A two-step high-risk immunization based on high-risk immunization

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Abstract In this paper, the two-step high-risk immunization was investigated based on high-risk immunization for the SIRS model in small-world networks and scale-free networks. First, the effects of various immunization schemes are studied and compared. When the number of immune is same, the research result shows that the immune effect of the two-step high-risk immunization strategy is not the best nor the worst. However, the practicability is better compare with others. Furthermore, by changing the proportional of immunization the optimal immune effect can be achieved in the two-step high-risk immunization. Computation results verify that the two-step high-risk immunization is effective, and it is economic and feasible in practice.

Keywords Epidemic · Immunization · Two-step · High-risk

1 Introduction

Epidemic is one of the most important issues related to our real lives. From SARS in 2003, to 2005 H1N1 flu, to today's H7N9, the epidemic problem has attracted a lot of attention. The dynamical behaviors of epidemic diseases have been studied for a long time; it also has been a hot topic [1–14].

For the problem of epidemic, the networks and its dynamics are some basic issue need to be solved firstly. The traditional study of networks includes lattices, regular tree, and ER random graph [15] and then, many real networks were investigated, such as the Internet, the www, the scientific web, and so on. The small-world phenomenon and scale-free property are two major statistical topological characteristics, and the corresponding network models were named as small-world network [16–18] and scale-free network [19–21], respectively. So many epidemic spreading problems are investigated on the two networks.

The epidemic system can be represented as a network where nodes stand for individuals, and edges stand for the relationships between them. Based on this hypothesis, several models have been proposed for epidemic dynamics. The typical Epidemic mode for the disease dynamics includes the susceptible-infected (SI) model [22], susceptible-infected-recovered (SIR) model [23], and susceptible-infected-recovered-susceptible (SIRS) model [24], which represent the development of each individual's disease in a network. In this paper, the SIRS model was selected as our study object. In this model, an infected individual (I) is infective for some period of time and then is recovered (R), and no longer be infected by other infected individuals (I), and then he (she) would lose the immunization and become a susceptible individual again.

The purpose of immune strategy is protecting the part of network nodes would not be infected by cutting off the propagation path of virus. It is one of effective

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tive methods to prevent the virus from spreading on the population. In fact, an efficient immunization strategy can not only be applied to the computer network, but also have an important significance to the diseases spread and control in the human social networks. Currently, various immunization strategies have been proposed, for example: In 2000, Callway proposed a random immunization [25] which the nodes to be vaccinate are selected randomly, while in order to control the spread of the disease we almost vaccinate to all individuals. Obviously, it is not practical. Lately, Pastor-Satorras and Vespignani proposed targeted immunization schemes [26], which immune the large degree nodes firstly. Although there are good effects, but it needs to obtain the whole network topology structure information. In response to this issue, Cohen designs an efficient immunization strategy named “acquaintance immunization” [27]. In this scheme, first randomly select some nodes, then randomly immune one of the nodes from their acquaintances. It is evident that the nodes with large degree have more chance to be vaccinated. In recent years, some other immune strategies have been proposed. For example: J. Gómez-Gardenes put forward on top of real communication networks the immune strategy [28] based on covering algorithm, the effect is basically consistent with the targeted immunization strategy. Madar and Kalisky also proposed the double acquaintance immunization [29] that first randomly selected some nodes, and then randomly immune their two acquaintances. The double acquaintance immunization shows a higher fraction of endemic states than the targeted, but its fraction of infected individuals is still lower than that obtained with the acquaintance immunization strategy. And we proposed a high-risk immunization strategy [30], which the individuals with infected neighbors were looked as high-risk groups, and by immunizing the high-risk groups to controls the spread of the disease. However, the neighbor’s neighbors were not considered in our last work. In this paper, the two-step high-risk immunization strategy was designed based on high-risk immunization. In this scheme, not only the high-risk neighbors were immunized, but also the indirect neighbors of infected individual were immunized too.

The rest of the paper is organized as follows: in Sect. 2, the two-step high-risk immunization in different WS small-world complex networks is investigated, and the standard SIRS model is built. In Sect. 3, we investigate the behaviors of density of infected indi-

viduals on different BA scale-free networks, and the theoretical analysis and computer simulations are given. Finally, conclusions are given in last section.

