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Cellular automata modelling of SEIRS*

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In this paper the SEIRS epidemic spread is analysed, and a two-dimensional probability cellular automata model for SEIRS is presented. Each cellular automation cell represents a part of the population that may be found in one of five states of individuals: susceptible, exposed (or latency), infected, immunized (or recovered) and death. Here studied are the effects of two cases on the epidemic spread. i.e. the effects of non-segregation and segregation on the latency and the infected of population. The conclusion is reached that the epidemic will persist in the case of non-segregation but it will decrease in the case of segregation. The proposed model can serve as a basis for the development of algorithms to simulate real epidemics based on real data. Last we find the density series of the exposed and the infected will fluctuate near a positive equilibrium point, when the constant for the immunized is less than its corresponding constant τ_0. Our theoretical results are verified by numerical simulations.

Keywords: cellular automata, epidemic, modelling, SEIRS modelling
PACC: 0550

1. Introduction

Throughout the 20th century, epidemics constituted a very important topic in mathematics and physics, biology, engineering, medicine. Epidemics have been modelled using differential equations. However, this approach has some serious drawbacks in that it neglects the local characteristics of the spreading process, and it does not include variable susceptibility of individuals. Cellular automata (CA) can overcome the above drawbacks and have been used by several researchers as an alternative method of modelling epidemics, and thus bridge a gap between the studies of microcosmic and macroscopical phenomena.

CA (von Neumann, 1966) are models of physical systems, where space and time are discrete and interactions are localized. They have been extensively used as models for complex systems. Many scholars have obtained good results in some fields, such as manager science,[1] fluid mechanics,[2] the chemistry oscillation, penetration and movement,[3] reaction-diffusion systems[4] etc. Sirakoulis *et al*[5] studied the effects of population movement and vaccination on epidemic spread by use of a cellular automaton model; Ahmed and Agiza[6] studied the SIR model, where the S, I and R stand for susceptible, infections, recovered respectively with CA. A differential equation about SEIRS model, where the SEIRS stand, respectively for susceptible, exposed, infections, recovered, susceptible, was studied by Langlais and Suppo,[7] and the global stability of an infection-free periodic solution was analyzed about the SEIRS model with time delay by Wang.[8]

The aim of this work is to use the CA approach to study the effect of population on epidemic spread. We develop a two-dimensional CA model, in which each cell represents a part of population that may be found in one of five states: susceptible, latency, infected, recovered and death. As the parts of population move randomly in the CA lattice, the disease spreads. We study the effects of two cases on the epidemic spread. i.e. the non-segregation and the segregation of population on the latency and the infected. We conclude that the epidemic will spread in the case of non-segregation but it will decrease in the case of segregation. The proposed model can serve as a basis for the development of algorithms to simulate real epidemics based on real data.

The paper is organized as follows. All necessary backgrounds of CA are given in Section 1. The basic

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notions of cellular automata are given in Section 2. A summary of the differential equations used to model the epidemic spread is given in Section 3. The model is described in Section 4. Based on this model, we develop an algorithm for the simulation of epidemic spreading. The effects of two cases on the epidemic spread. i.e. the effects of the non-segregation and the segregation of population on the latency and the infected are described in Section 5. Finally, the discussion about the results and the conclusions obtained in the previous sections are presented in Section 6.

2. Basic notions of cellular automata

First, we give the notions and definitions:

Lattice We set up a two-dimensional lattice denoted by L^2. Every cell in L^2 is denoted by $c(i,j)$, (for short by c,) where (i,j) is the position of the cell in the lattice, i is the row to the cell c, j is the column. Obviously $i \in N^+$ and $j \in N^+$ (N^+ refers to positive integer).

Neighbourhood By a cell neighbourhood we refer to a set of cells that affect the evolution of the central cell. The neighbourhood of the cell c is denoted by $N(c(i,j))$. For the cell c, a neighbourhood is then defined by the mapping

$$c \to N(c) = \{c(i-1,j), c(i+1,j), c(i,j), c(i,j+1), c(i,j-1)\}. \quad (1)$$

From Eq.(1) we illustrate the neighbourhood of von Neumann as shown in Fig.1.

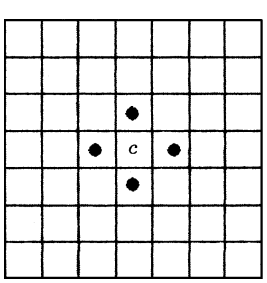


Fig.1. von Neumann neighbourhood.

