

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/2148264>

A Cellular Automata Model with Probability Infection and Spatial Dispersion

Article in *Chinese Physics* · May 2006

DOI: 10.1088/1009-1963/16/5/017 · Source: arXiv

CITATIONS

16

READS

69

3 authors, including:



Zhen Jin

Shanxi University

322 PUBLICATIONS 4,763 CITATIONS

[SEE PROFILE](#)



Mainul Haque

University of Nottingham

65 PUBLICATIONS 1,411 CITATIONS

[SEE PROFILE](#)

Some of the authors of this publication are also working on these related projects:



Predicting COVID-19 epidemics [View project](#)



Mathematical modeling and control of COVID-19 [View project](#)

A Cellular Automata Model with Probability Infection and Spatial Dispersion

Jin Zhen^{*} and Liu Quanxing[†]

*Department of mathematics, North University of China,
Taiyuan, Shan'xi, 030051, People's Republic of China*

Mainul Haque[‡]

*Department of Mathematics, Krishnath college,
Berhampore, Mursidabad, West Bengal, India-742101.*

(Dated: February 8, 2008)

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

PACS numbers: 05.50.+q, 87.23.Cc, 87.18.Hf, 89.75.Fb

I. INTRODUCTION

The foundation of modern mathematical epidemiology based on the compartment models were laid in the early 20th century. From the middle 20th century, mathematical epidemiology has been growing exponentially. Various mathematical models have been proposed and analyzed. Although epidemic models are studied too much from that time but little attention has been paid on the localized processes of pathogen transmission between susceptible, 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 are 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, spatial 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 of habitable sites, where no more than one individual can occupy any particular site. Spatially structured pathogen transmission says that the probability of susceptible host acquires 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 by exogenously, H . The varieties of the local host densities are very important to understand 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 [8] has studied an SIS epidemic model with varying pupulation density and obtained the condition for which the pathogen extinct. Similar results have been shown by Mollison [3] and Ahmed and Agiza [9]. Keeling [10] have studied the effects of local spatial structure on epidemiological invasions in an epidemic model. Here we have considered an SEIS epidemic model with infectious force in the exposed and infected period, like as in [11, 12]. In this SEIS model we have considered the spatial dispersion and obtained the MF transmission threshold for epidemic persistence, \mathcal{R}_c , by using probability cellular automata. We have also shown the nature of solution of the system around the trivial equilibrium (0,0) by using Lyapunov function. Finally numerical simulations have been performed by the help of experimental data for the SARS disease in China.

II. ANALYSIS FOR THE MODEL

In order to get the same neighborhood 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 neighborhood for each site k are shown in Figure 1 and Figure 2. The Figure 3 shows the same fact with a special black point neighborhood for site k . Let us suppose that the individual dispersion in the sites

^{*}Electronic address: jinzhn@263.net

[†]Electronic address: liuqx315@sina.com

[‡]Electronic address: mainul.haque@rediffmail.com

of the two-dimensional torus for the global state set is denoted by $Z(t)$ at time t . Then we have

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

In the above global set, the $z_k(t)$ 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 recoveree from pathogen infection does not confer immunity, so the recovered individuals will again be treated as susceptible individual.

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

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

Note that in the Eq. (1), $J > N(t)$, therefore we get $H(t) \leq 1$. In this model, we assume that the natural birth rate equal to it's natural death rate and d is the mortality rate due to the infection.

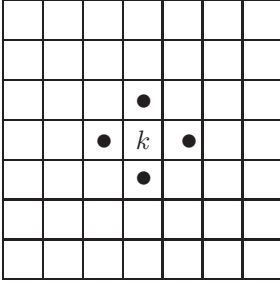


FIG. 1: Von Neumann Neighborhood.

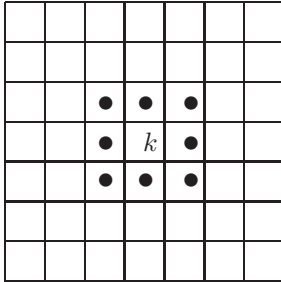


FIG. 2: Moore Neighborhood.

