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Dynamics and Genealogy of Strains in Spatially Extended Host–Pathogen Models

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We examine the dynamics of evolution in a generic spatial model of a pathogen infecting a population of hosts, or an analogous predator–prey system. Previous studies of this model have found a range of interesting phenomena that differ from the well-mixed version. We extend these studies by examining the spatial and temporal dynamics of strains using genealogical tracing. When transmissibility can evolve by mutation, strains of intermediate transmissibility dominate even though high-transmissibility mutants have a short-term reproductive advantage. Mutant strains continually arise and grow rapidly for many generations but eventually go extinct before dominating the system. We find that, after a number of generations, the mutant pathogen characteristics strongly impact the spatial distribution of their local host environment, even when there are diverse types coexisting. Extinction is due to the depletion of susceptibles in the local environment of these mutant strains. Studies of spatial and genealogical relatedness reveal the self-organized spatial clustering of strains that enables their impact on the local environment. Thus, we find that selection acts against the high-transmissibility strains on long time-scales as a result of the feedback due to environmental change. Our study shows that averages over space or time should not be assumed to adequately describe the evolutionary dynamics of spatially distributed host–pathogen systems.

Introduction

Evolution in host–pathogen systems is a topic of great interest because pathogen generation times are short, and hence adaptation can occur rapidly (Anderson & May, 1982; Levin *et al.*, 1997). There are many medically and ecologically relevant examples of pathogen evolution, such as the emergence of drug-resistant strains (Schrag & Perrot, 1996) and the decreased virulence of introduced control agents (Fenner, 1983). Host–pathogen systems are typically not well mixed, but rather are spatially distributed. Mutant pathogen strains arise locally, and considerable variation in type is possible from one locality to another (Pielou, 1974). Moreover, host and pathogen densities are inhomogeneous and dynamic. It has become apparent in recent research that inhomogeneities in spatially distributed populations can fundamentally change the dynamics of ecological systems (Kareiva, 1990; Tilman & Kareiva, 1997), and host–pathogen systems are no exception (Mollison, 1977; Comins *et al.*, 1992). Spatial extent can also

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fundamentally change evolutionary dynamics (Sayama *et al.*, 2000). We note, in particular, that the characteristics of the pathogen can greatly affect the spatial and temporal dynamics of the host, which in turn affects the evolution of the pathogens. Host–pathogen systems can be considered as a type of predator–prey system, and these characteristics and our investigation also apply to predator–prey systems.

There have been a number of recent theoretical studies relevant to the discussion of evolution in spatially extended host–pathogen systems (Rand *et al.*, 1995; Boots & Sasaki, 2000; Haraguchi & Sasaki, 2000), including studies of predator–prey analogues of host–pathogen systems (Savill & Hogeweg, 1998). We will extend these studies by focusing on the dynamics and genealogy of strains. Most pathogenic species are found to consist of a number of distinct strains, distinguished from one another by a mutation or set of mutations. Our study of strain dynamics will allow us to discuss the relationship of local spatial effects and the long-term behavior of the system. In particular, we will show that mutant strains can arise that (1) increase in number rapidly over many generations; (2) are spatially clustered; (3) become extinct over longer times due to the local extinction of hosts. The evolutionary dynamics can be understood as a selection process that favors different types at different time-scales. There are two regimes, with a sharp transition between them: a short time regime in which mutant strains with high reproduction ratios dominate, and a long time regime in which environmental feedback causes those strains to be selected against.

The Model

We consider a simple spatially extended model of a pathogen spreading through a host population. This model allows for the mutation of a single quantitative trait, the transmissibility of the pathogen from one host to another. Similar to other recent studies (Savill & Hogeweg, 1998; Haraguchi & Sasaki, 2000; Boots & Sasaki, 2000), mutation is part of the dynamics of the model. The evolving population is composed of different types of pathogens. We will study the mechanisms that give rise to this composition.

The class of models we consider assume that reproduction of hosts and infection of pathogens occur locally in space (e.g. by contact or airborne transmission rather than waterborne transmission). We also assume that infection is ultimately fatal, so that our models are relevant to the case where infection is at least usually fatal and not to the case where infected hosts normally recover with or without immunity. As a model of predator–prey systems, it is relevant to the case where predators are capable of causing the local extinction of their prey, and when a local population is "infected" with predators it cannot recover. Our main results are insensitive to the detailed aspects of the model, including not only parameter values but also the inclusion of additional factors such as uninfected host death, limited local movement of hosts, occasional long-range dispersal of hosts, and different lattice structures. Such changes affect specific values of measured quantities, but not the generic behavior of the model. Although it could be considered a model of specific systems, our investigation is one of generic properties common to many spatially distributed systems.

THE MODEL WITHOUT MUTATION

We use a spatially extended susceptible-infected-removed (SIR) model (Sato *et al.*, 1994; Rand *et al.*, 1995; Andjel & Schinazi, 1996) with local reproduction of hosts. The model is a probabilistic cellular automaton with possible states 0 (empty), S (susceptible host), and I (infected host). At each time step, healthy hosts reproduce into each empty neighboring cell with probability g; this occurs independently for each neighboring cell. To model the carrying capacity of the environment, each cell can have at most one host individual. Alternately, each cell can be considered to represent local populations, either absent or at carrying capacity. An infected host dies with probability v (virulence). Finally, an infected host I causes a neighboring uninfected host to become infected with probability τ (transmissibility). The state transition

probabilities are

$$P(0\to S) = 1-(1-g)^n,$$
$$P(S\to I) = 1-(1-\tau)^m,$$
$$P(I\to 0) = v, \qquad (1)$$

where n is the number of uninfected host neighbors, and m is the number of infected neighbors. Rand *et al.* (1995) note that asynchronous updating does not significantly change the dynamics. The model differs from that in Haraguchi *et al.* (2000) only in the use of discrete time and the lack of death of susceptibles.

