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Critical behaviour of a probabilistic automata network SIS model for the spread of an infectious disease in a population of moving individuals

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Abstract. A probabilistic automata network SIS model for the spread of an infectious disease in a population of moving individuals is studied. The local rule consists of two subrules. The first one, applied synchronously, models infection and recovery. It is a probabilistic cellular automaton rule. The second, applied sequentially, describes the motion of the individuals. The model contains three parameters, the probabilities p_i to get infected and p_r to recover, and the average number of tentative moves per individual m. Depending upon the values of these parameters, in the infinite-time limit, the system is either in the disease-free state or in the endemic state. It goes from one state to the other through a transcritical bifurcation similar to a second-order phase transition characterized by a non-negative order parameter, whose role is played, in this model, by the stationary density of infected individuals. The (p_i, p_r) phase diagram and the critical behaviour of the stationary density of infectives in the neighbourhood of the phase transition, are studied as a function of m. According to whether the individuals perform short- or long-range moves, it is found that the parameters characterizing the transition have a qualitatively different behaviour as m varies. When m is very large, the correlations created by the application of the subrule modelling infection and recovery are destroyed, and, as expected, the behaviour of the system is then correctly predicted by a mean-field-type approximation which assumes a homogeneous mixing of the individuals. When m is not large, this assumption is no longer correct.

1. Introduction

This paper discusses a probabilistic automata network SIS model, i.e. a model in which, after recovery, infected individuals (I) become susceptibles (S) again to catch the disease (as, e.g., with the common cold). This model exhibits a transcritical bifurcation between a endemic state and a disease-free state. The emphasis is on the influence of motion of the individuals on the critical behaviour of the model in the neighbourhood of the bifurcation point.

In models formulated in terms of differential equations, the motion of the individuals is usually taken into account by incorporating a diffusion term in the evolution equation. A typical example is Murray's model for the spatial spread of rabies among foxes in Europe (Källén *et al* 1985, Murray *et al* 1986). These models, which have unquestionably contributed to our understanding of the spread of an infectious disease, do not, however, take into account correctly the short-range character of the infection process. This is manifest when the system exhibits bifurcations. In phase-transition theory it is well known that in the vicinity of a bifurcation point—i.e. a second-order transition point—certain physical

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quantities exhibit a singular behaviour (Boccara 1976). It is only above a certain spatial dimensionality—the upper critical dimensionality—that the behaviour of the system may be correctly described by a partial differential equation.

One way to take the short-range character of the infection process into account correctly is to discretize space, and to represent the spread of an epidemic as the growth of a random cluster on a lattice if, after recovery, infected individuals become permanently immune (Grassberger 1983, Cardy and Grassberger 1985). This model exhibits non-trivial critical behaviour. It is in the same universality class as percolation cluster growth models. Its upper critical dimensionality is equal to 6 (Cardy 1983).

The relationship of the spatial spread of an epidemic to the percolation process was first noticed by Mollison (1977), and after the publication of his paper, and the introduction of random graphs—which are graphs with randomly coloured edges—by Gertsbakh (1977), several papers have appeared in the mathematical literature on the so-called spatial general epidemic model (see, e.g., Kuulasmaa 1982, Kuulasmaa and Zachary 1984, Cox and Durrett 1988).

All these models, however, neglect the motion of the individuals which, for the general epidemic model, has been found to be an important factor (Boccara and Cheong 1992).

2. Description of the model

In an SIS model, based on disease status, the individuals are divided into two disjoint groups:

(S) the *susceptible* group, i.e. those individuals who are not infected but who are capable of contracting the disease and become infective; and

(I) the *infective* group, i.e. those individuals who are capable of transmitting the disease to susceptibles.

The model is formulated in terms of automata networks (Goles and Martínez 1990). Automata networks are discrete dynamical systems in time and space. They may be defined as follows.

Let $G = (V, E)$ be a graph, where V is a set of vertices and E a set of edges. Each edge joins two vertices not necessarily distinct. An automata network, defined on V, is a triple $(G, Q, \{f_i | i \in V\})$, where G is a graph on V, Q a finite set of states and $f_i: Q^{|U_i|} \to Q$ a mapping, called the local transition rule associated to vertex i. $U_i = \{j \in V | \{j, i\} \in E\}$ is the neighbourhood of i, i.e. the set of vertices connected to i, and $|U_i|$ denotes the number of vertices belonging to U_i. The graph G is assumed to be locally finite, i.e. for all $i \in V$, $|U_i| < \infty$.

