Selective Predation and Productivity Jointly Drive Complex Behavior in Host-Parasite Systems

Spencer R. Hall,[1,*] Meghan A. Duffy,[2,†] and Carla E. Cáceres[1,‡]

1. School of Integrative Biology, University of Illinois at Urbana-Champaign, Urbana, Illinois 61801;
2. Kellogg Biological Station and Department of Zoology, Michigan State University, Hickory Corners, Michigan 49060

Submitted April 12, 2004; Accepted August 12, 2004; Electronically published November 16, 2004

Online enhancements: appendix, tables.

abstract: Successful invasion of a parasite into a host population and resulting host-parasite dynamics can depend crucially on other members of a host's community such as predators. We do not fully understand how predation intensity and selectivity shape host-parasite dynamics because the interplay between predator density, predator foraging behavior, and ecosystem productivity remains incompletely explored. By modifying a standard susceptible-infected model, we show how productivity can modulate complex behavior induced by saturating and selective foraging behavior of predators in an otherwise stable host-parasite system. When predators strongly prefer parasitized hosts, the host-parasite system can oscillate, but predators can also create alternative stable states, Allee effects, and catastrophic extinction of parasites. In the latter three cases, parasites have difficulty invading and/or persisting in ecosystems. When predators are intermediately selective, these more complex behaviors become less important, but the host-parasite system can switch from stable to oscillating and then back to stable states along a gradient of predator control. Surprisingly, at higher productivity, predators that neutrally select or avoid parasitized hosts can catalyze extinction of both hosts and parasites. Thus, synergy between two enemies can end disastrously for the host. Such diverse outcomes underscore the crucial importance of the community and ecosystem context in which host-parasite interactions occur.

Keywords: Allee effects, catastrophes, host/parasite interactions, invasion, persistence, selective predation.

* Corresponding author; e-mail: srhall@uiuc.edu.

† E-mail: meghan.duffy@kbs.msu.edu.

‡ E-mail: caceres@life.uiuc.edu.

Am. Nat. 2005. Vol. 165, pp. 70–81. 0003-0147/2005/16501-40397$15.00.

In general, predator selectivity and predation intensity can dramatically shape community structure and ecosystem properties (Zaret 1980; Sih et al. 1985). Predation becomes particularly interesting in host-parasite systems because predators often preferentially cull parasitized hosts (Hudson et al. 1992; Murray et al. 1997). When predators prefer to eat infected hosts, incidence of parasitism typically drops (Hudson et al. 1992; Arneberg et al. 1998; Packer et al. 2003). Additionally, predation intensity itself can strongly alter population dynamics of hosts and parasites (Ives and Murray 1997; Hudson et al. 1998; Packer et al. 2003; Dwyer et al. 2004). When sufficiently selective and dense, predators may even prevent successful invasion of parasites into host populations (Duffy et al., forthcoming). Here, we study how invasion success, persistence, and dynamics of parasites in host systems depend on the nature and intensity of selective predation as modulated by ecosystem productivity.

In most theoretical studies of host-parasite-predation interactions, predator behavior is simplified and isolated from an ecosystem context. Typically, host-parasite models represent feeding behavior of predators with a linear, mass-action functional response (Hudson et al. 1998; Packer et al. 2003; but see Ives and Murray 1997; Dwyer et al. 2004). However, the feeding rate of real predators often saturates (Case 2000). In food chain models, the dynamical consequences of this saturating response become accentuated at high ecosystem productivity (e.g., through the paradox of enrichment), but host-parasite-predation models do not typically examine these links. Without predators, epidemiological models predict that increases in productivity can modify host-parasite dynamics in unpredictable ways (Lafferty and Holt 2003). It seems important, then, to consider potential synergies between predator behavior and productivity in host-parasite-predator models.

Our goal was to explore the range of theoretical behaviors predicted from interactions between hosts, microparasites, and predators. Although it was intentionally kept simple, we built our model around key biological elements of a microparasite-zooplankton-fish system. *Daphnia dentifera*, the host, commonly dominates zooplankton grazer

communities of northern temperate lakes in the midwestern and northeastern United States (Tessier and Woodruff 2002). Daphniid populations reproduce continuously and facultatively asexually (thus meeting several assumptions of continuous-time models), and their birthrates are density dependent. These hosts are susceptible to several bacterial and fungal microparasites. Infected *Daphnia* rarely recover, and parasites usually kill their hosts and can greatly reduce host reproduction (Ebert et al. 2000). Additionally, in many midwestern lakes, bluegill sunfish prey on *Daphnia dentifera*. These visually oriented predators also selectively prey on infected hosts because many parasites turn normally translucent *Daphnia* opaque, making them more visible (Duffy et al., forthcoming). Feeding rate of bluegill saturates at moderate densities of *Daphnia* (Mittelbach 1981), and the generation time of the fish predator greatly exceeds that of its zooplankton prey.

We incorporated several features of this natural history into a standard host-microparasite framework. We added density-dependent regulation to the host (Begon and Bowers 1995; Greenman and Hudson 1997), and we compared predators with linear (mass action) and saturating (type II) feeding behavior (Case 2000). We assumed that dynamics of the fish predator occur so slowly that we could break the three species system into its slow and fast components (Ludwig et al. 1978; Rinaldi and Scheffer 2000; Scheffer et al. 2000). Thus, predator density appears as a parameter, not a variable, that is varied with the predator's preference for parasitized hosts (selectivity). The effect of predators is modulated by ecosystem productivity, which we equated with relaxation of the strength of density dependence on the host's birthrate. Interplay between these three factors yields a diverse array of biologically relevant behaviors, including oscillations, Allee effects, and catastrophic extinction of parasites and hosts. Thus, predators may play an even more critical role in host-parasite dynamics than previously thought.

Theory

We build on predator-prey-like epidemiological models that track population dynamics of susceptible (S) and infected (I) hosts (Anderson and May 1991). We first consider behavior of the host-microparasite system without predation; then, we add a predator with a linear functional response. The two models produce simple behavior and yield analytically tractable results (Packer et al. 2003). Here, these well-known findings (presented in app. A in the online edition of the *American Naturalist*) provide contrast to those from the new model with selective, saturating predation. We explore behavior of this more complex model along gradients of predator densities, selective foraging, and productivity using analysis of nullclines and bifurcations with simulations.