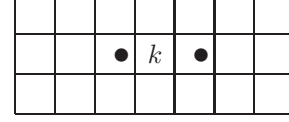


FIG. 3: $r = 1/2$.

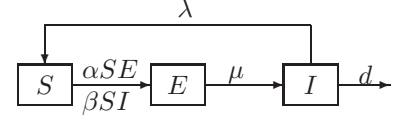


FIG. 4: Schematic flow diagram of transmission for SEIS model.

Here the δ represents the maximum number individual of in neighborhood for site k , then the number of sites in δ_k is $\delta = |\delta_k| - 1$, where the δ_k is the k -th neighborhood. There is no variation in size among 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 like as follows: (i) if the k -th site is non-empty, then we can assume that a exposed or infected k -th site transmits the pathogen to a susceptible individual within δ_k and (ii) if the k -th site is not infected or exposed, then it acquires the pathogen (infection) from a exposed or infected individual within δ_k . In our model, let us assume that δ_k is symmetric. We also assume that the site k (or k -th site) with it's center is like as it is shown in Figure 1 and Figure 2. There are the same transmit rule for all δ_k . When the δ_k is asymmetric, a host at site k is infected from a sets differing from the set to which the pathogen can be transmitted. Therefore it will be difficult to discuss the mode of transmission in term of mathematical equations. So for the sake of simplicity, we have assumed that δ_k is symmetric. Now we define the local function f as follows

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

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

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

The n_k hosts on δ_k include s susceptibles, e exposed and i infectives; $n_k = s + e + i$. Here α is the probability, per discrete time interval, that an exposed on δ_k transmits the disease to the susceptible at site k . β is the probability, per discrete time interval, that an infective on δ_k transmits the disease to susceptible at site k . μ is the probability, per time interval, that an exposed individuals convert to a infected one. Since the

infectives act independently, so the susceptible acquires the pathogen in a single time interval with conditional probability $1 - (1 - \alpha)^e(1 - \beta)^i$. The unconditional prob-

ability of infecting the focal susceptible in a discrete time interval is ε_k :

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

III. THE APPROXIMATION OF THE GLOBAL DENSITY

Mean-field analysis have different forms, here we adopt a ‘local-dispersal’ approximation [13]. Let us assume $S(t)$, $E(t)$, $I(t)$ 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

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

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

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

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

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

$$\begin{aligned} x(t+1) &= F_1(x(t), y(t)) = x(t)(1 - \mu) + (H(t) - x(t) - y(t)) \cdot \sum_{\xi=0}^{\delta} \sum_{\eta=0}^{\delta} \\ &\quad \cdot \binom{\delta}{\xi} \binom{\delta-\xi}{\eta} x(t)^{\xi} y(t)^{\eta} (1 - x(t) - y(t))^{\delta-\xi-\eta} [1 - (1 - \alpha)^{\xi} \cdot (1 - \beta)^{\eta}] \\ &= x(t)(1 - \mu) + (H(t) - x(t) - y(t)) \cdot \left[\sum_{\xi=0}^{\delta} \sum_{\eta=0}^{\delta} \binom{\delta}{\xi} \binom{\delta-\xi}{\eta} x(t)^{\xi} y(t)^{\eta} (1 - x(t) - y(t))^{\delta-\xi-\eta} \right. \\ &\quad \left. - \sum_{\xi=0}^{\delta} \sum_{\eta=0}^{\delta} \binom{\delta}{\xi} \binom{\delta-\xi}{\eta} x(t)^{\xi} y(t)^{\eta} (1 - x(t) - y(t))^{\delta-\xi-\eta} (1 - \alpha)^{\xi} (1 - \beta)^{\eta} \right] \\ &= x(t)(1 - \mu) + (H(t) - x(t) - y(t)) \cdot \left[1 - (1 - \alpha x(t) - \beta y(t))^{\delta} \right]. \end{aligned} \quad (7)$$

and

$$y(t+1) = F_2(x(t), y(t)) = y(t)(1 - \lambda - d) + \mu x(t) \chi(8)$$

From Eq. (7) and Eq. (8) we can obtain that the global density of exposed and infective are given as follows:

