Two-dimensional SIR epidemics with long range infection

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We extend a recent study of susceptible-infected-removed epidemic processes with long range infection (referred to as I in the following) from 1-dimensional lattices to lattices in two dimensions. As in I we use hashing to simulate very large lattices for which finite size effects can be neglected, in spite of the assumed power law $p(x) \sim |x|^{-\sigma-2}$ for the probability that a site can infect another site a distance vector $x$ apart. As in I we present detailed results for the critical case, for the supercritical case with $\sigma = 2$, and for the supercritical case with $0 < \sigma < 2$. For the latter we verify the stretched exponential growth of the infected cluster with time predicted by M. Biskup. For $\sigma = 2$ we find generic power laws with $\sigma$-dependent exponents in the supercritical phase, but no Kosterlitz-Thouless (KT) like critical point as in 1-d. Instead of diverging exponentially with the distance from the critical point, the correlation length increases with an inverse power, as in an ordinary critical point. Finally we study the dependence of the critical exponents on $\sigma$ in the regime $0 < \sigma < 2$, and compare with field theoretic predictions. In particular we discuss in detail whether the critical behavior for $\sigma$ slightly less than 2 is in the short range universality class, as conjectured recently by F. Linder et al.. As in I we also consider a modified version of the model where only some of the contacts are long range, the others being between nearest neighbors. If the number of the latter reaches the percolation threshold, the critical behavior is changed but the supercritical behavior stays qualitatively the same.

I. INTRODUCTION

Epidemic spreading processes have attracted increasing attention in the statistical physics community [1]. In the simplest case of short range infection, no cooperative effects [2, 3], and ‘removal’ after the infection (i.e. either immunization or death), this ‘general epidemic process’ [4, 5] generates ordinary percolation clusters. In the present paper we shall deal with the generalization to the case where the process lives on a 2-d square lattice and at least some of the infections have long range. More precisely we shall study cases where each site can infect a finite number of other sites, and the probability for an infection to ‘jump’ a distance vector $x$ decreases for large $x$ as $p(x) \sim |x|^{-\sigma-d}$ with $d = 2$.

Models of this type were first suggested by [4]. A first calculation of critical exponents in [6] was flawed, as pointed out by Janssen [7], and numerical verifications of the exponents calculated in [7] where presented in [8].

These works were done in the spirit of the renormalization group (RG) for critical phenomena, and have to be seen in the context of other models with long range interactions, most prominently the Ising model [9–15]. As we shall discuss later in detail, they give most unambiguous results for $\sigma \approx 0$, where analytic results can be obtained by means of perturbative field theoretic RG.

Related to this was work on the case $\sigma = d$, which started already very early with papers by Ruelle [16], Dyson [17], Anderson [18], Thouless [19] and others. The main result obtained there was that the 1-dimensional Ising model with $\sigma = 1$ not only has a phase transition, but that the critical point is of Kosterlitz-Thouless (KT) type: Below the critical temperature one finds power laws with continuously varying exponents, while near the critical point the correlation length increases exponentially with the inverse distance from it. The most detailed numerical verification of the Ising model predictions is in [14]. Analogous mathematical results for the Potts model and for percolation were obtained in [20–23], but where not tested numerically for more than two decades, because of the obvious problem of simulating the very large lattices required to overcome finite size effects. They were verified only recently in [24] (denoted as I in the following), where hashing was used to simulate 1-d lattices of size $L = 2^{64}$.

Independently of the above mentioned work on SIR epidemics with long range contacts leading to power laws, increasingly much interest has been devoted recently to supercritical epidemics with $0 < \sigma < d$. Partly this comes from the interest in the navigability of small world networks [25], partly from interest in the spreading of various agents (viruses like influenza or HIV, computer viruses, rumors, and even money [26, 27]) on real world networks. But even apart from such epidemic-like processes, spatial networks with long range links have become powerful paradigms for complex real world systems [28]. Supercritical SIR epidemics with long range infection provide the easiest and most straightforward way to generate them.

The main objective of the present paper is to extend the simulations of I to two spatial dimensions. Again we shall use hashing, in order to simulate square lattices with $2^{48}$ sites. We could have easily increased this to $2^{128}$ sites with only minor loss of efficiency, but we checked that finite size effects can be safely neglected already with $2^{64}$ sites, for nearly all values of $\sigma$ (except for $\sigma$ very close
and below zero) and for all observables. While these simulations have no finite lattice size corrections, there are of course finite cluster size corrections, or equivalently corrections to scaling due to the finite duration of the epidemic. We thus complement these simulations also with simulations on finite lattices (up to $65536 \times 65536$) where we followed all epidemics until they died. In Sec. 2 we shall define the models in detail, and sketch the main results. Supercritical epidemics with $0 < \sigma < 2$ will be discussed in Sec. 3, while the case $\sigma = 2$ is treated in Sec. 4. Finally, the critical case is studied in Sec. 5, and our conclusions are drawn in Sec. 6.