In our model the set V is the two-dimensional torus Z_L^2, where Z_L is the set of integers modulo L. The neighbourhood of a given vertex (x, y) is the set of four vertices $\{(x \pm e_x, y \pm e_y) \mid e_x = 1, e_y = 1\}$. A vertex is either empty or occupied by an individual belonging to one of the two groups. The spread of the disease is governed by the following rules:

(i) Susceptibles become infective by contact, i.e. a susceptible may become infective with a probability p_i if, and only if, it is in the neighbourhood of an infective. More precisely, during one time step, the probability that a susceptible having z infected neighbours becomes infected is $(1 - (1 - p_i)^z)$. This hypothesis neglects incubation and latent periods, i.e. an infected susceptible becomes immediately infective.

(ii) Infectives recover and become susceptible again with a probability p_r. That is, at each time step, an infected individual either recovers with probability p_r or remains infected with probability $1 - p_r$. The number of time steps T during which he remains infected is a

random variable with a geometric distribution, i.e. the probability $P(T = k)$ that T is equal to the positive integer k is equal to $p_r(1 - p_r)^{k-1}$, and we have

$$E(T) = 1/p_r \qquad \mathrm{Var}(T) = (1 - p_r)/p_r^2$$

where, as usual, $E(T)$ and $\mathrm{Var}(T)$ denote, respectively, the mean and the variance of T. This assumption states that recovery is equally likely among infectives, it does not take into account the length of time the individual has been infective.

(iii) The time unit is the time step. In practical applications, the choice of the unit of time is related to the probabilities p_i and p_r. During one time step, the two preceding rules are applied synchronously, and the individuals move on the lattice according to a specific rule.

(iv) An individual selected at random may move to a vertex also chosen at random. If the chosen vertex is empty the individual will move, otherwise the individual will not move. The set in which the vertex is randomly chosen depends on the range of the move. To illustrate the importance of this range, we considered two extreme cases. The chosen vertex may either be one of the four neighbours or be any vertex of the graph. These two particular types of move will be called, respectively, *short-* and *long-range* moves. If N is the total number of individuals on Z_L^2, mN individuals, where m is a positive real number, are sequentially selected at random to perform a move. Since N is large (10^3–10^4), mN can still be an integer if m is small (10^{-2}). This sequential process allows some individuals to move more than others. In particular, the probability that s given individuals are not selected to perform a move is $(1 - s/N)^{mN}$, which tends to e^{-sm} as N tends to ∞. Since an individual may only move to an empty site, the parameter m represents the average number of *tentative* moves per individual during a unit of time.

This model is rather crude. Its purpose is to help understanding of the importance of motion. It assumes that the population is closed. It ignores births, deaths by other causes, immigrations or emigrations. It is a probabilistic automata network with a mixed transition rule. That is, at each time step, the evolution results from the application of two subrules. The first subrule determines which susceptibles become infectives and which infectives recover. It is a probabilistic three-state cellular automaton rule. It is applied *synchronously*. The second one specifies the motion of the individuals. It is applied *sequentially*. Both subrules are translation invariant, i.e. they do not depend upon the vertex (x, y).

3. Mean-field approximation

The mean-field approximation ignores space dependence and neglects correlations. It is, therefore, equivalent to the assumption of homogeneous mixing, which is considered a questionable assumption in epidemic modelling (Anderson and May 1991). In the case of a physical system exhibiting a phase transition the quantitative predictions of a mean-field approximation are not very good. However, for the SIS model described in the preceding section, since the second subrule represents a process that destroys the correlations created by the first subrule, in the limit $m \to \infty$, the mean-field approximation becomes exact.