Figure 1 shows snapshots of simulations after the long-term behavior is established, revealing how the geometry changes with differing transmissibility, virulence and reproduction rate. The system is spatially inhomogeneous, with host and pathogen distributed patchily, and spatial correlations in the distribution and reproduction of host and pathogen. As in all host–pathogen models, the pathogen must have a minimum transmissibility in order to propagate. In this model, the pathogen can drive the host to extinction if it exceeds a certain transmissibility (Sato *et al.*, 1994). Thus, there is a minimum and maximum transmissibility at which the pathogen and host can coexist. The region of parameter space in which there is coexistence was obtained by Haraguchi & Sasaki (2000).

Rand *et al.* compared simulations of the spatially extended model with a mean field (well-mixed) version of the model. In both cases, they considered the dynamics of a system with pathogens of transmissibility τ, and introduced pathogens with transmissibility $\tau\pm\Delta\tau$ ($\Delta\tau = 0.01$). In the well-mixed version, the higher-τ population always invades, driving the lower-τ one to extinction but itself surviving. By contrast, in the spatially extended version, there is a value of τ above which the mutant population does not successfully invade.

THE MODEL WITH DYNAMIC MUTATION

In real systems, characteristics of the pathogen can mutate, and this must be considered when making statements about the long-term behavior of a host–pathogen system. In order to investigate the evolutionary dynamics of the system, mutation should be incorporated into the dynamics of the model (Savill & Hogeweg, 1998; Haraguchi & Sasaki, 2000). The transmissibility becomes a variable quantitative trait which is part of an infected individual's state, rather than a global parameter. The states become $0, S$ and I_τ (host infected with pathogen of transmissibility τ). Mutation can be introduced by assuming that there is a probability μ that when a pathogen of transmissibility τ spreads, the newly infected individual has transmissibility $\tau\pm\varepsilon$:

$$P(0\to S) = 1-(1-g)^n,$$
$$P(S\to I_\tau) = \left[1-\prod_{\tau'}(1-\tau')^{m_{\tau'}}\right] \times \left[\frac{\frac{\mu}{2}p_{\tau-\varepsilon}+\frac{\mu}{2}p_{\tau+\varepsilon}+(1-\mu)p_\tau}{\sum_{\tau''}(\frac{\mu}{2}p_{\tau''-\varepsilon}+\frac{\mu}{2}p_{\tau''+\varepsilon}+(1-\mu)p_{\tau''})}\right],$$
$$P(I_\tau\to 0) = v, \qquad (2)$$

where $p_\tau = 1-(1-\tau)^{m_\tau}$ and m_τ is the number of infected neighbors of transmissibility τ. The assumption of incremental mutation will be extended later.

Behavior of the Model with Mutation

Mutation causes pathogens of differing transmissibility to coexist on the lattice. Haraguchi & Sasaki (2000) found that there is an evolutionarily stable value of τ, and that this value is somewhat lower than the maximum value for which pathogen and host can coexist. We show in Fig. 2 snapshots of simulations with dynamic mutation for different combinations of parameters. Each snapshot is taken after 10 000 generations, a time long enough to allow the evolved transmissibility to reach a stable value, aside from fluctuations. We find that in the presence of mutation, host and pathogen coexist for a wide range of virulence and host reproduction rate. The evolutionarily stable value can be substantially below the maximum possible value. Figure 3 shows two typical time series of the average, minimum and maximum transmissibility of the population. In each case, the average transmissibility is seen to approach

an evolutionarily stable value after several thousand generations, and then stay within 5% of this value. (For some combinations of parameters, the average varies over time by as much as 17%, but varies by no more than 5% for most.) We show in Fig. 3(a) and (b) that it will reach the same value whether the system starts with pathogens with transmissibility above or below this value.

Dynamics of Strains

An important clue to the evolutionary dynamics of the system can be seen in Fig. 4, a density plot of the distribution of pathogen transmissibilities over time. Most pathogens are within 0.05 of the evolutionarily stable value of $\tau = 0.3$. However, there is an additional temporal structure that is apparent in the figure: the population appears to have offshoots that persist for tens to hundreds of generations before disappearing. These offshoots are part of the characteristic behavior of the evolving population, even after it has converged to the evolutionarily stable average transmissibility. In the plot, an example of such an offshoot occurs at time $T = 26\,000$. The offshoots are visual traces of genetically related pathogens—strains. In particular, they reflect the presence of mutant strains which substantially exceed the evolutionarily stable value of τ, but then go extinct.

In the remainder of this paper, we will analyze the evolutionary dynamics by examining features related to properties of strains. We will examine: (1) the reproductive success of mutant pathogens and their descendants; (2) the lineage histories of strains; (3) the effect of pathogen phenotype on the local environment of susceptibles; and (4) the relationship of spatial and genealogical structure. Our analysis elucidates the mechanisms by which the population comes to be dominated by strains of intermediate transmissibility. We believe similar mechanisms may be at work in many natural systems.