II. THE MODEL AND BASIC FEATURES

We shall only deal with basic SIR epidemics on the square lattice with periodic boundary conditions. In all simulations the lattice size is $2^d$ sites, time is discrete and the infective period is one time step, after which sites become immune. We start always with a single infected site at $t = 0$, all other sites being susceptible. In each time step every infected site tries to infect in average $k$ neighbors other sites. If these sites are no longer susceptible, no replacements for them are chosen, i.e. the number of new infections is just reduced.

As in I, we consider two different models as how the target sites for new infections are selected. Most of the simulations were done for model (A), where each target site with distance vector $x$ from the infectious site is chosen randomly with probability $p(x)$. More precisely, we select first $k_0 = \lfloor k_{out} \rfloor$ such sites, and then one more site is chosen with probability $p = k_{out} - k_0$.

In model (B) (studied only in Sec. IV) we first infect each of the four neighbors with probability $q \in [0,1]$, and then choose $k_{out} - 4q$ additional targets as in model (A). For $q > 1/2$ this means that the process is always supercritical, and for $q = 1$ it implies that every epidemic always leads to an infinite cluster. We include this model for comparison with results in the mathematical literature [29, 33], where the emphasis was on supercritical long range percolation.

Following Linder et al. [8], we define $p(x)$ for $\sigma > 0$ implicitly by the following simple algorithm:

(i) We first chose two random numbers $u, v$ uniformly from $[0,1] \times [0,1]$.
(ii) If $w^2 \equiv u + v^2 > 1$, we discard them and choose a new pair until $w^2 < 1$.
(iii) Finally, we define

$$ x = \pm \frac{u}{w^{1+2/\sigma}}, \quad y = \pm \frac{v}{w^{1+2/\sigma}} $$

where all four sign combinations are chosen with equal probability. (iv) When computing the position of the new infected site, we added $x$ and used periodic boundary conditions.

For $\sigma \leq 0$ this can be modified suitably, but we shall not give any details as we will in the following show only simulations for $\sigma > 0$. These simulations took in total about four CPU years on modern PCs.

The phase diagram for model (A) is shown in Fig. 1. Below the continuous curve, i.e. for small values of $k_{out}$, there are only finite clusters with probability 1. Infinite epidemics can exist only above this critical curve $k_{out} = k_c(\sigma)$. For supercritical epidemics we have three regimes: For $\sigma < 0$ (i.e., to the left of the left dashed line) the process is of mean field type; for $0 < \sigma < 2$ (between the dashed lines) it is of intermediate type; and for $\sigma > 2$ it is basically as in short range percolation. For critical epidemics, one has three analogous regimes, but the boundaries between them are different. The boundary between mean field and intermediate-range epidemics is at point B, while the one between intermediate-range and short range is either at point D or near point C.

![Figure 1: Color online Phase diagram for model (A). Below the continuous curve, i.e. for small values of $k_{out}$, there are only finite clusters with probability 1. Infinite epidemics can exist only above this critical curve $k_{out} = k_c(\sigma)$. For supercritical epidemics we have three regimes: For $\sigma < 0$ (i.e., to the left of the left dashed line) the process is of mean field type; for $0 < \sigma < 2$ (between the dashed lines) it is of intermediate type; and for $\sigma > 2$ it is basically as in short range percolation. For critical epidemics, one has three analogous regimes, but the boundaries between them are different. The boundary between mean field and intermediate-range epidemics is at point B, while the one between intermediate-range and short range is either at point D or near point C.](image-url)
i.e. to the left of point B. For \( \sigma > 2/3 \) all critical exponents depend continuously on \( \sigma \), until the short range regime is reached. According to 8, this happens at point C which corresponds to \( \sigma_C = 43/24 = 1.79166 \ldots \) (\( \sigma_C = d - 2\beta/\nu \) in general), where \( \beta \) and \( \nu \) are the order parameter and correlation length critical exponents for ordinary (short-range) percolation. Our own simulations, presented in Sec. 5, suggest that the short range regime extends only down to \( \sigma = 2 \). The detailed critical behavior for \( \sigma_C < \sigma \leq 2 \) is unclear.

At \( \sigma = 0 \) and \( \sigma = d \) also the spatial structure of clusters of removed sites changes qualitatively. Assume that the epidemic started at \( x = 0 \). Then the density of removed sites \( R(x, t) \) decreases for any \( t > 0 \) asymptotically as

\[
R(x, t) \sim |x|^{-\sigma-d} \quad \text{for} \quad |x| \to \infty
\]

for all \( \sigma > 0 \), while \( R(x, t) \) becomes increasingly flat when \( \sigma < 0 \). The latter is illustrated for \( d = 1 \) in Fig. 1 of I, while the former is illustrated in Fig. 2.

Eq. (1) holds also for \( 0 < \sigma < 2 \) when \( |x| \gg t \), but at smaller distances the density decays faster than a power law, see Fig. 3. At least for supercritical epidemics this transition between the behaviors illustrated in Figs. 2 and 3 happens exactly at \( \sigma = 2 \). Whether this is still true for critical epidemics will be discussed in Sec. 5.

