Epidemic spreading with immunization and mutations

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The spreading of infectious diseases with and without immunization of individuals can be modeled by stochastic processes that exhibit a transition between an active phase of epidemic spreading and an absorbing phase, where the disease dies out. In nature, however, the transmitted pathogen may also mutate, weakening the effect of immunization. In order to study the influence of mutations, we introduce a model that mimics epidemic spreading with immunization and mutations. The model exhibits a line of continuous phase transitions and includes the general epidemic process (GEP) and directed percolation (DP) as special cases. Restricting to perfect immunization in two spatial dimensions, we analyze the phase diagram and study the scaling behavior along the phase transition line as well as in the vicinity of the GEP point. We show that mutations lead generically to a crossover from the GEP to DP. Using standard scaling arguments, we also predict the form of the phase transition line close to the GEP point. The protection gained by immunization is vitally decreased by the occurrence of mutations.

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I. INTRODUCTION

The modeling of epidemic spreading is a fascinating subject both in theoretical biology and statistical physics far from equilibrium [1,2]. A possible approach is the study of stochastic models that mimic the competition of infectious spreading and recovery by certain probabilistic rules. Depending on the rates for infection and recovery, the disease may either spread over the population or disappear after some time.

The simplest models for epidemic spreading assume that the individuals live on the sites of a *d*-dimensional lattice. At a given time, each individual can be either infected or healthy. The system evolves according to certain probabilistic rules that resemble infection of nearest neighbors and spontaneous recovery. If the susceptibility to infections is sufficiently large, the epidemic will spread while for low susceptibilities it will die out.

In most models, it is assumed that the contagious disease is transmitted exclusively by direct contact. This means that the disease, once extinct, cannot appear again. The system is then trapped in a fully recovered state, which can be reached but not be left. Such a state, where the system is dynamically trapped, is called *absorbing*. The two regimes of spreading and extinction are usually separated by a so-called absorbing phase transition. In the supercritical regime, where infections dominate, the epidemic may spread over the entire system, reaching a fluctuating stationary state with a certain average density of infected individuals. In the subcritical regime, where recovery dominates, the system eventually reaches the fully recovered absorbing state.

Close to the transition, the temporal evolution of the spreading process is characterized by large-scale fluctuations. Theoretical interest in epidemic spreading stems from the fact that this type of critical behavior is universal, i.e., it does not depend on the details of the model under consideration. The classification of all possible transitions from fluctuating phases into absorbing states is currently one of the major goals of nonequilibrium statistical physics [3,4].

Epidemic models without immunization and quenched randomness, where the pathogen (e.g., a virus) is transmitted to nearest neighbors, generically belong to the universality class of directed percolation (DP) [5]. The DP class is very robust and plays an important role not only in epidemiology but also in various other fields such as Reggeon field theory [6], interface depinning [7], population growth [8], catalytic reactions [9], or flowing sand [10] (for a review of possible experimental applications, see Ref. [11]).

As a next step towards a more realistic description of epidemic spreading, one can take the effect of immunization or weakening by infections into account [8,12–15]. The simplest way of implementing immunization is to change the initial susceptibility of an individual after the first infection and keep it constant thereafter. In the case of perfect immunization, where each individual can be infected only once, one obtains the so-called ''general epidemic process'' (GEP) [1,2,8]. It differs from DP insofar as the disease can only spread in those parts of the system which have not been infected before. Thus, starting with a single infected site in a nonimmune environment, the disease typically propagates in the form of a solitary wave, leaving a region of immune sites behind. The transition between spreading and extinction of the disease is a critical phenomenon which in this case belongs to the universality class of dynamical isotropic percolation [16]. Note that unlike DP models, a GEP running on a *finite* system has no fluctuating active state, instead the process terminates when it reaches the boundaries.

In nature, however, immunization is a much more complex phenomenon. For example, the protection by immunization may abate as time proceeds. Even more importantly, the strategy of immunization competes with the ability of the contagious pathogen to mutate so that it can no longer be recognized by the immune system of previously infected individuals, weakening the effect of immunization. The aim of the present work is to introduce and study a simple model which mimics epidemic spreading with immunization and

mutation. To this end, we generalize the model described in Refs. [14,15] by including mutations as well as a mechanism for the competition between different species of pathogens. The model is controlled by three parameters, namely, a first infection probability, a reinfection probability controlling the effect of immunization, and a probability for spontaneous mutations. Moreover, the model is defined in such a way that it includes DP and GEP as special cases.

The paper is structured as follows. In Sec. II, we first define the model. Restricting our analysis to the case of perfect immunization in two spatial dimensions, we discuss the phase diagram and the qualitative behavior of the model in Sec. III. In Sec. IV, the critical behavior at the phase transition line is studied in detail while crossover phenomena in the vicinity of the GEP point are investigated in Sec. V. The paper ends with concluding remarks in Sec. VI.