2 The two-step high-risk immunization on small-world networks

Suppose there are N nodes in the WS small-world network, each node is symmetrical connected to the nearest K nodes with probability p randomly re-connected to the each edge in the original network, and then the average degree of the network is $\langle k \rangle = 2K$. There are three node statuses: S represents susceptible state, I represents infected state, and R represents removed state. S state nodes are infected with a certain rate of β , I state nodes are cured with a cure rate γ , while the R state nodes are loss of immunity with a certain probability σ , then the model’s effective communication rate is $\lambda = \frac{\beta}{\gamma}$. Fig. 1 is the sketch of SIRS.

Like the high-risk immunization [30], define Ω is the probability that an arbitrary given node is a particular node’s neighbor.

The probability of an arbitrary node for access to immunization through vaccination is δ ; at the same time suppose p is the proportion that immunes the direct neighbors of high-risk people, and q is the proportion that immune the indirect neighbors. In homogeneous network, the two-step high-risk immunization model can be described as follows:

$$\begin{aligned} \frac{dS(t)}{dt} &= -\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle S(t)I(t) \\ &\quad + \sigma R(t) \\ \frac{dI(t)}{dt} &= \lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle S(t)I(t) \\ &\quad - I(t) \\ \frac{dR(t)}{dt} &= I(t) - \sigma R(t). \end{aligned} \quad (1)$$

Here, $S(t)$, $I(t)$, and $R(t)$, respectively, represent the average density of susceptible population, infected population, and removed population. In Eq. (1), the first

Fig. 1 Sketch of SIRS

term on the right side of the first equation considers the average density of an infected node infection produces new infections. The secondary term on the right side of the first equation considers the recovered nodes loss of immunity probability.

Because $S(t)$, $I(t)$, and $R(t)$ obey the normalization condition $S(t) + I(t) + R(t) = 1$, and the static conditions of no infectious diseases spread on networks are

$$\frac{dS(t)}{dt} = 0, \frac{dI(t)}{dt} = 0, \frac{dR(t)}{dt} = 0, \text{ And } I(t) = 0. \quad (2)$$

From the Eq. (1) and Eq. (2), we obtain

$$\begin{aligned} -\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle S(t)I(t) + \sigma R(t) &= 0 \\ \lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle S(t)I(t) - I(t) &= 0 \\ I(t) - \sigma R(t) &= 0. \end{aligned} \quad (3)$$

From the secondary equation of Eq. (3),

$$S(t) = \frac{1}{\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle}. \quad (4)$$

Substitute $S(t) + I(t) + R(t) = 1$ into Eq. (3), we obtain

$$-I(t) + \sigma[1 - I(t) - S(t)] = 0. \quad (5)$$

Substitute (4) into Eq. (5), we obtain

$$-I(t) + \sigma \left(1 - I(t) - \frac{1}{\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle} \right) = 0.$$

Because $I(t) = 0$, the above equation can be simplified as

$$\lambda = \frac{1}{(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle}.$$

So, the non-zero epidemic threshold is $\lambda_c = \frac{1}{(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \langle k \rangle}$. When $\sigma = 1$, the SIRS model is equivalent to the SIS model, and $\lambda_c = \frac{1}{(1 - p\delta\Omega - q\delta\Omega^2) \langle k \rangle}$.

If the vaccinating rate $\delta = 0$, obtain $\lambda_c = \frac{1}{\langle k \rangle}$.

From the results obtained by the above mathematical model, we can see that the non-zero epidemic threshold is associated with the average degree $\langle k \rangle$ of the network, the immune probability δ , the probability Ω of a node with specific nodes which are neighbors to each other, and the numbers of the direct neighbors and indirect neighbors. When the probability of $\langle k \rangle$, δ , and Ω is fixed, we can obtain the different non-zero epidemic threshold by change the immune proportion of neighbors and indirect neighbors. Then we can achieve the optimal immune effect.

