

Evolution in a spatially structured population subject to rare epidemics

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We study a model that gives rise to spatially inhomogeneous population densities in a system of host individuals subject to rare, randomly distributed disease events. For stationary hosts that disperse offspring over short distances, evolutionary dynamics can lead to persistent populations with a variety of spatial structures. A mean-field analysis is shown to account for the behavior observed in simulations of a one-dimensional system, where the evolutionarily stable state corresponds to the solution of a straightforward optimization problem. In two dimensions, evolution drives the system to a stable critical state that is less well understood.

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I. INTRODUCTION

This paper concerns a model recently developed in the context of research into the origins of spatial structure in populations of very simple biological organisms. The model consists of rules for the reproduction, natural death, and death due to disease (or other type of disturbance) of individual hosts living on a homogeneous lattice of sites. Although the model clearly does not accurately represent a real biological system, it illustrates two nontrivial effects that may be relevant for understanding the spatial distribution of some species. The first is the dramatic difference in the statistical steady state of “well mixed” populations (always randomly distributed in space) and “sessile” populations (in which organisms never move from their birthplace). In the sessile case a spatial structure emerges that permits persistence of the species in situations where the well-mixed model leads to rapid extinction [1]. The second effect is that evolutionary dynamics operating on the natural mortality rate selects for spatial structures with special properties. In one dimension (1D), a mean-field calculation of the steady state mortality rate based on a particular optimization principle compares well with simulation results. In 2D, evolution selects for a critical state that remains to be understood.

The discussion below treats a particular population dynamics model from a statistical physics perspective. The relation of the model to issues of ecological or biological interest is beyond the scope of this work. For recent papers and references to more biologically oriented studies, see Ref. [2] and [3]. References [3–7] discuss issues of spatial structure in the context of related ecological models.

The dynamics of a host-disease system necessarily involves several processes with logically distinct spatial scales (area occupied by an individual, distance over which offspring are dispersed, distance over which diseases can be transmitted between hosts) and temporal scales (birth and death rates, the frequency of diseases, the spreading rate of disease, mobility of hosts, rate of evolution of relevant traits). For the purposes of this paper, we are interested in

certain limiting cases that demonstrate some of the qualitatively different types of behavior that are possible. First, we assume that both the rate of disease spreading and the rate at which death occurs once a disease is contracted are extremely rapid compared to the reproduction rates of individual organisms. Our model treats entire epidemics as instantaneous events. Second, we assume that the length scales for offspring dispersal and disease transmission are approximately the same and both are equal to the size of a patch of territory occupied by an individual organism [8]. Modeling the system as a lattice of square patches, each of which can support a single organism, we assume that a new organism must be born on a patch directly bordering that of its parent. We also assume that the disease can spread from an organism on a given patch only to those that live on the patches directly bordering that patch. In addition, we assume that the disease is highly infectious and lethal; all organisms close enough to an infected one do contract the disease and die from it.

One might also introduce a time scale for the movement of organisms. In the well-mixed case, it is assumed that organisms move extremely rapidly compared to the rate of births, but extremely slowly compared to the spreading rate of diseases, and that they pay no attention to the positions of other organisms except to avoid having two occupying the same patch. Thus snapshots of the system separated by a small but finite time interval, would reveal entirely uncorrelated, randomly distributed populations, but these configurations would be frozen on the time scale required for any epidemic, no matter how large, to run its course.

In the sessile case, the organisms do not move at all. Both the rules that offspring live next to their parents and that diseases propagate through local contact only lead to nontrivial spatial correlations in the population. The effects of these correlations on evolutionary dynamics are the principal phenomena of interest in this paper.

II. DETAILED SPECIFICATION OF THE MODEL

The model consists of individual organisms that live on the sites of a regular (linear or square) lattice with periodic

boundary conditions. The total number of sites in the lattice is denoted by L . At any given time, there can be at most one organism on a given site; i.e., each site is either occupied by a single organism or unoccupied. The individual organisms are indexed by an integer label i and each is characterized by two positive real numbers m_i and b_i determining its mortality and reproductive rate.

In a single time step, one site is selected at random. The rate at which sites are selected is denoted by ρ . If the site is occupied by organism i , one of three things can happen.

(1) With probability m_i , the organism dies, leaving the site unoccupied.

(2) With probability b_i , the organism attempts to produce an offspring at a randomly selected nearest neighbor site. If the selected neighboring site is occupied, the offspring is aborted. If it is unoccupied, the offspring j is recruited there. The mortality and birth rate of the offspring are given by $m_j = m_i + \Delta_m$ and $b_j = b_i + \Delta_b$. A mutation $\Delta_x \neq 0$ occurs with probability p_x , in which case Δ_x is uniformly distributed in the interval $[-\mu_x, \mu_x]$, where μ_m and μ_b are constant parameters. If this procedure results in $m_j < 0$, then m_j is set to 0, and similarly for b_j .

