Long-range interactions and evolutionary stability in a predator-prey system

Erik M. Rauch[1,2] and Yaneer Bar-Yam[2]

[1]*MIT Computer Science and Artificial Intelligence Laboratory, 32 Vassar St., Cambridge, Massachusetts 02139, USA*
[2]*New England Complex Systems Institute, 24 Mt. Auburn St., Cambridge, Massachusetts 02138, USA*
(Received 31 December 2004; published 27 February 2006)

Evolving ecosystems often are dominated by spatially local dynamics, but many also include long-range transport that mixes spatially separated groups. The existence of such mixing may be of critical importance since research shows spatial separation may be responsible for long-term stability of predator-prey systems. Complete mixing results in rapid global extinction, while spatial systems achive long term stability due to an inhomogeneous spatial pattern of local extinctions. We consider the robustness of a generic evolving predator-prey or host-pathogen model to long-range mixing and find a transition to global extinction at nontrivial values implying that even if significant mixing already exists, a small amount of additional mixing may cause extinction. Our results are relevant to the global mixing of species due to human intervention and to global transport of infectious disease.

Most traditional approaches to biological modeling represent evolutionary and ecological systems using quantities averaged over space and time. Such systems are represented as the frequencies of various genes or types in the population. In this mean field formulation, the environment experienced by a particular individual is effectively the average over all environments [1]. However, real populations are distributed spatially; organisms experience different local environments, they consume resources locally, and they travel distances which are at most a fraction of the size of the habitat of their species. It has recently become recognized that spatial inhomogeneity can be crucial to the dynamics of ecological [2] and evolutionary [3] systems. In particular, spatial locality is necessary for stability (the coexistence of species) in a number of systems [4]. In the well-mixed case, predators become extinct in evolving predator-prey models [5,6]. (Whether spatial or well-mixed, these models describe discrete populations and thus sufficiently depleted populations go extinct, unlike the conventional Lotka-Volterra model where the continuum treatment does not allow depletion of prey to result in extinction.) As in earlier studies [7], evolutionary stability is defined by persistance over a time that grows exponentially with the size of the system (a remarkably long time), while extinction is characterized by systems whose populations expire over a time that is no greater than linear in the size of the system (a remarkably short time).

Many real spatially distributed systems, however, though interacting mostly locally, also have long-range interactions. In a biological context, long-range interactions can arise, for example, when individuals can disperse via spores or when their seeds are transported long distances or through transport by human beings, i.e., the unintentional or intentional introduction of invasive species from remote locations. In the limit of many long-range interactions, spatial models change to the behavior charasteristic of well-mixed systems. We explore in this paper the degree to which systems with mostly local interactions are robust to long-range interactions. Recent work on "Small-World" networks [8,9] has investigated the effect of long-range connections on the connectivity of natural and artificial networks, and has shown that it takes only a very small amount of global mixing to make a mostly locally interacting system behave as a well-mixed system in some respects. Since a small amount of mixing is likely in many cases, this finding makes suspect the relevance of spatial models, and raises the question of how evolutionary systems that depend on spatial separation are able to persist.

Previous studies of transitions arising from the introduction of global mixing have considered dynamic models without evolution, such as a spatial prisoner's dilemma with volunteering [10], a nonevolving host-pathogen model showing a transition to global oscilations [11], and a model showing a percolation transition [12]. In this work we focus on evolutionary coexistence in a predator-prey and/or host-pathogen model in which the dynamics can lead to extinction of the pathogen and severe depletion and sometimes extinction of the host as well. This model is of potential relevance to the ongoing evolution of "emergent" pathogens with human hosts as well as the evolutionary implications of invasive species. For the case of rapidly evolving emergent pathogens, host evolution in response to the pathogen cannot mitigate the extinction due to the short time scale over which pathogen evolution occurs.

