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Epidemic spreading in random walkers with heterogeneous interaction radius

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Abstract. We study epidemic spreading in a random walk network where agents with heterogeneous interaction radius randomly walk in a planar space. We obtain the explicit expression of epidemic threshold which indicates that the heterogeneity of interaction radius decreases the threshold. Concretely, the greater the variance of the radius distribution is, the smaller the epidemic threshold will be. Simulation results about the epidemic threshold match well with our theoretical results. In simulation study, the infection density in steady state, which is called the final density, is investigated. When there are two different values of radius, a larger mean value of radius increases the final density. However, although an increasing second order origin moment of radius makes the epidemic easier to outbreak, it also lowers the final density of infected individuals.

Keywords: Epidemic modelling, Computational biology

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Contents

1. Introduction

Amongst the most known works of investigating epidemic spreading, the earliest mathematical model was the Bernoulli equations proposed by Daniel Bernoulli to study small pox vaccination in 1760 [1]. Then cames the susceptible-infection-susceptible (SIS) and susceptible-infected-recovered (SIR) models which have attracted considerable attention since their presence [2, 3]. In the SIS model, an epidemic appears and subdivides the influenced population into two classes: susceptible and infected. Infected individuals convert back into the susceptible group after recovery. However, in the SIR model, a long-life immunity is gained after recovering from the infection. Also, there are other similar models including SI, SIRS, SEI, SIV and so on [4].

Mostly, the spreading of an epidemic needs physical contact, or say interaction, between individuals. A large-scale population forms an extremely complex interaction relationship. Traditional models like lattices, regular tree and classical random networks fail to model the epidemic spreading in large-scale networks with heterogeneity. Hence, in recent decades, complex networks have become a frequently used framework to study epidemic spreading since the introduction of the Watts–Strongatz (WS) small world network and the Barabási–Albert (BA) scale-free network [5, 6]. In [7, 8], Pastor-Satorras and Vespignani studied dynamical behavior of epidemic spreading in a BA scale-free network and found that the epidemic threshold disappears in the large-scale network. Also, many other works have investigated the epidemic spreading on networks with communities, small-world networks or multi-layer networks, adaptive networks, and the spreading of rumors and computer virus on complex networks [9–17].

However, most of the studies presented so far concern the case of a static network, i.e. the topological structure of network that are fixed in time, given once forever. However, the topological structure of networks, especially for human social networks, are time-varying. Most recently, quite a few works start to investigate some interesting phenomena, like the birth and death of individuals [18, 19], the weight-varying of links [20] and random walk networks or networks with moving nodes [21–31]. The random walk network, precisely, is a network where individuals constituting the nodes of the

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graph are here random walkers that are only able to interact with individuals falling within a given interaction radius apart from them [26].

Many researchers have delved deeply into the issue of epidemic spreading on a random walk network. To name a few, in [26], Mattia Frasca *et al* investigated the properties of a dynamical network with mobile individuals who are allowed to perform long-distance jumps. They presented the results of how long-distance jumps are combined with the local random walk to influence the structure and properties of the network. Based on the work in [26], Arturo Buscarino researched into the effect of local and long-distance motion on the SIR model. They found that the homogeneous-mixing approximation works well only when the velocity of movement is large enough [27]. In [24, 28], Zhou *et al* looked into communities with random walkers to simulate the realistic situation of multiple cities and to investigate the impact of the preference of an individual for public transport on the spread of infectious disease. Also, there are many other excellent works that investigate the epidemic spreading based on a random walk network [21–23, 25]. In conclusion, the propagation dynamics on random walk networks attract considerable attention in the area of epidemic spreading [32].

Almost all previous works based on random walk networks suppose that different individuals have the same interaction radius so as to better investigate other properties like the velocity of motion, the direction of motion and the density of the population. Nonetheless, in reality, the interaction radius of individuals are supposed to be heterogeneous. For example, individuals with poor hygiene habits tend to have a larger radius of contacting with infection sources. In wireless sensor networks, the transmission radius of each sensor depends on its available power. And thus, sensors with different power have different transmission raidus [33]. Since many examples show that uniform radius is not good enough to simulate well the system in real world, investigating heterogeneous interaction radius on random walk networks is necessary and has great practical significance. Thus in this paper, we focus on the heterogeneity of individuals' interaction radius.

