On the Critical Behavior of the General Epidemic Process and Dynamical Percolation

P. GRASSBERGER
Physics Department, University of Wuppertal, W. Germany and
Chemical Physics Department, The Weizmann Institute of Science, Rehovot, Israel

Received 10 February 1982; revised 20 July 1982

ABSTRACT

Scaling laws are formulated for the behavior of a space-dependent fluctuating general epidemic process near the critical point. Restricted to stationary properties, these laws describe also the critical behavior of random percolation. Monte Carlo calculations are used to estimate the critical exponents and the universal shape of the propagating wave, in the case of 2-dimensional space.

1. INTRODUCTION

The general epidemic process [1] (GEP) is a stochastic multiparticle process which should describe the essential features of a vast number of population growth phenomena. They all have in common that the population needs some "food supply" in order to survive and propagate, and that no additional food is provided during the process. Thus, in a finite environment, the population (or "epidemic" in the case of pests) can only reach a finite maximal size, after which it decreases and finally will become extinct. It can survive forever only in an infinite environment. If this is homogeneous (up to small fluctuations), it will move through it as a solitary wave, leaving an exhausted region behind it. Well-known examples are the growth of mushrooms in fairy rings, chemical solitary waves [2], forest fires, and, of course, epidemics.

The "food" in the "exhausted" region will not be completely used up. On the contrary, when the conditions are just barely favorable for the survival of the population, then its maximal density, the velocity of the solitary wave, and the amount of used "food" per unit volume will all be very small. The transition from possible survival to certain extinction is analogous to a second-order phase transition, and when the conditions for survival become marginal, we expect to observe a critical phenomenon [3].

It is this critical phenomenon which we shall study in the present paper.

52 Vanderbilt Ave., New York, NY 10017 0025-5564/83/02157+16$03.00

In a second-order phase transition, one has an intensive property (e.g. the density in a liquid-gas transition at the critical temperature, the magnetization in a ferromagnet, the laser field in a laser, etc.) called the "order parameter." When changing a control parameter (temperature or pumping rate in the above examples), the order parameter passes through a singularity, but remains bounded and continuous (in contrast to first-order transitions, where it is discontinuous). In the GEP, we can take e.g. the infection rate as control parameter analogous to the temperature, while the overall density of "food" used up can serve as an order parameter.

As one approaches the singularity, strong and long-ranged fluctuations build up. Any details of the system then become irrelevant if they are detectable only by looking at length scales much smaller than the correlation length of these fluctuations. If the system is homogeneous on large length scales, we thus expect the behavior to depend only on global properties like dimensionality, nature of the order parameter (e.g. scalar versus vector), or ergodicity (the GEP is of course not ergodic). This "universality" allows us to extract *quantitative* results from studies of highly simplified models, and to apply them in vastly varying circumstances without essential modifications.

There exist a large number of versions of the GEP in the literature. Some treat the process as deterministic, while others include fluctuations; some treat the fluctuations as homogeneous (corresponding to infinite mobility of the individuals), others as inhomogeneous; some assume that the individuals make random walks, others assume them to stay fixed in the space, the epidemic spreading only via nonlocal infections; etc.

In the critical case, fluctuations are of course essential, and one has to treat them inhomogeneously (the deterministic approximation is discussed in Section 4). But in analogy with other critical phenomena, it will not depend on many details of the model. The full extent of this "universality" is hard to assess, and in the nonrigorous treatment of the present paper we shall as usually rely on intuition.

In order to be more precise, we shall discuss in the next section a specific version of the GEP. In this section, we shall also elaborate on the relationship to percolation. In Section 3, the scaling laws pertinent to the critical behavior will be formulated. After a brief discussion of the deterministic approximation in Section 4, we shall present in Section 5 results of Monte Carlo simulations. In the final discussion in Section 6 we shall in particular try to estimate the size of the universality class, i.e. the range of models which should show the critical phenomena discussed in this paper.

2. THE BASIC MODEL AND PERCOLATION

The simplest stochastic and space-dependent model can be described as follows [1]. The individua live at the sites of an infinite d-dimensional cubic

lattice. Every lattice site is occupied by exactly one individuum, which cannot move away from it. The individua (or, as we shall briefly say, the sites) can be susceptible (denoted by X), infected (Y), or immune (Z). The process is assumed to be markoffian and proceeds via infection of nearest neighbors with rate σ:

$$X(\mathbf{n}) + Y(\mathbf{m}) \xrightarrow{\sigma} Y(\mathbf{n}) + Y(\mathbf{m}) \tag{2.1}$$

($\mathbf{n}, \mathbf{m}$ are nearest neighbors), and by recovery with rate ρ:

$$Y(\mathbf{n}) \xrightarrow{\rho} Z(\mathbf{n}). \tag{2.2}$$

Notice that there is no latency period.

Whether a single infected site can give rise to an infinite epidemic depends of course of the ratio σ/ρ. Let us assume ρ to be fixed. Then there exists a critical value σ_c such that for $\sigma < \sigma_c$ no infinite epidemic is possible. For $\sigma > \sigma_c$, an infinite epidemic will occur with probability $0 < P(\sigma) < 1$.

The problem of whether an infinite epidemic is possible is clearly a percolation [4] problem: the probability $P(\sigma)$ is just the chance that the original infected site belongs to an infinite cluster.

