Spreading in media with long-time memory

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We study the spreading of an agent in a medium whose susceptibility changes irreversibly at the first encounter with the agent. This can model epidemics with partial immunization or population growth with incomplete replenishment of food (in both cases the susceptibility for growth decreases after the first attack) or epidemics in which the resistance is weakened by the first infection (increased susceptibility). In such models one can have no growth at all, compact growth, or annular growth. We delineate the phase diagram and study the scaling behavior at the phase boundaries. Our arguments are supported by simulations in one and two dimensions. Although our model does not involve multiple absorbing states, we claim that our results explain the ''nonuniversal'' behavior seen in models with such states. [S1063-651X(97)07703-9]

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I. INTRODUCTION

Despite much effort and considerable recent progress, understanding the phase transitions in models for the spreading of a nonconserved agent in nonequilibrium systems remains an interesting challenge. Such models can describe the spreading of epidemics or forest fires [1–4], the growth of populations, the activity of catalyzers [5,6], and maybe even the formation of stars and galaxies [7]. In all cases, it is assumed that the agent cannot pop up spontaneously, but can multiply itself arbitrarily by local offspring production.

In the simplest case, a medium is considered to be without memory (i.e., without permanent consumption of resources or immunization) and without quenched randomness. One can then have an epidemic surviving *in loco*, provided the susceptibility to new infections is sufficiently high and the recovery rate is sufficiently low. The transition from the survival of the agent to its extinction is a critical phenomenon in the universality class of Reggeon field theory [8,9], the ''contact process'' [3,4], or directed percolation (DP) [10].

Nearly as simple is the case of perfect immunization [the ''general epidemic process'' (GEP) [2]]. Here the epidemic cannot, of course, survive *in loco*, but an infinite epidemic is nevertheless possible in the form of a solitary wave of activity. When starting from a punctual seed, this leads to annular growth, as seen, e.g., in the growth patterns (''fairy rings'') of some mushrooms. Again the transition between survival and extinction is a critical phenomenon, this time in the universality class of ordinary (undirected) percolation [11,12].

In the present paper, a generalization of these two cases is studied. More precisely, we shall discuss a process where the susceptibility changes after the first infection and remains constant thereafter. If it changes to zero, we have the GEP; if it is not changed at all, we have DP. In intermediate cases, we can still observe either annular or compact growth, or no growth at all. The situation is different if the susceptibility is *increased* by the first infection: in this case, annular growth is not possible. Instead, a tendency to even more compact growth is then observed since the epidemic finds it harder to invade new regions than to survive in regions it has already visited.

The main finding of this paper is that the critical susceptibility for sustained survival is strictly independent of the susceptibility to the first attack. This holds true for both cases, i.e., for increasing or decreasing susceptibility. On the other hand, scaling laws typically change. This is very similar to the behavior of spin systems in the presence of surfaces. While the critical temperature is still given by the bulk, there exist new surface critical exponents [13]. In this analogy (which should not be taken too seriously, of course), DP resembles the ''special point'' in a surface critical phenomenon.

Another interesting connection of our model is with generalizations of DP involving multiple absorbing states [14–20]. In DP, the ''dead'' state with no agent is unique and non-fluctuating. It is *absorbing* in the sense that a region in this state can leave it only by invasion at its boundaries. One of the best tested hypotheses in this field is that all continuous phase transitions in models with a unique absorbing state are in the DP universality class [22,23]. But this leaves the question open for models with multiple absorbing states. There, one must distinguish between models with *fluctuating* absorbing states (where ergodicity is not broken in the dead sector of phase space) and models with *frozen* absorbing states. In the former, there is indeed only a single absorbing macrostate and it is not surprising that they are also in the DP class [18,20].

In models with multiple frozen absorbing states, a dead configuration can change only if a new wave of activity passes through it. Ergodicity in the dead sector is broken and the universality with DP is much more subtle. In [15,18,19] it was found that scaling properties depend on the observables and on the initial states considered. In all cases, universality was found for the *stationary* behavior in the active phase and for the spreading behavior if the initial state coincided (statistically) with the dead state left after all activity had died out. If, however, the initial inactive state was ''atypical,'' then the spreading behavior was changed. Moreover, it was claimed in [19] that the critical point changes as

well and that one finds scaling behavior with nonuniversal critical exponents.

We contend that the latter is not fully correct and has to be interpreted as a slow cross-over effect. We argue that the multiplicity of absorbing states is not essential (for a related discussion, see [21]). What is important is that the spreading into an atypical initial state is characterized by a different effective susceptibility, whence we have precisely the behavior described in the first part of the Introduction.

In the next section we discuss an effective-field theory for this process. This will lead to a qualitative phase diagram after the presentation of a lattice model in Sec. III. Numerical simulations for two spatial dimensions are reported in Sec. IV A. They confirm the phase diagram and describe in more detail the behavior at the phase boundaries. The situation is slightly different in $d=1$. Simulations for this case are shown in Sec. IV B. The paper ends with a discussion in Sec. V.

II. FIELD THEORY

The field theory discussed here is essentially the same as for the spreading of the GEP presented in [24,25,21]. In this model we have just two fields, one for the spreading agent (ψ) and one for the ''debris'' left by the agent (ϕ). After being produced by the agent, the debris is completely inert: it neither diffuses, decays, nor reproduces itself. But it can act on the agent by modifying its reproduction rate ρ, its spontaneous death rate σ, and any other parameter influencing its spreading. For simplicity of discussion (and without restriction of the generality of the model), we shall assume that only ρ depends significantly on ϕ.