We consider an individual i has its own radius denoted by r^i. It can be infected by any other infected individuals within the circle defined by the location and r^i. This definition forms a directed network [34, 35] where that individual i can be infected by its neighbor but that does not guarantee the vise versa. In a directed network, the number of incoming links into a nodes is its in-degree and the number of outgoing links from a nodes is its out-degree [4]. A disease spreads from a node to other nodes through the outgoing links, and a node is infected by a disease from the incoming links [36].

The contributions of this paper are given as follows. A novel model is proposed to investigate the effect of heterogeneous interaction radius on epidemic spreading in random walkers. The dynamical process of epidemic spreading is modeled by the ordinary differential equation. Simulation shows that our model can well mimic the dynamical propagation process of epidemic. We obtain the correlation between epidemic threshold and the heterogeneous interaction radius, which indicates that the heterogeneity of interaction radius of individuals decreases the epidemic threshold. Moreover, the larger the second order origin moment of radius is, the lower the epidemic threshold would be, the easier the epidemic to outbreak. This result is verified by simulation. We also found that when there are two different values of radius, a larger mean value of radius increases thr final density. However, although an increasing second order origin

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moment of radius makes the epidemic easier to outbreak, it also lowers the final density of infected individuals.

The outline of this paper is as follows. In section 2, we establish the epidemic spreading model studied. In section 3, we show how epidemic threshold is decided by the density of population and the distribution of interaction radius by theoretical analysis. Simulation of this dynamical model is shown in section 4 where the results of epidemic threshold and final density are presented.

2. Model description

In order to realize a random walk network, there is a feasible and simple method used by previous works [22, 25–27, 29, 30]. Under this framework, we consider N moving individuals distributed in a planar space $\Gamma = \{(x, y) \in \mathbb{R}^2 : 0 \leqslant x \leqslant D, 0 \leqslant y \leqslant D\}$, with periodic boundary conditions, as is shown in figure 1. Because of periodic boundary conditions, although individual 2 in figure 1 is near to the southwest corner it can cover the areas in three other corners and when individual 7 moves beyond the east side boundary, it appears immediately in the west side at the corresponding location. Individuals are represented as point particles. The position of the ith individual at time t is denoted as $\Lambda_i(t) = (x_i(t), y_i(t)) \in \Gamma$ while its velocity $v_i(t) = (v\cos\theta_i(t), v\sin\theta_i(t))$, $i = 1, ..., N$. At time $t = 0$, the N particles are uniformly distributed in the planar space. Then, the coordinates and the velocity of individual i at time $t + 1$ are updated according to

$$\begin{aligned} x_i(t+1) &= x_i(t) + v_i(t)\cos\theta_i(t), \\ y_i(t+1) &= y_i(t) + v_i(t)\sin\theta_i(t), \\ \theta_i(t+1) &= \xi_i(t), \end{aligned} \tag{1}$$

where $\xi_i(t)$ is a random variable obeying the uniform distribution on the interval of $[-\pi, \pi]$ in each step. Each node has its own interaction radius. r^i is the interaction radius for the ith individual. In addition, considering the realistic phenomenon of travel with time scales shorter than those related to epidemic dynamics, we define the possibility of individuals performing long-distance jump $p_{\text{jump}} \in [0, 1]$, which many other works have discussed [22, 26, 27]. p_{jump} quantifies the probability of an individual's performing a jump to a random position in the space Γ.

At time t, the Euclidean distance between node i and j is expressed as

$$d_{ij}(t) = d_{ji}(t) = \|\Lambda_i(t) - \Lambda_j(t)\|_2 = \sqrt{(x_i(t) - x_j(t))^2 + (y_i(t) - y_j(t))^2}, \quad i, j \in 1, ..., N.\ i \neq j. \tag{2}$$

The in-degree of individual i [34] at time t depends on the number of other individuals within individual i's interaction radius, while its out-degree is defined as the number of individuals whose in-degree contains individual i. More clearly, individual i's in-degree is the number of individuals who satisfy $d_{ij}(t) \leqslant r^i, \forall\, j \neq i$, and out-degree is defined as the quantity of individuals who meet $d_{ij}(t) \leqslant r^j, \forall\, j \neq i$. We assume that, in our model, there are m different values of interaction radius which obey distribution $P(r_j)$, $j = 1, ..., m$ where $P(r_j)$ denotes the proportion of nodes with interaction radius r_j.