State set It is a nonempty finite set containing the values that may be taken as the cell's state at each time step, and denoted by Z. When the system evolves from an initial state to a final state according to the transition rule, the cell states are given at discrete times. The state of cell c at the time t is denoted by $Z_{c(i,j)}(t)$ and the state of its neighbourhood by $Z_{N(c)}(t)$.

Transition function (F) F is the transition rule, also called system dynamics, which governs the system evolution and determines how cells change their states in time, depending on their neighbourhood. It may be given by an analytical function, a matrix, or a set of transmit rules. The transition function F may be considered as the mapping

$$Z_{N(c)}(t) \to Z_c(t+1),$$
$$Z_c(t+1) = F(Z_{N(c)}(t)). \quad (2)$$

Where $Z_{N(c)}(t)$ is the state of the neighbourhood $N(c)$ at the time t, and $Z_c(t+1)$ is the state of the cell c at the time $t+1$.

In practice, when simulating CA rules, we cannot consider an infinite domain; thus, the lattice is finite and has a boundary. The initial condition may be given or randomly generated, and about the boundary condition, we can refer to Refs.[9,10].

As above, a cellular automaton is defined as the tuple $A = (L, Z, N, F)$. The specific basic notions and characteristics can be seen in Refs.[11] and [12].

3. Different equation for modelling epidemic spreading

Differential equation models are used to describe epidemic spreading, and the ordinary differential equation for this model is as follows:

$$\begin{cases} S' = \eta^P R - \lambda_1 \dfrac{SE}{N} - \lambda_2 \dfrac{SI}{N}, \\ E' = \lambda_1 \dfrac{SE}{N} + \lambda_2 \dfrac{SI}{N} - \gamma E, \\ I' = \gamma E - kI - \mathrm{d}I, \\ R' = kI - \eta^P R, \\ D' = \mathrm{d}I, \end{cases} \quad (3)$$

where $N = S(t)+E(t)+I(t)+R(t)$, d is the mortality. Here S is susceptible part, E the exposed part, I the infected part, R the recovered part, and D the death part of the population. We define the constant η^P for a full return to susceptible. Generally we studied the stability of the solution to Eq.(3), and the basic reproductive number R_0, known as the condition of the epidemic extinction or persistence. In this paper, we include the state by modifying the SEIRS, $S \to E \to I \to R \to S$, to a state $S \to E \to I \to R \xrightarrow{P} S$, where P is the stage of partial immunity. We use the cellular automata approach to give the condition for the persistence or extinction of epidemic.

4. Description of the mode

We analyse the mechanism of the epidemic spreading. Each individual resides in one of S, E, I, R, D states. In most SEIRS models, the spreading course of an epidemic disease goes as follows: after some susceptible individual has contacted an infected and exposed individual, or the infected has contacted a susceptible individual, the susceptible individual becomes exposed, then infected, and finally either dies of the disease or recovers and the recovered becomes susceptible. Here, we divide the exposed and infected into many states, as shown in Fig.2. For example, the spread of influenza and the SARS spreading among cites occurred in this form in 2003.[13]

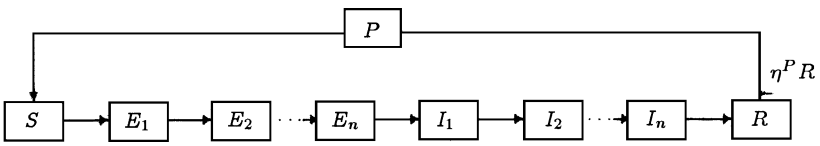


Fig.2. Flow chart for a generic SEIRS model.

Individuals is assumed to be distributed in the two-dimensional plane lattice of L^2, and each lattice represents an individual (or a cell), i.e. each lattice contains only one individual. So the number of lattice represents the individual number in the two-dimensional plane. The period of the exposed state equals $t_{\rm e}$, here $t_{\rm e} > 0$. After the $t_{\rm e}$ time elapsed the state of the cell becomes infected, the state of the cell is denoted by $Z_c^{\rm e}(t)$ when the cell c is exposed at the time t. The period of the infection is equal to $t_{\rm in}$, after the $t_{\rm in}$ time elapsed the state of the cell becomes recovered if the individual can survive. The state of the cell is denoted by $Z_c^{\rm i}(t)$ when the cell c is infected at the time t. Similarly, the state of the cell is denoted by $Z_c^{\rm s}(t)$ or $Z_c^{\rm r}(t)$ when the cell is susceptible or recovered at the time t. Next, we give the following state set:

$$S = \{0\}, \qquad R = \{r_1\} = \{0.5\}, \qquad D = \{-1\},$$
$$E = \{e_1, e_2, \ldots, e_n\} = \{1, 2, \ldots, n\},$$
$$I = \{\varepsilon_1, \varepsilon_2, \ldots, \varepsilon_n\} = \{n+1, n+2, \ldots, n+m\}.$$

where S is the susceptible state set, i.e. if $Z_c^{\rm s}(t) = 0$, it means that the cell c is susceptible at the time t. The E is the state set of the exposed parts, i.e. if $Z_c(t) \in E$, it means that the individual is exposed at the time t. I is the state set of the infected, i.e. if $Z_c \in I$, it means that the individual is the infected part at the time t. Similarly, R and D have their respective meanings.

