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Epidemic spreading on networks with vaccination*

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In this paper, a new susceptible-infected-susceptible (SIS) model on complex networks with imperfect vaccination is proposed. Two types of epidemic spreading patterns (the recovered individuals have or have not immunity) on scale-free networks are discussed. Both theoretical and numerical analyses are presented. The epidemic thresholds related to the vaccination rate, the vaccination-invalid rate and the vaccination success rate on scale-free networks are demonstrated, showing different results from the reported observations. This reveals that whether or not the epidemic can spread over a network under vaccination control is determined not only by the network structure but also by the medicine's effective duration. Moreover, for a given infective rate, the proportion of individuals to vaccinate can be calculated theoretically for the case that the recovered nodes have immunity. Finally, simulated results are presented to show how to control the disease prevalence.

Keywords: complex network, disease spreading, SIS model, epidemic modeling, vaccination, epidemic threshold

PACC: 0545

1. Introduction

The important issues of structures and dynamics of complex networks have been extensively investigated in recent years.[1−11] It was found that many real-world networks, like human sexual relationships,[1] e-mail networks,[2] the Internet,[3] the World Wide Web,[4] and food webs,[5] all have similar properties. They are referred to as scale-free networks,[6] and have been used to model networks of social association or contact.[12−17] On the other hand, disease spread on networks is a commonly concerned topic for research. In order to understand the underlying mechanisms of disease spreading, the susceptible-infected-susceptible (SIS),[18−21] susceptible-infected-recovered (SIR),[22,23] susceptible-infected (SI)[24−26] and susceptible-hidden-infected-susceptible (SHIS)[27] models over complex networks have all been investigated to date. Consequently, many effective immunization strategies (such as uniform immunization, proportional immunization, targeted immunization and immunization based on Euclidean distance) have been proposed in order to prevent and control the spreading diseases.[20,28]

In epidemic modeling, individuals are typically represented by nodes and possible contacts between individuals are linked by edges. Infectious diseases in nature are transmitted through contact with infected individuals or sources, such as rubella, herpes simplex, hepatitis B, AIDS, influenza, etc. In this work, an epidemic spreading model in the human population, treated as a scale-free network, is investigated, taking into account two different states of recovered nodes: immunity to the disease or not. A staged-progression SIS model is formulated and used to study the potential impact of imperfect vaccination. The vaccine is assumed to have the characteristic of protecting the nodes against infection.[29]

The following are the new findings in this study, which are different from the other existing reports: (i) the threshold of the effective spreading rate λ_c in this paper is correlated with the vaccination rate σ_1, the vaccination-invalid rate σ_2, and the vaccination success rate ε; (ii) in order to control the disease in the case that recovered nodes have immunity, the new model provides a formula for calculating the scales of σ_1, σ_2 and ε needed for vaccination when the other parameters are given; (iii) numerical simulation re-

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sults show that the epidemic spreading in scale-free networks is oscillating and periodic, showing how to control the different types of epidemic diseases.

The rest of the paper is organized as follows: Subsection 2.1 derives the mean-field epidemic spreading model as a scale-free Barabási–Albert (BA) network, in which recovered individuals have immunity to the disease, and analyzes its steady-state solutions. In this section, moreover, numerical simulations are given to show the effects of parameters σ_1, σ_2 and ε on the epidemic spreading. In Subsection 2.2, the case that recovered individuals do not have immunity to the disease is considered, and its steady-state solutions are analyzed. In this section, numerical simulations are also given to show the effects of parameters σ_1, σ_2 and ε on the epidemic spreading. Finally, Section 3 concludes the paper.

2. The SIS-like models

In most existing SIS-like models formulated on complex networks, one usually considers the case where the disease transmits without vaccination. But, more often than not, when an epidemic emerges, the authorities will take actions and measures, such as vaccination, to control it. In order to simplify the problem, the reported SIS models are employed, and the death of individuals caused by the disease is ignored, which is reasonable for some diseases such as influenza. Then the case where the network has vaccinated individuals and normal individuals is considered. More precisely, the new epidemic spreading model is defined as follows: there is one type of individuals, represented by nodes in a network, who are always in one of the three possible discrete states: susceptible without vaccination, susceptible with vaccination or infected. At each time step, a susceptible node without vaccination is infected with probability λ if it is connected to an infected node, and every infected node is cured and becomes susceptible again with probability δ. Parameter ε, $0 < \varepsilon < 1$ accounts for the efficacy of the vaccine-induced protection against infection (for a vaccine that offers 100% protection, $\varepsilon = 1$; generally in reality, $\varepsilon < 1$), and parameters σ_1 and σ_2 represent the susceptible node's vaccination rate and the vaccine-induced susceptible node's protection invalid rate, respectively. It should be noticed that the parameter ε should not be absorbed into the vaccination rate σ_1, because from a biological point of view, after a node is vaccinated one will not vaccine it again before knowing the vaccination result. Suppose that the unsuccessfully vaccinated nodes have the same infected rate as non-vaccinated nodes on the network. Then, all nodes run randomly through the SIS cycles. In order to consider the two scenarios where recovered individuals have immunity and do not have immunity, respectively, the susceptible nodes are separated into two sets: set A is composed of nodes who were not vaccinated or in the vaccine-invalid state, and set B is composed of nodes who were being vaccinated successfully.

2.1. SIS model for the case that recovered nodes have immunity

Suppose that after being recovered through medical treatment the nodes will not be infected again for a period of time even if they keep contact with infected nodes, such as with varicella. In this case, the recovered nodes are all susceptible nodes in set B. Here, assume that the recovered nodes have the same immunity as the vaccinated nodes to the same disease (see Fig.1).