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

This is a two-dimension nonlinear system. Since $D \ll N(t)$ for not too long time, so we can omit the change of total individual (see [14]) and let $H(t) = H = \text{constant}$, $x(t+1) = x(t) = x$, 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

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

Now in the above system, we have $\gamma = d + \lambda$. Since $\delta > 0$, $0 \leq x(t) < H$, $0 \leq y(t) < H$, so solving these above equations we get 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 the system (9) at $(0, 0)$. From the equation (7) and the equation (8) we obtain

$$\begin{aligned} \frac{\partial F_1}{\partial x(t)} &= (1 - \alpha x - \beta y)^\delta + \alpha \delta (H - x - y)(1 - \alpha x - \beta y)^{\delta-1} - \mu, \\ \frac{\partial F_1}{\partial y(t)} &= (1 - \alpha x - \beta y)^\delta + \beta \delta (H - x - y)(1 - \alpha x - \beta y)^{\delta-1} - 1, \\ \frac{\partial F_2}{\partial x(t)} &= \mu, \\ \frac{\partial F_2}{\partial y(t)} &= 1 - \gamma. \end{aligned}$$

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

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

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

$$\text{tr}\mathbf{A} = 2 - \mu - \gamma + H\delta\alpha \quad (11)$$

and

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

Now let us denote $\mathcal{R}_c = \frac{H\delta(\alpha\gamma + \beta\mu)}{\gamma\mu}$, as the persister threshold, then the following theorem holds true.

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

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

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

Let us consider a positive Lyapunov function $V = (\frac{\alpha}{\mu} + \frac{\beta}{\gamma})x + \frac{\beta}{\gamma}y$, then the time derivative of this function along

the solution of the system (13), we get

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

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

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

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

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

The above theorem gives us the condition for which the pathogen will extinct and when it will persist. The system (9) has an infection-free equilibrium in which the component of infectives is zero and exposed is zero. After analyzing the local stability of the system (9) around $(0, 0)$ we get a threshold value which determine when the disease dies out and when persist in the

system. These threshold conditions are characterized by the critical threshold \mathcal{R}_c , such that $(0,0)$ 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 .

