Parasite-Driven Extinction in Spatially Explicit Host-Parasite Systems

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ABSTRACT: General host-parasite theory suggests that parasites may be implicated in the extinction of their hosts by causing instability that leads to increased risk of stochastic extinction. In contrast, spatially explicit models suggest that the parasite may directly drive the host population to extinction. Here we examine the ecological characteristics of host-parasite interactions that favor parasite-driven host extinction. Pair approximations and simulations show that parasites only drive their hosts to extinction when they significantly reduce host reproduction. As a matter of interest, parasites that have a relatively small effect on host death rate are more likely to cause host extinction. Parasite-driven host extinction occurs at any population size, whereas extinction caused by stochastic effects is less likely to occur in large host populations. Populations may therefore be under threat from parasites that stop host reproduction, and this type of parasite may prove to be the most effective biological pesticide.

Keywords: ecology, pathogen, spatial model, sublethal effects, lattice.

Recently, the role of spatial heterogeneity has become one of the central issues in ecological theory (e.g., Kareiva 1990). This is mostly due to improvements in computational power and, to some extent, analytical techniques, since it has always been clear that spatial relationships are likely to be an important feature of most ecological interactions. However, much of the classical theory of host-parasite dynamics is for simplicity and tractability based on a mean-field approximation in which the individual's spatial relationships are ignored (see Anderson and May 1979, 1991). It is clear that local processes must play a role in most host-parasite interactions. In parasites (broadly defined to include pathogens such as viruses and bacteria) that spread by direct contact, the mean-field approximation seems especially unrealistic since local contacts will tend to be more likely than distant ones. With this in mind, some recent studies have explicitly included spatial structure into host-parasite interactions by examining, for example, metapopulations in coupled patches (White et al. 1996), while further work has examined the spread of infection through a host population using reaction-diffusion equations (Dwyer 1992; White et al. 1999). A third approach examines the role of the spatial structure of individual hosts within populations by using lattice/cellular automaton models (Sato et al. 1994; Rand et al. 1995; Schinazi 1996; Rhodes and Anderson 1997; Keeling 1999).

Without spatial structure, models predict that directly, horizontally transmitted parasites will not drive their hosts to extinction, since we can only have endemic, parasite-free states (Anderson and May 1979). This can be understood intuitively because, as the density of susceptible hosts becomes low, the overall transmission of the pathogen also becomes low. However, pathogens have been implicated in a number of complete extinctions of their hosts, including the extinction of the thylacine (a carnivorous marsupial; McCallum and Dobson 1995), African wild dogs (Burrows et al. 1995), and some amphibian species (Daszak and Cunningham 1999). Such extinctions, along with local population extinctions, are usually thought to occur because of stochastic events at low population sizes caused by parasite-induced mortality or population instability. In addition, when a parasite is shared by two species, a threatened population may also be driven to extinction by constant seeding of the pathogen from the larger reservoir population (McCallum and Dobson 1995).

Sato et al. (1994) were the first to show that the host can also be deterministically driven to extinction by a parasite once spatial relationships within populations are considered. This occurs because, although the overall density of susceptible hosts becomes low, the local density may remain high. Indeed, the parasite spreads in a disease-free host population without greatly reducing the density of

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susceptible individuals that are the direct neighbors of infected individuals. Consequently, the susceptible population may go to extinction before the local density of infected individuals falls. This form of parasite-driven extinction (PDE) contrasts with those normally considered in the general ecological literature that focus on population instability increasing the risk of extinction due to stochastic events at the low point of a population cycle. Furthermore, when spatial structure has been considered in terms of population extinctions, it has usually been thought to stabilize interactions and therefore reduce the risk of extinctions caused by, for example, predator-prey instabilities (see Hanski 1999). The deterministic nature of PDE along with the fact that spatial structure increases rather than decreases the risk of extinction are important characteristics that distinguish this form of extinction from other better-understood processes.

