Percolation with long-range correlations for epidemic spreading

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A percolation model with long-range correlations was introduced to investigate the phenomena of epidemic spreading by Monte Carlo simulations. The correlation exponent α and pathogenic ratio s correspond to different spreading methods and pathogenicity of variant epidemics. As the correlation changes from a weak one to a strong one, the patterns change from site percolation to Eden cluster when pathogenic ratio $s=1$, or Leath percolation cluster when $s<1$. Corresponding to change of patterns, the fractal dimension increases up to space dimension. The critical behavior in epidemic spreading has been examined based on the model. It is found that correlation has a great influence on the threshold of spreading percolation.

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I. INTRODUCTION

During the last two decades, fractal growth and aggregation phenomena have attracted considerable interest [1–3]. The fractal structure of aggregates strongly depends on the dynamics of the growth process. Many experiments, theoretic analysis, and computer simulations have been carried out to investigate the relationships between the geometry and mechanism. Great efforts have been directed to developing models for fractal growth and the aggregation process. There are three basic models of fractal growth: particle-cluster aggregation (PCA) [4,5], cluster-cluster aggregation (CCA) [6,7], and percolation [3]. Almost five decades ago, the concept of percolation was introduced to study the gelation process and the spreading of fluids in random media [3]. Then, many investigations were performed on the percolation model and its applications [8–11]. The percolation model is useful for describing some physical, chemical, and biological processes, such as the spreading of epidemics and forest fires, the gelation process, the invasion of water into oil in porous media, and other related problems [12–17].

Eden presented a model for the spreading of bacterial colonies on a homogeneous substrate that was represented by a lattice of sites [18,19]. In each growth step, one of the open sites at the perimeter of the cluster is chosen randomly and occupied. Eden cluster is compact and has a self-affine front [1]. Once the occupied probability is less than 1, Eden model changes into Leath percolation [3], which has been used to study epidemics, forest fire spreading, and similar phenomena [3,8,14,17].

Bunde *et al.* have introduced a spreading percolation in which epidemics spread by ''butterfly'' [14]. The model is interesting for the spatial correlation that is involved. But the correlation is limited for the ''butterfly'' and occupation can only occur at the sites nearest to those already in cluster, i.e., the cluster can only grow ''shell by shell.'' Recently, Makse *et al.* presented a correlated percolation to model urban growth [15]. Their results agreed well with the population distribution of Berlin in different periods. Very recently, Moore and Newman studied the epidemics in small-world networks by a percolation model [17]. Their study shows, that epidemics' behavior is strongly influenced by the networks.

In this paper, we introduced a percolation with long-range correlation to study epidemic spreading. In the model, spreading phenomena can occur through spatial distance, i.e., epidemics can spread to the sites that are not adjacent to the cluster of sick individuals. Our results will be useful to describe and understand the behavior of variant epidemic spreading.

II. MODEL AND SIMULATIONS

Monte Carlo (MC) simulations have been used for the present model. In simulations, we consider a square lattice. Each site can be empty or occupied by two types of particles, which are called sick (S) and immune (I) ones. At time $t=1$, an S particle is placed at the origin. An empty site (healthy particle) in the square lattice can be chosen randomly and converted into an S particle with probability sP, or an I particle with probability $(1-s)P$. Here, s is the pathogenic ratio that stands for the pathogenicity of viruses or germs of epidemics. P is the infected probability, which can be deduced as follows. In the percolation model with long-range correlations, the correlation probability p_i, with which a healthy particle is infected by the ith S particle, is expressed as

$$p_i = 1/r_i^{\alpha}, \tag{1}$$

where r_i is the distance from the empty site to ith S site and $\alpha(\alpha \geqslant 0)$ is the correlation exponent that relates to the ways of viruses or germs spreading. For the chosen empty site, the infected probability by the ith S particle is p_i, so the uninfected probability is $1-p_i$. Furthermore, considering the effects of all the S particles on a chosen particle, the total infected probability P takes the form

$$P = p_1 + (1-p_1)p_2 + (1-p_1)(1-p_2)p_3 + \cdots + (1-p_1) \times (1-p_2)\ldots(1-p_{N-1})p_N = 1 - \prod_{i=1}^{N}(1-p_i), \tag{2}$$

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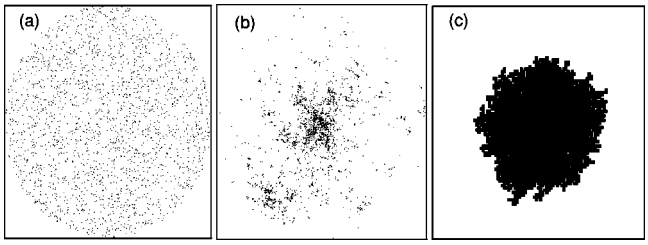

FIG. 1. Distribution of S particles for the percolation model with long-range correlations. $N=2500$, $s=1$, $\alpha=1.5$ (a), 2.5 (b), and 1000 (c). $L=500$ (a), 500 (b), and 100 (c).