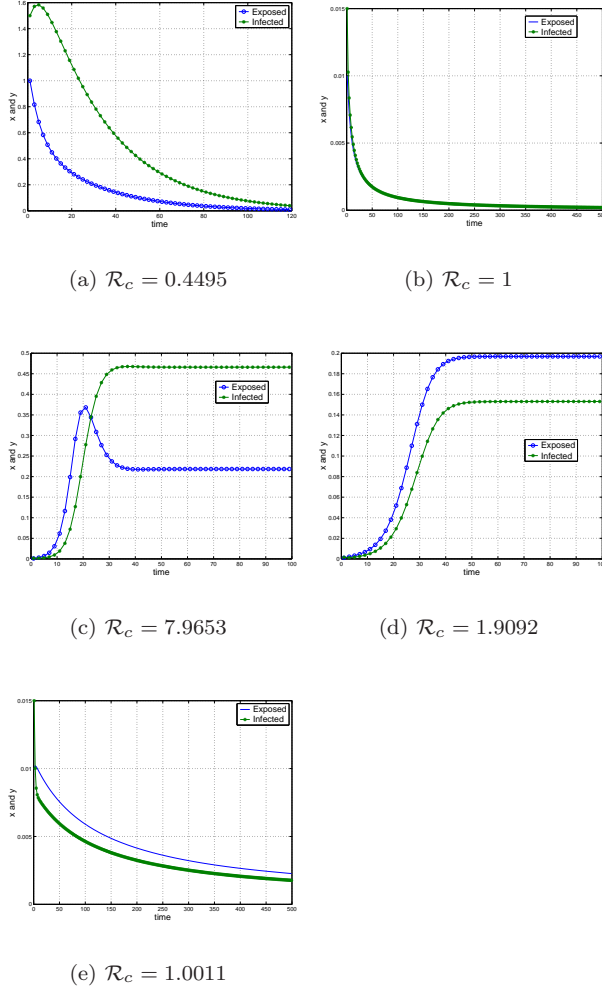


FIG. 4: For each figures we assume the following initial condition: $x(0) = 0.001$, $y(0) = 0.0015$. (a) Figure a: Figure a have drawn with the parameters values: $\alpha = 0.002$, $\beta = 0.004$, $\mu = 0.15$, $\delta = 8$, $H = 0.8$, $d = 0.0003$ and $\lambda = 0.07$. (b) For figure b: $\alpha = \beta = \mu = 0.64$, $d = 0.003$, and $\lambda = 0.637$. (c) For figure c: with the same parameters value that in figure (a) except $\alpha = 0.08$, $\beta = 0.05$. (d) For figure d: $\mathcal{R}_c = 1.9092$, $\mu = 0.35$, $\alpha = 0.5$, $\beta = 0.07$ and $\lambda = 0.45$, the value of other parameters are remaining same as in figure (a). (e) For figure e: $\mathcal{R}_c = 1.0011$, $\mu = 0.35$, $\alpha = 0.05$, $\beta = 0.0061105$, $x(0) = 0.01$ and $y(0) = 0.015$ and $\lambda = 0.45$, the value of other parameters are remaining same with figure (a).

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

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

Proof. The system of (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 the equation (15) can be written as

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

Now let us assume $g(x) = (H - x - hx)[1 - (1 - \alpha x - \beta hx)^\delta] - \mu x$. Since $0 < \alpha < 1$, $0 < \beta < 1$, $0 \leq x \leq 1$ and $0 \leq y \leq 1$, so $g(0) = 0$ and $dg(x)/dx$ is given by

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

Therefore

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

So, the function $g(x)$ is increasing at $(0,0)$. Therefore if we take $0 \leq \tau = \frac{H}{1+h} < 1$, then we get

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

Thus, there should be positive interior equilibrium of the system in the open interval $(0, \frac{H\mu}{1+h})$. Hence the proof. \square

Now we are interested to find out the positive interior equilibrium of the system in open interval $(0, \frac{H\mu}{1+h})$. Now for small values of α and β , taking Taylor expansion and keeping only the first two terms, we get

$$(1 - \alpha x - \beta y)^\delta = 1 - \delta(\alpha x + \beta y) + \dots \quad (16)$$

So from the systems (10), we obtain

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

Therefore when $\mathcal{R}_c > 1$, we can derive the positive interior equilibrium of the system (17) as follows

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

Together with the equation (5) and the equation (6), we get

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

From the equation (19) we can conclude that when $\mathcal{R}_c > 1$, then the global frequency of exposed and infective $(\frac{x^*}{H}, \frac{y^*}{H})$ increases with neighborhood size δ and with the pathogen transmission probability α and β . Figure 6 shows this fact. Again from Figure 6, we can conclude that infections decreases with the increasing of the recovery probability λ (see Figure 7(a)) but the size of the exposed individuals will increase with the increasing of λ (see Figure 7(b)) . This observation supports the observation of Ellner [17].

Now Since $0 < \alpha < 1, 0 < \beta < 1, 0 \leq x \leq 1$ and $0 \leq y \leq 1$, so $0 \leq (\alpha x + \beta y) < 1$. Now if the size of the interaction neighborhood and the environment increases, that is, $\delta \rightarrow J, J \rightarrow \infty$, then we get

$$1 - (1 - \alpha x - \beta y)^\delta \rightarrow 1, \quad (20)$$

as a consequence the equation (10) reduces to

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

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

$$\begin{cases} x^+ = \frac{H\gamma}{\mu\gamma + \gamma + \mu}, \\ y^+ = \frac{H\mu}{\mu\gamma + \gamma + \mu}, \end{cases} \quad (22)$$

which can be written as follows

$$\begin{cases} \frac{x^+}{H} = \frac{\gamma}{\mu\gamma + \gamma + \mu}, \\ \frac{y^+}{H} = \frac{\mu}{\mu\gamma + \gamma + \mu}. \end{cases} \quad (23)$$

Therefore the frequency of exposed and infected hosts are approximated to $([1 + \mu + \frac{\mu}{d+\lambda}]^{-1}, [1 + \gamma + \frac{d+\lambda}{\mu}]^{-1})$. Now the by comparison of the solution (19) with the solution (23), it is easy to show that they are approximately equal if $\delta \rightarrow \infty$. Note that the solution (19) is better than the solution (23), which shows the relation between exposed, infected and susceptible individuals.

