The most common friend first immunization*

Fu-Zhong Nian(年福忠)† and Cha-Sheng Hu(胡茶升)

School of Computer & Communication, Lanzhou University of Technology, Lanzhou 730050, *China*

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In this paper, a standard susceptible-infected-recovered-susceptible(SIRS) epidemic model based on the Watts–Strogatz (WS) small-world network model and the Barabsi–Albert (BA) scale-free network model is established, and a new immunization scheme — "the most common friend first immunization" is proposed, in which the most common friend's node is described as being the first immune on the second layer protection of complex networks. The propagation situations of three different immunization schemes — random immunization, high-risk immunization, and the most common friend first immunization are studied. At the same time, the dynamic behaviors are also studied on the WS small-world and the BA scale-free network. Moreover, the analytic and simulated results indicate that the immune effect of the most common friend first immunization is better than random immunization, but slightly worse than high-risk immunization. However, high-risk immunization still has some limitations. For example, it is difficult to accurately define who a direct neighbor in the life is. Compared with the traditional immunization strategies having some shortcomings, the most common friend first immunization is effective, and it is nicely consistent with the actual situation.

Keywords: epidemic, common friend, immunization, SIRS

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1. Introduction

Recently, the complex networks have attracted attention of many researchers who are familiar with the field. The so-called complex networks are large-scale networks with complex topological structures and dynamic behaviors.[1] The complex networks are composed of a large number of nodes standing for individuals, and edges standing for interactions between individuals,[2] such as the internet, social network,[3] wireless communication network, the telecommunication network, friend relationship network, epidemic spreading network,[4,5] and other complex networks. With the rapid development of society, our daily life is closely related to the networks. Therefore, the studies of the structure, characteristics and relationships of networks have played a significant role in our daily life and scientific research.[6–8]

The earliest research of complex networks can be traced back to the problem of seven-bridges of Konigsberg in the eighteenth century. The random graph theory is the key of the complex network theory which was created by Erdos and Renyi in the 1960s. Now, complex networks have been studied widely. In recent years, an important branch of complex networks — the transmission mechanisms and propagation dynamics — has been widely investigated.[5,6,9] Throughout history, any epidemic's outbreak is brought about by the social development of civilization. On the one hand, large-scale epidemics have a dramatic impact on the society. The network becomes an essential thing in our daily life, which promotes the continuous improvement of the modern health system and reduces the threat of epidemics. On the other hand, the increasingly developed social network makes the connection closer among people, which accelerates the great outbreaks of epidemics, such as SARS in 2003, H1N1 flu in 2005,[10] H7N9 in 2013,[11] HIV/AIDS,[12] and mad cow disease,[13] etc. These epidemics bring huge loss to the national economy and disaster to people's lives.[14] Therefore, the presence of epidemics is a problem we have to solve.[15–17]

In 1959, two famous Hungarian mathematicians (Erdos and Renyi) created the ER random graph network model.[18] Like the regular networks, the ER random graph can be classified as homogeneous complex networks, in which the degree of each node in the network is approximately equal to the average degree of the network. By the 1990s, with the rapid development of the computer, internet and other technologies, the advents of various databases enables people to obtain a variety of topological structures of real networks. In this context, two landmark models, small-world network model[19,20] and scale-free network model[21] have been proposed. The characteristics of small-world networks show that the distance between any two nodes is much smaller than the network size, and similar to the six degree separation theory in sociology. The degree distribution is a power-law one on scale-free networks, in which exist some nodes of larger degree. The small-world and scale-free network models were two major statistical topological models at the end of the twentieth century.[22] Therefore, many problems are investigated on the two prototype complex networks.

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†Corresponding author. E-mail: gdnfz@lut.cn

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Meanwhile, different propagation models are adopted in the various propagation behaviors. Classical propagation models were proposed by predecessors: susceptible-infected (SI),[23] susceptible-infected-susceptible(SIS),[24,25] susceptible-infected-recovered(SIR),[26] etc. Based on the above models, a lot of epidemic propagation models which are closer to the realistic ones are put forward, including susceptible-infected-recovered-susceptible(SIRS).[27] The transmission cases of three kinds of immunization schemes are studied by using the SIRS model.

Currently, with constant communications among people, more and more epidemics are spread to a wider range, which has brought huge influence to the human's life and the economy. Therefore, designing a highly effective immunization strategy has been of wide concern.[28–33] Typical immunization strategies are as follows: random immunization,[34] targeted immunization,[35] acquaintance immunization,[36] and other immunization strategies.[37,38] Random immunization is a simple strategy, which completely randomly selects a part of nodes over the whole network, and carries out immunization, but it is almost immunization of all the nodes to obtain a better immune effect. So, random immunization is not impractical. Targeted immunization is an effective strategy, which chooses a small number of key nodes to immunize first. However, the only drawback is that it needs to know the topology of the entire network. Acquaintance immunization is to vaccinate random acquaintances of random nodes. It is obvious that the nodes with larger degree are easier to select. Other immunization strategies contain the double acquaintance immunization,[39] the complex vaccination adoption mechanism,[38] etc. Since these classic immunization strategies have some drawbacks, the latest immunization strategies are presented, respectively, with high-risk immunization[40] and the two-step high-risk immunization.[41] High-risk immunization is an effective strategy, which treats direct susceptible neighbors with infected nodes as high-risk groups, and controls the spread of epidemics by immunizing high-risk groups. The two-step high-risk immunization is designed, which immunizes not only partial high-risk nodes, but also partial indirect neighbors of infected nodes. In this paper, a new immune strategy — the most common friend first immunization — based on the topology research of complex networks, is proposed. High-risk immunization must know all susceptible individuals whose neighbors have been infected, and these individuals are called "high-risk individuals" or "direct neighbors". In this paper, the most common friend first immunization only needs to know some direct neighbors and its neighbors. In real life, it is difficult to define all direct neighbors accurately, but it is very easy to find some direct neighbors and their neighbors. It is evident that the proposed method is more feasible than the high-risk immunization. Therefore, the most common friend first immunization is more operational, and is more suitable in real life.