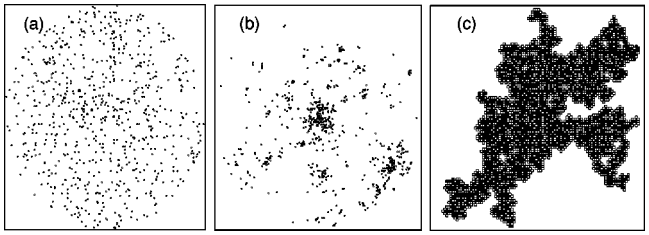

FIG. 2. Distribution of S and I particles for the percolation model with long-range correlations. Full square, S particles; open square, I particles. $s=p_c$, $\alpha=1.5$, (a) 2.5 (b), and 1000 (c). $L=200$ (a), 200 (b), and 100 (c).

where N is the number of S particles in existence. Choose an empty site, calculate its infected probability P, and convert it to an S with probability P or I particle with probability $1-P$. Repeat this process until the expected growth scale is attained or the epidemics cannot continue to spread.

The pathogenic ratio s and correlation exponent α can describe variety of types of epidemic spreading. There are two limits in the model: One is weak correlation limit, i.e., $\alpha=0$. In this case, this model degenerates to the site percolation in which the occupied fraction corresponds to the occupied probability [3]. The other one is strong correlation limit, i.e., $\alpha\rightarrow\infty$. In this limit case, the Eden cluster can be obtained with $s=1$, or the Leath percolation cluster with $s<1$.

III. RESULTS AND DISCUSSION

Numerical simulations are performed on a finite system of area of $L\times L$. The length of square particles is chosen to be the unit of length. The occupied fraction is given by $\phi=N/L^2$. In the simulation, the attention is focused on the S particles cluster, i.e., the distribution of S particles. L is chosen to be two values: 200 and 500. The results are almost the same. ϕ is taken as 0.01 in the simulations.

A. Morphology

The morphologies of the numerical simulations vary with the parameters s and α. Figure 1 shows the simulation patterns of epidemic spreading with the pathogenic ratio $s=1$ for several correlation exponents α. In the case of the weakly correlated percolation i.e., α is small, all S particles prefer to be dispersed randomly on the 2D world [see Fig. 1(a)]. It is a site percolation in which occupied the fraction corresponds to the occupied probability [3]. As α increases, the correlation between particles gets strong and the particles are not dispersed randomly any more. They are apt to form a dense pattern at some localities [as shown in Fig. 1(b)]. If α is large enough, the correlation becomes very strong. In this case, we get $P=1$ (when $r=1$) and 0 (when $r>1$) from Eqs. (1) and (2). It means that the S particles can only grow on the nearest sites to the cluster of S particles, which is just the growth rule of the Eden model [1,2,19,20] Therefore, the cluster is a very compact one and has a self-affine front [1] that is a reproduction of the Eden model [see Fig. 1(c)]. From the above, we generalize a conclusion. For the kind of epidemics that are very easy to spread (e.g., spread by winds) the disease can be transmitted through long distance, and the distribution of sick individuals is dispersive, which corresponds to the weakly correlated case. But for other kinds of epidemics that are mainly transmitted by contact (e.g., spread by body fluid, i.e., through sweat, food, sexual contact, and so on), the diseases can only spread through a very short distance from an S site to an empty one, which is relevant to the strongly correlated case. In this case, the sick particles are distributed thickly.

