Vol 15 No 6, June 2006
1009-1963/2006/15(06)/1248-09

Chinese Physics

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A cellular automata model of epidemics of a heterogeneous susceptibility*

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(Received 2 November 2005; revised manuscript received 7 March 2006)

In this paper we present a model with spatial heterogeneity based on cellular automata (CA). In the model we consider the relevant heterogeneity of host (susceptible) mixing and the natural birth rate. We divide the susceptible population into three groups according to the immunity of each individual based on the classical susceptible–infected–removed (SIR) epidemic models, and consider the spread of an infectious disease transmitted by direct contact among humans and vectors that have not an incubation period to become infectious. We test the local stability and instability of the disease-free equilibrium by the spectrum radii of Jacobian. The simulation shows that the structure of the nearest neighbour size of the cell (or the degree of the scale-free networks) plays a very important role in the spread properties of infectious disease. The positive equilibrium of the infections versus the neighbour size follows the third power law if an endemic equilibrium point exists. Finally, we analyse the feature of the infection waves for the homogeneity and heterogeneous cases respectively.

Keywords: cellular automata, epidemic spreading, SIR model, spatial heterogeneity, model evolution
PACC: 0550, 0250

1. Introduction

Any micro-organism or bacterium that can cause the infectious disease outbreaks is called pathogen. According to the statistics in 2000, there are already known 1709 kinds of pathogens that can cause the human body to catch a disease. The infectious disease is such a disease that live pathogens can reproduce in great quantities and spread from one host individual to another by direct or indirect contact, such as tuberculosis, vibrio cholerae, acquired immunodeficiency syndrome (AIDS), severe acute respiratory syndrome (SARS), syphilis and so on. There has been a great deal of discussion on the mechanisms of the 'emergence' of the these diseases. For example, recently the epidemic of syphilis across the United States has been studied by Nicholas *et al.*[1] Infectious disease accounts for 29 out of the 96 major causes of human morbidity and mortality listed by the World Health Organization and the World Bank[2] and 25% of global deaths (over 14 million deaths annually, WHO 2000).

Over the last few years, although a great deal of epidemic research has been done on the susceptible–infective–removed (SIR) type, susceptible–infected–susceptible (SIS) type and susceptible–exposed–infected–removed (SEIR) type by using an ordinary differential equations (ODE) model,[3−6] little research has been done on the spatial heterogeneity epidemic. James and Li[7] have performed a qualitative analysis by utilizing an appropriately chosen Liyapunov function, showing that the infection-free equilibrium of the heterogeneity susceptibility (HS) SIR model is globally asymptotically stable.

In Ref.[8], Ahmed and Agiza investigated the susceptible–infected–removed–susceptible (SIRS) model by one-dimensional cellular automata (CA), and gained a probability equilibrium point by computer simulation. But they did not give mathematical analysis of the stability. We have studied the infection disappearing in an isolable strategy for SEIRS models by two-dimensional CA.[9] We obtained a similar result to that from the ODE model of SEIRS.[10] Although useful insights have been gained into the dynamics,[11−13] more efforts are still needed to understand how the spatial heterogeneity and heterogeneity individual affect persistence or extinction across a va-

*Project supported by the National Natural Science Foundation of China (Grant No 10471040).
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riety of population dynamical regimes.

In fact, there are many factors which cause individuals to have very great difference in the antibody for the disease. This leads the susceptibilities to have different abilities immune from the pathogens, and each healthy individual can be infected if, among his nearest neighbours, somebody is infected with, for example, the syphilis.[1] We know that repeated epidemics of primary and secondary syphilis infection in the United States 50 years previously had been attributed to social and behavioural changes.[1] Reaction–diffusion models and other models that neglect individual interactions may be less effective in predicting the behaviour of a spatial system models.[14] Hiebeler described a generalization of the pair approximation,[15] where the longer-range correlations are taken into account, and an other moment-closure approximation has been developed.[16] In this article we develop a spatial heterogeneity (SH) model by using the mean-field method with the neighbour size of the individuals, and use the symbols α_i to denote the hazard of infection for susceptible individual in the ith population. We also consider the individual heterogeneity and the SH in this model. When we address the SH case δ is assumed variable (the δ means the neighbour structure of the cell or the degree distribution scale-free networks).

2. Heterogeneity and model

We divide the susceptibility population into three groups according to the immunity of each individual: high immunity, medium immunity and low immunity that are denoted by S_1, S_2 and S_3, respectively. The susceptibles between different groups are infected at different rates. The infectors are either completely removed or isolated, or have full immunity or die after they are recovered in the SIR model. Therefore, each cell within the system may exist in five states.

We suppose that the disease spreads by direct contact between a susceptible individual and an infector (or more infectors). At each time step, the infected transmission from the pathogen to the individual is of the S_1 type, S_2 type and S_3 type, which are denoted by symbols α_1, α_2 and α_3 respectively, when one or more individuals are infected among nearest neighbours. Meanwhile, an infected individual may recover at a rate μ. Some disease may cause so many deaths that the population is influenced. So it is necessary to take the birth and death rates into account. Here, we introduce two mortalities: d that is due to the infectious disease, and ε that stems from the other factors. The b means the birth rate, which will be used later.