In order to distinguish the identity of strains, we track the genealogy of pathogens. A strain is the set of individuals descended from a single common ancestor. One can choose any ancestor, but when studying evolutionary dynamics, it is particularly useful to consider a mutant strain to begin when a mutation occurs. A mutant descendant of this first mutant can be considered the beginning of a new strain.

To obtain adequate sampling of high-transmissibility cases, which are rare under incremental mutation because of selection, we modify the evolutionary model given in eqn (2) to have large, uniformly distributed mutations: mutations to a random value of transmissibility between 0.2 and 1.0 with a mutation rate $\mu = 0.002$. This rate is low enough that it is rare that a mutant strain itself mutates again. This modification does not significantly change the evolutionarily stable transmissibility.

REPRODUCTIVE SUCCESS OF MUTANTS AND THEIR DESCENDANTS

To gain insight into the reproductive success of mutants, we examine the net reproduction ratio R, which is defined as the average number of other individuals infected during the course of an individual's infection in a population where the infection is present. R has been commonly used as a measure of fitness in theoretical evolutionary studies of host–pathogen and many other ecological systems (Fisher, 1930; Brommer, 2000). In spatially homogeneous treatments of host–pathogen systems, selection will tend to increase R, and R increases with transmissibility (May & Anderson, 1983). The reason is that if two pathogens have the same number of susceptible neighbors (a condition that applies to the homogeneous version of the model), the one with the higher transmissibility has a greater probability of infecting. This is indeed the case of the reproduction ratio of initial mutants in the spatially distributed host–pathogen model; Fig. 5(a) plots the expected number of offspring of a mutant one generation after it arises. R increases rapidly and roughly linearly with τ, from a value of 1 at $\tau = 0.3$ to a value of 1.4 at $\tau = 1$. This must occur due to the equivalence of the environments into which all the mutants are introduced on average.

On the other hand, this conflicts with the observation that, in the model, pathogens with intermediate transmissibility dominate. The reproduction ratio averaged over many generations, in fact, has a peak at the evolutionarily

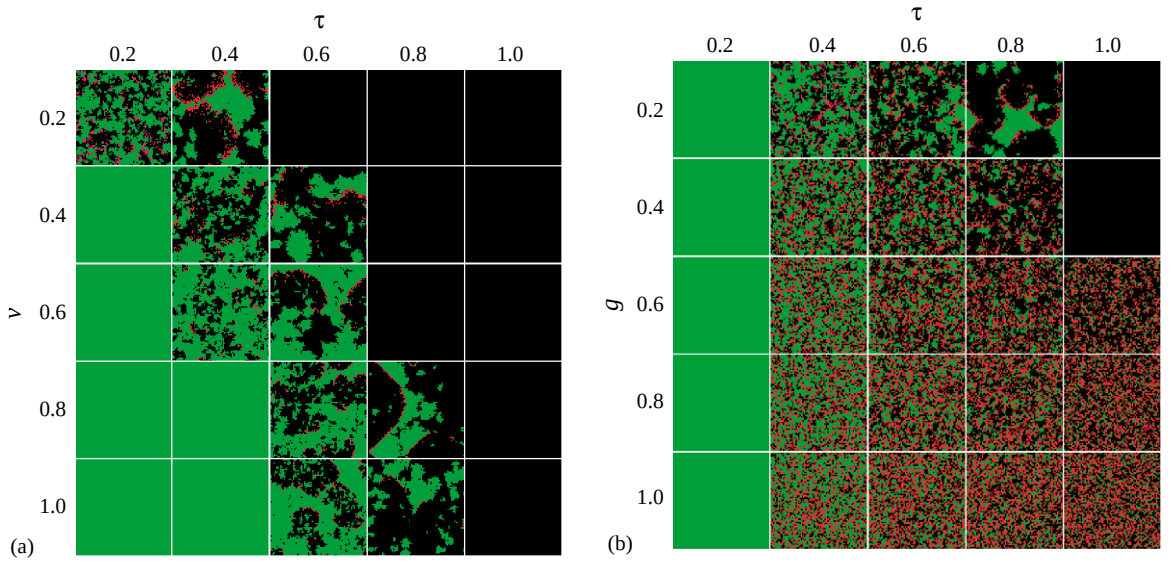

FIG. 1. Snapshots of the host-pathogen model with no mutation. Each of the 25 blocks in (a) and (b) is from a simulation with distinct parameter values. Green represents healthy hosts, red represents infected hosts, and black represents empty sites. The snapshots for those parameters for which hosts, but not pathogens, survive after 100 generations appear completely green. For those that appear black, the outcome is uncertain and can be either pathogen extinction or extinction of both pathogen and host. We use an $L \times L$ square lattice with periodic boundary conditions and a von Neumann neighborhood (north, south, east and west neighbors); here $L = 80$. (a) As a function of transmissibility τ and virulence v, with host reproduction rate g held at 0.05. (b) As a function of τ and g, with v held at 0.5.

