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IDEAS AND PERSPECTIVES

Keeping the herds healthy and alert: implications of predator control for infectious disease

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Abstract

Predator control programmes are generally implemented in an attempt to increase prey population sizes. However, predator removal could prove harmful to prey populations that are regulated primarily by parasitic infections rather than by predation. We develop models for microparasitic and macroparasitic infection that specify the conditions where predator removal will (a) increase the incidence of parasitic infection, (b) reduce the number of healthy individuals in the prey population and (c) decrease the overall size of the prey population. In general, predator removal is more likely to be harmful when the parasite is highly virulent, macroparasites are highly aggregated in their prey, hosts are long-lived and the predators select infected prey.

Keywords

Host–parasite, predator control, predator–prey.

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INTRODUCTION

Predator control is one of the oldest and most widespread wildlife management strategies in the world (Murie 1940). Large carnivores have been persistently persecuted in a worldwide attempt to protect domestic livestock (e.g. Anonymous 1972), and predators are frequently culled to protect populations of endangered wildlife (e.g. Simon 1962; Smuts 1975; Caughley & Sinclair 1994; Anonymous 2000). However, predator control can have unexpected consequences on the abundance of targeted prey species. For example, Sih *et al.* (1985) reviewed predator-removal experiments and found 54 of 135 systems in which prey populations subsequently declined. Similarly, Cote & Sutherland (1997) found that predator removal reduced prey populations in three of 11 controlled studies.

Here the consequences of predator removal on prey populations that are regulated by infectious diseases are examined. Predators are known to preferentially select diseased prey (e.g. Schaller 1972; Moore 2002), and several experimental studies have shown that parasite-induced morbidity increases vulnerability to predation. For example, antihelminthic treatment reduced the vulnerability of snowshoe hares (Murray *et al.* 1997) and red grouse (Hudson *et al.* 1992) to predators. If predators eliminate the most infectious individuals from the prey population, they will have an outcome equivalent to quarantine – whereby infectious individuals are removed from the healthy population and thereby prevented from spreading disease.

Mathematical models were used to characterize the circumstances in which parasites can become more harmful to host species after predators have been eliminated or reduced. It was assumed that predator abundance is decoupled from prey densities, which is appropriate for any predator that is sustained by a broad assemblage of prey species. Although predators may be

most likely to capture infected prey, possible consequences are considered when predators preferentially select healthy prey. Separate scenarios for microparasites and macroparasites were evaluated to determine if the different types of parasitic infections have different consequences on host abundance, and the impact of the predator's preference for infected vs. healthy prey and the role of macroparasite aggregation were considered.

EFFECTS OF PREDATOR REMOVAL ON HOST–PARASITE DYNAMICS

A. SI models of microparasitic infection

Consider a pathogen, where infected hosts either die or survive, but recovered survivors (if any) are susceptible to re-infection. Thus the host/prey population can be divided into two cohorts: S describes the number of susceptible individuals and I contains all infected individuals. The prey are exposed to C predators; healthy individuals have a birth rate of b and a death rate of $m(C)$, such that the death rate increases with C. Infected individuals give birth at rate b', and die at rate $m'(C)$. In the absence of predation, $C = 0$ and $m'(0) > m(0)$. A 'mass action' law determines the rate of new infections, and thus the net transmission rate is βSI, where β is the transmission coefficient. Finally, infected individuals lose the infection at rate γ and re-enter the pool of susceptibles.

With these assumptions, the dynamics of the susceptible and infected subpopulations are given by (Anderson & May 1978, 1981):

$$\frac{dS}{dt} = (b - m(C))S - \beta SI + (\gamma + b')I$$

$$\frac{dI}{dt} = \beta SI - (\gamma + m'(C))I$$

For notational convenience, we define several composite parameters: $d = \gamma + m'(C)$, the total rate at which infected individuals are removed from the population; $r = b - m(C)$, the intrinsic growth rate of susceptible individuals; $e = \gamma + b'$, the contribution to the growth of the healthy subpopulation from infected individuals, either by recovery or birth; and $r' = b' - m'(C)$, the contribution made by infected individuals to population growth. We assume that $r > 0$, so that the prey population can persist in the presence of the predator and in the absence of the infection. At equilibrium, the abundances of healthy and infected hosts are $S^* = d/\beta$ and $I^* = rd/\beta(d - e)$.

