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A cellular automata model with probability infection and spatial dispersion*

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In this article, we have proposed an epidemic model based on the probability cellular automata theory. The essential mathematical features are analysed with the help of stability theory. We have given an alternative modelling approach for the spatiotemporal system which is more realistic from the practical point of view. A discrete and spatiotemporal approach is shown by using cellular automata theory. It is interesting to note that both the size of the endemic equilibrium and the density of the individuals increase with the increase of the neighbourhood size and infection rate, but the infections decrease with the increase of the recovery rate. The stability of the system around the positive interior equilibrium has been shown by using a suitable Lyapunov function. Finally, experimental data simulation for SARS disease in China in 2003 and a brief discussion are given.

Keywords: cellular automata, SEIS model, stability, mean-field approximation, spatial epidemic
PACC: 0550

1. Introduction

The foundation of modern mathematical epidemiology, based on the compartment models, was laid in the early 20th century. Since the middle of the 20th century, mathematical epidemiology has been growing exponentially. Various mathematical models have been proposed and analysed. Although epidemic models were studied extensively afterwards, little attention has been paid to the localized processes of pathogen transmission between the susceptibly exposed and infected individuals. In the epidemic model, generally, it is assumed that the population is a continuous entity, and as a result it is often neglected that the population is composed of single interacting individuals. In fact, the spread of disease must be governed by the localized process.[1] The cellular automata (CA) or lattice gas cellular automata (LGCA) is a discrete approach to the time, space, and state. It shows the features of the epidemic model clearly with mathematical analysis and numerical simulations in a very simple way.[2] In a class of spatial epidemic models, it is supposed that the lattice has habitable sites, where no more than one individual can occupy any particular site. Spatially structured pathogen transmission says that the probability that the susceptible host suffers from some disease depends more on the local density of the exposed and infected host than that of the global.[3] However, in most of these models, local host density is treated as an equivalent to the global host density, since every lattice site is occupied with equal probability. The following spatial heterogeneity is generated exogenously, $H(t)$, which represents the spatial density of the host. The variation of the local host density is very important for understanding the role of spatial heterogeneity in the plants and animals communities.[4,5]

Harcourt[6] and Kobayashi[7] have studied an epidemic model on the citrus variegated chlorosis (CVC) by considering spatial dispersion and obtained an interesting negative binomial distribution. Duryea *et al*[8] has studied a susceptibles–infected–susceptibles (SIS) epidemic model with varying population density and obtained the condition under which the pathogen extincts. Similar results have been shown by Mollision[3] and Ahmed and Agiza.[9] Keeling[10] has studied the effects of local spatial structure on epidemiological invasions in an epidemic

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model. Here we consider an susceptibles–exposed–infected–susceptibles (SEIS) epidemic model with infectious force in the exposed and infected period, as in Refs.[11,12]. In this SEIS model we consider the spatial dispersion and obtain the transmission threshold for epidemic persistence, $\mathcal{R}_c$, by using a probability cellular automata and mean-field (MF) approximation. We also study the nature of solution of the system around the trivial equilibrium (0,0) by using Lyapunov function. Finally numerical simulations are performed by the help of experimental data for the SARS disease in China.

2. Analysis of the model

In order to gain the same neighbourhood for each site of the boundary, we treat the plane as a two-dimensional torus with J sites, where $J \gg 1$. The constant J is the carrying capacity of the environment, which is usually determined by the availability of the resources. In more general cases, the neighbourhood for each site k are shown in Figs.1 and 2. Figure 3 shows the same fact with a special black dot neighbourhood for site k. Suppose that the individual dispersion in the sites of the two-dimensional torus for the global state set is denoted by $Z(t)$ at time t. Then we have

$$Z(t) = \{z_1(t), z_2(t), \cdots, z_J(t)\}.$$

In the above global set, $z_k(t)$ denotes the k-th set of local states at time t. Each site of J is empty or occupied by at most one individual. So, in the epidemic each site state belongs to the set $\mathcal{S}$, where $\mathcal{S} = \{\text{susceptible}, \text{exposed}, \text{infected}, \text{empty}\}$. Hence, each $z_k(t)$ belongs to a finite set of elementary states. The recovery from pathogen infection does not retain immunity, so the recovered individuals will again be treated as susceptible individuals.

Let us assume that there exist N individual habitable sites (or cells) at time t and they are denoted by $N(t)$. Each of the N sites contains a single host, and no more than one individual can occupy any particular site. Then there are $J - N(t)$ number of empty sites at time t, with the empty site representing no individual host there. We denote $H(t)$ as the global density for the individuals at time t. Since J is the maximum number of individuals in the system, we have

$$H(t) = \frac{N(t)}{J}, \qquad (H(t) \leq 1). \tag{1}$$

Note that in Eq. (1) $J \geq N(t)$, therefore we have $H(t) \leq 1$. In this model, we assume that the natural birth rate equals its natural death rate and d is the mortality rate due to the infection.

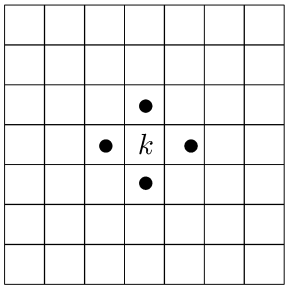


Fig.1. Von Neumann neighbourhood.