We treat the individuals as being spatially distributed on the two-dimensional loop with J ($J \gg 1$) sites, or J nodes in the plane where some points of contacts link up with each other. We topologically map the loop and complex network (or random network)[17,18] as a finite plane L^2 including J sites; this configuration can describe a system involving J individuals. Each site of L^2 is empty or occupied by at most one individual in this system.

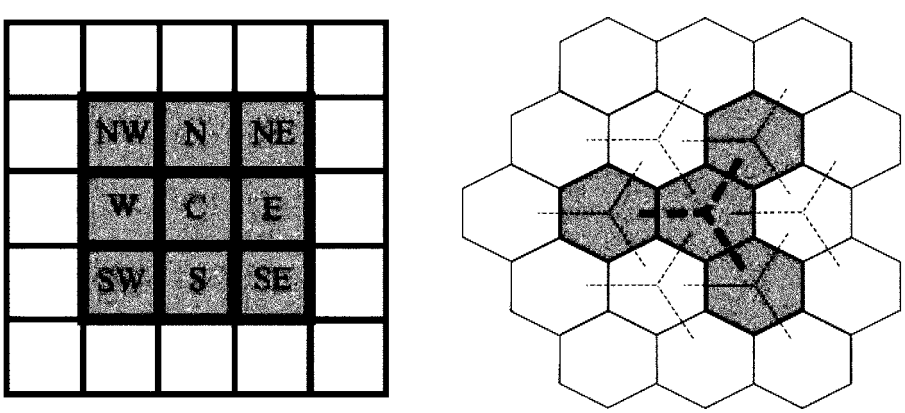


Fig.1. Two different kinds of neighbourhood of the individuals, where the grey cells represent the size of the contact neighbours.

The state configuration of the system at time t can be described by a set of numbers, which are denoted by $Z(t)$ as follows:

$$Z(t) = \{z_{1,1}(t), z_{1,2}(t), \cdots, z_{2,1}(t), z_{2,2}(t), \cdots, z_{i,j}(t)\},$$

$i, j \in N$. Here, $z_{i,j}(t)$ refers to the state of the site (i, j) at time t. We assign the following numbers to each site: –1, 0, 0.5, 1, 2 or 3, where "–1" represents the empty state. For any time t and site, there are $z_{i,j}(t) \in \{-1, 0, 0.5, 1, 2, 3\}$. In the system, each site can change its state at a certain rate (according to the states of the neighbour sites). An empty site can give birth to a susceptible offspring at a rate of b. We use the set of $\mathbf{X} = \{0, 0.5, 1\}$ to represent the susceptible, where the 0, 0.5 and 1 represent S_1, S_2 and S_3 respectively. Therefore, if the site (i, j) is susceptible at time t, then we have $z_{i,j}(t) \in \mathbf{X}$. States 2 and 3 represent the infected and the recovered respectively, i.e. $z_{i,j}(t) = 2$ represents that the individual of site (i, j) is infected at time t and $z_{i,j}(t) = 3$ represents that the individual of site (i, j) recovers at time t. We assume each occupied site can be annihilated at a rate of ε (natural mortality). An infected individual can be cured at a rate of μ or die at a rate of d due to this infectious disease. If the occupied site is annihilated, its state changes to –1 at the next time. Of course, each site can also maintain its state. We define $n_{i,j}(z, t)$ as the number of nearest neighbours of site (i, j) in the state z at time t, where $z \in \{-1, 0, 0.5, 1, 2, 3\}$. For example, nearest neighbours are shown in Fig.1, where the grey lattices represent nearest neighbours of the central cell. The rule we can give is similar to that in Ref.[9] as shown below.

$0 \longrightarrow 2$	at rate α_1	$0.5 \longrightarrow -1$	at rate ε
$0.5 \longrightarrow 2$	at rate α_2	$0 \longrightarrow -1$	at rate ε
$1 \longrightarrow 2$	at rate α_3	$-1 \longrightarrow 0$	at rate $p_1 b$
$2 \longrightarrow 3$	at rate μ	$-1 \longrightarrow 0.5$	at rate $p_2 b$
$2 \longrightarrow -1$	at rate $d + \varepsilon$	$-1 \longrightarrow 1$	at rate $p_3 b$
$1 \longrightarrow -1$	at rate ε	$3 \longrightarrow 0, 0.5, 1$	at rate ω
$3 \longrightarrow -1$	at rate ε		

In the above expression, the parameters are all non-negative. We assume that ε is relatively very small. It is not difficult to see that if $\omega \neq 0$, and α_2, α_3, p_2 and p_3 equal 0, this model turns into the SIRS model;[8,19] if $\omega = 0$, and α_2, α_3, p_2 and p_3 also equal 0, the model turns into the SIR model that has been widely studied[20,21] by cellular automata.