Thus we model the spreading by a Langevin equation for ψ similar to that of Reggeon field theory [8], but with ρ replaced by $\rho(\phi)$,

$$\frac{\partial\psi}{\partial t}=D\nabla^2\psi-\sigma\psi+\rho(\phi)\psi^2+\eta(x,t), \tag{1}$$

where D is a diffusion coefficient and $\eta(x,t)$ is a Gaussian noise whose variance is proportional to ψ,

$$\langle\eta(x,t)\,\eta(x',t')\rangle=\delta(x-x')\,\delta(t-t')\,\psi(x,t). \tag{2}$$

The equation for ϕ is simply

$$\frac{\partial\phi}{\partial t}=g\psi. \tag{3}$$

If we want to model a deteriorating (enhancing) influence of the debris, we should take $d\rho/d\phi<0$ ($d\rho/d\phi>0$). Actually, with this ansatz, the susceptibility for spreading would in general change at *each* infection, in contrast to our assumption that it changes only at the first. The latter could easily be taken into account by modifying Eq. (3), but we believe that the difference is irrelevant in any case.

The critical point of marginal *in loco* survival can depend only on the *asymptotic* value of ρ,

$$\rho_\infty=\lim_{\phi\to\infty}\rho(\phi). \tag{4}$$

Indeed, assume that the process is supercritical and that the epidemic does in fact survive *in loco*. Then, obviously, the density of debris will increase beyond any limit and the further evolution will depend only on ρ_∞. On the other hand, if the process is subcritical, the decay is controlled by $\rho(\phi)$ at some $\phi<\infty$. But, as we pass through the critical point, the final value of ϕ for surviving epidemics must diverge, thus proving our statement. Notice that this does *not* imply that the critical *behavior* is also governed by ρ_∞ alone. In contrast, the critical behavior will in general be controlled by the approach to ρ_∞.

Let us now assume that $d\rho/d\phi<0$. In this case, the spreading at the border of the activity region is easier than in its bulk. Thus the spreading of the border will be supercritical if the bulk is critical, while the bulk is subcritical if the edge is critical. As was shown in [24,25], the above model describes the critical spreading of the GEP in the latter case. Between these two cases, one has annular growth: the epidemic can survive in a propagating solitary wave (a ''front''), but it dies out in its wake. If $\rho_\infty=\rho_c$, the critical value for *in loco* survival, the density of activity at a fixed position in the bulk will decay with some power of time, which is *not* the same as for the decay of activity in DP (i.e., in the critical process with constant $\rho=\rho_c$). More precisely, we expect the decay to be *slower* than in DP (here we assume that the epidemic had started in a finite region; the case of infinite ''seeds'' will be discussed below).

For $d\rho/d\phi>0$, spreading is more difficult than survival. Thus there is no annular growth and we have a unique critical point. Again, this is attained when $\rho_\infty=\rho_c$, but this time the critical spreading will be slower than in DP and the decay of the activity at the critical point will be faster.

III. LATTICE MODEL

We have made no attempt to compute the critical behavior(s) in the above field theory analytically. Instead, we have simulated a lattice model that we believe to be in the same universality class.

In $2+1$ dimensions, this model is a generalization of directed bond percolation on the bcc lattice [26–28]. Stated differently, it is the spreading on a square lattice with discrete time such that each site stays active for only one unit of time after having been activated (infected). Each bond can transmit the agent with a certain prescribed but annealed probability. The generalization consists in allowing two different states for each site. Initially, all sites are in the *virgin* state v. After an agent has passed through them, they go over into the *used* state u in which they remain thereafter. The probability of transmission through a bond depends on the state of the site on the other side: it is q for bonds connecting to virgin sites and p for bonds connecting to used sites. For $q=p$, this model reduces to directed bond percolation as described in [26–28]. For $p=0$, on the other hand, it coincides with the spreading of ordinary bond percolation on the square lattice treated as a GEP [12,29].

According to the discussion of Sec. II, we expect the phase diagram to look qualitatively as in Fig. 1. For $p>p_c=0.287\,34$ [28], we have a finite probability for compact infinite growth of an epidemic starting from a single seed. For $p<p_c$ and $q<p_c$, the epidemic dies with probabil-

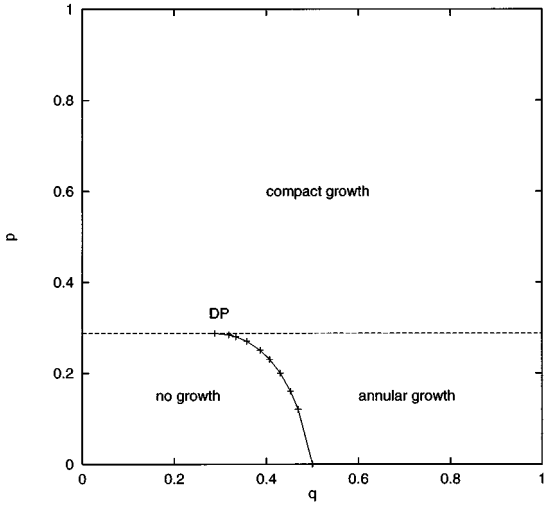

FIG. 1. Phase diagram for the model. All along the curved line, one has the critical behavior of the GEP, while DP behavior is only observed at the point where the straight and curved lines meet.

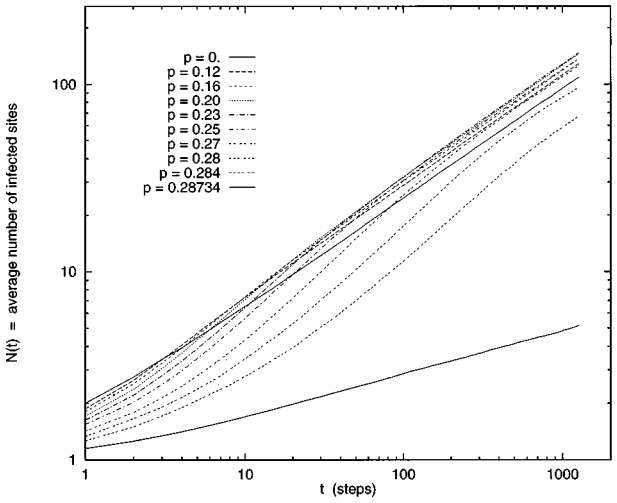

FIG. 2. log-log plot of the average number $N(t)$ of active sites at the no-growth–annular-growth boundary. Each curve corresponds to one of the ten points indicated in Fig. 1.

ity 1. The same is still true to the left of a curve $q=q_c(p)$, which connects the DP point $q=p=p_c$ with the critical point $q=0.5, p=0$ for bond percolation on the square lattice [11]. To the right of this line we have annular growth. All along the line $q=q_c(p)<p_c$, the critical behavior should be that of the GEP (i.e., of the spreading of ordinary percolation).