We represent changes in host density as a balance between self-regulation (negative density dependence) and regulation by parasites (Begon and Bowers 1995). To this structure, we add selective predation, yielding the system (see also tables 1 and B1, which is in the online edition of the *American Naturalist*):

$$\frac{dS}{dt} = bS[1 - c(S + I)] - dS - \beta SI - f_S(S, I, P), \quad (1a)$$

$$\frac{dI}{dt} = \beta SI - (d + \alpha)I - f_I(S, I, P). \quad (1b)$$

Change in population size of susceptible hosts (dS/dt; eq. [1a]) balances density-dependent production and losses. Growth rate of the host depends logistically on density as a function of maximal per capita birthrate (b) and strength of density dependence on birthrates (c). Productivity of the system for the host increases as this density dependence relaxes (i.e., as $1/c$ increases). Only susceptible hosts (S) reproduce (the bS term), a reasonable assumption for many parasites of *Daphnia* (Ebert et al. 2000). Infected hosts (I) do consume resources and therefore negatively affect host production. Losses from the susceptible host class come from three sources: density-independent mortality (at per capita rate d), infection by parasites through "pseudo–mass action" contact with infected hosts (the βSI term at rate β; Regoes et al. 2003), and losses to predation ($f_S[S, I, P]$). Change in population size of infected hosts (dI/dt; eq. [1b]) reflects the balance between gains from infection of susceptible hosts (βSI) and three sources of mortality: density-independent mortality (at rate d, equal to that of susceptible hosts), parasite-induced mortality (at rate α), and predation (at rate $f_I[S, I, P]$). We assume that infected hosts do not recover, lose infection, or become immune (Ebert et al. 2000; table B1).

To introduce predation, we first consider the commonly used linear form. If a predator's handling time for susceptible (T_S) and infected hosts (T_I) is 0, the predation terms follow a linear, mass-action functional response:

$$f_S(S, I, P) = a_S CS, f_I(S, I, P) = a_S \theta CI, \quad (2)$$

where C is the density of predators; a_S is the attack rate on susceptible hosts; and θ is the selectivity of the predator, the ratio of attack rates on infected to susceptible hosts (a_I/a_S). Thus, $\theta > 1$ implies that predators prefer infected hosts, $\theta = 1$ implies no prey preference at all, and $0 < \theta < 1$ means that predators prefer susceptible hosts to infected hosts. Avoidance of parasitized prey is highly unlikely in *Daphnia*-parasite-fish systems (Duffy et al., forth-

Table 1: Variables and parameters used in models of host-parasite-predator interactions

Variable/parameter	Units	Definition	Default value/range
a_j	Day^{-1}	Attack rate on prey j	…
b	Day^{-1}	Maximum birthrate of susceptible hosts	.4[a]
C	Number L^{-1}	Predator density	…
c	$(\text{Number L}^{-1})^{-1}$	Strength of density dependence	1/25–∞[a]
d	Day^{-1}	Loss rate not from predation or virulence	.05
$f_I(S, I, P)$	Day^{-1}	Predation rate on infected hosts	…
$f_S(S, I, P)$	Day^{-1}	Predation rate on susceptible hosts	…
h_S	Number L^{-1}	Half-saturation constant of predators for susceptible hosts	1[b]
I	Number L^{-1}	Density of infected host class	…
P	Number L^{-1} day^{-1}	Predation intensity (C/a_S or C/h_S)	0–2
S	Number L^{-1}	Density of susceptible host class	…
t	Day	Time	…
T_j	Day	Handling time of predator for prey j	…
α	Day^{-1}	Virulence (additional death rate due to infection)	.05[c]
β	Number L^{-1} day^{-1}	Transmission rate	.05[d]
γ	…	Ratio of handling times (T_I/T_S)	1[e]
θ	…	Predator preference for infected hosts (a_I/a_S)	.05–20

[a] In figures, range of $1/c$ is 0–25. Source: Tessier and Woodruff (2002).
[b] Estimated from Mittelbach (1981) for bluegill sunfish feeding on *Daphnia*.
[c] Source: M. A. Duffy and A. J. Tessier, unpublished data.
[d] Midrange estimate from Ebert et al. (2000).
[e] Assumes that handling time does not differ between susceptible and infected host classes.

coming). Yet, as shown in "Predators with a Saturating Functional Response," this case still yields theoretically interesting results. Below, we combine the $a_S S$ terms into a predation intensity term (P); P is a rate. We treat P as a parameter because P changes on a much slower timescale than S-I dynamics (May 1977; Ludwig et al. 1978; Rinaldi and Scheffer 2000; Scheffer et al. 2000; table B1).

Preliminaries: No Predation and Predation with a Linear Functional Response

Without predation, dynamics between susceptible and infected hosts are simple (see app. A). In a system without the parasite, equilibrial density of the susceptible host (S^*) increases linearly with productivity ($1/c$). Once a threshold density of susceptible hosts is reached, the parasite can invade. At this threshold, the basic reproductive ratio of the parasite (R_0) equals 1 (and the parasite invades when $R_0 > 1$). Coexistence of the host and parasite, when feasible, is stable, and S^* becomes decoupled from productivity. Instead, S^* becomes solely determined by traits of the infected host class (i.e., by parasites). Meanwhile, density of infected hosts becomes a monotonically increasing function of productivity.

A predator with a linear functional response quantitatively changes the density of susceptible and infected hosts and the parasite's ability to invade a host population. Otherwise, it does not fundamentally alter the qualitative dynamics of the host-parasite system (see app. A for details). When parasites and hosts coexist, density of susceptible hosts becomes a positive function of predation intensity (P) and selectivity (θ), while density of infected hosts decreases as P and θ increase (see also Packer et al. 2003). Additionally, at constant predator density (C), selectivity decreases the maximum predation intensity that infected hosts can withstand ($\hat{P}$ in app. A). This predation intensity constraint resembles the P^* of keystone-predation models (Grover 1997). Furthermore, at a given predation intensity, highly selected parasites require higher system productivity to invade and persist than parasites that predators neutrally select or avoid. However, host-parasite dynamics remain stable (see app. A for stability analysis).

Predators with a Saturating Functional Response

Although the assumption of linear, mass-action predation is mathematically convenient, predators often become satiated in nature. Thus, we can modify the predation terms $f_S(S, I, P)$ and $f_I(S, I, P)$ using a classical multiple-prey, type II functional response (Case 2000). With some manipulation, these predation terms become

$$f_S(S, I, P) = P\left(\frac{S}{h_S + S + \theta\gamma I}\right),$$

$$f_I(S, I, P) = P\left(\frac{\theta I}{h_S + S + \theta\gamma I}\right), \quad (3)$$

where

$$P \equiv \frac{C}{T_S},\ h_S \equiv \frac{1}{a_S T_S},\ \gamma \equiv \frac{T_I}{T_S},\ \theta \equiv \frac{a_I}{a_S}. \qquad (4)$$

Here, h_S is the predator's half-saturation constant for susceptible hosts, γ is the ratio of handling times of infected (T_I) to susceptible (T_S) hosts, θ remains the ratio of attack rates (as above), and C again is the density of predators. Note that predation intensity (P) now more naturally scales with feeding rate at saturation (which is $1/T_S$; Case 2000), not attack rate. Here, a selective predator attacks infected hosts at a different rate than susceptible hosts but does not handle these prey differently. This assumption likely applies to our *Daphnia*-parasite-fish system, so we set $\gamma = 1$. However, this restriction could be relaxed if predators more easily handled infected hosts once they have attacked them.