If the densities of the different groups of individuals are not space dependent, the state of the system at time t is characterized by the densities $S_{MFA}(t)$ and $I_{MFA}(t)$ of susceptibles and infectives, and the evolution equation of the density of infectives is

$$I_{MFA}(t+1) = I_{MFA}(t) + S_{MFA}(t)(1 - (1 - p_i I_{MFA}(t))^z) - p_r I_{MFA}(t) \qquad (1)$$

where z is the number of neighbouring vertices of a given vertex. For the two-dimensional square lattice considered in our simulations, $z = 4$. Note that, within the framework of this approximation, the incidence rate, i.e. the rate of new infection, represented by the term $S_{\mathrm{MFA}}(t)(1 - (1 - p_{\mathrm{i}} I_{\mathrm{MFA}}(t))^z)$, is not bilinear as in most models (Bailey 1975, Waltham 1974, Anderson and May 1991). Nonlinear incidence rates have recently been shown to exhibit very different dynamic behaviours (Hethcote and van den Driessche 1991).

Since the population is closed, the total density

$$C = S_{\mathrm{MFA}}(t) + I_{\mathrm{MFA}}(t) \tag{2}$$

is time independent. Eliminating $S_{\mathrm{MFA}}(t)$ between (1) and (2) yields

$$I_{\mathrm{MFA}}(t+1) = I_{\mathrm{MFA}}(t) + (C - I_{\mathrm{MFA}}(t))(1 - (1 - p_{\mathrm{i}} I_{\mathrm{MFA}}(t))^z) - p_{\mathrm{r}} I_{\mathrm{MFA}}(t). \tag{3}$$

In the infinite-time limit, the stationary density of infectives $I_{\mathrm{MFA}}(\infty)$ is such that

$$I_{\mathrm{MFA}}(\infty) = (1 - p_{\mathrm{r}}) I_{\mathrm{MFA}}(\infty) + (C - I_{\mathrm{MFA}}(\infty))(1 - (1 - p_{\mathrm{i}} I_{\mathrm{MFA}}(\infty))^z). \tag{4}$$

$I_{\mathrm{MFA}}(\infty) = 0$ is always a solution of equation (4). This value characterizes the *disease-free state*. It is a stable stationary state if, and only if, $zCp_{\mathrm{i}} - p_{\mathrm{r}} \leqslant 0$. If $zCp_{\mathrm{i}} - p_{\mathrm{r}} > 0$, the stable stationary state is given by the unique positive solution of equation (4). In this case, a non-zero fraction of the population is infected. The system is in the *endemic state*. For $zCp_{\mathrm{i}} - p_{\mathrm{r}} = 0$ the system, within the framework of the mean-field approximation, undergoes a transcritical bifurcation similar to a second-order phase transition characterized by a non-negative order parameter, whose role is played, in this model, by the stationary density of infected individuals $I_{\mathrm{MFA}}(\infty)$. This threshold theorem is a well known result for differential equation SIS models (Hethcote 1976).

It is easy to verify that, in the endemic state, when $zCp_{\mathrm{i}} - p_{\mathrm{r}}$ tends to zero from above, $I_{\mathrm{MFA}}(\infty)$ goes continuously to zero as $zCp_{\mathrm{i}} - p_{\mathrm{r}}$. In the $(p_{\mathrm{i}}, p_{\mathrm{r}})$ parameter plane,

$$zCp_{\mathrm{i}} - p_{\mathrm{r}} = 0 \tag{5}$$

is the equation of the second-order phase transition line.

In phase-transition theory, it is well known that constant interaction models exhibit a mean-field behaviour. In the appendix a constant interaction version of this SIS model is presented.

4. Simulations

In all our simulations, the total density of individuals $C = 0.6$, a value slightly greater than the site percolation threshold for the square lattice, which is equal to 0.593 (Stauffer 1979), in order to be able to observe cooperative effects when $m = 0$. Except to check possible size effects, all our simulations have been performed on a 100×100 lattice. All our results are averages over 10 measurements.

