Epidemic spreading in scale-free networks including the effect of individual vigilance*

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(Received 23 June 2011; revised manuscript received 30 July 2011)

In this paper, we study the epidemic spreading in scale-free networks and propose a new susceptible-infected-recovered (SIR) model that includes the effect of individual vigilance. In our model, the effective spreading rate is dynamically adjusted with the time evolution at the vigilance period. Using the mean-field theory, an analytical result is derived. It shows that individual vigilance has no effect on the epidemic threshold. The numerical simulations agree well with the analytical result. Furthermore, we investigate the effect of individual vigilance on the epidemic spreading speed. It is shown that individual vigilance can slow the epidemic spreading speed effectively and delay the arrival of peak epidemic infection.

Keywords: scale-free network, susceptible-infected-recovered model, individual vigilance, epidemic threshold

PACS: 02.50.Le, 05.45.–a **DOI:** 10.1088/1674-1056/21/1/010205

1. Introduction

Many real-world systems can be properly described as complex networks with vertices representing individuals or organizations and links mimicking the interactions among them. Recently, numerous power-law degree distribution networks in real life have attracted a lot of attention.[1−3] One of the primary reasons for studying network structures is to understand the mechanisms of epidemic spreading. To date, the epidemic spreading in scale-free networks has been studied by many researchers[4−18] and some constructive conclusions have been obtained using the susceptible-infected-susceptible (SIS),[4−10] the susceptible-infected-removed (SIR),[11−14] and the susceptible-infected (SI)[15−18] models. For example, the absence of an epidemic threshold has been observed in a wide range of SF networks of infinite size, except in Ref. [13], where the epidemic threshold $\lambda_c = 1/A$, with A being the constant infectivity of each node. In this paper, we mainly concentrate on the behaviour of the SIR model.

In the models mentioned above, the effective spreading rate is a constant in the whole epidemic spreading process. That is to say, each individual has no vigilant reaction to the epidemic spreading, or the vigilant reactions are not adjusted at all time steps. However, generally, in a real epidemic spreading process, the susceptible nodes generally have vigilant reactions against the epidemic and adjust the reactions according to the trend of the epidemic spreading. Individual vigilance increases if the epidemic spreads quickly and, conversely, vigilance will decrease or even disappear from the population. Therefore, not only does each susceptible individual has vigilant reactions to the epidemic infection, but also vigilant reactions are dynamical and change with the epidemic spreading time. In other words, at different time steps, the effective spreading rates are different.

By incorporating the factor of a node's dynamical vigilance to the epidemic infection to the standard SIR model, we propose a new SIR epidemic spreading model including the effect of individual vigilance in scale-free networks. Unlike Ref. [19], in our model, the individual vigilance is adaptively aware of the epidemic according to the speed of epidemic transmis-

*Project supported by the National Natural Science Foundation of China (Grant No. 60874091), the Six Projects Sponsoring Talent Summits of Jiangsu Province, China (Grant No. SJ209006), the Natural Science Foundation of Jiangsu Province, China (Grant No. BK2010526), and the Graduate Student Innovation Research Project of Jiangsu Province, China (Grant No. CXLX11_0417).

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sion. The proposed model has the following properties. i) Individual vigilance has no effect on the epidemic threshold. ii) Individual vigilance can slow the epidemic spreading speed effectively and delay the arrival of the epidemic infection peak value.

The rest of this paper is organized as follows. In Section 2, a new SIR model considering individual vigilance in scale-free networks is proposed. In Section 3, we analyze the proposed model theoretically using the mean-field approximation. In Section 4, simulations and results analysis are given. Section 5 concludes the paper.