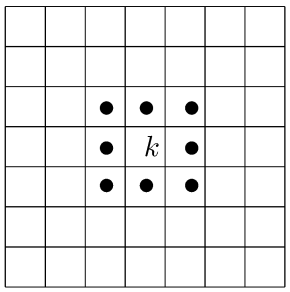


Fig.2. Moore neighbourhood.

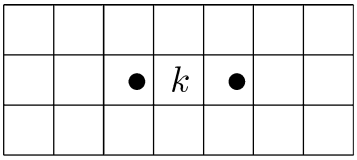


Fig.3. Neighbourhood of k for $r = 1/2$.

If δ represents the maximum number of individuals in the neighbourhood of site k, then the number of sites in δ_k is $\delta = |\delta_k| - 1$, where δ_k is the kth neighbourhood. There is no variation in size of the sites. The local transition rule governs the infection transmission about any site k. Let the mode of transmission of the infection within δ_k be as follows: (i) if the kth site is non-empty, then we can assume that an exposed or infected kth site transmits the pathogen to a susceptible individual within δ_k, and (ii) if the kth site is not infected or exposed, then it acquires the pathogen (infection) from an exposed or infected individual within δ_k. In our model, we assume that δ_k is symmetric. We also assume that the kth site with its centre is like that shown in Figs.1 and 2. There is the same transmission rule for all δ_k. When δ_k is asymmetric,

a host at site k may be infected differently from different neighbouring individuals. Therefore, it will be difficult to discuss the mode of transmission in term of mathematical equations. For the sake of simplicity, we assume that δ_k is symmetric. Now we define the local function f as follows:

$$f :\longrightarrow z_k(t+1) = f(z_k(t), z_{\delta_k}(t)). \tag{2}$$

Note that the spatial heterogeneity dispersion changes while the local density and individual interaction neighbourhoods are changed. The discrete random variable n_k counts the host in δ_k, with $0 \le n_k \le \delta$. Then the mean number of hosts per neighbourhood is given by

$$E\big[n_k(t)\big] = \frac{N(t)\delta}{J} = H(t)\delta. \tag{3}$$

The n_k hosts on δ_k include s susceptibles, e exposed and i infectives; $n_k = s + e + i$. Let α be the probability per discrete time interval, denoting that an exposed on δ_k transmits the disease to the susceptible at site k, β be the probability per discrete time interval, denoting that an infective on δ_k transmits the disease to susceptible at site k, and μ the probability per time interval, denoting that an exposed individual converts to an infected one. (see Fig.4)

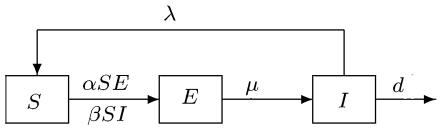


Fig.4. Schematic flow diagram of transmission for SEIS model.

Since the infectives act independently, the susceptible acquires the pathogen in a single time interval with conditional probability $1-(1-\alpha)^e(1-\beta)^i$. The unconditional probability of infecting the local susceptible in a discrete time interval is ε_k, expressed as

$$\varepsilon_k = \sum_{n=1}^{\delta}\sum_{e=1}^{n}\sum_{i}^{n}\Big[1-(1-\alpha)^e(1-\beta)^i\Big] \times \Pr[e_k = e, i_k = i | n_k = n\big] \cdot \Pr\big[n_k = n].$$

3. The approximation of the global density

Mean-field analyses have different forms, here we adopt a 'local-dispersal' approximation.[13] Assume that $S(t)$, $E(t)$ and $I(t)$ are the total susceptible, exposed and infected individuals at time t respectively. Then we have $N(t) = S(t) + E(t) + I(t)$. The global density of the exposed and the infected populations at time t are given by

$$x(t) = \frac{E(t)}{J}, \qquad y(t) = \frac{I(t)}{J}. \tag{4}$$

Thus, the global frequency of the exposed and the infected populations at time t are given by

$$\frac{E(t)}{N(t)} = \frac{E(t)}{H(t)J} = \frac{E(t)}{J}\,\frac{1}{H(t)} = \frac{x(t)}{H(t)}, \tag{5}$$

$$\frac{I(t)}{N(t)} = \frac{I(t)}{H(t)J} = \frac{I(t)}{J}\,\frac{1}{H(t)} = \frac{y(t)}{H(t)}. \tag{6}$$

For a given independence of sites, the probability that a host occupies a given site in δ_k is equal to the global density $H(t)$. The individual interaction acts as an independent event in δ_k, so (e, i) is an independent two-dimensional variable, and is denoted by (ξ, η), where $\xi+\eta \le n_k$. From the transmit rule f, we know that the number of exposed and infected individuals in δ_k has a (three-parameter) binomial distribution with parameters δ, $x(t)$, and $y(t)$. In the following, λ represents the probability (per time interval) that an infective recovers from infection and again becomes an susceptible. In the neighbourhood of δ_k, the exposed and infective at the time between $t+1$ and t are independent. Hence, we can obtain the global mean densities of the exposed and infective at time $t+1$, given as follows:

$$\begin{aligned} x(t+1) = F_1\Big(x(t), y(t)\Big) &= x(t)(1-\mu) + \Big(H(t) - x(t) - y(t)\Big) \\ &\times \sum_{\xi=0}^{\delta}\sum_{\eta=0}^{\delta}\binom{\delta}{\xi}\binom{\delta-\xi}{\eta}x(t)^{\xi}y(t)^{\eta}(1-x(t)-y(t))^{\delta-\xi-\eta}[1-(1-\alpha)^{\xi}(1-\beta)^{\eta}] \\ &= x(t)(1-\mu) + (H(t)-x(t)-y(t))\Big[\sum_{\xi=0}^{\delta}\sum_{\eta=0}^{\delta}\binom{\delta}{\xi}\binom{\delta-\xi}{\eta}x(y)^{\xi}y(t)^{\eta}(1-x(t)-y(t))^{\delta-\xi-\eta} \end{aligned}$$

$$-\sum_{\xi=0}^{\delta}\sum_{\eta=0}^{\delta}\Big(\begin{matrix}\delta\\ \xi\end{matrix}\Big)\Big(\begin{matrix}\delta-\xi\\ \eta\end{matrix}\Big)x(y)^{\xi}y(t)^{\eta}(1-x(t)-y(t))^{\delta-\xi-\eta}(1-\alpha)^{\xi}(1-\beta)^{\eta}\Big]$$

$$= x(t)(1-\mu)+(H(t)-x(t)-y(t))[1-(1-\alpha x(t)-\beta y(t))^{\delta}], \tag{7}$$

and

$$y(t+1)=F_2(x(t),y(t))=y(t)(1-\lambda-d)+\mu x(t). \tag{8}$$

From Eqs.(7) and (8) we can obtain the global densities of the exposed and infective, as follows:

$$\begin{cases} x(t+1)=x(t)(1-\mu)+(H(t)-x(t)-y(t))[1-(1-\alpha x(t)-\beta y(t))^{\delta}],\\ y(t+1)=y(t)(1-\lambda-d)+\mu x(t). \end{cases} \tag{9}$$

This is a two-dimensional nonlinear system. Since $D \ll N(t)$ for not too long a time, we can omit the change of the total of individuals (see Ref.[14]) and let $H(t)=H=$ constant, $x(t+1)=x(t)=x$, and $y(t+1)=y(t)=y$, where the symbol D represents the number of death up to time t. The equilibrium points of the system (9) satisfy the equations

$$\begin{cases} x=x(1-\mu)+(H-x-y)[1-(1-\alpha x-\beta y)^{\delta}],\\ y=y(1-\gamma)+\mu x(t). \end{cases} \tag{10}$$

Now in the above system , we have $\gamma=d+\lambda$. Since $\delta>0$, $0\le x(t)<H$, $0\le y(t)<H$, solving the above equations we have a zeros-fixed point (x_0,y_0):

$$\begin{cases} x_0=0,\\ y_0=0. \end{cases}$$

Now we consider the Jacobian matrix of system (9) at $(0,0)$. From Eqs.(7) and (8) we obtain

$$\frac{\partial F_1}{\partial x(t)}=(1-\alpha x-\beta y)^{\delta}+\alpha\delta(H-x-y)\times(1-\alpha x-\beta y)^{\delta-1}-\mu,$$

$$\frac{\partial F_1}{\partial y(t)}=(1-\alpha x-\beta y)^{\delta}+\beta\delta(H-x-y)\times(1-\alpha x-\beta y)^{\delta-1}-1,$$

$$\frac{\partial F_2}{\partial x(t)}=\mu,\quad \frac{\partial F_2}{\partial y(t)}=1-\gamma.$$

Therefore, the Jacobian Matrix $\boldsymbol{A}$ can be expressed at the disease-free equilibrium $(0,0)$ as follows:

$$\boldsymbol{A}=\begin{pmatrix} 1+H\alpha\delta-\mu & H\beta\delta\\ \mu & 1-\gamma \end{pmatrix}.$$

Then the trace and determinant of the matrix $\boldsymbol{A}$ are given by

$$\mathrm{tr}\boldsymbol{A}=2-\mu-\gamma+H\delta\alpha \tag{11}$$

and

$$\begin{aligned}\det\boldsymbol{A}&=(1-\mu+H\delta\alpha)(1-\gamma)-H\delta\beta\mu\\ &=1-\mu+H\delta\alpha-\gamma+\mu\gamma-H\delta(\alpha\gamma+\beta\mu).\end{aligned} \tag{12}$$

Now denote $\mathcal{R}_{\mathrm{c}}=H\delta(\alpha\gamma+\beta\mu)/\gamma\mu$ as the persister threshold, and then the following theorem holds.

Theorem 1 If $\mathcal{R}_{\mathrm{c}}<1$, then the system (9) is locally asymptotically stable around the disease-free equilibrium $(0,0)$, and unstable if $\mathcal{R}_{\mathrm{c}}>1$.