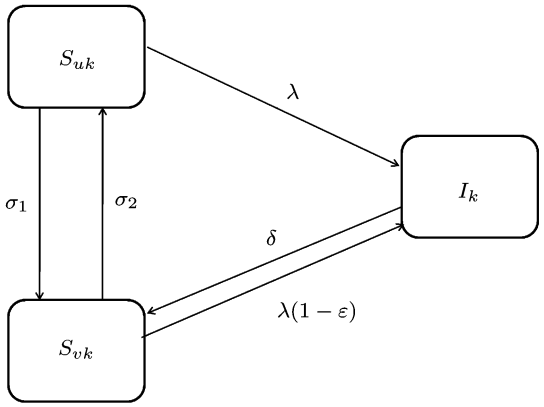


Fig.1. Schematic representation of the flow among different classes of susceptible nodes without vaccination (S_{uk}), susceptible nodes with vaccination (S_{vk}) and infected nodes ($I_k(t)$), which records the dynamic interaction between susceptible nodes and infected nodes. In this diagram, susceptible nodes without vaccination are infected by their neighboring infected nodes at a per capita rate λ and get vaccine-induced protection at a per capita rate σ_1, the susceptible nodes with vaccination lose their vaccinated protection at a per capita rate σ_2, the infected nodes are recovered at a per capita rate δ, the vaccinated nodes get infected at a per capita rate $\lambda(1-\varepsilon)$ because of unsuccessful vaccination. Here, it is supposed that the recovered nodes have the same immunity to the disease as the vaccinated nodes.

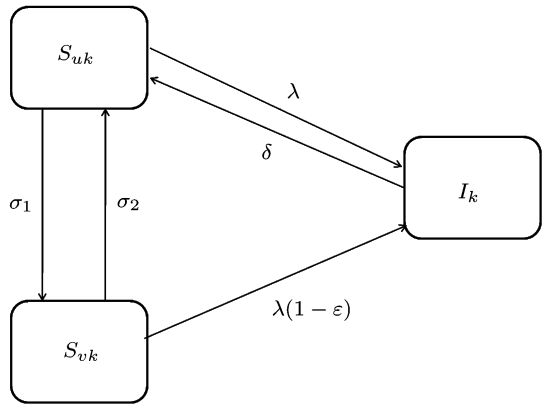


Fig.2. Schematic representation of the flow among different classes of susceptible nodes without vaccination (S_{uk}), susceptible nodes with vaccination (S_{vk}) and infected nodes (I_k), which records the dynamic interaction between susceptible nodes and infected nodes. In this diagram, susceptible nodes without vaccination are infected by their neighboring infected nodes at a per capita rate λ and get vaccine-induced protection at a per capita rate σ_1, the susceptible nodes with vaccination lose their vaccinated protection at a per capita rate σ_2, the infected nodes are recovered at a per capita rate δ, the vaccinated nodes get infected at a per capita rate $\lambda(1-\varepsilon)$ because of unsuccessful vaccination. Here, it is supposed that the recovered nodes do not have immunity to the disease.

In a scale-free network, the contact among nodes is related to the connectivity of the network. The number of infected nodes of degree (number of connections) k, denoted by $I_k(t)$, the number of susceptible nodes in set A with degree k, by $S_{uk}(t)$, and the number of susceptible nodes in set B with degree k, by $S_{vk}(t)$, all evolve according to the following transition patterns:

$$\begin{aligned}
\dot{S}_{uk(t)} &= -\lambda k\Theta_I S_{uk}(t) - \sigma_1 S_{uk}(t) + \sigma_2 S_{vk}(t),\\
\dot{S}_{vk(t)} &= \sigma_1 S_{uk}(t) - \sigma_2 S_{vk}(t)\\
&\quad - \lambda(1-\varepsilon)k\Theta_I S_{vk}(t) + \delta I_k(t),\\
\dot{I}_k(t) &= \lambda k\Theta_I S_{uk}(t) + \lambda(1-\varepsilon)k\Theta_I S_{vk}(t) - \delta I_k(t).
\end{aligned}$$

The number of nodes with degree k is $N_k(t) = S_{uk}(t) + S_{vk}(t) + I_k(t)$, which satisfies

$$\dot{N}_k(t) = 0,$$

where $0 < \lambda,\ \delta,\ \sigma_1,\ \sigma_2,\ \varepsilon < 1$. And the network size is $N(t) = \sum_k P(k)N_k(t)$, the number of susceptible nodes is $S(t) = \sum_k P(k)[S_{uk} + S_{vk}(t)]$, the number of infected nodes is $I(t) = \sum_k P(k)I_k(t)$, where $P(k)$ is the degree distribution. The probability $0 \le \Theta_I \le 1$ describes a link pointing to an infected individual, which satisfies the relation

$$\Theta_I = \sum_k \frac{kP(k)\dfrac{I_k(t)}{N_k(t)}}{\sum_s sP(s)}.$$

Let $x_k(t) = \dfrac{S_{uk}(t)}{N_k(t)}$, $y_k(t) = \dfrac{S_{vk}(t)}{N_k(t)}$ and $z_k(t) = \dfrac{I_k(t)}{N_k(t)}$. Then, since $x_k(t) + y_k(t) + z_k(t) = 1$, one has

$$\begin{aligned}
\dot{x_k}(t) &= -\lambda k\Theta_I x_k(t) - \sigma_1 x_k(t)\\
&\quad + \sigma_2 - \sigma_2 x_k(t) - \sigma_2 z_k(t),\\
\dot{z_k}(t) &= \lambda(1-\varepsilon)k\bar{\Theta}_I + \varepsilon\lambda k\Theta_I x_k(t)\\
&\quad - \lambda(1-\varepsilon)k\Theta_I z_k(t) - \delta z_k(t),
\end{aligned} \tag{1}$$