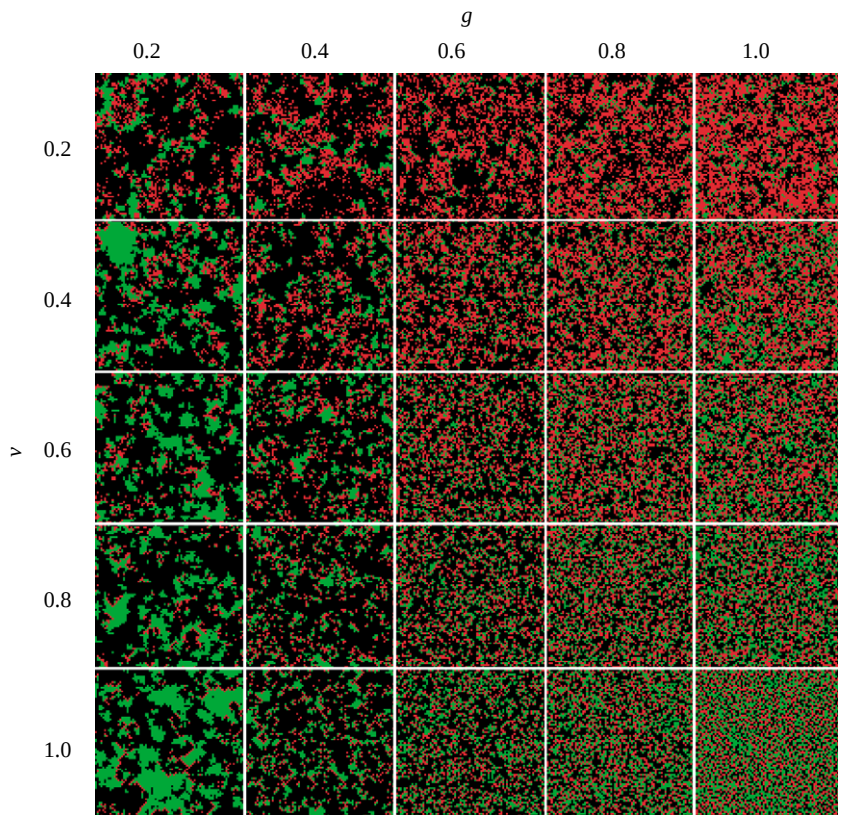

FIG. 2. Snapshots of the host–pathogen model with mutation after 10 000 generations. The transmissibility has evolved to an evolutionarily stable value. Each of the 25 blocks represents a simulation with different values of g and v as indicated. The dimension of the lattice L is 175, the mutation rate μ is 0.15 and the mutation increment ε is 0.005.

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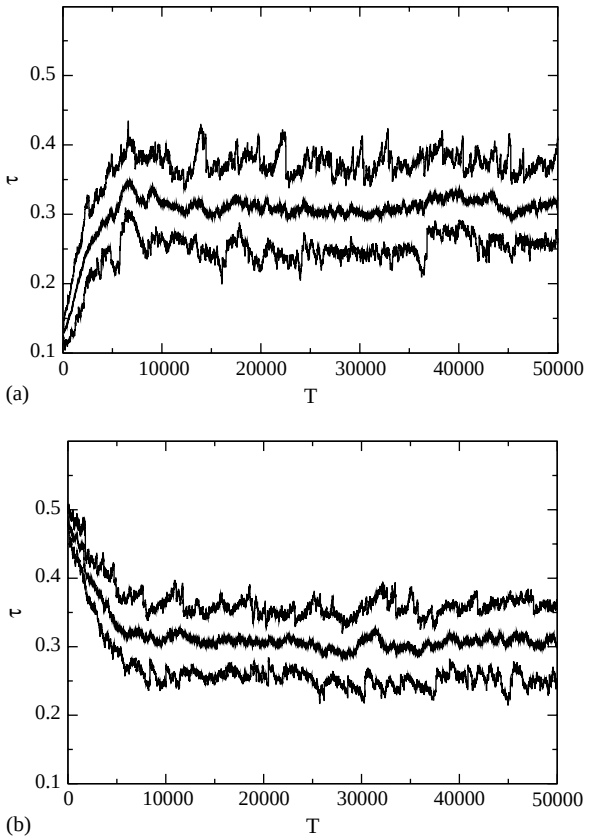

FIG. 3. Time series of transmissibility τ in the population, showing average, maximum, and minimum values. (a) τ is started at 0.15, below the evolutionarily stable value of 0.3. τ evolves upward to reach the evolutionarily stable value within 7000 generations. (b) τ started at 0.49; τ evolves downward to the evolutionarily stable value, again within 7000 generations. The virulence v is 0.2, host reproduction rate g is 0.05, lattice size L is 250, mutation rate μ is 0.15, and mutation increment ε is 0.005. All of the following figures use these parameters unless otherwise noted.

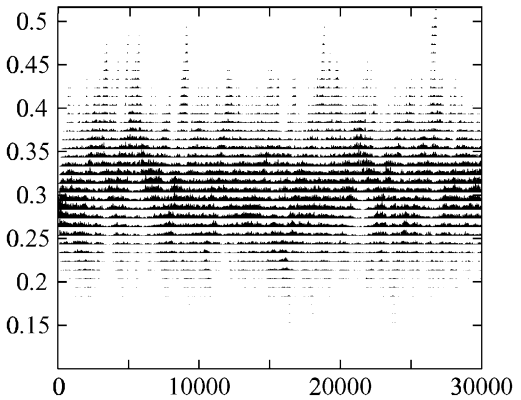

FIG. 4. Time series of the distribution of τ. Each vertical slice of this three-dimensional plot shows the distribution of transmissibilities at a given moment in time. Note that strains temporarily exceed the evolutionarily stable value of 0.3 but then go extinct. These correspond to the long excursions, for example at $T = 26\,000$. Parameters are as in Fig. 3.

stable transmissibility. Figure 5(b) shows R averaged over many generations. For values of τ centered on the evolutionarily stable value of 0.3, R is slightly greater than 1. (A self-sustaining stable population without mutation would have a reproduction ratio of exactly 1; here the reproduction ratio is greater than 1 because of mutated offspring which have lower reproduction ratios.) R is significantly lower for both higher and lower values of the transmissibility, consistent with the observation of the evolutionarily stable type.