The idea of the most common friend first immunization is described in Fig. 1. Set i as the infected node (i.e., red circle), the direct susceptible neighbor nodes adjacent to i as high-risk nodes (i.e., the green circle), the nodes of marking number 1, 2, and 3 as indirect susceptible neighbors. The indirect neighbor labeled with Arabic number indicates that an indirect neighbor has common friend or direct neighbors' total number. For example, the indirect neighbor marked 3 indicates that it has 3 high-risk nodes. In other words, this node has the most common friend, so it is the first immunization. One piece of advice is that each node is a susceptible node except the infected node.

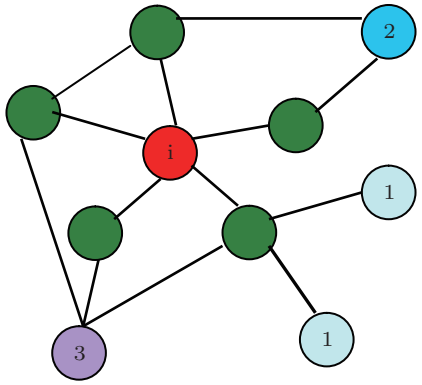


Fig. 1. (color online) Sketch of the most common friend first immunization.

The rest of this article is organized as follows. In Section 2, the most common friend first immunization on WS small-world network is introduced. In Section 3, the most common friend first immunization on BA scale-free network is described. In Section 4, the results and discussion of computer simulation on WS small-world network are presented. In Section 5, the results and discussion of computer simulation on BA scale-free network are given. Finally, according to the theoretical and simulation results, we can draw corresponding conclusions.

2. The most common friend first immunization on WS small-world network

In 1998, Watts and Strogatz (WS) put forward the concept of small-world networks. Meanwhile, the WS small-world network model was also established, and it is constructed in the following steps: (i) starting from the rule graph: the starting point is a ring with N nodes symmetrically connected with its $2K$ nearest neighbors, (ii) random rewiring: for every node, each link connected to a clockwise neighbor is rewired to a randomly chosen node with probability p, and preserved with probability $1-p$.

In the SIRS model, each node represents an individual, and each edge represents direct contact between two individuals. The number of all edges connected to a node is defined as

the degree of the node. Without considering isolated individuals, latent individuals, the birth and death rate of individuals in the propagation of epidemics, there are three node statuses, respectively, with susceptible status $S(t)$, infected status $I(t)$, and recovered status $R(t)$. The infection mechanism of the classic SIRS model is showed in Fig. 2.

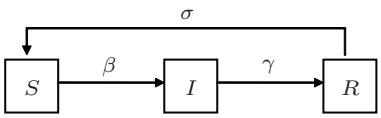


Fig. 2. Sketch of SIRS.

The disease propagation process is described as follows. At each time step, a susceptible individual S is infected into an infected individual with probability β, an infected individual I is cured into a recovered individual with probability γ, while a recovered individual R becomes a susceptible individual, due to losing immunity, with probability σ. Therefore, the model's effective transmission rate is $\lambda = \beta/\gamma$. In this paper, the probability with which any given node is a special node's neighbor is defined as ω, and the probability per unit time in which one node obtains immunity by vaccinating is defined as o; At the same time, the proportion at which the direct neighbors of the most common friend are immune is defined as p, and the proportion at which the indirect neighbors are immune is defined as q. The degree distribution of homogeneous network (ER random graph network and WS small-world network) has a peak at the average degree $\langle k\rangle$, but the exponent declines when the value of k is significantly smaller than $\langle k\rangle$, and far more than $\langle k\rangle$. So, we suppose that each node degree k_i is approximately equal to the average degree $\langle k\rangle$. Based on mean-field theory, when the network scale tends to infinity, the following dynamic equations can be obtained by ignoring the density correlation among different nodes:

$$\begin{aligned}
\frac{\mathrm{d}S(t)}{\mathrm{d}t} &= -\lambda(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle S(t)I(t) + \sigma R(t),\\
\frac{\mathrm{d}I(t)}{\mathrm{d}t} &= \lambda(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle S(t)I(t) - I(t),\\
\frac{\mathrm{d}R(t)}{\mathrm{d}t} &= I(t) - \sigma R(t). \qquad (1)
\end{aligned}$$

Suppose that o_{ij} is the number of common friends between the infected node i and indirect node j, $\sum_{j=1}^{k} o_{ij}$ is defined as the total number of common friends between the infected node i and all indirect nodes j, and k as the total number of indirect nodes. Therefore, $\xi = o_{ij}/\sum_{j=1}^{k} o_{ij}$ is defined as the correlation strength between the infected node i and indirect node j. Let Γ be a set of indirect neighbor node j, then

$$\psi_{\mathrm{j}} = \begin{cases} 1, & \text{the largest } \xi, j \in \Gamma \\ 0, & \text{other circums tances} \end{cases}$$

will be considered as the immune coefficient. Certainly, the largest ξ refers to being the first vaccinated.