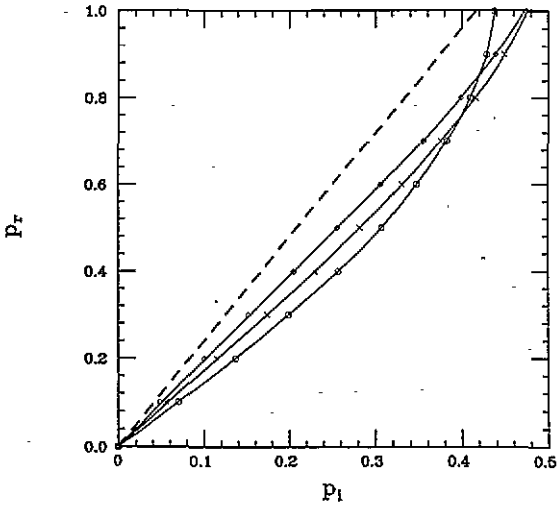

Figure 1. (p_i, p_r) phase diagram in the case of short-range moves for $m = 0$ (●), $m = 2$ (×), $m = 8$ (◇). The broken curve represents the mean-field approximation. Total density $C = 0.6$; lattice size, 100×100.

4.1. Short-range moves

Figure 1 represents the (p_i, p_r) phase diagram for different values of m in the case of short-range moves. Figure 2 shows a typical variation of the stationary density of infectives $I(m, \infty)$ as a function of p_i for given values of p_r and m. The slope at the critical point (i.e. the transcritical bifurcation point) seems to be infinite. If this is indeed the case, the critical exponent β defined by

$$\beta = \lim_{p_i - p_i^c \to 0^+} \frac{\log I(m, \infty)}{\log(p_i - p_i^c)} \tag{6}$$

which is equal to 1 within the mean-field approximation, is less than 1. Figure 3 shows a log–log plot of $I(m, \infty)$ as a function of $p_i - p_i^c$, where p_i^c is the critical value of p_i, for $p_r = 0.5$ and $m = 0.3$. It is found that $p_i^c = 0.302$ and $\beta = 0.6$. To check size effects, we have performed some measurements on a 200×200 lattice. The fluctuations, as expected, are reduced by a factor 2 but the mean values of the density of infectives is not modified.

It has been clearly established that the mean-field approximation, because it neglects correlations which play an essential role in the neighbourhood of a second-order phase transition, cannot predict correctly the critical behaviour of short-range interaction systems (Boccara 1976). For standard probabilistic cellular automata, this is also the case (Bidaux *et al* 1989, Martins *et al* 1991).

For a given value of p_r, the variations of β and p_i^c as functions of m (figure 4) exhibit two regimes reminiscent of crossover phenomena found phase-transition theory (Boccara 1976). In the small-m regime, i.e. for $m \lesssim 10$, p_i^c and particularly β have their $m = 0$ values. In the large-m regime, i.e. for $m \gtrsim 300$, p_i^c and β have their mean-field values. To check size effects on the crossover values, Boccara *et al* (1993) have studied the influence of m on the critical behaviour of a one-dimensional probabilistic elementary cellular automaton (diluted Rule 18). No effect has been found. As shown in figure 5, the exponent β does not seem to depend upon p_r in agreement with what is known from phase-transition theory: the value of a critical exponent does not change along a second-order transition line. The variation of p_i^c for $p_r = 1$, shown in figure 5, exhibits an interesting re-entrant effect. That is, there is a range of values for p_i such that, for small and large values of m, the system is in the endemic phase, whereas it is in the disease-free state for intermediate values of m.

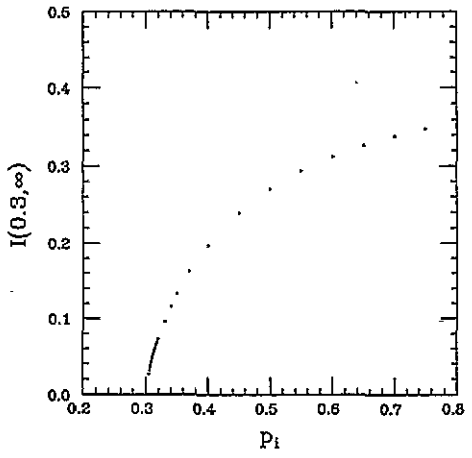

Figure 2. Typical variation of $I(m, \infty)$ as a function of p_i for given values of p_r and m in the case of short-range moves. Here $p_r = 0.5$, $m = 0.3$. The critical value of p_i is 0.3018. Total density $C = 0.6$; lattice size, 100×100.