Most simulations were done according to the single-seed spreading paradigm [31]. We started from a single infected site and stopped if the epidemic died out or if a preset maximum time was reached. This time was always chosen such that the boundary of the lattice was never reached, whence the simulations are free of finite-size (but, alas, not of finite-time) effects, with the potential difficulties, though, of kinetic roughening effects (see below).

The compact-growth–annular-growth border is much harder to study by means of simulations starting from a single active site. Very large lattices would be needed since the active region grows at a finite speed. In addition, since we have to simulate the *supercritical* annular growth, the simulations would involve very large numbers of active sites. This would make them very slow. (We should mention here that we used lists of active sites in order to speed up the simulations compared to a brute force updating of the entire lattice.) For this reason we have used a different geometry in this case. Instead of an infinite lattice with a single site seed leading to annular growth, we used a strip geometry with a planar growth front in a rectangular lattice of size $L_\perp \times L_\parallel$ $(L_\parallel > L_\perp)$ with periodic boundary conditions. The seed consisted of an entire line of active sites. Initially, two fronts emanated from this seed, but only one of them was followed. By ''cleaning'' the lattice ahead of the leading edge of this front, we ensured that it always invaded virgin territory. This cleaning of course eventually destroyed the other front, but it allowed us to follow the evolution for long times during which the activity looped several times across the lattice.

Irrespective of the geometry used, the critical behavior near the boundaries of the annular-growth region might *a priori* be influenced by the roughness of the interface separating the virgin territory ahead from the ''used'' sites. Typically, the width of this interface increases with time (to eventually saturate in strip geometry) and/or with size. This calls for a cautious interpretation of the simulations with respect to finite-time or finite-size effects.

IV. SIMULATIONS

A. $D=2$

In a first set of runs we determined precisely the GEP line connecting the DP point with the point $(p=0, q=1/2)$ and verified that all along this line we do indeed see a crossover to GEP behavior. For this we measured the average size $N(t)$ of the epidemic (number of infected sites at time t), the survival probability $P(t)$, and the squared spatial extension $R^2(t)$. The latter is defined as the average over $\mathbf{x}_i^2$, the average being taken over all active sites in surviving epidemics. These results are shown in Figs. 2–4. Each figure contains ten curves, each corresponding to one of the points indicated in Fig. 1. The two extreme points [for $p=q=p_c$ and for $(p=0, q=1/2)$] are indicated by solid lines in Figs. 2–4. They show the scaling behavior of DP and of GEP, respec-

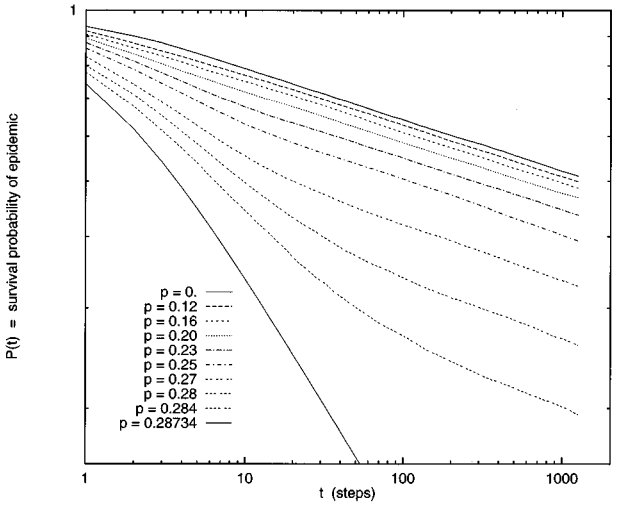

FIG. 3. Similar to Fig. 2, but for the survival probability $P(t)$.

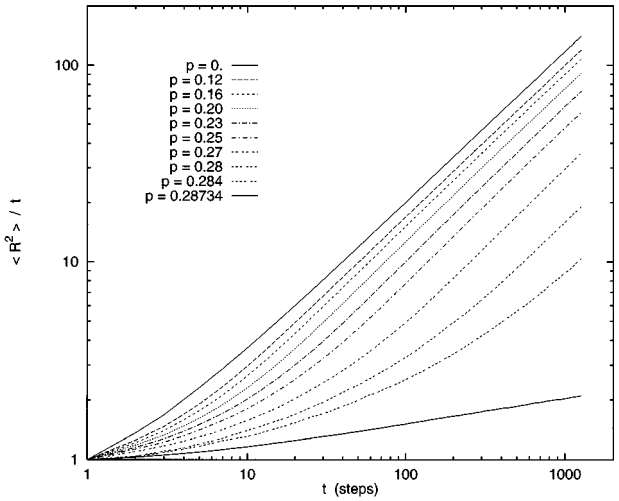

FIG. 4. Similar to Fig. 2, but for the ratio $R^2(t)/t$, where $R^2(t)$ is the average squared distance of active sites from the seed.