II. SIMULATION MODEL

Our model is meant to describe the spreading of an infectious disease that evolves as follows. Individuals can be healthy or infected with a certain pathogen. During their illness infected individuals may infect or reinfect neighboring individuals with certain probabilities. Moreover, there is a probability that a pathogen mutates during transmission. Because of the enormous number of possible mutations, one usually obtains an entirely new type of pathogen which has not been involved before. To simplify the model, we also assume that each individual can be infected at a given time by no more than a *single* type of pathogen. If the individual is exposed simultaneously to several competing pathogens, one of them is randomly selected.

In more technical terms, we assume that individuals live on the sites of a d-dimensional simple cubic lattice. Pathogens are represented by positive integers and each site keeps track of all species of pathogens by which it has been infected in the past. Therefore, the state of a site is characterized by an integer n together with a dynamically generated list of all previous types of infections. $n=0$ denotes a healthy individual while for $n>0$ the individual is infected with pathogen n. The model evolves in time by synchronous updates, i.e., in each time step the whole lattice is updated in parallel as follows.

Each infected individual at time t transmits its pathogen n to its $2d$ nearest neighbors (target sites). The transmitted pathogen reaches the target site at time $t+1$ with probability p (q) if it is a first infection (reinfection) with this species. If a target site is exposed to several transmitted pathogens, one of them is randomly selected with equal weight. Before infecting the target site, the selected pathogen mutates with probability λ, replacing n by a new integer number (drawn from a global counter) which has not been used before. In case of a first infection, the type of pathogen is added to the list of species against which the site will be immune in the future. Time of illness is a single time step.

III. PHASE DIAGRAM

In what follows, we restrict our analysis to the special case of perfect immunization $q=0$. In this case, the model is controlled by only two parameters, namely, the probability of first infections p and the probability of mutations λ. Moreover, we restrict ourselves to the case of $d=2$ spatial dimensions. The corresponding phase diagram is shown in Fig. 1. It comprises an active phase, where the epidemic spreads, and an inactive phase, where the disease dies out so that the system eventually enters the fully recovered absorbing state. Both phases are separated by a curved phase transition line.

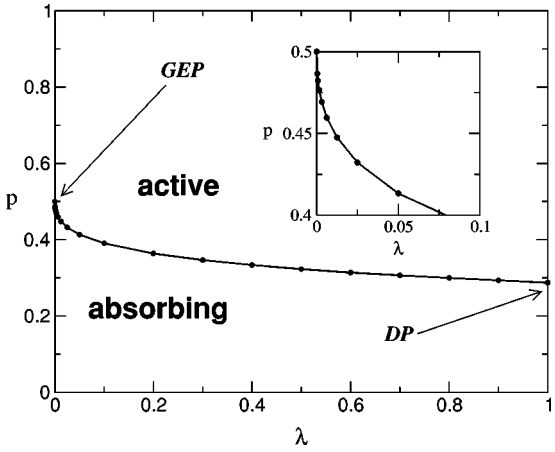

FIG. 1. Phase diagram of the model. The active and the inactive phases are separated by a curved line of continuous phase transitions, connecting the points of critical GEP and critical DP (see text). Circles mark the numerically determined critical points. The inset shows the phase transition line for small λ.

We first note that the end points of the phase transition line correspond to well-known special cases. On the one hand, for $\lambda=0$ the model reduces to the GEP on a square lattice [8], provided that only one type of pathogen is involved. In this case, the critical value of p is exactly given by $p_c=1/2$ [16]. On the other hand, for $\lambda=1$ all transmitted pathogens mutate, i.e., the target sites are always infected with a new species so that immunization has no influence. It is easy to see that in this case the model reduces to directed bond percolation on a square lattice with $p_c\simeq 0.287\,34$ [17]. The other points on the phase transition line in Fig. 1 were determined numerically using seed simulations (see Sec. IV A).

As already discussed in the Introduction, mutations generally weaken the effect of immunization. This explains why the critical value of p decreases monotonically with increasing mutation probability λ. Figure 1 clearly shows that introducing mutations in a GEP has a strong influence on p_c, especially if λ is very small. Since the phase transition line approaches the GEP point with infinite slope (see inset in Fig. 1), a tiny increase of λ reduces the corresponding critical value of p dramatically. This indicates that mutations are a relevant perturbation and thus the spreading behavior of the model for $\lambda>0$ is expected to differ from that of the GEP. On the other hand, for larger values $\lambda\gtrsim 0.1$ the critical value of p decreases only moderately with increasing λ, indicating that the behavior of the model in this region is essentially the same as for $\lambda=1$, where the transition is known to belong to DP.