Here, $S(t)$, $I(t)$, and $R(t)$, respectively, represent the average densities of susceptible individuals, infected individuals and recovered individuals. The first term on the right-hand side of the first equation denotes the average density of newly infected nodes generated by each infected node. The second term on the right-hand side of the first equation means the recovered nodes losing immunity with rate σ. The second term on the right-hand side of the second equation represents the infected nodes recovering with rate γ.

The most significant prediction of SIR is the presence of a nonzero epidemic threshold λ_{c}. If the value of λ is above λ_{c}, then the value of $R(t)$ is above zero; it means that the infected individuals will spread infectious diseases, and the total number of infected individuals in the whole network is finally stabilized to a certain equilibrium state. If the value of λ is below λ_{c}, then the value of $R(t)$ is equal to zero. It means that infectious diseases cannot spread, and the total number of infected individuals is infinitesimally small in the limit of very large populations. The $S(t)$, $I(t)$, and $R(t)$ obey the normalization principle: $S(t)+I(t)+R(t)=1$, the initial conditions of this equation are $R(t)=0$, $S(t)\approx 1$, and $I(t)\approx 0$.

When the final infected density would reach a steady value, equation (1) satisfies the non-epidemic stationary conditions: $\mathrm{d}S(t)/\mathrm{d}t=0$, $\mathrm{d}I(t)/\mathrm{d}t=0$, $\mathrm{d}R(t)/\mathrm{d}t=0$, and $I(t)=0$. The condition $I(t)=0$ shows that non-epidemic spreads, and it also indicates that the numbers of susceptible individuals, infected individuals and recovered individuals will no longer change. Bringing the equations of non-epidemic stationary conditions into Eq. (1), we obtain

$$\begin{aligned}
&-\lambda(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle S(t)I(t) + \sigma R(t) = 0,\\
&\lambda(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle S(t)I(t) - I(t) = 0,\\
&I(t) - \sigma R(t) = 0. \qquad (2)
\end{aligned}$$

From the second equation of Eq. (2), we obtain

$$S(t) = \frac{1}{\lambda(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle}. \qquad (3)$$

Substitute $S(t)+I(t)+R(t)=1$ into the third equation of Eq. (2), we obtain

$$-I(t) + \sigma[1-S(t)-I(t)] = 0. \qquad (4)$$

Substitute Eq. (3) into Eq. (4), we obtain

$$\begin{aligned}
&-I(t) + \sigma(1-I(t)\\
&-\frac{1}{\lambda(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle}) = 0. \qquad (5)
\end{aligned}$$

Because of the static condition $I(t)=0$, we obtain

$$\lambda = \frac{1}{(1-p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)\langle k\rangle}. \qquad (6)$$

When $\sigma = 0$, the SIRS model is equivalent to the SIS model. Substituting $\sigma = 1$ into Eq. (6), we obtain the non-zero epidemic threshold is

$$\lambda_c = \frac{1}{(1 - p\omega o - q\psi_j \omega^2 o)\langle k\rangle}.$$

When the vaccinating rate $o = 0$, $\lambda_c = 1/\langle k\rangle$.

From Eq. (6), we can see that the non-zero epidemic threshold is associated with the average degree $\langle k\rangle$ of the network, the immune probability o, the losing immunity probability σ, the proportion p of the direct neighbors, and the proportion q of the indirect neighbors.

3. The most common friend first immunization on BA scale-free network

Some phenomena of complex networks, especially of social networks, cannot be explained by WS small-world nor ER random graph network model. Because the degree distributions of the two network models obey the Poisson distribution, but the degree distributions of many networks in reality obey power-law distribution. For example, the transportation hub cities in China are limited in number, and these hub cities can reach many other cities, while the number of routes between the majority of common cities and other cities is relatively small. Another example is the case of the social network. In social networks, the public figures often have wide social circles and strong influence, while the social circles of ordinary people are relatively narrow and influence is relatively weak. To solve these problems, Barabasi and Albert proposed a scale-free network model based on the analysis of a large number of network data in 1999. The scale-free network model features a small number of hub nodes existing in the network. This model is proposed on the basis of two hypotheses: one is the growth pattern of the network: many networks grow, such as the production of new web pages in the internet, the addition of new friends in a friend's net, etc; the other is the preferential attachment mode: as new nodes join, they tend to be connected with more nodes. For instance, the new web pages generally link to the well-known Web pages, etc. So, SIRS' the most common friend first immunization epidemic model based on the scale-free network model is described as follows: constructing a BA scale-free network can lead to the growth and preferential connection. After enough iterations, we obtain a network composed of N nodes with degree distribution $P(k) = k^{-3}$ and the average degree $\langle k\rangle = 2$ m. According to mean-field theory, the dynamic equations of the BA scale-free network can be expressed as