2. The SIR model with effect of individual vigilance

We consider the SIR model defined on a scale-free network with general node degree distribution $P(k)$. In the SIR model, each node of the network has one of three possible states: susceptible (S); infected (I); and removed (R). At each time step, a susceptible node connected to an infected one will be infected with a probability v. At the same time, the infected nodes will be removed with a probability δ. An effective spreading rate $\beta = v/\delta$ (without lack of generality, we set $\delta = 1$) is defined as the effective infected probability per capita contact of infected individuals. So, we can easily obtain that a susceptible node a will be infected with probability $1-(1-\beta)^{k_i}$ at time step t, where k_i denotes the number of contacts between a and its infected neighbours at time step $t-1$. Let $I_k(t)$, $S_k(t)$, and $R_k(t)$ be the density of infected, susceptible, and removed nodes of degree k at time t, respectively. They satisfy the following normalization condition:

$$I_k(t) + S_k(t) + R_k(t) = 1. \tag{1}$$

Global quantities, such as the epidemic prevalence, are therefore expressed by the averages over the various degree classes, that is

$$\begin{cases} I(t) = \sum\limits_k P(k) I_k(t), \\ S(t) = \sum\limits_k P(k) S_k(t), \\ R(t) = \sum\limits_k P(k) R_k(t). \end{cases} \tag{2}$$

Now, by using the mean-field approximation, the rate equations for the partial densities in the scale-free network characterized by degree distribution $P(k)$ can be defined as[11]

$$\begin{cases} \dfrac{\mathrm{d}I_k(t)}{\mathrm{d}t} = -I_k(t) + \beta k S_k(t)\Theta(t), \\ \dfrac{\mathrm{d}S_k(t)}{\mathrm{d}t} = -\beta k S_k(t)\Theta(t), \\ \dfrac{\mathrm{d}R_k(t)}{\mathrm{d}t} = I_k(t), \end{cases} \tag{3}$$

where $\Theta(t)$ denotes the average probability of any given link pointing to an infected node. Neglecting the degree correlations in the network, $\Theta(t)$ can be expressed by[20,21]

$$\Theta(t) = \frac{\sum\limits_k kP(k)I_k(t)}{\langle k \rangle}. \tag{4}$$

So, the first equation in Eq. (3) can be rewritten as

$$\frac{\mathrm{d}I_k(t)}{\mathrm{d}t} = -I_k(t) + \beta k S_k(t) \frac{\sum\limits_k kP(k)I_k(t)}{\langle k \rangle}. \tag{5}$$

In such a standard SIR model, the effective spreading rate β is generally a constant in all time steps.

In our work, we represent individual vigilance with two parameters. i) The vigilance period τ: indicates the length of time when an individual is vigilant against infection. We assume that the vigilance period starts at T_1 time step and ends at T_2 time step. So, τ can be expressed as $\tau = T_2 - T_1$. At the same time, we suppose that all individuals have the same τ. ii) The vigilance function $g(\tau, t)$: indicates the individual vigilant level during τ. The $g(\tau, t)$ must be a piecewise continuous differentiable function. At the same time, it also satisfies $0 < g(\tau, t) < 1$ when $T_1 < t < T_2$ and $g(\tau, t) = 0$ when $t \le T_1$ or $t \ge T_2$. An example of individual vigilance function $g(\tau, t)$ is shown in Fig. 1.

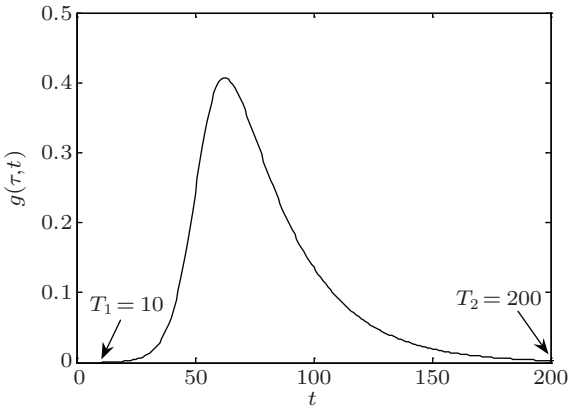


Fig. 1. An example of $g(\tau, t)$.