2.1. The mean-field approximation on regular lattice

Mean-field approximation is the simplest and most widely used cluster approximation in spatial problems. It is assumed that the states of any two individuals on the lattice are independent of each other, in other words, there exists the perfect mixing among the susceptible, the infector and the recovered. Despite the assumption made in the mean-field approximation being similar to that in the differential equation model, mean-field approximation is significantly different from differential equation model in that their mixing parameter can be a probabilistic variable. Unlike differential equation models where either all individuals diffuse or none at all, the decision to move around the mean-field is independently made among individuals. Mean-field approximation usually compares with the pair, the hybrid pairwise and the squarewise methods.[22] In this paper we give the model by using the mean-field method based on Ref.[15, 23] on a regular lattice, with the spatial heterogeneity fluctuation taken into consideration. We use the density $y(t)$ to replace the number of the infected individuals, and $x_i(t)$ to replace S_k individuals at time t, where the $x_i(t), y(t) \in [0, 1]$. Then

$$x_1(t) = \frac{N(0,t)}{J}, \quad x_2(t) = \frac{N(0.5,t)}{J},$$
$$x_3(t) = \frac{N(1,t)}{J}, \quad y(t) = \frac{N(2,t)}{J}. \tag{1}$$

If we use $H(t)$ to represent the global density of the system, then $H(t)$ can be written as

$$H(t) = \frac{N(t)}{J}, \tag{2}$$

where $N(t)$ is the total number of the individuals on the L^2 at time t, so $N(t) = \sum_{z=0,0.5,1,2,3} N(z, t)$.

We know that the α_k represents the probability of effective transmission of the disease between the susceptible of S_k type and any infector in a sampling interval. So $1-\alpha_k$ is the probability to avoid the transmission and remain S_k susceptible. For the S_k type, the probability that a susceptible avoids becoming infected by all of his neighbours with $n(2, t)$ infected individuals in the sampling interval is

$$q_k = (1 - \alpha_k)^{n(2,t)}, \tag{3}$$

where $n(2, t)$ is the number of the infected within nearest neighbours of the susceptible at time t. Here we denote the total number of the occupied sites within nearest neighbours by δ. The definition concerning δ

can be found in Ref.[20]. So for the S_k, the probability for the susceptible individual to gain the pathogen from its neighbours can be written as

$$1 - q_k = 1 - (1 - \alpha_k)^{n(2,t)}. \tag{4}$$

We assume that the inputs of the S_1 group, S_2 group and S_3 group respectively are $p_1 b$, $p_2 b$ and $p_3 b$, where $p_1 + p_2 + p_3 = 1$. The map of explaining the model is shown in Fig.2.

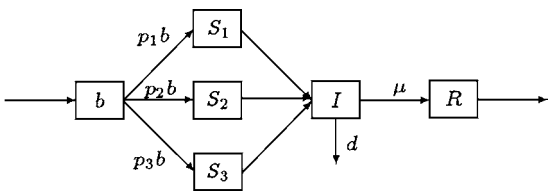


Fig.2. Schematic flow diagram of transmission model.

Therefore, the number of the susceptible in the next sampling interval $t+1$ will be the number of the susceptible individuals remaining at time t. Adding the input to the each type, we have

$$x_1(t+1) = (1 - H(t))p_1 b + x_1(t)(1 - K_1) - \varepsilon x_1(t), \tag{5a}$$

$$x_2(t+1) = (1 - H(t))p_2 b + x_2(t)(1 - K_2) - \varepsilon x_2(t), \tag{5b}$$

$$x_3(t+1) = (1 - H(t))p_3 b + x_3(t)(1 - K_3) - \varepsilon x_3(t), \tag{5c}$$

where the $K_k = \sum_{i=0}^{\delta} \binom{\delta}{i} y(t)^i (1 - y(t))^{\delta - i}[1 - (1 - \alpha_k)^i], k = 1, 2, 3$. From Eq.(2), we know that $H(t) = x_1(t) + x_2(t) + x_3(t) + y(t) + z(t)$. Here $1 - H(t)$ is the expression of the density of empty sites.

We suppose that those susceptible individuals leaving the susceptible type enter entirely into the infected type. Furthermore, we assume that the infected individual cure rate is μ, which is related to the infection period τ by $\mu \approx \tau^{-1}$. Then

$$\begin{aligned} y(t+1) =& (1 - \mu - d - \varepsilon)y(t) + x_1(t)K_1 \\ &+ x_2(t)K_2 + x_3(t)K_3. \end{aligned} \tag{6}$$