Assume each of lattices contains a single individual; the total lattice on the two-dimensional plane is denoted by J. Here we use the $E(t)$ and $I(t)$ to represent the total numbers of the exposed and the infected at the time t respectively. Analogous to the statistical mechanics of one-dimensional cellular automata,[14] the $E(t)$ and $I(t)$ can be respectively written as

$$E(t+1) = E(t) + E_{s\to i}(t) - E_{e\to i}(t),$$
$$I(t+1) = I(t) - I_{id}(t) - I_{ir}(t)).$$

Then the densities of the exposed and infected are given by the following equations:

$$x(t+1) = \frac{E(t+1)}{J} = \frac{E(t) + E_{\rm s\to i}(t) - E_{\rm e\to i}(t)}{J}, \tag{4}$$

$$y(t+1) = \frac{I(t+1)}{J} = \frac{I(t) - I_{\rm id}(t) - I_{\rm ir}(t))}{J}. \tag{5}$$

where $x(t)$ and $y(t)$ are the global densities of the exposed and the infected at the time t in the region respectively.

5. Transition function of cellular automata

There are five parameters $P_{\rm e}$, $P_{\rm in}$, $P_{\rm ir}$, η^P and $P_{\rm id}$ involved in the simulation. The $P_{\rm e}$ is the average probability of latency per discrete time interval in which the exposed transmits the disease to the susceptible in its neighbourhood. The $P_{\rm in}$ is the average probability of infection per discrete time interval in which the infected transmits the disease to the susceptible in its neighbourhood. Here, invoking the von Neumann neighbourhood as shown in Fig.1, the $c(i,j)$ cell at the next time step $t+1$ is affected by the states of all four cells in its neighbourhood at the present time step t. So, the local transition rule of cellular automata F comprises the transmission between the

states, as described in Eq.(6):

$$F:\ Z^5 \to Z \Rightarrow Z_{c(i,j)}(t+1) = F\Big(Z_{c(i,j)}(t), Z_{c(i-1,j)}(t), Z_{c(i+1,j)}(t), Z_{c(i,j-1)}(t), Z_{c(i,j+1)}(t)\Big). \quad (6)$$

Consider a susceptible individual is on the site $c(i,j)$, whose neighbourhood N_c include χ_1 exposed and χ_2 infected individuals. The infecting and exposing act independently, so the susceptible acquires the pathogen in a single time interval in an average probability of

$$P = \frac{\chi_1 P_e + \chi_2 P_{in}}{4}.$$

Here χ_1 is the number of the exposed individuals in the von Neumann neighbourhood of the susceptible and the χ_2 is the number of infected individuals in the von Neumann neighbourhood of the susceptible. Thus, we have $\chi_1 \in \{0,1,2,3,4\}$, $\chi_2 \in \{0,1,2,3,4\}$, but $\chi_1 + \chi_2 \le 4$.

We define the η^P as the probability that an infective individual recovers and reverts to a susceptible one per time interval. P_{ir} is the probability of the infected becoming recover, which is equal to $1/t_{in}$. P_{id} is the probability of disease-induced mortality. The automata rule of the spreading on the site $c(i,j)$ for the susceptible is as follows:

If $Z_{c(i,j)}(t) = 0$, then $Z_{c(i,j)}(t+1) = 1$ with the probability P, i.e. if rand $< P$, then the state of the individual in the cell (i,j) changes from susceptible to exposed at $t+1$ time, otherwise it remains in the susceptible, i.e. $Z_{c(i,j)}(t+1) = 0$. Here rand refers to a random number between 0 and 1.

The rules of cellular automata for the exposed, infection and recovers, respectively, are

If $Z_{c(i,j)}(t) \in I$, then $Z_{c(i,j)}(t+1) = -1$ (death) with probability P_{id} , or $Z_{c(i,j)}(t+1) \in R$ with probability P_{ir};

If $Z_{c(i,j)}(t) \in R$, then $Z_{c(i,j)}(t+1) = 0$ with probability η^P;

If $Z_{c(i,j)}(t) \in E$, then $Z_{c(i,j)}(t+1) = Z_{c(i,j)}(t)+1$, and after t_e time steps the cell $c(i,j)$ become to infection.