Thus, selection does not act instantaneously to favor pathogens of intermediate transmissibility. The difference between the time-averaged and mutant reproduction ratios points out the need to consider the reproductive success of pathogens over the lineage history of a strain.

LINEAGE HISTORY OF STRAINS

Figure 6 plots a measurement of the average population size $N(T, \tau)$ of a strain as a function of transmissibility τ and number of generations T since the beginning of the strain. Strains with higher τ grow much faster than ones with lower τ for a large number of generations. They reach a maximum and start declining after about 30 generations, and eventually go extinct. This is consistent with both Fig. 5(a) and (b). We plot in Fig. 7 a normalized net reproduction ratio of the mutant strain over the course of its lineage history $R(T, \tau) = N(T, \tau)^{1/T}$, representing the average number of offspring per generation from the beginning of the strain to time T. This measure of reproductive success can be seen to decrease below one for types significantly greater than the evolutionarily stable value at around $T = 200$ generations. The high-τ strains can grow in the short term, but die out in the long term, despite the fact that they have a higher net reproduction ratio for a significant time after they are first introduced.

Considering the reproductive success of strains as a function of time allows one to

characterize evolutionary systems in which the reproductive success of mutants differs on different time-scales. The populations of such systems can contain a mixture of strains, each of which is successful on a different time-scale. Figure 8 shows the most successful type as a function of time for the host–pathogen system. The distribution $P(\tau)$ of types, $\tau \neq \tau_{es}$, is given for low mutation rates by

$$P(p) = \frac{\mu \int_{T=0}^{\infty} N(T,\tau)}{n_{es} + \sum_{\tau' \neq \tau_{es}} \mu \left[\int_{T=0}^{\infty} N(T,\tau')\right]},$$

where n_{es} is the average number of individuals of the evolutionarily stable type.

RELATIONSHIP OF PATHOGEN TYPE AND SPATIAL STRUCTURE

To understand the lineage history of strains, it is helpful to examine the relationship of pathogen phenotype and the local environment of susceptible hosts. Figure 9 shows a mutant strain 50 generations after it arose, with a value of τ that is significantly above the evolutionarily stable type. This strain has arisen from a single ancestor at time t_0 which mutated from a lower value of τ. By time $t_0 + 250$, the strain has become extinct. The figure suggests that the local environment is significantly altered by the mutant type.

Figure 10 shows enlarged views of two panels of Fig. 1, where it can be seen that the local configuration of susceptibles that an average pathogen finds itself in changes with τ. Thus, the characteristics of the pathogen shape the host patches that they find themselves in. While a complete characterization of the local envriron-ment is difficult, we can consider the local density of susceptible hosts adjacent to an infected host as a first approximation. Using this measure, the effect of the pathogen phenotype on the local environment of susceptible hosts is apparent in Fig. 1, where only a single type is present in any one simulation. Figure 11 shows that the characteristic length scale of host patches also changes with τ.

Strains that arise by mutation are generally located in an area whose local environment has been determined by the strain it mutated from.

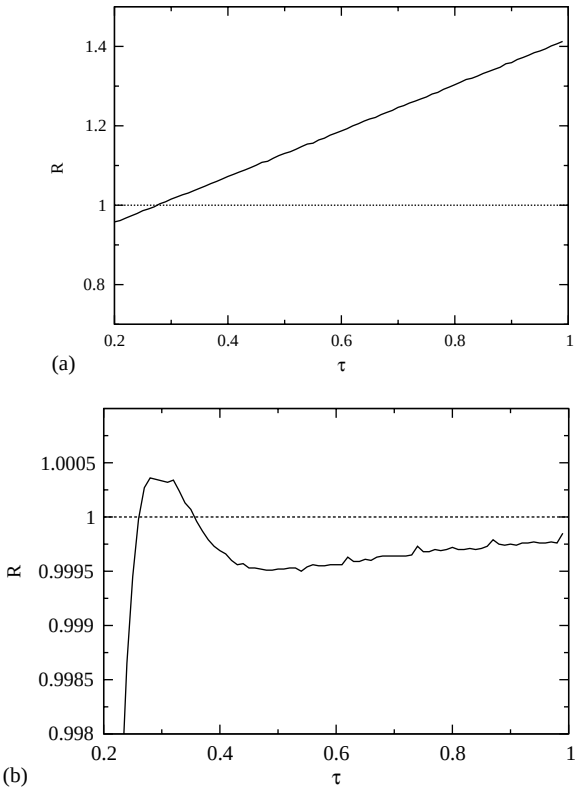

FIG. 5. The net reproduction ratio R in an evolving population, as a function of transmissibility τ. The dominant type has reached its evolutionarily stable value of $\tau = 0.3$. (a) For mutants, showing the expected number of offspring of a mutant one generation after it arises. (b) For all pathogens, averaged over 3×10^7 generations.

We find, however, that after the first mutant arises, the new strain changes the local environment, measured by the local density, to the environment that is characteristic of it. Figure 12 shows the local contact rate of susceptible hosts as a function of the time since the strain arose, where the change can be seen to take about 40 generations. Figure 13 shows an average over time of the local contact rate for the evolving system and compares it with the system with only one type. We see that, for all values of τ, the local contact rate for mixed systems (with mutation) is the same as that for homogeneous systems (without mutation), even though in the mixed system, many strains exist on the same lattice and individuals are constantly mutating.