Two components of these saturating predation terms introduce major changes to host-parasite dynamics. First, even in systems without parasites, predators can create alternative stable states for the host (May 1977; see app. A for details). These alternative stable states emerge because both an upper, stable equilibrium and a lower, unstable (saddle) equilibrium can coexist at intermediate predation intensity, P (fig. 1*A*). When these two equilibria coexist, the susceptible host experiences an Allee effect; if the susceptible host's initial population size starts or gets pushed below the saddle equilibrium, the predator will drive the host extinct (fig. 1*A*). Thus, the predator with a multiple-prey, type II functional response introduces minimum invasion sizes of susceptible hosts, and this invasion threshold increases with increasing P (fig. 1*A*). These Allee effects disappear at lower P (through a "transcritical bifurcation"; table B2 in the online edition of the *American Naturalist*), but the host cannot persist with overly high P (past a "fold bifurcation"; table B2; fig. 1*A*). Similar Allee effects for the parasite arise in the full host-parasite model. Second, the multiple prey functional response implies that predation rate on infected individuals saturates when predators become satiated, but predation rate depends on density of both susceptible (S) and infected (I) host classes. In particular, feeding rate on I is a negative function of S density (moving along isoclines of fig. 1*B*). Yet, feeding rate on I increases when predators prefer to eat infected hosts (fig. 1*B*).

The combination of a predator with a multiple-prey, type II functional response and very high selectivity on infected hosts produces complex but biologically relevant behavior in the host-parasite system. The model's behavioral repertoire is readily captured by a bifurcation diagram that delineates qualitative changes of the host-parasite system in relevant parameter space (see table B2 for descriptions of four key bifurcations and app. A for algebraic details). Here, these parameters are predation intensity (P) and the proxy for productivity ($1/c$). We describe four key regions of the bifurcation diagram containing points A–

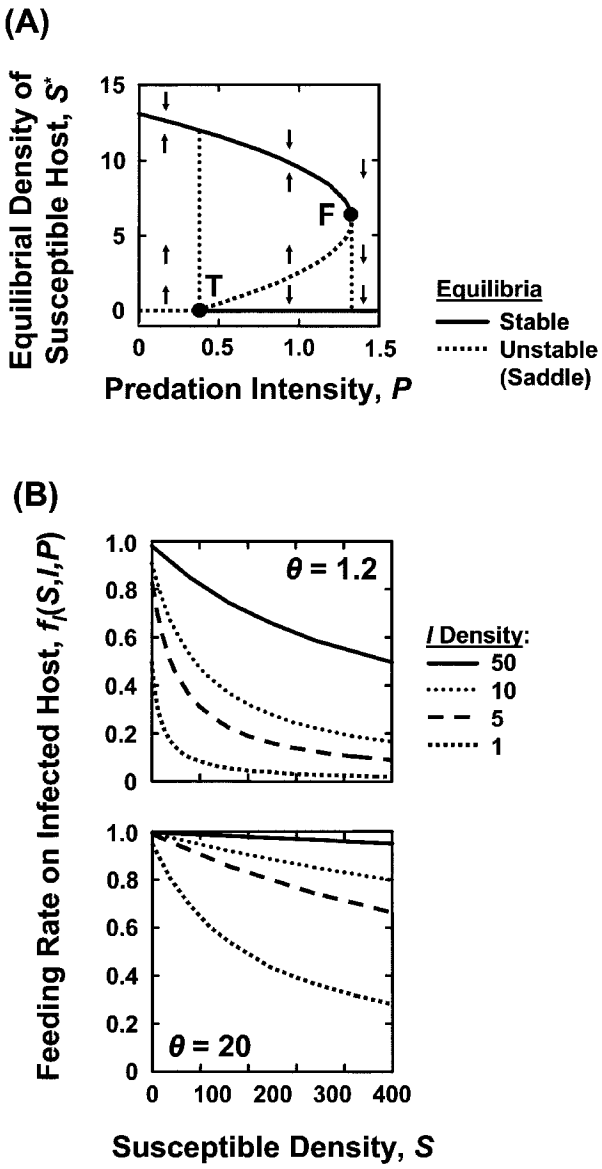

Figure 1: Two key components of the *S-I* model with saturating predation. *A*, In a system without parasites, predators can introduce alternative stable states for the host. At low predation intensity, a single stable equilibrium exists. As predation intensity increases past a transcritical bifurcation (point *T*), an additional equilibrium (a saddle) separates two alternative stable states and creates an Allee effect for the host. Predators always drive their prey extinct when P exceeds a fold bifurcation (point *F*). *B*, In the multiple-prey, type II functional response, feeding rate on infected hosts depends both on selectivity (θ) of predators and on density of susceptible and infected hosts. Other parameters are as in table 1.

D in detail using phase plots of nullclines and simulations (figs. 3, 4).

At low predation intensity, the *S*-*I*-predator system either behaves like the linear version of the model or begins to oscillate. In the region containing point *A* (fig. 2), susceptible and infected host nullclines cross only once at a stable, high *I*–low *S* equilibrium (fig. 3*A*). The point is stable because parasites exert most of the control on *S*-*I* dynamics, and the system behaves qualitatively like the case with a predator feeding with a linear functional response. At higher predation intensity and productivity (point *B*, fig. 2), the sole interior equilibrium becomes unstable (through a Hopf bifurcation; table B2). There, *S* and *I* populations begin to oscillate in stable limit cycles (fig. 3*B*), in part due to an interaction of predators and parasites. As the parasite begins to lose control of the *S*-*I* system due to mortality from selective predators, the host periodically escapes, starting a cycle. When density of susceptible hosts becomes high, parasites can infect them quickly on a per capita basis because infection rate is high. As a result, the parasite quickly spreads, and *S* decreases while *I* increases. However, when *I* is high and *S* is low, parasites "starve" because they have depleted their resource. Additionally, predation rate on *I* becomes quite high (fig. 1*B*), so predation and resource depletion jointly cause the parasite to decline. At low *S* and *I*, susceptible hosts can increase rapidly because much of their carrying capacity remains unused. Once abundant, predators cannot completely control susceptible hosts because predators become satiated. Hence, the cycle begins again. This cycling requires a predator with a multiple-prey, type II functional response (app. A).

Parasites can experience Allee effects when increasing predator control creates an additional interior equilibrium. This new, low *I*–high *S* equilibrium arises in the parameter space containing point *C* (fig. 2) because nullclines for susceptible and infected hosts cross twice (above the interior *S*-*I* transcritical bifurcation; table B2; fig. 2). However, this new interior equilibrium is a saddle point, and it separates the stable, high *I*–low *S* equilibrium from a stable, *S*-only boundary equilibrium (fig. 3*C*). As a result, this interior saddle point controls an Allee effect for the parasite, which in turn implies that initial parasite invasion must exceed a threshold density for parasites to successfully invade the host-predator system. If the initial invasion size of infected hosts exceeds this threshold, it starts within the domain of attraction of the interior equilibrium (i.e., point *1*, *white area*, fig. 3*C*). If the invasion of infected hosts starts in the domain of attraction of the boundary, *S*-only equilibrium (*gray area*, fig. 3*C*), predators will drive the parasite extinct instead. Interestingly, the initial infection size can be too large to permit coexistence of all three species (e.g., point *2*, fig. 3*C*). In this case, a large population oscillation (driven by parasite-predator interactions) does not enter the domain of attraction of the stable, interior equilibrium.