6. Simulation results

We create the Matlab simulation by use of the rule of cellular automata in this modelling. The numbers of the exposed and infected state are assumed to be four and two respectively. Specifically $E = \{e_1,e_2,e_3,e_4\} = \{1,2,3,4\}$, $I = \{\varepsilon_1,\varepsilon_2\} = \{5,6\}$, $S = \{0\}$, $R = \{0.5\}$ and $D = \{-1\}$, where the states 1, 2, 3, and 4 refer to the state of individuals exposed, and the states 5 and 6 represent the state of individuals infected. Later the states 4 and 6 denotes the states of individuals with no disease due to segregation. The evolution takes place in the lattices of 100 by 100 cells, and the boundary is periodic.

6.1. Effects of non-segregation on the infected and exposed

Figure 3 gives an example of cellular automata evolution from disordered initial states in the L^2 plane, where $P_{id} = 0.004$, $P_{ir} = 0.6$, $\eta^P = 0.04$. The result is that 85 individuals are exposed and 60 individual are infected at the initial time, in which each site is randomly chosen to have a value from among one to six. From the statistical mechanics of cellular automata, the result demonstrates that the disease persists in this case. The global density time-series of the exposed and infected fluctuate around a positive equilibrium point as shown in Figs.3 and 4. Figure 4 gives an example of cellular automata evolution from disordered initial states, which have 85 exposed and 60 infected individuals at the initial time, with the parameters being the same as those in Fig.3, but for P_e and P_{in}. Comparing Fig.3 with Fig.4, we know that the positive equilibrium point increases with the increasing P_e and P_{in}. In order to compare with the segregation case, we give partial patterns of the evolution as shown in Fig.5, where the parameters are the same as those shown in Fig.3.

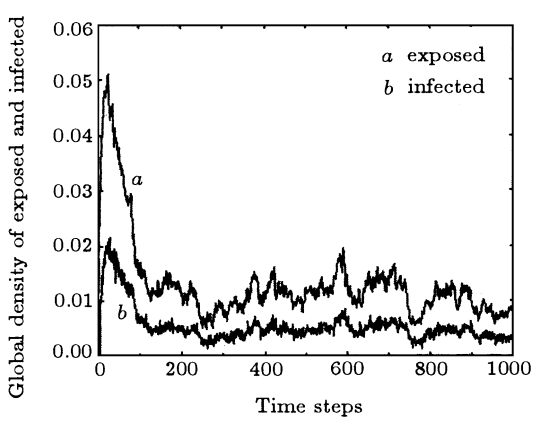


Fig.3. Global density time-series of the infected and exposed with $P_e = 0.4$, $P_{in} = 0.5$.

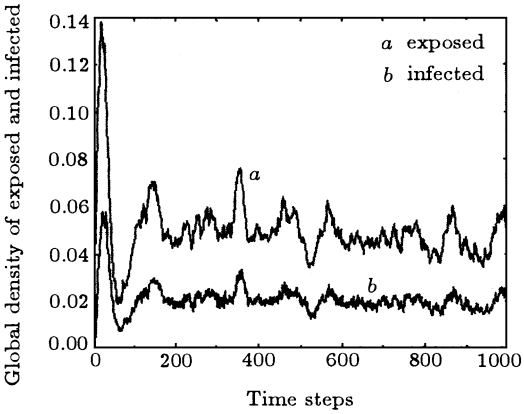


Fig.4. Global density time-series of the infected and exposed with $P_{\rm e} = 0.6$, $P_{\rm in} = 0.8$.

Consider the patterns of the evolution of cellular automata from single point or a small region containing a few individuals who are exposed and infected. The evolution of cellular automata from such an initial condition should provide models for a phenomenon. Figure 6 shows an examples of global density of the exposed and infected generated by the evolution of two-dimensional cellular automata from an initial state containing a few sites with infection, with the parameters being the same as those shown in Fig.6. Through the simulation we reach a conclusion: if we not take any drastic control measure, the disease will persist.