(3) With probability d , the organism contracts a disease. The disease spreads extremely rapidly to all neighboring organisms, and then to their neighbors, and so on, until it is stopped by a wall of unoccupied sites. Every organism that contracts the disease dies before the time step is completed. Thus the result of a disease is that an entire connected cluster of occupied sites becomes unoccupied in a single instant.

Note that we must always have $m_i + b_i + d \leq 1$ for all i in order for the probabilities to be well defined. If this condition is ever violated during the evolution of the system, we simply renormalize all the m_i , b_i , and d by a uniform factor and adjust the duration of the time step accordingly.

For most of this paper we consider the case of uniform birth probability; $b_i = b$ for all i and $p_b = 0$. We are particularly interested in the case where d is extremely small compared to b , but large compared to the rate of evolution of the m_i 's. In rough terms, the life cycle of an individual organism is typically short compared to the time between epidemics, but the full population dynamics including the effects of disease occurs against a backdrop of slower evolutionary processes.

III. QUANTITIES OF INTEREST

To characterize the behavior of the system we focus on three quantities: the total number of organisms N in the system; the average mortality rate $\langle m \rangle \equiv \sum_i m_i / N$; and the distribution of epidemic sizes $P(s)$. The size of an epidemic, s , is defined as the number of organisms that die as a result of a single disease event. $P(s)$ denotes the relative frequencies of epidemics with size equal to s observed during a suitably long time interval in the steady state, and is normalized to unity: $\sum_{s=1}^{\infty} P(s) = 1$.

Note that $P(s)$ is also closely related to the cluster size distribution for the system as it is commonly defined in site percolation studies. Let $C(s)$ be the normalized probability that a connected cluster chosen at random (with each cluster

given equal weight) has size s . Since any individual is equally likely to start an epidemic, the probability that an epidemic will have size s is

$$P(s) = sC(s)/N_p, \quad (1)$$

where $N_p \equiv \sum_{s=1}^{\infty} sC(s)$.

IV. STEADY STATES IN ONE DIMENSION

A. The well-mixed case

The 1D case where individuals are randomly distributed in space at every time step can be treated analytically [1]. Let $n(t)$ denote the fraction of sites occupied at time t and assume, for the moment, that $m_i = m$ (and $b_i = b$) for all i , and that $p_b = p_m = 0$ so there is no mutation.

In the infinite system size limit, for any finite set of sites the probability that a given site is occupied is n , independent of the other sites in the set. (If the set is infinite or if the system size is sufficiently small, the probabilities must be correlated to ensure that the density is indeed n .) To compute $C(s)$, suppose a site is selected at random from among the set of occupied sites that are immediately to the right of an unoccupied site. The probability that the cluster containing this site is of size s is equal to the probability that each of the $s-1$ sites directly to its right are occupied and the next one is unoccupied:

$$C(s) = n^{s-1}(1-n). \quad (2)$$

From Eq. (1) we obtain

$$P(s) = sn^{s-1}(1-n)^2, \quad (3)$$

which implies an average epidemic size

$$s_{avg} = \frac{1+n}{1-n}. \quad (4)$$

Note that $P(s)$ decays exponentially for large s .

Since the probabilities of occupation of adjacent sites are independent, the following equation describes the dynamics of n in the large system limit:

$$\frac{dn}{dt} = \rho n [b(1-n) - m - d s_{avg}], \quad (5)$$

where the first factor of n represents the probability of selecting an occupied site on a given time step and the factor of $(1-n)$ enters the birth term because offspring are created only if the target site selected for dispersal is unoccupied.

The steady state value n^* is obtained by setting dn/dt to zero, which yields

$$n^* = 1 - [m - d + \sqrt{(m-d)^2 + 8bd}]/(2b). \quad (6)$$

Regardless of the value of d , smaller m leads to larger n^* . Thus, although the average epidemic size grows with decreasing m , the total population density always increases. If m/b or d/b is too large, then dn/dt is always negative and the population decays to zero, or extinction. Henceforth we

shall assume $d/b \ll 1$ and m/b sufficiently small that extinction due to natural deaths does not occur.