As predator control increases further, parasites cannot persist permanently in a system, even if they can successfully invade. As *P* increases from point *C* in the bifurcation diagram (fig. 2), the host-parasite system starts to oscillate

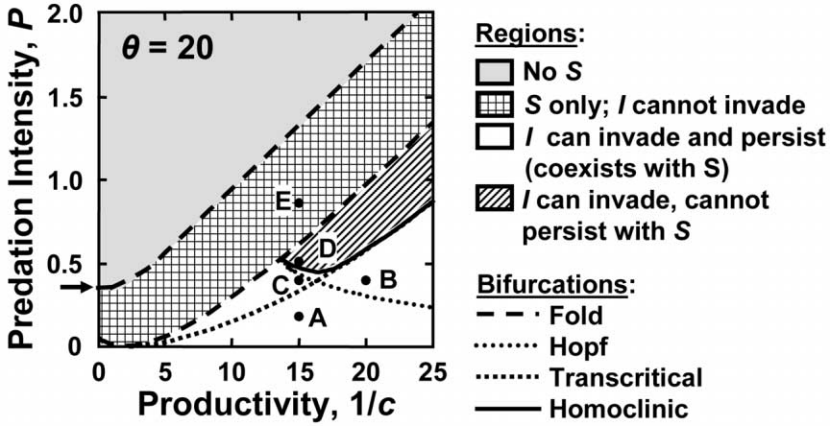

Figure 2: Bifurcation diagram of the *S*-*I* model with saturating predation and high selectivity ($\theta = 20$) in productivity ($1/c$)–predation intensity (P) parameter space. Fold bifurcations separate regions in which susceptible hosts (S) cannot persist with the predator (*gray*), the infected host (I) cannot persist with the susceptible host and predator (*hatched*), and both host classes might persist with the predator (*white* and *striped* regions). At levels of predation intensity above the arrow along the P-axis, multiple boundary equilibria emerge (through a transcritical bifurcation; see app. A for details). Points *A*–*D* are examined closely in figure 3. Other parameters are as in table 1.

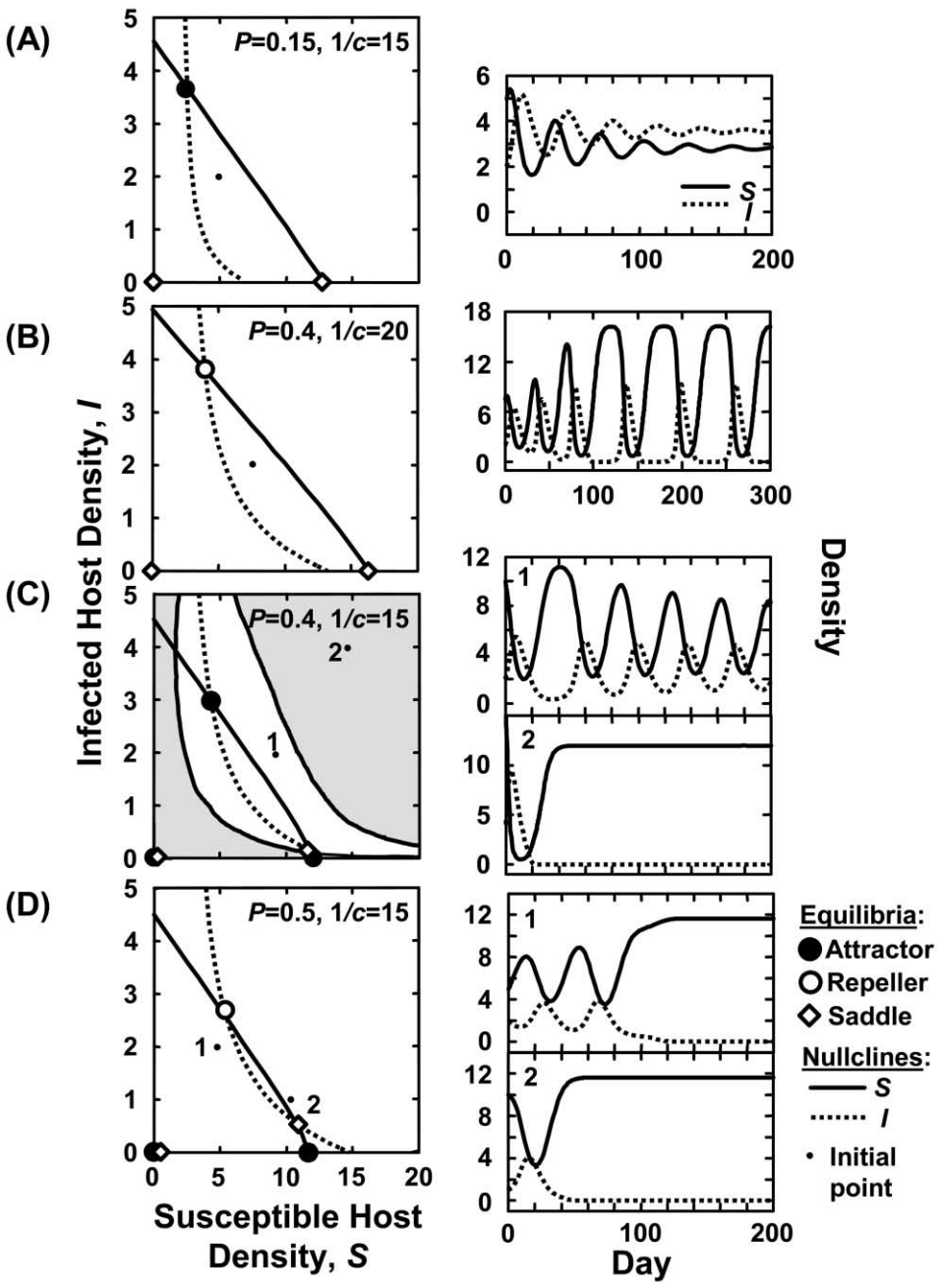

Figure 3: Four major behaviors of the *S-I* model with saturating predation and high selectivity ($\theta = 20$), as illustrated by nullclines in *S-I* state space and accompanying selected simulations. The letters correspond to points *A*–*D* on the associated bifurcation diagram (fig. 2). *A*, At low *P*, a single, stable interior equilibrium emerges, and the boundary equilibrium becomes a saddle. *B*, At higher $1/c$ and *P*, a Hopf bifurcation destabilizes this single equilibrium; stable limit cycles (oscillations) arise. *C*, As *P* increases further from point *A*, two interior equilibria emerge. The low *I*–high *S* saddle separates a stable high *I*–low *S* equilibrium from the stable boundary equilibrium. Thus, multiple stable states coexist, and Allee effects occur. Trajectories starting in the white region (e.g., point *1*) head to the interior equilibrium, while trajectories initiating in the shaded region (e.g., point *2*) lead to the boundary equilibrium. *D*, Once the homoclinic bifurcation emerges, trajectories starting near the unstable interior equilibrium eventually jump to the stable boundary equilibrium. Points starting close to the repeller (e.g., point *1*) may cycle several times more than those starting farther away (e.g., point *2*). All other parameters are at default values (table 1).