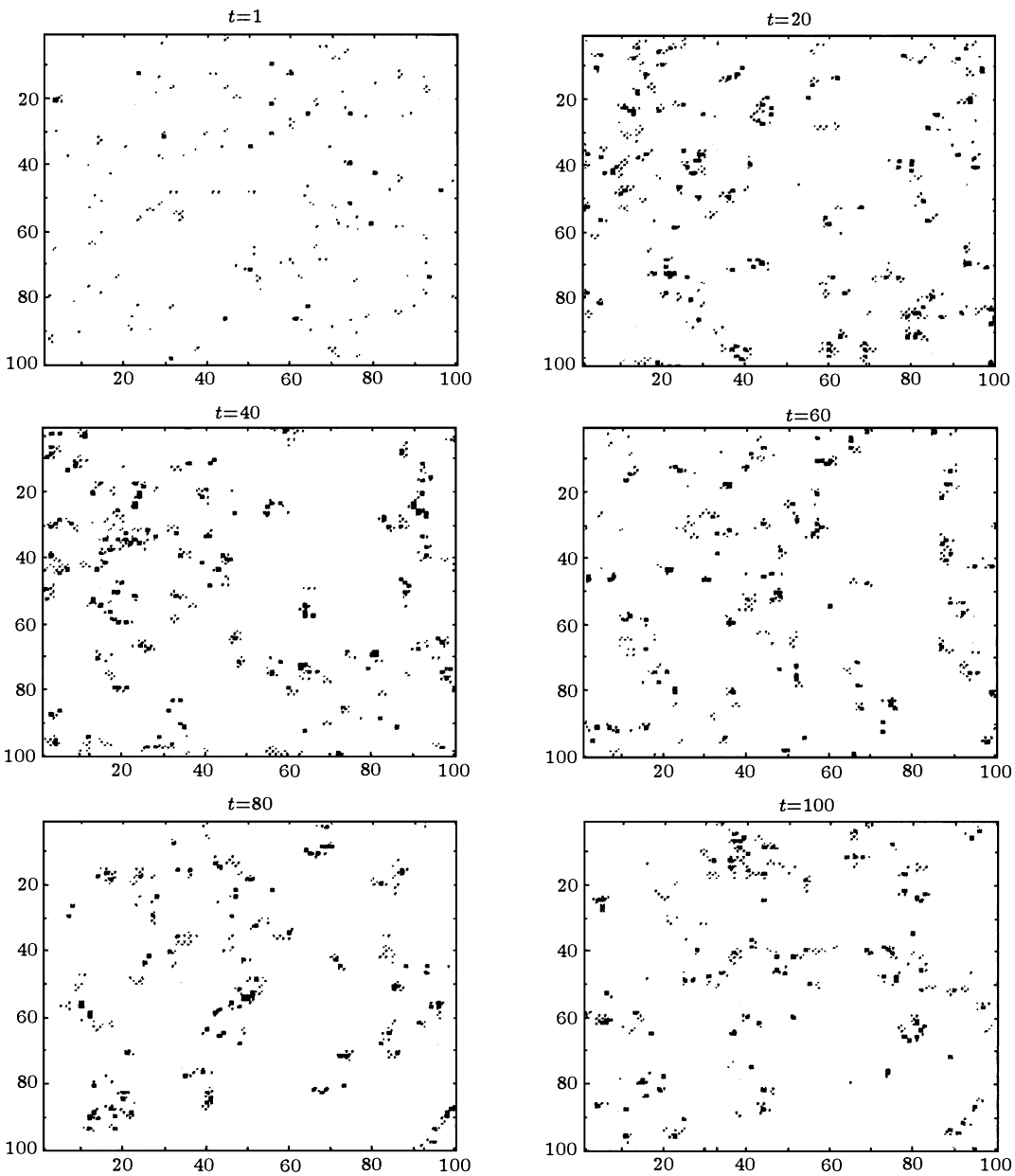


Fig.5. Examples of the evolution of the epidemics states from disordered initial states. White is for the susceptible or recovers, black is for infections, the grey for the exposed, where $P_{\rm e} = 0.4$, $P_{\rm in} = 0.5$, $P_{\rm id} = 0.004$, $P_{\rm ir} = 0.6$, $\eta^P = 0.04$. Here the coordinate plane represents the positions of the susceptible, exposed and infected individuals. The x-axis is denoted by i, the y-axis by j.

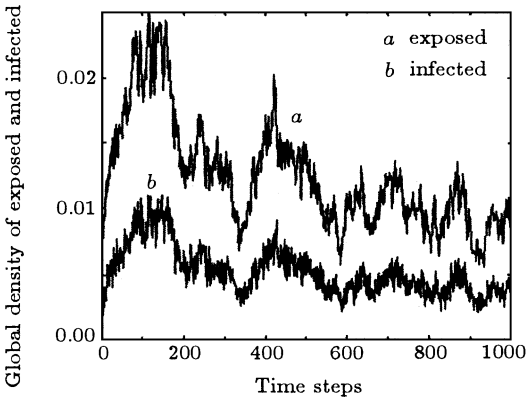


Fig.6. Example of global density from different initial conditions where 25 individuals are exposed and 10 individuals are infected.

