S - \beta SI + b_I I + b_R R,\\ \frac{dI}{dt} &= \beta SI - [\gamma + m_I(C)]I,\\ \frac{dR}{dt} &= \gamma I - m_R(C)R.\end{aligned} \tag{A1}$$

Here, b_S, b_I, and b_R are, respectively, birth rates of susceptible, infected, and recovered individuals, and m_S, m_I, and m_R are their mortality rates, which we again assume are increasing functions of predator abundance. For illustrative purposes, we assume as in the main text that mortalities increase linearly with predator abundance C, and we use m_{S0}, m_{I0}, and m_{R0} to denote density-independent mortalities and a_S, a_I, and a_R to denote the predator attack rates on the three classes of prey.

The change in total prey abundance $N = S + I + R$ is

$$\frac{dN}{dt} = [b_S - m_S(C)]S + [b_I - m_I(C)]I + [b_R - m_R(C)]R. \tag{A2}$$

We assume that the prey, when free of the infection, persists, so $b_S > m_S$ (the value of C at which $b_S = m_S(C)$ holds denotes the upper limit of predation intensity that the healthy host population can tolerate); hence, for the pathogen to regulate the host, deaths must exceed births for infected individuals, recovered individuals, or both.

The equilibrial values of infecteds, total host abundance, and prevalence are

$$I^* = \frac{m_R(C)[b_S - m_S(C)][\gamma + m_I(C)]}{\beta\{\gamma\ [m_R(C) - b_R] + m_R(C)[m_I(C) - b_I]\}}, \tag{A3}$$

$$N^* = \frac{1}{\beta}\left\{1 + \frac{[\gamma + m_I(C)][\gamma + m_R(C)][b_S - m_S(C)]}{\gamma[m_R(C) - b_R] + m_R(C)[m_I(C) - b_I]}\right\}, \tag{A4}$$

$$p^* \equiv \frac{I^*}{N^*} = \frac{m_R(C)[b_S - m_S(C)]}{[\gamma + m_R(C)][b_S - m_S(C)] + m_R(C)[m_I(C) - b_I] + \gamma[m_R(C) - b_R]}. \tag{A5}$$

For I^*, $N^* > 0$, and, hence, $p^* > 0$, the denominator in expression (A3) must satisfy $\gamma(m_R - b_R) + m_R(m_I - b_I) > 0$, which is equivalent to

$$[b_I - m_I(C)] + \frac{\gamma}{m_R(C)}[b_R - m_R(C)] < 0. \tag{A6}$$

The two bracketed terms on the left-hand side of inequality (A6) give the per capita contribution of the infected and recovered classes, respectively, to host population growth (see eq. [A2]), with the latter weighted by the ratio γ/m_R. For this equilibrium to exist, the overall demographic contribution of the combined infected and recovered classes must be sufficiently negative for the pathogen to regulate the host/prey population.

Algebraic expressions that describe when predation should increase or decrease disease prevalence for the above model are rather messy, and so we simply present some limiting cases and illustrative examples. Assume that the infectious agent does not affect mortality rates but does reduce the fecundity of infected individuals. If there is uniform predation on all hosts (so $m_S = m_I = m_R \equiv m$), the condition for an increase in disease prevalence with predator abundance, or $dp^*/dC > 0$, is

$$\frac{\gamma}{m(C)^2}[b_S - 2m(C)] > \frac{b_S - b_I}{b_S - b_R}. \tag{A7}$$

For inequality (A7) to hold, there must be demographic costs to recovered individuals, as measured by reduced fecundity. Such a reduction in fecundity can reflect lingering physiological effects or permanent damage from having been diseased. If this is true, and if in addition there is a high rate of recovery or low initial mortality (or both), inequality (A7) can hold, so that disease prevalence increases with predator abundance. When predation is focused exclusively on infected individuals, by contrast, an increase in predation always reduces prevalence. When predation is focused instead exclusively on immune individuals, prevalence increases with increasing predation when $b_S - m_S > b_R$. So a necessary condition for predation to increase prevalence is that recovery from infection and acquisition of immunity bear significant costs, as measured by reduced fecundity.

With uniform predation, the sign of dI^*/dC is governed by

$$-\left|(b_S - m)(b_I m^2 + b_R\gamma^2 + 2b_R\gamma m) + m(\gamma + m)[\gamma(m - b_R) + m(m - b_I)]\right|, \tag{A8}$$

which is always negative. So, as discussed in the main text, prevalence and the density of infected hosts can respond differently to changes in predation.