Going one step further, we investigated the influence of pathogenic ratio s on the distribution of sick and immune particles. Figure 2 gives the patterns of simulations with $s=p_c$ for variant α. p_c is the threshold of 2D percolation on the square lattice and has the value of 0.593 [3]. For small α, the system presents a weakly correlated percolation. In this case, S and I particles randomly lie on the 2D lattice [see Fig. 2(a)], and the distribution of S particles is similar to that in Fig. 1(a). The cluster consisting of S and I particles becomes denser as the correlation exponent α rises. The simulation with $s=p_c$ and $\alpha=2.5$ is shown in Fig. 2(b). The cluster of S particles in Fig. 2(b) is a little sparser than that in Fig. 1(b) ($s=1$ and $\alpha=2.5$). Figure 2(c) presents the pattern with $s=p_c$ and $\alpha=1000$. Comparing with Fig. 1(c) ($s=1$ and $\alpha=1000$), the cluster is sparser. In this case, an empty nearest site to the cluster of S particles is randomly chosen and is converted to an S particle with probability s or I one with $(1-s)$, which is the Leath method of generating single percolation [3]. Such a cluster is just the Leath percolation, which also has a threshold probability p_c. If $s<p_c$, the Leath method can only produce a finite single percolation cluster [3]. For the strong correlation limit, new S or I particles grow on the edge of the cluster of S particles with $P=1$, and the converted probabilities to S and I particles are s and $1-s$, respectively. It follows from this, if $s<p_c$, the epidemics cannot spread infinitely for the very strong correlation, i.e., the cluster of S particles is finite in scale. It can be seen that the pathogenic ratio s plays an important role in the spreading of diseases.

B. Fractal dimension

The static (geometric) property is expressed by fractal dimension D_f. The fractal dimension of a spreading percolation system can be calculated by the box-counting method that was used to study random sets in a box by Hamburger *et al.* recently [20]. It was found, for a low occupied fraction, apparent fractal behavior was observed between physically relevant cutoffs. The lower cutoff r_0 is presented by the length of particles. The upper cutoff r_1 is given by the aver-

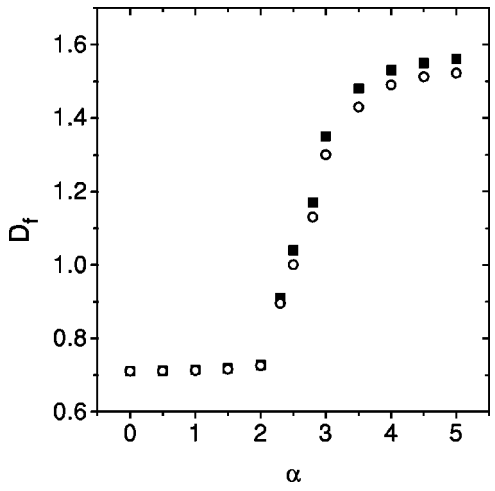

FIG. 3. The fractal dimension D_f of the cluster consisting of S particles as functions of the correlation exponent α for spreading percolation. Pathogenic ratio $s=1$ (full square), and 0.593 (open circle).

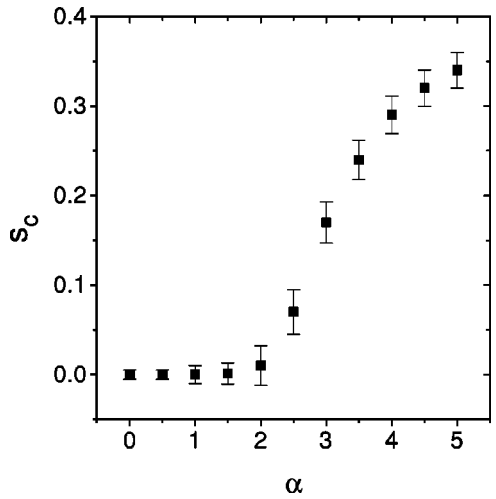

FIG. 4. The threshold s_c of pathogenic ratio *versus* the correlation exponent α for spreading percolation. $s_c \approx 0.593$ when $\alpha = 1000$.

age gap between adjacent particles [20]. The calculations below were completed by the box-counting method.

Figure 3 plots the fractal dimension D_f of the cluster consisting of S particles as functions of the correlation exponent α for pathogenic ratio $s=1$ and 0.593, respectively. It is found that, with α rising from 0 to infinite, D_f increases from 0.72 to 1.93, and a sharp step appears at $\alpha \approx 2$. We will show below that the behavior of D_f with α is reasonable. First, we check the case of the large α. When $\alpha \to \infty$, which corresponds to the limit of strong correlation, if $s=1$, our model reduces to the much-studied Eden model in which D_f is close to 2 (2 is the space dimension) [1,2,19]; if $s=p_c=0.593$, our model reduces to the Leath percolation on the threshold p_c in which $D_f \approx 91/48$ [3,14]. These results are in good agreement with our simulation results (1.93 and 1.88). Then we discuss the case of small α. When $\alpha=0$, our model reduces to the site percolation whose fractal dimension is related to occupied fraction ϕ [3,20]. When ϕ is small, D_f is small too. The simulation value (0.72) is reasonable.