Previous research has considered a variety of aspects of the evolutionary dynamics of spatial host-pathogen models. It has been shown that pathogen and host can coexist when the transmissibility evolves [13], and the mechanisms by which evolutionary stability and instability occur have been identified [5,6,14,15]. Other studies have considered the evolution of dispersal distance [16–19]. It has been shown [16] that when the dispersal distance itself can evolve in a system exhibiting spatial patterns consisting of spiral waves, this distance increases by selection and in some cases this may lead to the extinction of the population.

The spatially extended predator-prey model we use is chosen because it is simple yet exhibits nontrivial evolutionary behavior. In the model, the rate at which predators consume prey evolves; higher rates give a short-term advantage to predators since they can reproduce faster. However, in a globally mixed population, a rate that is too high leads to depletion of prey and hence the extinction of the predator. In the spatial case, because types are spatially segregated from each other, the overexploiting types cause their own local

extinction while the evolutionarily stable type survives. Thus, the long-term selection favors a sustainable level of predation. We will study the effect of long-range mixing on the evolutionary stability of the system, that is, the ability of predator and prey to coexist.

The model is a probabilistic cellular automaton [5,20], with possible states 0 (empty), H (prey), and P_τ (prey being consumed by predator with reproduction rate τ). At each time step, prey reproduce into each connected site with probability g if that site is not yet occupied. A predator P_τ can reproduce by colonizing a connected site with probability τ, if that site has prey. Prey being consumed by predators do not reproduce. Finally, a local predator population depletes its prey and hence dies out with probability v, leaving empty space. Explicit rules are in Ref. [5]. (The model can also be thought of as a host-pathogen system, with the predators being pathogens and the prey being hosts [21,7,22]; in these terms, v describes the virulence of the pathogen and τ the transmissibility.) To investigate the evolutionary dynamics of the system, we add mutations [13,5] that change the reproduction rate so that predators with reproduction rate τ have offspring at a value $\tau \pm \varepsilon$ with probability μ. This makes it possible to study the effect of selection on the predator-prey dynamics.

Several variations in the model were simulated without qualitative changes in results, including larger but still local reproduction neighborhoods and Gaussian distribution of the change in τ. We do not consider the evolutionary dynamics of g and v since they would simply evolve to arbitrarily large and small values, respectively, for inherent biological constraints [6].

In a well-mixed (homogeneous and/or mean-field) approximation [1] of the model [7,14], the reproduction rate τ monotonically increases [23]. The spatial model, by contrast, evolves to an evolutionarily stable value of τ. When high values of τ lead to extinction, τ does not increase to the point of extinction; the steady-state value is lower than the extinction limit. This self-organized survival results from local rather than global extinctions of overexploiting types and occurs for the entire region of parameter space where predator and prey coexist. Figure 1 shows an evolving system at intervals of 20 generations after it has reached the evolutionarily stable state, showing patches of prey growing and being depleted by predators of various types. Mutant, overexploiting strains continually arise but go extinct over many generations, but without causing the extinction of the entire population. The selective extinction results from self-consistent spatial segregation and the local depletion of prey [5,6]. Thus, selection acts only on long time scales, through environmental modification and feedback. We have previously shown using an analytic treatment that the key dynamical behaviors of the model can be accounted for by assuming a characteristic size of patches [14,15]. This motivates the discussion below of finite-size and patch-size scaling.

Preliminary to considering nonlocal reproduction, we characterized the effect of the size of the space on the stability of these systems. Coexistence requires a minimum amount of space. Below a lattice size L_{es}, the system is unstable [Fig. 2(a)] since an overexploiting mutant strain can grow to become the only strain in the whole space before
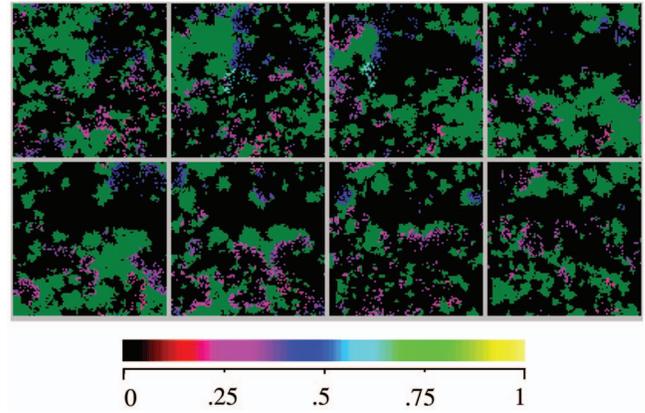