Here we focus on the ecological characteristics of the host-parasite interaction that determine whether extinction will occur in host-populations. We then aim to use these general characteristics to predict which parasites are likely to cause extinction in their host populations. In the previous lattice model that predicted pathogen-driven host extinction (Sato et al. 1994; Haraguchi and Sasaki 2000), infected individuals were unable to contribute to reproduction either directly or through recovery. This is appropriate to a number of host-parasite interactions including most larval diseases of insects (Fuxa and Tanada 1987; Boots and Norman 2000). However, the ability of infected individuals to reproduce is an important characteristic of many host-parasite interactions. Indeed, it is one characteristic that distinguishes host-parasite interactions from predator-prey interactions, since individuals that are preyed upon can clearly make no further contribution to reproduction. This is an important distinction, and we show that parasite-driven host extinction will not occur without a reduction in infected reproduction. Sato et al. (1994) further simplified their model by excluding the pathogenicity (disease-induced mortality) of the parasite. Their model is therefore only strictly applicable to a specialist group of parasites that stop reproduction but cause no increase in death rate. Here we first extend Sato et al. (1994) to include the pathogenicity of the parasite and then show that the degree of pathogenicity is central to the chance of host extinction.

Modeling

We consider a regular network of sites, each of which corresponds to either an individual host or an empty site. There are three possible states for each site: empty (0), occupied by a susceptible individual (S), or occupied by an infected individual (I). Host individuals reproduce into neighboring empty sites, while infection occurs from contact between infected and susceptible individuals at neighboring sites. Hosts do not move between sites, and infected individuals have a higher death rate than susceptibles. A site becomes empty when an individual dies and may be reoccupied by the progeny of individuals in neighboring sites. The analysis is based on pair approximation (Matsuda et al. 1992; Sato et al. 1994) and Monte Carlo simulation. A regular lattice with a periodic boundary is assumed so that each site has four nearest neighbors.

The state of each site is included in $\Sigma \equiv \{0, S, I\}$. Let $\rho_\sigma(t)$ ($\sigma \in \Sigma$) be the probability that a randomly chosen site has state σ at time t, and let $p_{\sigma\sigma'}$ be the probability that a randomly chosen site has state σ and its randomly chosen nearest-neighbor site has state σ' (σ and $\sigma' \in \Sigma$). Let $q_{\sigma/\sigma'}$ be a conditional probability that a randomly chosen nearest neighbor of a σ' site is a σ site. Let $q_{\sigma/\sigma'\sigma''}$ be a conditional probability that a randomly chosen nearest neighbor of the σ' site of a $\sigma'\sigma''$ pair is a σ site. The following differential equations for how the pairs of states change can then be written as

$$\begin{aligned}\frac{dp_{SS}}{dt} &= 2r\left[\theta + (1-\theta)q_{S/0S} + f(1-\theta)q_{I/0S}\right]p_{S0} \\ &\quad - 2dp_{SS} - \left[2\beta(1-\theta)q_{I/SS}\right]p_{SS}, \qquad (1)\end{aligned}$$

$$\begin{aligned}\frac{dp_{S0}}{dt} &= r\left[(1-\theta)q_{S/00} + f(1-\theta)q_{I/00}\right]p_{00} \\ &\quad + dp_{SS} + (d+\alpha)p_{IS} - dp_{S0} \\ &\quad - r\left[\theta + (1-\theta)q_{S/0S} + f(1-\theta)q_{I/0S}\right]p_{S0} \qquad (2) \\ &\quad - \left[\beta(1-\theta)q_{I/S0}\right]p_{S0},\end{aligned}$$

$$\begin{aligned}\frac{dp_{00}}{dt} &= 2dp_{S0} + 2(d+\alpha)p_{I0} \\ &\quad - 2r\left[(1-\theta)q_{S/00} + f(1-\theta)q_{I/00}\right]p_{00}, \qquad (3)\end{aligned}$$

$$\frac{dp_{II}}{dt} = 2\beta\left[\theta + (1-\theta)q_{I/SI}\right]p_{IS} - 2(d+\alpha)p_{II}, \qquad (4)$$

$$\begin{aligned}\frac{dp_{IS}}{dt} &= r\left[f\theta + (1-\theta)q_{S/0I} + f(1-\theta)q_{I/0I}\right]p_{I0} \\ &\quad + \left[\beta(1-\theta)q_{I/SS}\right]p_{SS} - (2d+\alpha)p_{IS} \qquad (5) \\ &\quad - \beta\left[\theta + (1-\theta)q_{I/SI}\right]p_{IS},\end{aligned}$$

$$\begin{aligned}\frac{dp_{I0}}{dt} &= \left[\beta(1-\theta)q_{I/S0}\right]p_{S0} + dp_{IS} \\ &\quad + (d+\alpha)p_{II} - (d+\alpha)p_{I0} \qquad (6) \\ &\quad - r\left[f\theta + (1-\theta)q_{S/0I} + f(1-\theta)q_{I/0I}\right]p_{I0}.\end{aligned}$$