Proof Let us linearize the system (9) at $(0,0)$, we have

$$\begin{cases} x(t+1)=(1+H\alpha\delta-\mu)x(t)+H\beta\delta y(t),\\ y(t+1)=\mu x(t)+(1-\gamma)y(t). \end{cases} \tag{13}$$

Consider a positive Lyapunov function

$$V=\Big(\frac{\alpha}{\mu}+\frac{\beta}{\gamma}\Big)x+\frac{\beta}{\gamma}y,$$

then the time derivative of this function along the solution of the system (13) we obtain

$$\begin{aligned}\Delta V&=V(t+1)-V(t)\\ &=\Big(\frac{\alpha}{\mu}+\frac{\beta}{\gamma}\Big)[x(t+1)-x(t)]\\ &\quad+\frac{\beta}{\gamma}[y(t+1)-y(t)]\\ &=-\Big(\frac{\alpha}{\mu}+\frac{\beta}{\gamma}\Big)\mu x+\Big(\frac{\alpha}{\mu}+\frac{\beta}{\gamma}\Big)H\delta(\alpha x+\beta y)\\ &\quad-\beta y+\frac{\beta}{\gamma}\mu x\\ &=-\alpha x-\beta y+(\frac{\alpha}{\mu}+\frac{\beta}{\gamma})H\delta(\alpha x+\beta y)\\ &=-(\alpha x+\beta y)(1-\mathcal{R}_{\mathrm{c}})<0.\end{aligned}$$

Again note that $\Delta V = 0$ at the origin $(0,0)$. According to Lyapunov LaSalle Theorem,[15] the origin of the system (9) is locally asymptotically stable.

Now if $\mathcal{R}_c > 1$, one of the Jury conditions[16] is violated

$$1 - \mathrm{tr}\boldsymbol{A} + \det\boldsymbol{A} = \gamma\mu - H\delta(\alpha\gamma + \beta\mu) > 0, \quad (14)$$

which implies that the matrix $\boldsymbol{A}$ has a pair of complex-conjugate eigenvalues lying outside the unit circle, which also implies that the equilibrium $(0,0)$ of the system (9) is unstable.

The above theorem shows us the condition under which the pathogen will extinct, and otherwise it will persist. The system (9) has an infection-free equilibrium in which the component of infectives is zero and the exposed is also zero. After analysing the local stability of the system (9) around $(0,0)$ we obtain a threshold value which determines when the disease dies out and when it persists in the system. These threshold conditions are characterized by the critical threshold $\mathcal{R}_c$, at which $(0,0)$ is locally asymptotically stable if $\mathcal{R}_c < 1$, and unstable if $\mathcal{R}_c > 1$. The numerical simulation shows this fact for different values of $\mathcal{R}_c$. (see Fig.5)