FIG. 1. (Color) Snapshots of the lattice for the evolving pathogen-host model. Twenty generations elapse between each frame. Prey populations by themselves are shown as green and prey being consumed by predators are colored depending on their value of the predator reproduction rate τ, as shown in the legend. In this and all subsequent figures, the system has settled to a steady state value of τ. We use an $L \times L$ square lattice with periodic boundary conditions and a von Neumann neighborhood (north, south, east, and west neighbors). In this figure, the lattice size $L=100$, prey depletion rate $v=0.2$, and prey reproduction rate $g=0.05$.

environmental feedback has time to cause its extinction. We have found that the minimum area for evolutionary stability L_{es}^2 is proportional to the average population of a predator strain summed over the course of its existence [Fig. 2(b)]. This indicates that evolutionary stability occurs when the space is large enough that nonoverexploiting strains can survive during an outbreak of an overexploiting mutant.

We introduce long-range dispersal by replacing the lattice of the model with a Small-World network [8]. We choose this static connectivity to relate our results to work on network theory, but we also consider dynamic long-range dispersal below. To generate the lattice, we begin with a network corresponding to a square lattice in which each site is connected to its four neighbors, and randomly "rewire" each link with probability p. Thus, there are $4pL^2$ long-range connections; a regular lattice is a special case with $p=0$. $p=1$ corresponds to a well-mixed case [7,14] in which higher-τ predator strains always outcompete lower-τ ones, and, for

TABLE I. Critical density p_c as a function of the model parameters, on (a) a Small-World network. (b) for long-range dispersal. 1 indicates stable for all p.

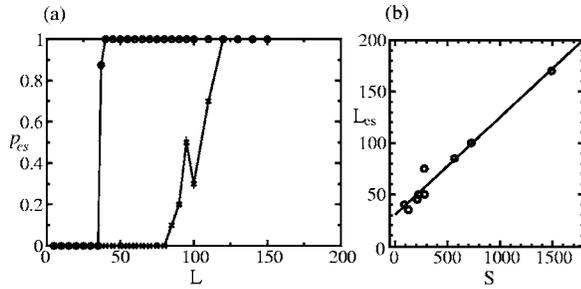| p_c	$g=0.05$	$g=0.01$	$g=0.2$
		(a)	
$v=0.1$	0.02	0.3	1
$v=0.2$	0.001	0.02	0.45
$v=0.4$	0.0001	0.002	0.25
		(b)	
$v=0.1$	0.1	0.8	1
$v=0.2$	0.005	0.2	1
$v=0.4$	0.001	0.02	0.9

FIG. 2. (a) Evolutionary stability as a function of lattice size L. Each point represents the probability p_{es} that the predator and prey will coexist for at least 100 000 generations, obtained from 10 runs. Any time substantially longer than a single generation will give similar results as there is a sharp transition between populations that survive for times that are exponential in system size and those that are not more than linear. There is a transition at a size L_{es}. For the left curve (circles), the prey reproduction rate $g=0.2$. For the right curve ($\times$ symbols), $g=0.05$. The depletion rate $v=0.2$. (b) The minimum lattice size for evolutionary stability L_{es} as a function of the average total population S of an overexploiting strain over the course of its existence. Results are for all combinations of $v=0.1,0.2,0.4$ and $g=0.05,0.1,0.2$. The line shows the fit $L_{es}=0.1S+30$ ($r^2=0.97$).