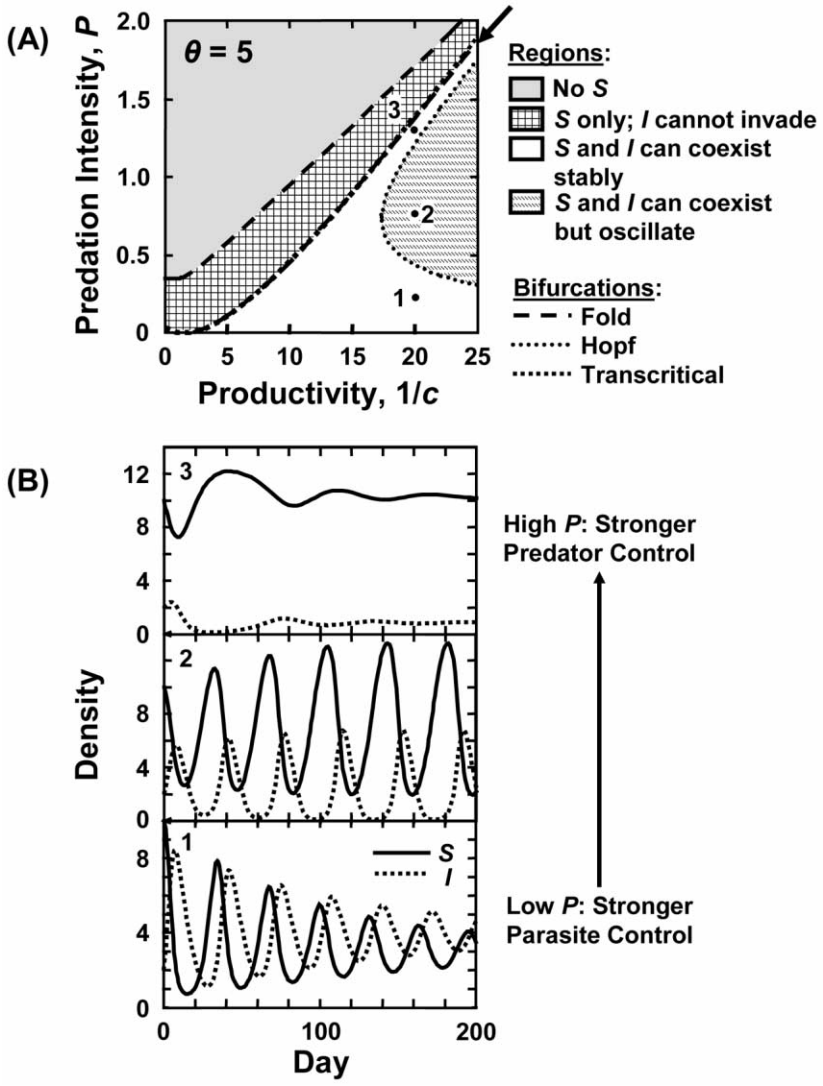

Figure 4: Dynamics of the *S-I* model with saturating predation and moderate selectivity ($\theta = 5$). *A*, Bifurcation diagram in productivity ($1/c$)–predation intensity (P) parameter space. In the coexistence region, a feasible interior saddle emerges only at productivity (*arrow*). A stable coexistence region surrounds a region of oscillating host-parasite dynamics. *B*, At $1/c = 20$, the *S-I* system transitions from damped oscillations (point *1*), to stable limit cycles (point *2*), and then back to damped oscillations (point *3*) along a gradient of increasing predation intensity. All other parameters remain at default values (table 1).

around the high *I*–low *S* equilibrium because a Hopf bifurcation is crossed (fig. 2). With further increases in predation past a critical threshold, these oscillations invariably shunt the *S-I* system toward the parasite-extinction point (e.g., the boundary, *S*-only equilibrium). Biologically, the parasites severely overexploit their resources, and then predators prevent parasites from rebounding. Mathematically, once the upper interior equilibrium becomes unstable, the lower, interior saddle separates these stable limit cycles from the stable boundary equilibrium. As P increases just a small amount more, the stable limit cycles grow until they run into this interior saddle, forming a global homoclinic bifurcation (fig. 2; table B2). If P increases above the homoclinic bifurcation (e.g., to point *D*, fig. 2), almost all *S-I* trajectories ultimately enter the domain of attraction of the stable boundary equilibrium (fig. 3*D*). Thus, a mathematical "catastrophe" occurs, and predators drive parasites extinct in a sudden, fast dynamic jump after one or more oscillations (depending on initial conditions; cf. points *1* and *2* of fig. 3*D*). This behavior resembles a "paradox of enrichment" for the parasite, although extinction occurs deterministically here. This

catastrophic behavior can resemble that in the system with alternative stable states (e.g., point *2*, fig. 3*C*), but the mathematical mechanisms differ between these cases.

At even higher predation (e.g., the region containing point *E*, fig. 2), the parasite can neither successfully invade nor coexist with a host and predator. In this case, an interior fold bifurcation is crossed, and the host and parasite nullclines do not even intersect. This state of the system superficially resembles the linear predation case once the critical predation density is reached (app. A, eq. [A8]) except that multiple, stable boundary (*S*-only) equilibria now exist (as in fig. 1*A*). Eventually, extremely high predation can eliminate susceptible hosts altogether (at the boundary fold bifurcation of fig. 2).

Predator selectivity and its interaction with productivity play a major role in determining diversity and relative importance of these complex, nonlinear behaviors. In contrast to the highly selective example ($\theta = 20$; figs. 2, 3), parasites can successfully invade host populations at higher predation intensity when the predator is intermediately selective (e.g., $\theta = 5$; fig. 4). This insight resembles findings from the linear model (app. A). Furthermore, in this case, when predation pressure just permits invasion of the parasite, *S*-*I* dynamics are stable (fig. 4). Such stability contrasts with the catastrophes predicted at higher selectivity (e.g., fig. 3*D*). Additionally, Allee effects for the parasite and multiple equilibria remain unlikely until productivity becomes quite high (they begin to emerge at the arrow in fig. 4).

At intermediate selectivity ($\theta = 5$), host-parasite (*S*-*I*) dynamics are either stable or will oscillate, depending on the degree of predator versus parasite control on the system and productivity. Once parasites successfully invade, dynamics of hosts and parasites are always stable at low productivity (fig. 4*A*). At higher productivity (e.g., $1/c = 20$), both *S*-*I* dynamics qualitatively shift along a gradient of predation pressure. At low *P*, parasites predominately control hosts. As a result, a stable, high *I*–low *S* equilibrium governs *S*-*I* dynamics (point *1*, fig. 4*B*). As predator intensity increases, parasites lose control of hosts, but predators cannot completely control parasites either (because of satiation), so the system oscillates (point *2*, fig. 4*B*). As *P* increases further, predator control dominates. Therefore, a stable equilibrium governs *S*-*I* dynamics again, with higher *S* and lower *I* density predicted (point *3*, fig. 4*B*).