$$\frac{\mathrm{d}S_k(t)}{\mathrm{d}t} = -\lambda\left(1 - \frac{p\omega o}{\sigma - \dfrac{q\psi_j\omega^2 o}{\sigma}}\right)kS_k(t)\Theta(t) + \sigma R_k(t),$$

$$\frac{\mathrm{d}I_k(t)}{\mathrm{d}t} = \lambda\left(1 - \frac{p\omega o}{\sigma - \dfrac{q\psi_j\omega^2 o}{\sigma}}\right)kS_k(t)\Theta(t) - I_k(t),$$

$$\frac{\mathrm{d}R_k(t)}{\mathrm{d}t} = I_k(t) - \sigma R_k(t). \tag{7}$$

Here, $S_k(t)$, $I_k(t)$, and $R_k(t)$, respectively, represent the densities of susceptible, infected, and recovered vertex of degree k. In Eq. (7), the first term on the right-hand side of the first equation represents the average density of fresh infected nodes generated by each infected node. The second term on the right-hand side of the first equation denotes the nodes losing immunity with rate σ. The first term on the right-hand of the third equation refers to the infected nodes recovering with rate γ.

In the BA scale-free network, the probability $\Theta(t)$ with which any given link points to an infected node can be expressed as

$$\Theta(t) = \frac{\sum_k kP(k)I_k(t)}{\sum_s sP(s)} = \frac{1}{\langle k\rangle}\sum_k P(k)I_k(t). \tag{8}$$

Here, $sP(s)$ is proportional to the probability with which a link points to a node with s links. That is to say, a randomly chosen link is more likely to be connected to an infected node with high connectivity.

In the BA scale-free network, $S_k(t)$, $I_k(t)$, and $R_k(t)$ obey the normalization principle: $S_k(t) + I_k(t) + R_k(t) = 1$. When there is no epidemic propagation in the network, $\mathrm{d}S_k(t)/\mathrm{d}t = 0$, $\mathrm{d}I_k(t)/\mathrm{d}t = 0$, $\mathrm{d}R_k(t)/\mathrm{d}t = 0$, and $I_k(t) = 0$. Bringing this equation into Eq. (7), we obtain

$$-\lambda(1 - p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)kS_k(t)\Theta(t) + \sigma R_k(t) = 0. \tag{9}$$

Combine the above normalization equations with Eq. (9), then we will obtain

$$S_k(t) = \frac{\sigma I_k(t) - \sigma}{-\lambda(1 - p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)k\Theta(t) - \sigma}. \tag{10}$$

From the second equation of Eq. (7), we obtain

$$S_k(t) = \frac{I_k(t)}{\lambda(1 - p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)k\Theta(t)}. \tag{11}$$

Substitute Eq. (11) into Eq. (10), we obtain

$$I_k(t) = \frac{\lambda(1 - p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)k\Theta(t)\sigma}{(1+\sigma)\lambda(1 - p\omega o/\sigma - q\psi_j\omega^2 o/\sigma)k\Theta(t) + \sigma}. \tag{12}$$

From Eq. (12), the solution $I_k(t) = 0$ and $\Theta(t) = 0$ always satisfies Eq. (12). For obtaining a non-zero static solution ($I_k(t) \neq 0$), we will see both sides of Eq. (12) as a function $G(\Theta)$ ($0 \prec \Theta \leq 1$), which allows a non-trivial solution. Therefore, it requires the following inequality to hold true

$$\frac{\mathrm{d}G(\Theta)}{\mathrm{d}\Theta}|_{\Theta=0} \geq 1. \tag{13}$$

In other words,

$$\left.\frac{\mathrm{d}\left\{\frac{1}{\langle k\rangle}\sum_k kP(k)\times\left\{\frac{\lambda(1-p\omega o/\sigma)k\Theta(t)\sigma}{(\sigma+1)\lambda(1-p\omega o/\sigma-q\psi_j\omega^2 o/\sigma)k\Theta(t)+\sigma}\right\}\right\}}{\mathrm{d}\Theta}\right|_{\Theta=0} = \frac{\lambda}{\langle k\rangle}\sum_k k^2P(k)(1-p\omega o/\sigma-q\psi_j\omega^2 o/\sigma)\geq 1.$$

Let ω be the probability with which any given node is a neighbor of some specific node, then we will have

$$\omega = \frac{kP(k)}{N\langle k\rangle}. \tag{14}$$

Here, $P(k)=2m^2/k^3$.