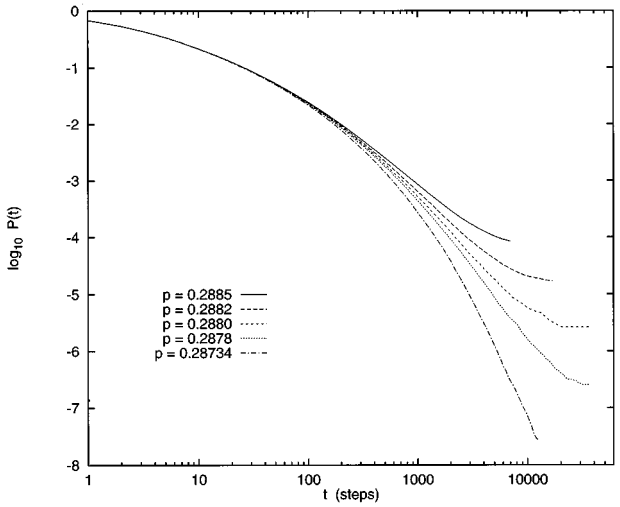

FIG. 6. Similar to Fig. 5, but for the survival probability $P(t)$.

tively. For the other eight curves, the value of p was chosen first and $q_c(p)$ was determined such that $P(t)$ was parallel to the GEP curve for large t. In all three plots, the expected crossover is clearly seen: close to the DP point and for small t we have approximately DP behavior, but for larger times and/or for points not close to DP we observe GEP scaling. This is most easily seen in the behavior of $R^2(t)$, but it is also evident from the other two plots. Of course our method to determine q_c favored GEP-type scaling for $P(t)$, but even for this variable it is far from trivial that we could find any values of q where such a clear crossover is seen. In fact, we could not find values of q for which DP scaling is observed at large times.

Next we tried to verify the prediction that the compact-growth–no-growth transition is at $p=p_c$, independent of the value of q. This seems counterintuitive, as one might expect that the critical value of p is increased when $q<p$, an expectation indeed claimed to be verified in [19]. We thus fixed q at 0.25 (a value well below p_c) and performed runs at several values of $p \geqslant p_c$. Again we measured $N(t), P(t)$, and $R^2(t)$. Results are shown in Figs. 5–7. In none of these plots do we see any hint of scaling. If we were looking for scaling behavior, we would thus have to choose another (bigger) value of p. But even then we would have to accept significant deviations from scaling. The fact that the transition is indeed at $p=p_c=0.287\,34$ is most clearly seen from Fig. 5: for all values $>p_c$, $N(t)$ first decays, but then turns sharply to increase at very late times. This is easily understood. Since $q<p_c$, the cluster at first (when nearly all sites are virgin) has a very small probability of survival and $N(t)$ drops much faster than for critical DP. But if $p>p_c$, some clusters will survive nevertheless, and once they have reached a critical size, they will even have a chance to grow in the now friendly environment. Figures 6 and 7 support this picture, though much less clearly.

A more quantitative argument is as follows. Assume a cluster has survived, in a run at $p=p_c+\epsilon$, up to a time t_0 at which it has reached a size $R(t_0) \gg 1$. Inside this cluster we have essentially unmodified DP, hence the density of active sites is $N \sim \epsilon^{\beta}$ and the total number of active sites is $\sim R^d \epsilon^{\beta}$ (we keep the argument general by allowing any dimensionality d). The fluctuations inside are normal (we are

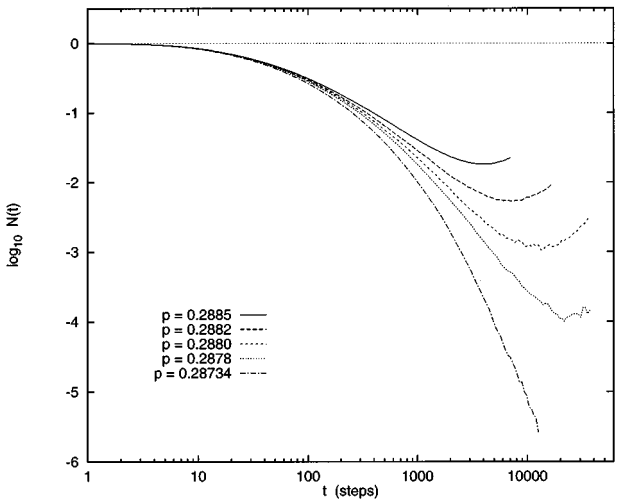

FIG. 5. log-log plot of the average number $N(t)$ of active sites for $q=0.25$, for five values of p at or slightly above p_c.

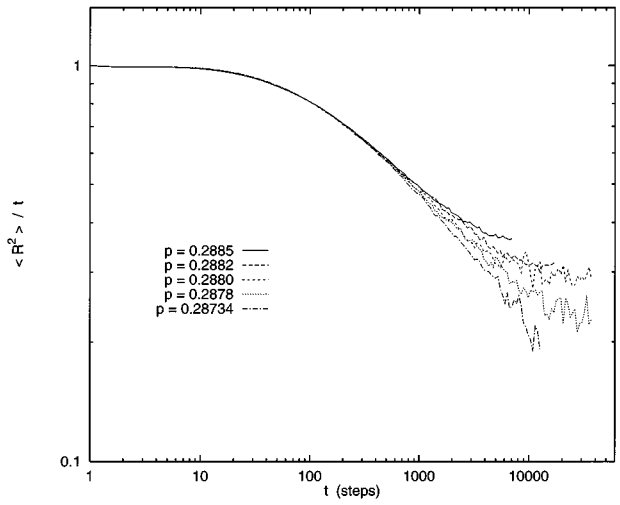

FIG. 7. Similar to Fig. 5, but for the ratio $R^2(t)/t$.