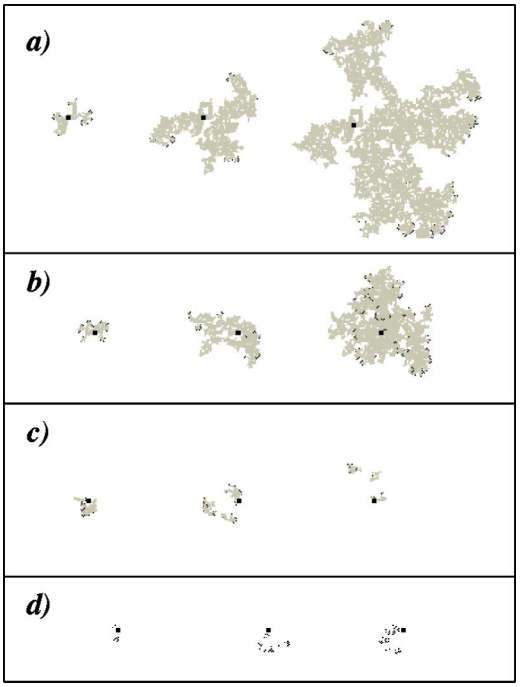

FIG. 2. Snapshots of seed simulations at the phase transition. The black square marks the position of the seed. Each row shows a simulation for a fixed value of λ and times $t=30$, $t=100$, and $t=225$ (from left to right). From (a) to (d), the values of λ are 0 (GEP), 0.003 125, 0.05, and 1 (DP). Black (gray) dots denote active (immune) individuals (details in the text).

Figure 2 shows snapshots of simulations at the transition for different times and various values of λ. Infected individuals are represented by black dots. Healthy individuals which are immune against at least one active type of pathogen are marked by gray dots. The snapshots suggest the following qualitative behavior.

(a) For $\lambda=0$ (GEP), the process creates a growing cluster of immune sites with infected individuals located at the edges. In the active phase, this region is compact while it is fractal at the transition.

(b) For small λ, the disease first behaves as a GEP (for $t\leqslant 100$) while for larger times the spreading behavior changes and clearly differs from the GEP. There still is a region of immune individuals but it does no longer provide efficient protection, since it is reinvaded by mutated pathogens.

(c) Increasing λ further, the process changes its appearance already at an early stage. There are only small patches of immune sites and the process reminds more of DP than GEP.

(d) Finally, for $\lambda=1$ every transmitted pathogen mutates into a new one. In this case, immunization has no influence and the process reduces to DP.

Based on these phenomenological observations, we expect the model to behave initially in the same way as a GEP. After a certain time mutations become relevant, allowing former immune areas to be reinvaded. Especially close to the transition, the process survives long enough to reach this crossover time. The visual appearance of the process is then increasingly similar to that of a DP process. The time it takes to observe the crossover from the GEP to DP grows with decreasing λ and eventually diverges in the limit $\lambda\to 0$.

IV. CRITICAL BEHAVIOR ALONG THE PHASE TRANSITION LINE

Phase transitions into absorbing states are usually characterized by simple scaling laws. In models for epidemic spreading, an important quantity is the probability $P_s(t)$ that an epidemic starting from a single infected seed in a healthy and nonimmune environment survives at least until time t.

Let us first recall the standard scaling laws for the survival probability. Let $\Delta_p=p-p_c$ denote the distance from criticality. If $|\Delta_p|\ll 1$, the survival probability first decays as a power law $P_s(t)\sim t^{-\delta}$ until a certain time scale $\xi_{||}\sim|\Delta_p|^{-\nu_{||}}$ is reached from where on it either saturates at $P_s(\infty)\sim\Delta_p{}^{\delta\nu_{||}}$ for $\Delta_p>0$ or decays exponentially for $\Delta_p<0$, reaching a spatial extension $\xi_\perp\sim|\Delta_p|^{-\nu_\perp}$. Assuming scaling invariance, this behavior can be described in terms of a scaling form

$$P_s(t)=t^{-\delta}\Phi(\Delta_p t^{1/\nu_{||}}), \tag{1}$$

where $\Phi(\zeta)$ is a scaling function with the asymptotic behavior

$$\Phi(\zeta)\sim(\text{const},\zeta^{\delta\nu_{||}},0) \quad \text{for} \quad \zeta\to(0,+\infty,-\infty) \tag{2}$$

such that the time dependence drops out for $\zeta\to+\infty$. The scaling function $\Phi(\zeta)$ and the triplet of critical exponents $(\nu_{||},\nu_\perp,\delta)$ are believed to characterize the universality class of the phase transition under consideration. This scaling form is known to be valid both for the GEP and DP, although with different sets of critical exponents [18]:

$$(\nu_{||},\nu_\perp,\delta)\simeq\begin{cases}(1.506,4/3,0.092) & \text{for GEP}\\ (1.295,0.734,0.451) & \text{for DP.}\end{cases} \tag{3}$$

For the density ρ in DP, a similar scaling form as in Eq. (1) is valid.

