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Automata network SIR models for the spread of infectious diseases in populations of moving individuals

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Abstract. Automata network SIR models for the spread of infectious diseases are studied. The local rule consists of two subrules. The first one, applied sequentially, describes the motion of the individuals, the second is synchronous and models infection and removal (or recovery). The spatial correlations created by the application of the second subrule are partially destroyed according to the degree of mixing of the population which follows from the application of the first subrule. One- and two-population models are considered. In the second case, individuals belonging to one population may be infected only by individuals belonging to the other population as is the case, for example, for the heterosexual propagation of a venereal disease. It is shown that the occurrence of the epidemic in one population may be triggered by the occurrence of the epidemic in the other population. The emphasis is on the influence of the degree of mixing of the individuals which follows from their diffusive motion. In particular, the asymptotic behaviours for very small and very large mixing are determined. When the degree of mixing tends to infinity the correlations are completely destroyed and the time evolution of the epidemic is then correctly predicted by the mean-field approximation.

1. Introduction

The majority of epidemic models are formulated in terms of either differential equations or stochastic processes (Bailey 1975, Waltman 1974). This paper deals with automata network SIR models for the spread of infectious diseases within a population of moving individuals. The emphasis is on the influence of motion. This factor is usually neglected in epidemic models (Grassberger 1983, 1985). In an SIR model, based on disease status, the individuals are divided into three 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;

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

(R) the *removed* group, i.e. those individuals who have had the disease and are dead, or are isolated, or have recovered and are permanently immune.

The possible evolution of an individual may, therefore, be represented by the following transfer diagram:

$$S \xrightarrow{p_i} I \xrightarrow{p_r} R$$

where p_i and p_r denotes, respectively, the probability of being infected and the probability to be removed.

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The models discussed in this paper are formulated in terms of automata networks (Goles and Martínez 1990). Automata networks consist of graphs with a discrete variable at each vertex. Each vertex variable evolves in discrete time steps according to a definite rule involving the value of neighbouring vertex variables. The vertex variables may be updated sequentially or synchronously.

Automata networks are discrete dynamical systems, which may be defined more formally 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 $f_i: Q^{|U_i|} \rightarrow Q$ 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 models the set V is the two-dimensional torus $\mathbf{Z}_L^2$, where $\mathbf{Z}_L$ is the set of integers modulo L. A vertex is either empty or occupied by an individual belonging to one of the three 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. This hypothesis neglects latent periods, i.e. an infected susceptible becomes immediately infective.

(ii) Infectives are removed (or become permanently immune) with a probability p_r. This assumption states that removal is equally likely among infectives. In particular, it does not take into account the length of time the individual has been infective.

(iii) The time unit is the time step. During one time step, the two preceding rules are applied after the individuals have moved 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 nearest 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 $\mathbf{Z}_L^2$, mN individuals, where m is a real positive number, are sequentially selected at random to perform a move. This sequential process allows some individuals to move more than others. 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. It is a measure of the *degree of mixing* which follows from the application of this rule.

This model assumes that the population is closed. It ignores births, deaths by other causes, immigrations, or emigrations.

It is straightforward to extend this model to more than one population. For instance, the heterosexual spread of a venereal disease involves the obligatory switching of infection back and forth between two distinct populations. In this case, the probability for a susceptible of population 1 (respectively 2) to become infective by contact with an infective of population 2 (respectively 1) is denoted by $p_{1,i}$ (respectively $p_{2,i}$), and the probability for an infective of population 1 (respectively 2) to be removed is denoted by $p_{1,r}$ (respectively $p_{2,r}$). A less crude model for the heterosexual spread

of a venereal disease may be obtained, for example, by separating males and females into different age groups and assuming that a male (respectively female) susceptible belonging to a given age group can catch the disease from a female (respectively male) infective if, and only if, the infective belongs to neighbouring age groups.

These models are automata networks with mixed transition rules. That is, at each time step, the evolution results from the application of two subrules. The first subrule specifies the motion of the individuals. It is applied *sequentially*. The second one determines which susceptibles become infectives and which infectives are removed. It is applied *synchronously*. Both subrules are probabilistic. In all the models, the transition rules are translation invariant, i.e. they do not depend upon the vertex i.

2. Mean-field approximation

The mean-field approximation ignores space dependence and neglects correlations. In the case of a system exhibiting a phase transition the quantitative predictions of a mean-field approximation are not very good, but, for the SIR models described in the preceding section, since the first subrule represents a process that destroys the correlations created by the second subrule, if m tends to ∞, the mean-field approximation should become exact.

If the densities of the different groups of individuals are not space-dependent, the state of the population α at time t is characterized by the respective densities $S_\alpha(t)$, $I_\alpha(t)$ and $R_\alpha(t)$ of susceptible, infective and removed individuals. $C_\alpha = S_\alpha(t) + I_\alpha(t) + R_\alpha(t)$ is the density of individuals of population α. Since the population is closed, it is time-independent.

2.1. *The one-population model*

Since we are considering one population, we omit the index α. We have

$$S(t+1) = C - I(t+1) - R(t+1) \tag{1}$$

$$R(t+1) = R(t) + p_r I(t) \tag{2}$$

$$I(t+1) = I(t) + S(t)(1 - (1 - p_i I(t))^z) - p_r I(t) \tag{3}$$

where z is the number of neighbouring vortices of a given vortex.

From equations (1)–(3), it follows that $S(t)$ is positive non-increasing whereas $R(t)$ is positive non-decreasing. Therefore, the infinite-time limits $S(\infty)$ and $R(\infty)$ exist. Since $I(t) = C - S(t) - R(t)$, it follows also that $I(\infty)$ exists and satisfies the relation

$$R(\infty) = R(\infty) + p_r I(\infty)$$

which shows that $I(\infty) = 0$.