Substitute Eq. (14) and this equation into inequality (13), then we will obtain

$$\begin{aligned}
&\frac{\lambda}{\langle k\rangle}\sum_k k^2P(k)(1-p\omega o/\sigma-q\psi_j\omega^2 o/\sigma)\\
&=\frac{\lambda}{\langle k\rangle}\left(\sum_k k^2P(k)-\sum_k k^2P(k)p\omega o/\sigma\right.\\
&\quad\left.-\sum_k k^2P(k)q\psi_j\omega^2 o/\sigma\right)\\
&=\frac{\lambda}{\langle k\rangle}\left[\langle k^2\rangle-\sum_k k^2P(k)\frac{op}{\sigma}\frac{kP(k)}{N\langle k\rangle}\right.\\
&\quad\left.-\sum_k k^2P(k)\frac{oq}{\sigma}\psi_j\left(\frac{kP(k)}{N\langle k\rangle}\right)^2\right]\\
&=\frac{\lambda}{\langle k\rangle}\left[\langle k^2\rangle-\sum_k k^2P(k)\frac{op}{\sigma}\frac{k2\mathrm{m}^2}{N\langle k\rangle k^3}\right.\\
&\quad\left.-\sum_k k^2P(k)\frac{oq}{\sigma}\psi_j\left(\frac{k2\mathrm{m}^2}{N\langle k\rangle k^3}\right)^2\right]\\
&=\frac{\lambda}{\langle k\rangle}\left[\langle k^2\rangle-\frac{op}{\sigma}\frac{2\mathrm{m}^2}{N\langle k\rangle}-\frac{oq}{\sigma}\psi_j\frac{4m^4}{N^2\langle k\rangle^2k^2}\right]\geq 1.
\end{aligned}\tag{15}$$

When equation (15) is taken to be an equation, we obtain

$$\frac{\lambda_\mathrm{c}}{\langle k\rangle}\left[\langle k^2\rangle-\frac{op}{\sigma}\frac{2m^2}{N\langle k\rangle}-\frac{oq}{\sigma}\psi_j\frac{4m^4}{N^2\langle k\rangle^2k^2}\right]=1. \tag{16}$$

From the above equation, we obtain

$$\lambda_\mathrm{c}=\frac{\langle k\rangle}{\left[\langle k^2\rangle-\frac{op}{\sigma}\frac{2m^2}{N\langle k\rangle}-\frac{oq}{\sigma}\psi_j\frac{4m^4}{N^2\langle k\rangle^2k^2}\right]}. \tag{17}$$

When $\sigma=1$ and $o=0$, $\lambda_\mathrm{c}=\langle k\rangle/\langle k^2\rangle$. Here,

$$\begin{aligned}
\langle k\rangle&=\sum_k kP(k)=\sum_k 2m^2/k^2=2m^2\sum_k\frac{1}{k^2},\\
\langle k^2\rangle&=\sum_k k^2P(k)=\sum_k k^2 2m^2/k^3=2m^2\sum_k\frac{1}{k}.
\end{aligned}$$

Substitute the above equation into Eq. (17), then we will obtain

$$\lambda_\mathrm{c}=\frac{2m^2\sum_k\frac{1}{k^2}}{2m^2\sum_k\frac{1}{k}-\frac{op}{\sigma}\frac{1}{N\sum_k\frac{1}{k^2}}-\frac{oq}{\sigma}\psi_j\frac{1}{N^2\left(\sum_k\frac{1}{k^2}\right)^2k^2}}. \tag{18}$$

So,

$$\begin{aligned}
\frac{1}{\lambda_\mathrm{c}}&=\frac{2m^2\sum_k\frac{1}{k}-\frac{op}{\sigma}\frac{1}{N\sum_k\frac{1}{k^2}}-\frac{oq}{\sigma}\psi_j\frac{1}{N^2\left(\sum_k\frac{1}{k}\right)^2}}{2m^2\sum_k\frac{1}{k^2}}\\
&=\frac{\sum_k\frac{1}{k}}{\sum_k\frac{1}{k^2}}-\frac{op}{2N\mathrm{m}^2\left(\sum_k\frac{1}{k^2}\right)^2\sigma}\\
&\quad-\frac{oq\psi_j}{2N^2\mathrm{m}^2\left(\sum_k\frac{1}{k}\right)^2\sum_k\frac{1}{k^2}\sigma}.
\end{aligned}\tag{19}$$

In the continuous k approximation, we calculate

$$\begin{aligned}
\sum_k\frac{1}{k}&\to\int_m^M\frac{1}{k}\mathrm{d}k=\ln\frac{M}{m},\\
\sum_k\frac{1}{k^2}&\to\int_m^M\frac{1}{k^2}\mathrm{d}k=\frac{1}{m}-\frac{1}{M}=\frac{M-m}{mM}.
\end{aligned}\tag{20}$$

Here, M is the maximum degree, substitute Eq. (20) into Eq. (19), then we will obtain

$$\begin{aligned}
\frac{1}{\lambda_\mathrm{c}}&\approx\frac{\ln\frac{M}{m}}{\frac{M-m}{mM}}-\frac{op}{2Nm^2\sigma\left(\frac{M-m}{mM}\right)^2}\\
&\quad-\frac{oq\psi_j}{2N^2m^2\left(\ln\frac{M}{m}\right)^2\frac{M-m}{mM}\sigma}\\
&=\frac{\mathrm{m}M\ln\frac{M}{m}}{M-m}-\frac{oM^2p}{2N\sigma(M-m)^2}\\
&\quad-\frac{oMq\psi_j}{2N^2m\sigma(M-m)\left(\ln\frac{M}{m}\right)^2}.
\end{aligned}\tag{21}$$

When M is big enough, $M-m\approx M$. Substitute this equation into Eq. (21), then we will obtain

$$\frac{1}{\lambda_\mathrm{c}}=m\ln\frac{M}{m}-\frac{o}{2N\sigma}\left(p+\frac{q\psi_j}{2Nm(\ln\frac{M}{m})^2}\right). \tag{22}$$

From Eq. (22), we can see that the non-zero epidemic threshold is associated with the number of the existing nodes (m), the immune probability (o), the losing immunity probability (σ), network size (N), the proportion of direct neighbors (p), and the proportion of indirect neighbors (q).