Here $\theta = 1/z$, where z is the number of nearest neighbors for each site ($z = 4$ for a two-dimensional regular lattice), r is the reproductive rate, d is the natural mortality rate of susceptibles, β is the transmission rate, and α is the added mortality caused by infection (pathogenicity). Reproduction depends on the number of susceptibles ($q_{S/0S}$, $q_{S/0I}$, and $q_{S/00}$) and infecteds ($q_{I/0S}$, $q_{I/0I}$, and $q_{I/00}$) around each empty site. Infecteds may reproduce at the same rate ($f = 1$) as susceptibles, not at all ($f = 0$), or at a reduced rate ($0 < f < 1$).

From equations (1)–(6) for how the pairs change (the doublet dynamics), we can write equations that describe the way in which the global densities of the susceptibles ($p_S = p_{S0} + p_{SS} + p_{SI}$) and infecteds ($p_I = p_{I0} + p_{II} + p_{IS}$) change as

$$\frac{dp_S}{dt} = rp_Sq_{0/S} + rfp_Iq_{0/I} - \beta p_Sq_{I/S} - dp_S, \tag{7}$$

$$\frac{dp_I}{dt} = \beta p_Iq_{S/I} - (d + \alpha)p_I. \tag{8}$$

If we replace the local densities ($q_{0/S}$, $q_{I/S}$, and $q_{S/I}$) with their global equivalents (p_0, p_I, and p_S), we can examine the model without spatial structure. From this, it is easy to see that our model reduces to a classical, density-dependent mean-field host-parasite model (i.e., Anderson and May 1981), with the possibility of a reduced birthrate by infected individuals (with $0 \le f \le 1$). Microscopically, the infection process obeys a mass-action rule with infection proportional to the time that a susceptible individual is next to an infected individual. This is a biologically transparent and realistic assumption for which there is no reasonable alternative. However, macroscopically, there is no mass-action assumption in the global dynamics of the spatial model since we expect inequalities where $q_{I/S} < p_I$ and $q_{0/S} < p_0$ because of spatial clumping. Our results are therefore not dependent on a mass-action assumption concerning the transmission process that may be criticized because of a lack of empirical support (e.g., Knell et al. 1996).

Since the doublet dynamics (eqq. [1]–[6]) depend on triplet densities, we need to make an approximation to complete the pair approximation and then use the doublet dynamics to predict the behavior of the model. The tendency for susceptible individuals to cluster will tend to make $q_{S/00}$ smaller than $q_{S/0}$, while $q_{S/0S}$ will be larger than $q_{S/0}$ (see Sato et al. 1994). We decouple the triplet densities as $q_{S/00} = \varepsilon q_{S/0}$ (and $q_{I/00} = \varepsilon q_{I/0}$), where ε is a constant to embed the triplet correlation into doublet dynamics; it is determined from the critical fecundity necessary to maintain the population in basic contact processes (Sato et al. 1994). It has been shown that $\varepsilon = 0.8093$ for a two-dimensional regular lattice (see Sato et al. 1994). The other triplet densities are decoupled as $q_{S/0S} = 1 - \varepsilon q_{0/0} - q_{I/0}$, $q_{I/S\sigma} = q_{I/S}$ for any σ, and $q_{S/0I} = q_{S/0}$ (see Sato et al. 1994), with corresponding decouplings for infecteds when they reproduce. By constructing the local density dynamics from the doublet density dynamics (in which the conditional probabilities relate to the overall proportions; eqq. [1]–[6]), combining them with the global density dynamics (eqq. [7] and [8]), and linearizing near the equilibrium, we can examine the local stability of the disease-free and population extinction equilibrium (see Sato et al. 1994 for details). The borders between different outcomes can then be drawn. For example, in figure 1, the population extinction equilibrium is locally stable for transmission rates greater than the upper border, while the disease-free equilibrium is locally stable for transmission rates below the lower border. We then check the predictions of the pair approximations by simulation of the model in a regular lattice with periodic boundaries.