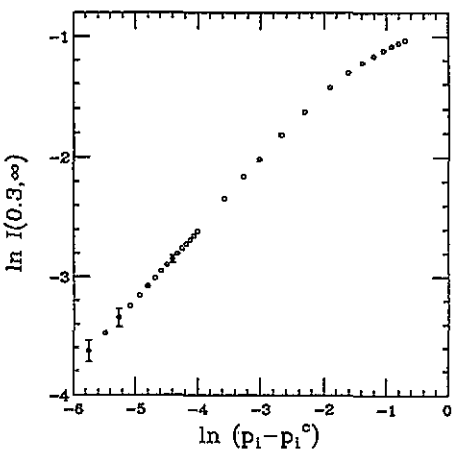

Figure 3. Typical log–log plot of $I(m, \infty)$ as a function of $p_i - p_i^c$ for given values of p_r and m in the case of short-range moves. Here $p_r = 0.5$, $m = 0.3$. The critical value of p_i is 0.3018. Typical bars are represented. Total density $C = 0.6$; lattice size, 100×100.

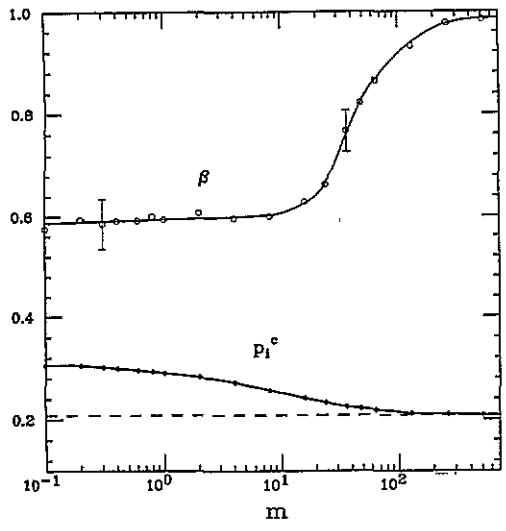

Figure 4. Variations of β and p_i^c as functions of m for short-range moves: $p_r = 0.5$; $C = 0.6$, lattice size, 100×100. For β, typical error bars have been represented.

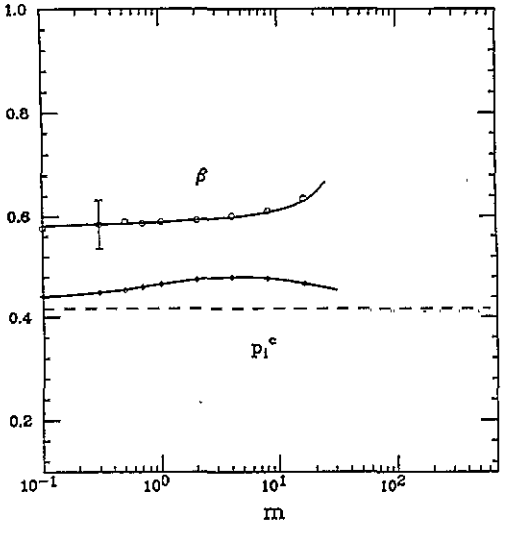

Figure 5. Variations of β and p_i^c as functions of m for short-range moves: $p_r = 1$; $C = 0.6$; lattice size, 100×100. For β, typical error bars have been represented.

Concerning the value of the critical exponent β the following two points should be stressed.

(i) The fact that, for small values of m, the exponent β, approximately equal to 0.6, is much less than its mean-field value illustrates how wrong the assumption of homogeneous

mixing is. This model, which takes into account the fluctuations in the number of contacts in space and time, neglects, however, all other causes of heterogencity.

(ii) When $m = 0$, the value of β for this model is equal to the value of β for two-dimensional directed percolation (Bease 1977). This result strongly suggests that the critical properties of our model are universal, i.e. model independent.

For given values of p_i and p_r, we have also studied the asymptotic behaviour of the stationary density of infectives, for both small and large values of m. More precisely, we have determined the following two exponents

$$\alpha_0 = \lim_{m\to 0} \frac{\log(I(m,\infty) - I(0,\infty))}{\log m} \tag{7}$$

$$\alpha_\infty = \lim_{m\to \infty} \frac{\log(I(\infty,\infty) - I(m,\infty))}{\log m}. \tag{8}$$

The log–log plots in figures 6 and 7 show that $\alpha_0 = 0.177 \pm 0.015$ and $\alpha_\infty = -0.945 \pm 0.065$.