SPATIAL AND GENEALOGICAL STRUCTURE

In order for the strains to systematically modify the local spatial structure of their

environment, we expect that they are at least partially spatially segregated. To study this directly, we track genealogical distance in the following way. For each pair of individuals, the number of generations since their most recent common ancestor defines a genealogical distance between them, or coalescence time (Donnelly & Tavare, 1995). To track genealogical relatedness, we map each pair of individuals to their genealogical distance from each other. At each time step, the genealogical distances are updated so that the genealogical distance of each pair is one generation more than the genealogical distance of the respective parents of the pair. Two offspring of the same parent receive a genealogical distance of one.

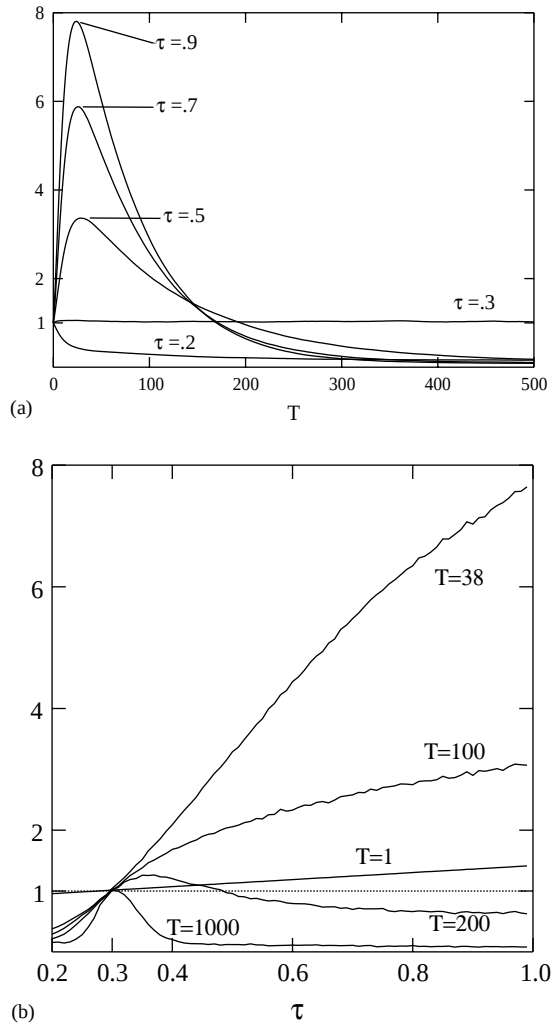

FIG. 6. The average population of mutant strains as a function of time since the first (ancestor) mutant arose. (a) The average population is plotted as a function of time T, with curves for various transmissibilties τ. High-transmissibility strains initially grow rapidly, but reach a maximum and then decline after about 30 generations. (b) The average population is plotted as a function of τ, with curves for various T. Between $T = 100$ and 200, it can be seen that selection changes from favoring higher-transmissibility mutant strains to favoring strains of intermediate transmissibility. In order to collect data for all τ, mutations are large—mutants' transmissibility is set to a random value between 0.2 and 1. μ is 0.002; other parameters are as in Fig. 3.

We show in Fig. 14, a representation of the spatial structure of genealogical distance. In this picture, the colors show the degree of genealogical relatedness to a particular pathogen. The left and right panels show this for two different individuals at the same time in an evolving population, simulated using incremental mutations. Figure 15 plots the average genealogical distance as a function of physical distance between the two individuals in space, averaged over all pairs at that physical distance. The genealogical distance is small for short distances, reflecting the likelihood that nearby individuals are genetically related because of the locality of reproduction. It increases for longer spatial distances, indicating that strains are physically clustered on short and medium time-scales. We note that physical distance remains small for genealogical distances of hundreds of generations. This implies that the typical lifespan of mutant strains (200 generations) is small enough so that strains go extinct before spreading throughout the space.

Conclusion

We have shown that the evolutionary dynamics of a generic host–pathogen model can be understood by characterizing the reproductive success of strains on different time-scales. Neither spatially averaged properties nor time-averaged local properties can reveal the mechanisms responsible for the long-term composition of the population.

In particular, we have shown that (a) the time-averaged reproduction ratio has a maximum at intermediate transmissibilities, but the reproduction ratio of mutants when they first arise increases with transmissibility; (b) the lineage history of high-transmissibility strains shows

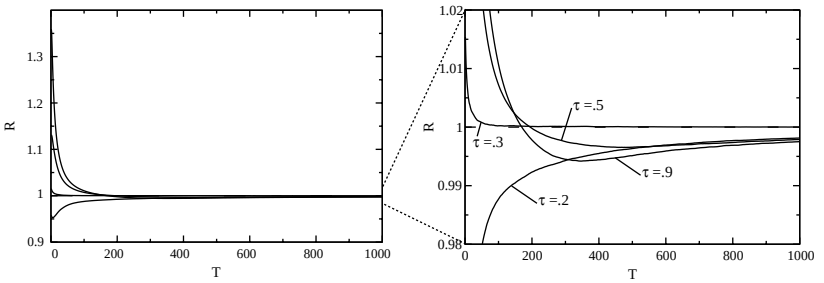

FIG. 7. The normalized per-generation reproduction ratio of a mutant strain $R(T,\tau) = N(T,\tau)^{1/T}$. Within 200 generations, this value drops below 1 for transmissibilities significantly greater than the evolutionarily stable type. The plot on the left shows $R(T,\tau)$ for the full range of R. To show that $R(T,\tau)<1$ when τ is not the evolutionarily stable value, the right plot shows the same data with a truncated R-axis. Parameters are as in Fig. 6.