We now analyze the critical behavior of the model, assuming that the scaling form (1) is valid everywhere in the vicinity of the phase transition line. Although the qualitative discussion in Sec. I suggests DP behavior for $0<\lambda\leqslant 1$, we note that this would be a nontrivial result, since the so-called DP conjecture does not apply in the present case. The DP conjecture [19] states that phase transitions in two-state systems with a reachable absorbing state and short-range interactions belong to DP, provided that memory effects, nonconventional symmetries, and quenched disorder are absent. Contrarily, the present model has many absorbing states and memorizes previous infections over a long time.

A. Seed simulations

Seed simulations start with a single infected site at the origin in a nonimmune environment. Each run is stopped when it dies out or reaches a preset maximum time. We average over many runs with different realizations of ran-

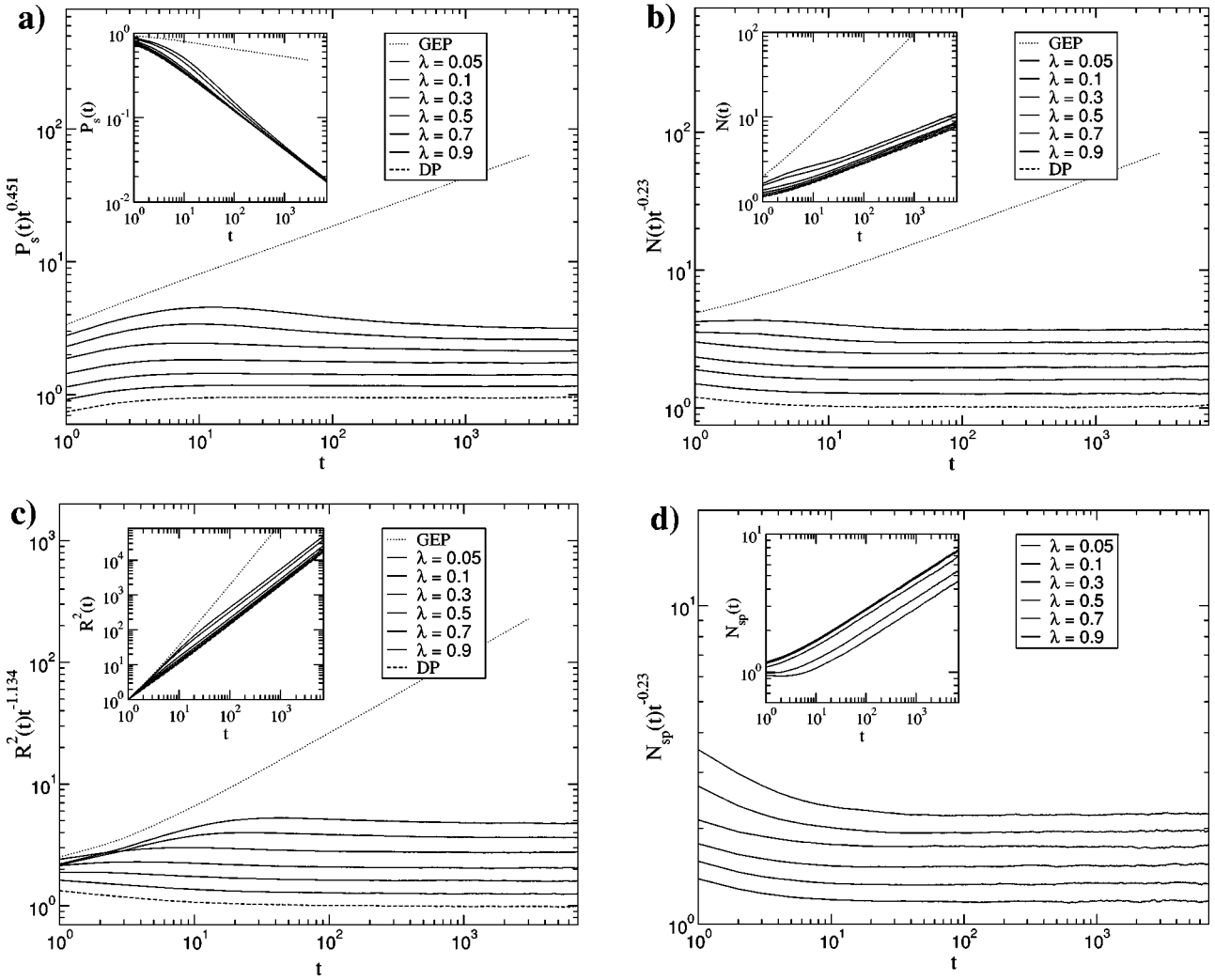

FIG. 3. Seed simulations: $P_s(t)$, $N(t)$, $R^2(t)$, and $N_{\mathrm{sp}}(t)$ at the phase transition for different values of λ. The data are multiplied by the expected asymptotic power law and vertically shifted. The insets show the original data.