Results

Let us start by considering the case when infected individuals do not reproduce at all ($f = 0$). This model is similar to one considered by Sato et al. (1994), except that we add biological realism by including the pathogenicity (disease-induced death rate) of the parasite. Figure 1 shows the boundaries of different outcomes predicted from pair approximation (eqq. [1]–[6]). Similar to models without spatial structure, we have a disease-free region when the transmission rate of the pathogen and birthrate of the host are low. For a given birthrate, as we increase the transmission rate, we enter another region—endemic—where the parasite is now able to maintain itself in the host population. As transmission increases still further, we enter a new region where the host and therefore the parasite both go to extinction (pathogen-driven extinction). This does not occur in equivalent nonspatial models.

Figure 1 also shows the results of simulations of the model on a 100 × 100 regular lattice. The pair approximation predicted the disease-free region and the endemic region well, and the simulations confirm that extinction occurs at higher transmission rates. However, the region for parasite-driven extinction observed in simulations is wider than that predicted by pair approximation. In addition, when the transmission rate of the pathogen is high and the birthrate low, simulations predict a disease-free region rather than the pathogen-driven extinction predicted by pair approximation. Both these deviations from the predictions of the pair approximations can be ascribed to the effect of density fluctuations in a finite population (fade-out). We therefore have both certain (in the region predicted by pair approximations) and stochastic (at high

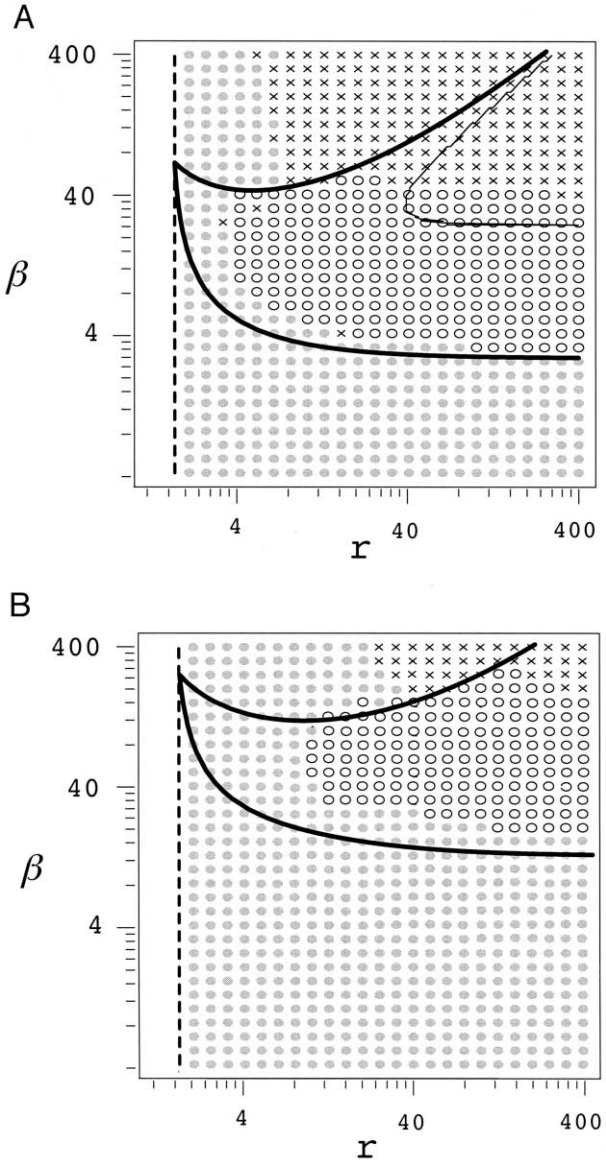

Figure 1: Boundaries between disease-free, endemic, and extinction regions drawn by pair approximations and results of simulation (in 100 × 100 lattices) in transmission rate (β) and host reproduction (r) parameter space. The outcome of each simulation run is classified as endemic (*open circle*; both susceptible and infected individuals persisted until $t = 50$), disease-free (*gray circle*; infected individuals go to extinction, leaving a disease-free susceptible population), or parasite-driven (*hatch*; susceptible and infected individuals go to extinction, leading to the extinction of the entire population) host extinction. In the few cases where there is variation in the results of the simulations—near the borderlines between the states—the majority outcome is presented. In both cases, there is no reproduction from infecteds ($f = 0$). In *A*, $d = 1.0$ and $\alpha = 1.0$, while in *B*, there is a higher pathogenicity so that $\alpha = 10.0$. The thin line in figure 1*A* encloses the region where pair approximation predicts stable limit cycles.

transmission and low host birthrates) extinction of the disease. Similarly, we have certain (parasite-driven) and stochastic (fade out) host extinction. In the region of stochastic host extinction, pair approximation predicts endemic limit cycles (fig. 1*A*).