Since the infected individuals leaving the infected type enter entirely into the recovered type, then

$$z(t+1) = z(t) + \mu y(t) - \varepsilon z(t). \tag{7}$$

If we use $x(t)$ to represent the density of the total susceptibility at time t, then

$$x(t) = x_1(t) + x_2(t) + x_3(t). \tag{8}$$

From Eqs.(5a)–(7), this model may be described by a set of nonlinear discrete equations as follows:

$$\begin{aligned} x_1(t+1) &= (1 - x_1(t) - x_2(t) - x_3(t) - y(t) - z(t))p_1 b + x_1(t)(1 - \alpha_1 y(t))^{\delta} - \varepsilon x_1(t), \\ x_2(t+1) &= (1 - x_1(t) - x_2(t) - x_3(t) - y(t) - z(t))p_2 b + x_2(t)(1 - \alpha_2 y(t))^{\delta} - \varepsilon x_2(t), \\ x_3(t+1) &= (1 - x_1(t) - x_2(t) - x_3(t) - y(t) - z(t))p_3 b + x_3(t)(1 - \alpha_3 y(t))^{\delta} - \varepsilon x_3(t), \\ y(t+1) &= (1 - \mu - d - \varepsilon)y(t) + x_1(t)[1 - (1 - \alpha_1 y(t))^{\delta}] + x_2(t)[1 - (1 - \alpha_2 y(t))^{\delta}] + x_3(t)[1 - (1 - \alpha_3 y(t))^{\delta}], \\ z(t+1) &= z(t) + \mu y(t) - \varepsilon z(t). \end{aligned} \tag{9}$$

From Eqs.(1) and (2) it is seen that $x(t)$, $y(t)$ and $z(t)$ represent the density of the susceptibility, the infected and the recovered at time t respectively. So

$$x(t) + y(t) + z(t) = H(t) \leq 1. \tag{10}$$

In Eq.(9), let $x_1(t) = x_1(t+1) = x_1$, $x_2(t) = x_2(t+1) = x_2$, $x_3(t) = x_3(t+1) = x_3$, $y(t) = y(t+1) = y$ and $z(t) = z(t+1) = z$, then the fixed point will satisfy the following equations:

$$\begin{aligned} x_1 &= (1 - x_1 - x_2 - x_3 - y - z)p_1 b + x_1(1 - \alpha_1 y)^{\delta} - \varepsilon x_1, \\ x_2 &= (1 - x_1 - x_2 - x_3 - y - z)p_2 b + x_2(1 - \alpha_2 y)^{\delta} - \varepsilon x_2, \\ x_3 &= (1 - x_1 - x_2 - x_3 - y - z)p_3 b + x_3(1 - \alpha_3 y)^{\delta} - \varepsilon x_3, \\ y &= (1 - \mu - d - \varepsilon)y + x_1[1 - (1 - \alpha_1 y)^{\delta}] + x_2[1 - (1 - \alpha_2 y)^{\delta}] + x_3[1 - (1 - \alpha_3 y)^{\delta}], \\ z &= z + \mu y - \varepsilon z. \end{aligned} \tag{11}$$

We obtain the fixed point solutions, i.e. disease-free equilibrium I_0 as follows:

$$I_0 := \left\{ x_1 = \frac{p_1 b}{\varepsilon + b}, x_2 = \frac{p_2 b}{\varepsilon + b}, x_3 = \frac{p_3 b}{\varepsilon + b}, y = 0, z = 0 \right\}. \tag{12}$$

Performing Taylor expansion to power series of Eq.(11) and keeping only the first two terms, then we obtain

$$\begin{aligned}
x_1 &= (1 - x_1 - x_2 - x_3 - y - z)p_1 b + x_1(1 - \delta\alpha_1 y) - \varepsilon x_1, \\
x_2 &= (1 - x_1 - x_2 - x_3 - y - z)p_2 b + x_2(1 - \delta\alpha_2 y) - \varepsilon x_2, \\
x_3 &= (1 - x_1 - x_2 - x_3 - y - z)p_3 b + x_3(1 - \delta\alpha_3 y) - \varepsilon x_3, \\
y &= (1 - \mu - d - \varepsilon)y + \delta\alpha_1 x_1 y + \delta\alpha_2 x_2 y + \delta\alpha_3 x_3 y, \\
z &= z + \mu y - \varepsilon z.
\end{aligned} \tag{13}$$

From the last equation in Eq.(13), we have

$$z = \frac{\mu}{\varepsilon} y. \tag{14}$$

Hence, Eq.(13) can be written as follows:

$$\begin{aligned}
\delta\alpha_1 x_1 y + \varepsilon x_1 &= (1 - x_1 - x_2 - x_3 - \lambda y)p_1 b, \\
\delta\alpha_2 x_2 y + \varepsilon x_2 &= (1 - x_1 - x_2 - x_3 - \lambda y)p_2 b, \\
\delta\alpha_3 x_3 y + \varepsilon x_3 &= (1 - x_1 - x_2 - x_3 - \lambda y)p_3 b, \\
\alpha_1 x_1 + \alpha_2 x_2 + \alpha_3 x_3 &= \frac{\gamma}{\delta},
\end{aligned} \tag{15}$$

where $\lambda = 1 + \dfrac{\mu}{\varepsilon}$ and $\gamma = \mu + d + \varepsilon$. From Eq.(15) we can obtain