### III. SUPERCritical Behavior for \( 0 < \sigma < 2 \)

Although most studies of percolation and epidemic processes in the physics literature deal only with critical cases, it is clear that supercritical processes are of utmost practical importance. Indeed, it can be argued that most real epidemics like rabies, HIV, or various strands of influenza are far supercritical, take place in two dimensions of space, and are transmitted – due to long distance travels – by effective contacts with long range. Accordingly, there are several papers in the recent physics literature where either the spreading or the topology of the generated clusters were studied.

Typical examples for the former are 34, 35 and 19, where front propagation was studied by mean field type arguments. While a finite speed of propagation was claimed in 27, velocities that increase exponentially with time were found in 34, 35. As regards the topology of the cluster of removed sites, the main discussion was whether it forms a small world network (with graph diameter increasing logarithmically with the number of sites), has dimension two (as for short range epidemics), or is in between 36, 40.

As seen from Fig. 2 the spatial structure of cluster is rather simple. For small distances from the seed, there is a region of constant density smaller than 1, while there is a power law decay at larger densities. Indeed, Fig. 2 and similar plots for other values of \( \sigma \in [0, 2] \) are compatible, for large \( t \), with a scaling form

\[
R(x, t) \sim \phi(|x|/\xi(t))
\]

with

\[
\phi(z) = \begin{cases} 
\text{const} & \text{for } z \ll 1, \\
z^{-2-\sigma} & \text{for } z \gg 1,
\end{cases}
\]

and a smooth cross-over between the two regimes. The fact that \( \phi(z) < 1 \) for all \( z \) can be shown easily. Indeed, since the chance for site \( x \) to get infected by site...
$y$ is $p(y - x)$, the probability for it to have never been infected is

$$1 - \lim_{t \to \infty} R(x) \geq \prod_{y} (1 - p(y - x)), \quad (4)$$

giving

$$\log[1 - \lim_{t \to \infty} R(x)] \geq \sum_{y} \log[1 - p(y)] > -\infty. \quad (5)$$

According to Eq. (2), the function $\xi(t)$ also controls the geometric average radial size,

$$r(t) \equiv \langle |x(t)|_{\text{geom}} \rangle \equiv \exp(\langle \ln |x(t)| \rangle \sim \xi(t). \quad (6)$$

It also controls the size of the cluster of infected sites at any given time, which is a fuzzy ring of radius $\xi(t)$.

If the size would increase exponentially with time, as obtained in mean field theory [34, 35], this would mean that the cluster has the small world property, since its graph diameter is $\leq 2t$. On the other hand, if the radius would increase linearly [27], its dimension would be $\leq 2$. Actually, as proven rigorously in [28, 33], neither is correct. Instead, the size increases for any dimension $d$ like a stretched exponential,

$$r(t) \sim \exp(a t^{\gamma}) \quad (7)$$

with

$$\gamma = \gamma(\sigma) = \frac{1}{\log_{2} \frac{d + \sigma}{2d}}. \quad (8)$$

For $\sigma \to 0$ one has $\gamma \to 1$, i.e. one obtains the small world behavior of mean field theory. For $\sigma \to 2$, on the other hand, $\gamma \to 0$. Qualitatively similar behavior was suggested in [40] on the basis of numerical simulations, but the detailed functional form of $\gamma(\sigma)$ obtained in [40] was different.

Verifying Eqs. (7) and (8) numerically is not easy:

- First of all, for finite $t$ slightly different results are obtained when $r(t)$, the number $n(t)$ of active sites, or the number $N(t)$ of removed (‘immune’) sites is considered, although they all should show the same asymptotic behavior up to powers of $t$. In the following we shall concentrate on $n(t)$.

- Secondly, as found also in I, directly fitting $n(t)$ with a stretched exponential gives much too large estimates for $\gamma$. It is much better to define an effective growth rate

$$\alpha(t) = \ln \left[ \frac{n(t + 1/2)}{n(t - 1/2)} \right]. \quad (9)$$

which should, according to Eq. (7), decrease as $\alpha(t) \sim t^{1-\gamma}$.

\begin{itemize}
  \item For small values of $\sigma$, $n(t)$ does increase exponentially with $t$ for very long times, if $k_{\text{out}}$ is not very large. The reason is that the deviation from exponential increase is due to saturation effects, and these set in very late for small $\sigma$ and $k_{\text{out}}$. This is clearly seen in Fig. 4, where $\alpha(t)$ is plotted against $t$ on a log-log plot. The straight dashed line is the prediction of Eqs. (7) and (8), and it gives a decent fit only for $t > 30$. Due to this effect, we were not able to verify Eq. (5) for $\sigma < 0.3$.

  \item As seen in the insert in Fig. 4, even for large values of $t$ there are strong systematic deviations from the predicted asymptotic behavior (notice that statistical errors in Fig. 4 are much smaller that the line width). Similar systematic corrections were also found for all other values of $\sigma$ and $k_{\text{out}}$. It seems that they were underestimated in I and are responsible for most of the systematic overestimation of $\gamma$ found in that paper.
\end{itemize}

In spite of these problems, our final results shown in Fig. 5 are fully compatible with Eqs. (7), (8) and definitely rule out the alternative conjecture of [40].