In discussing percolation, one usually assumes that it is decided from the very beginning whether a certain site (or link) can be passed or not, it being a property of the lattice. In the GEP, one assumes in contrast that the link between two sites is established (or not) only at the moment when the infection is to pass across it. Since infection can however pass only once through any link, this difference does not matter mathematically.

The GEP defined by Equations (2.1) and (2.2) corresponds to neither site nor bond percolation. Notice that Equation (2.2) implies that the times of illness are exponentially distributed with mean value $T = 1/\rho$. Let us now replace this by the assumption—which for many diseases is more realistic anyhow—that these times are all equal to a fixed time T. Then the chances that an infection can pass from site $\mathbf{n}$ to a neighbouring site $\mathbf{m}$ (provided the latter is still susceptible) are all independent and equal to

$$p = 1 - e^{-\sigma T}. \tag{2.3}$$

It is easy to see that this is precisely the problem of bond percolation, with probability p for the bonds to be unbroken.

On the other extreme, one might keep the exponential distribution of times of illness, but assume that every infection hits *all* susceptible next neighbors *simultaneously*. Then $p = \sigma/(\sigma + \rho)$ is the chance that an infection can pass through a site to all its neighbors, and we deal with site percolation.

This model is very close to the model of Alexandrowicz [5]. The "branch tips" of Reference [5] correspond to our infected sites, the boundary of the cluster as defined in Reference [5] corresponds to those immune cells which have not infected their neighbors.

3. CRITICAL BEHAVIOR

Assuming universality (which at least for site and bond percolation is well established [4], we know the scaling properties of the distribution of immune individuals *after* the epidemic. We shall only mention a few results, referring to Reference [4] for more details.

For $\sigma > \sigma_c$, both the probability $P(\sigma)$ of an infinite epidemic and the overall average density $Z(\sigma)/P(\sigma)$ of immunes in the case of the latter are proportional[1] to the percolation probability:

$$P(\sigma) \propto \frac{Z(\sigma)}{P(\sigma)} \propto (\sigma - \sigma_c)^{\beta} \qquad \text{for} \quad \sigma \downarrow \sigma_c. \tag{3.1}$$

The density $Z(\mathbf{n}; \sigma)$ of immunes at a distance $\mathbf{n}$ from the original infected site is equal[1] to the pair connectedness and behaves at $\sigma = \sigma_c$ like

$$Z(\mathbf{n}; \sigma_c) \propto |\mathbf{n}|^{-2\beta/\nu} \qquad \text{for} \quad |\mathbf{n}| \to \infty. \tag{3.2}$$

For $\sigma < \sigma_c$, finally, the average size of the epidemic (again arising from a single infected site) is equal[1] to the mean cluster size:

$$\sum_{\mathbf{n}} Z(\mathbf{n}; \sigma) \propto (\sigma_c - \sigma)^{-\gamma}, \qquad \gamma = d\nu - 2\beta, \tag{3.3}$$

while its spatial extension scales like[2]

$$\langle |\mathbf{n}| \rangle \equiv \frac{\sum_{\mathbf{n}} Z(\mathbf{n}; \sigma)|\mathbf{n}|}{\sum_{\mathbf{n}} Z(\mathbf{n}; \sigma)} \propto (\sigma_c - \sigma)^{-\nu}. \tag{3.4}$$

For $d = 2$, the critical exponents are [6,4] (at least approximately)

$$\nu = \tfrac{4}{3} \tag{3.5}$$

---

[1] Up to corrections which are inessential at the critical point.

[2] $\langle X \rangle$ is used throughout this paper for the expectation value of a quantity X.

and

$$\beta = \tfrac{5}{36} = 0.139. \tag{3.6}$$

While these "static" scaling laws have been studied in much detail, much less is known about the dynamical behavior.

Let us again assume that there was a single infected site at time $t = 0$, located at $\mathbf{n} = 0$. Then standard arguments [3] suggest that the density of immune sites can be written close to the critical point as

$$Z(\mathbf{n}, t; \sigma) \approx |\mathbf{n}|^{-2\beta/\nu} F\left(\frac{|\mathbf{n}|^{\tau}}{t^{\nu}}, \varepsilon t^{1/\tau}\right), \tag{3.7}$$

where

$$\varepsilon = \frac{\sigma - \sigma_c}{\sigma_c}, \tag{3.8}$$

τ is a new critical exponent (introduced first in Reference [5]), and $F(\xi, \zeta)$ is a universal[3] scaling function.

The arguments of F are usually chosen somewhat different. With our choice, we can assume that the same analytic function $F(\xi, \zeta)$ describes the subcritical ($\zeta < 0$) and the supercritical ($\zeta > 0$) domains.[4] In the usual formulation, one would use $|\mathbf{n}|\varepsilon^{\nu}$ and $t\varepsilon^{\tau}$ as variables of F, making an analytic extrapolation through $\varepsilon = 0$ impossible.

Analogously, we assume for the probability $P(t; \sigma)$ that there is at least one infected at time t (i.e. that the epidemic is not yet extinct at t):

$$P(t; \sigma) \approx t^{-\beta/\tau} \psi(\varepsilon t^{1/\tau}), \tag{3.9}$$

with another universal scaling function $\psi(\xi)$.