If the initial conditions are

$$R(0) = 0 \qquad \text{and} \qquad I(0) \ll S(0)$$

$I(1)$ is small, and we have

$$I(1) - I(0) = (z p_i S(0) - p_r) I(0) + \mathrm{O}\left(I^2(0)\right). \tag{4}$$

Hence, according to the initial value of the density of susceptibles, we may distinguish two cases:

(i) If $S(0) < p_r/zp_i$ then $I(1) < I(0)$. Since $S(t)$ is a non-increasing function of time $I(t)$ goes monotonically to zero as t tends to ∞. That is, no epidemic occurs.

(ii) If $S(0) > p_r/zp_i$ then $I(1) > I(0)$. The concentration $I(t)$ of infectives increases as long as the density of susceptibles $S(t)$ is greater than the threshold p_r/zp_i and then tends monotonically to zero.

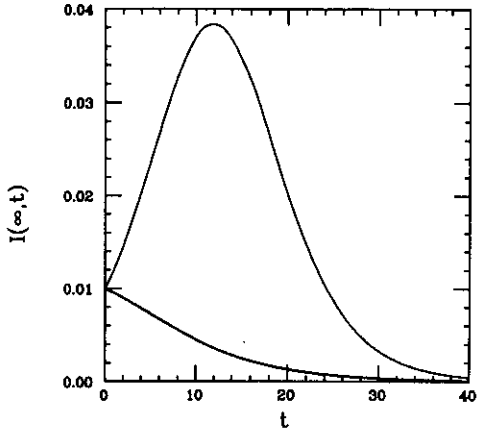

Figure 1. Time evolution of the density of infectives for the one-population model using the mean-field approximation. $C = 0.6$; $I(0) = 0.01$; $z = 4$; $p_i = 0.3$. (*a*) $p_r = 0.5$ ($S(0) > p_r/zp_i$). (*b*) $p_r = 0.75$ ($S(0) < p_r/zp_i$).

This shows that the spread of the disease occurs only if the initial density of susceptibles is greater than a threshold value. This threshold theorem has been established for the first time by Kermack and McKendrick (1927) using an epidemic model formulated in terms of a set of three differential equations. $I(t)$ being, in general, very small, equation (3) is well approximated by

$$I(t+1) = I(t) + zp_iS(t)I(t) - p_rI(t) \tag{3'}$$

which shows that the mean-field approximation is equivalent to a time discrete formulation of the Kermack–McKendrick model. Figure 1 shows two typical time evolutions of the density of infectives.

2.2. The two-population model

Here $\alpha = 1, 2$. We have

$$S_\alpha(t+1) = C_\alpha - I_\alpha(t+1) - R_\alpha(t+1) \tag{5}$$

$$R_\alpha(t+1) = R_\alpha(t) + p_{\alpha,r}I_\alpha(t) \tag{6}$$

$$I_\alpha(t+1) = I_\alpha(t) + S_\alpha(t)\left(1 - (1 - p_{\alpha,i}I_\beta(t))^z\right) - p_{\alpha,r}I_\alpha(t) \tag{7}$$

where α and β are equal to one or two with $\alpha \neq \beta$.

As for the one-population model, it follows that, for $\alpha = 1, 2$, $S_\alpha(t)$ is positive non-increasing whereas $R_\alpha(t)$ is positive non-decreasing. Therefore, the infinite-time

limits $S_\alpha(\infty)$ and $R_\alpha(\infty)$ exist. Since $I_\alpha(t) = C_\alpha - S_\alpha(t) - R_\alpha(t)$, it also follows that $I_\alpha(\infty)$ exists and satisfies the relation

$$R_\alpha(\infty) = R_\alpha(\infty) + p_{\alpha,r} I_\alpha(\infty)$$

which shows that $I_\alpha(\infty) = 0$.

Due to the coupling between the two populations a wider variety of situations may occur. For instance, if the initial conditions are

$$R_\alpha(0) = 0 \qquad \text{and} \qquad I_\alpha(0) \ll S_\alpha(0)$$

$I_\alpha(1)$ is small, and we have

$$I_\alpha(1) - I_\alpha(0) = z p_{\alpha,i} S_\alpha(0) I_\beta(0) - p_{\alpha,r} I_\alpha(0) + O(I_\beta^2(0)) \tag{8}$$

where α and β are equal to one or two with $\alpha \neq \beta$. Hence, according to the initial values $S_1(0)$, $S_2(0)$, $I_1(0)$ and $I_2(0)$, we may observe the following behaviours:

(i) If $z p_{1,i} S_1(0) I_2(0) - p_{1,r} I_1(0) < 0$ and $z p_{2,i} S_2(0) I_1(0) - p_{2,r} I_2(0) < 0$, then $I_1(1) < I_1(0)$ and $I_2(1) < I_2(0)$. Since S_1 and S_2 are non-increasing functions of time, $I_1(t)$ and $I_2(t)$ go monotonically to zero as t tends to ∞. No epidemic occurs.

(ii) If $z p_{1,i} S_1(0) I_2(0) - p_{1,r} I_1(0) > 0$ and $z p_{2,i} S_2(0) I_1(0) - p_{2,r} I_2(0) > 0$, then $I_1(1) > I_1(0)$ and $I_2(1) > I_2(0)$. The densities of infectives in both populations increase as long as the densities of susceptibles and infectives satisfy the relations

$$z p_{1,i} S_1(t) I_2(t) - p_{1,r} I_1(t) > 0 \qquad \text{and} \qquad z p_{2,i} S_2(t) I_1(t) - p_{2,r} I_2(t) > 0$$

and then tend monotonically to zero.