Fig. 2 The comparison of random immunization, high-risk immunization, and the two-step high-risk immunization in WS small-world network (when $N = 2,000$, $\langle k \rangle = 4$, $\sigma = 0.2$. Infection density varies with propagation probability)

Based on the analysis, the corresponding experiments are performed to further verify it. Our simulations are implemented at fixed $N = 2,000$, $\langle k \rangle = 4$, and the rewiring rate is 0.1. Initially, 10 % of the susceptible nodes in the network are infected. Suppose that the chance of being infected by its neighbor is equal for all nodes. In other words, in each iteration, the probability of being infected by its neighbor is the same. Simulations were computed averaging over 30 different starting configurations, performed on 20 different realizations of the network.

Figure 2 is the comparison of random immunization, high-risk immunization, and the two-step high-risk immunization. Under the condition that the vaccinating number is same, the effect of the high-risk immunization is best, while the effect of the two-step high-risk immunization is not only slightly inferior to high-risk immunization but also better than random immunization. This is consistent with the actual situation, the high-risk immunization directly cut off the route of transmission of the virus from the source, greatly reducing the spread of the virus, so the effect is best; while in the similar condition, the two-step high-risk immunization not only immunizes their directly neighbors but also immunizes their indirectly neighbors, so compared with high-risk immunization cut off more decentralized transmission. So the immune effect is slightly inferior. However, who is the directed neighbor is difficult to identify in fact. So, the two-step high-risk immunization is more feasible.

Fig. 3 **a** When p from 0.1 to 0.9 and q from 0.9 to 0.1, infection densities vary with propagation probability (p represents the proportion of immune direct neighbor node, q represents the proportion of immune indirect neighbor node). **b** The changing of total number of immune nodes with p and q

Figure 3a is the comparison of the immune effect when we change the immune proportion of direct neighbor and indirect number in the two-step high-risk immunization. From Fig. 3a, we can see, when the proportion of immune direct neighbor nodes from 0.1 to 0.9 increased, and the proportion of immune indirect neighbor nodes from 0.9 to 0.1 decreased, the epidemic threshold λ increases at begin and then decreases. Figure 3b is changing of total number of immune nodes with p and q . From Fig. 3b, we can see that the total numbers generally equalled when the immune proportion is changed.

Here, p represents the proportion of immune direct neighbor node; q represents the proportion of immune indirect neighbor node.

Figure 3 indicates the immune effect when the number of vaccinated nodes is same or similar. For the stronger infectious virus, the more intimate neighbors ($p = 0.9, q = 0.1$) or more indirect neighbors ($p = 0.1, q = 0.9$) can be vaccinated to achieve the optimal immune effect; for the not strong infectious virus, the spread of virus can be controlled effectively when evenly choose the number of direct and indirect neighbors individuals ($p = 0.5, q = 0.5$).

3 The two-step high-risk immunization on scale-free networks

The characteristic of WS small-world model is the connectivity distribution can be approximately represented by the Poisson distribution. The distribution has a peak in the average value $\langle k \rangle$, then rapid degradation exponentially, which means that when $k \gg \langle k \rangle$, it almost not exists a node which degree is K . So the network is also known as the Homogeneous network. In recent years, another major discovery is in many complex networks, including Internet, WWW, and the new supersedes the old network, connection degree distribution function has the power-law form. Because in these network the nodes' degree without significantly characteristic length, so called scale-free networks [19]. Barabási and Albert proposed the BA scale-free model based on the characteristic of growth and preferential attachment. We study the SIRS two-step high-risk immunization infectious disease model based on the scale-free network model is described as follows: constructs a BA scale-free network, it satisfies the growth and preferential connection characteristic, after iterating this scheme a sufficient number of times, we obtain a networks composed of N nodes with degree distribution $P(k) = k^{-3}$ and degree $\langle k \rangle = 2m$. Let $S_k(t)$, $I_k(t)$, and $R_k(t)$ be the densities of susceptible, infected, and recovered vertex of degree k . Then we have the following dynamics model:

$$\begin{aligned} \frac{dS_k(t)}{dt} &= -\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)kS_k(t)\Theta(t) \\ &\quad + \sigma R_k(t) \\ \frac{dI_k(t)}{dt} &= \lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)kS_k(t)\Theta(t) \\ &\quad - I_k(t) \\ \frac{dR_k(t)}{dt} &= I_k(t) - \sigma R_k(t). \end{aligned} \quad (6)$$