$$y^* = \frac{b - (b + \varepsilon)x^*}{\gamma + \lambda b}, \tag{16}$$

where $x^* = x_1^* + x_2^* + x_3^*$. Substituting the expression (16) in to Eq.(15) yields

$$x_1^* = \frac{[(1 - x^*)(\gamma + \lambda b) - \lambda(b - (b + \varepsilon)x^*)]p_1 b}{\delta\alpha_1(b - (b + \varepsilon)x^*) + \gamma\varepsilon + \lambda b\varepsilon},$$

and through computing we discover x_1^* to monotonically and gradually increase with ε. Generally, we only consider the death rate due to the disease, so we let $\varepsilon = 0$, and have

$$x_1^* = \frac{\gamma p_1}{\delta\alpha_1}. \tag{17}$$

Obviously, $x_1^* > 0$. Similarly $x_2^* > 0$, and $x_3^* > 0$, i.e. the system has a positive fixed point.

Here the asterisk denotes positive equilibrium point. We can easily obtain $z^* = \dfrac{\mu}{\varepsilon} y^*$, when $\varepsilon \neq 0$. In Section 4 we test the positive equilibrium of the infection versus the neighbour size δ of the cell with computer simulation.

3. Mathematical properties for the model

At the end of the epidemic we may investigate its effect by means of the difference between the initial and final sizes of the population and neighbour sizes (or the degree distribution of the scale-free network) δ. In order to analyse the stability condition of the fixed point I_0 in the mean-field model, we linearize Eq.(9) as follows:

$$\begin{aligned}
x_1(t+1) &= (1 - x_1(t) - x_2(t) - x_3(t) - y(t) - z(t))p_1 b + x_1(t)(1 - \delta\alpha_1 y(t)) - \epsilon x_1(t), \\
x_2(t+1) &= (1 - x_1(t) - x_2(t) - x_3(t) - y(t) - z(t))p_2 b + x_2(t)(1 - \delta\alpha_2 y(t)) - \epsilon x_2(t), \\
x_3(t+1) &= (1 - x_1(t) - x_2(t) - x_3(t) - y(t) - z(t))p_3 b + x_3(t)(1 - \delta\alpha_3 y(t)) - \epsilon x_3(t), \\
y(t+1) &= (1 - \mu - d - \epsilon)y(t) + x_1(t)\delta\alpha_1 y(t) + x_2(t)\delta\alpha_2 y(t) + x_3\delta\alpha_3 y(t), \\
z(t+1) &= z(t) + \mu y(t) - \varepsilon z(t).
\end{aligned} \tag{18}$$

3.1. Stability analysis of I_0

From Eq.(12) we know there exists a disease-free equilibrium point I_0 in the deterministic CA model. If we denote the linearized matrix around the fixed point I_0 of Eq.(9) by $\mathcal{J}$ for the fixed point I_0 the Jacobian matrix is

$$\mathcal{J} = \begin{pmatrix} 1-p_1b-\varepsilon & -p_1b & -p_1b & -p_1b\left(1+\frac{\delta\alpha_1}{b+\varepsilon}\right) & -p_1b \\ -p_2b & 1-p_2b-\varepsilon & -p_2b & -p_2b\left(1+\frac{\delta\alpha_2}{b+\varepsilon}\right) & -p_2b \\ -p_3b & -p_3b & 1-p_3b-\varepsilon & -p_3b\left(1+\frac{\delta\alpha_3}{b+\varepsilon}\right) & -p_3b \\ 0 & 0 & 0 & 1-\gamma+\frac{\delta b}{b+\epsilon}\Psi & 0 \\ 0 & 0 & 0 & \mu & 1-\varepsilon \end{pmatrix}. \tag{19}$$

In this matrix, $\Psi = \alpha_1p_1 + \alpha_2p_2 + \alpha_3p_3$ and $\gamma = \mu + d + \varepsilon$. In the simple SIR model, the basic reproductive ratio R_0 is the most fundamental quantity in epidemiology. Unfortunately, $\mathcal{J}$ is a five-order matrix, and it is difficult to obtain the corresponding eigenvalue formula, but we can calculate the eigenvalues of the matrix $\mathcal{J}$ instead. For the stability at I_0, the disease-free equilibrium is locally asymptotically stable. The parameters must satisfy the spectral radius of $\mathcal{J}$ in the inequality $\rho(\mathcal{J}) < 1$. We denote the eigenvalues of matrix $\mathcal{J}$ by λ_1, λ_2, $\cdots$, λ_5. The inequality $\rho(\mathcal{J}) < 1$ indicates the magnitude of the largest eigenvalue to be less than 1, i.e.

$$\max(|\lambda_1(\delta,\epsilon,d,\alpha_1),\cdots|,|\lambda_2(\delta,\epsilon,d,\alpha_1),\cdots|,\cdots) < 1. \tag{20}$$

4. Numerical simulations

In order to highlight some of the properties of the CA model and to compare the results obtained from the model, we give the numerical simulation in this section. The numerical values are chosen to be in a reasonable range, but do not represent true values corresponding to any particular epidemic for the humankind and the terrestrial wild creature species.