(iii) If $z p_{1,i} S_1(0) I_2(0) - p_{1,r} I_1(0) < 0$ and $z p_{2,i} S_2(0) I_1(0) - p_{2,r} I_2(0) > 0$, then $I_1(1) < I_1(0)$ and $I_2(1) > I_2(0)$, but, since $I_1(t+1)$ depends on $I_2(t)$, the density of infectives in population 1 does not necessarily goes monotonically to zero. After having decreased for few time steps, due to the increase of the density of infectives in population 2, it may increase if $z p_{1,i} S_1(t) I_2(t) - p_{1,r} I_1(t)$ becomes positive. The spread of the disease in population 2 may trigger the epidemic in population 1. If, however, the increase of the density of infectives in population 2 is not high enough, then the density of infectives in population 1 will decrease monotonically whereas the density of infectives in population 2 will increase as long as the density of susceptibles $S_2(t)$ is greater than $p_{2,r} I_1(t) / z p_{2,i} I_1(t)$, and then tends monotonically to zero. The disease spreads only in population 2 whereas no epidemic occurs in population 1.

Figures 2(*a*)–(*d*) show some typical time evolutions of the density of infectives in both populations.

In phase transition theory, it is well known that infinite-range interaction models exhibit a mean-field behaviour. In the appendix infinite-range interaction versions of the one- and two-population SIR models are presented.

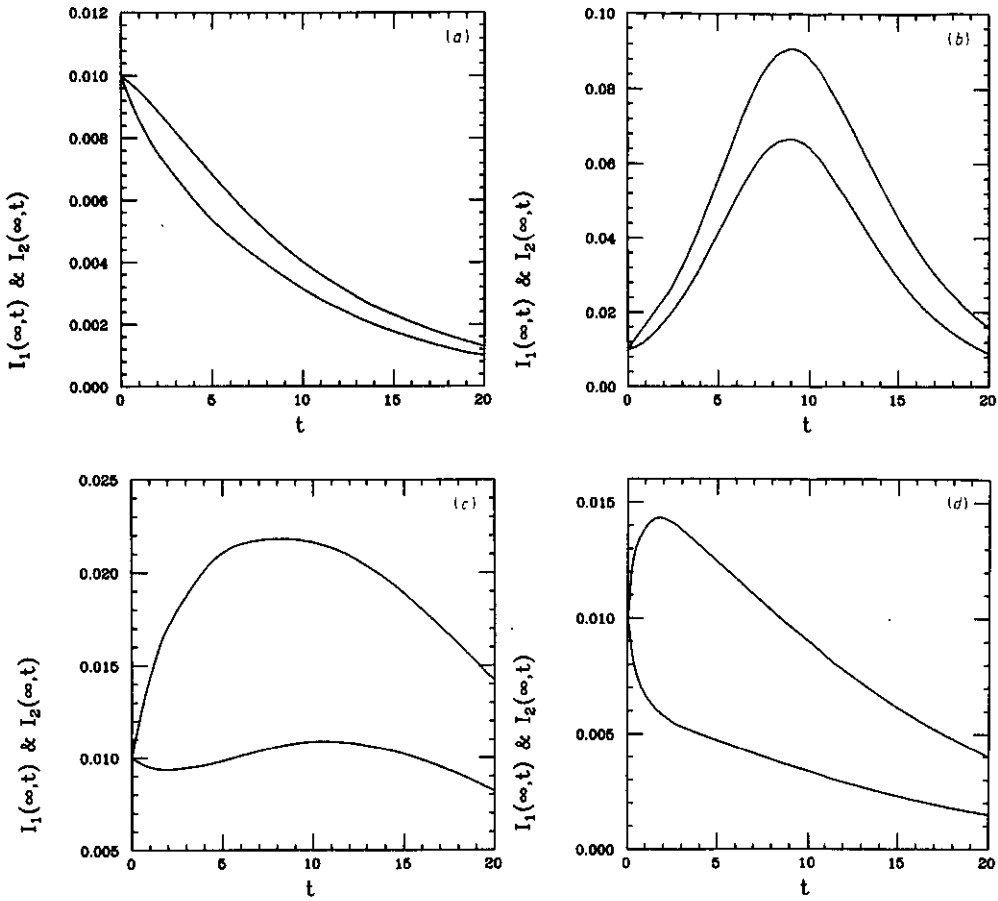

Figure 2. Time evolution of the densities of infectives for the two-population model using the mean-field approximation. $Q_1 = zp_{1,i}S_1(0)I_2(0) - p_{1,r}I_1(0)$; $Q_2 = zp_{2,i}S_2(0)I_1(0) - p_{2,r}I_2(0)$; $z = 4$; $S_1(0) = S_2(0) = 0.29$; $I_1(0) = I_2(0) = 0.01$. (*a*) $Q_1 < 0$ and $Q_2 < 0$, $p_{1,i} = 0.37$, $p_{2,i} = 0.23$, $p_{1,r} = 0.6$, $p_{2,r} = 0.3$. (*b*) $Q_1 > 0$ and $Q_2 > 0$, $p_{1,i} = 0.5$, $p_{2,i} = 0.8$, $p_{1,r} = 0.35$, $p_{2,r} = 0.25$. (*c*) $Q_1 < 0$ and $Q_2 > 0$, $p_{1,i} = 0.13$, $p_{2,i} = 0.8$, $p_{1,r} = 0.27$, $p_{2,r} = 0.35$. (*d*) $Q_1 < 0$ and $Q_2 > 0$, $p_{1,i} = 0.15$, $p_{2,i} = 0.6$, $p_{1,r} = 0.5$, $p_{2,r} = 0.3$.