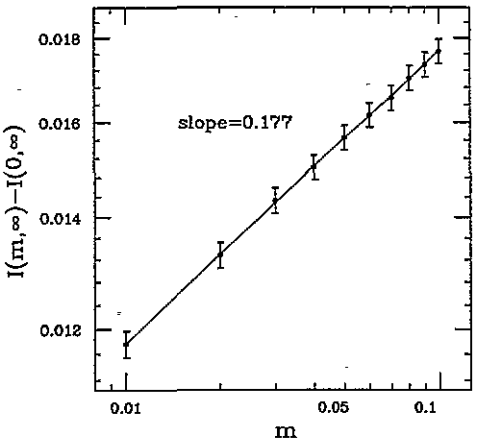

Figure 6. Log–log plot of $I(m,\infty) - I(0,\infty)$ as a function of m for given values of p_i and p_r in the case of short-range moves. The slope is 0.177 ± 0.015. Here $p_i = p_r = 0.5$. Total density $C = 0.6$; lattice size, 100×100.

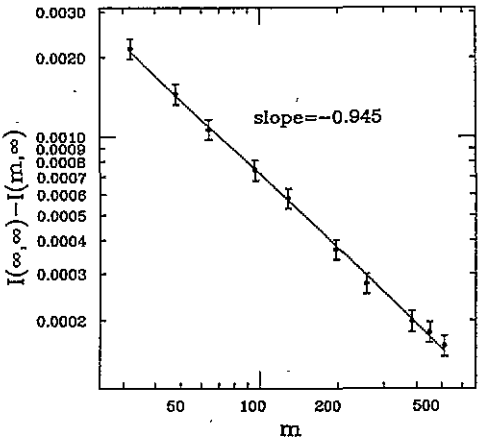

Figure 7. Log–log plot of $I(\infty,\infty) - I(m,\infty)$ as a function of m for given values of p_i and p_r in the case of short-range moves. The slope is -0.945 ± 0.065. Here $p_i = p_r = 0.5$. Total density $C = 0.6$; lattice size, 100×100. Error bars are represented.

The fact that α_0 is rather small shows the importance of motion in the spread of a disease. The stationary number of infectives increases dramatically when the individuals start to move. In other words, we may say that the response $\partial I(m,\infty)/\partial m$ of the stationary density of infectives to motion of the individuals tends to ∞ when m tends to 0. The asymptotic behaviour of $I(m,\infty)$ for small m is related to the asymptotic behaviour of $I(0,t)$ for large t, as shown by the following approximate argument. $I(0,\infty)$ is an average computed over all the configurations belonging to the attractor of the probabilistic cellular automaton rule modelling infection and recovery (first subrule). Between two successive applications of this rule, moving the individuals changes the stationary density of infectives and, if m is small, it may be assumed that the rate with which the density changes is proportional to m. Therefore, we may write

$$\lim_{t\to\infty} \frac{\partial I(m,t)}{\partial t} = \mathrm{O}(m). \tag{9}$$

If, for large t, we have (see figure 8)

$$\lim_{m\to 0} I(m,t) = \mathrm{O}(t^{-\gamma}) \tag{10}$$

then $t^{-\gamma-1} = \mathrm{O}(m)$. Thus $\lim_{t\to\infty} I(m,t) = \mathrm{O}(m^{\gamma/(\gamma+1)})$. Therefore, this simple argument yields

$$\alpha_0 = \gamma/(\gamma+1). \tag{11}$$

We have found $\gamma = 0.26 \pm 0.06$, that is $\gamma/(\gamma+1) = 0.206 \pm 0.06$. Considering the large error on γ (about 25%) (11) is approximately verified.