One might guess that a further decrease in predator selectivity should continue to stabilize *S*-*I*-predation dynamics. Surprisingly, when the predator is nearly neutrally selective (e.g., $\theta = 1.2$), a new catastrophic behavior emerges; predation may drive both hosts and parasites extinct. Thus, when predators are neutrally selective, a parasite invasion can lead to extinction of a host that otherwise coexisted stably with its predator. The associated nullclines in this region (above the homoclinic bifurcation; fig. 5*A*, 5*B*) help explain this behavior. The sole interior equilibrium is a repeller, and both *S*-only equilibria are saddles. Oscillating *S*-*I* trajectories eventually crash into the smaller *S*-only saddle and head toward the only stable attractor in the system—an extinction point for both *S* and *I* (fig. 5*B*). If predation pressure becomes higher, this catastrophe may occur fairly rapidly; if predation pressure drops, eventual collapse of the *S*-*I* system may take much longer (point *1* vs. *2* of fig. 5*A*, 5*C*). If predators avoid eating parasitized hosts (e.g., $\theta = 0.05$), the situation becomes worse for persistence of the *S*-*I* system because predators push it toward extinction at even lower productivity and predation intensity (fig. 5*D*). These catastrophic outcomes depend on productivity because at lower productivity, parasites and hosts can coexist stably with a predator (fig. 5*A*, 5*D*).

Discussion

Successful invasion of a parasite into a host population and the resulting parasite-host dynamics can depend crucially on other members of a host's community. Theoretical and empirical studies have shown that a host's competitors can greatly impact invasion success of a parasite (Holt and Pickering 1985; Bowers and Turner 1997; Greenman and Hudson 1997; Gilbert et al. 2001; Tompkins et al. 2002; Holt et al. 2003). Now, our theoretical results show how predators induce complex dynamics in otherwise simple host-parasite interactions. By incorporating realistic, saturating (type II) foraging behavior of the predator, we found that a parasite's ability to invade and persist in a host population depends sensitively on intensity of predation pressure, selectivity of the predator, and productivity.

For instance, highly selective predators can introduce Allee effects and alternative stable states for the parasite. As a result, parasites not only require a critical density of hosts to invade (i.e., the R_0 threshold in many host-parasite models) but also a minimal invasion size of infected individuals. If the invasion does not exceed this minimal size (or if it exceeds it by too much), the invasion will ultimately fail. Allee effects can arise in host-parasite systems because of nonlinear transmission, immunological response of the host, local spatial interactions, and stochasticity (Holmes 1997; Gubbins et al. 2000; Regoes et al. 2002). We show that a third species, predators, can also induce them. However, the model shows that successful invasion does not guarantee persistence of the parasite. Particularly at high productivity, parasites can overexploit their host resources (causing oscillations that selective predators exacerbate), but then predators prevent recovery

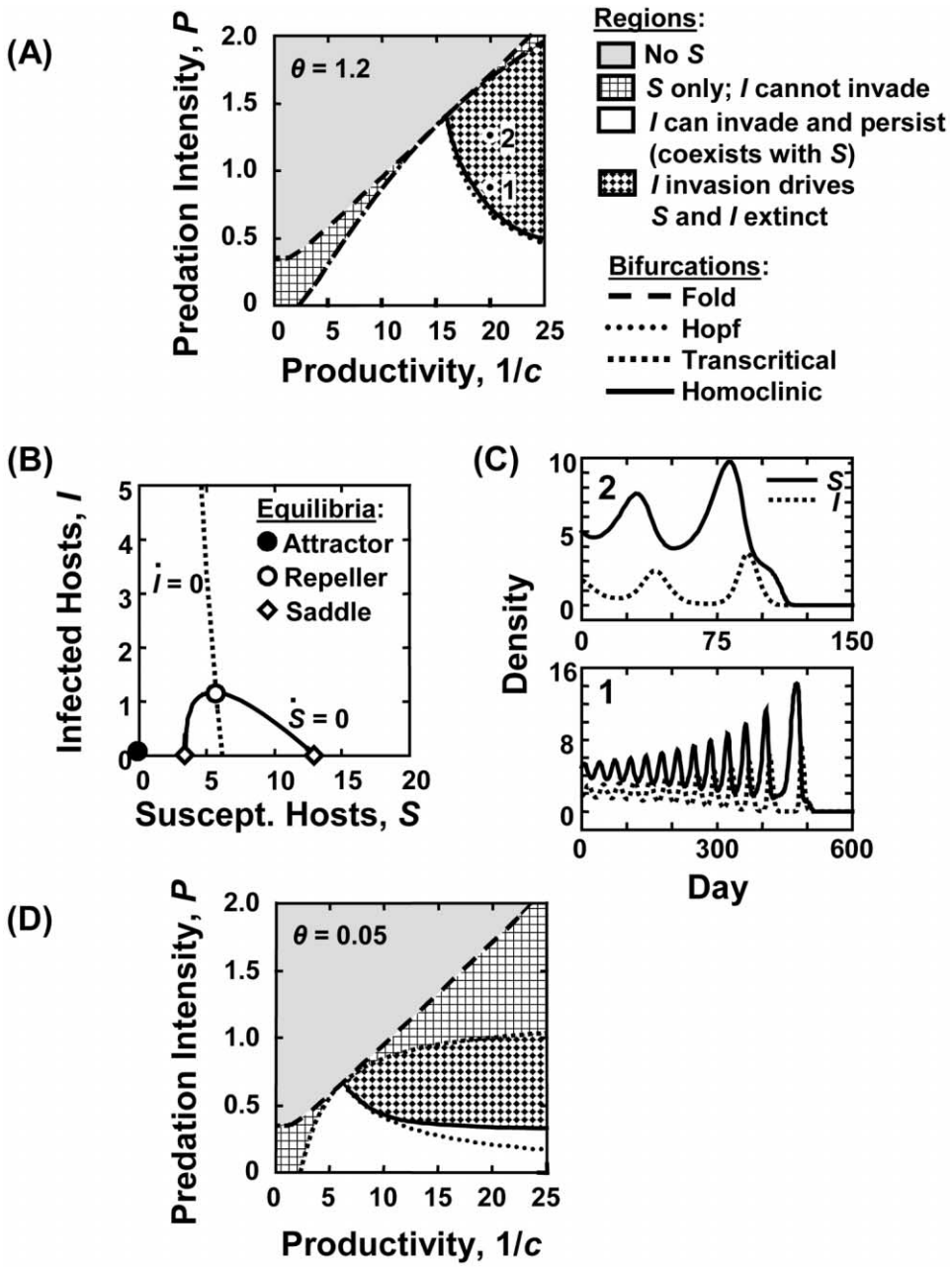

Figure 5: Dynamics of the *S*-*I* model with saturating predation where the predator neutrally selects ($\theta = 1.2$) or avoids parasitized hosts ($\theta = 0.05$). *A*, Bifurcation diagram in productivity ($1/c$)–predation intensity (P) parameter space with a neutrally selective predator. In this case, the homoclinic bifurcation emerges as a limit cycle collides with the smaller boundary (*S*-only) equilibrium, which is a saddle. *B*, Nullclines of the system at point *2* in the bifurcation diagram. The sole stable equilibrium is a mutual extinction state. *C*, Simulations at two points above the homoclinic bifurcation. The system takes a longer time to reach the mutual extinction point when predation intensity is lower than when it is higher. *D*, Bifurcation diagram in $1/c$-P parameter space with a predator that avoids parasitized hosts.