In Eq. (6), the first term on the rhs of the first equation considers the average density of newly infected nodes generated by each infected node. And the probability is $\Theta(t)$ that any given link points to an infected node. The secondary term on the rhs of the first equation represents the nodes losing immunity with rate σ . The first term on the rhs of the last equation of Eq. (6) denotes the infected nodes recovering with unit rate.

As we know, in scale-free networks a randomly chosen link is more likely to be connected to an infected node with high connectivity, yielding [31]:

$$\Theta(t) = \frac{\sum_k k P(k) I_k(t)}{\sum_s s P(s)} = \frac{1}{\langle k \rangle} \sum_k k P(k) I_k(t). \quad (7)$$

Also, $S_k(t)$, $I_k(t)$, and $R_k(t)$ obey the normalization condition for each k

$$S_k(t) + I_k(t) + R_k(t) = 1. \quad (8)$$

While non-epidemic stationary conditions can be given as follows: $\frac{dS_k(t)}{dt} = 0$, $\frac{dI_k(t)}{dt} = 0$, $\frac{dR_k(t)}{dt} = 0$ And $I_k(t) = 0$ then, from the first equation of Eq. (6), we obtain

$$-\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)kS_k(t)\Theta(t) + \sigma R_k(t) = 0. \quad (9)$$

Combine above equation with Eq. (8), we obtain

$$S_k(t) = \frac{\sigma I_k(t) - \sigma}{-\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)k\Theta(t) - \sigma}. \quad (10)$$

From the second equation of Eq. (6)

$$S_k(t) = \frac{I_k(t)}{\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)k\Theta(t)}. \quad (11)$$

Substitute (11) into (10) in the following. We obtain the following equation:

$$I_k(t) = \frac{\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)k\Theta(t)\sigma}{(\sigma + 1)\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)k\Theta(t) + \sigma}. \quad (12)$$

From the formula, the solution $I_k(t) = 0$ and $\Theta(t) = 0$ always satisfies the Eq. (12). In order to get a non-

zero static solution, to both sides of the equation (12) represents a function $F(\Theta)$, The range of this function is $0 < \Theta \leq 1$. Therefore, a nontrivial solution that exists provided is [30]

$$\frac{dF(\Theta)}{d\Theta} \Big|_{\Theta=0} \geq 1. \quad (13)$$

Must be fulfilled. i.e.,

$$\begin{aligned} \frac{d}{d\Theta} \left\{ \frac{1}{\langle k \rangle} \sum_k k P(k) \right. \\ \times \left. \left\{ \frac{\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)k\Theta(t)\sigma}{(\sigma + 1)\lambda(1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma)k\Theta(t) + \sigma} \right\} \right\} \Big|_{\Theta=0} \\ = \frac{\lambda}{\langle k \rangle} \sum_k k^2 P(k) (1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \geq 1. \end{aligned} \quad (14)$$

Ω is the probability of any given node which is a neighbor of some specific node. Therefore,