3. Simulations

3.1. The one-population model

In all our simulations, the total density of individuals is above 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 $m = 0$. Figure 3 shows that the influence of the parameter m on the time evolution of an epidemic with permanent removal for short-range moves. As m increases the density of infectives as a function of time tends to the mean-field result. Figure 4 shows that the convergence to the mean-field result is much faster for long-range moves. Mixing is more effective with long-range moves. If, instead of permanent removal, infectives recover with the probability p_r and become permanently immune the convergence to the mean-field result is slower (figure 5) since the presence of the inert immune population on the lattice interfere

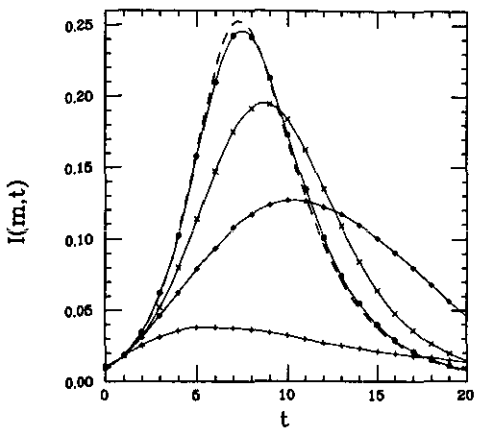

Figure 3. Time evolution of an epidemic for the one-population model for different values of m. Short-range moves and permanent removal. $C = 0.6$; $I(0) = 0.01$; $p_i = 0.5$; $p_r = 0.3$, 100×100 lattice. Each point represents the average of 10 experiments: +, $m = 0$; ◇, $m = 5$; ×, $m = 25$; ○, $m = 250$. The broken curve corresponds to the mean-field approximation.

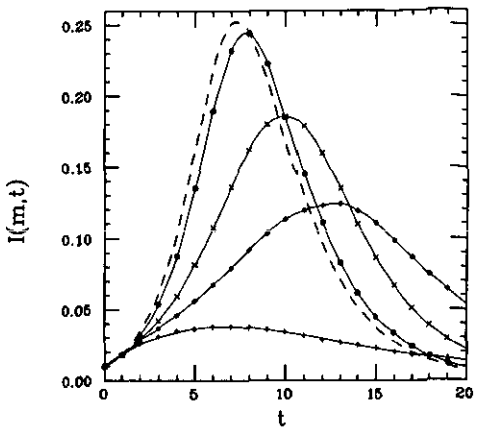

Figure 4. Time evolution of an epidemic for the one-population model for different values of m. Long-range moves and permanent removal. $C = 0.6$; $I(0) = 0.01$; $p_i = 0.5$; $p_r = 0.3$, 100×100 lattice. Each point represents the average of 10 experiments: +, $m = 0$; ◇, $m = 0.2$; ×, $m = 0.5$; ○, $m = 2$. The broken curve corresponds to the mean-field approximation.

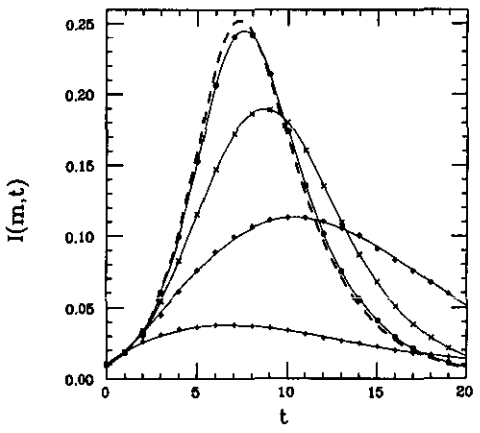

Figure 5. Time evolution of an epidemic for the one-population model for different values of m. Short-range moves and permanent recovery. $C = 0.6$; $I(0) = 0.01$; $p_i = 0.5$; $p_r = 0.3$, 100×100 lattice. Each point represents the average of 10 experiments: +, $m = 0$; ◇, $m = 5$; ×, $m = 25$; ○, $m = 250$. The broken curve corresponds to the mean-field approximation.

with the mixing.

Note that, since the initial configuration is random, for any type of move and any value of m, the value of density of infectives after the first time step is correctly predicted by the mean-field approximation.

As shown by Kermack and McKendrick (1927) the spread of the disease does not stop for lack of a susceptible population. As the time t tends to infinity, the stationary density of susceptibles $S(m,\infty)$ for a given value of m is positive. The variation of $S(m,\infty)$ as a function of m is represented in Figure 6 in the case of permanent removal and short-range moves. As expected $S(m,\infty)$ tends to the mean-field value as m tends to ∞. More precisely, the log–log plot, represented in Figure 7, shows

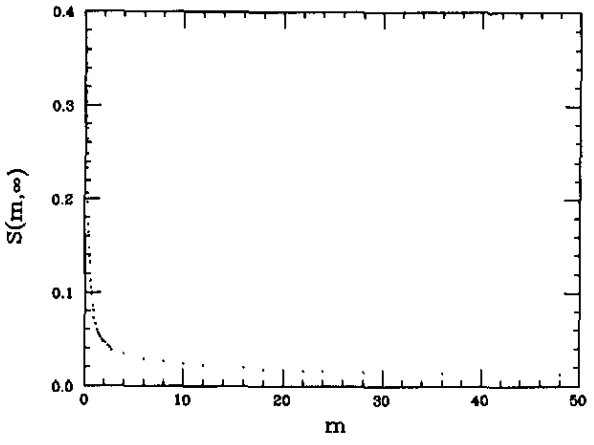

Figure 6. *Stationary density* of susceptibles for the one-population model as a function of m in the case of permanent removal and short-range moves.

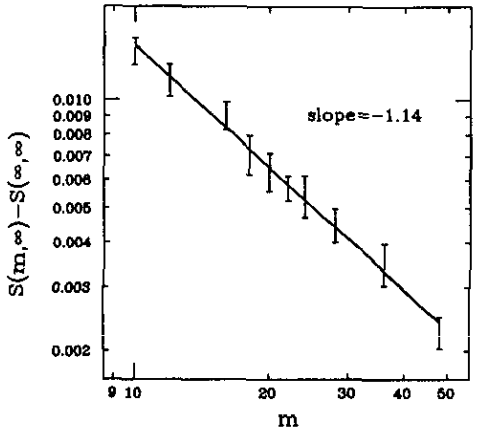

Figure 7. Asymptotic behaviour as m tends to ∞ of the stationary density of susceptibles for the one-population model in the case of permanent removal and short-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 100 x 100 lattice. Each point represents the average of 20 experiments. Slope $= -1.14 \pm 0.11$.