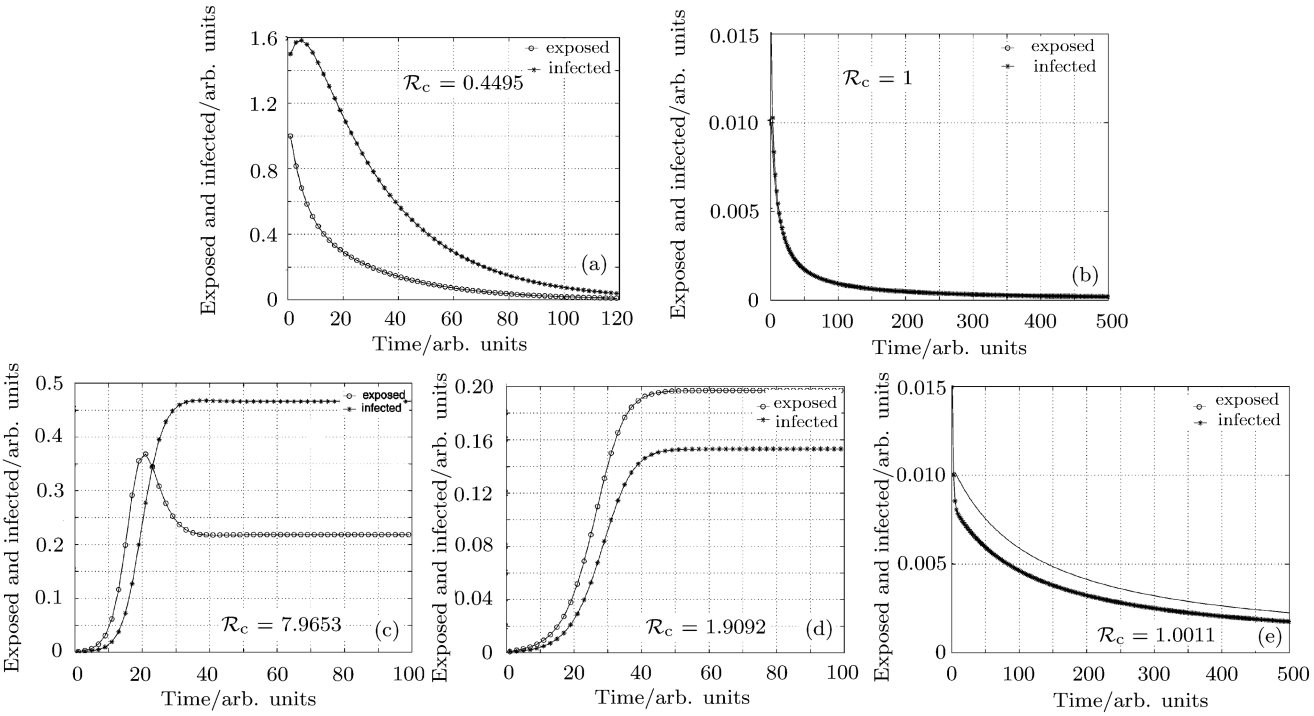


Fig.5. The numerical solution of the system (9) with different $\mathcal{R}_c$. For each figure we choose the following initial condition: $x(0) = 0.001$, $y(0) = 0.0015$. Parameters: (a) $\alpha = 0.002$, $\beta = 0.004$, $\mu = 0.15$, $\delta = 8$, $H = 0.8$, $d = 0.0003$ and $\lambda = 0.07$; (b) $\alpha = \beta = \mu = 0.64$, $d = 0.003$, and $\lambda = 0.637$; (c) with the same parameter values as in Fig.5(a) except $\alpha = 0.08$, $\beta = 0.05$; (d) $\mu = 0.35$, $\alpha = 0.5$, $\beta = 0.07$ and $\lambda = 0.45$, the values of other parameters are the same as in Fig.5(a); (e) $\mu = 0.35$, $\alpha = 0.05, \beta = 0.0061105$, $x(0) = 0.01$ and $y(0) = 0.015$ and $\lambda = 0.45$, the values of other parameters are the same as in Fig.5(a).

Now we are in a position to show the behaviour of the system (9) around the positive interior equilibrium.

Theorem 2 If $\mathcal{R}_c = H\delta(\alpha\gamma + \beta\mu)/\gamma\mu > 1$, the system (9) possesses a positive interior equilibrium (x^*, y^*).

Proof The system (10) can be expressed as

$$\begin{cases} \mu x = (H - x - y)[1 - (1 - \alpha x - \beta y)^{\delta}], \\ y = hx, \end{cases} \quad (15)$$

where $h = \mu/\gamma$. Again Eq.(15) can be written as

$$\mu x = (H - x - hx)[1 - (1 - \alpha x - \beta hx)^{\delta}].$$

Assume $g(x) = (H - x - hx)[1 - (1 - \alpha x - \beta hx)^{\delta}] - \mu x$. Since $0 < \alpha < 1, 0 < \beta < 1, 0 \le x \le 1$ and $0 \le y \le 1$, we have $g(0) = 0$, and $g'(x)$ given by

$$\begin{aligned} g'(x) = & -(1+h)[1 - (1 - \alpha x - \beta hx)^{\delta}] \\ & + (H - x - hx)\delta(\alpha + \beta h) \\ & \times (1 - \alpha x - \beta hx)^{\delta - 1} - \mu. \end{aligned}$$

Thus

$$\begin{aligned} g'(0) &= H\delta(\alpha+\beta h)-\mu \\ &= H\delta\Big(\alpha+\frac{\mu\beta}{\gamma}\Big)-\mu \\ &= \frac{1}{\gamma}[H\delta(\alpha\gamma+\beta\mu)-\mu\gamma]>0. \end{aligned}$$

One can conclude that the function $g(x)$ increases at $(0,0)$. If taking $0\le\tau=[(H/(1+h)]<1$, we obtain

$$g(\tau)=-\frac{H\mu}{1+h}<0.$$

Hence, it has been proven that there should be positive interior equilibrium of the system in the open interval $(0, H\mu/(1+h))$.

Now we wish to find the positive interior equilibrium of the system in the open interval $(0, H\mu/(1+h))$. For small values of α and β, taking Taylor expansion and keeping only the fist two terms, we obtain

$$(1-\alpha x-\beta y)^{\delta}=1-\delta(\alpha x+\beta y)+\cdots. \tag{16}$$

Putting it in Eq.(10), we obtain

$$\begin{cases} x=x(1-\mu)+\delta(H-x-y)(\alpha x+\beta y), \\ y=y(1-\gamma)+\mu x. \end{cases} \tag{17}$$

When $\mathcal{R}_{\rm c}>1$, we can derive the positive interior equilibrium of system (17) as follows:

$$\begin{cases} x^*=\dfrac{H\gamma}{\gamma+\mu}-\dfrac{\gamma^2\mu}{\delta(\gamma+\mu)(\alpha\gamma+\beta\mu)}, \\ y^*=\dfrac{H\mu}{\gamma+\mu}-\dfrac{\mu^2\gamma}{\delta(\gamma+\mu)(\alpha\gamma+\beta\mu)}. \end{cases} \tag{18}$$

Together with Eqs.(5) and (6), the only positive equilibrium point becomes

$$\begin{cases} \dfrac{x^*}{H}=\dfrac{\gamma}{\gamma+\mu}-\dfrac{\gamma^2\mu}{H\delta(\gamma+\mu)(\alpha\gamma+\beta\mu)}, \\ \dfrac{y^*}{H}=\dfrac{\mu}{\gamma+\mu}-\dfrac{\gamma\mu^2}{H\delta(\gamma+\mu)(\alpha\gamma+\beta\mu)}. \end{cases} \tag{19}$$

From Eq.(19) we can conclude that when $\mathcal{R}_{\rm c}>1$, the global frequency of the exposed and infective $\left(\frac{x^*}{H},\frac{y^*}{H}\right)$ increases with the neighbourhood size δ, and the pathogen transmission probabilities α and β are shown in Fig.6. Again from Fig.7, we can conclude that the infections decrease with the increase of the recovery probability λ (see Fig.7(a)), but the size of the exposed individuals will increase with the increase of λ (see Fig.7(b)). This observation supports that of Ellner.[17]