Figure 1*B* shows how an increased parasite-induced death rate (pathogenicity) affects the chance of extinction. Comparing figures 1*A* and 1*B*, we can see that extinction is more likely when the pathogenicity (disease-induced death rate) is low. This is caused by the lower death rate increasing the infectious period of the parasite. This important result suggests that mild parasites that prevent their hosts from reproducing are more likely to drive their hosts to extinction than severe ones. It also emphasizes the importance of our inclusion of pathogenicity in this model, in contrast to Sato et al. (1994), who only consider the limit when $\alpha = 0$, which is the limit at which host extinction is most likely. It follows that if there is a tradeoff for the parasite between pathogenicity and transmission rate (such that high transmission rates are associated with high pathogenicity), parasite-driven host extinction is less likely (fig. 1). This relationship also leads to the conclusion that at any fixed R_0 (the basic reproductive number of the parasite), the asymptotic outcome will be the same (see Haraguchi and Sasaki 2000).

Distinguishing between parasite-driven and stochastic host extinction from any given time series is difficult, but the two processes can be distinguished by increasing the lattice size (host population size). In larger populations, parasite-driven extinction still occurs (fig. 2*A*–2*B*), while in the stochastic extinction region, the population is protected and an endemic equilibrium is found (fig. 2*B*–2*C*). Fluctuations in population densities are often seen at the endemic equilibrium (fig. 2*E*). As the lattice size and therefore the carrying capacity of the host increase, the fluctuations in the global densities become less pronounced (fig. 2*E*). This suggests that local fluctuations become asynchronous within larger populations spread over a wider region and therefore damp the overall, global fluctuations.

When the infected individual's reproduction is unaffected ($f = 1$), pair approximation does not predict a region of parasite-driven host extinction (fig. 3*A*). Again, simulations confirm the predictive power of the pair approximation in the disease-free and endemic areas, and they further confirm that there is no parasite-driven host extinction in this model (fig. 3*A*). However, simulations show that a disease-free region again occurs when β is high and r is low (fig. 3*A*). The parasite is therefore most likely to be maintained at intermediate transmission rates. Pathogen-driven extinction of the host can occur when infected individuals reproduce but only when this is at a substantially lower rate (fig. 3*B*). However, the host-extinction region is much smaller than when there is no