Now we discuss the case where the parameters i.e. P_{id} and η^P are changed. When the birth rate approximates to the mortality, i.e. $P_{\mathrm{id}} \to 0$, then the global density series of the exposed and infected generated by evolution from several different disordered initial states, according to this model, are almost qualitatively similar to those under the condition of the same parameters, though the simulation indicates that if $\eta^P > \tau_0$ (the τ_0 approximately equals to 0.07) the global density series of the exposed and infected reach the same value in the limit. Figure 7 illustrates this case.

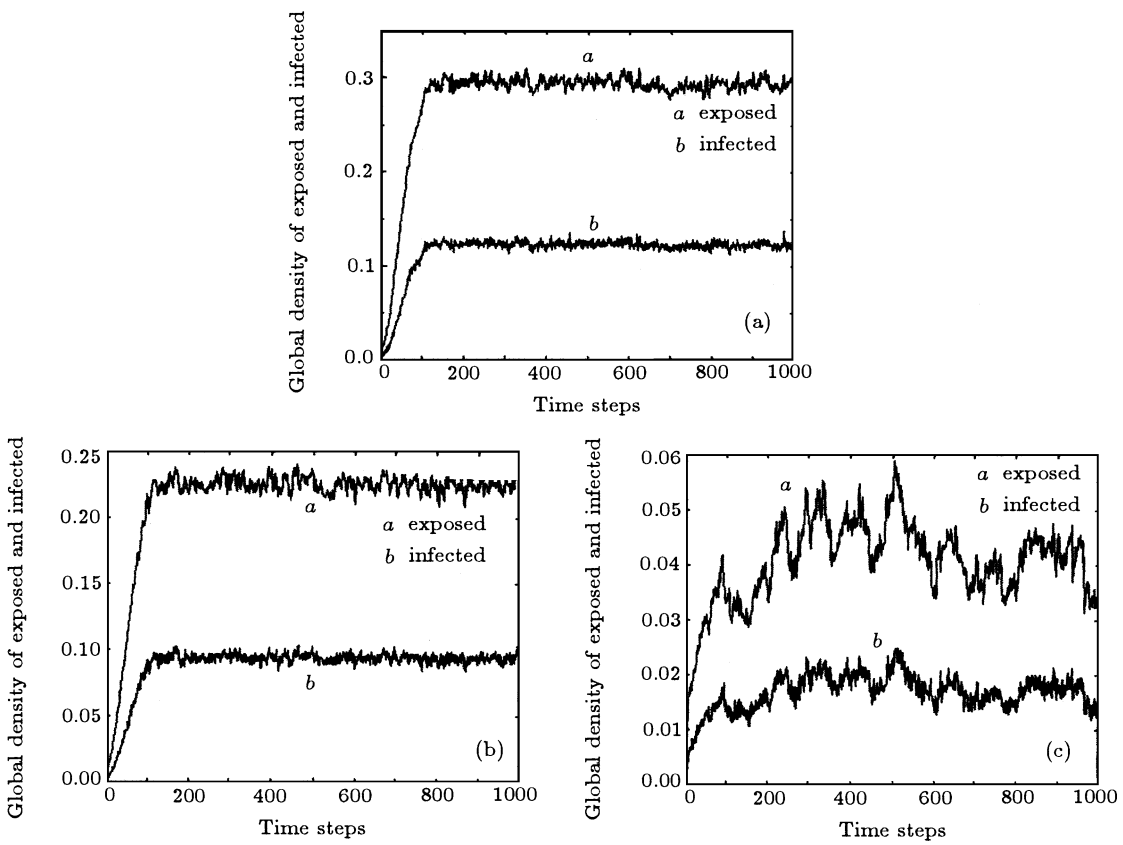


Fig.7. Global density time-series of the infected and exposed with $P_{\mathrm{e}} = 0.4$, $P_{\mathrm{in}} = 0.5$, $P_{\mathrm{id}} = 0.00001$. (a) with $\eta^P = 0.8$. (b) with $\eta^P = 0.4$. (c) with $\eta^P = 0.06$. Here the initial states are the same as those in Fig.3.

6.2. The effects of segregation on the exposed and infection

6.2.1. The effects of segregation on infection

When considering only the effect of segregation on the infection, we need to add the local rule:

If $Z_{c(i,j)}(t) \in \varepsilon_1$, then $Z_{c(i,j)}(t+1) \in \varepsilon_2$ with probability $P_{\varepsilon_1 \to \varepsilon_2}$.