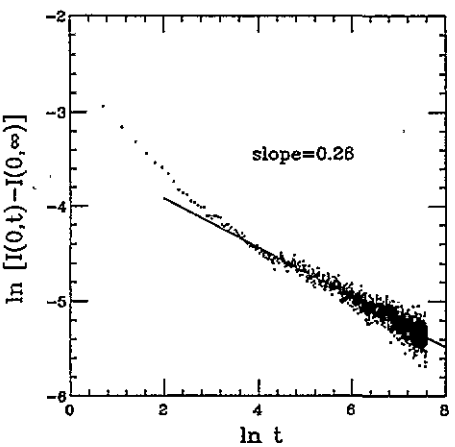

Figure 8. Asymptotic behaviour of the density of infectives $I(0,t)$ for large t. Here $p_1 = p_r = 0.5$. Total density $C = 0.6$; lattice size, 1000×1000. Initial density of infectives $I(0,0) = 0.8C$. The slope is -0.26 ± 0.06.

Since random short-range moves correspond to diffusion, the value of α_∞, which is close to -1, is not surprising. Indeed, for large m, this diffusive motion destroys correlations in a volume which behaves as $m^{d/2}$, where d is the space dimensionality. Therefore, if the spatial correlations created by the first subrule decrease rapidly enough, $I(m,\infty)$ should tend to $I(\infty,\infty)$ as $1/m$.

4.2. Long-range moves

For long-range moves, the variations of β and p_i^c as functions of m, for a fixed value of p_r, are very different from those for short-range moves. Figure 9 shows that β and p_i^c reach their mean-field values very rapidly. Whereas for short-range moves, β and p_i^c do not vary in the small-m regime, here, in contrast, the derivatives of β and p_i^c with respect to m tend to ∞ as m tends to 0. For small m, the asymptotic behaviour of β and p_i^c may, therefore, be characterized by an exponent. Figures 10 and 11 show log–log plots of, respectively, $\beta(m) - \beta(0)$ and $p_i^c(0) - p_i^c(m)$ as functions of m. Both exponents are close to 0.5.

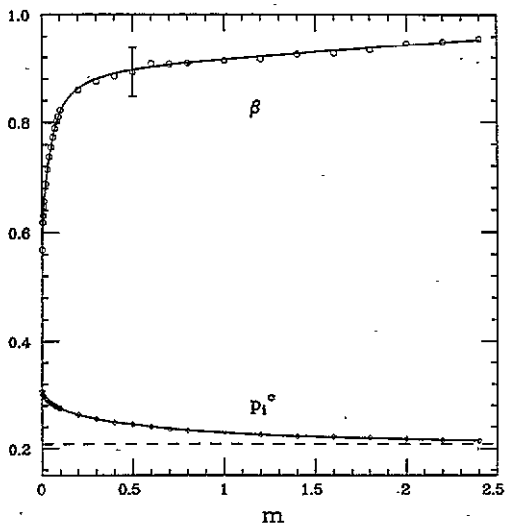

Figure 9. Variations of β and p_i^c as functions of m for long-range moves. $p_r = 0.5$, $C = 0.6$, lattice size, 100×100. For β, a typical error bar is represented.

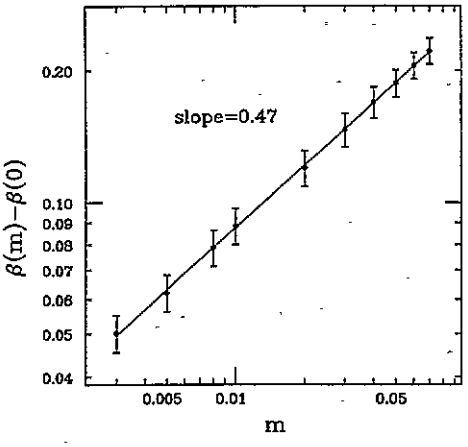

Figure 10. Log–log plot of $\beta(m) - \beta(0)$ as a function of m for a given value of p_r in the case of long-range moves. The slope is 0.47 ± 0.05. Here $p_r = 0.5$. Total density $C = 0.6$; lattice size, 100×100. Error bars are represented.