IV. THE CASE $\sigma = 2$

In the supercritical phase, the transition between intermediate and short-range behaviors happens at $\sigma = d$. For $d = 1$ we found in I that the number of infected sites increases for $\sigma = 1$ with a power law

$$n(t, k_{\text{out}}) \sim t^{\gamma(k_{\text{out}})}, \quad (10)$$
provided \( k_{\text{out}} \) was larger than a critical value \( k_c \). The power \( \eta \) depended continuously on \( k_{\text{out}} \), and the behavior near the critical point \( k_c \) was of Kosterlitz-Thouless type with the critical length (and time) increasing like a stretched exponent with the distance from criticality. The order parameter (the density of the infinite cluster) was a constant \( >0 \) at criticality, showing that the transition is first order.

Indeed, except for details this behavior for \( d = 1 \) had been predicted long ago, and some aspects had even been proven rigorously \([20][23]\). Comparably detailed predictions are not available for \( d = 2 \). It was conjectured in \([30]\) that scaling with continuously varying exponents as in Eq. (10) holds for model (B) with \( q = 1 \) (i.e., when every site is connected to each of its neighbors, so that \( k_c = 4 \)), but neither the dependence of \( \eta \) on \( k_{\text{out}} \) nor the behavior in model (A) are known.

Values of \( n(t) \) for model (A) and for different values of \( k_{\text{out}} \) are shown in Fig. 5. We see that indeed all curves for \( k_{\text{out}} \geq 1.503 \) show power laws for large \( t \), with exponents decreasing with \( k_{\text{out}} \). As \( k_{\text{out}} \) approaches \( k_c \approx 1.5007 \) from above, this asymptotic power law sets in later and later. At the same time, in this limit a different power law is observed for small \( t \), with an exponent \( \eta(k_c) \approx 0.6 \) which is compatible within errors with the exponent for critical SIR epidemics with short range contacts \([41]\) (a more detailed comparison with short range SIR epidemics will be given in the next section).

For model (B) with \( q > 0.5 \) the behavior is slightly different. In that case the process is always supercritical, and thus the asymptotic power laws hold down to \( t = O(1) \). There is no time regime for small \( k_{\text{out}} \) where the asymptotic power law is replaced by a different power law. This is not true for model (B) with \( q = 0.5 \). Although the process is also in this case supercritical for any number of long range contacts (and thus the threshold value of \( k_{\text{out}} \) is also trivial), we found that otherwise the behavior is similar to the one shown in Fig. 6. When \( k_{\text{out}} \) converges from above towards the critical value \( k_c = 2 \), \( \eta \) converges to a value that is larger than the value for short range critical SIR epidemics. Indeed this value seems to be compatible with the limit found for model (A), i.e.

\[
\eta_c \equiv \lim_{k_{\text{out}} \to k_c} \eta(k_{\text{out}}) = 1.108(2)
\]

for both model (A) and model (B) with \( q = 1/2 \).

The exponents \( \eta(k_{\text{out}}) \) are plotted against \( k_{\text{out}} \) in Fig. 6 (see the uppermost curve corresponding to the largest \( k_{\text{out}} \)). Statistical errors are comparable to or smaller than the line width.

\[
\eta \approx 2.13 + 0.667 \ln k_{\text{out}}.
\]

Indeed, this behavior is common to both models, because short range bonds (which make the entire difference between models (A) and (B)) are irrelevant when \( k_{\text{out}} \gg 1 \).

The detailed threshold behavior of \( \eta \) cannot be seen from Fig. 7, therefore we show in Fig. 8 the same data plotted on a log-log plot, where we also changed the abscissa from \( k_{\text{out}} \) to \( k_{\text{out}} - k_c \) and the vertical axis from \( \eta \) to \( \eta - \eta_c \). We see clear indications for power laws in model (A) and in model (B) with \( q = 1 \), while the data suggest a different behavior for model (B) with \( q = 1/2 \) (more precisely, the data for model (B) with \( q = 1 \) suggest

\[
\eta - \eta_c \sim (k_{\text{out}} - k_c)^{0.3},
\]

while for model (A) we find

\[
\eta - \eta_c \sim (k_{\text{out}} - k_c)^{1/2},
\]

For \( d = 1 \), the percolation transition at \( \sigma = d \) is known to be discontinuous \([22][23]\), as found also numerically in...
The huge corrections to scaling visible in Fig. 9 indicate \( \sigma > 1 \), surprising, as we expect this scaling for all ordinary (short range) critical percolation. This is not compatible with the order parameter exponent \( \beta \) that we should be careful with any quick conclusion. The detailed behavior at the critical point will be discussed in the next section.

V. CRITICAL BEHAVIOR

A. “Easy” regions and general overview

First we shall discuss the “easy” regions, i.e. values of \( \sigma \) far from the transition points B, C and D in Fig. 1, while the vicinities of these points will be discussed in later subsections.