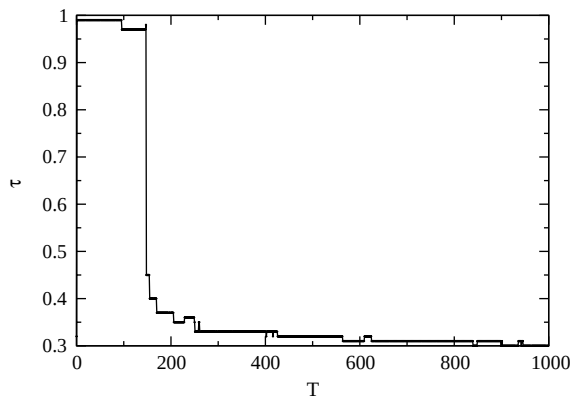

FIG. 8. The most successful type $\tau_{opt} = max_{\tau}(R(T,\tau))$ as a function of time since the beginning of the strain, showing that types with τ close to 1 dominate on short time-scales, and types with τ close to 0.3 dominate on long time-scales, with a sharp transition at about $T = 150$. Parameters are as in Fig. 6.

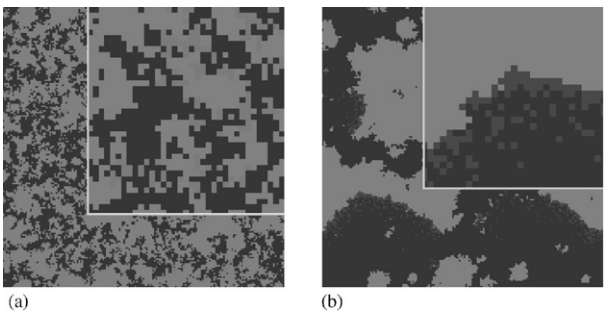

FIG. 10. Magnification of the model with no mutation, showing an example of how transmissibility governs the local spatial structure of susceptible hosts the pathogens find themselves in. The transmissibility τ is (a) 0.2, (b) 0.45.

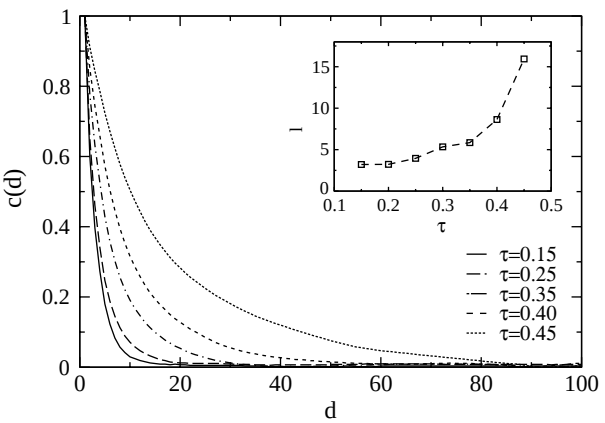

FIG. 11. Spatial auto-correlation $c(d)$ of healthy hosts as a function of distance d. The decrease with distance shows the characteristic length scale of host patches. The inset shows the characteristic length scale l of the patches, the length at which the correlation drops to $1/e$, as a function of t.

that they grow faster than intermediate-transmissibility ones for a significant length of time before declining and going extinct; (c) the characteristics of the pathogen determine the spatial distribution of the host, and hence the environment that the pathogens find themselves in. Strains that reproduce and grow over several generations change the local environment over time, and their local effect is the same whether or not there are other types on the lattice; (d) the genealogical distance between pathogens is correlated with their spatial distance.

In summary, we find that high-transmissibility strains change their environment in a way that is ultimately detrimental to their survival. However, there is a significant time delay before this change leads to their extinction. During this time, these strains take advantage of the host spatial structure generated by the evolutionarily stable type and are able to propagate rapidly before going extinct. Since new mutant strains arise by mutation, it is possible for strains which,

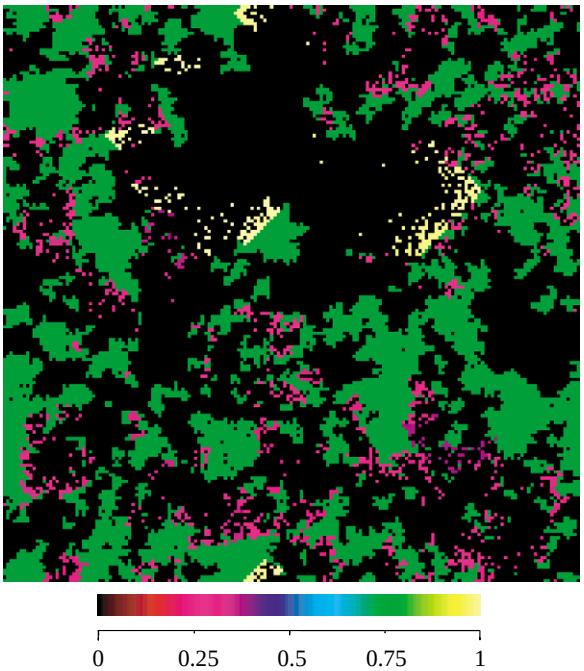

FIG. 9. A snapshot of the model with mutation, with τ shown as color as indicated in the legend. Yellow represents a high-transmissibility ($\tau = 0.9$) mutant strain which arose 50 generations ago. Hosts are shown as dark green. The lattice size L is 175. We see that the mutant strain is spatially clustered and is depleting the hosts from its local environment. This environmental change leads to the eventual extinction of the strain.