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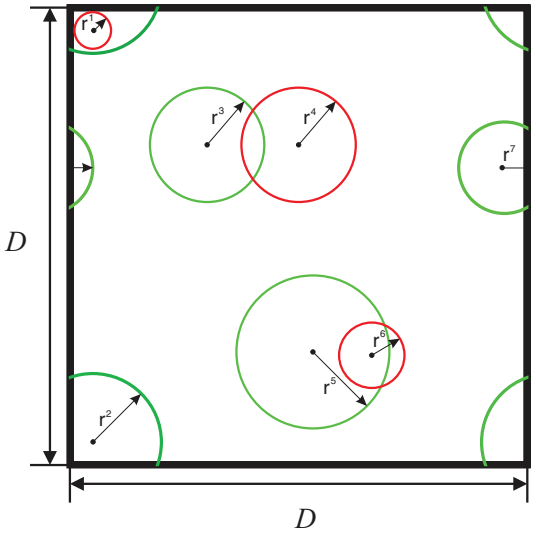

Figure 1. Illustrative plot for the study of epidemic spreading in a population of individuals who perform random walk in a planar space with periodic boundary conditions. Green represents the individual who is susceptible while red for the infected one. There are only seven nodes plotted for illustration purpose.

Epidemic spreading is modeled according to the SIS model, which, as mentioned before, partitions the N nodes into two disjointed compartments [2, 3]: susceptible (S) and infected (I). We indicate $N_S(t)$ and $N_I(t)$ as, respectively, the number of individuals in the two compartments at time t. Obviously, we have the total number of individuals $N_S(t) + N_I(t) = N$, which remains constant over time. A susceptible individual can be infected by each contact with an infected individual with probability β. An infected one, at each time step, could be recovered with probability λ.

The process through which the epidemic spreads can be summarized as follows. The interaction radius defines the network of contacts: at each time step t, individual i can be infected by contacting with infective neighbors located within a disk-shaped neighborhood of radius r^i. In the framework given in figure 1, individuals 2 and 5, respectively, have a chance of being infected by individuals 1 and 6. However, since individual 4 is located outside individual 3's neighborhood, epidemic is impossible to spread from individual 4 to individual 3. Since interaction radius indicates the neighborhood of an individual who could be infected by infected neighbors within this neighborhood, we also name it as susceptibility radius.

To further elucidate the system behavior, we derive a mean-field approximation under the homogeneous mixing (HM) hypothesis, whereby each individual has the same probability of contacting any other [22]. If high mobility ($p_{\text{jump}} \to 1$ or $v \simeq D$) is satisfied, the contact network is annealed into an averaged one that can be well approximated by HM hypothesis [22, 26, 27, 37].

Homogeneous mixing defines that individuals have same probability of contacting any other. In our model, P_{jump} quantifies the probability of an individual's performing a jump to a random position in the space. That is, when $P_{\text{jump}} = 1$, each individual locates at a random position independently. The definition of $P_{\text{jump}} = 1$ makes it equivalent that individuals have the same probability of contacting any other. Thus, the results under the two different circumstances are the same. However, different from our model,

other works [26, 27] define that individuals perform long distance jump in other ways. They travel with a much larger velocity v_{M} and with a random angle. For example, in [26], $v/v_{\mathrm{M}} = 0.03$, instead of appearing in a random position. We believe that, in the circumstance of $P_{\mathrm{jump}} = 1$, only if $v_{\mathrm{M}} \simeq D$ or $v_{\mathrm{M}} \gg D$, each individual who performs a long distance jump has equal chance of contacting any other. The movement of individuals just accords with the homogeneous mixing hypothesis.

In this hypothesis, we have $\rho = N/D^2$. $N_{\mathrm{S}}(r, t)$ and $N_{\mathrm{I}}(r, t)$ are respectively the number of susceptible individuals and the number of infected individuals with susceptibility radius r. $N_r := N_{\mathrm{S}}(r, t) + N_{\mathrm{I}}(r, t)$ which is a constant. We define $s(r, t) = N_{\mathrm{S}}(r, t)/N_r$ and $i(r, t) = N_{\mathrm{I}}(r, t)/N_r$ as the fractions of susceptible and infected nodes at time t. Obviously, $s(r,t) + i(r,t) = 1$. Under the HM hypothesis, we calculate the probability of not being infected for individuals with radius r which is $(1-\beta)^{k_r^{\mathrm{inf}}}$. $(1-\beta)^{k_r^{\mathrm{inf}}}$ means the probability of an individual with radius r not being infected by any of its neighbors, where k_r^{inf} is the number of its infected neighbors and $k_r^{\mathrm{inf}} = N\sum_{j=1}^{m} P(r_j) i(r_j, t) r^2\pi/D^2 = N_{\mathrm{I}}(t) r^2\pi/D^2$. Then, the fraction of susceptible individuals with radius r that enter the infected group at time $t+1$ is $s(r, t)[1-(1-\beta)^{k_r^{\mathrm{inf}}}]$ while that of infected individuals with radius r that enter the susceptible group is $\lambda i(r, t)$. Therefore, the complete mean-field model is