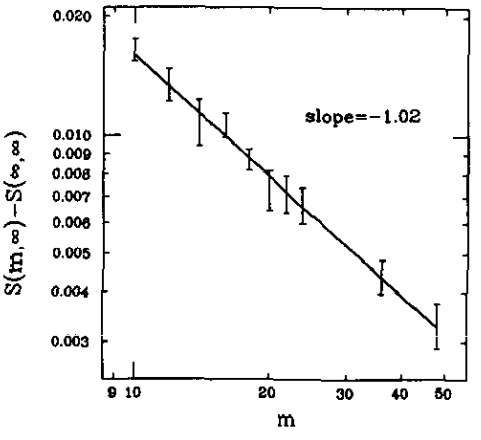

Figure 8. Asymptotic behaviour as m tends to ∞ of the stationary density of susceptibles for the one-population model in the case of permanent recovery and short-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 100 x 100 lattice. Each point represents the average of 20 experiments. Slope $\approx -1.02 \pm 0.11$.

that $S(m,\infty)$ tends to $S(\infty,\infty)$ as $m^{-\alpha}$, where $\alpha = 1.14 \pm 0.11$.

For comparison we have also studied the asymptotic behaviour as m tends to ∞ of $S(m,\infty) - S(\infty,\infty)$ if we have permanent recovery and short-range moves (figure 8) or permanent removal and long-range moves (figure 9). These two log–log plots show that the exponent α is equal to 1.02 ± 0.11 in the first case whereas it is equal to 1.73 ± 0.11 in the second one. The value of the exponent α characterizes the approach of the stationary density of susceptibles $S(m,\infty)$ to its mean-field value. α seems to depend upon the range of the move but not upon the fact that we have permanent recovery or permanent removal. For short-range moves which correspond to a diffusive motion, α is close to 1. When m is large, this diffusive motion destroys correlations in a volume which behaves as $m^{d/2}$, where d is the space dimensionality. One should expect a $1/m$ behaviour for $S(m,\infty) - S(\infty,\infty)$

when $d = 2$ (Grassberger 1991) if the spatial correlations decrease fast enough. We found that the approach of the stationary density of susceptibles to its mean-field value is faster for long-range moves. This is reasonable since mixing is more effective in this case.

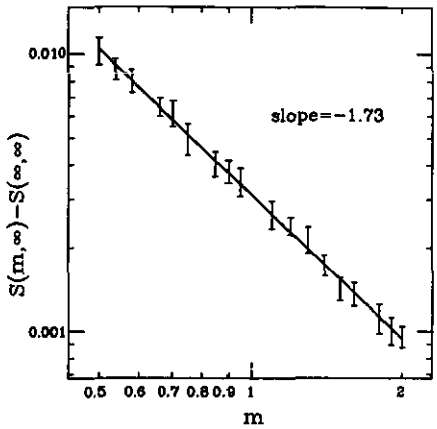

Figure 9. Asymptotic behaviour as m tends to ∞ of the stationary density of susceptibles for the one-population model in the case of permanent removal and long-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 100 x 100 lattice. Each point represents the average of 20 experiments. Slope = -1.73 ± 0.11.

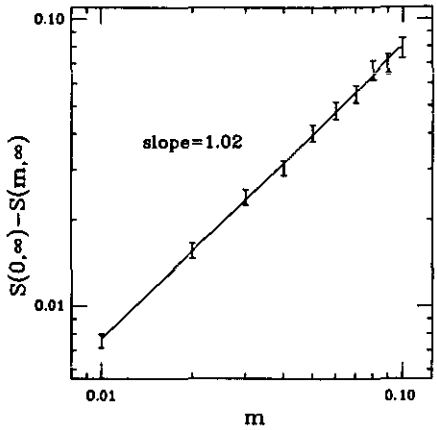

Figure 10. Asymptotic behaviour as m tends to 0 of the stationary density of susceptibles for the one-population model in the case of permanent removal and short-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 100 x 100 lattice. Each point represents the average of 400 experiments. Slope = 1.02 ± 0.05.

The log–log plot of $S(0,\infty) - S(m,\infty)$, represented in figure 10, shows that, for small m, $S(m,\infty)$ seems to behave linearly (slope = 1.02 ± 0.05). The absolute value of the derivative with respect to m of the stationary density of susceptibles at the origin is finite but it is, however, very large. That is, as soon as the individuals start to move, the spread of the disease increases dramatically.

The asymtotic behaviour of $S(0,\infty) - S(m,\infty)$ for small values of m is related to the asymptotic behaviour of $S(0,\infty) - S(0,t)$ for large values of t. More precisely, we argue that

$$S(0,\infty) - S(0,t) \sim \mathrm{e}^{-at} \iff S(0,\infty) - S(m,\infty) \sim m$$

where a is a positive constant. This result can be established as a consequence of the following two assumptions (Boccara *et al* 1992):

(i) The function

$$(m, \mathrm{e}^{-at}) \mapsto \Delta(m, \mathrm{e}^{-at}) = S(0,\infty) - S(m,t)$$

is a homogeneous function of m and e^{-at} such that

$$\Delta(\lambda^x m, \lambda \mathrm{e}^{-at}) \equiv \lambda \Delta(m, \mathrm{e}^{-at}).$$

(ii)

$$\lim_{t\to\infty} \frac{\partial}{\partial t} S(m,t) \sim m.$$

From the first assumption it follows that

$$\Delta(m, e^{-at}) = e^{-at}\Delta\left(me^{axt}, 1\right)$$

$$\Delta(m, e^{-at}) = m^{1/x}\Delta\left(1, e^{-at}/m^{1/x}\right).$$

That is, $S(0,\infty) - S(0,t) \sim e^{-at}$ and $S(0,\infty) - S(m,\infty) \sim m^{1/x}$.