Here the state of ε_2 ($\varepsilon_2 = 6$) is segregation, so no disease spreads to the susceptible. Figure 8 describes the case where the infected individuals are partly segregated ($P_{\varepsilon_1 \to \varepsilon_2} = 0.8$), and Fig.9 is for the case of $P_{\varepsilon_1 \to \varepsilon_2} = 1$, in which the parameters are the same as those in Fig.8, but for $P_{\varepsilon_1 \to \varepsilon_2}$. Figure 8 and Figure 9 demonstrate that the segregation has a great effect on controlling the disease spreading.

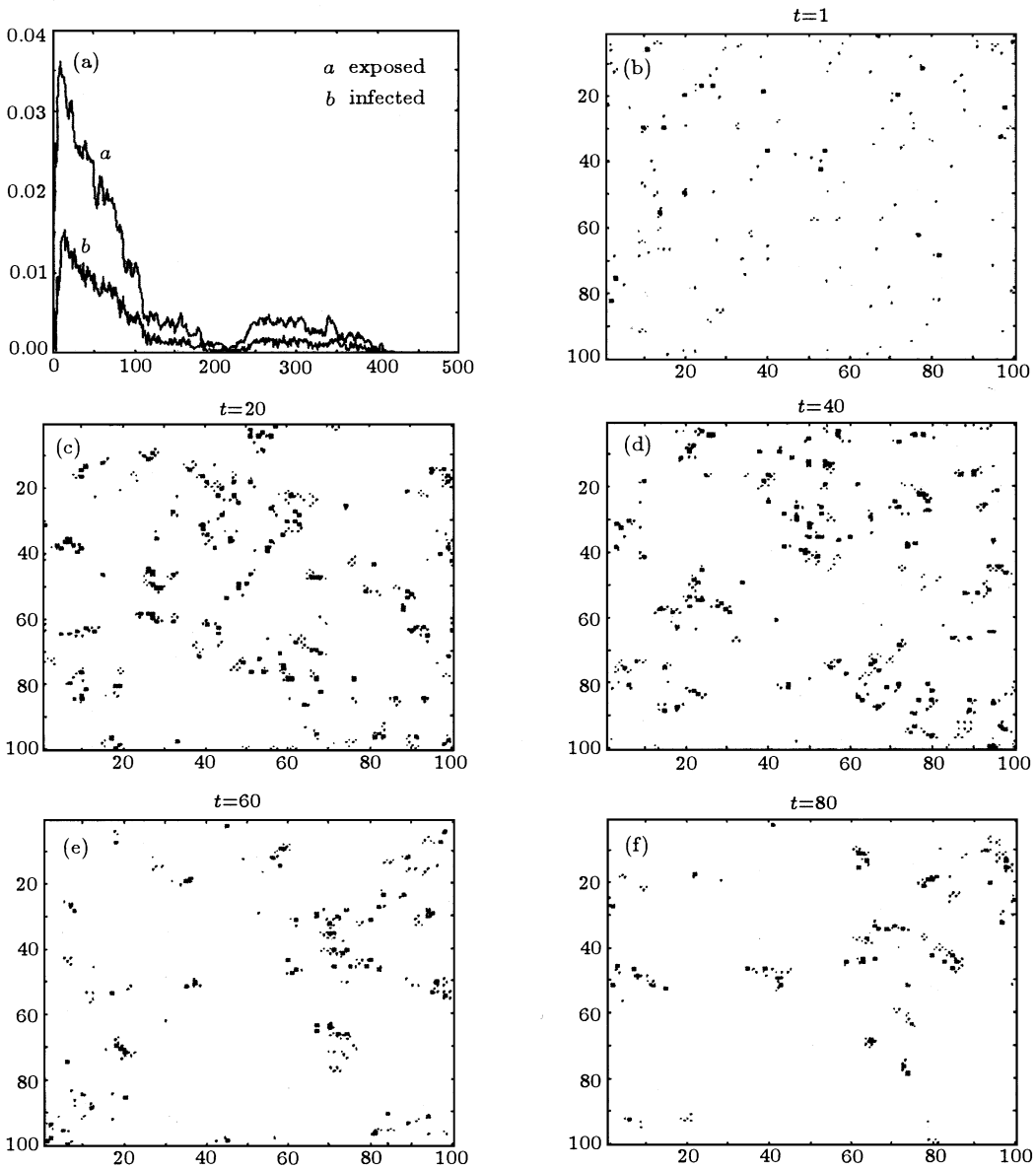


Fig.8. The white represents the susceptible or recovers, the black the infected, the grey the exposed. $P_{\rm id} = 0.004$, $P_{\rm ir} = 0.6$, $\eta^P = 0.004, P_{\varepsilon_1 \to \varepsilon_2} = 0.8$. (a) The global density time-series of the infected and exposed with $P_{\rm e} = 0.4$ and $P_{\rm in} = 0.5$. (b)–(f) show the evolution with time. Here the coordinate plane represents the positions of the susceptible, exposed and infected individuals. The x-axis is denoted by i, the y-axis by j.