domness. In order to eliminate finite size effects, the lattice is always chosen large enough so that the epidemic never reaches its boundaries. As usual in this type of simulation we measure the survival probability $P_s(t)$, the number of active sites $N(t)$ averaged over all runs, and the mean square spreading from the origin $R^2(t)$ averaged over all active sites in surviving runs [20]. The scaling form (1) implies that these quantities vary at criticality algebraically as

$$P_s(t)\sim t^{-\delta}, \quad N(t)\sim t^{\theta}, \quad R^2(t)\sim t^{2/z}, \tag{4}$$

where $z=\nu_{\parallel}/\nu_{\perp}$ and $\theta=d/z-2\delta-1$ for the GEP and $\theta=d/z-2\delta$ for DP [21].

Figure 3 shows our results of seed simulations along the phase transition line. Note that for each site, one has to deal with the whole list of previous infections which is continuously updated. This makes these simulations numerically challenging.

In order to determine the critical threshold $p_c(\lambda)$, we kept λ fixed and varied p until $P_s(t)$ displayed the expected slope of DP in a log-log plot (dashed lines in Fig. 3). Although this procedure is in favor of DP scaling, the mere fact that it works consistently for all quantities in Eq. (4) confirms that the transition does belong to DP for any $0<\lambda\leqslant 1$ while GEP scaling can be ruled out. The data also show the expected crossover. For small λ, the curves roughly display the slope expected for the GEP (dotted lines) before they cross over to DP, confirming the crossover scenario discussed in the previous sections. Thus the introduction of mutations in a GEP is a relevant perturbation in the sense that it changes the asymptotic critical behavior of the model. At the phase transition, the interplay between immunization and mutations drives the system towards DP.

The involvement of different species of pathogens in a spreading process with mutations allows one to introduce the number of active species $N_{\mathrm{sp}}(t)$ as an additional order parameter. As shown in panel (d) of Fig. 3, the number of active species at criticality increases in the same way as the number of infected individuals. Thus their quotient tends to a λ-dependent constant $c(\lambda)=\lim_{t\to\infty} N/N_{\mathrm{sp}}$.

B. Full-lattice simulations

In this type of simulation, we use a finite system with periodic boundary conditions. The initial configuration is a

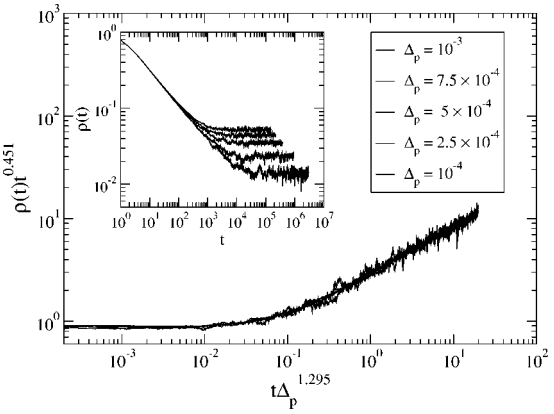

FIG. 4. Data collapse of $\rho(t)$ for $\lambda=0.5$ using the critical exponents of DP, i.e., $\delta=0.451$ and $\nu_\parallel=1.295$.

fully occupied lattice where all individuals are infected by different types of pathogens. (If all sites were occupied with the same type of pathogen, the process would immediately be trapped in the absorbing state.) We measure the density of active sites $\rho(t)$ and the density of active species $\rho_{\rm sp}(t)$. In the case of directed bond percolation $\lambda=1$, it is known that the density of active sites decays as $\rho(t)\sim t^{-\delta}$. Exemplarily, we performed a full-lattice simulation for $\lambda=0.5$ at the critical point, confirming this type of decay with $\delta=0.451$. Moreover, like in seed simulations, the density of active species also decays as $\rho_{\rm sp}(t)\sim t^{-\delta}$.

The three exponents δ,θ,z in Eq. (4) that govern the spreading behavior at the transition depend only on two of the three independent critical exponents that are needed to characterize the universality class of DP [see Eq. (3)]. To roughly check the value of the third independent exponent, we performed off-critical full-lattice simulations for $\lambda=0.5$ and different values of $0<\Delta_p\ll1$. As is shown in Fig. 4 using the critical exponents δ and $\nu_\parallel$ of DP one obtains a reasonable data collapse of the density $\rho(t)$. Though the data in Fig. 4 cannot be used for a precise analysis, it is strongly suggested that indeed all three exponents governing the scaling behavior of the spreading process for $\lambda>0$ are that of DP.