most model parameters, become extinct. Figure 3 shows the behavior of the system as p is increased from 0 as measured by the probability of coexistence over a long-time scale (100,000 generations) as a function of p. There is a sharp transition to extinction at a value p_c which depends on the parameters (Table I). This critical density is plotted in Fig. 4(a) as a function of the characteristic spatial scale of the system: the area of a contiguous host patch in which a pathogen is located. For a wide range of parameters, a single functional dependence is found—a power law. When patches are larger in the absence of long-range links, the system is more sensitive to the addition of links [Table I(a)]. The results we found are quite different from studies of Small-World networks that focus on connectivity. Similar to previous studies of nonevolving systems [10–12], we find a sharp transition at a value of $p \equiv p_c$ that depends on model parameters. Figure 3(a) shows that the system is robust to the introduction of small amounts of mixing. Indeed, p_c can be quite high; for example, for $g=0.2$, the system is still stable when the probability of long-range dispersal is 0.45.

While the stability of the system does not follow the behavior of connectivity in Small-World networks, a relationship can still be made to this area of research. Studies have shown [24–28] that the Small-World phenomenon of shortened path lengths occurs only between nodes that are more than a certain crossover distance $\xi(p)$ from each other on the underlying lattice before addition of long-range links. Below this distance, the path length scales linearly in the distance on the underlying lattice. We have found that the crossover distance at the critical density $\xi(p_c)$ is approximately linear in the average diameter $\sqrt{a_p}$ of patches in the evolutionary model [Fig. 4(b)], and that $\xi(p_c)$ is, in general, larger than $\sqrt{a_p}$. This can be understood if we interpret $\xi(p)$ as the scale below which the system has spatial behavior and is not strongly affected by the long-range mixing. If $\xi(p)$ is smaller
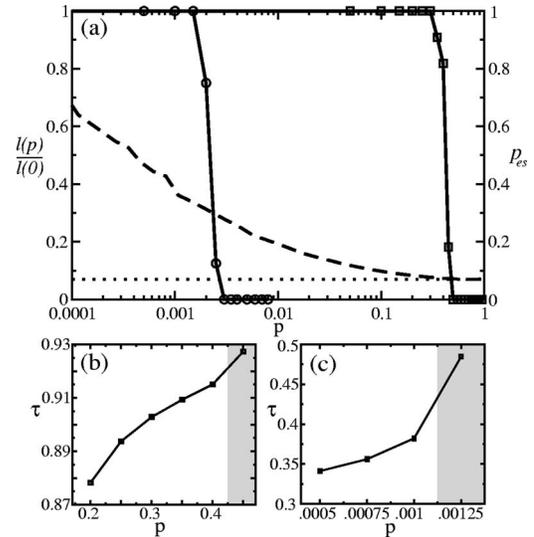

FIG. 3. Evolutionary stability on a two-dimensional Small-World network. (a) The probability p_{es} that predator and prey coexist for 100 000 generations, as a function of p, averaged over 11 runs, for $g=0.05$ (circles) and $g=0.2$ (squares) (depletion rate $v=0.2$, lattice size $L=250$). Note the logarithmic scale of p. We identify the point of transition to instability p_c as the density such that for all $p>p_c$, $p_{es}<1/2$. For comparison, the average path length $l(p)$ between nodes is plotted as a fraction of $l(0)$ (dashed line, same scale). For comparison, the dotted line shows $l(1)/l(0)$, that is, the value for a random network. (b) The evolutionarily stable reproduction rate τ_{es} as a function of p on a two-dimensional (2D) Small-World network, averaged over the last 200 generations of 10 runs of 100 000 generations. τ_{es} is also plotted for values of p for which the predators go extinct (shaded region); the average of the last 200 generations before extinction is plotted. (c) As (b), but for $g=0.05$.

than the patch size, then the addition of long-range links reduces the effective size of the system below the size of a single patch on average, leading to extinction.