The powers of $|\mathbf{n}|$ and t in front of the scaling functions in Equations (3.7) and (3.9) have been fixed so that one recovers the scaling laws (3.1)–(3.4) for $t \to \infty$.

For $\sigma > \sigma_c$, and for $t \gg \varepsilon^{-\tau}$ and $|\mathbf{n}| \gg \varepsilon^{-\nu}$, we expect that the immune sites will be essentially uniformly distributed [with density $Z(\sigma)$] in a sphere with radius growing linearly with time. At the edge of this sphere, there will

[3] F is universal only up to rescaling. That is, a different model in the same universality class will have a scaling function $\bar{F}(\xi, \zeta) = aF(b\xi, c\zeta)$, with nonuniversal constants a, b, and c. Analogous remarks hold for all other scaling functions in this paper.

[4] For the epidemic process with recovery but without immunization—which is in the same universality class as Reggeon field theory [7] and directed percolation in $d + 1$ dimensions [8]—this has indeed been verified by Monte Carlo simulations [7].

be a solitary wave of infected sites. It moves outward with velocity v, and has a width Γ. Thus we expect

$$Z(\mathbf{n}, t; \sigma) \approx Z(\sigma) \cdot \vartheta\left(\frac{|\mathbf{n}| - vt}{\Gamma}\right) \tag{3.10}$$

with $\vartheta(\xi)$ being a universal kinklike scaling function:

$$\vartheta(\xi) \approx \begin{cases} 1 & \text{for } \xi < 0, \\ 0 & \text{for } \xi > 1. \end{cases} \tag{3.11}$$

Comparing this with Equation (3.7), we find

$$v \propto \varepsilon^{\tau - \nu} \tag{3.12}$$

and

$$\Gamma \propto \varepsilon^{-\nu}. \tag{3.13}$$

Finally, we might mention that, exactly at the critical point, the size of the epidemic increases according to Eq. (3.7) like

$$\sum_{\mathbf{n}} Z(\mathbf{n}, t; \sigma_c) \propto t^{\gamma/\tau}, \tag{3.14}$$

while its spatial extension increases like

$$\langle |\mathbf{n}| \rangle_t \propto t^{\nu/\tau}. \tag{3.15}$$

The mean number and distribution $Y(\mathbf{n}, t; \sigma)$ of infected sites can immediately be found from the above by using

$$Y(\mathbf{n}, t; \sigma) = \rho \frac{\partial}{\partial t} Z(\mathbf{n}, t; \sigma). \tag{3.16}$$

Alternatively, assume that at $t = 0$ the entire hyperplane $n_1 = 0$ is infected, and that the infection spreads into the positive n_1-direction. Instead of Equation (3.7), we then have

$$Z(n_1, t; \sigma) \approx n_1^{-\beta/\nu} G\left(\frac{n_1^\tau}{t^\nu}, \varepsilon t^{1/\tau}\right). \tag{3.17}$$

At the critical point, the mean number of immunes per unit hyperplane increases then like

$$\sum_{n_1 = 0}^{\infty} Z(n_1, t; \sigma_c) \propto t^{(\nu - \beta)/\tau}, \tag{3.18}$$

while both the average value of n_1 and its dispersion increase like

$$\langle n_1 \rangle_t \propto \left[V_{ar}(n_1) \right]^{1/2} \propto t^{\nu/\tau}. \tag{3.19}$$

4. DETERMINISTIC APPROXIMATION

In the next section, we shall present Monte Carlo calculations which support these scaling laws and yield values for the critical exponents and scaling functions. Before doing this, let us discuss briefly the critical behavior of the deterministic approximation.

Treating Equations (1.1) and (1.2) as deterministic, the equations of motion are [1]

$$\dot{X}(\mathbf{n}, t) = -\sigma X(\mathbf{n}, t) \sum_{\mathbf{m}}{}' Y(\mathbf{m}, t), \tag{4.1}$$

$$\dot{Y}(\mathbf{n}, t) = -\rho Y(\mathbf{n}, t) + \sigma X(\mathbf{n}, t) \sum_{\mathbf{m}}{}' Y(\mathbf{m}, t), \tag{4.2}$$

$$\dot{Z}(\mathbf{n}, t) = \rho Y(\mathbf{n}, t). \tag{4.3}$$

The sums in Equations (4.1)–(4.2) and in Equation (4.5) below extend over the next neighbors of $\mathbf{n}$, and the densities are subject to the constraint

$$X(\mathbf{n}, t) + Y(\mathbf{n}, t) + Z(\mathbf{n}, t) = 1. \tag{4.4}$$

One easily verifies that the quantities

$$\mathcal{K}(\mathbf{n}) = X(\mathbf{n}, t) \exp\left\{ \frac{\sigma}{\rho} \sum_{\mathbf{m}}{}' Z(\mathbf{m}, t) \right\} \tag{4.5}$$

are conserved: $\dot{\mathcal{K}}(\mathbf{n}) = 0$. Thus, if only the origin was infected at $t = 0$, one has

$$\left(1 - Z(\mathbf{n}, t) - \frac{1}{\rho} \dot{Z}(\mathbf{n}, t) \right) e^{(\sigma/\rho) \Sigma' Z(\mathbf{m}, t)} = 1 - \delta_{\mathbf{n},0}. \tag{4.6}$$