4. Results and discussion of computer simulation on WS small-world network

From Eq. (6), we can see that the average degree $\langle k \rangle$ of the network, the immune probability o, the losing immunity probability σ, the proportion of the direct neighbors p and the proportion of the indirect neighbors q affect the dynamic behaviors of epidemics. So, in this part, some sensitivity analyses are performed in terms of the model parameters.

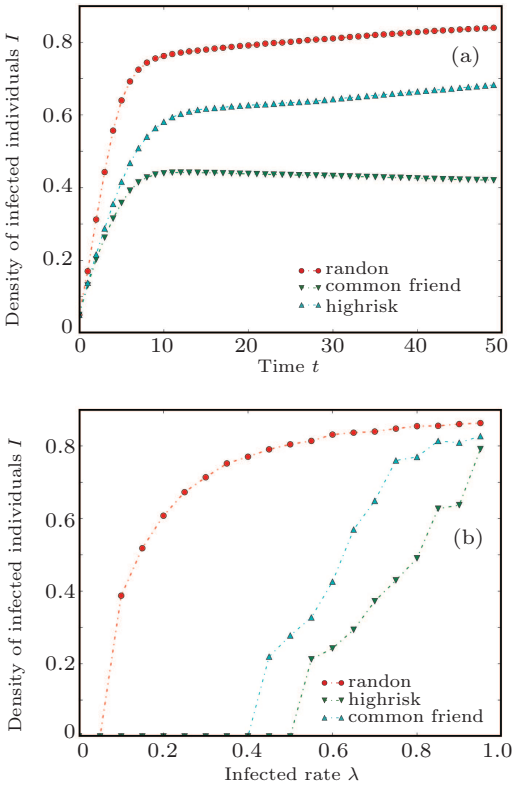


Fig. 3. (color online) (a) Time-dependent and (b) infected-rate-dependent densities of infected individuals, respectively, for random immunization, high-risk immunization, and the most common friend first immunization on WS small-world network ($N = 2000$, $\langle k \rangle = 4$, and the rewiring rate is 0.1).

Based on the above analysis of mathematical theory, the corresponding experiments are also testified. Without loss of generality, a small-world network model is constructed: network scale $N = 2000$, the average degree $\langle k \rangle = 4$, and the rewiring rate is 0.1. At the beginning, 5% of the susceptible nodes are infected. In each iteration, the susceptible node has the same probability with which it is infected by its neighbors. The prevalence is computed by averaging over 50 different starting configurations, performed on 20 different realizations of the network. Figure 3(a) shows the changing of infected density in a homogeneous network, respectively, with random immunization, high-risk immunization and the most common friend first immunization scheme. From Fig. 3(a), we can see that random immunization is located on the top, which indicates that the infected density is the largest when non-epidemic spreads. So, random immunization's effect is the worst. The high-risk immunization is located at the bottom, which indicates that the infected density is the least when non-epidemic spreads, and its immune effect is the best. The most common friend first immunization is located in the middle, which demonstrates that the immune effect is worse than the high-risk immunization. It is difficult to accurately define who a direct neighbor in the life is, but it is rather easy to find some direct neighbors and its neighbors. Hence, the most common friend first immunization is more suitable to the actual case.

Figure 3(b) shows the variations of the density of infected individuals with infected rate for three immunization strategies. Figure 3(b) indicates that high-risk immunization's effect is best, and the most common friend first immunization's effect is slightly worse than the high-risk immunization's, but better than the random immunization's. Because the high-risk nodes are immune, the high-risk immunization can cut off spreading routes from the root, which reduces the spread of epidemics. So, the immune effect of high-risk immunization is the best. The random immunization randomly immunizes the nodes of networks, and it has more transmission routes, so it is difficult to curb the spread of epidemics. Hence, random immunization's effect is the worst. However, the most common friend first immunization effectively cuts off transmission routes of epidemics. Compared with the high-risk immunization, the most common friend first immunization relatively cuts off dispersed transmission routes, and the range of epidemic transmission is relatively wide. Therefore, the most common friend first immunization's effect is slightly poorer than high-risk immunization's.

Figure 4 shows the variations of densities of infected individuals with infected rate infected density for $o = 0.05$, $o = 0.08$ and $o = 0.1$. Figure 4 points out that as the vaccinating rate o becomes larger, the effect of immunization is better. This is also consistent with the actual case.