Half von Neumann neighborhood: Let us consider a special case when $\delta = 2$, the neighborhood become one-dimensional which is half of the standard von Neumann neighborhood. Here in our case the neighborhood have only horizontal neighbors or isotropic neighbors, and it have the qual probability for each step. Hence we define the radius $r = 1/2$, the equation of (10) can be written as

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

Solving the equation (24) we get

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

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

where

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

IV. NUMERICAL SIMULATIONS

In this section we have shown the numerical simulation of our CA model with the help Matlab programme. Here we have considered the case for a common place like any public organization, school, company and so on, hence we assume $J = 10000$. We have also simulated for a large value of J but have the similar results like in our other paper [18]. The sites are updated synchronously. Figure 5 shows that the global frequency of exposed and infected individuals increase with the increasing of the size of the neighborhood provided $\mathcal{R}_c > 1$. The Figure 6 shows that infections decreases with the increasing of the recovery probability λ (see Figure 7(a)) but the size of the exposed individuals will increase with the increasing of λ (see Figure 7(b)) . Figure 7 and Figure 8 are showing the role of spatial heterogeneity with $\delta = 8$. The Figure 8(a) and figure 8(c) are showing the disorder dispersions of the hosts at initial time.

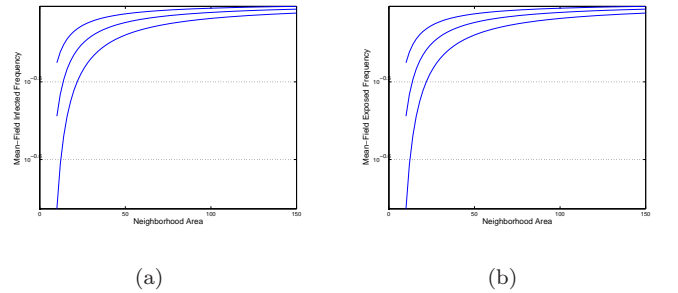


FIG. 5: The frequency of exposed and infected individuals with a logarithmic scale at mean-field equilibrium. The parameters values are: $H=0.7$, $\lambda = 0.6$, $d = 0.0003$, $\mu = 0.4$ for each figure. In the above figures, the upper line is drawn with $\alpha = \frac{1}{4}$ and $\beta = \frac{1}{2}$; the middle line with $\alpha = \frac{1}{6}$ and $\beta = \frac{1}{4}$; the lowermost line with $\alpha = \frac{1}{8}$ and $\beta = \frac{1}{8}$.