$$\begin{aligned} \frac{\mathrm{d}i(r, t)}{\mathrm{d}t} &= -\lambda i(r, t) + s(r, t)[1-(1-\beta)^{k_r^{\mathrm{inf}}}], \quad r = r_1, ..., r_m, \\ \frac{\mathrm{d}s(r, t)}{\mathrm{d}t} &= \lambda i(r, t) - s(r, t)[1-(1-\beta)^{k_r^{\mathrm{inf}}}], \quad r = r_1, ..., r_m. \end{aligned} \tag{3}$$

3. Threshold analysis

In this section, we provide a theoretical analysis on the epidemic threshold of our mean-field model. Since $s(r,t) + i(r,t) = 1$, (3) can be rewritten as

$$\frac{\mathrm{d}i(r, t)}{\mathrm{d}t} = -\lambda i(r, t) + [1 - i(r, t)][1-(1-\beta)^{k_r^{\mathrm{inf}}}], \quad r = r_1, ..., r_m. \tag{4}$$

When the ratio β/λ is fixed, different pais of β and λ only affect the definition of the time scale of the disease propagation [28, 38]. Therefore, we can set β and λ to be sufficiently small so as to use the approximation $[1-(1-\beta)^{k_r^{\mathrm{inf}}}] \to \beta k_r^{\mathrm{inf}}$. In other words, we can maintain the same results (except for the time scale) as long as the ratio β/λ is fixed. This approximation has been widely adopted in [28, 37, 39, 40]). Thus, we obtain

$$\frac{\mathrm{d}i(r, t)}{\mathrm{d}t} = -\lambda i(r, t) + [1 - i(r, t)]\beta k_r^{\mathrm{inf}}, \quad r = r_1, ..., r_m. \tag{5}$$

By letting the right-hand side of (5) be zero, we have a steady-state shown as follow

$$i_r^* = \frac{\beta k_r^{\mathrm{inf}}}{\beta k_r^{\mathrm{inf}} + \lambda} = \frac{\beta N \frac{r^2\pi}{D^2} \sum_{j=1}^{m} P(r_j) i_r^*}{\beta N \frac{r^2\pi}{D^2} \sum_{j=1}^{m} P(r_j) i_r^* + \lambda}. \tag{6}$$

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We denote $I(t) \in [0, 1]$ as the fraction of infected individuals among N individuals and I^* as its steady-state value. Obviously,

$$I(t) = \sum_{j=1}^{m} P(r_j) i(r_j, t). \tag{7}$$

By combining (6) and (7) we get a self-consistency equation [4]:

$$I^* = \sum_{j=1}^{m} P(r_j) \frac{\beta N \frac{r_j^2 \pi}{D^2} I^*}{\beta N \frac{r_j^2 \pi}{D^2} I^* + \lambda}. \tag{8}$$

The trivial solution $I^* = 0$ always satisfies (8). A non-zero steady density can be obtained when the right-hand side and the left-hand side of (8), expressed as functions of I^*, cross in the interval $0 < I^* \leqslant 1$, allowing a nontrivial solution. Let

$$f(I^*) = \sum_{j=1}^{m} P(r_j) \frac{\beta N \frac{r_j^2 \pi}{D^2} I^*}{\beta N \frac{r_j^2 \pi}{D^2} I^* + \lambda}, \tag{9}$$

and it is easy to know that this corresponds to the following inequality:

$$\left. \frac{\mathrm{d}f(I^*)}{\mathrm{d}I^*} \right|_{I^*=0} > 1 \Longrightarrow \sum_{j=1}^{m} P(r_j) N \frac{r_j^2 \pi}{D^2} \frac{\beta}{\lambda} = \rho \frac{\beta}{\lambda} \sum_{j=1}^{m} P(r_j) r_j^2 \pi > 1. \tag{10}$$

Hence, the threshold of infection probability β_c could be written as

$$\beta_c = \frac{\lambda}{\rho \pi \sum_{j=1}^{m} P(r_j) r_j^2}. \tag{11}$$

The threshold in the HM model with homogeneous radius have been proposed by previous works [22, 24, 28, 37]

$$\beta_c = \frac{\lambda}{\rho \pi r^2}. \tag{12}$$

Obviously, when $m = 1, P(r) = 1$ which means all individuals share the same susceptibility radius, (11) degenerates into (12).