of the parasites. Thus, a parasite may successfully invade but then catastrophically disappear from an ecosystem in a way resembling the paradox of enrichment. Perhaps these catastrophes can account for collapses of macroparasites of grouse, even when the per capita net reproductive rate of the parasite would suggest persistence (Hudson et al. 1998).

At the other extreme, interactions with both its predator

and parasite may propel an otherwise persistent host toward extinction. We show that parasite invasion can drive its host to catastrophic extinction in the presence of a neutrally selective predator or one that prefers uninfected hosts. This parasite-driven extinction does not require explicit representation of space (as does the other known example of parasite-driven extinction; Boots and Sasaki 2002). Our result seems counterintuitive at first; why should predators that avoid parasitized hosts drive both host and parasite extinct? It stems again from overexploitation of hosts by the parasite and density-dependent predation on nonreproductive, infected hosts. This time, a predator-driven Allee effect in the susceptible host itself becomes crucial. This behavior resembles the "enemy release hypothesis" (Elton 1958; Keane and Crawley 2002): a species may successfully invade and persist with one natural enemy (e.g., the predator) only if it leaves its other natural enemy (e.g., the parasite) behind.

Predator-induced Allee effects introduce a constraint for a parasite, but they also beg another question: What controls its invasion size in the first place? Some parasites have a free-living infective stage (Anderson and May 1991; Dobson and Hudson 1992). If this stage can survive for long intervals and act as a source of colonists, a parasite might persist through a storage effect (Chesson 2000). Alternatively, some parasites can arrest development in larval stages (Dobson and Hudson 1992). Furthermore, migration from other parasitized systems might promote local parasite persistence in environments that are otherwise inhospitable to the parasite (through a "rescue effect"; Earn et al. 1998). A metapopulation perspective has already flourished in epidemiological theory and has yielded major insight into human diseases (Hess 1996; Grenfell and Harwood 1997; Keeling and Gilligan 2000). Metacommunity approaches may naturally extend to multispecies host-parasite systems linked by dispersal. Such a perspective will likely emphasize the importance of asynchrony for parasite persistence (Earn et al. 1998). In some systems, however, seasonality may still overpower rescue effects.

Seasonal changes in temperature and predation intensity most likely shape the freshwater *Daphnia*-parasite-fish system. Duffy et al. (forthcoming) hypothesized that highly selective predation by fishes may control invasion of a bacterial parasite in temperate lake ecosystems. In several lakes, epidemics of parasites begin as host death rate drops, and these drops occur at the end of summer when temperature declines and bluegill predators move inshore. Yet, these epidemics end after only a single oscillation. Our model readily predicts that a catastrophe and/or an Allee effect could terminate the epidemics after one oscillation.

Although designed around a *Daphnia*-microparasite-fish system, the model explored here provides general insight into potential dynamical behaviors of other host-parasite-predator systems. Such insight is pertinent because two case studies have yielded differing conclusions about selective predation-induced, host-parasite dynamics. In a grouse-nematode system, reduction of predators destabilized population dynamics of host and parasite (Dobson and Hudson 1992; Hudson et al. 1992, 1998). Therefore, predators dampened the intrinsically oscillatory nature of that system. In contrast, in a hare-nematode-predator system, parasites induced oscillations into otherwise stable predator-prey dynamics (Ives and Murray 1997; Murray et al. 1997). Ives and Murray (1997) proposed two explanations for this apparent discordance. First, behavior of host-macroparasite-predator systems may depend on lethality of the parasites and strength of coupling between predator and host dynamics. Alternatively, generalist predators in the grouse-nematode may have stabilized host-macroparasite dynamics (but generalist predators can also generate complex dynamics in host-parasite systems; Dwyer et al. 2004).

Our minimal model produces several additional, general insights into switches between oscillating and stable host-parasite dynamic with predators. Parasites may destabilize otherwise stable host-predator interactions, yet predators may destabilize otherwise stable host-parasite interactions. The specific behavior of the three-species combination depends crucially on the degree of selectivity of the predator, intensity of predation pressure, and ecosystem productivity. Perhaps these factors also influence macroparasite systems. Here, this combination provoked oscillatory dynamics and thus provided one of only a few other deterministic mechanisms that can destabilize parasite-host interactions (Anderson and May 1981; Greenman and Hudson 1997). At higher productivity, host-parasite dynamics may shift from stable to oscillatory back to stable dynamics along a gradient of increasing predator control. Thus, the model predicts that community and ecosystem context can drive either oscillatory or stable host-parasite dynamics.

Analysis of these types of models will continue to provide important guides to the range of possible outcomes in host-parasite systems. As ecologists and managers become increasingly interested in preventing outbreaks of nonhuman diseases or using parasites for biological control, they will likely place strong emphasis on a priori predictions of invasion dynamics of disease (Russell et al. 2003). Success of such efforts may depend on an understanding of the direct and indirect effects of other species on hosts and parasites. Our analysis shows that selective predators may play a critical role in invasion and persistence of both parasites and hosts. Here, this role hinged on predation intensity, predator foraging behavior (selectivity and satiation), and ecosystem productivity. By considering interactions among these factors, a priori predic-

tions of disease dynamics may have a greater chance of success.

Acknowledgments

Thanks go to G. Dwyer, P. Hudson, A. Tessier, and an anonymous referee for valuable comments on previous versions of the manuscript. Primary funding for this study came from National Science Foundation (NSF) grant OCE 02-35039 to C.E.C. M.A.D. was supported by an NSF graduate research fellowship and NSF graduate training grant DIR-9602252. This is contribution 1155 from the Kellogg Biological Station.

Literature Cited

Anderson, R. M., and R. M. May. 1981. The population dynamics of microparasites and their invertebrate hosts. Philosophical Transactions of the Royal Society of London B 180:340–413.

———. 1991. Infectious diseases of humans: dynamics and control. Oxford University Press, Oxford.

Arneberg, P., A. Skorping, B. Grenfell, and A. Read. 1998. Host densities as determinants of abundance in parasite communities. Proceedings of the Royal Society of London B 265:1283–1289.

Begon, M., and R. G. Bowers. 1995. Beyond host-pathogen dynamics. Pages 478–509 *in* B. T. Grenfell and A. P. Dobson, eds. Ecology of infectious diseases in natural populations. Cambridge University Press, Cambridge.