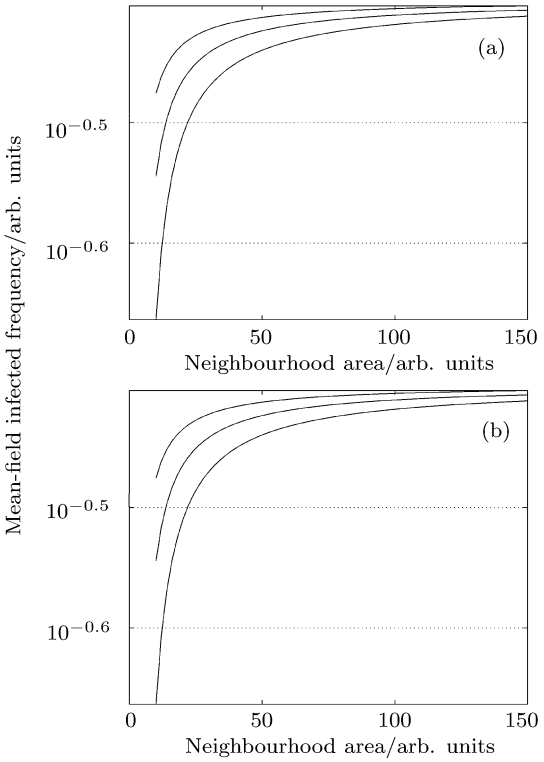


Fig.6. The frequency of the exposed and infected individuals in a logarithmic scale at the mean-field equilibrium. The parameters are: $H=0.7$, $\lambda=0.6$, $d=0.0003$, $\mu=0.4$ for each figure. The upper line is drawn with $\alpha=1/4$ and $\beta=1/2$; the middle line with $\alpha=1/6$ and $\beta=1/4$; the lower line with $\alpha=1/8$ and $\beta=1/8$.

Since $0<\alpha<1, 0<\beta<1$, $0\le x\le 1$ and $0\le y\le 1$, we have $0\le(\alpha x+\beta y)<1$. If the size of the interaction neighbourhood and the environment increases, that is, $\delta\to J, J\to\infty$, we gain

$$1-(1-\alpha x-\beta y)^{\delta}\to 1, \tag{20}$$

and as a consequence, Eq.(10) reduces to

$$\begin{cases} x=x(1-\mu)+(H-x-y), \\ y=y(1-\gamma)+\mu x. \end{cases} \tag{21}$$

Now solving the above equation and denoting the positive interior equilibrium as (x^+,y^+), we obtain

$$\begin{cases} x^+=\dfrac{H\gamma}{\mu\gamma+\gamma+\mu}, \\ y^+=\dfrac{H\mu}{\mu\gamma+\gamma+\mu}, \end{cases} \tag{22}$$

which also can be written as follows:

$$\begin{cases} \dfrac{x^+}{H}=\dfrac{\gamma}{\mu\gamma+\gamma+\mu}, \\ \dfrac{y^+}{H}=\dfrac{\mu}{\mu\gamma+\gamma+\mu}. \end{cases} \tag{23}$$

From the above formula, one can obtain that the frequency of the exposed and infected hosts can be approximated to $([1+\mu+\mu/(d+\lambda)]^{-1}, [1+\gamma+(d+\lambda)/\mu]^{-1})$. Comparing Eq.(19) with Eq.(23), it is easy to show that they are approximately equal if $\delta \to \infty$. Note that Eq.(19) is better than Eq.(23), since the former includes the effect of local environment, δ.

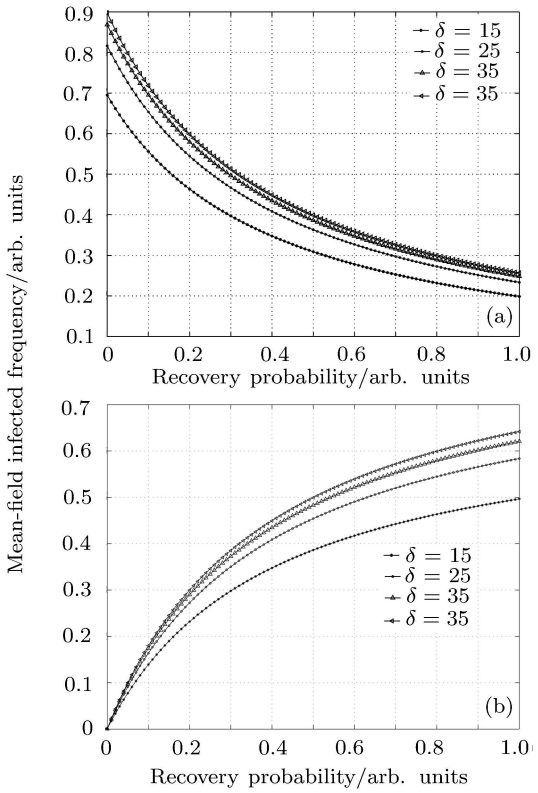


Fig.7. The effects of recovery probability on the global frequency for different values of δ, with $H=0.7$, $\mu=0.8$, $\alpha=1/6$, $d=0.0003$ and $\beta=1/4$.