In the numerical simulations, some of the parameter values are fixed at baseline values that are given in Table 1. In the numerical simulation we assume that the mortality due to the infectious disease is greater than that resulting from the other factors, i.e. $d > \varepsilon$.

Table 1. Baseline parameter values for the numerical simulation.

parameter	value	meaning
ε	0.0003	Natural mortality rate
b	0.005	Birth rate

First, we give simulations to the stability of the disease-free equilibrium (I_0). The eigenvalues of the 5×5 Jacobian matrix $\mathcal{J}$ are calculated. If $\delta \leq 4$, the spectral radius is less than one; the disease-free equilibrium is locally asymptotically stable. Figure 3 gives the result when $\rho(\mathcal{J}) < 1$. Table 2 gives the values of the spectral radius for several different values of the neighbour size, where $\mu = 0.12$. For $\delta = 6$, the spectral radius is greater than one; the disease-free equilibrium is unstable as shown in Fig.4.

Table 2. Spectral radius ρ of the 5×5 Jacobian matrix for disease-free equilibrium, when $\mu = 0.12$.

the neighbour size	3	4	5	6	7	8
spectral radium ρ	0.9990	0.9990	1.0260	1.0560	1.0860	1.1160

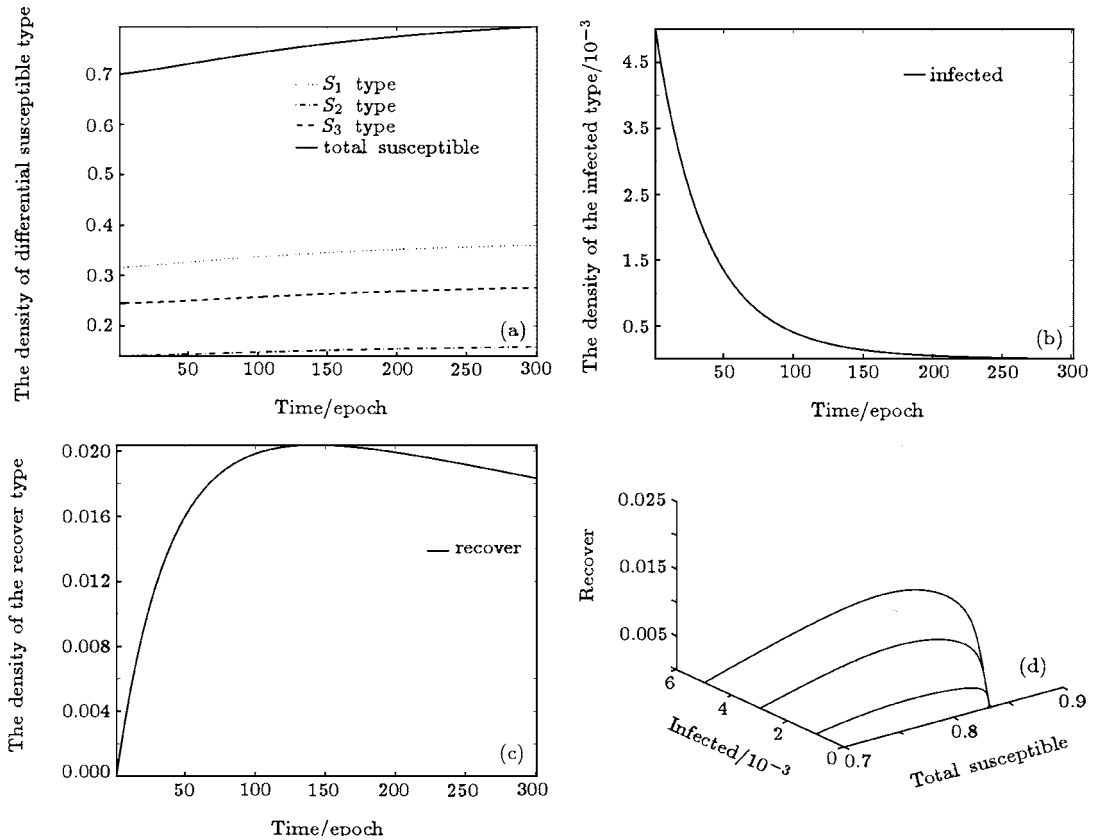


Fig.3. The simulation results for Eq.(9) on condition that $\rho(\mathcal{J})$=0.9990, with parameters $\delta = 4$, $p_1 = 0.45$, $p_2 = 0.2$, $p_3 = 0.35$, $b = 0.005$, $\varepsilon = 0.001$, $d = 0.003$, $\alpha_1 = 0.02$, $\alpha_2 = 0.04$, $\alpha_3 = 0.05$, $y(0) = 0.001$, $\mu = 0.12$, and $H(0) = 0.801$. (a)–(c) show time series of the susceptibles, infected and recovered individuals from t=1 to 300 epoch. (d) shows the phase trajectory for this system.