The second assumption expresses the fact that, when m is small, the variation of the density of susceptibles per unit of time is proportional to m. This assumption seems reasonable if the approach to the attractor may be analysed in terms of annihilating particules or defects (Boccara *et al* 1991). Since

$$\frac{\partial}{\partial t}S(m,t) = -\frac{\partial}{\partial t}\Delta(m, e^{-at})$$

it follows that

$$\lim_{t\to\infty}\frac{\partial}{\partial t}S(m,t) \sim m^{1/x}.$$

Hence $x = 1$, that is $S(0,\infty) - S(m,\infty) \sim m$.

If, instead of an exponential behaviour, $S(0,\infty) - S(0,t)$ tends to zero as $t^{-\gamma}$ as t tends to ∞, where γ is a positive real, then, assuming that $(m,t) \mapsto S(0,\infty) - S(m,t)$ is a homogeneous function of m and t, it follows that $S(0,\infty) - S(m,\infty) \sim m^{\gamma/\gamma+1}$, which shows that, in this case, the derivative with respect to m of the asymptotic density of susceptibles for $m = 0$ is infinite.

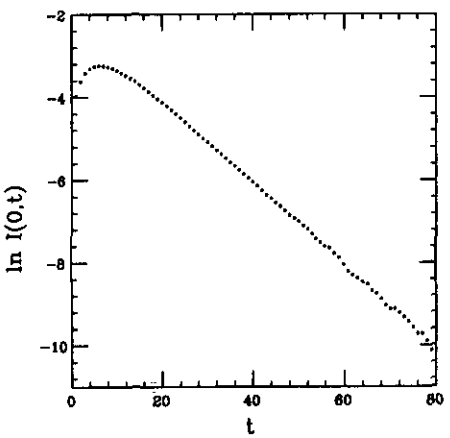

Figure 11. Variation of $\log I(0,t)$ as a function of t for the one-population model in the case of permanent removal and short-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 500 x 500 lattice. Each point represents the average of ten experiments.

Figure 11, which represents the variation of $\log I(0,t)$ as a function of t, shows that $I(0,t)$ tends to zero exponentially as t tends to ∞. Since

$$S(0,0) - S(0,t) = \int_0^t I(0,\tau)\,d\tau$$

it follows that

$$S(0,\infty) - S(0,t) \sim e^{-at}.$$

Here again for comparison we have also studied the asymptotic behaviour as m tends to zero of $S(0,\infty) - S(m,\infty)$ if we have permanent recovery and short-range moves (figure 12) or permanent removal and long-range moves (figure 13). These two log–log plots show that, for small m, $S(0,\infty) - S(m,\infty) \sim m$ (more precisely the slopes are, respectively, 1.01 ± 0.05 and 0.97 ± 0.10. This result does not depend, in particular, upon the range of the moves. This is reasonable since in the above proof there is no need to specify how the small mixing is created.

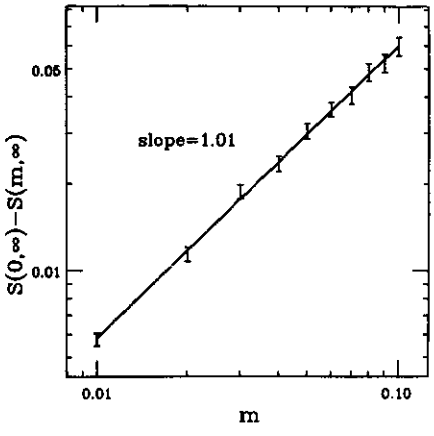

Figure 12. Asymptotic behaviour as m tends to 0 of the stationary density of susceptibles for the one-population model in the case of permanent recovery and short-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 100 x 100 lattice. Each point represents the average of 200 experiments. Slope = 1.01 ± 0.05.

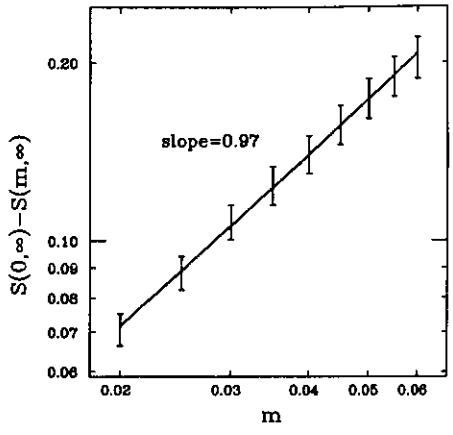

Figure 13. Asymptotic behaviour as m tends to 0 of the stationary density of susceptibles for the one-population model in the case of permanent removal and long-range moves. $C = 0.6$; $p_i = 0.5$; $p_r = 0.3$, 100 x 100 lattice. Each point represents the average of 300 experiments. Slope = 0.97 ± 0.10.

3.2. The two-population model

Figures 14–17 represent the time evolution of the densities of infectives for the two-population model for $m = 1$ in the case of short-range moves with permanent removal. Figures 18(*a*) and 18(*b*) show the influence of the parameter m on the time evolution of an epidemic when the occurrence of the epidemic in one population is triggered by the occurrence of the epidemic in the other population in the case of short-range moves with permanent removal. As for the one-population model, when m increases the density of infectives as a function of time tends to the mean-field result. For the values of the various probabilities chosen in this case, the epidemic in population 2 is not triggered by the occurrence of the epidemic in population 1 if $m = 0$ whereas it is when m is large enough.