where $x(t) = \sum_k P(k)x_k(t)$ is the density of susceptible nodes in set A, $y(t) = \sum_k P(k)y_k(t)$ is the density of susceptible nodes in set B, $z(t) = \sum_k P(k)z_k(t)$ is the density of total infected nodes, and

$$\Theta_I = \sum_k \frac{kP(k)z_k(t)}{\sum_s sP(s)}. \tag{2}$$

In the steady state, $x_k(t)$ and $z_k(t)$ are functions of λ, δ, σ_1 and σ_2, thus the probability Θ_I becomes an implicit function of the parameters λ, δ, σ_1 and σ_2. Letting the stationarity condition be $\dot{x}_k(t) = 0$ and $\dot{z}_k(t) = 0$, one has

$$\begin{aligned}
&- \lambda k\Theta_I x_k - \sigma_1 x_k + \sigma_2 - \sigma_2 x_k - \sigma_2 z_k = 0,\\
&\lambda(1-\varepsilon)k\Theta_I + \varepsilon\lambda k\Theta_I x_k - \lambda(1-\varepsilon)k\Theta_I z_k - \delta z_k = 0,
\end{aligned} \tag{3}$$

giving solutions

$$\begin{aligned}
x_k &= \frac{\sigma_2\delta}{(\sigma_1+\sigma_2+\lambda k\Theta_I)[\delta+\lambda(1-\varepsilon)k\Theta_I] + \sigma_2\varepsilon\lambda k\Theta_I},\\
z_k &= \frac{\lambda(1-\varepsilon)k\Theta_I}{\delta+\lambda(1-\varepsilon)k\Theta_I} + \frac{\varepsilon\lambda k\Theta_I}{\delta+\lambda(1-\varepsilon)k\Theta_I}\cdot\frac{\sigma_2\delta}{(\sigma_1+\sigma_2+\lambda k\Theta_I)[\delta+\lambda(1-\varepsilon)k\Theta_I] + \sigma_2\varepsilon\lambda k\Theta_I}.
\end{aligned}$$

From relation (2) and $\langle k\rangle = \sum_s sP(s)$, one has

$$\Theta_I = \sum_k \frac{kP(k)z_k(t)}{\sum_s sP(s)}$$
$$= \frac{1}{\langle k\rangle}\sum_k kP(k)\Bigg\{\frac{\lambda(1-\varepsilon)k\Theta_I}{\delta+\lambda(1-\varepsilon)k\Theta_I} + \frac{\varepsilon\lambda k\Theta_I}{\delta+\lambda(1-\varepsilon)k\Theta_I}$$
$$\times \frac{\sigma_2\delta}{(\sigma_1+\sigma_2+\lambda k\Theta_I)[\delta+\lambda(1-\varepsilon)k\Theta_I]+\sigma_2\varepsilon\lambda k\Theta_I}\Bigg\}. \quad (4)$$

Obviously, $\Theta_I = 0$ is a solution of Eq.(4). Another stationary prevalence ($x_k \neq 0$) is obtained when the right-hand side and left-hand side of Eq.(4), expressed as function Θ_I, over the interval $0 < \Theta_I \leq 1$, allowing a nontrivial solution. It is easy to vertify that this corresponds to the inequality:

$$\frac{\mathrm{d}}{\mathrm{d}\Theta}\Bigg\{\frac{1}{\langle k\rangle}\sum_k kP(k)\Bigg[\frac{\lambda(1-\varepsilon)k\Theta_I}{\delta+\lambda(1-\varepsilon)k\Theta_I} + \frac{\varepsilon\lambda k\Theta_I}{\delta+\lambda(1-\varepsilon)k\Theta_I}$$
$$\times \frac{\sigma_2\delta}{(\sigma_1+\sigma_2+\lambda k\Theta_I)[\delta+\lambda(1-\varepsilon)k\Theta_I]+\sigma_2\varepsilon\lambda k\Theta_I}\Bigg]\Bigg\}\Bigg|_{\Theta_I=0} \geq 1,$$

that is,

$$\frac{\lambda(\sigma_1+\sigma_2-\varepsilon\sigma_1)\langle k^2\rangle}{\delta(\sigma_1+\sigma_2)\langle k\rangle} \geq 1,$$

where $\langle k^2\rangle = \sum_k k^2P(k)$. Let λ_c be the minimum value of λ satisfying the above inequality. Then

$$\lambda_c = \frac{\delta(\sigma_1+\sigma_2)\langle k\rangle}{(\sigma_1+\sigma_2-\varepsilon\sigma_1)\langle k^2\rangle}. \quad (5)$$

In an infinite-sized scale-free network with degree distribution $P(k) \sim k^{-\gamma}$ where $0 < \gamma \leq 1$, one has $\langle k^2\rangle \to \infty$,[20] so that the epidemic threshold λ_c of this new SIS model disappears for any fixed values of σ_1, σ_2, δ and ε, which is consistent with the other typical SIS models for scale-free networks. At the same time, one can easily find their difference — the new model relies on the infectivity parameters δ, σ_1 and σ_2, but the others do not.