$$\Omega = \frac{k P(k)}{N \langle k \rangle}. \quad (15)$$

As for BA scale-free network,

$$P(k) = \frac{2m^2}{k^3}. \quad (16)$$

Substitute Eqs. (15) and (16) into (14), we obtain

$$\begin{aligned} \frac{\lambda}{\langle k \rangle} \sum_k k^2 P(k) (1 - p\delta\Omega/\sigma - q\delta\Omega^2/\sigma) \\ = \frac{\lambda}{\langle k \rangle} \left(\sum_k k^2 P(k) - \sum_k k^2 P(k) p\delta\Omega/\sigma \right. \\ \left. - \sum_k k^2 P(k) q\delta\Omega^2/\sigma \right) \\ = \frac{\lambda}{\langle k \rangle} \left[\langle k^2 \rangle - \sum_k k^2 P(k) \frac{\delta p}{\sigma} \frac{k P(k)}{N \langle k \rangle} \right. \\ \left. - \sum_k k^2 P(k) \frac{\delta q}{\sigma} \left(\frac{k P(k)}{N \langle k \rangle} \right)^2 \right] \\ = \frac{\lambda}{\langle k \rangle} \left[\langle k^2 \rangle - \sum_k k^2 P(k) \frac{\delta p}{\sigma} \frac{k 2m^2}{N \langle k \rangle k^3} \right. \\ \left. - \sum_k k^2 P(k) \frac{\delta q}{\sigma} \left(\frac{k 2m^2}{N \langle k \rangle k^3} \right)^2 \right] \\ = \frac{\lambda}{\langle k \rangle} \left[\langle k^2 \rangle - \frac{\delta p}{\sigma} \frac{2m^2}{N \langle k \rangle} - \frac{\delta q}{\sigma} \frac{4m^4}{N^2 \langle k \rangle^2 k^2} \right] \geq 1. \end{aligned} \quad (17)$$

The value of λ yielding the inequality (17), i.e.,

$$\frac{\lambda_c}{\langle k \rangle} \left[\langle k^2 \rangle - \frac{\delta p}{\sigma} \frac{2m^2}{N \langle k \rangle} - \frac{\delta q}{\sigma} \frac{4m^4}{N^2 \langle k \rangle^2 k^2} \right] = 1.$$

From above equation, the critical epidemic threshold λ_c , that is given by

$$\lambda_c = \frac{\langle k \rangle}{\left[\langle k^2 \rangle - \frac{\delta p}{\sigma} \frac{2m^2}{N \langle k \rangle} - \frac{\delta q}{\sigma} \frac{4m^4}{N^2 \langle k \rangle^2 k^2} \right]}. \quad (18)$$

When $\sigma = 1$ and $\delta = 0$, $\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$

Here,

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

Substitute the above two equations into Eq. (18)

$$\lambda_c = \frac{2m^2 \sum_k \frac{1}{k^2}}{\left[2m^2 \sum_k \frac{1}{k} - \frac{\delta p}{\sigma} \frac{1}{N \sum_k \frac{1}{k^2}} - \frac{\delta q}{\sigma} \frac{1}{N^2 (\sum_k \frac{1}{k^2})^2 k^2} \right]}.$$

Further

$$\begin{aligned} \frac{1}{\lambda_c} &= \frac{2m^2 \sum_k \frac{1}{k} - \frac{\delta p}{\sigma} \frac{1}{N \sum_k \frac{1}{k^2}} - \frac{\delta q}{\sigma} \frac{1}{N^2 (\sum_k \frac{1}{k^2})^2}}{2m^2 \sum_k \frac{1}{k^2}} \\ &= \frac{\sum_k \frac{1}{k}}{\sum_k \frac{1}{k^2}} - \frac{\delta p}{2Nm^2 (\sum_k \frac{1}{k^2})^2 \sigma} \\ &\quad - \frac{\delta q}{2N^2 m^2 (\sum_k \frac{1}{k})^2 \sum_k \frac{1}{k^2} \sigma}. \end{aligned} \quad (19)$$

In the continuous k approximation, calculate the approximation of $\sum_k \frac{1}{k}$ and $\sum_k \frac{1}{k^2}$

$$\sum_k \frac{1}{k} \rightarrow \int_m^M \frac{1}{k} dk = \ln \frac{M}{m} \quad (20)$$

$$\sum_k \frac{1}{k^2} \rightarrow \int_m^M \frac{1}{k^2} dk = \frac{1}{m} - \frac{1}{M} = \frac{M-m}{mM} \quad (21)$$