For many biological scenarios, such as airborne dispersal in pathogens, it is more realistic that only the predator disperses long distances, and disperses to a dynamically chosen site instead of over a fixed set of long-range connections. We model this as follows: p describes the probability that a predator will have a chance to disperse to a randomly chosen site, and occupies the site with probability τ if that site contains a prey population. This leads to similar results, although the value of p_c changes [Table I(b)].

The transition to instability can be studied by examining the average reproduction rate τ_{es} of the predators in the long-time steady-state limit. Increasing long-range dispersal increases τ_{es} as seen in Figs. 3(b) and 3(c). This is consistent with the idea that exploiting strains are increasingly able to escape local extinction through long-range dispersal.

The relevance of the spatial scale of patch size in the absence of long-range links to the critical density of long-range dispersal can be readily understood. As patch size increases, a predator strain that overexploits a patch has more opportunities to maintain itself by invading a distant patch before local extinction, and so requires a lower rate of long-range dispersal. Thus, the larger the scale of spatial structure in the distribution of predator and prey, the smaller the den-
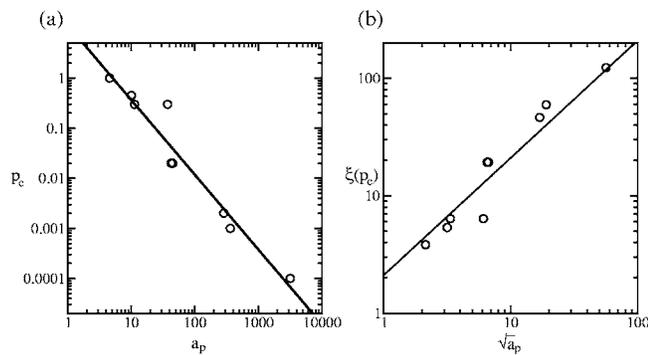
FIG. 4. (a) The critical density p_c as a function of the average patch area a_p. The line is the least-squares fit $p_c \sim a_p^{-1.5}$ ($r^2 = 0.94$). (b) The length scale of the crossover between "small-world" and "large-world" scaling of distances at the critical density p_c, as a function of the average patch diameter $\sqrt{a_p}$, i.e., $\xi(p_c) = ln(4\pi p_c L^2)/2\sqrt{\pi p_c}$. The line shows the fit $\xi(p_c) = 2.1\sqrt{a_p}$ ($r^2 = 0.97$). Please note the logarithmic horizontal scale.

sity of long-range links needed to destabilize the system. Moreover, the robustness of the predator-prey system is affected by the time scale at which selection acts against an overexploiting strain [5]. When such strains are successful for a large number of generations before going extinct, they have more chances to disperse to a distant patch before they locally deplete their prey.

Our results suggest that spatially extended models are relevant to studies of systems with long-range interactions, as long as the density of such interactions is not too high. There are many other processes in spatially extended biological

systems that operate on particular length scales, and the characteristic behavior of such systems may also be robust to limited long-range mixing. Examples include the formation of patterns in ecosystems by local activation and long-range inhibition [29] and pattern formation in excitable media, such as spiral waves [30].

The transition from spatial to homogeneous behavior can, however, be sudden and can occur even in systems that already have a significant density of long-range interactions. Thus, one should not conclude that a system that already has long-range mixing will be stable to additional mixing.

According to our simulations, when global mixing increases beyond the critical density, overexploiting predator strains escape local extinction and replace sustainable strains globally, leading to their own extinction and decimation of the prey population. Our results apply directly to simple evolutionary models, but similar considerations apply to the phenomena of emergent diseases (such as Ebola, SARS [31], and Avian Flu [32]), most of which evolve on short time scales, and may also apply to invasive species, which have been of widespread ecological concern [33,34]. While the demonstration that some long-range connections do not always destablize evolving systems provides some reassurance, the danger from additional connections suggests that a system may cross the transition and become unstable with little warning as global mixing increases in frequency.