These expressions provide several insights:

i. Effects of predation on parasite induced host regulation. The prey population can only be regulated at a stable equilibrium if $d > e$, or $b' < m'(C)$; thus infected prey must die at a rate exceeding their own birth rate (i.e. $r' < 0$); otherwise, the infected subpopulation would grow exponentially. Thus, predator removal (which reduces d) may make it more difficult for an ineffective parasite to regulate its host.

ii. Effects of predation on the abundance of healthy prey. If predators increase the death rate of infected prey, the expression for equilibrial abundance, $S^* = d/\beta$ shows that predation will always increase the abundance of healthy, susceptible prey. Infected prey live longer in the absence of predators and can thus infect many more susceptible prey animals (see example in Fig. 1).

iii. Effects of predation on the incidence of disease in the host population. The incidence of the disease is $q = I^*/(I^* + S^*) = r/(r + d - e)$. As r decreases with increasing C, and d increases with C, q always decreases with increasing C (as can be checked by evaluating the sign of dq/dC. In short, reduced predator pressure always leads to increased incidence of disease in the prey population (as in Fig. 1).

iv. Effects of predation on total prey abundance. The standard goal of predator removal is to increase prey numbers, but our model shows that this result is not inevitable. From (ii) above, the number of susceptible prey will decline in the absence of predation. For total host numbers to increase, the number of infected individuals must increase sufficiently to overcome the decline in healthy animals. The total host population size is $H^* = S^* + I^* = (rd + d(d - e))/\beta(d - e)$.

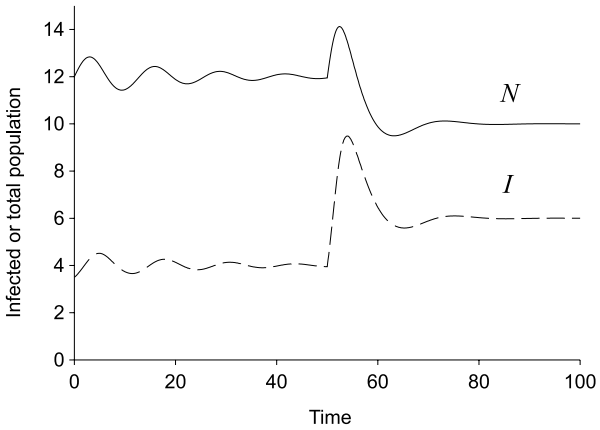

Figure 1 Impact of predator removal in a simple SI host–parasite model. The solid line is N, the total population size of the prey ($S + I$); the dashed line is I, the number of infecteds. Predator removal occurs at 50 years. The parameters are $r = 0.3$, $\beta = 0.1$, $e = 0.2$ and $d = 0.8$ until time 50, at which point d is decreased to 0.4. In this example, predator removal: (i) increases the number of infected hosts, I; (ii) increases disease prevalence, I/N and (iii) decreases the total prey population, N. Results (i) and (ii) are general features of predator–host–pathogen systems, whereas result (iii) occurs in some but not all cases (see text).

Now consider two limiting cases:

1 The predator only consumes healthy prey because it risks incidental infection from infected prey, as may happen with anthrax or bovine tuberculosis. Thus d is independent of C, and r increases with decreasing C. In this case, predator removal will increase prey abundance.
2 The disease may be so virulent that few individuals recover, and infected individuals fail to breed. In this case, the parameter e is approximately zero, and total host number is simply $N^* = (r + d)/\beta$. Assuming that r declines with C, whereas d increases with C, the net effect upon total host number is determined by the relative magnitudes of $\partial r/\partial C$ and $\partial d/\partial C$. If predators concentrate exclusively on healthy prey, predator removal increases total prey numbers (as before). If predators feed equally upon healthy and infected prey, the two mortality effects balance out, and predator removal will not influence prey population size. But if predators can more easily capture infected prey, predation increases total prey population size (Fig. 1). If predators exclusively capture infected prey, predation will increase total prey numbers whenever $d > e + \sqrt{er}$. Predation causes infected individuals to die at a younger age and, thus, to infect fewer conspecifics.

Similar effects emerge from a range of models (e.g., Hochberg *et al.* 1990). Anderson & May (1981), for instance, describe variants of our SI model that include vertical transmission, latent infection period and free-living parasite stages. In all cases, predator removal (mimicked by a decrease in density-independent mortality rates for either infected or susceptible hosts) increases disease prevalence, and there are ranges of parameter values where predator removal depresses total host abundance because of an upsurge of infection (R.D. Holt, unpublished data). Thus, an emergent 'rule-of-thumb' is that predation quite generally reduces the incidence of infection by specialist pathogens (hence, predators make for 'healthy herds') and in some circumstances predation can increase total prey population size.