The introduction of mutations in m_i has a dramatic effect on the long-term behavior of the well-mixed model. Evolution will select for smaller values of m_i in this case. Since individuals with different m 's are randomly mixed throughout the system, the rates of birth and the effects of epidemics are the same for all individuals. Thus the individuals with longer life expectancies and their offspring will gradually take over the system. This argument is supported by a standard invasion analysis in which one assumes a small concentration $n_1 \ll 1$ of organisms with $m_i = M_1$ in a sea of organisms n_2 with $m_i = M_2$ [1,9]. Letting $n = n_1 + n_2$, the dynamical equations for the population densities are

$$\frac{dn_x}{dt} = \rho n_x [b(1-n) - M_x - d s_{avg}] \quad (7)$$

for $x=1,2$ and s_{avg} still given by Eq. (11).

Consider a system with $m_i = M_2$ for all i that has reached a steady state. The term in brackets on the right-hand side of Eq. (7) then vanishes for $x=2$. If a very small density $n_1 \ll n_2$ of organisms with $m_i = M_1$ is now added to the system, the right-hand side of the equation for $x=1$ is obviously positive if and only if $M_1 < M_2$. Thus mutant organisms with smaller values of m grow in concentration, while mutants with larger values of m will decay to extinction.

Let $\langle m \rangle(t)$ be the average value of m_i over the current population. In a system where mutation occurs slowly enough that the distribution of m_i 's remains narrow and the system is always near a steady state corresponding to $m_i = \langle m \rangle$ for all i , $\langle m \rangle$ will exhibit steady decay toward zero. In a finite system with sufficiently small d , this implies that $\langle m \rangle$ will eventually become so small that birth events will fill the lattice, forming one big cluster that will be wiped out in an epidemic. Thus the evolutionary dynamics in the well-mixed model drives any finite system to extinction and no steady state is reached.

Note that the probabilistic nature of the model guarantees that extinction will eventually occur in any finite system due to a large fluctuation. When we speak of a statistical steady state here, we mean that the system would persist indefinitely in that state if it were infinite in extent. Indefinite persistence does not occur for the well-mixed model with evolution, since $\langle m \rangle$ never reaches a statistically stable value. Using finite simulations, however, extinctions caused by evolution toward $\langle m \rangle = 0$ and by statistical fluctuation with fixed $\langle m \rangle$ can be distinguished. Extinction occurs much more rapidly in the former case, and the time to extinction grows much more slowly than the system size. (We have not measured the precise dependence.) For a stable $\langle m \rangle$, the time to extinction due to fluctuations grows exponentially with system size.

B. The sessile case

When the organisms are considered immobile, *a priori* analysis becomes more difficult. Because each offspring is born next to its parent and stays there, the probability of a site being occupied depends on the probability that its neighbors are occupied. Even for identical individuals, when m_i

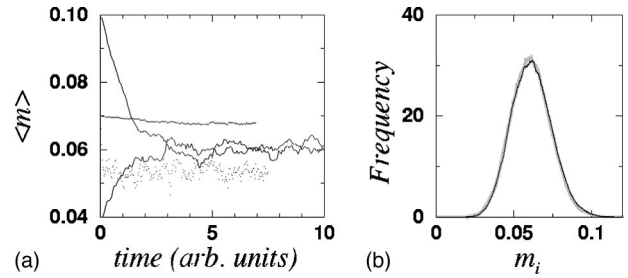


FIG. 1. Evolutionary behavior of mortality rates in the 1D sessile case. Plots are shown for a lattice of 10^4 sites, with $b=1$, $p_m=0.1$, and $d=10^{-4}$. (a) $\langle m \rangle(t)$ for different mutation rates and different initial conditions. The upper trace (dots) is for $\mu_m = 0.001$. The lower trace (dots) is for $\mu_m = 0.02$. The two longer traces (solid lines) are for $\mu_m = 0.005$ with different initial uniform values of m_i . (b) The final distribution of m_i in each of the two $\mu_m = 0.005$ runs, averaged over the time interval from 12 to 24 (not shown) for each run.

$= \langle m \rangle$ for all m , calculation of n^* is beyond the scope of this work. Nevertheless, the correlation between the occupancies of sites decays exponentially with the distance between them, so a self-consistent calculation of the average cluster size is possible, and an understanding of the long-term result of evolutionary dynamics can be obtained.

Numerical simulations reveal a striking difference between the long-term evolution in the sessile and well-mixed cases. In the sessile case, $\langle m \rangle$ fluctuates about a stable value and the system reaches a statistical steady state. (Reference [10] discusses another example of the selection of an intermediate rates in a parasite-host system.) Figure 1(a) shows time traces of $\langle m \rangle$. The longer, solid curves show that different initial conditions with $\langle m \rangle$'s above and below the selected value converge to statistically indistinguishable states. The dotted traces show that the steady state $\langle m \rangle$ depends upon the value of mutation rate. When the mutation rate (or μ_m) is sufficiently small that there is effectively no evolution on the scale of the disease rate d , we expect $\langle m \rangle$ to be independent of μ_m . Figure 1(b) shows the distribution of m_i in the steady state for the two longer runs shown in (a).