For large t and $|\mathbf{n}|$, one can neglect the time and space dependence in this equation, and gets [9]

$$\left[1 - Z(\mathbf{n}, t) \right] e^{(\sigma \mathcal{N}/\rho) Z(\mathbf{n}, t)} = 1, \qquad \mathcal{N} = \text{coordination number}. \tag{4.7}$$

For $\sigma \mathcal{N}/\rho < 1$, this has only the solution $Z = 0$. For $\sigma \mathcal{N}/\rho > 1$, one has in addition a solution with $0 < Z < 1$. Thus the critical point is

$$\sigma_c = \rho / \mathcal{N}. \tag{4.8}$$

In the critical region, $Z(\mathbf{n}, t)$ is small and slowly varying. Thus, Equation (4.6) can be approximated as

$$\frac{\varepsilon}{\mathcal{N}} \sum_{\mathbf{m}} Z(\mathbf{m}, t) - \frac{1}{\rho} Z(\mathbf{n}, t) + \tfrac{1}{2} Z^2(\mathbf{n}, t) \approx 0. \tag{4.9}$$

Inserting the scaling law (3.7), we find

$$\tau = 2\beta = 2\nu = 1. \tag{4.10}$$

This implies in particular that the velocity of the solitary wave scales like

$$v \propto (\sigma - \sigma_c)^{1/2}, \tag{4.11}$$

as discussed by Mollison [1]. Including fluctuations, the results of the next section yield instead in 2 dimensions $v \propto (\sigma - \sigma_c)^{0.173}$, while Reference [5] implies for 3 dimensions $v \propto (\sigma - \sigma_c)^{0.31}$.

One must however be careful: in the deterministic approximation, an infected site will always lead to an epidemic (provided $\sigma > \sigma_c$), and thus Equations (3.1) and (3.9) do not make sense. More generally, as the concept of a cluster is inappropriate in the deterministic approximation, the straightforward connection to percolation is lost. Accordingly, the value of β in Equation (4.10) is not the "classical" value for percolation.

5. MONTE CARLO CALCULATIONS

For convenience, we did not perform simulations for the basic model as it is defined in Section 2, but for two models which should be in the same universality class. Both have discrete time in addition to discrete space. We only performed calculations for $d = 2$.

In the first model, we chose at each step $t \to t + 1$ a site at random. If this site was susceptible or immune, we went on to $t + 2$. If the site was infected, we first allowed it to infect one of its four neighbors, provided it was susceptible. After this we allowed it to recover with probability p, and went on to $t + 2$. Notice that in the limit of an infinite lattice this is equivalent to a continuous-time model.

We made runs with two different initial conditions. In the first case, we started from one infected site in the middle of the lattice. In the other, we started with the whole edge $n_1 = 0$ of the lattice infected, and observed the epidemic growing into the lattice.

A typical result is shown in Figure 1, where the average number of infected sites, $\langle Y \rangle_t = \sum_{\mathbf{n}} Y(\mathbf{n}, t; \sigma)$, is shown on a log-log plot versus time. At $p = 0.6575$, 0.665, and 0.67, we have averaged over 400 runs each, every run

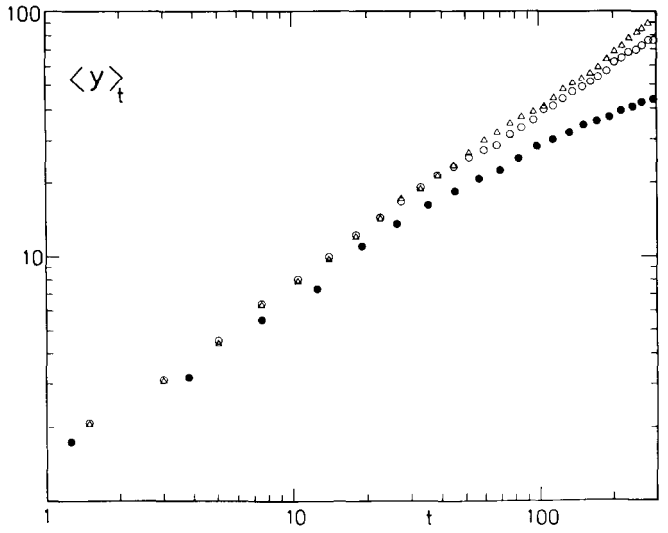

FIG. 1. First model; average number of infected sites versus time for $p = 0.6575$ (●), $p = 0.665$ (○), and $p = .67$ (△) on a log-log plot. The runs started with 1 infected site at $t = 0$, all others being susceptible.

starting with a single infected site at $t = 0$. Notice that the expected scaling law $\langle Y \rangle_t \propto t^{\gamma/\tau - 1}$ corresponds to a straight line in this plot. Similar graphs were obtained for the number of immunes, the survival probability $P(t; \sigma)$, and for the spatial extension of the epidemic. They all showed agreement with the scaling laws postulated in Section 3, with $p_c = 0.667 \pm 0.01$, $\nu = 1.4 \pm 0.1$, $\tau = 1.5 \pm 0.1$, and $\beta = 0.12 \pm 0.03$ (the errors are only rough estimates). The values for ν and β are in agreement with the more precise values of References [4] and [5].