B. SIR models of microparasitic infection

With pathogens such as rinderpest or distemper, infected hosts can recover and become immune to further infection. Preliminary investigation of a model that incorporated host immunity by adding the abundance of recovered hosts, R, to model (1) suggests that the basic results still apply to the density of susceptible and infected individuals in the population. Predator removal increases the number of infecteds and decreases the number of susceptibles, thereby increasing disease incidence. However, the survivors of the infection form a third segment of the population, the 'recovereds', who are released from a major source of mortality, generally leading to an overall increase in the total population (Fig. 2). A more detailed exploration of this model will be presented elsewhere.

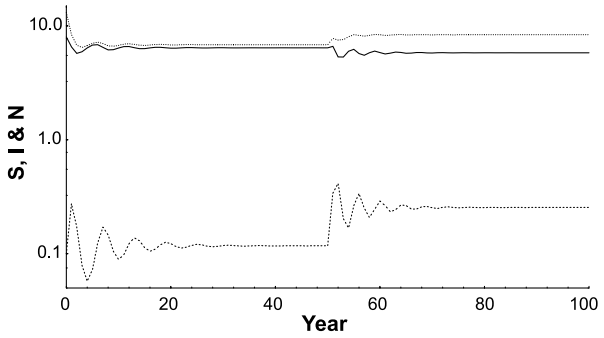

Figure 2 The effect of predator removal in an SIR model. The lowest line corresponds to infected individuals, I; the middle (solid) to susceptibles, S and the top to the total population, N. In this example, $r = 1.2$, $\beta = 0.08$, $e = 2.5$, $d = 0.7$. Predator removal occurs at 50 years, leading to an increased density of I, a decrease in S, and a net increase in N because of the release of recovered individuals from predation.

C. Macroparasites

Macroparasites tend to induce morbidity rather than mortality, thus making the host more vulnerable to predation. Macroparasites are invariably aggregated in their hosts (Shaw & Dobson 1995). As such, if predators selectively remove the most heavily infected prey, they will remove a higher proportion of the parasite population than of the host population, thereby releasing the hosts from the regulatory role of parasitism. Here the interaction between predation and parasitism in a directly transmitted macroparasite, where the parasite increases vulnerability to predation – but is not trophically transmitted to the predator – is explored. The situation where predators avoid infected hosts is also examined.

The Anderson and May 'macroparasite' model (Anderson & May 1978; May & Anderson 1978) considered the dynamics of a parasite in a host population with constant birth and death rates (designated b and d). In the absence of parasitism, it is again assumed that per capita host mortality is an increasing function of predator density, $m(C)$. Note that $m(0) = d$, the death rate in the absence of predation and of parasitism. Host mortality from parasitism is given by $m'(C)$, and $m'(0) = \alpha$ measures the impact of parasites on host mortality in the absence of predation. The adult parasite has a fecundity of λ and a mortality rate of μ. Transmission is via free-living infective stages and can be

subsumed within the saturation parameter for host abundance, H_0. With a random distribution of parasites, the dynamics of the system may be described by two equations:

$$\frac{dH}{dt} = (b - m(C))H - m'(C)P$$

$$\frac{dP}{dt} = \frac{\lambda HP}{(H + H_0)} - (d + \mu + m'(C))P - m'(C)\frac{P^2}{H}$$

The first equation describes the dynamics of the hosts, H, that are preyed upon by predators, C, and parasitized by a helminth species, P, whose changes in abundance are described by the second equation. As in the SI model, there is no acquired immunity by hosts to parasitism. By setting $dH/dt = dP/dt = 0$, we obtain expressions for the equilibrium abundance of hosts, H^*, and mean intensity of infection, P^*/H^*:

$$\frac{P^*}{H^*} = (b - m(C))/m'(C)$$

This expression implies that a decreased predation pressure will increase the mean parasite burdens, regardless of the pattern of predator preference. A more complicated expression is obtained for equilibrium host abundance:

$$H^* = \frac{H_0[(m' - m) + (d + \mu + b)]}{\lambda - [(m' - m) + (d + \mu + b)]}$$

As with the microparasites, a decrease in predator abundance, C, can lead to a decline in the host population (Fig. 3a). Reduced predation also decreases the threshold population density required for the infection to be maintained: the parasite's birth rate, λ, no longer has to be high enough to compensate for the mortality of parasites removed by predators, so $\lambda > [(m' - m) + (d + \mu + b)]$. There are limitations on this result as excessive predation can have a greater impact than parasitism (in which case the population is regulated by the predators). Thus, 'host release' is only expected when initial predation rates are low to moderate (Fig. 3b).