Now, we consider a scale-free BA network model[30,31] with $P(k) = 2m^2/k^3$, where m is the minimum number of connections of individual nodes, and $\langle k\rangle = \int_m^\infty kP(k)\mathrm{d}k = 2m$. Clearly, $\langle k^2\rangle \simeq 2m^2\ln(K_c/m)$, $K_c \to \infty$. Substituting them into Eq.(5) yields

$$\lambda_c \approx \frac{\delta(\sigma_1+\sigma_2)}{(\sigma_1+\sigma_2-\varepsilon\sigma_1)m\ln(K_c/m)}.$$

In order to consider the dependence of network size N on λ_c, one has to relate the maximum connectivity K_c to N, which is given by $K_c \simeq mN^{\frac{1}{2}}$,[32] so

$$\lambda_c \simeq \frac{2\delta(\sigma_1+\sigma_2)}{(\sigma_1+\sigma_2-\varepsilon\sigma_1)m\ln N}. \quad (6)$$

Obviously, $\frac{\mathrm{d}\lambda_c}{\mathrm{d}\varepsilon} > 0$ and

$$\frac{\mathrm{d}\lambda_c}{\mathrm{d}\sigma_1} = \frac{\varepsilon\delta\sigma_2}{(\sigma_1+\sigma_2-\varepsilon\sigma_1)^2}\cdot\frac{\langle k\rangle}{\langle k^2\rangle} > 0,$$
$$\frac{\mathrm{d}\lambda_c}{\mathrm{d}\sigma_2} = \frac{-\varepsilon\sigma_1\delta}{(\sigma_1+\sigma_2-\varepsilon\sigma_1)^2}\cdot\frac{\langle k\rangle}{\langle k^2\rangle} < 0.$$

Here, λ_c is monotonously increasing with σ_1 and ε, and monotonously decreasing with σ_2. That is, for some special diseases, in order to control their spreading, one may try to enhance the efficacy of the vaccine-induced protection mechanism against infection ε and vaccination rate σ_1, to lower the vaccination-invalid rate σ_2. This is reasonable from a biological point of view.

While for a given endemic disease, the infective rate λ is known in advance. So, from relation (5), for given σ_2 and ε, it is easy to calculate the minimum value of σ_1, which gives some guideline of how many individuals to be vaccinated in order to control the disease. From relation (6), it can be concluded that the larger the network size, the easier the epidemic outbreaks.

In order to visualize the effect of vaccination, numerical simulations are carried out with the BA network model for the case that recovered nodes have immunity to disease. The network size is $N = 2\times10^3$, and the average degree $\langle k\rangle = 6$. All data are averaged over 10 independent runs.

Figure 3 shows the effective vaccination on the epidemic spreading for the case that the recovered nodes have immunity. The top (first) curve is the density $z(t)$ of infected nodes as a function of time

when there is no vaccination. Clearly, the disease will outbreak over the whole network. When the vaccination rate $\sigma_1 = 0.05$ and the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$ are imposed, from relation (6), one can easily calculate the maximal vaccination invalid-rate σ_2, which may control the disease, resulting in $\sigma_2^* = 9.6014\times10^{-4}$. Here, let $\sigma_2 = 9 \times 10^{-4} < 9.6014 \times 10^{-4}$, then the density $z(t)$ is plotted as the second curve. When the vaccination rate $\sigma_1 = 0.1$ and the vaccination-invalid rate $\sigma_2 = 0.01$ are imposed, from relation (6), one can easily calculate the efficacy of the vaccine-induced protection against infection ε, which may control the disease, resulting in $\varepsilon^* = 0.9830$. Here, let $\varepsilon = 0.99 > 0.9830$. The density $z(t)$ is plotted as the third curve. When the vaccination invalid rate $\sigma_2 = 0.01$ and the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$ are imposed, from relation (6), one can easily calculate the minimal vaccination-invalid rate σ_1, which may control the disease, resulting in $\sigma_1^* = 0.5211$. Here, let $\sigma_1 = 0.6 > 0.5211$, the density $z(t)$ curve is plotted as the one in the bottom. One can easily see that with the parameters calculated from relation (6), the density of the infected nodes decreases distinctly and the disease will be eliminated, revealing the necessary amount of vaccine to use for controlling the epidemic spreading for the case that the recovered nodes have immunity. Thus relation (6) gives a useful formula for effective control of epidemic spreading over scale-free networks.

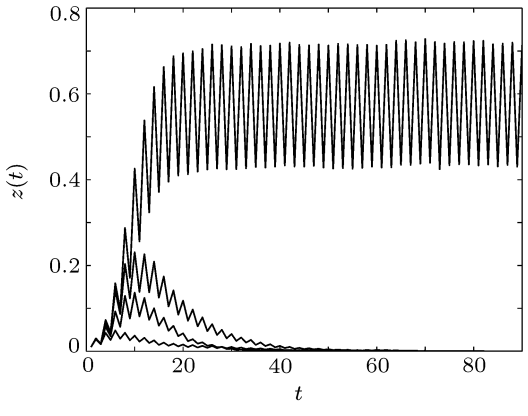


Fig.3. Density $z(t)$ of infected nodes under effective vaccination as a function of time in a BA network, for the case that the recovered nodes have immunity. The recovered rate $\delta = 0.4$, and the infected rates $\lambda = 0.3$.

By comparing Fig.3 with Fig.4, the theoretic and the simulated results have almost the same variation trend, although the bottom curve's end is a little higher in the theoretical result. Hence, using the mean-field analysis for the BA networks is quite effective.

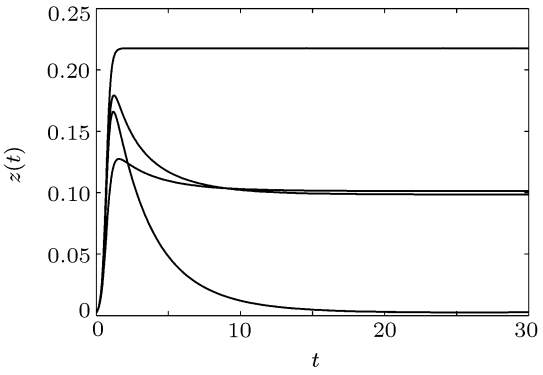


Fig.4. Density $z(t)$ of infected nodes under effective vaccination as a function of time for Eq.(1). All parameters are the same as those in Fig.3, and the curves are the same as those in Fig.3, from top to bottom.