We shall not present any more details on these results, as much more extensive runs were performed with the second model. This was chosen so as to coincide, with respect to its static properties, with bond percolation, where the critical percolation probability is rigorously known [10] to be $p_c = 0.5$. While knowledge of the exact value of p_c is of no great importance per se (populations usually do not live on square lattices), it helps of course enormously in estimating the critical exponents.

In this second model, each iteration $t \to t + 1$ implied a systematic sweep through the whole lattice. In this sweep, we first stored the updated state of each lattice site in a separate array, and only after the sweep did we replace the old state by the updated state. In one sweep, we allowed each infected site to infect *all* neighboring susceptible sites, each with probability p, and to recover with probability 1.

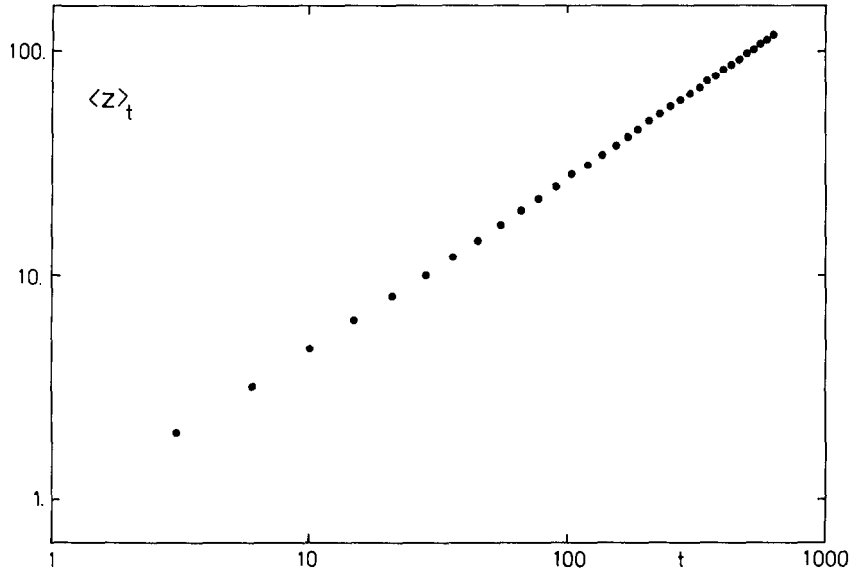

FIG. 2. Results from second model, with runs starting with the whole edge $n_1 = 0$ infected, and with $p = p_c = 0.5$. Statistical errors are only shown in Fig. 2b. Figure 2a. average numbers of immunes (including those at $n_1 = 0$) versus time.

We used multispin coding [11] in order to store the state of 30 sites in one 60-bit word, and to speed up the iterations. We started all runs with the whole edge $n_1 = 0$ infected, on lattices of size up to 350×870, with cylindrical boundary conditions, and performed up to 630 iterations.

The results of 200 such runs at $p = p_c = 0.5$ are shown in Figure 2. We find perfect agreement with the scaling laws (3.18) and (3.19), with

$$\frac{\nu - \beta}{\tau} = 0.807 \pm 0.01 \tag{5.1}$$

and

$$\nu/\tau = 0.885 \pm 0.01. \tag{5.2}$$

Using the values $\nu = \frac{4}{3}$ and $\beta = \frac{5}{36}$, we get

$$\tau = 1.494 \pm 0.015, \tag{5.3}$$

in agreement with the less precise value of Reference [5].

Among the scaling functions, the most interesting seems to be the function $\vartheta(\xi)$ describing the kink in Equation (3.10), or rather its derivative describing

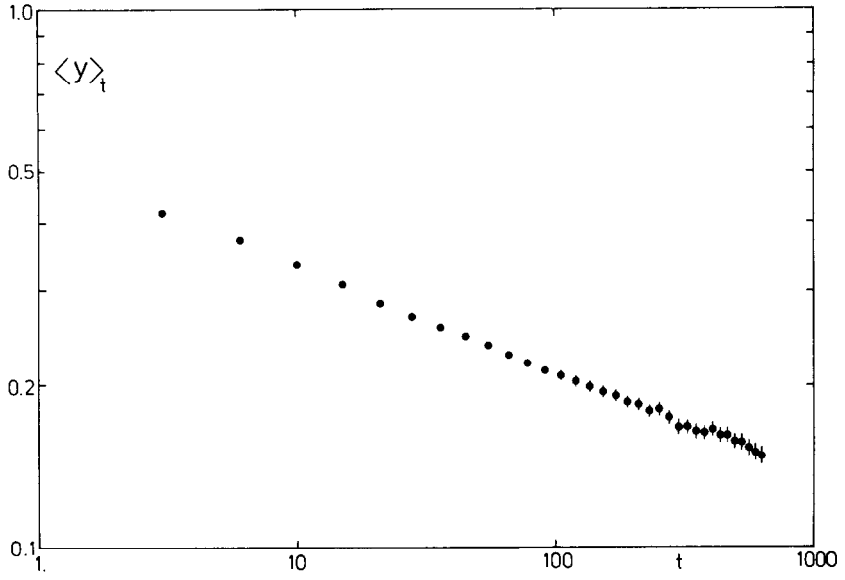

FIG. 2b. Average number of infected sites.