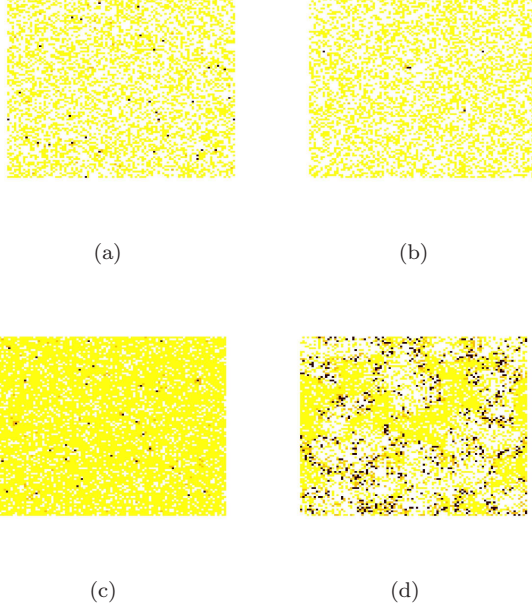


FIG. 7: In the above figures the space dispersion are shown in details with parameters values $\alpha = \frac{1}{8}, \beta = \frac{1}{5}, \mu = 0.4, d = 0.1332, \lambda = 0.45$ and $J = 10000$. Note that a given site may be empty (white), occupied by a susceptible (grey), or occupied by an exposed, or infected individuals (black). (a) and (b) are for low global density $H(0) = 0.4$, (b) The same after 50th iteration of the previous. (c) and (d) are for high global density $H(0) = 0.85$, (d) The same after the 50th iteration of the previous.

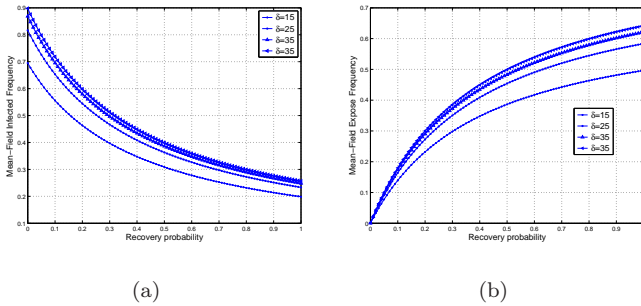


FIG. 6: The effects of recovery probability. The above figures are drawn for different values of δ with $H = 0.7, \mu = 0.8, \alpha = 1/6, d = 0.0003$ and $\beta = 1/4$.

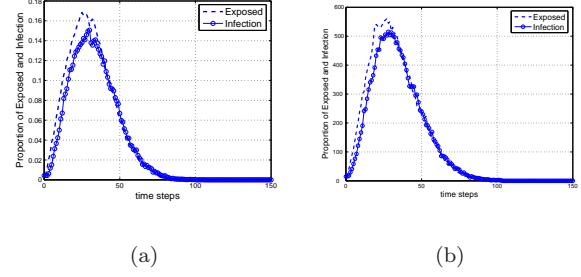


FIG. 8: In the above simulations we have taken the global density $H = 0.7$ and the mortality rate $d = 0.1332$ (for Hong Kong city). The value of the other parameters are $\alpha = 1/8, \beta = 1/5, \mu = 0.4, \lambda = 0.5$. (a) The global frequency of the exposed and infected with respect to time. (b) The numbers of exposed and infected with respect to time.

In the Figure 7 the white, shaded and black portion represent the empty site of susceptible, exposed and infected individuals respectively. In Figure 7(a) average density of the host is 0.4 and in Figure 7(c) it is 0.85 at initial time $t=0$. We have got the Figure 7(b) and Figure 7(d) at $t=50$ with the same parameters values as in Figure 7(a) and Figure 7(c) respectively. The Figure 8 is the most important figure because it is an example of simulation for the SARS disease spreading at China in 2003, where the global density is equals to 0.7 (i.e $H = 0.7$). The number of the recovery of SARS is so small that we can ignore it, so the state of the SARS is processing as the Figure 4 shows. The detail rule to the CA model is similar to [18]. The Figure 9 is collect from the papers of Steven et. al [19] where they have studied about the SARS disease in Hong Kong city. Our Figure 8 is similar to their figure Figure 9 but not same. Similar works have performed by Lipsitch et. al [20]. Therefore our results are supported by their works.

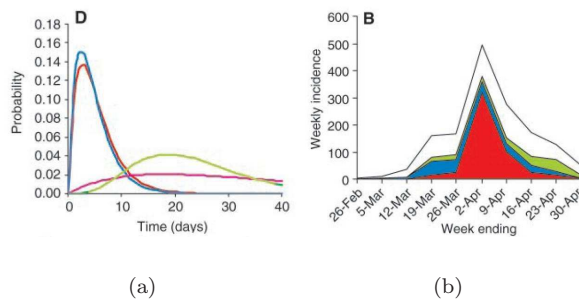


FIG. 9: (a) Distributions for the waiting times of the compartments of the stochastic model shown in see [19]. Distributions shown are estimates for the start of the epidemic. The onset-to-hospitalization distribution changes during the epidemic as a result of more rapid hospital admission. (b) Weekly incidence (by time of hospital admission) with the color coding used in (a).