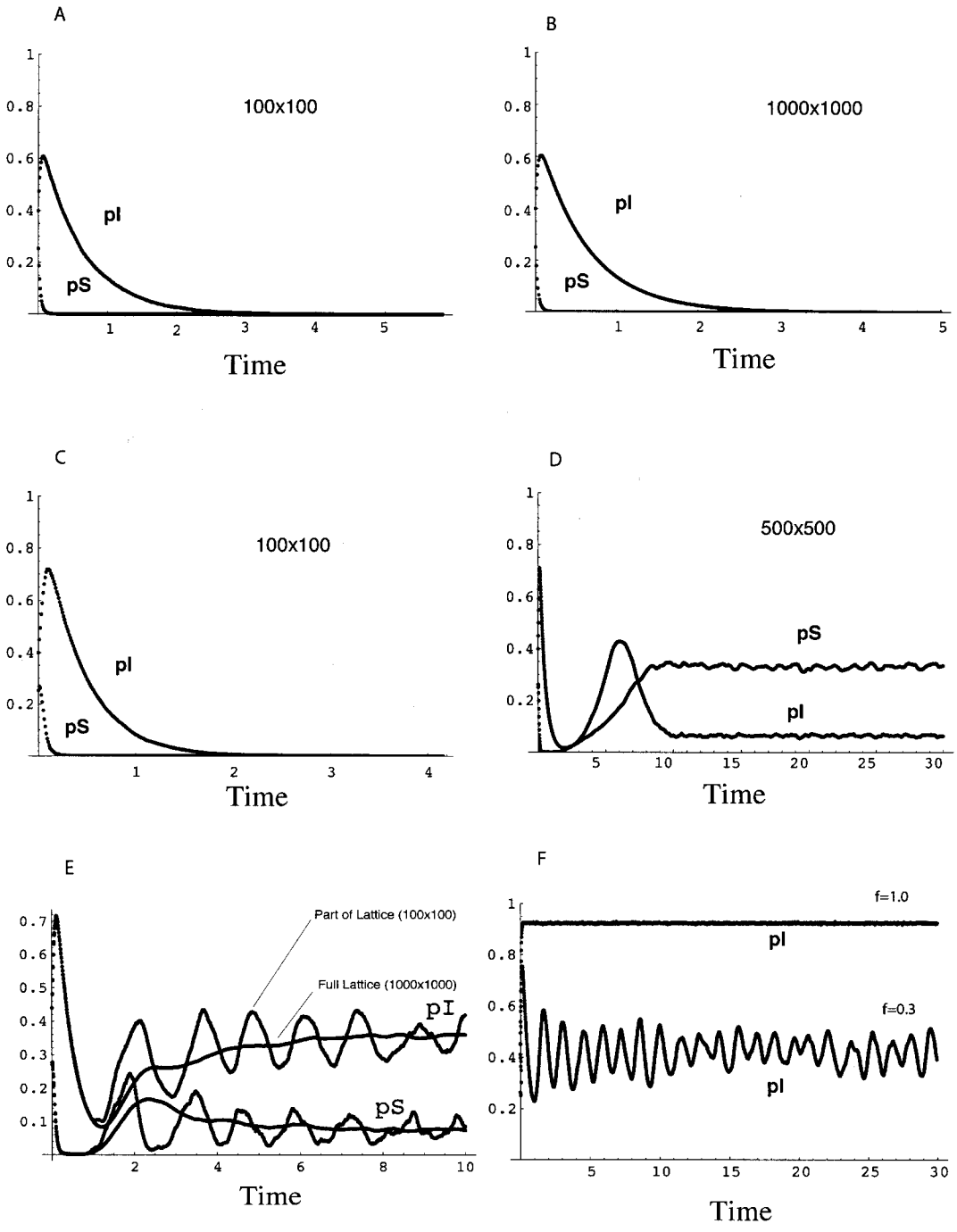

Figure 2: Proportion of infected (pI) and susceptible (pS) individuals through time showing the deterministic and stochastic host extinction predicted in figure 1*A*. In *A* and *B*, deterministic extinction is predicted at $r = 24$ and $\beta = 120$, and we find extinction when the lattice size is 100 × 100 (*A*) and 1,000 × 1,000 (*B*). In *C* and *D*, at $r = 80$ and $\beta = 48$, we have stochastic extinction with a lattice size of 100 × 100 (*C*) but endemic equilibrium (as predicted by pair approximations in fig. 1*A*) at an increased lattice size of 500 × 500 (*D*). Other parameters are the same as in figure 1*A*. In *E*, the proportion of infected (pI) and susceptible (pS) individuals through time are shown for an entire lattice (1,000 × 1,000) and part of the same lattice (100 × 100) superimposed. Other parameters are $r = 80$, $\beta = 40$, $\alpha = 1.0$, and $d = 1.0$ (same as in fig. 1*A*). In *F*, the proportion of infected (pI) individuals through time shows how sublethal effects ($f = 0.3$) increase fluctuations in a 150 × 150 lattice. Other parameters are $r = 80$, $\beta = 40$, $\alpha = 1.0$, and $d = 1.0$.