Therefore, the epidemic outbreaks when $\beta > \beta_c$, while in the condition that $\beta < \beta_c$ the infection will end up zero and the infectious disease will not be epidemic. From (11), we can see that the infection threshold can only correlate with the density of moving individuals ρ, the recovery rate λ and the second order moment of radius $\Sigma P(r_j) r_j^2$. In previous works, usually, λ is set as a constant and we do so in this work.

Intuitively, when other parameters are given, the larger the population density is, the smaller the infection threshold will be, and it is easier for disease to spread. Another interesting result underneath (11) is that heterogeneous interaction radius lowers the infection threshold compared with identical radius even if the mean value of the distribution of radius is equal to the identical radius i.e. $\sum_{j=1}^{m} P(r_j) r_j^2 \geqslant \bar{r}^2$, where

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$\bar{r} = \sum_{j=1}^{m} P(r_j) r_j$, is always satisfied, when $r_j > 0, \forall\, j = 1, ..., m$ according to Jensen's inequality.

Remark 3.1. We find that when the mean value of radius distribution $\bar{r}$ and m is fixed, the larger the variance of radius distribution is, the smaller the infection threshold would be. We assume that we have two sets of radius distribution, $P_1(r_i), i = 1, ..., m$, and $P_2(r_j), j = 1, ..., m$. They have the same mean value: $\sum_{i=1}^{m} P_1(r_i) r_i = \sum_{j=1}^{m} P_2(r_j) r_j = \bar{r}$. δ_1 and δ_2 are respectively the variance of distribution set 1 and 2. $\delta_1 = \sum_{i=1}^{m} P_1(r_i)(r_i - \bar{r}^2)$ and $\delta_2 = \sum_{j=1}^{m} P_2(r_j)(r_j - \bar{r}^2)$. By subtracting two variances, we obtain $\delta_1 - \delta_2 = \sum_{i=1}^{m} P_1(r_i) r_i^2 - \sum_{j=1}^{m} P_2(r_j) r_j^2$. Thus, we can conclude a larger variance lowers the infection threshold.

4. Numerical simulations

The epidemic thresholds above are obtained under HM hypothesis. In this section, we first simulate the case when high mobility is not satisfied. In our simulation, the length of the side of the planar space D is set to be 30 while recovery rate λ is a constant, say 0.1. Velocity v is supposed to be 0.1. These parameters are fixed. To be more brief, we will not mention them in the following parts of simulation. We start our simulations with a small number of infected individuals and we follow the system until the number of infected individuals approaches a stable level. Precisely, 5% of individuals is set to be infective at $t = 0$ to provide the seed of the infection, while all the others start from the susceptible state.

During the simulation, if a disease breaks out, the number of infected goes up, and reaches a stable value. A typical case is shown in figure 2, where it can be noticed that at the end of the simulation the number of infected reaches a dynamic equilibrium. In figure 2, we have considered a population of random walkers with $N = 900$, $\rho = 1$,and different values of p_{jump}. Obviously, when the system reaches a stable level, the number of infected individuals increases for increasing values of p_{jump}, which means that for larger p_{jump} the disease exhibits a higher infection density. Also, the case with $p_{\text{jump}} = 1$ is in perfect agreement with the situation of homogeneous mixing and this result backs up the correctness of (3). The same result is also obtained under the condition of identical radius in other works [26, 27, 37, 41]. The effect of p_{jump} on epidemic spreading is frequently investigated by previous works and they state that long-distance jump, due for example to aviation traffic, can strongly influence the propagation of disease among the world [22, 24, 27, 37, 41].

In figure 3, we study how P_{jump} affects the final density. The main increase of final density completes before p_{jump} reaches 0.1. Besides, we could observe that a larger $\sum P(r) r^2$ decreases the final density. When P_{jump} increases, the difference caused by different distribution becomes more evident.

Since long-distance jump has been over studied and a small value of p_{jump} could make the case of random walkers similar to the case under HM hypothesis [27], in later simulations, we will only investigate the case with $p_{\text{jump}} = 1$ and the case of HM hypothesis. By doing so, we could better focus on investigating the effect of heterogeneous radius on epidemic spreading.