The results of these models are more striking when the parasites are highly aggregated within the host population. The degree of aggregation, k, was incorporated in the model by using a negative binomial distribution (following Anderson & May 1978). This parameter has an inverse relationship with aggregation such that as $k \to 0$, aggregation increases, and a smaller number of hosts is infected by a greater number of parasites. This relationship is incorporated into the model as $(k + 1)/k$ so that:

$$(\alpha + m'(C))\frac{P^2}{H} \xrightarrow{becomes} (\alpha + m'(C))\frac{P^2}{H}\frac{k+1}{k}$$

The expression for the mean parasite burden remains unchanged while the equilibrium host abundance is now given by:

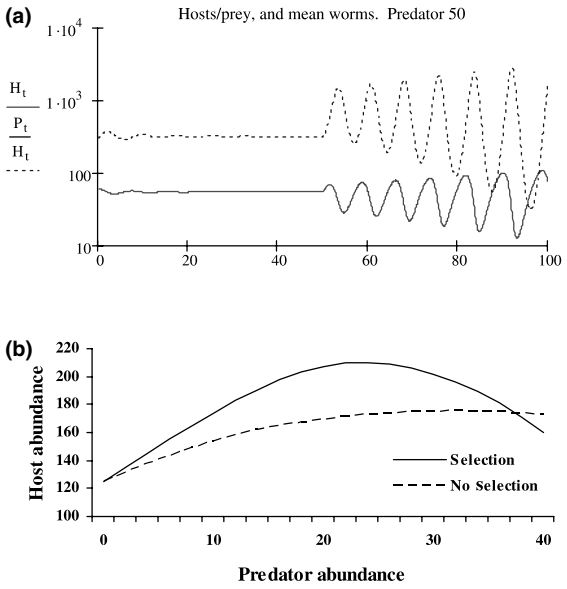

Figure 3 Effects of predator removal in a macroparasite model. (a) Changes in host population size (solid line) and mean number of parasites per host (dotted line) with predator removal at 50 years. Here, $r = 0.75$, $\beta = 0.1$, $d = 1.05$; individuals do not recover from infection, so there is no equivalent of e; remaining parameters are set according to Hudson *et al.* (1998). Note that predator removal causes population cycles in both prey and parasites. (b) Release from macroparasite regulation with increasing levels of predation. The solid line shows host abundance when predators selectively remove the most heavily infected individuals; dotted line shows abundance when predators select prey at random. At low to moderate predation levels, predators increase the size of the host population, and selective predators remove such a high proportion of the parasites that the host population rises even faster. Simulations run with a weakly aggregated distribution ($k = 1$).

$$H^* = \frac{H_0[(m' - m) + (d + \mu + b)\frac{k+1}{k}]}{\lambda - [(m' - m) + (d + \mu + b)\frac{k+1}{k}]}$$

This means that as parasites become more aggregated in the host population, predation provides greater release from parasitic regulation, permitting the host population to rise faster. Thus if the predator selects the most heavily infected 10% of the prey population, it removes an increasing proportion of the parasite population. Theoretically, predation could drive extremely aggregated parasites to extinction by removing just a few heavily infected hosts. However, this outcome is unlikely because of various selective pressures preventing parasites from becoming excessively aggregated (Shaw & Dobson 1995).

As selective predation effectively increases parasite-induced mortality, predators will also tend to stabilize the dynamics of host populations (paralleling the effect of predation in the microparasite model). Macroparasites tend to cause morbidity, reducing the fecundity of their hosts and

weakly aggregated parasites tend to destabilize host dynamics (May & Anderson 1978, Dobson & Hudson 1992). Models that include a generalist predator with a constant rate of predation suggest that increased predation will reduce the destabilizing impact of the parasite on host fecundity and thereby stabilize host dynamics (Hudson *et al.* 1992). Incorporating a functional response by the predator can lead to more interesting dynamics and destabilize host dynamics even further, for example, by generating 10-year cycles in host abundance.