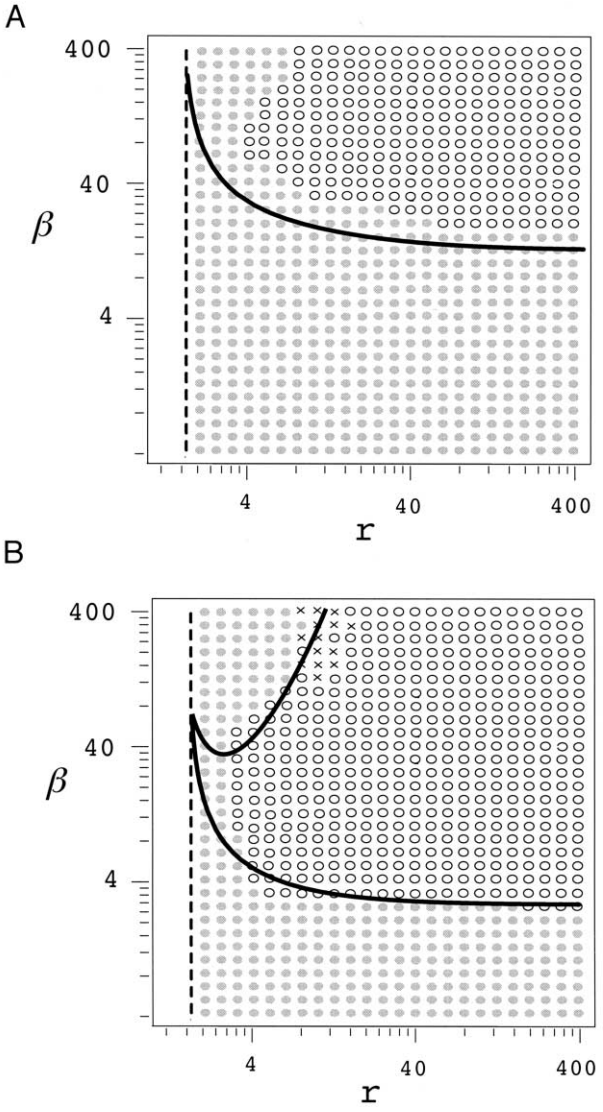

Figure 3: Boundaries between disease-free, endemic, and extinction regions drawn by pair approximations and the results of simulation (in 100 × 100 lattices) in transmission rate (β) and host reproduction (r) parameter space. The outcome of each simulation is classified as endemic (*open circle*; both susceptible and infected individuals persisted until $t = 50$), disease-free (*gray circle*; infected individuals go to extinction, leaving a disease-free population), or parasite-driven (*hatch*; susceptible and infected individuals go to extinction, leading to the extinction of the entire population) host extinction. In the few cases where there is variation in the results of the simulations—near the borderlines between the states—the majority outcome is presented. In *A*, the infected individual's reproduction is unaffected ($f = 1$), with $d = 1.0$ and $\alpha = 10.0$, while in *B*, they reproduce, but at a much lower rate ($f = 0.2$), with $d = 1.0$ and $\alpha = 1.0$.

reproduction and negligible at moderate degrees of reduced reproduction. The sublethal effects of the parasite on host reproduction ($f < 1$) also destabilize the host dynamics (fig. 2*D*), a result not found in equivalent host-parasite models that lack spatial structure (Anderson and May 1981).

Discussion

Our models show the importance of spatial structure in general to the equilibrium and dynamical behavior of host-parasite interactions as well as the role of specific ecological characteristics in determining these behaviors. A key finding is that a parasite may drive its host and therefore itself to extinction if infected individuals do not reproduce or reproduce at a much lower rate than susceptible individuals. When the infected individual's reproduction is not strongly affected, the host is not driven to extinction. In all cases, high transmission rates and low host birthrates lead to the extinction of just the parasite. In models that are not spatially explicit, we do not find this type of parasite-driven host extinction. Our results therefore emphasize how explicitly considering space in ecological theory can change the established inferences of classical models.

Our results are important for two further reasons. First, spatial population structure—in particular, spatially explicit metapopulations—has generally been thought to reduce the chance of population extinction (see Hanski 1999). While clearly this will often be the case, our results show that spatially explicit structure can also lead to the completely opposite outcome. This is because parasite-driven host extinction is not a process that increases instability in population dynamics and therefore is not related to the normal processes that increase the risk of the more commonly considered stochastic extinctions. Second, this article has emphasized the importance of analytical analysis by approximation of spatial models. Without the pair approximations used here, it is unlikely that by simulation alone we would have been able to ascertain that there were two distinct types of extinction occurring (parasite-driven and stochastic). The extinction region in the pair dynamics led directly to our understanding of the processes involved in these two types of extinction. Pair approximations are equally likely to prove to be useful tools in the analysis of spatial models in many contexts of ecological theory, especially in terms of finding distinct outcomes not predicted from mean-field theory.