4. Conclusion

We have studied automata network SIR models for the spread of infectious diseases in populations of moving individuals. The local rule of the automaton consists of two subrules. The first, applied sequentially, describes the different types of moves the

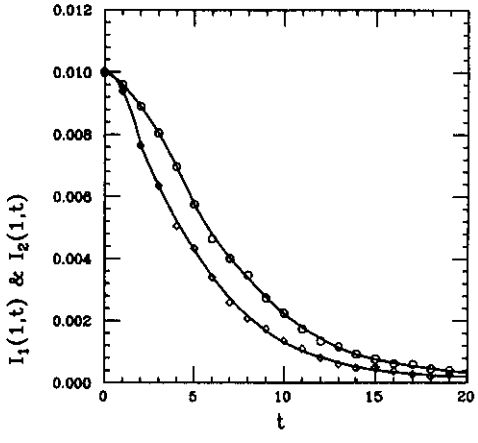

Figure 14. Time evolution of the densities of infectives for the two-population model for $m = 1$. Short-range moves and permanent removal. $C_1 = C_2 = 0.3$; $I_1(0) = I_2(0) = 0.01$; $p_{1,i} = 0.37$; $p_{1,r} = 0.52$; $p_{2,i} = 0.23$; $p_{2,r} = 0.30$, 100×100 lattice. Each point represents the average of 20 experiments: ⋄, population 1; ○, population 2.

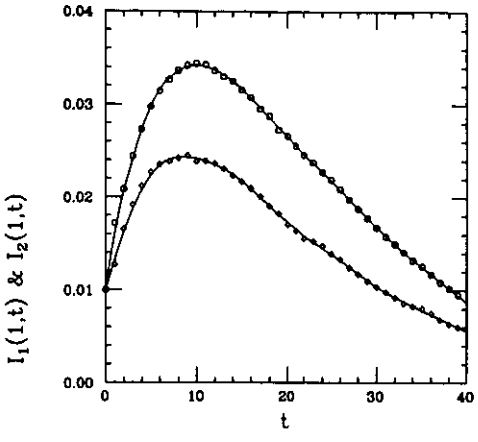

Figure 15. Time evolution of the densities of infectives for the two-population model for $m = 1$. Short-range moves and permanent removal. $C_1 = C_2 = 0.3$; $I_1(0) = I_2(0) = 0.01$; $p_{1,i} = 0.50$; $p_{1,r} = 0.30$; $p_{2,i} = 0.80$; $p_{2,r} = 0.20$, 100×100 lattice. Each point represents the average of 20 experiments: ⋄, population 1; ○, population 2.

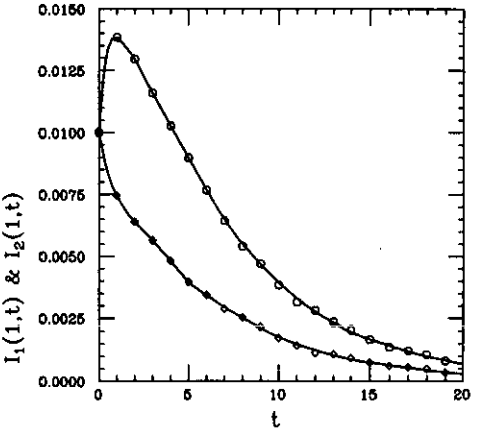

Figure 16. Time evolution of the densities of infectives for the two-population model for $m = 1$. Short-range moves and permanent removal. $C_1 = C_2 = 0.3$; $I_1(0) = I_2(0) = 0.01$; $p_{1,i} = 0.16$; $p_{1,r} = 0.45$; $p_{2,i} = 0.60$; $p_{2,r} = 0.30$, 100×100 lattice. Each point represents the average of 20 experiments: ⋄, population 1; ○, population 2.

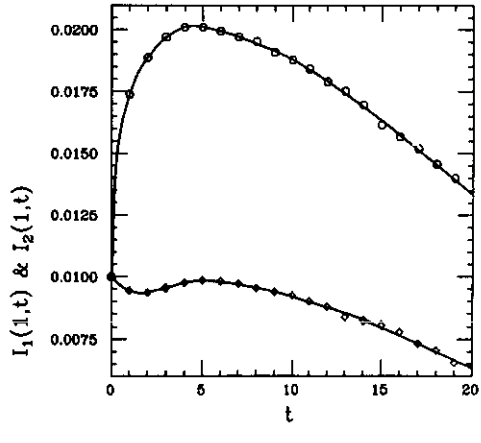

Figure 17. Time evolution of the densities of infectives for the two-population model for $m = 1$. Short-range moves and permanent removal. $C_1 = C_2 = 0.3$; $I_1(0) = I_2(0) = 0.01$; $p_{1,i} = 0.10$; $p_{1,r} = 0.19$; $p_{2,i} = 0.80$; $p_{2,r} = 0.16$, 100×100 lattice. Each point represents the average of 20 experiments. ⋄, population 1; ○, population 2.

individuals may perform whereas the second, which is synchronous, models infection and removal (or recovery). We have considered one- and two-population models. In the second case, individuals belonging to one population may be infected only by individuals belonging to the other population as is the case for the heterosexual