Figure A1 shows several examples. When there is selective predation focused on infecteds alone (dashed lines in fig. A1), both prevalence and infected abundance decline with increasing predation. When there is indiscriminate predation, by contrast, and a substantial demographic cost of past infection for recovered hosts, prevalence increases with C over a range of low to moderate densities (solid line in fig. A1*A*), even though the abundance of infecteds declines (fig.

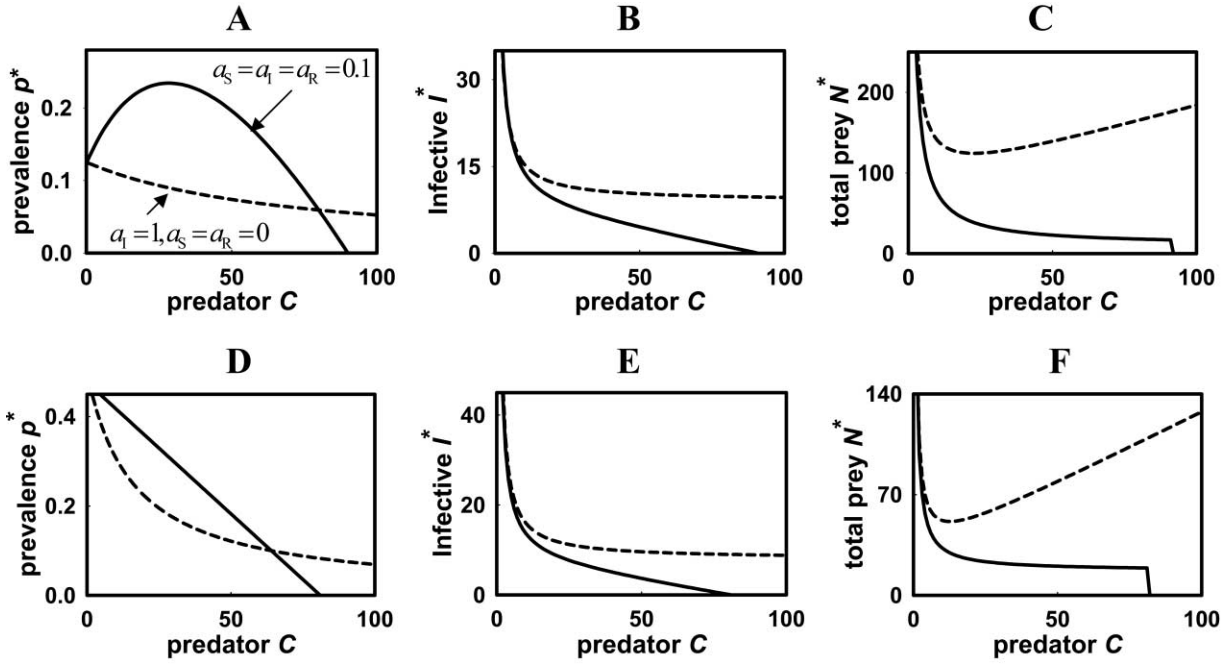

Figure A1: Equilibrium values for p^*, I^*, and N^* given by equations (A3)–(A5). It is assumed that the pathogen is regulatory. The dependence of equilibrial values on predation level C is shown for both indiscriminate predation (*solid lines*) and selective predation on the infected class (*dashed lines*). Two different sets of parameter values are shown: in *A–C*, $b_S = 10$, $b_I = 0.1$, $b_R = 1$, $m_{S0} = m_{I0} = m_{R0} = 1$, $\beta = 1$, $\gamma = 7$; in *D–F*, $b_S = b_R = 9$, $b_I = 1$, $m_{S0} = m_{R0} = 1$, $m_I = 10$, $\beta = \gamma = 1$. Linear mortality is assumed throughout. With the parameter values used in these figures, the mortality rate $m_S(C)$ of the susceptible class exceeds its birth rate b_S for $C > 87$ under indiscriminate predation, and the prey population goes extinct at this point (*solid lines*). When there is a permanent cost of having once been infected but having recovered, one can observe opposing effects of increased predation on disease prevalence and the absolute abundance of infected individuals (cf. *A* and *B*) and also cases where predators indirectly facilitate their prey, as measured by total prey/host numbers (as in *C*).

A1*B*). If there are no such demographic costs, both prevalence and abundance decline with increasing predation (solid lines in fig. A1*D*, A1*E*). An additional effect illustrated in the figure is that an increase in selective predation can increase total host numbers because of the way in which predation reduces the importance of the disease in regulating host abundance (see dashed line in fig. A1*C*). So predators can in effect be mutualists of their prey when a specialist regulatory pathogen is present. This effect does not seem to occur for indiscriminate predation.

Similar results occur for unequal predation rates. The algebra is messier, so we do not present it here.

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