Our goal is to calculate the steady state value of $\langle m \rangle$. The key is to identify an appropriate optimization problem that can be solved on the basis of plausible assumptions concerning the cluster size distribution for fixed m . We argue that the relevant quantity to optimize is the *growth rate of a colony surrounded by unoccupied sites*. Consider a group (not necessarily a single cluster) of organisms with $m_i = m$ that is contained within a finite interval on the lattice. Let $x(t)$ be the position of the rightmost organism at time t . The growth rate of the colony is defined as the average value of dx/dt for large t . We wish to find the value of m that maximizes the growth rate for given values of b and d .

Note that maximizing the growth rate is not logically equivalent to minimizing the total death rate from natural causes and diseases. The interior structure of the growing colony is not important in the sessile case in 1D. Since there is no way for a competing colony to invade unoccupied sites between the leftmost and rightmost members of a colony, faster growing colonies win. For completeness, one must

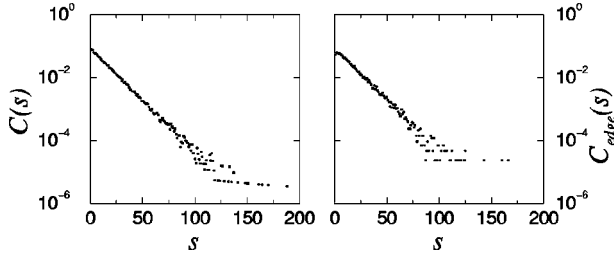


FIG. 2. Cluster size distributions in the 1D sessile case with $b = 1.0$ and $d = 10^{-4}$. Normalized $C(s)$ (left) and $C_{edge}(s)$ obtained from clusters in a growing colony.

also note that when two colonies come in contact, they influence each other through epidemics initiated in the cluster where they are joined, which consists of two “subclusters,” one from each colony. In this configuration, the subcluster from one colony can be destroyed by a disease initiated on the adjoining subcluster from the other colony. The effects of such epidemics are neutral on average. In any given configuration the product of the length of a subcluster and the probability that subcluster will be destroyed by a disease initiated in a subcluster from the other colony is the same for both subclusters (proportional to the product of the sizes of the two subclusters). Thus the epidemics that span the front dividing the two colonies do not, on average, affect the position of the front; the motion is determined entirely by how fast the colonies grow into a gap that separates them.

To estimate the value of m that gives rise to the fastest growth rate, we make three simplifying assumptions. First we make the approximation that the distribution of m_i in the steady state is a δ function at $m_i = \langle m \rangle$. Second, we assume that $C(s)$ decays exponentially in the steady state corresponding to $\langle m \rangle$; i.e., that

$$C(s) = A e^{-\alpha s}, \quad (8)$$

where $A = 1/\sum_{s=1}^{\infty} C(s)$ is a normalization constant. The latter assumption can be checked numerically and is confirmed to the degree illustrated in Fig. 2. Notice that the exponential form holds down to rather small s . If this were not the case, the calculations below would contain additional numerical factors determined by the precise form of the distribution. Finally, we assume that the statistics of clusters at the edge of a growing colony are the same as those in the bulk steady state. This assumption is also borne out by the numerical simulations summarized in Fig. 2.

To calculate c , the average cluster size in the bulk steady state, we begin with the rate equation for n , rewritten in a way that does not depend upon the assumption of uncorrelated site occupation:

$$\frac{dn}{dt} = \rho n \left[b \frac{1}{c} - m - d 2c \right]. \quad (9)$$

The coefficient of b is just the probability of selecting an occupied site at the edge of a cluster and selecting a neighboring unoccupied site for the offspring. Each edge of the cluster contributes $b/2$ to the rate because exactly one of its two neighboring sites is unoccupied. The precise coefficient

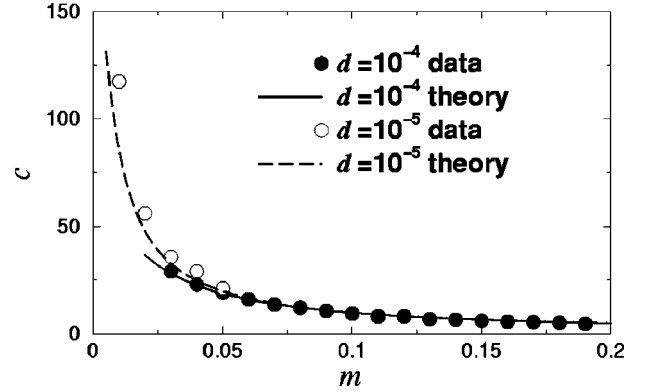


FIG. 3. Comparison of calculated average cluster size with simulations. Plots are shown for the case $b = 1.0$. For each data point, all organisms in the system have the same fixed value of m —there is no evolution.