For \( \sigma \ll 2/3 \) one is far in the mean field regime. We do not show any data, but it suffices to say that all critical exponents agreed with their predictions to very high accuracy. There was also no problem near point A in Fig. 1, where the supercritical model changes from mean field to the intermediate phase. We expect of course highly non-trivial behavior as one goes from the critical line into the supercritical phase, when \( 0 < \sigma < 2/3 \) (see I for the analogous situation in \( d = 1 \)), but this seems to leave no traces on the critical line. Similarly, there is no problem for \( \sigma \gg 2 \), where we recover short range behavior.

For intermediate values \( 2/3 < \sigma < 2 \) we do not have exact predictions for the critical exponents, but in the central part of this region, say \( 0.9 < \sigma < 1.4 \), we find rather clean scaling laws with only moderate corrections to scaling. Typical results obtained for \( \sigma = 1.25 \), e.g., are shown in Figs. 10 to 15.
are shown in Figs. 11 and 12. They show that power laws (geometric) average radius of the cluster of infected sites \( k \) are indeed observed for the same value of \( k \). Also they do not prevent precise estimates of \( k \) the data for \( t \) reaches the critical point. More precisely, in order to do justice to the very small error bars, we plotted \( n(t)/t^n \) for various values of \( t \) as a function of \( t \). We see a good data collapse both in Fig. 13 and in Fig. 14. In order to improve the accuracy, we plotted \( n(t, \epsilon) \approx t^n F(\epsilon t^{1/\nu}) \),

\[
n(t, \epsilon) \approx t^n F(\epsilon t^{1/\nu}),
\]

where \( F(z) \) is an everywhere analytic universal scaling function.

The most precise way to measure it in ordinary percolation is to relate \( \partial n(t, \epsilon)/\partial \epsilon \) to correlations between cluster sizes and cluster perimeter lengths \( [42,44] \). In the present case this cannot be used, so we had to estimate it from data collapse plots, although these are notoriously unreliable in the presence of strong corrections to scaling. Results for \( \sigma = 1.25 \) are shown in Fig. 13 and analogous results for ordinary bond percolation are shown for comparison in Fig. 14. In order to improve the accuracy, we plotted in both cases not \( t^{-\eta} n(t, \epsilon) \) against \( x \equiv \epsilon t^{1/\nu} \), but we multiplied the former by a suitable exponential \( e^{ax} \), where the constant \( a \) is chosen such as to make the scaling function \( F(z) \) horizontal at \( z = 0 \).

We see a good data collapse both in Fig. 13 and in Fig. 14 with obvious deviations near \( z = 0 \) due to scaling violations at small \( t \). In Fig. 14 we only plotted data for \( t > 5 \). For bond percolation we used of course the exactly known values for \( p_c \) and the estimates of the critical exponents from \([11]\), while we used in Fig. 13

In Fig. 10 we show the average number of infected sites as a function of \( t \) for various values of \( k_{out} \) near the critical point. More precisely, in order to do justice to the very small error bars, we plotted \( n(t)/t^n \), where \( n \) is an estimate for the critical exponent. If it is chosen correctly, the data for \( k_{out} = k_c \) should be horizontal for large \( t \). We see of course considerable corrections for \( t < 200 \), but they do not prevent precise estimates of \( n \) and \( k_c \).

Similar plots for the survival probability and for the (geometric) average radius of the cluster of infected sites are shown in Figs. 11 and 12. They show that power laws

\[
P_{\text{surv}}(t) \sim t^{-\delta}, \quad r(t) \sim t^{1/z}
\]

are indeed observed for the same value of \( k_{out} \). Also they satisfy the hyperscaling relation \( \eta = d/z - 2\delta - 1 \).

While \( \eta, \delta \), and \( z \) are sufficient to describe scaling exactly at the critical point, one more exponent is needed to describe scaling near the critical point. For this we can use e.g. the order parameter exponent \( \beta \) or the correlation length exponent \( \nu \). For our growth simulations, the most convenient exponent is, however, \( \nu \), which describes how the correlation time diverges as \( k_{out} \to k_c \). It also describes how fast the different curves in Figs. 10 to 12 diverge at \( t \to \infty \). Technically it is defined by defining first \( \epsilon = k_{out} - k_c \), and using then the finite-time scaling ansatz

\[
n(t, \epsilon) \approx t^n F(\epsilon t^{1/\nu}),
\]
the parameters determined from Fig. 10. We obtained \( \nu_t = 1.176(14) \).

Finally, let us discuss the densities of removed sites, i.e. the densities of the percolation cluster at different times (the densities of active sites are trivially related). In Fig. 15 we see two different power laws (as also found in I for the 1-d case). More precisely, we find the same scaling law Eq. (2), but with

\[
\phi(z) = \begin{cases} 
  z^{-2+\sigma} & \text{for } z \ll 1, \\
  z^{-2-\sigma} & \text{for } z \gg 1,
\end{cases}
\]

Again \( \xi(t) \) can be taken equal to \( r(t) \), the geometric average cluster radius. As in the 1-d case this gives immediately a relation between the critical exponents [7],

\[
(1 + \eta)z = \sigma
\]

(17)

(notice that \( z \) was defined differently in I, as \( r \sim t^{z} \), while we now use the more conventional definition \( t \sim r^{z} \)). This is satisfied within statistical errors.