Boots, M., and A. Sasaki. 2002. Parasite-driven extinction in spatially explicit host-parasite systems. American Naturalist 159:706–713.

Bowers, R. G., and J. Turner. 1997. Community structure and the interplay between interspecific infection and competition. Journal of Theoretical Biology 187:95–109.

Case, T. J. 2000. An illustrated guide to theoretical ecology. Oxford University Press, Oxford.

Chesson, P. 2000. Mechanisms of maintenance of species diversity. Annual Review of Ecology and Systematics 31:343–366.

Dobson, A. P., and P. J. Hudson. 1992. Regulation and stability of a free-living host-parasite system: *Trichostrongylus tenuis* in red grouse. II. Population models. Journal of Animal Ecology 61:487–498.

Duffy, M. A., S. R. Hall, A. J. Tessier, and M. Huebner. Forthcoming. Selective predators and their parasitized prey: are epidemics in zooplankton under top-down control? Limnology and Oceanography.

Dwyer, G., J. Dushoff, and S. H. Yee. 2004. Generalist predators, specialist pathogens, and insect outbreaks. Nature 430:341–345.

Earn, D. J. D., P. Rohani, and B. T. Grenfell. 1998. Persistence, chaos and synchrony in ecology and epidemiology. Proceedings of the Royal Society of London B 265:7–10.

Ebert, D., M. Lipsitch, and K. L. Mangin. 2000. The effect of parasites on host population density and extinction: experimental epidemiology with *Daphnia* and six microparasites. American Naturalist 156:459–477.

Elton, C. S. 1958. The ecology of invasions by animals and plants. Methuen, London.

Gilbert, L., R. Norman, K. M. Laurenson, H. W. Reid, and P. J. Hudson. 2001. Disease persistence and apparent competition in a three-host community: an empirical and analytical study of large-scale, wild populations. Journal of Animal Ecology 70:1053–1061.

Greenman, J. V., and P. J. Hudson. 1997. Infected coexistence instability with and without density-dependent regulation. Journal of Theoretical Biology 185:345–356.

Grenfell, B., and J. Harwood. 1997. (Meta)population dynamics of infectious diseases. Trends in Ecology & Evolution 12:395–399.

Grover, J. P. 1997. Resource competition. Chapman & Hall, New York.

Gubbins, S., C. A. Gilligan, and A. Kleczkowski. 2000. Population dynamics of plant-parasite interactions: thresholds for invasions. Theoretical Population Biology 57:219–233.

Hess, G. 1996. Disease in metapopulations: implications for conservation. Ecology 77:1617–1632.

Holmes, E. E. 1997. Basic epidemiological concepts in a spatial context. Pages 111–136 *in* D. Tilman and P. Kareiva, eds. Spatial ecology: the role of space in population dynamics and interspecific interactions. Princeton University Press, Princeton, NJ.

Holt, R. D., and J. Pickering. 1985. Infectious diseases and species coexistence: a model of Lotka-Volterra form. American Naturalist 126:196–211.

Holt, R. D., A. P. Dobson, M. Begon, R. G. Bowers, and E. M. Schauber. 2003. Parasite establishment in host communities. Ecology Letters 6:837–842.

Hudson, P. J., A. P. Dobson, and D. Newborn. 1992. Do parasites make prey vulnerable to predation? red grouse and parasites. Journal of Animal Ecology 61:681–692.

———. 1998. Prevention of population cycles by parasite removal. Science 282:2256–2258.

Ives, A. R., and D. L. Murray. 1997. Can sublethal parasitism destabilize predator-prey population dynamics? a model of snowshoe hares, predators, and parasites. Journal of Animal Ecology 66:265–278.

Keane, R. M., and M. J. Crawley. 2002. Exotic plant invasions and the enemy release hypothesis. Trends in Ecology & Evolution 17: 164–169.

Keeling, M., and C. A. Gilligan. 2000. Metapopulation dynamics of bubonic plague. Nature 407:903–906.

Kot, M. 2001. Elements of mathematical ecology. Cambridge University Press, Cambridge.

Lafferty, K. D., and R. D. Holt. 2003. How should environmental stress affect the population dynamics of disease? Ecology Letters 6:654–664.

Ludwig, D., D. D. Jones, and C. S. Holling. 1978. Qualitative analysis of insect outbreak systems: the spruce budworm and forest. Journal of Animal Ecology 47:315–322.

MathWorks. 1999. MATLAB 5.3: the language of technical computing. MathWorks, Natick, MA.

May, R. M. 1977. Thresholds and breakpoints in ecosystems with a multiplicity of stable states. Nature 269:471–477.

Mittelbach, G. G. 1981. Foraging efficiency and body size: a study of optimal diet and habitat use by bluegills. Ecology 62:1370–1386.

Murray, D. L., J. R. Cary, and L. B. Keith. 1997. Interactive effects of sublethal nematodes and nutritional status on snowshoe hare vulnerability to predation. Journal of Animal Ecology 66:250–264.

Packer, C., R. D. Holt, P. J. Hudson, K. D. Lafferty, and A. P. Dobson. 2003. Keeping the herds healthy and alert: implications of predator control for infectious disease. Ecology Letters 6:797–802.

Regoes, R. R., D. Ebert, and S. Bonhoeffer. 2002. Dose-dependent

infection rates of parasites produce Allee effect in epidemiology. Proceedings of the Royal Society of London B 269:271–279.

Regoes, R. R., J. W. Hottinger, L. Sygnarski, and D. Ebert. 2003. The infection rate of *Daphnia magna* by *Pasteuria ramosa* conforms with the mass-action principle. Epidemiology and Infection 131: 957–966.

Rinaldi, S., and M. Scheffer. 2000. Geometric analysis of ecological models with fast and slow processes. Ecosystems 3:507–521.

Russell, C. A., D. L. Smith, L. A. Waller, J. E. Childs, and L. A. Real. 2003. *A priori* prediction of disease invasion dynamics in a novel environment. Proceedings of the Royal Society of London B 271: 21–25.

Scheffer, M., S. Rinaldi, and Y. A. Kuznetsov. 2000. Effects of fish on plankton dynamics: a theoretical analysis. Canadian Journal of Fisheries and Aquatic Sciences 57:1208–1219.

Sih, A., P. Crowley, M. McPeek, J. Petranka, and K. Strohmeier. 1985. Predation, competition, and prey communities: a review of field experiments. Annual Review of Ecology and Systematics 16:269–311.

Tessier, A. J., and P. Woodruff. 2002. Cryptic trophic cascades along a gradient of lake size. Ecology 83:1263–1270.

Tompkins, D. M., A. W. Sainsbury, P. Nettleton, D. Buxton, and J. Gurnell. 2002. Parapoxvirus causes a deleterious disease in red squirrels associated with UK population declines. Proceedings of the Royal Society of London B 269:529–533.

Zaret, T. M. 1980. Predation and freshwater communities. Yale University Press, New Haven, CT.

Associate Editor: Benjamin M. Bolker