Generally, the higher vigilance an individual has, the lower value β would be. So, we redefine β in the following form:

$$\beta'(t) = \beta(1 - g(\tau, t)). \tag{6}$$

Replacing β with $\beta'(t)$ in Eq. (3), we can construct the novel SIR model with individual vigilance as follows:

$$\begin{cases} \dfrac{\mathrm{d}I_k(t)}{\mathrm{d}t} = -I_k(t) + \beta(1 - g(\tau, t))kS_k(t)\dfrac{\sum\limits_k kP(k)I_k(t)}{\langle k\rangle}, \\ \dfrac{\mathrm{d}S_k(t)}{\mathrm{d}t} = -\beta(1 - g(\tau, t))kS_k(t)\dfrac{\sum\limits_k kP(k)I_k(t)}{\langle k\rangle}, \\ \dfrac{\mathrm{d}R_k(t)}{\mathrm{d}t} = I_k(t). \end{cases} \tag{7}$$

Clearly, $\beta'(t) = \beta$ when $g(\tau, t) = 0$, meaning that all individuals have no vigilance corresponding to the standard SIR model. In addition, $\beta'(t) = 0$ when $g(\tau, t) = 1$, which describes that all of the individuals have the highest vigilance level, leading to no extra individual being infected.

3. Epidemic threshold of the model

At the beginning of an epidemic spreading, the infected individuals are very few, so the initial conditions for Eq. (7) are $R_k(0) = 0$, $I_k(0) \cong 0$, $S_k(0) = 1 - R_k(0) - I_k(0) = 1$ and $g(\tau, 0) = 0$. Combined with these initial conditions, the equation for density $S_k(t)$ can be drawn from the second equation in Eq. (7),

$$S_k(t) = \exp\left[-k\beta \int_0^t (1 - g(\tau, u)) \times \frac{1}{\langle k\rangle} \sum_k kP(k)I_k(u)\,\mathrm{d}u \right]. \tag{8}$$

Define auxiliary function $\phi(t)$ as

$$\phi(t) = \int_0^t (1 - g(\tau, u)) \frac{1}{\langle k\rangle} \sum_k kP(k)I_k(u)\,\mathrm{d}u. \tag{9}$$

From the third equation in Eq. (7), and combined with initial conditions, we can rewrite Eq. (9) as ($t \geq T_2$)

$$\begin{aligned} \phi(t) &= \int_0^t (1 - g(\tau, u)) \frac{1}{\langle k\rangle} \sum_k kP(k)\,\mathrm{d}(R_k(u)) \\ &= (1 - g(\tau, t)) \frac{1}{\langle k\rangle} \sum_k kP(k)R_k(t) + M, \end{aligned} \tag{10}$$

where $M = \int_{\mathrm{T}_1}^{\mathrm{T}_2} \frac{1}{\langle k\rangle} \sum\limits_k kP(k)R_k(u)\,\mathrm{d}(g(\tau, u))$.

Focusing on Eqs. (9) and (10), calculating the time derivative of function $\phi(t)$ in Eq. (10), we can obtain

$$\begin{aligned} \frac{\mathrm{d}\phi(t)}{\mathrm{d}t} &= (1 - g(\tau, t)) \frac{1}{\langle k\rangle} \sum_k kP(k)I_k(t)\,\mathrm{d}t \\ &= \frac{1}{\langle k\rangle} (1 - g(\tau, t)) \sum_k kP(k)(1 - S_k(t) - R_k(t)) \\ &= \frac{1}{\langle k\rangle} (1 - g(\tau, t)) \sum_k kP(k)(1 - S_k(t) - R_k(t)) \\ &= (1 - g(\tau, t)) - \frac{(1 - g(\tau, t))}{\langle k\rangle} \\ &\quad \times \sum_k kP(k)S_k(t) - \phi(\mathrm{t}) + M. \end{aligned} \tag{11}$$

Since $I_k(\infty) = 0$, we have $\lim\limits_{t\to\infty} \mathrm{d}\phi(t)/\mathrm{d}t = 0$. According to the definition of $g(\tau, t)$, $\lim\limits_{t\to\infty} g(\tau, t) = 0$. Therefore, we can obtain the following equation from Eq. (11):

$$\phi(\infty) = 1 - \frac{1}{\langle k\rangle} \sum_k kP(k)S_k(\infty) + M. \tag{12}$$

From Eqs. (8) and (12), we can obtain the following self-consistent equation for ϕ_∞:

$$\begin{aligned} \phi(\infty) &= 1 - \frac{1}{\langle k\rangle} \sum_k kP(k) \exp(-\beta k\phi(\infty)) + M \\ &\equiv F(\phi(\infty)). \end{aligned} \tag{13}$$