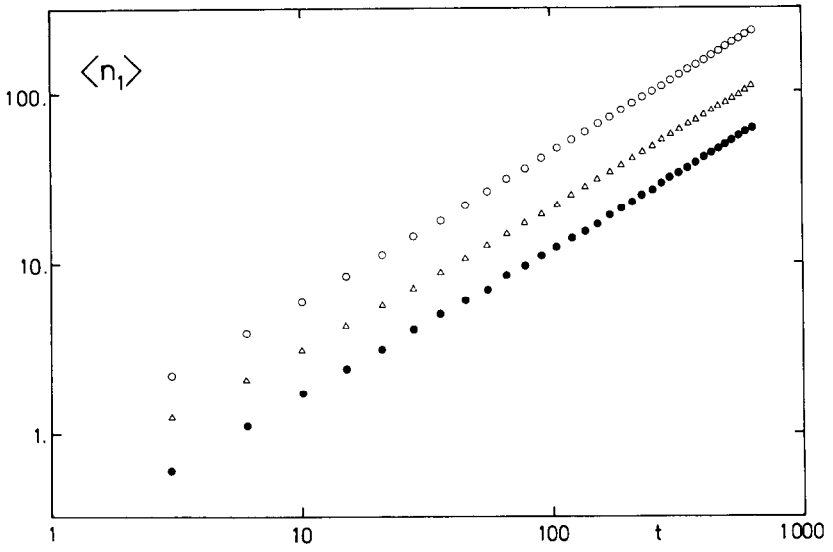

FIG. 2c. Average value of n_1 (excluding $n_1 = 0$) for infected sites (○) and for immunes (△), and dispersion of n_1 for infected sites (●).

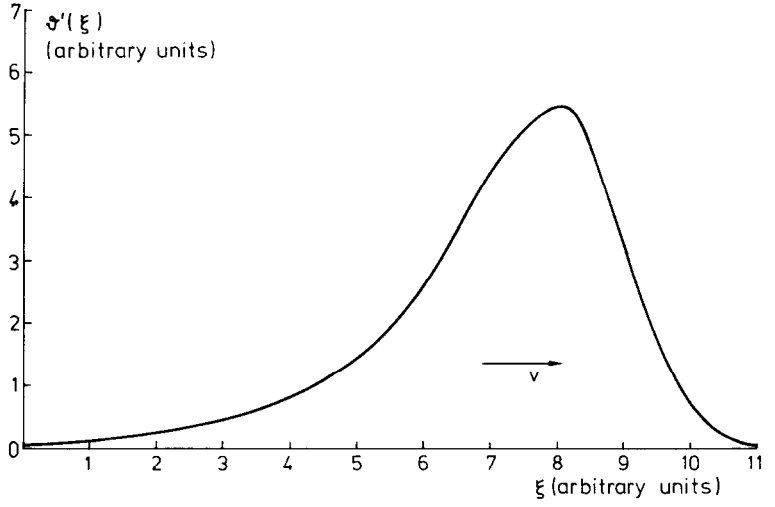

FIG. 3. Scaling function $\frac{d\vartheta(\xi)}{d\xi}$ describing the solitary wave of infected sites. The wave propagates from left to right.

the solitary wave of infected sites. The result from 100 runs with $p = p_c + 2^{-5}$ on a 200×870 lattice, and 100 runs with $p = p_c + 2^{-6}$ on a 260×870 lattice, are shown in Figure 3. Here, as the wave passes through a small area, the increase of the number of infected is much faster than the subsequent decrease. This is in contrast to the deterministic case, where at the critical point the wave has a symmetrical shape.

Finally, we present two graphs which support the scaling law (3.17) and our claim that scaling functions like $F(\xi, \zeta)$ and $G(\xi, \zeta)$ are analytic in ζ at $\zeta = 0$. From Equations (3.16) and (3.17), we expect for the mean number of infected sites

$$\sum_{n_1} Y(n_1, t; \sigma) \approx t^{(\nu-\beta)/\tau-1} \chi(\varepsilon t^{1/\tau}), \qquad \varepsilon = \frac{p - p_c}{p_c}, \tag{5.4}$$

and for their average distance from the border $n_1 = 0$ we expect

$$\langle n_1 \rangle \approx t^{\nu/\tau} \varphi(\varepsilon t^{1/\tau}). \tag{5.5}$$

The values of $t^{(\beta+\tau-\nu)/\tau} \sum_{n_1} Y(n_1, t; \sigma)$ are plotted in Figure 4(a) versus the variable $\zeta = \varepsilon t^{1/\tau}$, for seven different values of ε. Similarly, the values of $t^{-\nu/\tau} \langle n_1 \rangle$ are shown in Figure 4(b). We see that they indeed fall (at least for $t \gtrsim 15$) on common functions $\chi(\zeta)$ and $\varphi(\zeta)$, respectively, which are perfectly