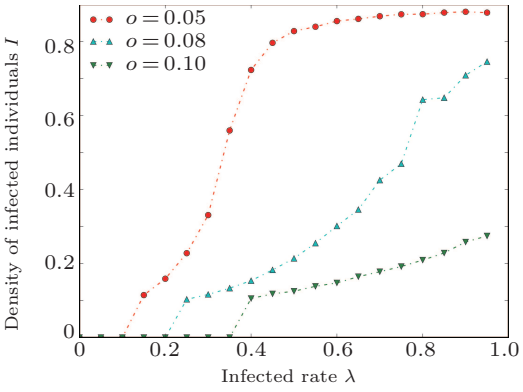


Fig. 4. (color online) Variations of the density of infected individuals with infected rate for the most common friend first immunization on WS small-world network ($N = 2000$, $\langle k \rangle = 4$, the rewiring rate is 0.1), respectively, in the cases of $o = 0.05$, $o = 0.08$, and $o = 0.1$

Figure 5 shows the variations of the density of infected individuals with infected rate for the most common friend first immunization at different values of immune proportion p of direct neighbors and immune proportion q of indirect neighbors. In order to compare with other immunization strategies,

the same number of immune individuals is selected for each immunization strategy. The parameters p and q are not independent of each other, but $p+q=1$, which means that the sum of the number of immune direct and indirect neighbors is equal to the total number of immunization. From Fig. 5, when p decreases from 0.9 to 0.1 and q increases from 0.1 to 0.9, the effect of immunization becomes worse. One significant conclusion can be drawn from Eq. (6): λ_c increases with p or q increasing. Because p and q are independent of each other in Eq. (6), this is not contradictory with the simulation results.

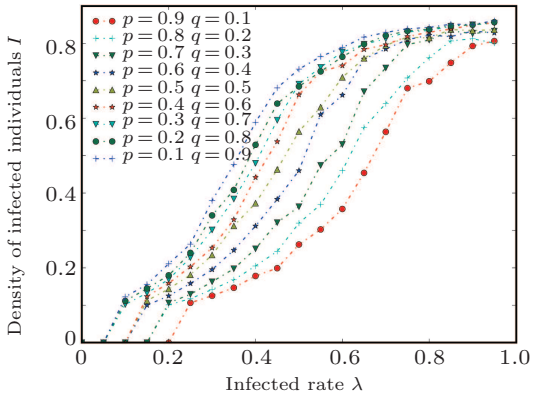


Fig. 5. (color online) Variations of density of infected individuals with infected rate for the most common friend first immunization on WS small-world network ($N=2000$, $\langle k\rangle=4$, the rewiring rate is 0.1), respectively, at different values of p and q (when p ranges from 0.9 to 0.1 and q from 0.1 to 0.9).

5. Results and discussion of computer simulation on BA scale-free network

From Eq. (22), it can be seen that the immune probability o, the losing immunity probability σ, network scale size N, the proportion p of direct neighbors, and the proportion q of indirect neighbors affect the epidemic threshold. So, in this section, some sensitivity analyses are performed in terms of the model parameters.

A scale-free network model is constructed with the following parameters: network size $N=2000$ and the initial node size $m=5$. At the beginning, 5% of the susceptible nodes are infected. In each iteration, the susceptible node has the same probability with which it is infected by its neighbors. The prevalence is computed by averaging over 50 different starting configurations, performed on 20 different realizations of the network. From Fig. 6, under the same conditions ($\lambda=0.7$, $\sigma=0.02$, $o=0.18$, $p=0.8$, and $q=0.2$), the high-risk immunization's effect is the best, and the most common friend first immunization's effect is slightly worse than the high-risk immunization's, but better than the random immunization's. This is consistent with the theoretical analysis.

According to the theory of critical value of transmission, the transmission rate is larger than the critical value, and the infected individuals can spread. As described in Fig. 7, the critical value of high-risk immunization is larger than that of the most common friend first immunization, also larger than that of random immunization. Consequently, the effect of high-risk immunization is the best. But in real life, it is difficult to find out all the high-risk nodes. In addition, the most common friend first immunization strategy can be immune indirect node having the most public friend in turn, and its effect of immunization is better. So, the most common friend first immunization is economic and effective. Moreover, the critical value of random immunization on BA scale-free network is 0, which means that it needs to be immune to all the nodes of network mostly to guarantee the final elimination of the epidemics.

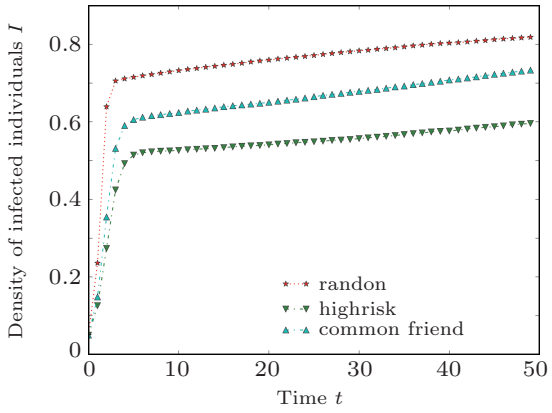


Fig. 6. (color online) Time-dependent densities of infected individuals, respectively, for random immunization, high-risk immunization and the most common friend first immunization on BA scale-free network ($N=2000$, $m=5$).