V. CRITICAL BEHAVIOR IN THE VICINITY OF THE GEP POINT

In this section, we investigate the influence of mutations in the vicinity of the GEP point in order to address two questions, namely, how does the system cross over from the critical GEP to a nontrivial fluctuating active state and why does the phase transition line terminate in the GEP point with an infinite slope.

A. Decay at the GEP point

Already at the GEP point our model exhibits an additional feature, namely, the competition of different types of infections in full-lattice simulations (see Sec. IV B). One observes a coarsening process of competing species, leading to a slow decay of the density of active sites and active species. As

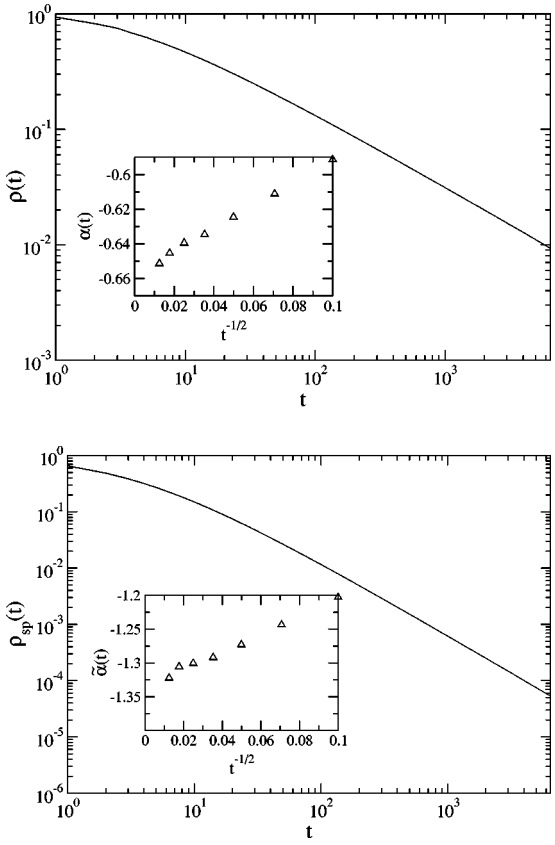

FIG. 5. Full-lattice simulations at the GEP point. The decay of ρ and $\rho_{\rm sp}$ may suggest a possible power-law behavior in the limit $t\to\infty$. The effective exponents can be extrapolated visually for $1/\sqrt{t}\to0$ (see insets).

shown in Fig. 5, the decay of these quantities suggests possibly asymptotic power laws, although the data display a considerable curvature in both cases.

Assuming asymptotic power laws

$$\rho(t)\sim t^{-\alpha},\quad \rho_{\rm sp}(t)\sim t^{-\tilde{\alpha}} \tag{5}$$

at the GEP point, we extrapolate the effective exponents $\alpha(t),\tilde{\alpha}(t)$ visually for $t\to\infty$ (see insets of Fig. 5), obtaining the estimates

$$\alpha=0.66(2),\quad \tilde{\alpha}=1.33(4), \tag{6}$$

suggesting that $\tilde{\alpha}=2\alpha$.

Regarding the limited accuracy of our numerical simulations, the conjecture of an asymptotic algebraic decay has to be taken with care. However, indirect support comes from the one-dimensional case. Here the GEP transition is shifted to $p_c=1$ and the dynamics of competing species reduces to a ballistic coalescence process [22], for which asymptotic power laws could be derived exactly.

B. λ-controlled transition at the GEP point

Let us now turn to the critical behavior in the vicinity of the GEP point. The GEP point in Fig. 1 can be approached

either vertically by varying p or horizontally by varying λ. The critical behavior in vertical direction has been studied in detail in Refs. [8,12,13] and can be described in terms of the scaling form (1). Contrarily, moving in horizontal direction by varying λ and keeping $p=1/2$ fixed one encounters mutations as an additional feature, leading to a nontrivial fluctuating active state.