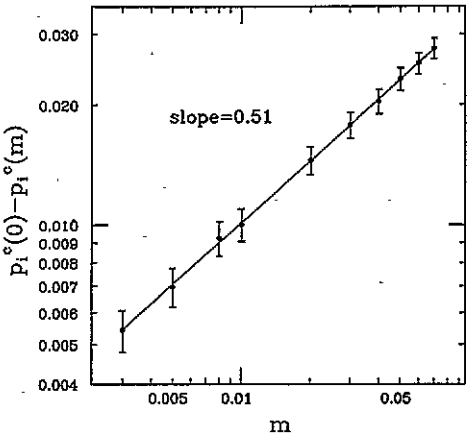

Figure 11. Log–log plot of $p_i^c(0) - p_i^c(m)$ as a function of m for a given value of p_r in the case of long-range moves. The slope is 0.51 ± 0.05. Here $p_r = 0.5$. Total density $C = 0.6$; lattice size, 100×100. Error bars are represented.

5. Conclusion

We have studied an automata network SIS model for the spread of infectious diseases in populations of moving individuals. The local rule of the automaton consists of two subrules. The first, which is synchronous, models infection and recovery, the second, applied sequentially, describes the different types of moves the individuals may perform. The model contains three parameters. p_i and p_r, which are, respectively, the probabilities to

be infected by contact and to recover, characterize the probabilistic cellular automaton rule modelling infection and recovery. The mixing process, which follows from the application of the second subrule, is characterized by a parameter m that represents the average number of tentative moves per individual. Depending upon the values of these parameters, in the infinite-time limit, the system is either in the disease-free state or in the endemic state. It goes from one state to the other through a transcritical bifurcation similar to a second-order phase transition characterized by a non-negative order parameter, whose role is played, in this model, by the stationary density of infected individuals.

Our main results emphasize the influence of m, i.e. the importance of motion. In particular, we have found that the derivative of the stationary density of infectives with respect to m, which characterizes the response of the system to motion of the individuals, tends to ∞ as m tends to zero, and, therefore, the asymptotic behaviour of the stationary density of infectives for small m may be characterized by an exponent which has been determined.

In the neighbourhood of the phase transition the system exhibits a critical behaviour due, for any finite value of m, to the local character of the subrule modelling infection and recovery. For $m = 0$, the critical exponent β has the value found for two-dimensional directed percolation, suggesting that the critical behaviour of our SIS model is universal, i.e. the same for a large class of two-dimensional models. The (p_i, p_r) phase diagram and the critical behaviour of the stationary density of infectives have been studied as functions of m. According to whether the individuals perform short- or long-range moves, we have found that, for a fixed value of p_r, the threshold p_i^c and the exponent β have qualitatively different behaviour as m varies. When m is small, the derivatives of p_i and β are, to the precision of our results, equal to zero for short-range moves, whereas they are infinite for long-range moves. When m becomes very large, the correlations created by the application of the subrule modelling infection and recovery are destroyed and, as expected, the behaviour of the system is then correctly predicted by the mean-field approximation.

Appendix. Constant interaction SIS model

In a constant interaction model, the neighbourhood U_i of a given vertex i consists of all the other vertices. That is, $U_i = V - \{i\}$. Hence, if $|V|$ denotes the total number of vertices, $|U_i| = |V| - 1$. Since the number of neighbours is very large, the probability to become infective by contact must be very small. More precisely, when $|V|$ tends to infinity, this probability should behave as $1/|V|$. Therefore, equation (3) becomes

$$I_{CI}(t+1) = (1-p_r)I_{CI}(t) + (C - I_{CI}(t))\left(1 - \left(1 - \frac{p_i I_{CI}(t)}{|V|}\right)^{|V|-1}\right) \tag{A1}$$

where C is the total density and I_{CI} denotes the density of infectives. In the limit $|V| \to \infty$, equation (A1) is replaced by

$$I_{CI}(t+1) = (1-p_r)I_{CI}(t) + (C - I_{CI}(t))(1 - \exp(-p_i I_{CI}(t))). \tag{A2}$$

Note that, in this model, the parameter p_i does not represent a probability. That is, it may take values greater than 1.

Depending upon the values of the different parameters, the stationary density of infectives $I_{CI}(\infty)$ for this model exhibits a behaviour similar to that obtained using the mean-field approximation. In particular, in the (p_i, p_r) parameter plane,

$$Cp_i - p_r = 0 \tag{A3}$$

is the equation of the second-order phase-transition line.

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