C. Critical behavior in epidemic spreading

As described in Sec. III A, in the case of the strong correlation limit ($\alpha \to 0$), if pathogenic ratio $s < p_c$, the epidemics cannot spread infinitely, i.e., it is the critical phenomena and the threshold $s_c = p_c$. Does threshold s_c exist for other cases of nonzero variant correlation exponent α?

To answer this question, we consider the epidemic spreading over the range $0 \leqslant \alpha < \infty$. It is supposed that, if the growth time Δt of an S particle is longer than $20\overline{\Delta t}$ ($\overline{\Delta t}$ is the mean growth time of previous S particles), the disease cannot spread any longer. The supposition is based on the fact that the life-span of viruses and germs are finite. To check the effect of Δt on s_c, two simulations with different Δt ($=20\overline{\Delta t}$ and $40\overline{\Delta t}$) have been done. Obtained values of s_c are almost the same. This shows that the supposition is reasonable.

Based on this assumption, the numerical simulations were performed and the results are shown in Fig. 4. It is found that, when $\alpha < 2$, s_c is close to 0, and when α is very large s_c becomes close to p_c. There is a sharp step in the $s_c - \alpha$ curve too. The relation of s_c to α is easy to understand. For the limit of strong correlation, our model reduces to Leath percolation that has a threshold p_c [3]. If $s < p_c$, the cluster of S particles can only grow to a finite size. Thus, in this case, $s_c \to p_c$. For the weak correlation limit, the S particles can be randomly transmitted through a very long distance. It is very difficult to prevent the epidemics from spreading, and we can expect that $s_c \to 0$. When α changes from 0 to a large value, s_c increases from 0 to p_c. These expectations agree well with the results of our simulations. From the above, for different epidemics, the threshold differs greatly for the variant spreading ways. For the diseases spreading by touch (strong correlation), the threshold is large and it is not too difficult to control the disease spreading. But for the diseases that can be transmitted through long distances (weak correlation), the spreading phenomena is very difficult to control.

It appears that in Figs. 3 and 4, there is a sharp step at the point $\alpha_c \approx 2$ for $D_f - \alpha$ and $s_c - \alpha$ curves. α_c is the transition value, which can be deduced using a simple analysis. We consider the growth probability P_d of S particles at the sites whose distance from the nearest S particle are larger than a certain value d. Then P_d can be given by

$$P_d = \sum_{r=d}^{\infty} sp(r) \Big/ \sum_{r=1}^{\infty} sp(r). \tag{3}$$

Using integrals instead of summation approximately, Eq. (3) becomes $P_d = (r^{2-\alpha}|_d^\infty)/(r^{2-\alpha}|_1^\infty)$. Thus, as a rough estimate, $P_d \approx 1$ for the range $\alpha < 2$ and $P_d \approx d^{2-\alpha}$ for the range $\alpha > 2$. For the case $\alpha = 2$, $P_d = \lim_{\alpha \to 2} r^{2-\alpha}|_d^\infty / r^{2-\alpha}|_1^\infty = 1$. $\alpha_c = 2$ is the transition point from a random dispersed distribution to a compact one of particles. The distribution of particles is reflected by the fractal dimension. So the sharp step appears at $\alpha \approx 2$ in the $D_f - \alpha$ curve. Analogously, we can explain the behavior of s_c in Fig. 4. When $\alpha < 2$, the system belongs to the universality class of a random graph with the constant probability of a given pair of sites being connected. In this case, the S particles are dispersed everywhere and the S particles continue to produce new S particles. Thus the

system grows infinitely and $s_c=0$. When $\alpha>2$, we explain the same universality class at the square lattice, since most connections are local. The model can also be performed in a d dimension lattice. Similar results can be obtained. More generally, $\alpha_c=d$.

IV. CONCLUSION

In this paper, a percolation model with long-range correlations was introduced to investigate the epidemic spreading by Monte Carlo simulations. Our model gave a variety of patterns that can help us to describe and understand how different diseases spread. The critical behavior in spreading phenomena was discussed based on the model. It may be useful to control epidemic spreading and describe similar spreading phenomena.

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