Our results suggest the need for concerted response, including medical developments, and, perhaps, societal changes. Due to increasing global transportation, human beings may cross the transition without much warning and suffer large pandemics unless preventive actions are taken that either limit global transportation or its impact.

[1] Y. Bar-Yam, Adv. Complex Syst. **2**, 277 (1999).
[2] *Spatial Ecology: The Role of Space in Population Dynamics and Interspecific Interactions*, edited by D. Tilman and P. Kareiva (Princeton University Press, Princeton, NJ, 1997).
[3] H. Sayama *et al.*, Phys. Rev. E **62**, 7065 (2000).
[4] R. Durrett and S. A. Levin, Theor Popul. Biol. **46**, 363 (1994).
[5] E. M. Rauch *et al.*, Phys. Rev. Lett. **88**, 228101 (2002).
[6] E. M. Rauch *et al.*, J. Theor. Biol. **221**, 655 (2003).
[7] D. A. Rand *et al.*, Proc. R. Soc. London, Ser. B **259**, 55 (1995).
[8] D. J. Watts and S. H. Strogatz, Nature (London) **393**, 440–442 (1998).
[9] M. E. J. Newman *et al.*, Phys. Rev. Lett. **84**, 3201 (2000).
[10] G. Szabo and J. Vukov, Phys. Rev. E **69**, 036107 (2004). The word "evolution" as used to reproduction without evolution in the model we study.
[11] M. Kuperman and G. Abramson, Phys. Rev. Lett. **86**, 2909 (2001).
[12] D. H. Zanette, Phys. Rev. E **64**, 050901(R) (2001).
[13] Y. Haraguchi and A. Sasaki, J. Theor. Biol. **203**, 85–96 (2000).
[14] M. A. M. de Aguiar *et al.*, Phys. Rev. E **67**, 047102 (2003).
[15] M. A. M. de Aguiar *et al.*, J. Stat. Phys. **114**, 1417 (2004).
[16] N. J. Savill and P. Hogeweg, Proc. R. Soc. London **265**, 25 (1998); Theor Popul. Biol. **56**, 243 (1999).

[17] J. C. Koella, Proc. R. Soc. London **267**, 1979 (2000).
[18] D. J. Murrel *et al.*, Oikos **97**, 229 (2002).
[19] F. Rousset and S. Gandon, J. Evol. Biol. **15**, 515 (2002).
[20] J. E. Satulovsky and T. Tome, Phys. Rev. E **49**, 5073 (1994).
[21] H. N. Comins *et al.*, J. Anim. Ecol. **61**, 735 (1992).
[22] M. P. Hassell and H. B. Wilson, see Ref. [2].
[23] R. M. May and R. M. Anderson, Proc. R. Soc. London, Ser. B **219**, 281 (1983).
[24] M. Barthelemy and L. A. N. Amaral, Phys. Rev. Lett. **82**, 3180 (1999); **82**, 5180 (1999).
[25] C. F. Moukarzel, Phys. Rev. E **60**, R6263 (1999).
[26] M. E. J. Newman and D. J. Watts, Phys. Lett. A **263**, 341 (1999).
[27] M. A. de Menezes *et al.*, Europhys. Lett. **50**, 574 (2000).
[28] P. Sen and B. Chakrabarti, J. Phys. A **34**, 7749 (2001).
[29] Y. Bar-Yam, *Dynamics of Complex Systems* (Westview, Boulder, 2003), Chap. 7.
[30] E. Meron, Phys. Rep. **218**, 1 (1992).
[31] D. M. Morens *et al.*, Nature (London) **430**, 242 (2004).
[32] T. Horimoto and Y. Kawaoka, Clin. Microbiol. Rev. **14**, 129 (2001).
[33] H. A. Mooney and E. E. Cleland, Proc. Natl. Acad. Sci. U.S.A. **98**, 5446 (2001).
[34] D. Normile, Science **306**, 968 (2004).