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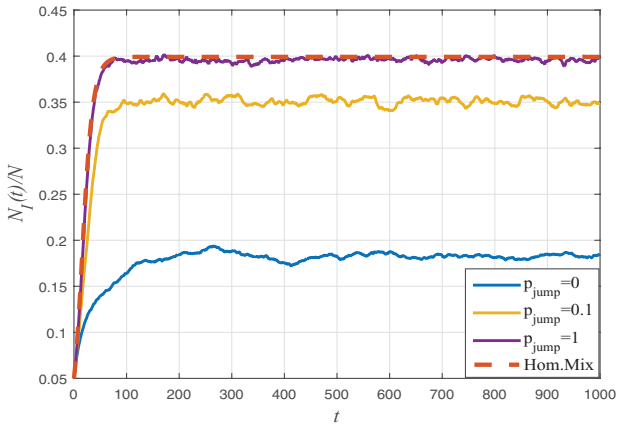

Figure 2. Density $N_I(t)/N$ of infected individuals as a function of time for a system of random walkers with $N = 900$, $\beta = 0.06$, $m = 3$, $r_1 = 0.5$, $r_2 = 1$, $r_3 = 1.5$, and their distribution is $P(r_1) = 0.3$, $P(r_2) = 0.4$, $P(r_3) = 0.3$. Results are averaged over 50 iterations with different initial conditions. The dash line represents the simulation result of (3) i.e. the homogeneous-mixing approximation.

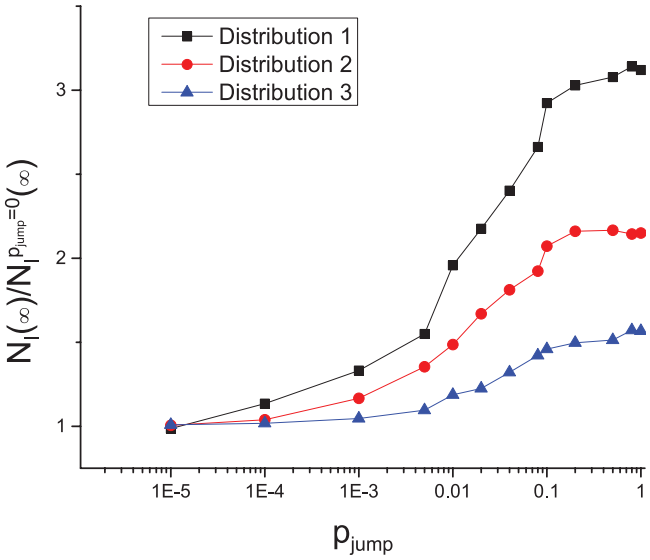

Figure 3. The ratio of final density $N_I(\infty)/N_I^{p_{jump}=0}(\infty)$ of infected individuals as a function of P_{jump} for a system of random walkers with $N = 900$, $\beta = 0.06$, $m = 3$, where $N_I^{p_{jump}=0}(\infty)$ means the final number of infected individuals when the system with $p_{jump} = 0$ becomes stable. We study three distributions: 1. identical interaction radius distribution 2. distribution with $r = [0.5\ 1\ 1.5]$ and $P(r) = [0.3\ 0.4\ 0.3]$ 3. distribution with $r = [0.5\ 1\ 1.5]$ and $P(r) = [0.4\ 0.2\ 0.4]$. Results are averaged over five iterations with different initial conditions.

Further, we investigate the threshold beyond which the disease will outbreak. We could know from figure 4 how the threshold decreases with an increasing ρ. The dash line is plotted according to (11) that we obtain through threshold analysis. Dot scatters are simulated by running (3) and the inverted triangle represents the case of $p_{jump} = 1$ that is derived from mechanism simulation. Figure 4 shows that the relation between ρ

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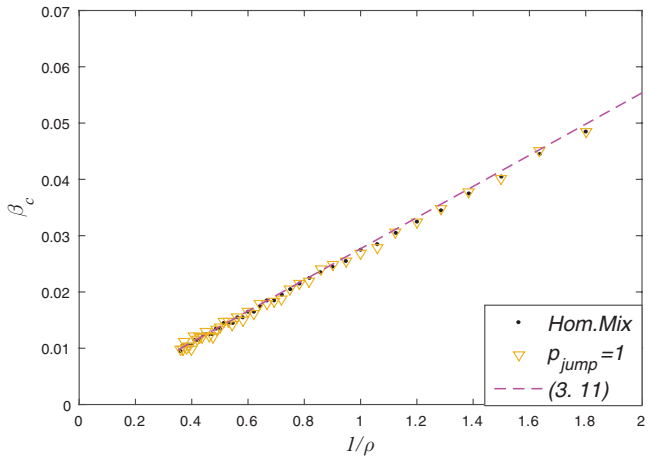

Figure 4. Epidemic threshold as a function of density ρ for a system of random walkers with $m = 3$, $r_1 = 0.5$, $r_2 = 1$, $r_3 = 1.5$, and their distribution is $P(r_1) = 0.3$, $P(r_2) = 0.4$, $P(r_3) = 0.3$. Results are obtained from (11), (3) and the case with $p_{\text{jump}} = 1$. The result of $p_{\text{jump}} = 1$ is averaged over 50 independent runs.