As usual in critical phenomena, the additional control parameter λ is associated with a novel critical exponent $\mu_\parallel$. Like $\nu_\parallel$ this exponent is defined in such a way that

$$\xi_\parallel \sim \lambda^{-\mu_\parallel} \tag{7}$$

is the correlation time in the stationary state for $p=1/2$ and $0<\lambda \ll 1$. Similarly, the corresponding spatial correlation length is expected to scale as $\xi_\perp \sim \lambda^{-\mu_\perp}$, where $\mu_\perp = \mu_\parallel / z$. According to standard scaling theory, the survival probability $P_s(t)$ should obey the scaling form

$$P_s(t) \simeq t^{-\delta} \Psi(\lambda t^{1/\mu_\parallel}), \tag{8}$$

where $\delta \simeq 0.092$ is the density decay exponent of the GEP. With an appropriate scaling function $\Psi(\zeta)$, Eq. (8) implies that the survival probability eventually saturates at a value $P_s(\infty) \sim \lambda^{\delta\mu_\parallel}$. Using this scaling form, we collapse datasets for seed simulations at $p=1/2$ for $\lambda = 5\times 10^{-4}$ and $\lambda = 10^{-4}$ (not shown as a figure), obtaining the estimate

$$\mu_\parallel = 0.63(3). \tag{9}$$

In the case of full-lattice simulations, provided that the power-law behavior in Eq. (5) is correct, we expect that $\rho(t)$ and $\rho_{\rm sp}(t)$ obey the scaling forms

$$\rho(t) = t^{-\alpha} \Omega(\lambda t^{1/\mu_\parallel}), \quad \rho_{\rm sp}(t) = t^{-\tilde{\alpha}} \tilde{\Omega}(\lambda t^{1/\mu_\parallel}), \tag{10}$$

where $\Omega(\zeta)$ and $\tilde{\Omega}(\zeta)$ are appropriate scaling functions such that eventually the densities reach stationary values $\rho(\infty) \sim \lambda^{\alpha\mu_\parallel}$ and $\rho_{\rm sp}(\infty) \sim \lambda^{\tilde{\alpha}\mu_\parallel}$. Using the value of $\mu_\parallel = 0.63$ [Eq. (9)], these scaling forms lead to reasonable data collapses, as shown in Fig. 6.

We now suggest an explanation for the numerically determined value of $\mu_\parallel = 0.63$. Initially the process behaves as a critical GEP until mutations become relevant at a typical time $\xi_\parallel$. Our argument is based on the assumption that $\xi_\parallel$ scales in the same way as the typical time at which the first mutation occurs. With the mutation probability λ, one needs, on average, λ^{-1} infections until the first mutation occurs. As the process initially behaves as a critical, GEP, the number of infections grows as $\int dt\, N(t) \sim t^{\theta+1}$. Hence, we are led to $\xi_\parallel \sim \lambda^{-1/(\theta+1)}$ with $\theta = d/z - 2\delta - 1 = 0.587$. This implies the scaling relation

$$\mu_\parallel = \frac{1}{\theta+1} \simeq \frac{1}{1.587} = 0.630, \tag{11}$$

which is in perfect agreement with the numerical estimation in Eq. (9).

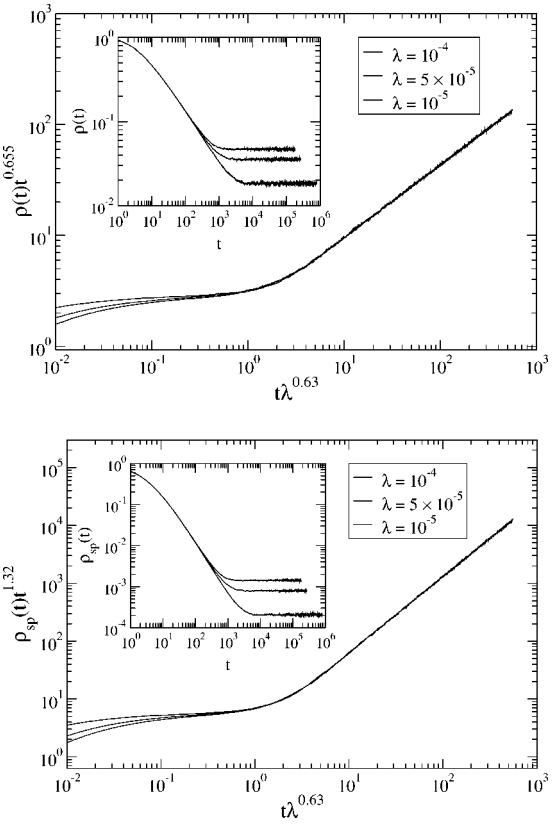

FIG. 6. Data collapse of ρ and $\rho_{\rm sp}$ based on the scaling forms (10) using the exponents of Eqs. (6) and (9).