In general, we define the pair connectedness exponent \( \eta_{\text{pair}} \) by

\[
R(x, t) \sim r^{-\eta_{\text{pair}}}
\]

(18)

0.9
 0.1
 0.2
 0.3
 0.4
 0.5
 0.6
 0.7
 0.8
 0.5  1  1.5  2  2.5
η
σ
GEP
27/32 (σ-2/3)
FIG. 16. (Color online) The growth exponent \( \eta \) plotted against \( \sigma \). For \( \sigma \leq 2/3 \), the mean field prediction is \( \eta = 0 \). For ordinary percolation (“GEP”) the result of [41] is \( \eta = 0.58435(50) \). The leading order \( \epsilon \)-expansion result is as indicated by the tilted straight line.

FIG. 15. (Color online) Log-log plots of densities of removed sites for \( \sigma = 1.25 \) at \( t = 2000, 7000, \) and 22000. As in Figs. 2 and 3, the fluctuations at small \( r \) are artifacts. The straight lines indicate the power laws for small and large \( r \).

FIG. 14. (Color online) Plot analogous to Fig. 13 but for ordinary bond percolation. Here we used the exact value \( p_c = 1/2 \) and the critical exponent estimates from [41].

FIG. 13. (Color online) The quantity \( t^{-\eta} n(t, \epsilon) e^{3x} \) plotted against \( x \equiv \epsilon^{1/z} \), for \( \sigma = 1.25 \). Here, \( \epsilon = k_{\text{out}} - k_c \), and \( k_c \) and \( \eta \) were as determined from Fig. 10. The factor \( e^{3x} \) was added in order to make the scaling function horizontal at the origin and increasing thereby the visible resolution.

FIG. 12. The quantity \( n(t) / t^\eta \) plotted against \( x \equiv \epsilon t^{1/\nu} \), for \( \sigma = 25 \). Here, \( \epsilon = k_{\text{out}} - k_c \), and \( k_c \) and \( \eta \) were as determined from Fig. 10.
for \( r < r(t) \). We also obtain the correlation length exponent \( \nu \) by \( \nu = \nu_t / z \). Using these, we summarize our numerical results for the critical exponents in Figs. 16 to 22. In these plots we also indicate the results for ordinary percolation (the “General Epidemic Process, GEP”) and for mean percolation, and the \( \epsilon \)-expansion results of [7]. While these results need no further comment in the “easy” regions, the results in the “hard” regions will be discussed in the following subsections. Here we just point out that all exponents are very precisely given by the mean field values for \( \sigma < 3/2 \). For \( \sigma > 2 \) there are much larger corrections to the (presumably exact) GEP values, hinting at considerable finite-time corrections. We should say that the error bars in the “hard” regions are dominated by uncertainties in the extrapolation \( t \to \infty \). They are not straightforward statistical errors, and their estimation is highly subjective, as for all critical exponents. It is quite obvious that some error bars (e.g. those for \( \sigma > 2 \)) are wrong, but we included them on purpose, stressing thereby that meaningful error bars are virtually impossible. As we said, details are given in the following subsections.

In the intermediate region, different exponents follow the predictions of the \( \epsilon \)-expansion to varying degree. Overall, the agreement is best for \( \eta_{\text{pair}} \), while it is worst for \( z \).
FIG. 21. (Color online) The correlation length exponent \( \nu \) obtained by \( \nu = \nu_t/z \). We include it, and the exponent \( \beta \) in the next figure, as the only exponents that are not measured directly. The GEP value is \( \nu = 4/3 \).

FIG. 22. (Color online) The order parameter exponent \( \beta \) obtained by the hyperscaling relation \( \beta = \nu \eta_{\text{pair}}/2 \). The GEP value is \( \nu = 5/36 \). In the mean field regime \( \beta = 1 \), violating there the hyperscaling relation.

FIG. 23. (Color online) Log-linear plots of \( n(t) \) for \( \sigma = 2/3 \), for several values of \( k_{\text{out}} \) near \( k_c \). The central (near-horizontal) curve is obtained from \( \approx 10^9 \) clusters, accordingly its statistical errors are smaller than the line thickness. The dotted straight line is one (but not the preferred) possibility for logarithmic corrections.