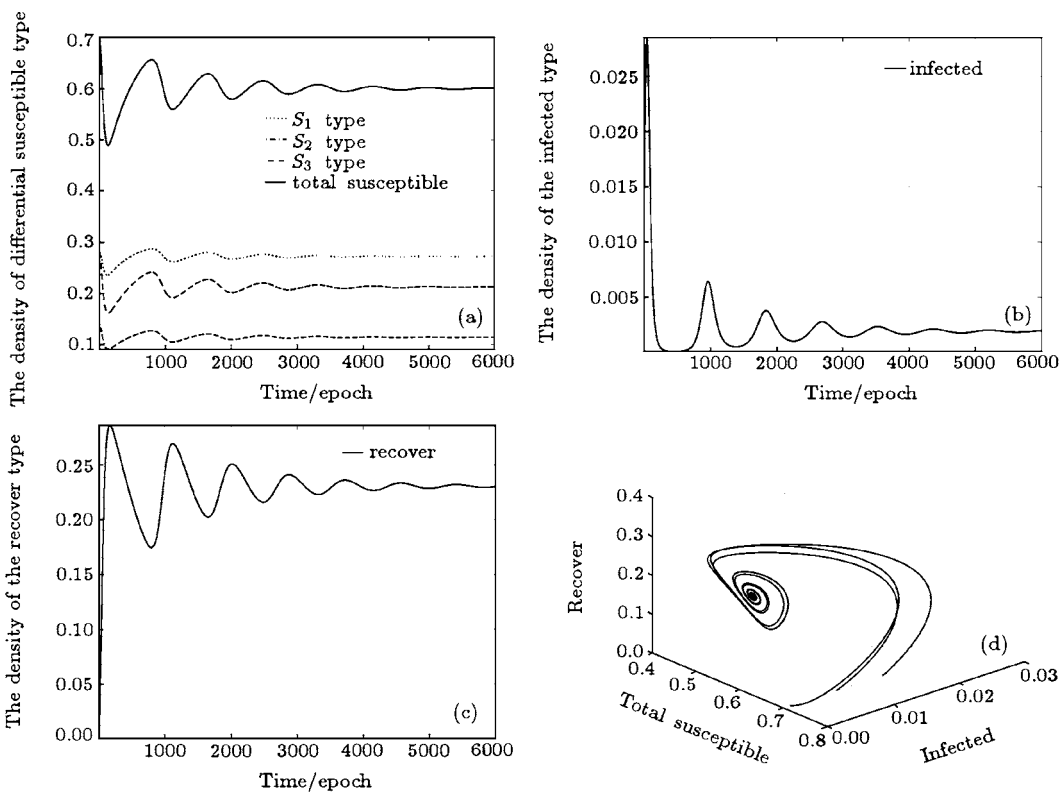


Fig.4. Simulation results of Eq.(9) on condition that $\rho(\mathcal{J})$=1.0560, with parameters $\delta = 6$, $p_1 = 0.4$, $p_2 = 0.2$, $p_3 = 0.4$, $b = 0.005$, $\varepsilon = 0.001$, $d = 0.003$, $\alpha_1 = 0.02$, $\alpha_2 = 0.04$, $\alpha_3 = 0.05$, $y(0) = 0.001$, $\mu = 0.05$ and $H(0) = 0.801$.

To investigate the effects of neighbour size, we fix the other parameters but let δ be variable. We can analyse the model by direct computer simulation. Figure 5 illustrates the results of computer simulation.

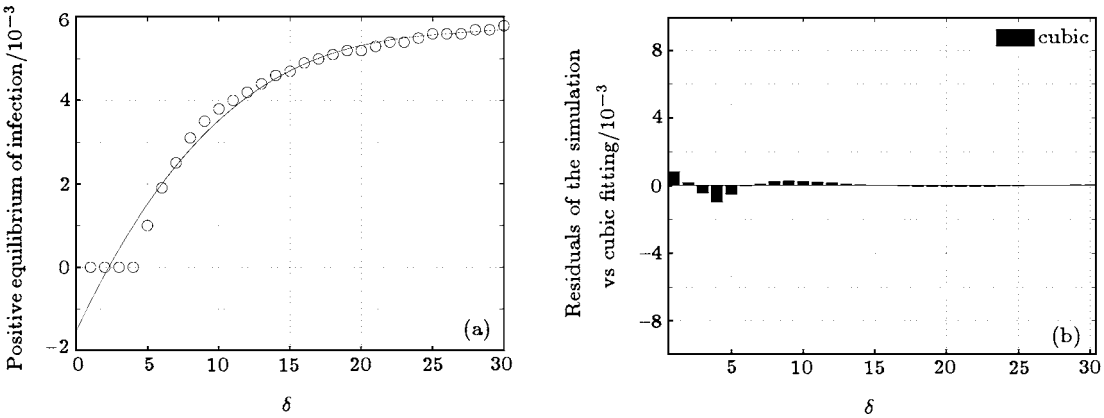


Fig.5. The parameters are the same as those in Fig.3, but for δ. (a) The horizontal axis and vertical axis represent neighbour size of individuals and positive equilibrium of infection respectively. The solid line indicates the estimated level of cubic fitting. The circles show results of computer simulation. (b) The error bars are the same as those in (a).