Predators may avoid sick hosts to escape infection by generalist parasites or by parasites that utilize predators as the next link in their life cycle. The host population will decline whenever the predator selectively captures uninfected hosts. Again the consequences can be explored by simply setting $m'(C) = \alpha$ in the macroparasite model, so that

$$\frac{P^*}{H^*} = \frac{r - m(C)}{\alpha}$$

$$H^* = \frac{H_0(d + \mu + \alpha + b - m(C))}{\lambda - (d + \mu + \alpha + b - m(C))}$$

These expressions suggest that 'fussy' predators still reduce mean parasite burdens through their impact on reducing host population size.

EMPIRICAL EVIDENCE: THE EFFECT OF PREDATION ON RED GROUSE AND THEIR PARASITES

Hudson *et al.* (1992) examined the interaction between red grouse, the parasitic nematode, *Trichostrongylus tenuis*, and their predators. Gamekeepers are employed by private estates to control predators, and the grouse populations exhibit cyclic fluctuations caused predominantly by a nematode-induced reduction in host fecundity (Hudson *et al.* 1998). Comparative data from within study areas show that predators selectively remove the heavily infected individuals from the population. If gamekeepers are removed or predators are allowed to increase, the grouse population is less likely to cycle (Hudson 1992), but the tendency to oscillate resumes after gamekeeping is resumed. Between-site comparisons show that the proportion of heavily infected grouse increases with the density of gamekeepers, an inverse (albeit indirect) estimate of predation pressure (Hudson *et al.* 1998).

DISCUSSION

Pathogens may often be more important than predation in regulating host populations. For example, herbivores in the Serengeti were held to about one-fifth of their carrying capacity by repeated outbreaks of rinderpest (Sinclair 1979) while predation only had minimal impact on these prey (Kruuk 1972 Schaller 1972). Our SI and macroparasite models show that predator removal can increase the regulatory role of parasites to the point of lowering host population size. This effect is most conspicuous when predators had selectively removed infected prey, but non-selective predation would also have been beneficial to the host population by reducing the lifespan of infected individuals. The elimination of low to moderate predation rates is generally harmful to herd health, but prey population sizes will increase when predation pressure initially exceeded the impact of parasitism.

Although the SIR models also predict that predator removal will generally reduce the number of healthy individuals, the total host population size will typically be increased, in contrast with the other models. This effect arises from rapid growth of the 'recovered' segment of the population – animals that have become immune to further infection but are still vulnerable to predation. In general, predation is beneficial if it is exceeded by parasite-inflicted mortality, but this is impossible for the class of recovered, immune hosts.

No replicated, randomized large-scale experiments have yet been performed to measure the changes in infection rate and host abundance in response to altered predation rates. But comparative data do show changes in host population dynamics in the absence of predation. Besides the red grouse study outlined above, some studies (e.g. Tapper *et al.* 1997) have shown that predator removal increases prey productivity but does not seriously affect prey population size. However, no data were available on rates of parasitic infection.

While predation will generally reduce the parasite load in the prey population, this can only translate into increased prey population size when the force of predation is lower than parasite-inflicted mortality. When diseases are host-specific, and predators are generalists, predation is likely to inflict greater mortality than parasites at low prey population densities (e.g. Tegner & Levin 1983; Lafferty & Kushner 2000). Specialist diseases are expected to decline at low host densities, while generalist predators can be sustained by alternative prey species when the host species is scarce. This situation requires more complex models than those considered here (e.g. Hochberg 1989), but Choo *et al.* (2003) have recently shown that prey and parasite numerical responses to predation can even have consequences for the evolution of virulence.

However, many parasitic species are generalists and the force of these infections can remain high even at low densities of a particular host species. Such pathogens are particularly relevant to conservation biologists, as large populations of domesticated hosts often live near closely related taxa of wild animals (Cleaveland *et al.* 2001; Lafferty

& Gerber 2002). In cases of rare or endangered species, the impact of generalist diseases from domestic animals may often swamp the effects of predation. Thus, if predators primarily capture common prey species and reduce their level of infection, the predators may reduce the potential impact of 'spillover' infection to rare prey species. Ironically, domesticated stock is especially likely to benefit from predation on wild herbivores. Livestock husbandry is largely focused on minimizing contact with wild carnivores, but it is often difficult to protect stock from multi-host pathogens. By removing infectious individuals from the wildlife population, predators not only reduce the force of infection in wildlife, but indirectly reduce the impact of disease on the domesticated species.

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