Half of von Neumann neighbourhood Let us consider a special case when $\delta=2$; the neighbourhood becomes one-dimensional which is half of the standard von Neumann neighbourhood. In our case the neighbourhood consists of only horizontal neighbours or isotropic neighbours, and it has an identical probability for each step. Hence we define the radius as $r=1/2$, and Eq.(10) can be written as

$$\begin{cases} x = x(1-\mu) + (H-x-y)[1-(1-\alpha x-\beta y)^2], \\ y = y(1-\gamma) + \mu x. \end{cases} \tag{24}$$

Solving Eq.(24), we obtain

$$\begin{cases} x_1 = \dfrac{\gamma((2+H\alpha)\gamma + (2+H\beta)\mu - \sqrt{A})}{2(\gamma+\mu)(\alpha\gamma+\beta\mu)}, \\ y_1 = \dfrac{\mu((2+H\alpha)\gamma + (2+H\beta)\mu - \sqrt{A})}{2(\gamma+\mu)(\alpha\gamma+\beta\mu)}; \end{cases}$$

$$\begin{cases} x_2 = \dfrac{\gamma((2+H\alpha)\gamma + (2+H\beta)\mu + \sqrt{A})}{2(\gamma+\mu)(\alpha\gamma+\beta\mu)}, \\ y_2 = \dfrac{\mu((2+H\alpha)\gamma + (2+H\beta)\mu + \sqrt{A})}{2(\gamma+\mu)(\alpha\gamma+\beta\mu)}, \end{cases}$$

where

$$A = (H\beta-2)^2\mu^2 + \gamma^2(4-4H\alpha+H^2\alpha^2+4\mu) + 2\gamma\mu(H^2\alpha\beta - 2H(\alpha+\beta) + 2(2+\mu)).$$

4. Numerical simulations

In this section we present the numerical simulation for the CA model with the help of Matlab programme. Here the programme is performed on a 100×100 lattice ($J=10000$). The choice of the number of individuals is reasonable for real systems such as the public organization, school, company and so on. We have also simulated using a large value of J and the qualitative results is the same (for large J see Ref.[18]). Figure 6 shows that the global frequency of the exposed and infected individuals increases with the increase of the size of the neighbourhood provided that $\mathcal{R}_c > 1$. Figure 7 shows that infections decrease with the increase of the recovery probability λ (see Fig.7(a)), but the size of the exposed individuals will increase with the increase of λ (see Fig.7(b)). Figures 8 and 9 show the role of spatial heterogeneity with $\delta=8$. Figures 8(a) and 8(c) illustrate the disordered dispersions of the hosts at the initial time.

In Fig.8 the white, grey, and black lattice sites represent the susceptible, exposed, and infected individuals respectively. Figures 8(a) and 8(c) show the spatial distributions with $H=0.4$ and $H=0.85$ at the initial time $t=0$, respectively. We give the evolution results in Figs.8(b) and 8(d) at $t=50$ that start from Fig.8(a) and Fig.8(c) respectively. Figure 9 is the most important figure because it is an example of simulation for the SARS disease spreading in China in 2003, where the global density is equal to 0.7 (i.e. $H=0.7$). The number of people immune from SARS is so small that we can ignore it, and the state of the SARS is processing as Fig.4 shows. The detailed rules in the CA model is similar to those in Ref.[18]. Figure 10 is taken from the paper of Riley *et al*[19] where they have studied about the SARS disease in Hong Kong. Figure 9 is similar to their figure (i.e. see Fig.10) obtained by using different method in their paper. Similar works have been performed by Lipsitch *et al.*[20] Therefore we can say our results support their works.

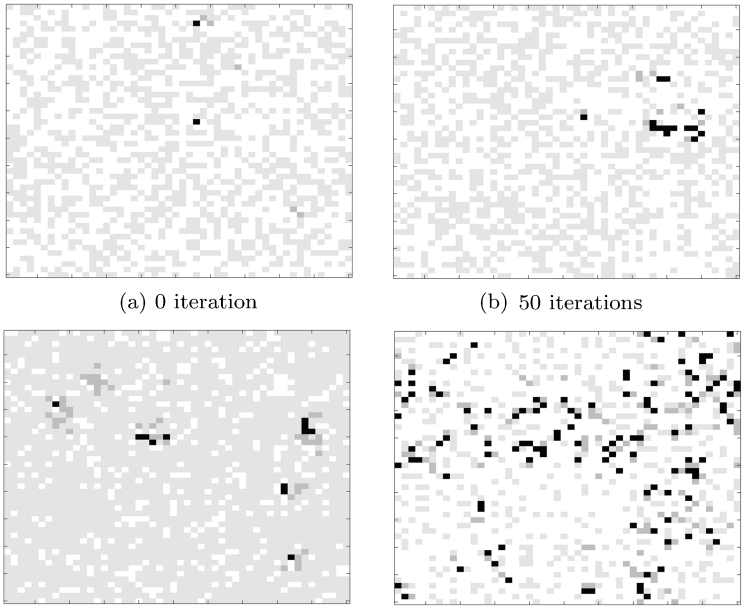


Fig.8. The evolution of the spatial distribution with parameters $\alpha = 1/8$, $\beta = 1/5$, $\mu = 0.4$, $d = 0.1332$, $\lambda = 0.45$ and $J = 10000$. Note that a given site may be empty (white), occupied by a susceptible (grey), or occupied by an exposed, or infected individual (black). (a) and (b) are for low global density $H(0) = 0.4$, (c) and (d) are for high global density $H(0) = 0.85$.