of d should be $n \sum_s s P(s)$, or $n s_{avg}$, where $P(s)$ is obtained from $C(s)$ via Eq. (1). Using the form of C in Eq. (8), we obtain

$$c = \frac{1}{1 - e^{-\alpha}} \quad (10)$$

and

$$s_{avg} = \frac{1 + e^{-\alpha}}{1 - e^{-\alpha}}. \quad (11)$$

For our present purposes, we consider the case $\alpha \ll 1$, corresponding to the large c 's that arise for $d \ll b$. It then becomes reasonable to write $s_{avg} = 2c$. [Note that for the well-mixed case, Eq. (2) has the form of Eq. (8) with $\alpha = \log n$, and Eqs. (5) and (9), with $2c$ replaced by s_{avg} , are equivalent.]

In the steady state, the right-hand side of Eq. (9) vanishes, implying

$$c = \frac{m}{4d} \left(-1 + \sqrt{1 + \frac{8bd}{m^2}} \right). \quad (12)$$

Figure 3 shows a comparison of this formula with simulation results for $d = 10^{-4}$ and 10^{-5} . Note that for $d \ll m^2/b$ we have $c \approx b/m$ and the cluster size distribution becomes independent of d .

For a colony growing into unoccupied territory, the growth rate is given on average by

$$\frac{dx}{dt} = \rho \left[\frac{b}{2} - m [1 + C(1)g] - d(2c^2 + gc) \right], \quad (13)$$

where g is the average size of a “gap,” a string of unoccupied sites between two clusters. The coefficient of b accounts for the fact that x advances only if the birth occurs to the right of the rightmost organism. The coefficient of m is unity except when the rightmost organism forms a cluster of size 1, in which case x decreases additionally by the size of the gap to its left. The coefficient of d is $\int s(s+g)C(s) ds$, the first factor of s representing the probability of selecting a site in the cluster and the $s+g$ term representing the resulting

TABLE I. Cluster and gap size data for 1D sessile model with different disease rates d . Data were compiled from single runs on a system of 10^4 sites with parameters $b=1.0$, initial m_i all set to 0.1, $p_m=0.1$, $\mu_m=0.001$. Following a transient of 10^5 epidemics, data were averaged over 200 configurations separated in time by 2500 epidemics.

d	Average cluster c	Average gap g	Fraction of size 1 clusters $C(1)$
10^{-3}	6.2	3.1	0.14
10^{-4}	14.5	2.4	0.07
10^{-5}	35.4	2.8	0.03

size of the change in x . [Note the difference between this coefficient, which is order c^2 , and the coefficient of d in Eq. (9). The difference arises because $nL=N_c c$ has been factored out in Eq. (9), where N_c is the number of clusters in the system.]

For $d \ll m$, the distribution of gap sizes is dominated by the statistics of gaps produced by natural deaths rather than diseases. During the periods between diseases we expect the m_i to mutate toward lower values, so $\langle m \rangle$ becomes substantially smaller than b . Moreover, when a disease creates a large gap, that gap can only be filled in from the ends; there is no way to break the large gap into two smaller ones by inserting organisms in the middle of it. The result is that the vast majority of gaps have size 1 and g remains of order unity even though c can be much larger. In addition, since $b > \langle m \rangle$, isolated organisms are rarely produced and do not remain isolated for long, so that $C(1) \ll 1$. These arguments become increasingly unreliable as d is increased, in which case the selected $\langle m \rangle$ increases and c decreases, but we are interested in the limit of small d . Table I shows values obtained from simulations with $d=10^{-3}$, 10^{-4} , and 10^{-5} .

For sufficiently small d (which generates large c), we may neglect the contributions proportional to g in Eq. (13). Figure 4 shows a comparison of Eq. (13) with simulations