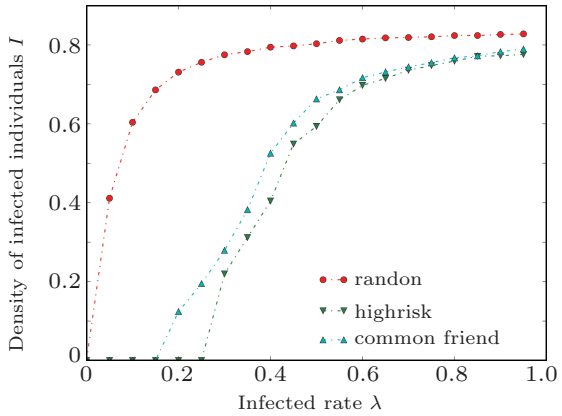


Fig. 7. (color online) Variations of density of infected individuals with infected rate, respectively, for random immunization, high-risk immunization, and the most common friend first immunization on BA scale-free network ($N=2000$, $m=5$).

Figure 8 shows the variations of the density of infected individuals for the most common friend first immunization in the heterogeneous network, with the initial node size $m=4$, $m=6$, and $m=8$. From the above theory, we can draw the conclusion that λ_c increases with m ($\langle k\rangle=2m$) decreasing, the simulation result can confirm this point.

Figure 9 shows the variations of the density of infected individuals with infected rate for the most common friend first

immunization strategy, respectively, with the network scale sizes of $N = 2000$ and $N = 3000$. It shows that an epidemic threshold λ_c decreases with N increasing, which is consistent with the above analysis. In other words, the bigger the network size N, the smaller the epidemic threshold λ_c will be, so the immune effect is worse. For example, in a crowd, the epidemic is vulnerable to spread. This is consistent with our reality.

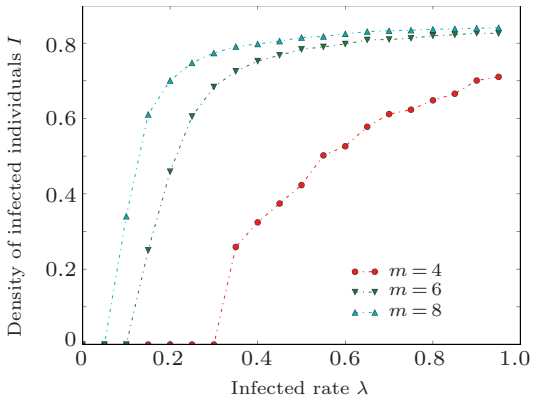


Fig. 8. (color online) Variations of the density of infected individuals with infected rate for the most common friend first immunization on BA scale-free network ($N = 2000$), respectively, with $m = 4$, $m = 6$, and $m = 8$.

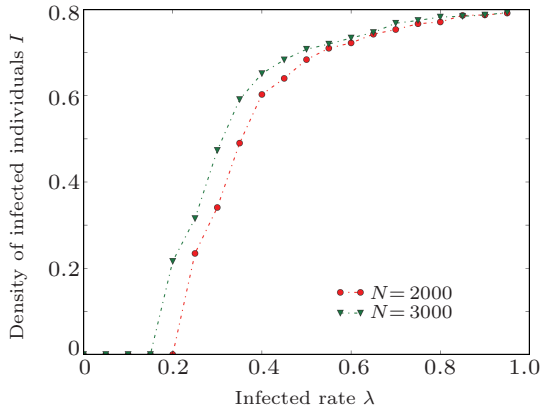


Fig. 9. (color online) Variations of density of infected individuals with infected rate for the most common friend first immunization on BA scale-free network, respectively, with $N = 2000$ and $N = 3000$.

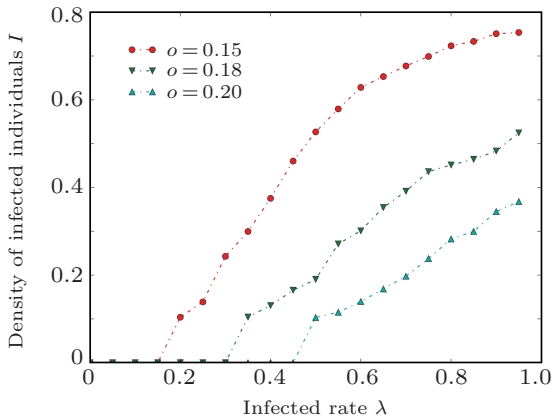


Fig. 10. (color online) Variations of density of infected individuals with infected rate for the most common friend first immunization on BA scale-free network ($N = 2000$, $m = 5$), respectively, with $o = 0.15$, $o = 0.18$, and $o = 0.2$.

Figure 10 shows the variations of the density of infected individuals for the most common friend first immunization, respectively, with $o = 0.15$, $o = 0.18$, and $o = 0.2$. It shows that λ_c increases with o increasing, which is consistent with the above analysis.

6. Conclusions

The adoption of appropriate immunization strategy is significant to prevent and control infectious diseases. In this paper, the most common friend first immunization scheme is proposed. According to the analysis above, the transmission cases of three immunization schemes based on small-world and scale-free network are simulated, respectively, with random immunization, high-risk immunization and the most common friend first immunization. The simulation results indicate that the immune effect of the most common friend first immunization is better than that of random immunization, but inferior to that of the high-risk immunization. The high-risk immunization must know all susceptible individuals whose neighbors have been infected. However, the most common friend first immunization only needs to know some direct neighbors and their neighbors. So, the most common friend first immunization is more feasible in reality.

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