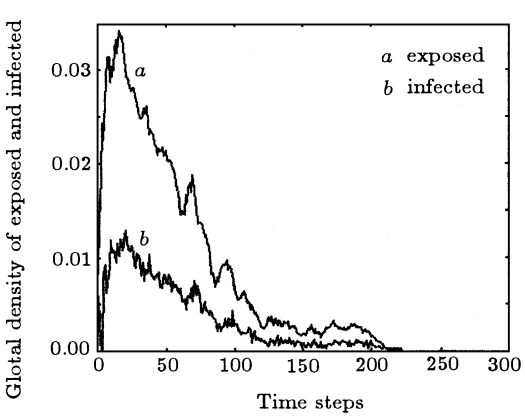


Fig.9. Example of global density time-series of the exposed and infected, where $P_{\varepsilon_1 \to \varepsilon_2} = 1$.

6.2.2. The effects of segregation on the infected and exposed

If we segregate the exposed and the infected, we need to add the spreading rule:

If $Z_{c(i,j)}(t) \in E$ and $Z_{c(i,j)}(t) \overline{\in} e_4$, then $Z_{c(i,j)}(t+1) = e_4$ with probability $P_{\rm e \to seg}$.

Where e_4 is the state segregated from the exposed. Here it should be pointed out that e_1, e_2 and e_3 are not the states segregated from the exposed. In this case, the computer simulation results are shown in Fig.10.

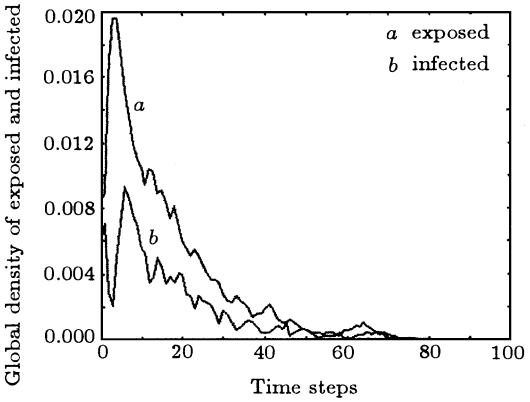


Fig.10. Global density time-series of the infected and exposed with the same parameters as those in Fig.9, but for $P_{\varepsilon_1 \to \varepsilon_2} = 0.4, P_{\mathrm{e} \to \mathrm{seg}} = 0.8$.

7. Discussion and conclusions

By analysing the numerical simulation results we know that if we not take any control measure for the epidemic, as $t \to \infty$, the global density time-series of the exposed and the density time-series of the infected will fluctuate near a positive equilibrium point; furthermore, the disease will be persistent. When the P_{in} equals 0.4 and P_{e} equals 0.5, the maximum value of the infections approximates 0.05, and the positive equilibrium point of the infections approximates 0.005. The classes of the exposed have the same characteristics; however, the maximum value of the exposed approximates 0.05, and the positive equilibrium point of fluctuation approximates 0.01 as shown in Fig.3. Furthermore, the increase of the P_{in} and P_{e} will lead to the increase of the positive equilibrium point of fluctuation as can be seen in Figs.3 and 4. When the infected individuals are partly segregated, the disease will become extinct, but slowly. Figure 8(a) shows the case where the $P_{\varepsilon_1 \to \varepsilon_2}$ equals 0.8. When the infected individuals are completely segregated, the disease will quickly become extinct, and the maximum value for the exposed just approximates 0.035, and the maximum value for the infected just approximates 0.015, at the same time if we segregated the exposed individuals, then the extinction of the disease will speed up (see Fig.10). Finally, from Fig.7 we can see there exists a constant τ_0, and when $\eta^P \geq \tau_0$ the global density series of the exposed and infected attain the same value, which has been addressed from the theorem and will be described in our later publication. Otherwise the density series of the exposed and infection will fluctuate near a positive equilibrium point, when the constant for the immunized is less than the τ_0.

This model is also used to investigate the effect of segregation measure on the epidemic. We believe that the approach introduced in this paper will be also applicable to other epidemic models beyond the SEIRS type and we intend to address this question in future publication.

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