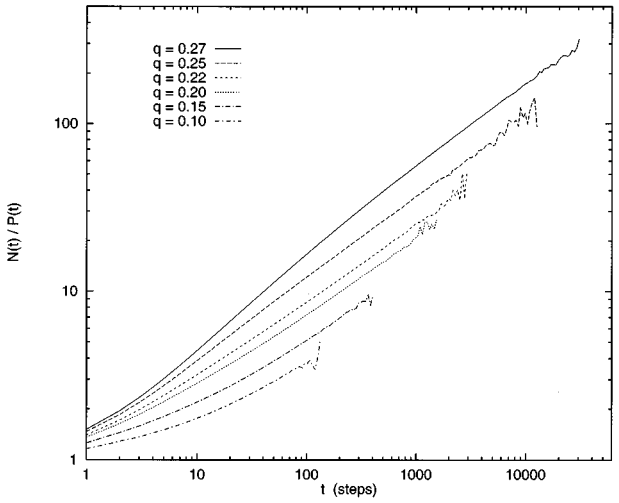

FIG. 8. log-log plot of the average number of active sites *per surviving cluster* $N(t)/P(t)$, for $p=p_c=0.287\,34$ and for six values of q below p_c.

not at the critical point) and thus the rate of extinction by spontaneous fluctuations is given by $dP/dt\sim -Pe^{-t/T}$ with a characteristic time $T\sim\exp(N)$. If the cluster does not become extinct, on the other hand, its radius will increase linearly. Even if this increase is arbitrarily slow, the limit $\ln P(\infty)=\ln P(t_0)-\int_{t_0}^{\infty}dte^{-t/T}$ will be finite, hence the cluster will have a finite chance to survive forever.

After having accepted that the critical value of p is equal to p_c for all $q\leqslant p_c$, we can now study the q dependence of the critical behavior. Figures 8–10 display the three quantities $N(t)$, $P(t)$, and $R^2(t)/t$. For ease of interpretation, Fig. 8 now shows the ratio $N(t)/P(t)$, which is the number of active sites per surviving epidemic. Each curve corresponds to a different value of q, while $p=p_c$ for all of them.

The clearest indication of scaling is for $N(t)/P(t)$, for which we get a reasonably good fit with

$$N(t)/P(t)\sim t^{\alpha},\quad \alpha=0.47. \tag{5}$$

The situation is worse for $R^2(t)$. Here the curves are strongly bent except for very small q. But they seem to follow

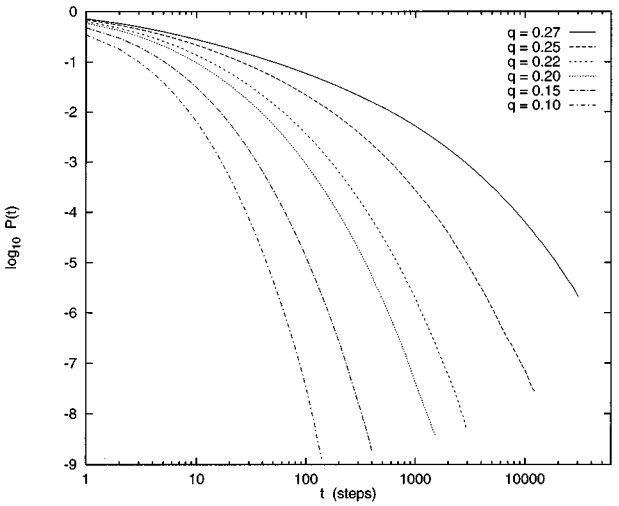

FIG. 9. Similar to Fig. 8, but for the survival probability $P(t)$.

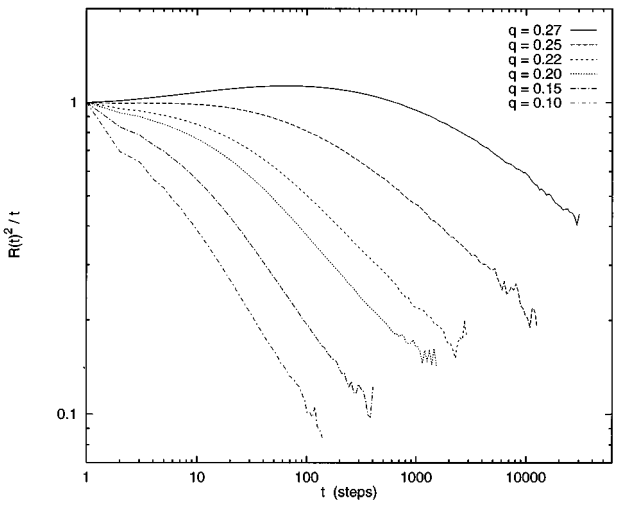

FIG. 10. Similar to Fig. 8, but for the ratio $R^2(t)/t$.

straight lines for large t, suggesting an asymptotic power law $R^2(t)\sim t^z$. The exponent z seems to depend weakly on q. It increases from $z\approx 0.46$ for $q=0.1$ to $z\approx 0.65$ for $q\geqslant 0.22$. All these numbers should of course be taken with some caution. Since the density inside a surviving cluster should decrease with time, we must have $\alpha<z$, which is just verified for small q in the above data. We warn the reader that we have no good theoretical argument for power laws to hold and the data might well follow some other law asymptotically. For $P(t)$, indeed, Fig. 9 suggests no power law at all. We tried stretched exponentials for this observable, without much more success and, we should say, with no better theoretical arguments either. Needless to say, the absence of a power law for $P(t)$ prevents any hyperscaling relation from holding.

For the annular-growth–compact-growth transition, we used the strip geometry. We first checked that $p=p_c$ is indeed the threshold for sustained activity *in loco* for all $q>p_c$. For $p<p_c$, the density of active sites decays to zero in the wake of the activity wave, while it tends to a positive value for $p>p_c$.