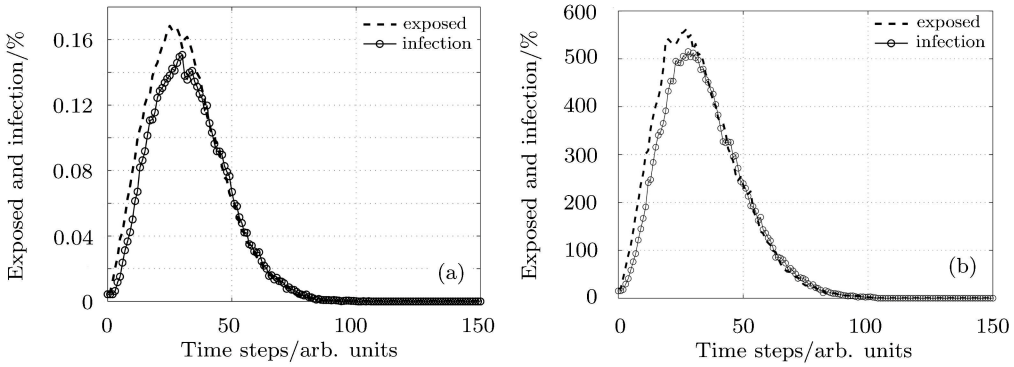


Fig.9. Numerical simulation by using the CA method in two-dimensional space with the global density $H = 0.7$ and the mortality rate $d = 0.1332$ (for Hong Kong). The values of the other parameters are $\alpha = 1/8$, $\beta = 1/5$, $\mu = 0.4$, $\lambda = 0.5$. (a) The global frequency of the exposed and infected against time. (b) The numbers of the exposed and infected against time.

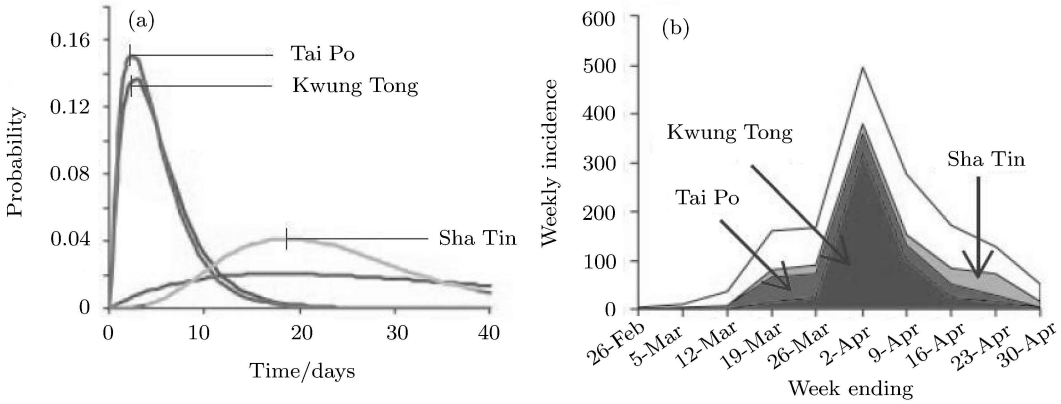


Fig.10. The time series of the exposed and infected. (a) Distributions of the waiting times of the compartments of the stochastic model. Distributions shown are estimates for the start of the epidemic. The onset-to-hospitalization distribution changes during the epidemic as a result of more rapid hospital admission. Weekly incidence (by the time of hospital admission) of Fig.10(b) corresponds to Fig.10(a). Reproduced with permission from Steven Riley *et al*, Ref.[19].

5. Conclusion and discussion

We have proposed and analysed an epidemic model by using cellular automata theory. We have considered the dispersion of the individuals with infectious force in a latent period. The standard **Moore neighbourhood** is used in this paper. The readers can compare other methods of the approximation to spatio-temporal models for epidemic with local spread, such as PA, SA, HPA.[21] Recently, the complex networks method is a hot topic and it is widely used in the research of the epidemic spreading.[22,23]

The method of approximation to the spatial model is proven to be true via the simulation. Its analytical results, as well as the numerical simulations suggest that the frequency of the exposed and infected host increases with the increase of the heterogeneity (density) and the size of the neighbourhood (see Figs.6, 7 and 8).

It has been observed that the infection will spread and persist when the epidemiological threshold $\mathcal{R}_c > 1$. (see Ref. [24]).

The effect of dispersion factor depends on the size of neighbourhoods and global density of the host. In Ref.[25], we studied the law governing the size of the neighbourhood under a heterogeneous susceptibility. Here, Fig.8 has been performed with different values of the global density of the host as one of the spatial heterogeneity factors that is demonstrated to be an important factor for epidemic models.

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