The question remains as to whether parasites have in the past or will in the future drive their hosts' populations to extinction. Obviously, the stable host-parasite interactions that we can observe in nature exclude ones that have previously gone extinct. Haraguchi and Sasaki (2000) ex-

amined the evolution of the parasite transmission rate in a spatial model that showed parasite-driven host extinction. They predicted that the parasite would often evolve to a transmission rate just below the boundary of host extinction. A similar result was found in work by Rand et al. (1995), although in their case only parasite extinction was predicted. This suggests that parasites will not evolve sufficiently high transmission rates to cause the extinction of their hosts. However, we consider the situation where the host birthrate falls, perhaps because of a reduction in the quality of the environment. In this case, without the chance for evolution to occur, the interaction could move out of the endemic parameter region, and parasites that stop reproduction may now drive the host to extinction. There is also the chance of the emergence of noncoevolved diseases through host transfer by parasites. In this case, there is the possibility that parasites that have jumped to a new host will have characteristics that by chance fall within the region of parasite-driven host extinction.

Parasites, including pathogens such as viruses, bacteria, and microsporidians, are of great importance for their use and potential use as biological pest control agents (Payne 1988). The role of genetic engineering in altering the transmission efficiency of a parasite to improve its effectiveness as a control agent is of great interest. Here we have shown that if the chosen parasite also stops the host from reproducing, a highly effective control agent may be produced that is able to cause the extinction of the pest population. Since this is a deterministic process intrinsic to the host-parasite interaction, it will occur without the need for inundatory use of the control agent. However, when the parasite chosen allows significant reproduction from infected individuals, an artificial increase in the transmission rate may lead to the loss of the control agent itself because of an increased risk of epidemic fade-out.

This study has emphasized the importance of the nonlethal effects of parasites on the population dynamics of their hosts. The effect of the parasitic infection on reproduction is crucial in determining whether the host or just the parasite goes extinct. In addition, the destabilizing role of sublethal effects is clearly shown since we see an increased chance of unstable population dynamics as $f \to 0$. When sublethal infection has been previously implicated as a destabilizing force in host-parasite interactions, it has generally been in combination with transmission by free-living infective stages (Anderson and May 1981; Hudson et al. 1998). By explicitly considering spatial structure, however, we see that sublethal effects may destabilize host populations even in parasites that are not propagated by free-living infective stages.

Another important and, perhaps at first sight, counterintuitive result of this work is that parasites with lower pathogenicity are more likely to cause the extinction of their hosts. This can be intuitively understood to be caused by the fact that, in parasitic infections that may cause extinction, infected individuals are unable to reproduce (or reproduce at a much lower rate). Parasite-driven extinction comes about because of very high levels of infection, and therefore the increased infectious period when pathogenicity is low always enables the process. It is often considered that there may be a trade-off between a parasite's pathogenicity and its transmission rate so that low pathogenicity will usually be associated with low transmission rates. Clearly, a low transmission rate reduces the chance of parasite-driven extinction, and therefore the nature of any trade-off in the parasite will be important in determining the chance of extinction. For example, if there is a linear trade-off (and therefore a nearly constant R_0), the asymptotic states become more or less insensitive to changes in α and β (see fig. 4*b* of Haraguchi and Sasaki 2000). There may be an understandable tendency to consider mild pathogens that cause low mortality to be unimportant to their hosts. Our results emphasize that in combination with strong sublethal effects on reproduction, mild pathogens may have considerable effects on their host populations.

Distinguishing between parasite-driven and stochastic extinction from time series or spatial relationships in natural populations is difficult because simulations show similar spatial patterns and time series. However, when the population is close to deterministic extinction, the rate of change of the global susceptible density will always be negative. From equation (7), this is most likely to occur when new infections are outstripping new births ($rp_Sq_{0/S} + rfp_Iq_{0/I} < \beta p_Sq_{I/S}$). However, stochastic extinction can occur when equation (7) is positive as $p_S \to 0$ since although the population has the potential to grow, the finite number of sites and small population size cause extinction. A relatively large number of new infections when the population is close to extinction may therefore be indicative of deterministic extinction. However, it is not always possible to distinguish between these two processes in a particular finite population since there are a number of possible paths to stochastic extinction, and these may have the characteristics of deterministic extinction by chance. For example, high numbers of infections may occur when the population is close to extinction by chance, leading to a form of stochastic extinction that is difficult to distinguish from deterministic extinction. Nevertheless, it is important to distinguish between parasite-driven and stochastic host extinction in the field because this will have consequences in terms of the management protocols used in any program to prevent extinction. Broadly, if stochastic extinction is occurring, increasing the population size by introduction may be appropriate. This will not prevent extinction under parasite-driven extinction, whereas any

increase in host local reproduction may. In addition, the breakdown of the spatial structure of the host population—by transplantation, for example—may reduce the chance of deterministic extinction.

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