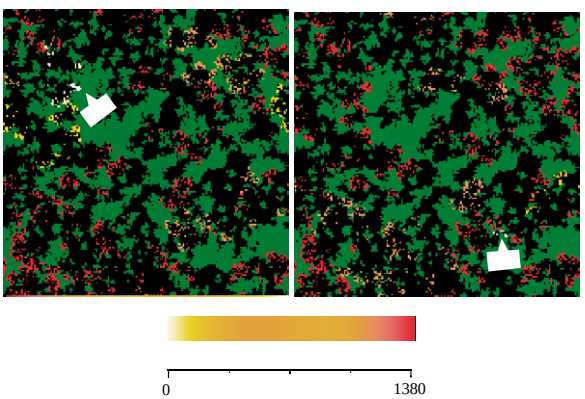

FIG. 14. Genealogical distance between individuals in space. Distance from the individual marked by the arrow is shown as color. Yellow indicates pathogens that have a recent ancestor in common with the pathogen indicated by an arrow; red represents ones that have the most distant common ancestor (a distance of 1380 generations). Pathogens of the same color are not necessarily related to each other. The two plots show relatedness from two different individuals at the same time.

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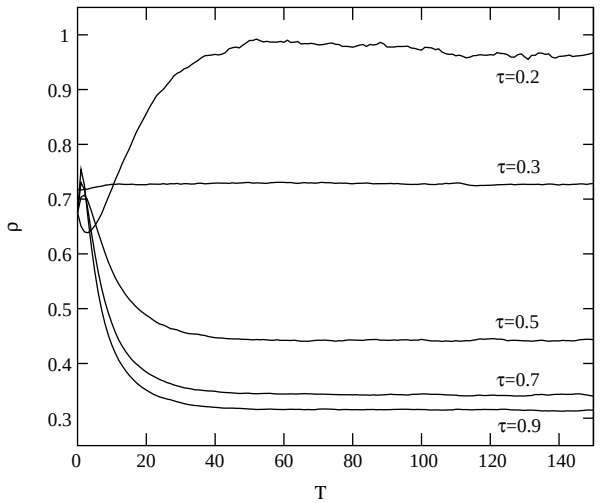

FIG. 12. The contact rate, ρ (the number of neighboring susceptible hosts), averaged over all individuals infected with a strain of a particular type, as a function of time since the strain first arose. Within 40 generations, the local environment in the vicinity of the strain has been changed from the value characteristic of the evolutionarily stable type to a value characteristic of the mutant strain. This characteristic value is plotted in Fig. 13. Parameters are as in Fig. 6.

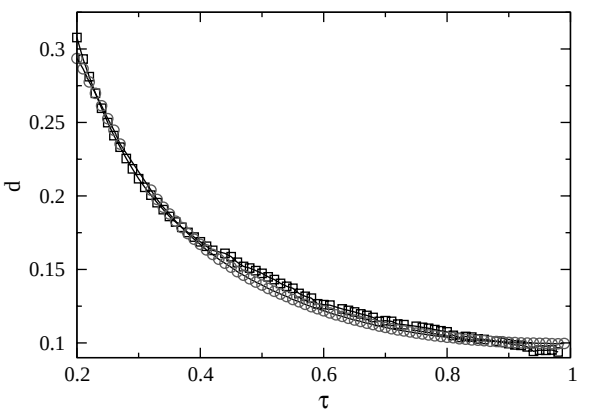

FIG. 13. The contact rate, ρ, as a function of transmissibility τ. Squares represent data measured in non-mutating populations where all pathogens are of the same type, and circles represent data taken in evolving populations, where many other strains with different τ are present. Parameters are as in Fig. 6. (Data for high τ in homogeneous systems are more variable since the pathogen drives the host to extinction and hence a shorter time series is available).

by themselves, would drive the host to extinction, to be continually present in a population if mutations are frequent enough. In systems like the one studied, reproductive success must be thought of as a function of time. The composition of types of this, and, we believe, many natural systems, can be understood as a mixture of types, each of which is successful on a particular time scale.

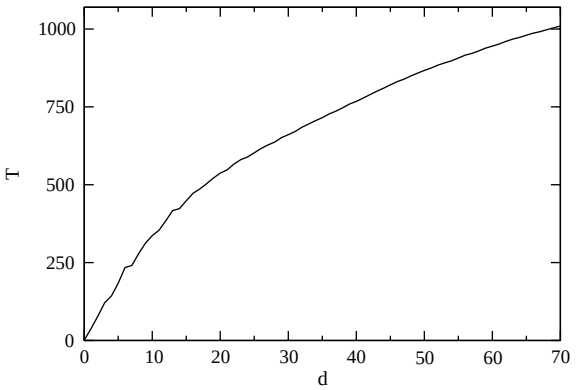

FIG. 15. The average number of generations since a pair of individuals had their most recent common ancestor (coalescence time), as a function of their distance from each other. Since the size of the system being simulated is only 100×100, the levelling off of the curve may be due to the finite system size. Other parameters are as in Fig. 3.

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