In Fig.5, compared with the top curve (no vaccination), the lower curve shows the impact of vaccine on the epidemic spreading. Although the infected rate $\lambda = 0.5$ is above the theoretical threshold $\lambda_c = 0.0516$, from relation (6), which means that the disease must outbreak over the whole network, it can still make the density of the infected nodes decrease distinctly. This result shows that it is necessary to impose vaccination for the case that the recovered nodes have immunity, whether it can prevent the epidemic spreading or not, at least it can decrease the density of the infected nodes.

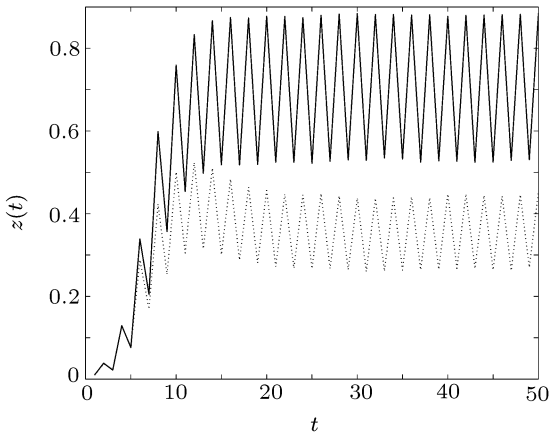


Fig.5. Density $z(t)$ of infected nodes as a function of time in a BA network for the case that the recovered nodes have immunity. The infected rate $\lambda = 0.5$, the recovered rate $\delta = 0.5$, the vaccination rate $\sigma_1 = 0.05$, the vaccination-invalid rate $\sigma_2 = 0.25$, the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$ (lower curve). And for the case without vaccination, $\lambda = 0.5$, $\delta = 0.5$, $\sigma_1 = 0$, $\sigma_2 = 0$ and $\varepsilon = 0$ (upper curve).

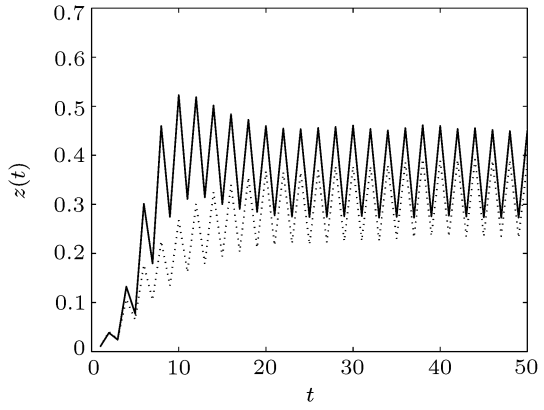


Fig.6. Density $z(t)$ of infected nodes as a function of time in a BA network for the case that the recovered nodes have immunity. The infected rate $\lambda = 0.5$, the recovered rates $\delta = 0.5$, the vaccination-invalid rate $\sigma_2 = 0.25$, the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$, and the vaccination rate $\sigma_1 = 0.50$, and 0.05 (lower to upper).

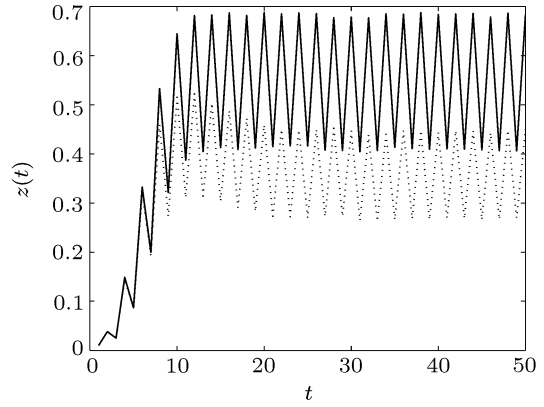


Fig.7. Density $z(t)$ of infected nodes as a function of time in a BA network for the case that the recovered nodes have immunity. The infected rate $\lambda = 0.5$, the recovered rates $\delta = 0.5$, the vaccination rate $\sigma_1 = 0.5$, the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$, and the vaccination-invalid rate $\sigma_2 = 0.25$, and 0.55 (lower to upper).

Figures 6–8 show the effects of the vaccination rate σ_1, the vaccination-invalid rate σ_2 and the efficacy of the vaccine-induced protection against infection ε on the epidemic spreading. From both theoretical analysis and numerical simulations, one can make effort to control the epidemic spreading by increasing the vaccination rate σ_1, decreasing the vaccination-invalid rate σ_2, or increasing the efficacy of the vaccine-induced protection against infection ε.

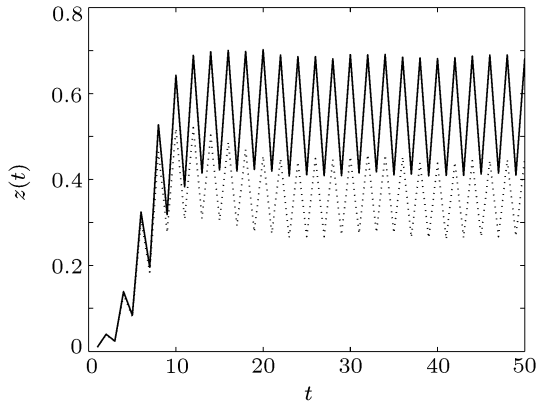


Fig.8. Density $z(t)$ of infected nodes as a function of time in a BA network for the case that the recovered nodes have immunity. The infected rate $\lambda = 0.5$, the vaccination rate $\sigma_1 = 0.05$, the vaccination-invalid rate $\sigma_2 = 0.25$, the recovered rate $\delta = 0.4$, and the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9, 0.6$ (lower to upper).