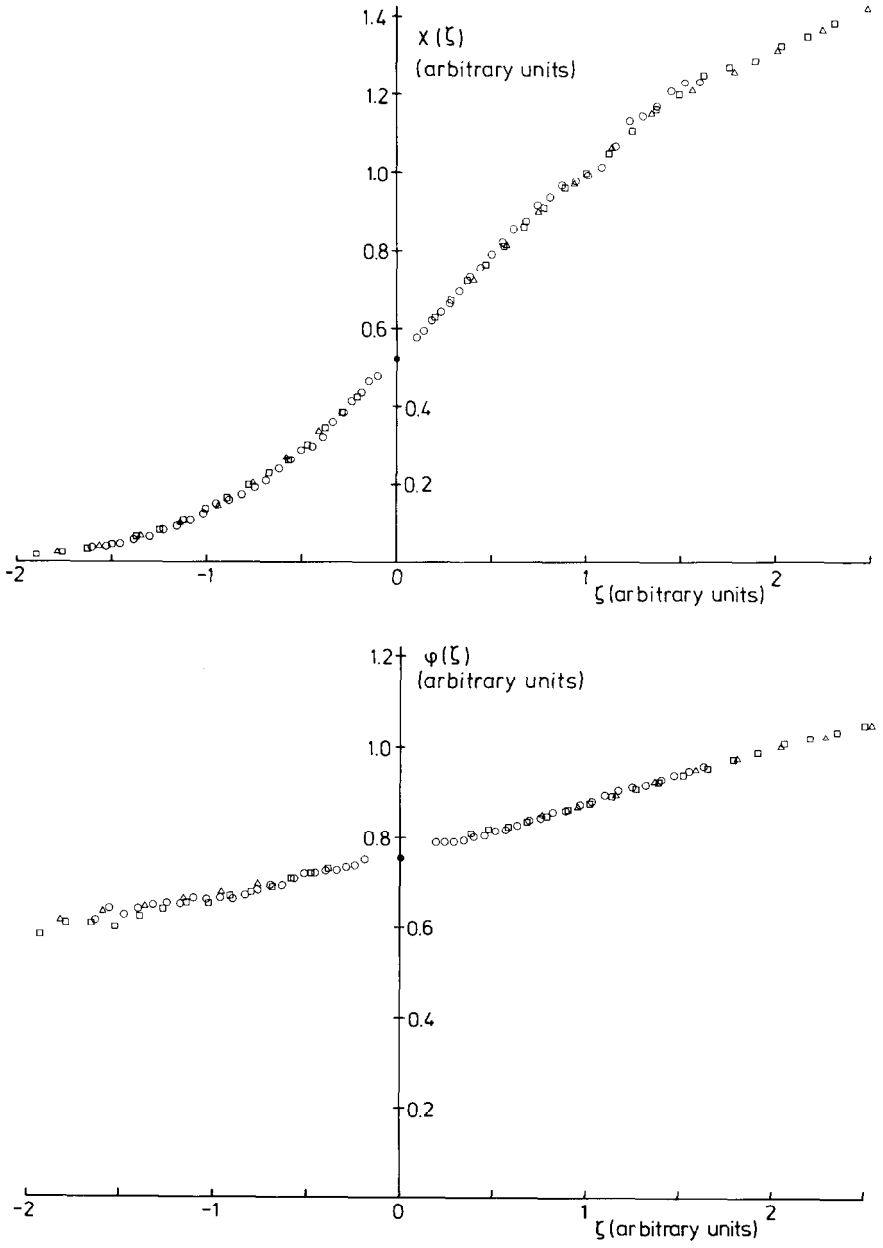

FIG. 4. Values of $t^{(\beta+\tau-\nu)/\tau}\Sigma_{n_1} Y(n_1, t; \sigma)$ (Figure 4a) resp. of $t^{-\nu/\tau}\langle n_1 \rangle$ (Figure 4b), versus $\zeta = \varepsilon t^{1/\tau}$. ●: $\varepsilon = 0$ (200 runs). ○: $\varepsilon = \pm 2^{-5}$ (100 runs each). □: $\varepsilon = \pm 2^{-4}$ (-''-). △: $\varepsilon = \pm 2^{-3}$ (100 resp. 500 runs). In Figure 4a (4b) only results for $t \geqslant 6$ ($\geqslant 15$) are shown.

smooth at $\zeta = 0$. Using instead of $\nu = \frac{4}{3}$ the alternative value $\nu = 1.3547\ldots$ proposed by Klein et al. [12], we found marginally worse agreement.

6. DISCUSSION

The reason for making such extensive Monte Carlo calculations of critical behavior is of course the hope that the results might be applied to more realistic models, without any essential modification. This hope is not founded on rigorous mathematical arguments, but on vast experience with other critical phenomena [3].

Critical phenomena arise typically when fluctuations are strong and have a coherence length (and time) much larger than any microscopic length (or time) of the model. The lack of "appropriate" scales then leads to the renormalization (semi-)group, which then implies the scaling laws. Thus one expects all models to have identical critical behavior (i.e. the same critical exponents and scaling functions), which can be distinguished only by "microscopic" details. In our case, this means that the scaling laws of Section 3 and the values of the exponents should not be altered when making e.g. the following modifications:

(1) altering the markoffian nature of the process by introducing e.g. latency periods or age-dependent infection or recovery rates—provided the additional time scales introduced thereby are finite;

(2) replacing the cartesian lattice by a different regular 2-dimensional lattice [4], or even by the continuous [13] space R^2 [in the latter case, Equation (2.1) must be replaced by

$$X(\mathbf{a}) + Y(\mathbf{b}) \overset{\sigma V(|\mathbf{a}-\mathbf{b}|)}{\rightarrow} Y(\mathbf{a}) + Y(\mathbf{b}) \tag{6.1}$$

with a "contact distribution" $V(|\mathbf{a}|)$ which has short range];

(3) distributing the susceptibles at $t = 0$ not in a regular but in a random fashion;

(4) introducing e.g. carriers which transmit the infection, provided they can travel only finite distances and live only for finite times.