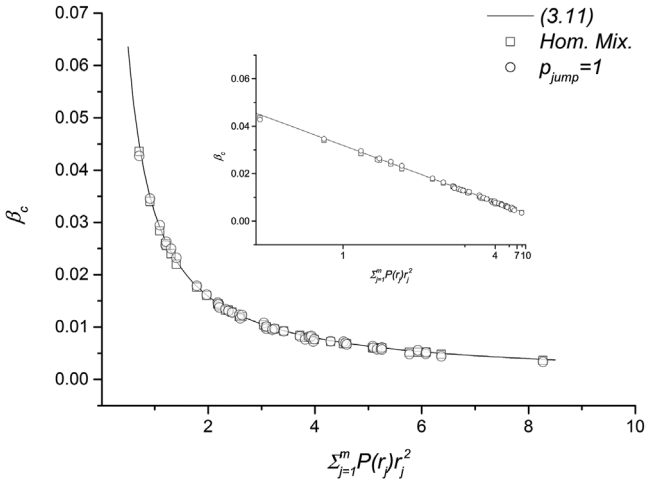

Figure 5. Epidemic threshold as a function of $\sum_{j=1}^{m} P(r_j) r_j^2$ for a system of random walkers with $N = 900$, i.e. $\rho = 1$, $m = 4$ and the values of r_j and $P(r_j)$ are randomly chosen to obtain different values of $\sum_{j=1}^{m} P(r_j) r_j^2$. Results are obtained from (11), (3) and the case with $p_{\text{jump}} = 1$. The result of $p_{\text{jump}} = 1$ is averaged over 50 independent runs.

and β_c we get from epidemic analysis is accurate and $\beta_c \propto \frac{1}{\rho}$. In the case of figure 4, based on that $\lambda = 0.1$, $P(r) = [0.3\ 0.4\ 0.3]$ and $r = [0.5\ 1\ 1.5]$, we conclude that $\beta_c = 0.0277/\rho$.

Also, how the heterogeneous radius affect the epidemic threshold is presented in (11), which shows that $\beta_c \propto 1/\sum_{j=1}^{m} P(r_j) r_j^2$. See figure 5 where the relation between epidemic threshold β_c and $\sum_{j=1}^{m} P(r_j) r_j^2$ is plotted. The line, square and circle, respectively, represent the (11) we get from theoretical analysis, the result obtained by running (3), and the result of mechanism simulation according to the model we defined. Apparently, a larger $\sum_{j=1}^{m} P(r_j) r_j^2$ corresponds to a lower threshold which means the

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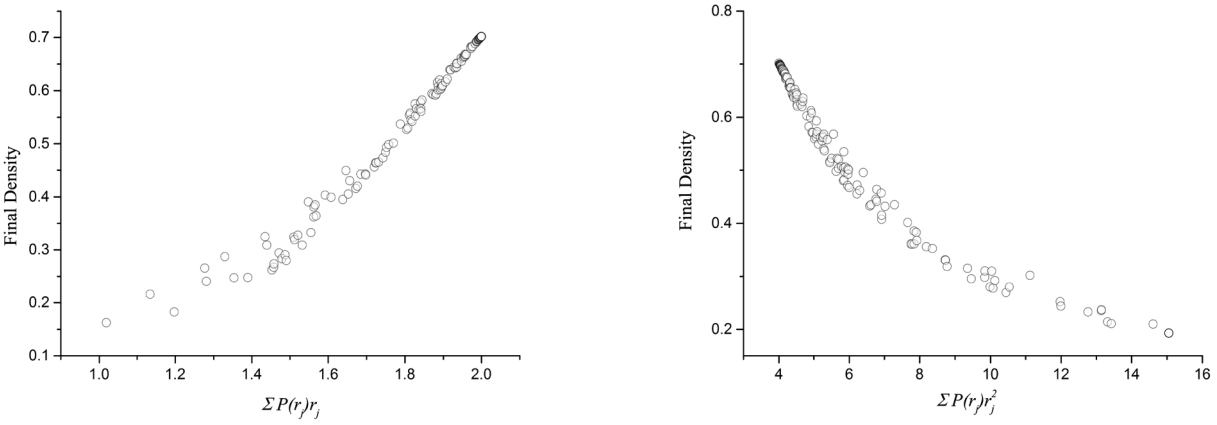