FIG. 24. (Color online) Log-log plots of \( t^{-\eta} n(t) \) for \( \sigma = 0.7692 \) and \( \eta = 0.074 \), for several values of \( k_{\text{out}} \) near \( k_c \). None of the curves is linear, showing that the scaling limit is not yet reached even for \( t = 10^4 \). Curves for \( k_{\text{out}} \leq 0.058892 \) finally turn upward, showing that \( k_c \) is larger than this value. If we assume conservatively that the curve for \( k_{\text{out}} = 0.058895 \) becomes asymptotically horizontal, then

B. The critical case for \( \sigma \approx 2/3 \)

As we said, we encountered no problems for \( \sigma < 2/3 \). Results exactly at \( \sigma = 2/3 \) are shown in Fig. 23. We see that indeed \( \eta = 0 \) at the critical point, with very small but clearly visible corrections. These corrections are compatible with being logarithmic,

\[
n(t, k_c) \sim 1.074 + .007 \ln t,
\]

but the smallness of the amplitude suggests that they are more likely to be log-log corrections,

\[
n(t, k_c) \sim \text{const} + a \ln \ln t
\]

with \( a \approx 0.1 \). Precise fits of this form are if course meaningless, unless they were guided by theory.

The situation is much worse for \( \sigma \) slightly larger than \( 2/3 \), as also seen from Figs. 16 to 22. In Fig. 24, we show \( t^{-0.074} n(t) \) for \( \sigma = 0.7692 \). None of the curves in this plot is linear, showing that the scaling limit is not yet reached even for \( t = 10^4 \). Curves for \( k_{\text{out}} \leq 0.058892 \) finally turn upward, showing that \( k_c \) is larger than this value. If we assume conservatively that the curve for \( k_{\text{out}} = 0.058895 \) becomes asymptotically horizontal, then
the value \( \eta = 0.074 \) used in this plot is the correct exponent. But it is smaller than the value 0.087 predicted by the \( \epsilon \)-expansion, indicating that corrections to scaling are even larger than suggested by Fig. 24. Similar problems were seen also for \( \sigma = 0.714 \) and 0.833, and had been found also in I for \( d = 1 \).

C. The intermediate to short range cross-over

Even worse corrections to scaling were found at the cross-over from intermediate to short range behavior, near points C and D in Fig. 1.

The growth of \( n(t) \) at \( \sigma = 2 \) is shown in Fig. 25. More precisely, we show there \( t^{-0.64} n(t) \), where the power of the prefactor was chosen so that the most straight curves are roughly horizontal at large \( t \). But none of the curves is really straight. Moreover, for \( \sigma = 2 \) we expect all scaling laws to agree with short range epidemics. The scaling for the latter, corresponding to \( \eta = 0.58435(50) \), is indicated by the dashed-dotted line. Similar plots were obtained for all \( \sigma \in [1.5, 2.5] \). Results for

\[
\sigma = \sigma_C \equiv \frac{43}{24} = 1.7916 \ldots
\]

are e.g. shown in Fig. 26.

According to Ref. 8, the ordinary (short range, GEP) percolation universality class prevails for all \( \sigma > \sigma_C \). The main reason for this is that

\[
\eta_{\text{pair}} = 2 - \sigma
\]
\[ n(t) / t^{\eta} \exp\left[ 1.3 x \right] = (k_{out} - k_c) t^{1/\nu} \]

In Panel a the exponent \( \eta = 0.64 \) suggested by Fig. 25 is used (and \( \nu \) is fitted as \( \nu = 1.497 \)), while in Panel b the GEP exponents \( \eta = 0.5844 \) and \( \nu = 1.5078 \) are used. The curves correspond to the following values of \( k_{out} \): 1.492, 1.496, 1.498, 1.499, 1.4995, 1.500, 1.5004, 1.50055, 1.5007, 1.5008, 1.5010, 1.5020, 1.5030, 1.5050, and 1.510 (from left to right).

Lattices, for which finite lattice effects became a problem for large \( t \), while the above results are free of any finite lattice corrections. Still, the effects seen e.g. in Figs. 25 and 26 are hard to miss.

D. Finite lattice simulations at \( \sigma = \sigma_C \)

Finally, we performed also simulations on finite lattices, in order obtain independent estimates for the fractal dimension of the percolation cluster and for exponents \( \beta \) and \( \nu \). In I we had refrained from such simulations, because it was not clear how finite size effects should be described in the Kosterlitz-Thouless type transition that holds there at \( \sigma = d \). For \( d = 2 \), however, the transition is a standard second order phase transition (except for possible logarithmic corrections), and we can assume that the usual finite size scaling (FSS) applies.

In the following we show only results for \( \sigma = \sigma_C \), since there the discrepancy between the scenarios proposed in [8] and in the present paper is most clear. At face value, the simulations presented in the last subsection suggest that \( \beta > 5/36 \) and \( D_f < 91/48 \) (\( D_f \) is the fractal dimension of the cluster of “removed” sites), while according to [8] these deviations should vanish in the scaling limit \( L, t \to \infty \). In the above simulations we studied the limit \( L \to \infty \) for finite \( t \), while simulations on finite lattices allow us to study the limit \( t \to \infty \) for finite \( L \). It is hoped that both limits together can clarify the situation better than either limit by itself.