2.2. SIS model for the case that recovered nodes have no immunity

Suppose that after having recovered through medical treatment the nodes will be infected once again immediately if they contact with infected nodes. In this case, the recovered nodes are all susceptible nodes in set A (see Fig.2). All nodes will evolve according to the following transition patterns:

$$\begin{aligned}
\dot{S_{uk}}(t) &= -\lambda k \Theta_I S_{uk}(t) - \sigma_1 S_{uk}(t) + \sigma_2 S_{vk}(t) + \delta I_k(t), \\
\dot{S_{vk}}(t) &= \sigma_1 S_{uk}(t) - \sigma_2 S_{vk}(t) - \lambda(1-\varepsilon) k \Theta_I S_{vk}(t), \\
\dot{I_k}(t) &= \lambda k \Theta_I S_{uk}(t) + \lambda(1-\varepsilon) k \Theta_I S_{vk}(t) - \delta I_k(t).
\end{aligned}$$

Let $x_k(t) = \dfrac{S_{uk}(t)}{N_k(t)}$, $y_k(t) = \dfrac{S_{vk}(t)}{N_k(t)}$ and $z_k(t) = \dfrac{I_k(t)}{N_k(t)}$. Then using the same method as in subsection 2.1, by imposing the stationarity condition of $\dot{x_k}(t) = 0$ and $\dot{z_k}(t) = 0$, one has

$$\begin{aligned}
x_k &= \frac{\sigma_2\delta + \delta(1-\varepsilon)\lambda k \Theta_I}{(\sigma_1 + \sigma_2 + \lambda k \Theta_I)[\delta + \lambda(1-\varepsilon) k \Theta_I] + \sigma_2 \varepsilon \lambda k \Theta_I - \delta \varepsilon \lambda k \Theta_I}, \\
z_k &= \frac{\lambda(1-\varepsilon) k \Theta_I}{\delta + \lambda(1-\varepsilon) k \Theta_I} + \frac{\varepsilon \lambda k \Theta_I}{\delta + \lambda(1-\varepsilon) k \Theta_I} \cdot \frac{\sigma_2\delta + \delta(1-\varepsilon)\lambda k \Theta_I}{(\sigma_1 + \sigma_2 + \lambda k \Theta_I)[\delta + \lambda(1-\varepsilon) k \Theta_I] + \sigma_2 \varepsilon \lambda k \Theta_I - \delta \varepsilon \lambda k \Theta_I}.
\end{aligned}$$

The minimum value of λ is given by

$$\lambda_c = \frac{\delta(\sigma_1 + \sigma_2)\langle k\rangle}{(\sigma_1 + \sigma_2 - \varepsilon\sigma_1)\langle k^2\rangle}. \tag{7}$$

Compared with Subsection 2.1, the SIS model under these two transmitting modes on scale-free networks has the same epidemic threshold λ_c, but their equilibrium points are different. These results reveal that whether or not the recovered nodes have immunity to the disease is not a determining factor for disease prevalence; it only influences the number of infected nodes in the whole network.

To see the effects of vaccination, numerical simulations are carried out on a scale-free BA network for the case that the recovered nodes have no immunity to the disease, with the network size $N = 2 \times 10^3$ and the average degree $\langle k\rangle = 6$. All data are averaged over 10 independent runs.

Figure 9 shows the effective vaccination to the epidemic spreading for the case that the recovered nodes have no immunity. The top (first) curve is the density $z(t)$ of infected nodes as a function of time when there is no vaccination, where the disease will outbreak over the whole network. The second, third and the bottom curves have the same parameters as those in Fig.3, but the infected node density decreases not as distinctly as in Fig.3. This result reveals that different transmitting modes should receive vaccination to different extents, although the same amount of vaccine may not have the same impact on these two types of diseases; they can all decrease the density of the infected nodes.

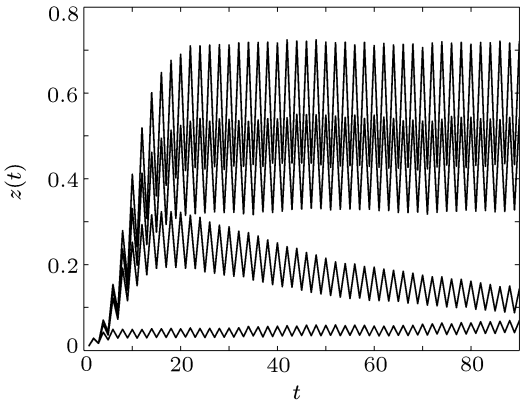


Fig.9. Density $z(t)$ of infected nodes under effective vaccination as a function of time in a BA network for the case that the recovered nodes have no immunity. The recovered rate $\delta = 0.4$ and the infected rates $\lambda = 0.3$.

By comparing Fig.9 with Fig.10, the theoretical and the simulated results have almost the same variation trend, although the bottom curve's end is a little higher in the theoretical result. Hence, using the mean-field analysis on the BA networks is quite effective. And comparing Fig.10 with Fig.4, and Fig.9 with Fig.3, shows the same phenomenon that the disease is prone to transmit for the case that the recovered nodes have no immunity.