C. Curvature of transition line at the GEP point

So far the numerical analysis suggests that in the vicinity of the GEP point, the epidemic process with mutations is invariant under scaling transformations of the form $x \to x' = bx$, $t \to t' = b^z t$, and

$$\Delta_p \to \Delta_p{}' = b^{-1/\nu_\perp} \Delta_p, \tag{12}$$

$$\lambda \to \lambda' = b^{-1/\mu_\perp} \lambda, \tag{13}$$

where b is a scaling factor, $\Delta_p = p - 1/2$, δ and z are the critical exponents of the GEP, and $\mu_\perp = \mu_\parallel / z$. In addition, the order parameters have to be rescaled appropriately. In seed simulations, this leads to the combined scaling form for the survival probability

$$P_s(t, \Delta_p, \lambda) = t^{-\delta} \tilde{\Phi}(\Delta_p t^{1/\nu_\parallel}, \lambda t^{1/\mu_\parallel}) \tag{14}$$

in the vicinity of the GEP point. Similar relations should be valid for the densities $\rho(t)$ and $\rho_{\rm sp}(t)$ in full-lattice simulations, provided that the conjecture of asymptotic power-law behavior in Eq. (5) is correct.

As usual in the theory of critical phenomena, the phase transition line itself has to be invariant under scaling transformations. Comparing Eqs. (12) and (13), we are led to the conclusion that the form of the transition line for small values of λ is given by

$$\Delta_p \sim \lambda^\gamma, \tag{15}$$

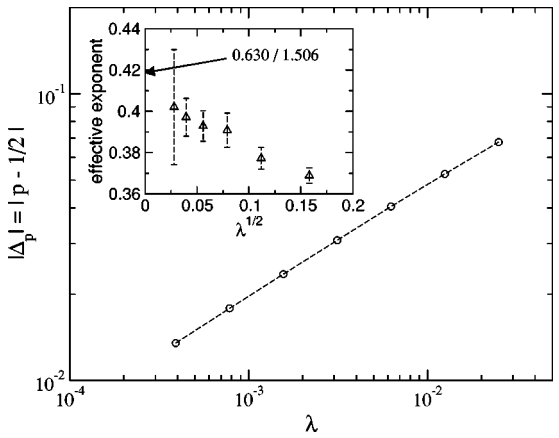

FIG. 7. Double-logarithmic plot of the phase transition line $|\Delta_p(\lambda)|$ for $\lambda \ll 1$. The effective exponent can be extrapolated for $\sqrt{\lambda} \to 0$ (see inset).

where

$$\gamma = \frac{\mu_\perp}{\nu_\perp} = \frac{\mu_\parallel}{\nu_\parallel} \simeq \frac{0.63}{1.506} = 0.42. \tag{16}$$

Since $\partial \Delta_p / \partial \lambda \sim \lambda^{-0.58}$, the phase transition line indeed terminates at the GEP point with an infinite slope.

In order to confirm relations (15) and (16), we plotted $|\Delta_p|$ versus $\lambda \ll 1$ in a double-logarithmic representation in Fig. 7. The local slope of this curve leads to the effective exponent (inset in Fig. 7) which can be extrapolated and leads to the estimation $\gamma \approx 0.41(3)$, in agreement with the prediction in Eq. (16).

VI. DISCUSSION AND CONCLUSION

In this paper, we have introduced a minimal model for epidemic spreading with immunization and mutations. Apart from the probabilities for first infections and reinfections p and q, the model is controlled by a probability λ that a transmitted pathogen mutates, creating a new pathogen which was not involved before. The model includes the GEP ($\lambda = 0, q = 0$) and DP ($\lambda = 1$ or $q = p$) as special cases.

Restricting the analysis to the case of perfect immunization and two spatial dimensions, we have shown that at the transition between survival and extinction our model shows DP scaling everywhere along the phase transition line except for the point where the model reduces to the critical GEP. In the vicinity of the GEP point, even a small mutation probability drives the system away from criticality into a fluctuating active state. This crossover can be described in terms of a suitable scaling theory, which involves a new exponent $\mu_\parallel$. This crossover exponent also determines the form of the phase transition line in the vicinity of the GEP point. We suggested an explanation for the value of $\mu_\parallel$ which turns out to be in perfect agreement with the numerical analysis.

Although the model presented here is highly idealized (individuals on a square lattice, homogeneous infection probabilities, nearest-neighbor infections, etc.), there is an important conclusion to be drawn regarding realistic spreading of epidemics in nature. As in the model, realistic epidemic spreading starts at a certain threshold determined by various parameters such as the average susceptibility, the interaction frequency, and the degree of immunization and/or vaccination. Mutations weaken the effect of immunization, thereby decreasing this threshold. An important message of our paper is that for a population which is mainly stabilized by immunization and/or vaccination, this threshold varies *nonlinearly* with the mutation rate, in the present case roughly as the square root of λ. Thus even a small rate of mutations can significantly weaken the stability of a population at the onset of epidemic spreading.

As a possible extension of the present study, it would be interesting to investigate whether these properties can also be observed in epidemic processes with long-range infections [23] if mutations are introduced. Moreover, it would be interesting to study the surface critical behavior at the system's boundaries [24].

ACKNOWLEDGMENTS

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