In Fig. 28 we show a data collapse based on the FSS ansatz

\[ M(L, k_{out}) = L^{D_f} G((k_{out} - k_c)L^{1/\nu}). \] (23)

where \( M \) is the mass of the cluster of “removed” (i.e. previously infected) sites. We see substantial scaling violations far away from the critical point, which was of course to be expected. The main plot of the figure suggests that these scaling violations are very small in the central (scaling) region of the plot. The insert shows that this is not quite true, but that the main violations come
from small values of \( L \). The data for large \( L \) suggest that

\[
D_f = 1.715(30) \quad \text{and} \quad \nu = 1.42(3). \quad (24)
\]

From these we obtain \( \beta = (2 - D_f) \nu / 2 = 0.202(20) \). These values are significantly different from their values for ordinary percolation, but are fully consistent with the values obtained in the last subsection.

While this is a further indication that the cross-over to ordinary percolation does not happen at \( \sigma = 1 \), it is not very convincing since scaling corrections are notoriously easy to miss in such data collapse plots. Therefore we also show in Fig. 29 log-log plots of \( L^{-\nu} M(L, k_{\text{out}}) \) against \( L \), for several values of \( k_{\text{out}} \) close to \( k_C \). Asymptotically, these curves should veer up (down), if \( k_{\text{out}} \) is larger (smaller) than \( k_C \). From Fig. 26 (and from the corresponding plot for \( P_{\text{surv}} \)) we see two main alternatives.

- Either (a): \( \nu_t \) assumes the GEP value, in which case \( k_C \leq 1.40835 \);
- Or (b): \( \nu_t \approx 0.67 \), in which case \( k_C = 1.40838(15) \).

The data in Fig. 26 clearly indicate that the value 1.4085 is supercritical, while 1.408 and 1.4083 are subcritical, in perfect agreement with Fig. 26. But they also suggest strongly that \( k_{\text{out}} = 1.40838 \) is very close to critical. In this case the fractal dimension would be \( D_f = 1.707(7) \), which is definitely smaller than the value 0.91/48 = 1.896 of ordinary percolation. On the basis of Fig. 26 alone, a critical value 1.40835 seems unlikely but not excluded. But it would give an even smaller value of \( D_f \) and is thereby clearly excluded. Our final estimate is \( D_f = 1.707(17) \), where the uncertainty is mainly due to the uncertainty of \( k_c \).

VI. DISCUSSION AND CONCLUSIONS

This work was triggered by recent discussions in the physics literature of spatially embedded networks with long range connections \([23, 36, 40, 45, 49]\) and supercritical epidemic processes leading to such networks \([27, 34, 35]\). Our simulations showed that most of these speculations are obsolete, but in retrospect this was to be expected. The correct topology of such networks was known rigorously since 2004 \([29]\). If the probability to infect a neighbor at distance \( x \) decays as \( x^{-\sigma} \) with \( 0 < \sigma < s \), then the number of nodes reached by a path of length \( t \) increased like a stretched exponential with known (and non-trivial) exponent. Our simulations fully agree with this prediction, in spite of the notorious difficulty to fit stretched exponentials.

Another class of problems is concerned with the supercritical behavior at \( \sigma = d \). It was observed already in the late 1960’s that the case \( \sigma = d \) is special. For instance, the 1-d Ising model shows a phase transition for \( \sigma < 1 \), while it has no transition for \( \sigma > 1 \). For 1-d percolation, a seminal early result was that there is a discontinuous phase transition at \( \sigma = 1 \) with Kosterlitz-Thouless like behavior in the supercritical regime \([22]\).

No analogous result was known in two dimensions, although there are conjectures \([30]\) that there also might exist continuously varying exponents in the supercritical phase. In the present paper we verify this conjecture and suggest various scaling laws related to it.

In contrast to the 1-d case, the percolation transition in the 2-d case with \( \sigma = 2 \) is continuous. Indeed, there are very strong theoretical arguments that the critical exponents at this point are exactly those of ordinary percolation or, as far as temporal aspects are concerned, of the “general epidemic process” (GEP). On the other hand, there is a long-standing debate for the Ising model (where the situation in this respect should be very similar) whether the ordinary short-range behavior should end at \( \sigma = 2 \) or extend some way into the region \( \sigma < 2 \) \([9-13, 15]\). This debate is mostly centered around field theoretic arguments, but the most recent simulations \([15]\) seemed to have settled the problem: The ordinary short-range behavior extends a finite amount into the region \( \sigma < 2 \). The same conclusion was reached for percolation in \([8]\).

Our present simulations show rather convincingly that this is wrong – at least for percolation, but the theoretical analogy suggests also for the Ising model. There is a singularity at \( \sigma = 2 \), and at least some of the critical exponents are different for all \( \sigma < 2 \) from those for ordinary percolation. While we are rather confident about this basic claim \([17]\), details are much harder to pin down due to huge corrections to scaling. This represents the main open problem related to the present paper.

The field theory that had given rise to the above debate can be treated by renormalization group methods near \( \sigma = d/3 \) \([7]\), where a field theoretic e-expansion predicts anomalous critical exponents up to first or-
In summary, percolation with long range infection is a fascinating problem. It touches basic questions of renormalization group theory, it has applications to real-world epidemics, and it sheds light on the structure of real-world complex networks. And, finally, it still shows a number of open questions after having been studied for more than 40 years.

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