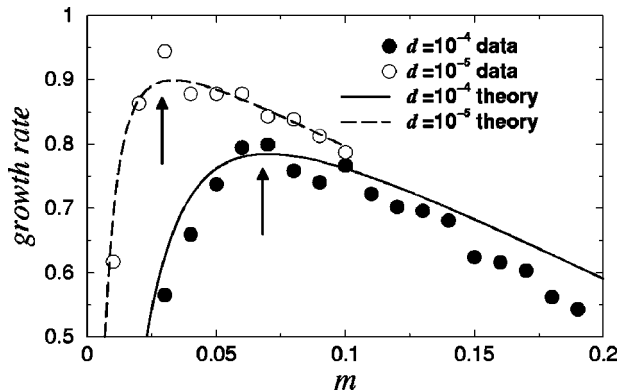


FIG. 4. Comparison of calculated growth rate with simulations. Plots are shown for the case $b=1.0$. For each data point, all organisms in the system have the same fixed value of m —there is no evolution. The theory curves are obtained from Eq. (13) under the assumption that the g terms are negligible. The arrows indicate the average values of m measured in the steady state of systems of 10^4 lattice sites with $p_m=0.1$ and $\mu_m=0.001$.

for $d=10^{-4}$ and 10^{-5} and several values of m , where we have used Eq. (12) to express dx/dt in terms of m . The agreement is quite good.

Substituting for c from Eq. (12) we obtain the following equation for m_0 , the value of m that maximizes dx/dt :

$$m_0^3 + dm_0^2 + 8bdm_0 + 8bd^2 - 4b^2d = 0. \quad (14)$$

Note that b is of order unity. We make the ansatz $m_0 \sim d^{1/3}$ and count powers of d in each term. Neglecting terms of power $4/3$ or higher in d , we find

$$m_0 \approx 4^{1/3} b^{2/3} d^{1/3}, \quad (15)$$

justifying the ansatz. The approximation neglects terms a factor of $d^{1/3}$ smaller than the result.

Equation (15) represents a parameter-free calculation of the value of $\langle m \rangle$ that should be selected by evolution for sufficiently small d . This may be compared with average values measured in simulations. For $d=10^{-4}$ and 10^{-5} (with $b=1$), we calculate $m_0=0.074$ and 0.034 , respectively. Simulations on a lattice of 10^4 sites with $p_m=0.1$ and $\mu_m=0.001$ yield $\langle m \rangle=0.068 \pm 0.002$ and 0.029 ± 0.003 . Note from Table I, however, that for these values of d , the quantity $C(1)g$ is not negligible, being of order 0.1. When the measured values are used in Eq. (13), the calculated values of m_0 shift to 0.070 and 0.033. The reasonably good agreement with simulations indicates that the evolutionary dynamics is consistent with our picture of the 1D system. In particular, the system retains a finite correlation length (or average cluster size) for any nonzero d , and a treatment neglecting correlations in the sizes of adjacent clusters is adequate for understanding the steady state.

V. STEADY STATES IN TWO DIMENSIONS

A. Self-organized criticality

The analyses of the well-mixed and sessile cases in 1D do not transfer easily to two dimensions. In the well-mixed case, however, the basic phenomenology is the same. As in 1D, the lack of spatial correlation among closely related offspring implies that it is always advantageous to live longer. Evolution drives $\langle m \rangle$ toward zero, resulting in increasingly large fluctuations in the population size and rapid extinction.

In the sessile case, the situation is quite different. As in 1D, colonies of organisms with lower m_i become more dense and epidemics propagate more easily through them, which provides a mechanism for suppressing the evolutionary pressure toward $\langle m \rangle=0$. For fixed $m_i=\langle m \rangle$, two qualitatively different behaviors are observed in simulations. (See Fig. 5.) For relatively small $\langle m \rangle$ as in Fig. 5(a), the population is best described as consisting of distinct colonies that grow into large empty spaces and sometimes merge. The density within a colony is sufficiently high that almost all the organisms in it die in a single epidemic, with just a few survivors around the edges. It then takes a long time, comparable to the disease rate, for the new colonies spawned by the survivors to fill in the emptied regions. For relatively large $\langle m \rangle$ as in Fig. 5(b), the population density remains low and is roughly ho-