Clearly, when $\phi_\infty = 0$, from Eq. (9), we can obtain $I_k(t) \equiv 0$ and then $R_k(t) \equiv 0$, which means $M = 0$. That is to say, $\phi_\infty = 0$ is always a solution (trivial solution) to Eq. (13). We are very interested in the conditions in which a nontrivial solution to Eq. (13) exists. From Eq. (13), we can obtain that if the epidemic spreading reaches a steady state, it must satisfy condition $\phi_\infty > 0$ ($0 < \phi_\infty \leq 1$). It is easy to realize that this corresponds to the inequality

$$\left. \frac{\mathrm{d}F(\phi(\infty))}{\mathrm{d}\phi(\infty)} \right|_{\phi(\infty)=0} \geq 1 \tag{14}$$

being satisfied. That is

$$\left. \frac{\mathrm{d}}{\mathrm{d}\phi(\infty)} \left(1 - \frac{1}{\langle k\rangle} \sum_k kP(k) \exp(-\beta k\phi(\infty)) + M \right) \right|_{\phi(\infty)=0} \geq 1. \tag{15}$$

Then, we will obtain the following inequality:

$$\frac{\beta}{\langle k\rangle}\sum_k k^2 P(k) \geq 1. \tag{16}$$

The value of β yielding the equality in Eq. (17) defines the critical epidemic threshold β_c, which is given by

$$\beta_c = \frac{\langle k\rangle}{\langle k^2\rangle}. \tag{17}$$

If β is below β_c, the epidemic will gradually die out. On the contrary, if β is above β_c, the epidemic will break out and spread on the network, finally reaching a special steady state. From Eq. (17), we can draw the conclusion that the epidemic threshold in our proposed model is identical to the value in the standard SIR model.[11] This means that individual vigilance has no effect on the epidemic threshold.

4. Simulations and analysis

To further assist understanding the proposed model, we give a set of simulations based on the Barabasi–Albert (BA) network[22] to validate the above analysis result. The parameters used to generate the BA network are: $N = 1000$, $m_0 = m = 3$ and $\langle k\rangle = 6$. Each simulation run begins with one randomly chosen infected node in the network and each simulation plot shown is averaged over 100 independent simulation runs.

For comparison, we choose vigilance function $g(\tau, t)$ as follows:

(I) $g(\tau,t) = \begin{cases} \delta I(t - T_1), T_1 \leq t \leq T_2, \\ 0, t < T_1 \ \text{ or } \ t > T_2, \end{cases}$

(II) $g(\tau, t) = 0$.

In the simulation, function $g(\tau, t)$ does not always need to satisfy continuity and differentiability strictly.

Case (II) means that the individual has no vigilance. Here, we set $\delta = 0.5$, $T_1 = 10$ and $T_2 = 200$, then we have $\tau = T_2 - T_1 = 190$. The relations between R_∞ and effective spreading rate β are shown in Fig. 2.

From Eq. (17) and the set simulation parameters, we can obtain the epidemic threshold $\beta_c = 0.0745$. In Fig. 2, the diamond and the circle lines denote cases (I) and (II) (no vigilance) for $g(\tau, t)$, respectively. As the arrow denotes in the insert of Fig. 2, we can see clearly that the epidemic thresholds both are almost equal to the theoretical value of $\beta_c = 0.0745$, although case (I) has considered individual vigilance. This means that the simulations agree well with the predictions of our epidemic spreading model in Section 3. That is to say, individual vigilance has no effect on the epidemic threshold.

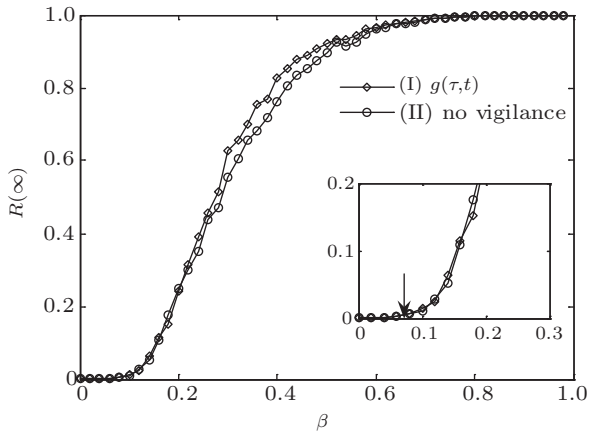


Fig. 2. Relations between $R(\infty)$ and β for different vigilance function $g(\tau, t)$. The insert shows details in the small β part.