Next, we were interested in the density $\rho(\xi,t)$ of active sites at a distance ξ behind the leading edge of the wave. At threshold, we expect this quantity to reach a limit $\rho(\xi)$ for $t\to\infty$, which scales as

$$\rho(\xi)\sim\xi^{-\delta}, \tag{6}$$

where $\delta=0.451\pm 0.003$ [28] is the exponent governing the decay of the density of active sites in DP in $2+1$ dimensions, starting from an entire active line perpendicular to the growth direction. Equation (6) can be understood as follows. For $q>p=p_c$, the front propagates at a finite speed v, so that ξ is up to a factor $1/v$ equal to the time elapsed between the passage of the front and the measurement of the density. On the other hand, the evolution of DP clusters is slow compared to a propagation with finite speed $t\sim x^{z/2}\sim x^{0.566}$ against $t\sim x$, whence the passage of the front through a given point can be considered as instantaneous from the point of view of an evolving DP cluster. Thus $\rho(\xi)$ indeed measures

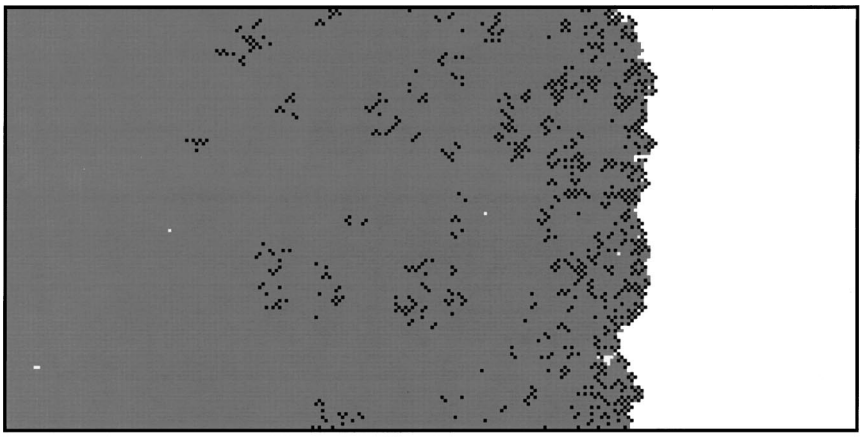

FIG. 11. Snapshot of the lattice in the annular-growth regime ($p=0.27$, $q=0.6$). The transverse size is $L_\perp=128$. Active, virgin, and used sites are in black, white and gray, respectively. The front is moving from left to right.

the density of active sites at time $\tau=\xi/v$ after all sites had been infected simultaneously and decays according to Eq. (6).

Measuring $\rho(\xi,t)$ is not easy. Figure 11 shows a typical snapshot of the activity wave in the annular-growth regime near threshold and Fig. 12 the corresponding (averaged) density profiles. In such a regime, i.e., for q not too close to p_c, the leading edge of activity is almost identical to the virgin-used interface. Its profile, which is close to Gaussian, provides a measure of its roughness, which is simply the width w. For $q=1$, the interface is flat ($w=0$). For $p_c<q<1$, w initially grows with time and then saturates at a value $w(p,q,L_\perp)$. On general grounds, we expect the interface to be in the universality class of the Kardar-Parisi-Zhang model [30] and thus $w^2\sim L_\perp$. For $(q,p)\to(p_c,p_c)$, the situation is much more complex, as we have $v\to 0$, $w\to\infty$, even at fixed transverse size. This intricate limit is left for future work, together with the study of the interface along the annular-growth–no-growth boundary.

At any rate, the study of the annular-growth–compact-growth boundary, even for q not too close to p_c, already involves a rather delicate limit in which ξ, $L_\perp$, and t all have to go to infinity, but not in an arbitrary order. We have to take $L_\perp\to\infty$, since otherwise the front will die with probability 1. We have used typically $L_\perp=1024$, $\xi\leqslant L_\parallel=2048$, and $t\leqslant 2\times 10^5$. With this value of $L_\perp=1024$ and for the values of q studied ($0.5\leqslant q\leqslant 1$), the roughness of the front was typically $\leqslant 50$. This is indeed much smaller than $L_\parallel$, but it induces an inherent uncertainty in the definition of ξ, which renders difficult the estimation of critical exponents. In practice, the absolute position of the activity wave can be measured in several ways. For the regimes of interest here, we used the position $x_{\max}$ of the leading edge (which advances regularly with velocity v). However, to check the expected scaling (6), we need to set an ''effective zero'' for ξ. In view of this, we have adopted a rather empirical procedure. We have defined $\xi=x_{\max}-\Delta(q)-x$, where $\Delta(q)$ is a positive constant determined such that $\rho(\xi)$ showed the best scaling (i.e., produced the straightest lines for large ξ on a log-log plot). Not surprisingly, we find that $\Delta(q)$ is of the same order and behaves exactly as $w(p_c,q,L_\perp)$. The results of this procedure are shown in Fig. 13. We indeed see nice scaling with an exponent close to $\delta\approx 0.45$, independent of q.

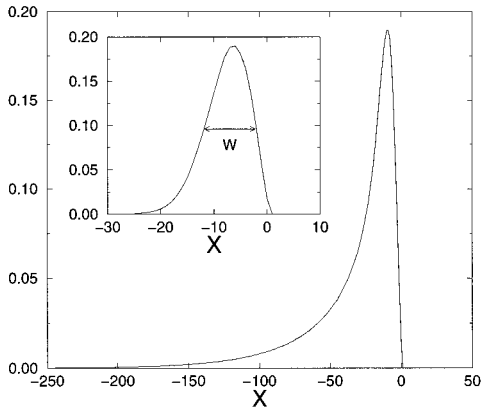

FIG. 12. Averaged (normalized) density profile of active sites (main graph) and distribution of the x location of the sites of the virgin-nonvirgin interface (inset). Same parameters as in Fig. 11. The quantity x plotted horizontally is the distance from the leading edge $x_{\max}$.

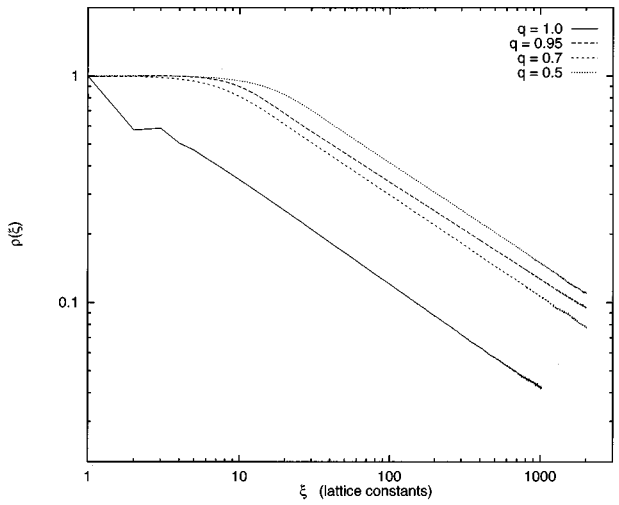

FIG. 13. log-log plots of the density $\rho(\xi)$ of active sites a distance ξ behind the progressing front of activity. The normalization is set to $\rho(1)=1$ for all curves. See the text for the precise definition of ξ. All curves are for $p=p_c$.