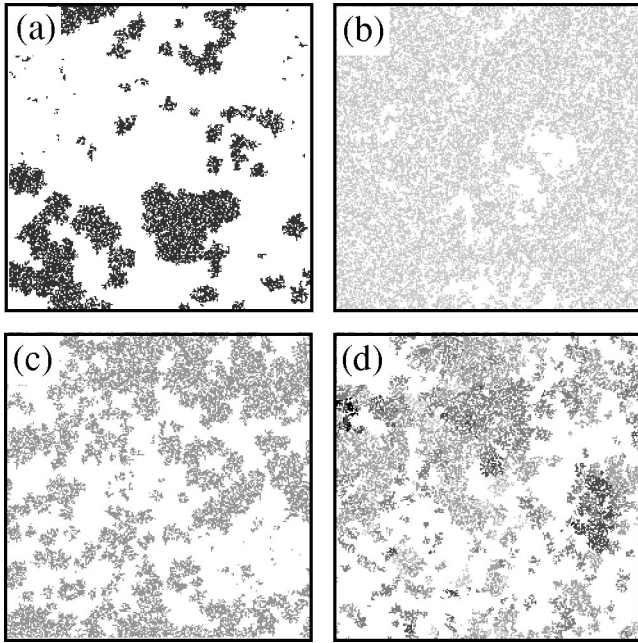


FIG. 5. Snapshots of a 256×256 system after approximately 10^4 epidemics. Parameters are $b=0.5$ and $d=10^{-5}$. Gray scale indicates the value of m_i . Darkest gray indicates m_i near 0.09; lightest gray indicates m_i near 0.2. (a) All m_i fixed at 0.09: dense, separate colonies. (b) All m_i fixed at 0.20: homogeneous density with small holes due to epidemics. (c) All m_i fixed at 0.15: critical structure corresponding to the average value of m selected by evolution. (d) Evolved system with $p_m=0.1$ and $\mu_m=0.01$.

mogeneous in space. Epidemics carve out relatively small empty regions, which are filled on time scales that may be slow compared to the birth rate, but are fast compared to the disease rate.

In two dimensions, colonies are not protected from invasion the way they are in 1D. Any gaps in the boundary of the growing colony permit invasions by organisms with different m_i . Thus there is less justification for using the colony growth rate as a criterion for selecting the evolutionarily stable state. Moreover, if the same argument applied above to the 1D case is attempted for 2D, the self-consistency of the approximations is not maintained. In addition to the technical difficulty of estimating the number of sites available for new offspring in a typical cluster, a fundamental difficulty is encountered: the assumption of an exponentially decaying $P(s)$ is violated in the evolutionarily stable state. For sufficiently small d and large system size, the system evolves to a state where $P(s)$ has a power-law tail, and may be classified as an example of a *self-organized critical state* [11]. The power law in $P(s)$ may be cut off either by the finite system size or by nonzero d . (See Ref. [12] for analysis of another host-pathogen model in which evolution of the disease transmissibility drives the system to a critical state.)

Figure 6 shows the distribution of epidemic sizes in the steady state, which has a power-law regime $P(s) \propto s^{-\nu}$ with $\nu \approx 1$. For this figure, $d=3 \times 10^{-6}$ was chosen (by trial and error) such that the cutoff due to d was roughly the same as the cutoff due to the finite system size. Plots for $d=1 \times 10^{-5}$ and 1×10^{-6} are shown for comparison. Studies with

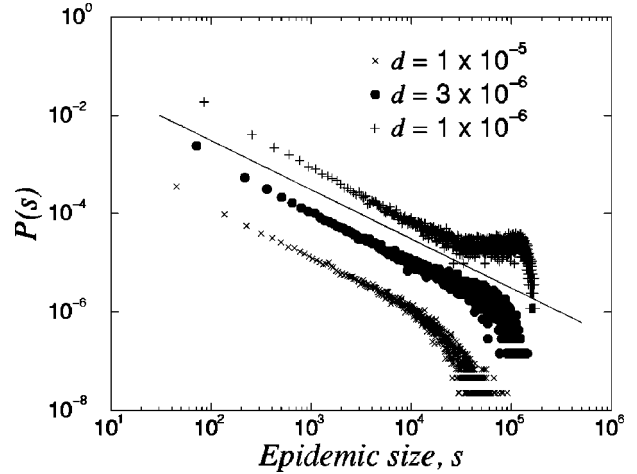


FIG. 6. Normalized epidemic size distributions from simulations in the 2D sessile case on a 512×512 lattice for the three values of d indicated in the legend. Parameters are $b=0.5$, $p_m=0.1$, and $\mu_m=0.02$. The central curve shows the power law corresponding to the smallest d that permits data uncorrupted by finite size effects. The upper (lower) curve is shifted up (down) by a factor of 10 for clarity. The solid line of slope -1 is a guide to the eye.

smaller system sizes indicate that the peak at large sizes observed for the smallest d would disappear if the system size were increased. For the $d=3 \times 10^{-6}$ run, $\langle m \rangle$ began at $0.2b$, reached a value of roughly $0.3b$ after approximately 1000 epidemics, then fluctuated between $0.30b$ and $0.32b$ over the course of the following 50 000 epidemics.

We note that the observed value of ν is consistent with that measured by Henley for a ‘‘self-organized percolation’’ (SOP) model [13]. The SOP model differs from ours in three respects: (i) new hosts are generated at each unoccupied site at a steady rate, independently of the presence of other hosts in the system; (ii) the rate at which disturbances occur, d in our model, is assumed to approach zero; and (iii) there is no evolutionary mechanism in the system. We have not yet gathered enough data to determine whether the two models are in different universality classes.