Here, M is the maximum degree, substitute Eqs. (20) and (21) into Eq. (19)

$$\begin{aligned} \frac{1}{\lambda_c} &\approx \frac{\ln \frac{M}{m}}{\frac{M-m}{mM}} - \frac{\delta p}{2Nm^2 \sigma (\frac{M-m}{mM})^2} \\ &\quad - \frac{\delta q}{2N^2 m^2 (\ln \frac{M}{m})^2 \frac{M-m}{mM} \sigma} \\ &= \frac{mM \ln \frac{M}{m}}{M-m} \\ &\quad - \frac{M^2 \delta p}{2N \sigma (M-m)^2} - \frac{M \delta q}{2N^2 m \sigma (M-m) (\ln \frac{M}{m})^2}. \end{aligned}$$

When M is big enough (when m is constant, M increases with N), $M-m \approx M$ thus,

$$\frac{1}{\lambda_c} \approx m \ln \frac{M}{m} - \frac{\delta}{2N \sigma} \left(p + \frac{q}{2Nm (\ln \frac{M}{m})^2} \right). \quad (22)$$

From Eq. (22), we can see

(1) λ_c increases with σ decreasing

In our real life, the longer the time immune in our body, the harder the virus transmit in the crowd, namely there is a higher spreading threshold; while the shorter the time with the immune in our body, the easier the virus transmit in the crowd, which means the spreading threshold is lower.

(2) λ_c increases with δ increasing.

δ is disease vaccination rate. If the vaccination rate is very high namely to have more individual acquired immunity, the disease is not easy to spread and the threshold higher.

(3) λ_c decreases with N increasing.

The more population, the more easily outbreak infectious diseases, this is consistent with our real life.

(4) λ_c increases with p increasing or increases with q increasing, and exists a optimal proportion p and q of control the spread of the disease, makes λ_c maximum.

When immunizing the more direct neighbors, the disease is not easy to spread. When immunizing the more indirect neighbors, the disease is not easy to spread too. While when the total immunity number is same or similar, an optimal immune proportion can be achieved by changing the immune proportion of direct neighbors and indirect neighbors. In this case,

Fig. 4 The comparison of random immunization, high-risk immunization, and the two-steps high-risk immunization in BA scale-free network (when $N = 2,000$, $\langle k \rangle = 4$, $\sigma = 0.2$ Infection density varies with propagation probability)

the spread of virus can be most effectively controlled, and the propagation threshold reached the maximum.

Based on the above analysis of mathematical theory, the corresponding experiments also were implemented to verification. The network was selected as scale-free network with $N = 2,000$, average degree $\langle k \rangle = 4$, and the initial node $m = 5$. The $0.1 \cdot N$ infected nodes were selected randomly from the network. Suppose the chance of be infected by infected neighbor is equal to all nodes, and the probability of infection is the same. Simulations were computed averaging over 30 different starting configurations, performed on 20 different realizations of the network.

Figure 4 is the comparison of random immunization, high-risk immunization, and the two-step high-risk immunization effect. Under the condition that the vaccinating number is same, the results are similar to the small-world network; the effect of the high-risk immunization is best, while the effect of the two-step high-risk immunization is not only slightly inferior to high-risk immunization but also better than random immunization.

Figure 5a is the comparison of the immune effect when we change the immune proportion of direct neighbors and indirect numbers in the two-step high-risk immunization. In Fig. 5a, when the proportion of immune direct neighbor nodes from 0.1 to 0.9 increased, and the proportion of immune indirect neighbor nodes from 0.9 to 0.1 decreased, the epidemic threshold λ decreases at begin and then increases.

Fig. 5 **a** When p from 0.1 to 0.9 and q from 0.9 to 0.1, infection densities vary with propagation probability ($N = 2,000$, $\langle k \rangle = 4$, $\sigma = 0.2$, p represents the proportion of immune direct neighbor node; q represents the proportion of immune indirect neighbor node.) **b** The changing of total number of immune nodes with p and q