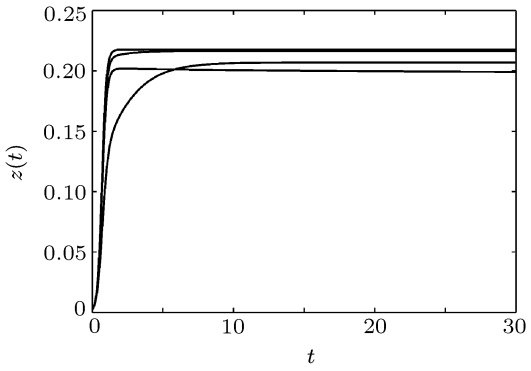


Fig.10. Density $z(t)$ of infected nodes under effective vaccination as a function of time for the theoretical mean-field model discussed in Subsection 2.2. All parameters are the same as those in Fig. 9, and the curves are the same as those in Fig.3 from top to bottom.

Figure 11 shows the effects of vaccination on the epidemic spreading, where the density of the infected nodes is distinctly decreasing with the vaccination. But, comparing it with Fig.5, one can easily see that in order to attain almost the same density of $z(t)$, for the case that the recovered nodes have no immunity, the vaccination rate σ_1 is larger than, the vaccination-invalid rate σ_2 is smaller than, and the efficacy of the vaccine-induced protection against infection ε is larger than the corresponding values in Fig.5.

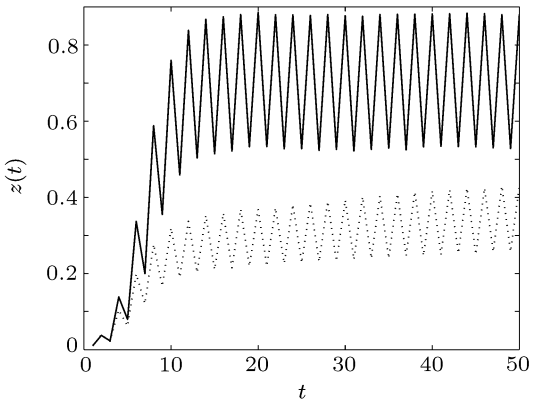


Fig.11. Density $z(t)$ of infected nodes as a function of time in a BA network for the case that the recovered nodes have no immunity. The infected rate $\lambda = 0.5$, the recovered rates $\delta = 0.5$, and the vaccination rate $\sigma_1 = 0.25$, the vaccination-invalid rate $\sigma_2 = 0.05$, the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.99$ (lower). And for the case without vaccination, $\lambda = 0.5$, $\delta = 0.5$, $\sigma_1 = 0$, $\sigma_2 = 0$ and $\varepsilon = 0$ (upper).

Figures 9 and 11 show that in order to control epidemic spreading in the case that the recovered nodes have no immunity in the BA network, more efforts have to be made. It is also clear that, the theoretical relation (6) is very important to the case that the recovered nodes have immunity to eliminate the disease, but not to the case without immunity.

Figure 12 shows the effect of vaccination rate σ_1 on the epidemic spreading. It is easy to see that increasing the vaccination rate can decrease the density of the infected nodes to some extent.

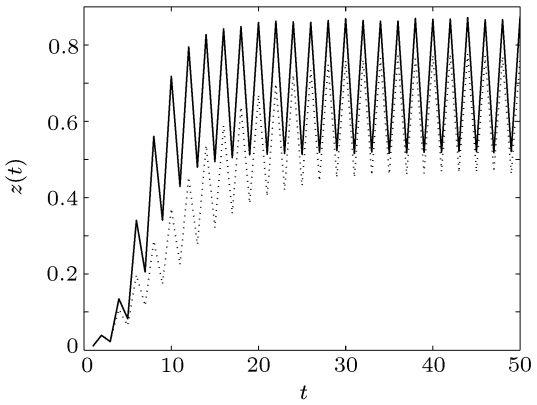


Fig.12. Density $z(t)$ of infected nodes as a function of time in a BA network for the case that the recovered nodes have no immunity. The infected rate $\lambda = 0.5$, the recovered rates $\delta = 0.5$, the vaccination-invalid rate $\sigma_2 = 0.25$, the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$, and the vaccination rate $\sigma_1 = 0.50$, and 0.05 (lower to upper).

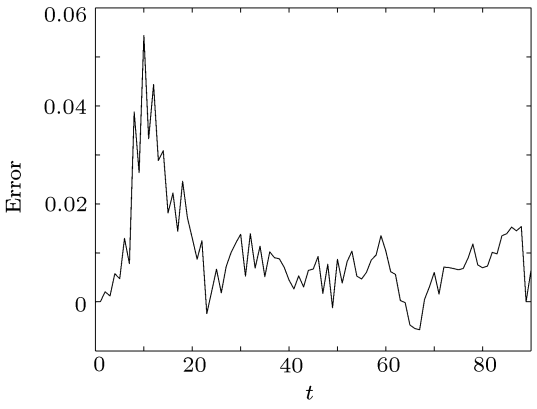


Fig.13. The error of infected node density as a function of time in a BA network for the case that the recovered nodes have no immunity, when the vaccination-invalid rate changes from $\sigma_2 = 0.25$ to 0.55. The infected rate $\lambda = 0.5$, the recovered rates $\delta = 0.5$, the vaccination rate $\sigma_1 = 0.5$, and the efficacy of the vaccine-induced protection against infection $\varepsilon = 0.9$.

Figures 13 and 14 show the errors of $z(t)$ when the vaccination-invalid rate σ_2 and the efficacy of the vaccine-induced protection against infection ε are changed, respectively, where the error is very small, which suggests that the epidemic spreading is robust against σ_2 and ε. Hence, by decreasing σ_2 or increasing ε to control the disease is more difficult; the best effort would be to increase the vaccination rate σ_1.