It may be tempting to relate the self-organized critical behavior to ordinary site percolation in 2D, but the connection is tenuous. As Fig. 5(c) clearly shows, the spatial structure of the population in the steady state is far from homogeneous. There are relatively dense regions where diseases have not appeared for some time, and there are sparse regions that are in the process of being refilled after large epidemics have swept through. Thus we do not expect the average density in the critical state to be directly related to the site percolation threshold.

B. Evolving b and m together

It is instructive to consider what happens in this model when b and m are *both* allowed to evolve. The system passes through three distinct regimes: (1) an initial rapid adjustment of $\langle b \rangle$ and $\langle m \rangle$ to bring their ratio to a critical value as in the case where b is held fixed ($\langle b \rangle / \langle m \rangle \approx 2.5$); (2) a period in which $\langle b \rangle$ and $\langle m \rangle$ grow in tandem with their ratio roughly constant, generating a power-law distribution of epidemic

sizes with an increasing cutoff size; and (3) a period in which the epidemic size distribution is dominated by finite size effects (rather than cut off due to nonvanishing d), population density fluctuations become extremely large, and eventually extinction becomes probable.

During the last period, b and m increase faster than exponentially in time, and $\langle b \rangle / \langle m \rangle$ increases rapidly. (A similar situation occurs in the 1D system as well.) In effect, the dynamics is dominated by the rapid evolution between disturbance events, which leads to extremely high birth rates. The epidemic size distribution in this regime is dominated by system-wide events, one of which eventually causes the extinction. The time to extinction by this mechanism does depend upon the initial value of d , but due to the exponential growth of the birth and mortality rates during the second regime listed above, it is impossible to delay the extinction for long. It is clear from the previous section, however, that a persistent population can be obtained if a maximum birth rate is imposed, which is clearly a reasonable restriction for a biological system.

VI. CONCLUSION: SPATIAL STRUCTURE, KIN SELECTION, AND OPTIMIZATION PRINCIPLES

The emergent spatial structure in the model we have investigated is of interest for both biologists and physicists. First, it is a crucial ingredient in the evolutionary stability of this simple system. Second, it is an example of a nonequilibrium physical structure that arises as a solution to a complex optimization problem. We conclude with some remarks on each of these issues.

In the well-mixed model, if a disease infects an individual it is transmitted to a neighbor that is a randomly selected individual. However, in the sessile model neighbors are likely to be closely related because of the contact process nature of reproduction. When a disease infects an individual, the individual passes it on to relatives, and this interaction brings up issues of “kin selection.” In the well-mixed model, selection drives the mortality rate to zero because an individual’s fitness is just a matter of producing more offspring than other individuals. In the sessile model, an individual’s fitness is influenced by siblings passing diseases on to one another, and an effective strategy for passing on more

surviving offspring is to die and leave holes that stop disease transmission. Previous kin selection studies have focused primarily on the competition between siblings that results when offspring are produced locally [14–16]; these models typically examine evolution for dispersal, and the concepts behind kin selection may help explain the dispersal of offspring even when such dispersal is risky. In a similar manner individuals in our model have a decreased fitness when offspring are produced locally because death by disease is mediated by neighbors. In our case we have not incorporated evolution of dispersal; however, if we were to fix mortality rates, we strongly suspect (and preliminary runs show) that selection would favor higher dispersal. Unfortunately, higher dispersal causes the mortality rate to evolve to lower levels, which in turn causes extinction.

The present study may also be relevant for future investigations into the origin of power-law damage distributions in evolving systems. Two intriguing but rather different explanations for the generic occurrence of power-law distributions have been suggested in the recent physics literature. The theory of self-organized criticality (SOC) explains the power laws by placing the model in a broad class of systems that exhibit avalanche dynamics when driven very slowly [11]. In such models, power-law distributions of avalanche sizes emerge at the slowest driving rates for generic parameter values. A second proposal is that power laws are indicative of a system that has resulted from a design process that produces states that minimize the expected damage in an uncertain environment, states exhibiting “highly optimized tolerance” (HOT) [17]. One may argue that natural selection in the biological world acts to create HOT configurations at the organism or ecosystem level.

Our 2D results are characteristic of SOC systems. Evolution of the mortality to a critical value is roughly analogous to the evolution of the slope of a granular pile to the value that produces power-law distributions of avalanches [18]. It is interesting to note, however, that the 1D model with the same dynamical rules does not yield SOC. Our current model is not expected to give rise to HOT states, at least in part because the system is spatially homogeneous. In future studies we plan to seek evidence for rudimentary HOT states resulting from evolution in systems with a slow gradient in the disease rate.

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