A somewhat more subtle question is whether one can allow also for mobility of the individua. Clearly, if the immunes can move around, laws like Equations (3.2) and (3.4) describing their spatial distribution at $t \to \infty$ become inapplicable. Nevertheless, the time-dependent scaling laws for the infected population obtained from (3.7) by differentiation should still hold, if the motion is diffusive with finite diffusion constant. The reason is that close to the critical point Equation (3.7) predicts a typical spread $\langle |\mathbf{n}| \rangle \propto t^{\nu/\tau} \propto t^{0.9}$, while the diffusive spread is only $\langle |\mathbf{n}| \rangle \propto \sqrt{t}$.

A feature which does influence the critical behavior is the dimensionality. Thus, the critical exponents in 3 dimensions are different from those above. Also, different behavior would be obtained if we allowed the infection only to spread in a certain angular region (directed percolation). The same holds if we either neglect recovery altogether, or allow for recovery but assume that the recovered individua are not immune. The latter case is in the same universality class as directed percolation in $d+1$ dimensions. Also in this latter class are both Schlögl models [14], reggeon field theory [7,15], and the basic contact model [16], which is isomorphic to the reggeon spin model of Reference [17] (as can seen from the stochastic reinterpretation of the reggeon spin model [7]).

Even if the scaling laws of Section 3 do apply asymptotically, one has still to find the domain (in space, time, and the control parameters) where they represent good approximations. Certainly, this can only be the case if the lengths and times considered are much larger respectively than the inherent length and time scales of the model. This is however not sufficient. In a second-order phase transition, one has the so-called Ginzburg criterion [3] which determines the critical domain. For the GEP, the analogous criterion is not yet known.

For epidemics in human populations, it seems hard to realize the situation where the inherent length scales become small. The situation may be much more favorable for spreading of plant parasites, e.g. in large-scale monocultures. Even better candidates for the observation of critical behavior are the spread of bacteriophages on cultures of killed bacteria, or chemical waves [2], in particular in reactions involving large molecules in order to suppress diffusion.

I am much indebted to Dr. D. Stauffer for bringing Reference [5] to my attention, and for carefully reading the manuscript. To him and to Drs. J. Kertesz, J. Boissonade, and H. J. Herrmann, I am also indebted for a long and very instructive discussion.

REFERENCES

1 D. Mollison, *J. Roy. Statist. Soc. Ser. B* 39:283 (1977); N. T. J. Bailey, *The Mathematical Theory of Infectious Diseases*, Griffin, London, 1975.

2 B. Hess and A. Boiteux, *Ber. Bunsen Ges.* 84:346, 392 (1980); A. Hanna and K. Showalter, talk at conference on "nonlinear phenomena in chemical dynamics," Bordeaux, Sept. 1981.

3 H. E. Stanley, *Introduction to Phase Transitions and Critical Phenomena*, Oxford U.P., London, 1971 (2nd ed. in preparation); C. Domb and M. S. Green (Eds.), *Phase Transitions and Critical Phenomena*, Vols. 1–6, Academic, London, 1972–.

4 J. W. Essam, *Rep. Progr. Phys.* 43:833 (1980); D. Stauffer, *Phys. Rep.* 54C:1 (1979).

5 Z. Alexandrowicz, *Phys. Lett.* 80A:284 (1980).

6 M. P. M. den Nijs, *J. Phys. A* 12:1857 (1979); H. W. J. Blöte et al., *J. Phys. A* 14:L45 (1981); P. D. Eschbach et al., *Phys. Rev. B* 23:422 (1981).
7 P. Grassberger and A. de la Torre, *Ann. Physics* 122:373 (1979).
8 J. Cardy and R. L. Sugar, *J. Phys. A* 13:L423 (1980).
9 D. G. Kendall, *J. Roy. Statist. Soc. Ser. A* 120:64 (1957).
10 H. Kesten, *Comm. Math. Phys.* 74:41 (1980).
11 L. Jacobs, M. Creutz, and C. Rebbi, *Phys. Rev. Lett.* 42:1390 (1979); R. Zorn, H. J. Herrmann, and C. Rebbi, *Comput. Phys. Comm.* 23:337 (1981).
12 W. Klein et al., *Phys. Rev. Lett.* 41:1145 (1978).
13 E. T. Gawlinski and H. E. Stanley, *J. Phys. A* 14:L291 (1981).
14 F. Schlögl, *Z. Phys.* 253:147 (1972); P. Grassberger, *Phys. Lett.* 84A:459 (1981).
15 M. Moshe, *Phys. Rep.* 37C:255 (1978); P. Grassberger and K. Sundermeyer, *Phys. Lett.* 77B:220 (1978).
16 D. Griffeath, *Springer Lecture Notes in Mathematics*, Vol. 724, 1979.
17 D. Amati et al., *Nuclear Phys. B* 112:107 (1976); 114:483 (1976); R. C. Brower et al., *Phys. Rev. D* 15:1756 (1977).