Figure 5b is changing of total number of immune nodes with p and q . From Fig. 5b, we can see that the total numbers change from 400 to 1,000 when the immune proportion is changed as above. This phenomenon is mainly determined by the characteristics of scale-free network itself. In the construction of scale-free networks there are two important features which are the growth and preferential attachment, so some degree of nodes is relatively large. This leads to a rapidly increase in the number of direct neighbors and indirect neighbors we looked for, which affect the immune quantity. So if a smaller proportion of direct neighbors and a larger proportion of indirect neighbors ($p = 0.1$, $q = 0.9$) are vaccinated, it means that almost all the points

are vaccinated in the network, at this time the propagation threshold is large. On the contrary when a larger proportion of direct neighbors and a smaller proportion of indirect neighbors ($p = 0.9, q = 0.1$) are vaccinated, the number of immune nodes is relatively small, but the virus transmission route be cut off from the root, and the propagation threshold is large too; once the virus break through the first layer of immune defense, the spread of the virus will unbridle. This is also the reason why the infection density sudden increases when $p = 0.9, q = 0.1$ in Fig. 5a.

Here, p represents the proportion of immune direct neighbor node; q represents the proportion of immune indirect neighbor node.

Figure 5b is changing of total number of immune nodes with p and q . In Fig. 5b the total number of immune nodes is so large difference, while in practical application, the immune effect is expected to compare in similarity immune nodes. So immune number controlled as is shown in Fig. 6. Figure 6a shows the variation of infection density with propagation probability when the proportion of immune direct neighbor nodes from 0.1 to 0.7 increased and the proportion of immune indirect neighbor nodes from 0.55 to 0.25 decreased. From Fig. 6b we can see that the total number changes from 500 to 600 generally not big fluctuations when the immune proportion is changed as above.

Figure 6 indicates the immune effect when the number of vaccinated nodes is same or similar. In Fig. 6, for the stronger infectious virus, more indirect neighbors can be immunized to active the optimal immune effect ($p = 0.1, q = 0.55$); for not strong infectious virus, more direct neighbors can be immunized to control the spread of disease effectively ($p = 0.7, q = 0.25$).

4 Conclusion

In this paper, the two-step high-risk immunization strategy was proposed by experimenting in the WS small-world network model and BA scale-free network model; further the effects of various immunization schemes are compared; at the same time the application effect of the two-step high-risk immunization is discovered. According to the different strengths of infectious disease, the different immunization proportional can be taken to achieve the optimal immune effect. The results verified that the two-step high-risk

Fig. 6 a When p from 0.1 to 0.7 and q from 0.55 to 0.25, infection densities vary with propagation probability ($N = 2,000, \langle k \rangle = 4, \sigma = 0.2$, p represents the proportion of immune direct neighbor node; q represents the proportion of immune indirect neighbor node.) b The total number of immune nodes varies with p and q

immunization not only has its own advantages, but also is effective, economic, and feasible.

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References

1. Chu, X., Zhang, Z., Guan, J., et al.: Epidemic spreading with nonlinear infectivity in weighted scale-free networks. *Phys. A* **390**(3), 471–481 (2011)