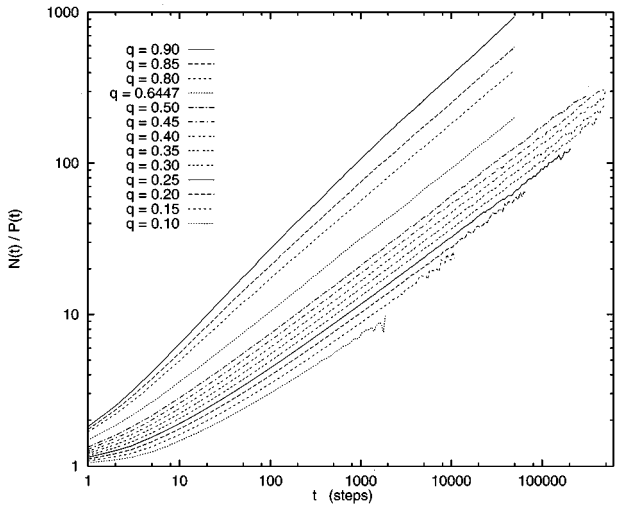

FIG. 14. log-log plot of the average number of active sites per surviving cluster $N(t)/P(t)$, for spreading in one dimension at $p=p_c=0.6447$ and for 13 values of q. Of these, three are above p_c and nine are below.

B. $D=1$

The above arguments should be slightly modified in the case of one spatial dimension. In this case there is no undirected percolation spreading (the critical point is at $q=1$), so there can be no annular growth either. Thus the phase diagram shown in Fig. 1 has to be modified such that the curved boundary is horizontal. As a result, only transitions between no growth and compact growth are possible. These transitions are different depending on whether q is below, at, or above p_c.

We have simulated essentially the same model as in Sec. III. The threshold for directed percolation is now at $p_c=0.644\,701$ [32]. We show only the results from simulations at $p=p_c$. For all $p>p_c$, one has compact growth, and for all $p<p_c$ and $q<1$, the process dies out exponentially fast.

In Figs. 14–16 we show the by-now familiar quantities $N(t)/P(t), P(t)$, and $R(t)^2/t$. As expected, we again find a

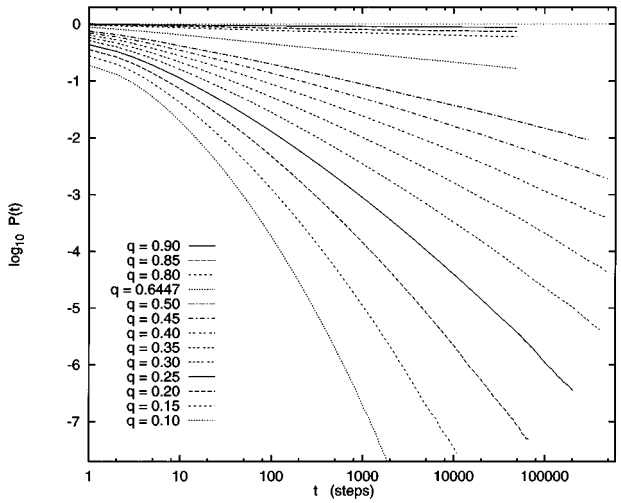

FIG. 15. Similar to Fig. 14, but for the survival probability $P(t)$.

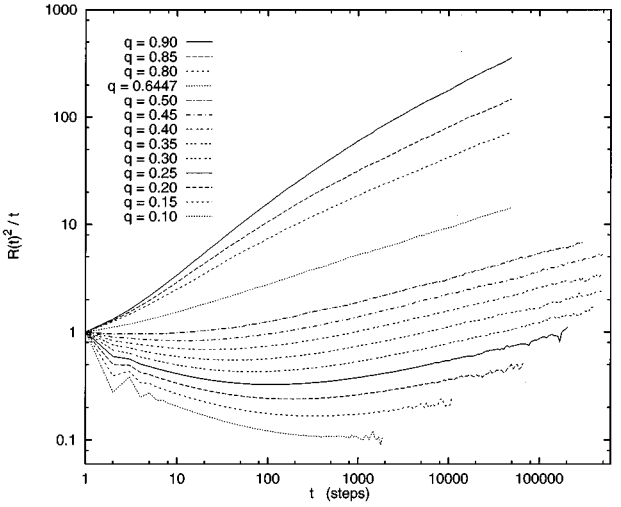

FIG. 16. Similar to Fig. 14, but for the ratio $R^2(t)/t$.

decrease of $P(t)$ that is faster than any power of t for all $q<p_c$, while it seems to follow a nonuniversal power for $q>p_c$. More surprising than this is the behavior of $N(t)/P(t)$ and of $R(t)^2/t$: after marked deviations from any power laws for small t, they both seem to scale for $t\to\infty$, with the critical exponents of DP. Thus the nonuniversal behavior seems in this case to affect only $P(t)$ and the corrections to scaling, but not the asymptotic behavior of $N(t)/P(t)$ and $R(t)^2/t$. This is in very strong contrast to the behavior for $d=2$, where $R(t)^2$ clearly does not show DP scaling for $q<p_c$.

The most likely explanation for this surprising difference is that an epidemic in one dimension must create a compact set of used sites on which it survives, while a two-dimensional epidemic can survive on a fractal set of used sites. Thus the bulk region in a surviving one-dimensional epidemic never undergoes the anomalous influence of virgin sites, while it is permanently exposed to it in two dimensions. Unfortunately, we see no way to deduce quantitative results from this heuristic argument.