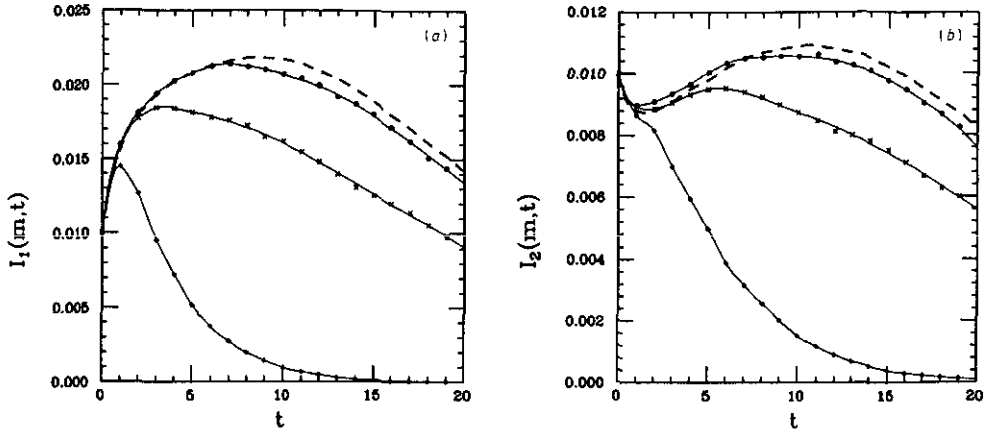

Figure 18. Time evolution of an epidemic for the two-population model for different values of m, when the occurrence of the epidemic in one population is triggered by the occurrence of the epidemic in the other population. Short-range moves and permanent removal. $C_1 = C_2 = 0.3$; $I_1(0) = I_2(0) = 0.01$; $p_{1,i} = 0.80$; $p_{1,r} = 0.35$; $p_{2,i} = 0.13$; $p_{2,r} = 0.27$, 100 x 100 lattice. Each point represents the average of 10 experiments. ◇, $m = 0$; ×, $m = 15$; ○, $m = 125$. The broken curve corresponds to the mean-field approximation. (*a*) Population 1. (*b*) Population 2.

propagation of a venereal disease. We have shown that the occurrence of the epidemic in one population may be triggered by the occurrence of the epidemic in the other population. Our main results, however, emphasize the influence of the degree of mixing which follows from the application of the first subrule. The degree of mixing is measured by a parameter m that represents the average number of tentative moves per individual. If m goes to ∞ then the time evolution of the epidemic is exactly described by the mean-field approximation. The approach of the mean-field value for the stationary density of susceptibles $S(m,\infty)$ is given by a power law behaviour, that is $S(m,\infty) - S(\infty,\infty)$ behaves as $m^{-\alpha}$, where the exponent α is close to one if the motion of the individuals is diffusive, that is for short-range moves. For long-range moves the approach to the mean-field value is faster. The behaviour of $S(m,\infty)$ for small values of m has also been studied. The derivative with respect to m of $S(m,\infty)$ is negative and very large showing that as soon as the individuals start to move, the spread of the disease increases dramatically. If we assume that $S(m,t) - S(0,\infty)$ is a homogeneous function of m and $e^{-\alpha t}$, and that the variation of the density of susceptibles per unit of time is proportional to m, which seems to be a reasonable assumption if the approach to the attractor may be analysed in terms of annihilating defects, then the linear behaviour of $S(m,\infty)$ for small values of m follows from the fact that $S(0,t)$ tends to $S(0,\infty)$ as $e^{-\alpha t}$.

Acknowledgments

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Appendix. Infinite-range interaction models

In an infinite-range 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 of becoming infective by contact must be very small. More precisely, when $|V|$ tends to infinity, this probability should behave as $1/|V|$. Therefore, in the case of a one-population model, equations (1)–(3) become

$$S(t+1) = C - I(t+1) - R(t+1) \tag{A1}$$

$$R(t+1) = R(t) + p_r I(t) \tag{A2}$$

$$I(t+1) = I(t) + S(t)\left(1 - \left(1 - \frac{p_i I_t}{|V|}\right)^{|V|-1}\right) - p_r I(t) \tag{A3}$$

where C is the total density. In the limit $|V| \to \infty$, equation (A3) is replaced by

$$I(t+1) = I(t) + S(t)\left(1 - \exp(-p_i I(t))\right) - p_r I(t). \tag{A4}$$

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

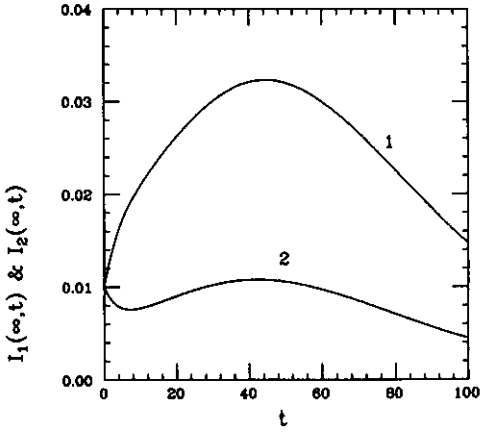

Figure 19. Typical time evolution of an epidemic for the two-population infinite-range model when the occurrence of the epidemic in population 2 is triggered by the occurrence of the epidemic in population 1. $C_1 = C_2 = 0.3$; $I_1(0) = I_2(0) = 0.01$; $p_{1,i} = 0.90$; $p_{1,r} = 0.06$; $p_{2,i} = 0.20$; $p_{2,r} = 0.14$.

The equations for the corresponding two-population model read

$$S_\alpha(t+1) = C_\alpha - I_\alpha(t+1) - R_\alpha(t+1) \tag{A5}$$

$$R_\alpha(t+1) = R_\alpha(t) + p_{\alpha,r} I_\alpha(t) \tag{A6}$$

$$I_\alpha(t+1) = I_\alpha(t) + S_\alpha(t)\left(1 - \exp(-p_{\alpha,i} I_\beta(t))\right) - p_{\alpha,r} I_\alpha(t) \tag{A7}$$

where C_α is the density of population α. α and β are equal to one or two with $\alpha \neq \beta$.

Depending upon the values of the different parameters, the densities of infectives as a function of time for these two models exhibit similar behaviour as those obtained

using the mean-field approximation. For instance, figure 19 shows, for the two-population model, a typical time evolution of an epidemic when the occurrence of the epidemic in population 2 is triggered by the occurrence of the epidemic in population 1.

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