Figure 5 shows that the neighbour size of the individual is very important in the epidemics spreading. The positive fixed point of the infection follows the third power law vs the neighbour size when $\delta \geq 5$. For $\delta < 5$ the asymptotic density of infected goes to zero, but if $\delta > 5$ it becomes positive.

Figure 6 shows the spatiotemporal spread of the epidemics from point of view of cellular automata. In this figure, outcome is obtained from a lattice with the size 200×200 at $t = 20, 60, 120$ epoch. As we can see, there are different spatial patterns for the heterogeneity and homogeneity even if the initial value and the parameters are the same (the infection rate α_k and infection period $1/\tau$). We suppose there being 100 individuals who are infected and 10 per cent of sites are empty in the system. The localized disease outbreaks are diffusive from the centre.

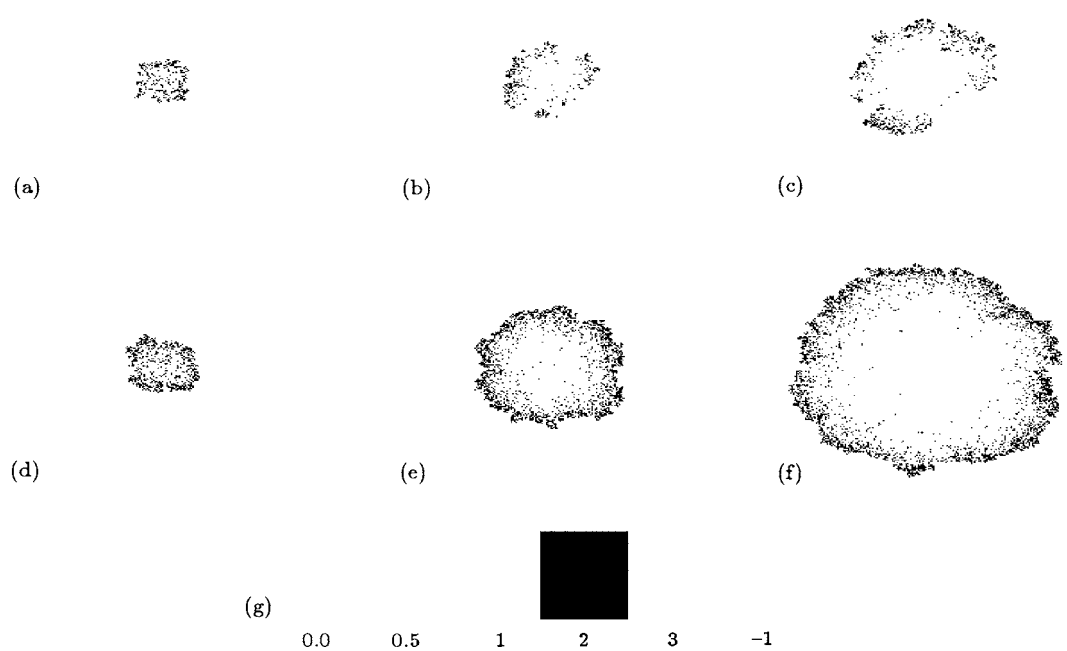


Fig.6. The time evolution of spatial patterns. (a)–(c) show the heterogeneity case and (d)–(e) show the homogeneity case under the same parameters. (g) represents the state of the cell, what the colors represent is as follows: black squares, infected; light grey squares, empty sites.

As we can see, the trait of the spatial patterns is that the infection waves are narrower, and occasionally the wave breaks in the heterogeneity case; Contrarily, under the homogeneity condition these waves are broader and do not easily break. At the same time, we easily discover that the spread of homogeneity is faster than that of heterogeneity, which can cause the spread of the large-scale spatiotemporal patterns.

5. Discussion

The main finding from our simulation is that the positive equilibrium of the disease decreases with the decrease of the contact neighbour size δ. Here we consider the hazard of infection for susceptible individuals is constant, but it is variable[1] in this model for different diseases. For example, we can replace the constant α_i by a time-dependent expression such as $\alpha_i(t) = b_0(1 + \xi \cos 2\pi t)$ for measles,[24] where b_0 is the average contact rate and ξ is the seasonal component. Recently, the pairwise method,[22] the rate equation[23] and the moment methods[16] are used to predict the spatial problem. The finite size complex network is also studied in the epidemic spread by Li *et al*, in which the neighbour size is equal to the degree k.[18] The shape of the neighbours has many configurations, if we extend the goal site (or cluster) to a larger one ($a_1 \times b_1$ block) then there will be more than one possible tiling to the goal site (or cluster). Hiebeler gave the particular discussion in Ref.[15] in this case.

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