V. DISCUSSION AND CONCLUSIONS

We have given heuristic—but we believe convincing—theoretical arguments for the conjecture that the threshold for critical spreading in the case of infinite memory effects is only given by the susceptibility after repeated infection, independent of the susceptibility of the initial virgin medium. The latter will, however, influence the detailed critical behavior. This is strikingly similar to surface critical phenomena in lattice spin systems, with DP being analogous to the ''special'' point [13].

We have supported this conjecture by simulations of spreading in one- and two-dimensional lattice models. In these models we have found very clear evidence that critical behavior for spreading is indeed changed with respect to DP. But while all critical exponents seemed to be affected in $d=2$, it seems that only one of the two exponents governing the behavior exactly at $p=p_c$ is changed. We have not studied the critical behavior governing the approach to $p=p_c$.

We have argued that these results apply also to models with multiple absorbing states. Indeed, the main effect of the

multiplicity of absorbing states in these models is to create an *effective* memory-dependent susceptibility. (This assumes that the ''static'' behavior of these models is in the DP universality class, as claimed in [15,18,19]. This might not be strictly true, and there might be very small differences even in the static behavior. In that case, the effect of the multiplicity of absorbing states would be much more subtle. But we have no reason to suspect such problems.) Thus we claim that the initial state dependence of the critical point *locations* found in [19] is a crossover effect. On the other hand, the nonuniversality of critical *exponents* found there and in [15,18] is real, though the actual values of the exponents should again be strongly influenced by crossover effects that were not correctly taken into account.

In [33], a different model was introduced for simulating the changed effective susceptibility at active region boundaries in systems with multiple absorbing states. But in contrast to the model treated in the present paper, the medium at site **x**, in this model, ''forgot'' whenever the epidemic receded beyond **x**: Only the active sites at the *current* boundary of the epidemic ''feel'' the modified susceptibility, whether they have been visited before or not. This is somewhat awkward to handle in two and more dimensions, thus the authors studied only one-dimensional systems. They found that their results could not explain the phenomena seen in [15,18,19]. We believe that this is a consequence of their specific assumptions that make the model of [33] very different from ours and, we believe, less natural.

We should finally point out that different results are expected in the case of slowly decaying memory effects. If the susceptibility relaxes after an infection to its original values with a power law, we expect both the location of the critical point and the critical exponents to be modified.

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[1] S.R. Broadbent and J.M. Hammersley, Proc. Cambridge Philos. Soc. **53**, 629 (1957).
[2] D. Mollison, J. R. Stat. Soc. B **39**, 283 (1977).
[3] T.M. Liggett, *Interacting Particle Systems* (Springer, New York, 1985).
[4] R. Durrett, *Lectures on Particle Systems and Percolation* (Wadsworth, Pacific Grove, CA, 1988).
[5] R.M. Ziff, E. Gulari, and Y. Barshad, Phys. Rev. Lett. **56**, 2553 (1986).
[6] I. Jensen, H.C. Fogedby, and R. Dickman, Phys. Rev. A **41**, 3411 (1990).
[7] L.S. Schulman and P.E. Seiden, J. Stat. Phys. **19**, 293 (1978).
[8] M. Moshe, Phys. Rep. C **37**, 255 (1978).
[9] P. Grassberger and K. Sundermeyer, Phys. Lett. **77B**, 220 (1978).
[10] W. Kinzel, Z. Phys. B **58**, 229 (1985).
[11] D. Stauffer and A. Aharony, *Introduction to Percolation Theory*, 2nd ed. (Taylor & Francis, London, 1992).
[12] P. Grassberger, Math. Biosci. **62**, 157 (1983).
[13] H.W. Diehl, in *Phase Transitions and Critical Phenomena*, edited by C. Domb and J.L. Lebowitz (Academic, New York, 1986).
[14] I. Jensen, Phys. Rev. Lett. **70**, 1465 (1993).
[15] I. Jensen and R. Dickman, Phys. Rev. E **48**, 1710 (1993).
[16] D. ben-Avraham and J. Köhler, J. Stat. Phys. **65**, 839 (1992).
[17] K. Yaldram, K.M. Khan, N. Ahmed, and M.A. Khan, J. Phys. A **26**, L801 (1993).
[18] J.F.F. Mendes, R. Dickman, M. Henkel, and M.C. Marques, J. Phys. A **27**, 3019 (1994).
[19] R. Dickman, Phys. Rev. E **53**, 2223 (1996).
[20] P. Grassberger, J. Stat. Phys. **79**, 13 (1995).
[21] M.A. Muñoz, R. Dickman, G. Grinstein, and R. Livi, Phys. Rev. Lett. **76**, 451 (1996).
[22] H.K. Janssen, Z. Phys. B **42**, 151 (1981).
[23] P. Grassberger, Z. Phys. B **47**, 365 (1982).
[24] J. Cardy and P. Grassberger, J. Phys. A **18**, L267 (1985).
[25] H.K. Janssen, Z. Phys. B **58**, 311 (1985).
[26] H. Chaté and P. Manneville, Phys. Rev. A **38**, 4351 (1988).
[27] P. Grassberger, J. Phys. A **22**, 3673 (1989).
[28] P. Grassberger and Y.-C. Zhang, Physica A **224**, 169 (1996).
[29] P. Grassberger, J. Phys. A **25**, 5867 (1992).
[30] See, e.g., T. Halpin-Healy and Y.-C. Zhang, Phys. Rep. **254**, 215 (1995).
[31] P. Grassberger and A. de la Torre, Ann. Phys. (N.Y.) **122**, 373 (1979).
[32] J.W. Essam, A.J. Guttmann, and K. de'Bell, J. Phys. A **21**, 3815 (1988).
[33] J.F.F. Mendes, R. Dickman, and H. Herrmann, Phys. Rev. E **54**, R3071 (1996).