Figure 6. (a) A scatter plot on the density $N_{\mathrm{I}}(\infty)/N$ of infected individuals at the steady state under the change of $\sum_{j=1}^{m} P(r_j)r_j = \bar{r}$ for a system of random walkers with $N = 900$. $m = 2$. $P(r_1)$, $P(r_2)$, r_1, r_2 are randomly chosen under the condition that $P(r_1)r_1^2 + P(r_2)r_2^2 = 4$. Thus, $\sum_{j=1}^{m} P(r_j)r_j^2$ is fixed and $\sum_{j=1}^{m} P(r_j)r_j = \bar{r}$ changes. (b) A scatters plot on the density $N_{\mathrm{I}}(\infty)N$ of infected individuals at the steady state under the change of $\sum_{j=1}^{m} P(r_j)r_j^2$ for a system of random walkers with $N = 900$. $m = 2$. $P(r_1)$, $P(r_2)$. r_1, r_2 are randomly chosen under the condition that $P(r_1)r_1 + P(r_2)r_2 = 2$. Thus, $\sum_{j=1}^{m} P(r_j)r_j = \bar{r}$ is fixed and $\sum_{j=1}^{m} P(r_j)r_j^2$ changes.

larger $\sum_{j=1}^{m} P(r_j)r_j^2$ is, the more easily a disease outbreaks. We have proved in threshold analysis that the heterogeneity of interaction radius lowers the epidemic threshold. Thus, this gives us a guidance of epidemic prevention that if with the same $\bar{r}$, choosing a system with a set of $P(r_j)$ and r_j with lower variance could prevent the outbreaks of disease.

According to (11), as $\sum_{j=1}^{m} P(r_j)r_j^2$ increases, epidemic threshold β_c declines reciprocally. In figure 5, a subfigure with a reciprocal x-axis is plotted which proves the accuracy of (11).

An interesting result is shown in figure 6(a). We let $m = 2$. In figure 6(a), r and $P(r)$ are randomly chosen. We uniformly choose $P(r)$ at the interval of [0.2, 0.8]. r_1 is generated uniformly at the interval $[0, \sqrt{4/P(r_1)}]$. We have known that when $\sum_{j=1}^{m} P(r_j)r_j^2$ is fixed, the change of $\bar{r}$ has no impact on the epidemic threshold. The epidemic threshold is excusively decided by $\sum_{j=1}^{m} P(r_j)r_j^2$, ρ and λ. However, we could observe from figure 6(a) that a larger $\bar{r}$ produces a higher final density even though $\sum_{j=1}^{m} P(r_j)r_j^2$ is fixed.

In figure 6(b), $P(r_1)$ is uniformly chosen at the interval [0.2, 0.8] and r_1 at the interval $[0, 2/P(r_1)]$ under the case of $\sum_{j=1}^{m} P(r_j)r_j = 2$. We could observe that a larger second order origin moment of radius decreases the final density of infection. We have concluded that a larger $\sum P(r)r^2$ decreases β_c. This gives us a guidance that if $\sum P(r)r^2$ cannot be small enough to stop the diseases' outbreak, we could increase it to downsize their prevalence.

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5. Conclusion

In summary, we have studied the effects of diversity in interaction radius on epidemic spreading by proposing a model which separates individuals into a few parts according to their interaction radius. Based on the interaction rule of random walkers and homogeneous mixing theory, an ordinary differential equation was established to model the spreading process of the epidemic. The model is effective in describing the propagation dynamics according to the simulation result. We have obtained the epidemic threshold from which we could find guidance of prevention and control of epidemic outbreaks. The epidemic threshold obtained by theoretical analysis is verified by the result of epidemic threshold studied through simulation. In the final density study, simulation results indicate that when there are two different values of radius, the second order origin moment of radius $\Sigma P(r_j)r_j^2$ affects the epidemic threshold negatively and final density positively. A larger second order origin moment of radius $\Sigma P(r_j)r_j^2$ makes epidemic easier to outbreak. However, it can reduce the scale of infection. This result is supposed to be helpful in both the prevention of outbreak and the control of the scale of infection.

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