In order to further demonstrate the analysis results about the epidemic threshold under individual vigilance, we use case (I) vigilance function. At the same time, we set T_1 to 0, T_2 to 0, 50, 100 and 200, respectively, and $\delta = 0.8$. The relations between R_∞ and effective spreading rate β are shown in Fig. 3.

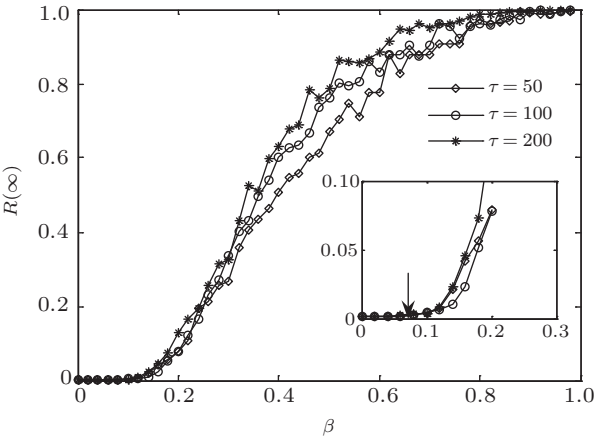


Fig. 3. Relations between $R(\infty)$ and β for different τ with the same vigilance function. The insert shows details in the small β part.

Analyzing the curves in Fig. 3, we can conclude that the epidemic threshold does not change with the vigilance period increasing. This also validates the result that individual vigilance has no effect on the epidemic threshold.

In order to investigate the effect of individual vigilance on the epidemic spreading process, we further

simulate the evolution process of the epidemic spreading. The vigilance function is the same as the previous simulations. At the same time, the other parameters are set to $\beta = 0.2$, $\delta = 0.5$, $T_1 = 0$ and $T_2 = 200$. Simulation results are shown in Fig. 4.

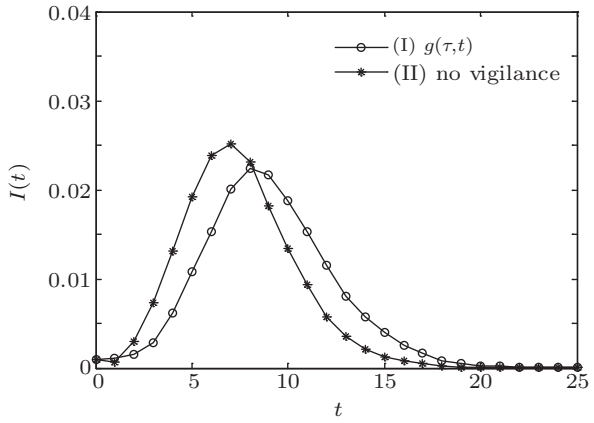


Fig. 4. Relation between $I(t)$ and evolution time for different $g(\tau, t)$.

Since the infected node removal probability is $\delta = 1$, $I(t)$ means the density of newly infected nodes. In the other words, $I(t)$ is the speed of the epidemic spreading. Apparently, in Fig. 4, we can find that if each individual is vigilant to the epidemic, the epidemic infection peak time will delay and its value will reduce as compared with those in the case of no vigilance. This may leave us enough time to develop control measures (such as vaccination).

5. Conclusion

In this paper, considering the effect of individual vigilance, we have investigated epidemic spreading in scale-free networks and propose a new SIR model, in which the effective spreading rate is dynamically adjusted with the time evolution. Individual vigilance was expected to decrease the epidemic threshold. In fact, what is interesting, the epidemic threshold remains unchanged, which has been proved by theoretical analysis and the numerical simulation in this paper. In addition, we have also studied the effect of individual vigilance on the epidemic spreading speed. It has been shown that individual vigilance can slow the epidemic spreading speed effectively and delay the arrival of the epidemic infection peak value.

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