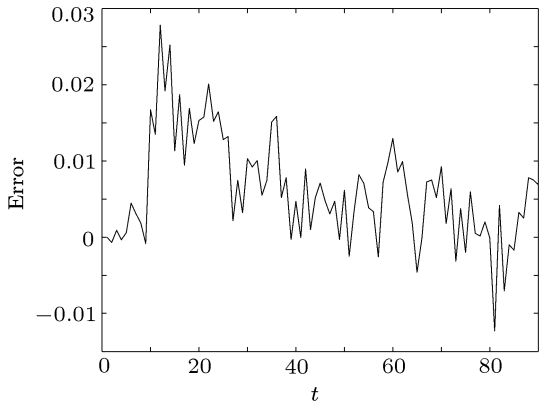


Fig.14. The error of infected node density as a function of time in a BA network for the case that the recovered nodes have no immunity, when the efficacy of the vaccine-induced protection against infection changes from $\varepsilon = 0.9$ to 0.6. The infected rate $\lambda = 0.5$, the vaccination rate $\sigma_1 = 0.05$, the vaccination-invalid rate $\sigma_2 = 0.25$, and the recovered rates $\delta = 0.4$.

All numerical simulation results for BA networks have shown that the epidemic spreading in scale-free networks is oscillating and periodic, for both cases that the recovered nodes have or have not immunity. Relation (6), obtained by theoretical analysis, gives a formula for controlling epidemic spreading for the case that recovered nodes have immunity, but not for the case without immunity. When the network size is finite, as verified by numerical simulations, the disease can be treated as endemic.

3. Conclusion

In this paper, a new epidemic spreading SIS-like model has been proposed and simulated by taking into account the impact of imperfect vaccination. The disease transmission dynamics in the two cases that the recovered nodes have immunity or have not, have been evaluated for scale-free networks. The vaccination rate, the vaccination-invalid rate, and the efficiency of the vaccine-induced protection against infection are shown to be important factors for disease outbreak. Analysis has led to a useful formula for calculating the vaccination rate needed for disease prevention, and simulated results have shown that, for the case that the recovered individuals have immunity to disease,

there are three effective ways to prevent disease prevalence: increasing the vaccination rate, decreasing the vaccination-invalid rate, and increasing the efficacy of the vaccine-induced protection against infection, but for the case without immunity, increasing the vaccination rate alone could be the best possible solution. Finally, based on both analytic and simulated results, it is concluded that the disease can be prone to transmit for the case that recovered nodes have no immunity, which is consistent with the common sense.

References

[1] Liljeros F, Edling C R, Amaral L A N, Stanley H E and Aberg Y 2001 *Nature* (London) **411** 907

[2] Ebel H, Mielschand L I and Bornholdt S 2002 *Phys. Rev.* E **66** 035103

[3] Faloutsos M, Faloutsos P and Faloutsos C 1999 *Comput. Commun. Rev.* **29** 251

[4] Lawrence S and Giles C L 1998 *Science* **280** 98

[5] Montoya J M and Solé R V 2002 *J. Theor. Biol.* **214** 405

[6] Dorogovtsev S N and Mendes J F F 2002 *Adv. Phys.* **51** 1079

[7] Li Y, Liu Y, Shan X M, Ren Y, Jiao J and Qiu B 2005 *Chin. Phys.* **14** 2153

[8] Marder M 2007 *Phys. Rev.* E **75** 066103

[9] Draief M 2006 *Physica* A **363** 120

[10] Xu X J, Wu Z X and Chen G R 2007 *Physica* A **377** 125

[11] Yan G, Fu Z Q, Ren J and Wang W X 2007 *Phys. Rev.* E **75** 016108

[12] Karsai M, Juhász R and Iglói F 2006 *Phys. Rev.* E **73** 036116

[13] Piccardi C and Casagrandi R 2008 *Phys. Rev.* E **77** 026113

[14] Bai W J, Zhou T and Wang B H 2007 *Physica* A **384** 656

[15] Sun H J and Gao Z Y 2007 *Physica* A **381** 491

[16] Bab M A and Albano E V 2008 *J. Phys.* A: *Math. Theor.* **41** 045001

[17] Florian L, Johannes T G, Silvio R D and Haye H 2008 *J. Phys.* A: *Math. Theor.* **41** 185005

[18] Pastor-Satorras R and Vespignani A 2001 *Phys. Rev. Lett.* **175** 3200

[19] Pastor-Satorras R and Vespignani A 2001 *Phys. Rev.* E **63** 066117

[20] Pastor-Satorras R and Vespignani A 2002 *Phys. Rev.* E **65** 036104

[21] Shi H J, Duan Z S and Chen G R 2008 *Physica* A **387** 2133

[22] Yang R, Wang B H, Ren J, Bai W J, Shi Z W, Xu W and Zhou T 2007 *Phys. Lett.* A **364** 189

[23] Pei W D, Chen Z Q and Yang Z Z 2008 *Chin. Phys.* B **17** 373

[24] Barthélemy M, Barrat A, Pastor-Satorras R and Vespignani A 2004 *Phys. Rev. Lett.* **92** 178701

[25] Barthélemy M, Barrat A, Pastor-Satorras R and Vespignani A 2005 *J. Theor. Biol.* **235** 275

[26] Zhou T, Liu J G, Bai W J, Chen G R and Wang B H 2006 *Phys. Rev.* E **74** 056109

[27] Hayashi Y, Minoura M and Matsukubo J 2004 *Phys. Rev.* E **69** 016112

[28] Guo W P, Li X and Wang X F 2004 *Physica* A **380** 684

[29] Gumel A B, McCluskey C C and Driessche P van den 2006 *Bull. Math. Biol.* **68** 2105

[30] Barabási A L and Albert R 1999 *Science* **286** 509

[31] Barabási A L, Albert R and Jeong H 1999 *Physica* A **272** 173

[32] Takeuchi Y, Ma W B and Beretta E 2000 *Nonl. Anal.* **42** 931