2. Liu, J., Zhang, T.: Epidemic spreading of an SEIRS model in scale-free networks. *Commun. Nonlinear Sci. Numer. Simul.* **16**(8), 3375–3384 (2011)
3. Wang, L., Li, X., Zhang, Y.-Q., et al.: Evolution of scaling emergence in large-scale spatial epidemic spreading. *PloS one* **6**(7), e21197 (2011)
4. Saumell-Mendiola, A., Serrano, M.Á., Boguñá, M.: Epidemic spreading on interconnected networks. *Phys. Rev. E* **86**(2), 026106 (2012)
5. Zhu, G., Fu, X., Chen, G.: Spreading dynamics and global stability of a generalized epidemic model on complex heterogeneous networks. *Appl. Math. Modell.* **36**(12), 5808–5817 (2012)
6. Granell, C., Gómez, S., Arenas, A.: Dynamical interplay between awareness and epidemic spreading in multiplex networks. *Phys. Rev. Lett.* **111**(12), 128701 (2013)
7. Li, C.-H., Tsai, C.-C., Yang, S.-Y.: Analysis of epidemic spreading of an SIRS model in complex heterogeneous networks. *Commun. Nonlinear Sci. Numer. Simul.* **19**(4), 1042–1054 (2014)
8. Sun, Y., Liu, C., Zhang, C.-X.: Epidemic spreading on weighted complex networks. *Phys. Lett. A* **378**(7), 635–640 (2014)
9. Xia, C., Wang, L., Sun, S., et al.: An SIR model with infection delay and propagation vector in complex networks. *Nonlinear Dyn.* **69**(3), 927–934 (2012)
10. Zhou, X., Cui, J.: Analysis of stability and bifurcation for an SEIV epidemic model with vaccination and nonlinear incidence rate. *Nonlinear Dyn.* **63**(4), 639–653 (2011)
11. Nian, F., Liu, T.: High-risk immunization strategies for multiethnic regions. *Abstract Appl. Anal.* **2014**, 6 (2014)
12. Wang, T.: Pattern dynamics of an epidemic model with nonlinear incidence rate. *Nonlinear Dyn.* 1–10 (2014)
13. Sun, G.-Q.: Pattern formation of an epidemic model with diffusion. *Nonlinear Dyn.* **69**(3), 1097–1104 (2012)
14. Chen, Q., Teng, Z., Wang, L., et al.: The existence of codimension-two bifurcation in a discrete SIS epidemic model with standard incidence. *Nonlinear Dyn.* **71**(1–2), 55–73 (2013)
15. Erdős, P., Rényi, A.: On the evolution of random graphs. *Magyar Tud. Akad. Mat. Kutató Int. Közl* **5**, 17–61 (1960)
16. Newman, M.E.: Models of the small world. *J. Stat. Phys.* **101**(3–4), 819–841 (2000)
17. Watts, D.J., Strogatz, S.H.: Collective dynamics of ‘small-world’ networks. *Nature* **393**(6684), 440–442 (1998)
18. Newman, M.E., Watts, D.J.: Renormalization group analysis of the small-world network model. *Phys. Lett. A* **263**(4), 341–346 (1999)
19. Barabási, A.-L., Albert, R.: Emergence of scaling in random networks. *Science* **286**(5439), 509–512 (1999)
20. Barabási, A.-L.: Scale-free networks: a decade and beyond. *Science* **325**(5939), 412–413 (2009)
21. Wang, N.-N., Chen, G.-L.: Presented at the Proceedings of the 2012 International Conference on Communication, Electronics and Automation Engineering, 2013 (unpublished)
22. Chen, L., Sun, J.: Global stability of an SI epidemic model with feedback controls. *Appl. Math. Lett.* **28**, 53–55 (2014)
23. Harko, T., Lobo, F. S., Mak, M.: Exact analytical solutions of the Susceptible-Infected-Recovered (SIR) epidemic model and of the SIR model with equal death and birth rates. *arXiv preprint arXiv:1403.2160* (2014)
24. Xu, R., Ma, Z.: Global stability of a delayed SEIRS epidemic model with saturation incidence rate. *Nonlinear Dyn.* **61**(1–2), 229–239 (2010)
25. Callaway, D.S., Newman, M.E., Strogatz, S.H., et al.: Network robustness and fragility: percolation on random graphs. *Phys. Rev. Lett.* **85**(25), 5468 (2000)
26. Pastor-Satorras, R., Vespignani, A.: Immunization of complex networks. *Phys. Rev. E* **65**(3), 036104 (2002)
27. Cohen, R., Havlin, S., Ben-Avraham, D.: Efficient immunization strategies for computer networks and populations. *Phys. Rev. Lett.* **91**(24), 247901 (2003)
28. Gómez-Gardenes, J., Echenique, P., Moreno, Y.: Immunization of real complex communication networks. *Eur. Phys. J. B-Condens. Matter Complex Syst.* **49**(2), 259–264 (2006)
29. Madar, N., Kalisky, T., Cohen, R., et al.: Immunization and epidemic dynamics in complex networks. *Eur. Phys. J. B-Condens. Matter Complex Syst.* **38**(2), 269–276 (2004)
30. Nian, F., Wang, X.: Efficient immunization strategies on complex networks. *J. Theor. Biol.* **264**(1), 77–83 (2010)
31. Pastor-Satorras, R., Vespignani, A.: Epidemic spreading in scale-free networks. *Phys. Rev. Lett.* **86**(14), 3200 (2001)