V. CONCLUSION AND DISCUSSION

In this article we have proposed and analyzed an epidemic model by using cellular automata theory. We have considered here the dispersion of the individuals with infectious force in latent period. We have also used the

standard **Moore neighborhood**. The readers can compare the another methods of the approximation to spatio-temporal models for epidemic with local spread, such as PA, SA, HPA in [21].

We have made some approximation which are very logical for the system. The analytical findings and as well as the numerical simulations suggest us that the frequency of exposed and infected host increase with the increasing of heterogeneity (density) and the size of neighborhood (See the figure 6, figure 7 and figure 8) .

It has been observed that to spread the infection, the epidemiological threshold $\mathcal{R}_c > 1$. This Mean-Field per-sister threshold (R_c) property is to some extent related to the “basic reproductive ratio R_0 ” of the epidemic theory, see [22].

Generally, the effect of dispersion factor depends on the size of neighborhood and global density of host. The Figure 7 have been performed with different values of the global density of the host as the heterogeneity is an important factor for epidemic models.

VI. ACKNOWLEDGMENTS

This work is supported by the National Natural Science Foundation of China under Grant No 10471040 and part of this work have been done at that time when author M. Haque is visiting North University of China.

-
- [1] T. Caraco, M. Duryea, G. G. adn William Maniatty, and B. K. Szymanski, J. Theor. Biol. **351-61**, 351 (1998).
 - [2] H. Fuk s and A. T. Lawniczak, Discrete Dynamics in Nature and Society **6**, 191 (2001).
 - [3] D. Mollison, Mathematical Biosciences **107**, 255 (1991).
 - [4] R. Anderson, D. Gordon, M. Crawley, and M. Hassell, Nature **296**, 245 (1982).
 - [5] M. Hutchings, *Plant population biology* (Blackwell Scientific Publications (28th Symposium of the British Ecological Society), 1988).
 - [6] D. Harcourt, Can. Ent. **93**, 945 (1961).
 - [7] S. Kobayashi, Res. Popul. Ecol. **7**, 109 (1965).
 - [8] M. Duryea, T. Caraco, G. Garder, W. Maniatty, and B. Szymanski, Physica D **132**, 511 (19999).
 - [9] E. Ahmed and H. Agiza, Physica A **253**, 347 (1998).
 - [10] M. Keeling, Proc. Roy. Soc. Lond. B **266**, 859 (1999).
 - [11] Y. San-Ling, J. Biol. Math. **16**, 392 (2001).
 - [12] L. Guihua and J. Zhen, Chaos, Solitons and Fractals **25**, 1177 (2005).
 - [13] D. Hiebeler, J. Theor. Biol. **187**, 307 (1997).
 - [14] O. Diekmann and J. Heesterbeek, *Mathematical Epidemiology of Infectious Disease-Model Building, Aalysis and Interpretatio*, Wiley series in mathematical and Computational Biology (John Wiley and Son, Ltd, 2000).
 - [15] J. Laselle, *The stability of dynamical system* (Hmilton Borlin press, 1993).
 - [16] E. Jury, *Inners and stability of dynamic systems* (Wiley, New york, 1974).
 - [17] S. P. Ellner, J. Theor. Biol. **210**, 435 (2001).
 - [18] L. Quanxing and J. Zhen, Chinese Physics **15**, 1370 (2005).
 - [19] S. Riley, C. Fraser, and C. A. Donnelly, **300**, 1966 (2003).
 - [20] M. Lipsitch, T. Cohen, and B. Cooper, Science **300**, 1966 (2003).
 - [21] J. Filope and G. Gibson, Bulletin of Mathematical Biology **63**, 603 (2001).
 - [22] R. Anderson and R. May, Proc. Roy. Soc. Lond. B **291**, 451 (1981).