Vaccination strategies of a modified SIS model on complex networks

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***Abstract*— A new susceptible-infected-susceptible model is researched in this paper, which has an infective vector. And it describes epidemics (e.g. malaria) transmitted through an infective vector (e.g. mosquitoes) on complex networks. We compare the modified model with the standard SIS model having an infective vector. We also study and compare the effects of the uniform immunization and targeted immunization on complex networks. Then, analytical and simulated results are given to show that the uniform immunization strategy to the modified model is very effective on scale-free networks.**

***Keywords*— Complex network, Disease spread, Immunization, Infective vector.**

I. INTRODUCTION

In recent years, how the infectious diseases transmitted has attracted much attention and interest both medical practice and academia. The development of such mathematical models [1],[2],[3] is aimed at understanding the mechanisms of infectious diseases. These models predict the consequences of the presentation of public health interventions to control the transmission of diseases, and they help us to expound on effective strategies to minimize the destructiveness caused by various infectious diseases.

Many studies on complex networks, such as susceptible-infected-susceptible (SIS) [4]–[8], susceptible-infected-removed (SIR) [9],susceptible -infected (SI) [10]–[12], have been researched during the past years. Cooke proposed a model for the transmission of an infectious disease [13]. Busenberg and Cooke[14], Marcati and Pozio [15] considered the extensions of the model. Only the spread of an infectious disease transmitted by a vector was considered by them. Actually, many factors influence the dynamics of some diseases (e.g. malaria, yellow and dengue fever), such as humans, the vector and the blood transfusion transmission, as well as the environment. It affects directly or indirectly these elements and the interrelations among them [20],[21].

Other hosts such as mosquitoes mediate disease transmission in many infectious human diseases [22]. On this occasion, diseases transmission not only by contacts between individuals, but also by contacts between individuals and vector. Although the human's contacts can be considered as SF, a mosquito may bite a person without any selectivity. To research such a spreading characteristic, this paper extends the standard model to qualitatively understand and describe the mechanism of epidemics spread by applying two immunizations strategies (uniform immunization and targeted immunization).

The following SIS model with an infective vector on complex networks [23] is discussed:

$$\begin{cases} \dot{I}_k(t) = -I_k(t) + \lambda k[1-I_k(t)]\Theta(\rho(t)) + \gamma_1[1-I_k(t)]v(t) \\ \dot{v}(t) = -v(t) + \gamma_2[1-v(t)]\Theta(\rho(t)) \end{cases} \quad (1)$$

In the above model, both individuals and infective vector are considered as the same nodes in the network, but it is not the case in reality. Actually, the human contacts can be considered as scale-free, but the infective vector will bite a person no matter what the degree of this person is. However, they assume that a mosquito is likely to bite a person with large degree in [23]. A modified SIS model with an infective medium was proposed by Yang et al. [24] aimed at above model.

Based on the above SIS model, we investigate a modified SIS model in this paper with an infective medium on complex networks. Furthermore, we will show that the main features and theoretical results obtained are different from those presented

in the above SIS model by applying uniform immunization and targeted immunization to the modified model.

The rest of the paper is organized as follows. In Section 2, we construct the SIS model with an infective vector on complex networks. We discuss the effect of the uniform immunization strategy and Targeted immunization strategy on the modified model in Section 3. Finally, Section 4 concludes the paper.

II. THE MODEL

Generally speaking, in most of SIS-like models there is only one type of node-individuals [22] and the epidemic can only be diffused by individual contacts. We usually neglect the influence of the infected vector such as mosquitoes.

In this paper, the epidemic model is defined as follows: there are two types of nodes in a network, one is composed of individuals and the other is composed of female mosquitoes (only female mosquitoes infect humans) [20], [22]. All nodes can only exist in one of the two discrete states, susceptible (i.e. healthy) or infected. The model in this paper completely neglects the details of the infection mechanism within each individual. And the disease transmission is described in the following way: At each time step, a susceptible individual is infected with probability γ if it is connected to an infected individual, and infected individuals are cured and become susceptible again with probability δ. Therefore $\lambda=\gamma/\delta$ (without lack of generality, we set $\delta=1$) is considered as an effective spreading rate. Different from other models, at each time step here, susceptible individuals are infected with probability γ_1 due to the bites by infected mosquitoes. Moreover, susceptible mosquitoes are infected with probability γ_2 due to biting on infected persons. Then, all individuals as well as mosquitoes run through the susceptible-infected-susceptible cycles stochastically. Here, we will not incorporate the possibility of individual removal due to birth and death or acquired immunization. Also it is assumed that there is no infection spreading between mosquitoes. Moreover, we assume that the infected female mosquitoes will recover to susceptible with the probability μ and that there is no natural death or disease-related death with respect to mosquitoes, so the number of female mosquitoes is constant. The transmission sketch is shown in Fig.1.

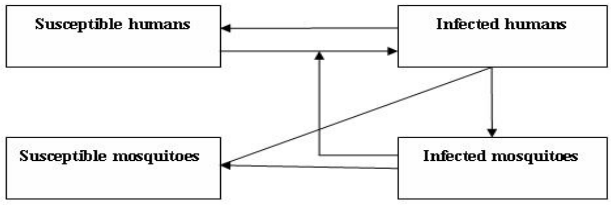

Fig. 1 Flowchart of disease transmission between individuals and mosquitoes. All individuals as well as mosquitoes run stochastically through the susceptible-infected-susceptible cycles.

Let $S_k(t)$, $I_k(t)$ denote the densities of susceptible and infected nodes with degree k at time t, respectively. And let $V(t)$ be the density of the infective medium at time t. Then

$$I_k(t)+S_k(t)=1,$$

and the mean-field equations for infected nodes with degree k can be written as

$$\begin{cases}\dot{I}_k(t)=-I_k(t)+\lambda k[1-I_k(t)]\Theta(\rho(t))+\gamma_1[1-I_k(t)]\upsilon(t),\\ \dot{\upsilon}(t)=-\upsilon(t)+\gamma_2[1-\upsilon(t)]\Theta(\rho(t)).\end{cases} \quad (2)$$

Here, λ is the infection rate, $\rho(t)=\sum p(k)I_k(t)$ is the density of infected individuals on the network, λ_1 and λ_2 are constants and according to [12], $\Theta(t)$ can be written as

$$\Theta(t)=\sum\frac{kp(k)I_k(t)}{\sum kp(k)}=\frac{1}{\langle k\rangle}\langle kI_k(t)\rangle \quad (3)$$

Where $p(k)$ is the connectivity distribution, $\langle k\rangle$ is the average degree of the network.

By imposing the stationarity condition

$$\begin{cases}\dot{I}_k(t)=0,\\ \dot{\upsilon}(t)=0,\end{cases} \quad (4)$$

the first equation of system (2) yields

$$I_k(t)=\frac{\lambda k\Theta(t)+\gamma_1\upsilon(t)}{1+\lambda k\Theta(t)+\gamma_1\upsilon(t)} \quad (5)$$

The second equation of system (2) yields

$$\upsilon(t)=\frac{\gamma_2\rho(t)}{1+\gamma_2\rho(t)} \quad (6)$$

Substituting (6) into (5), one obtains

$$I_k(t)=\frac{\lambda k\Theta(t)+\lambda k\gamma_2\rho(t)\Theta(t)+\gamma_1\gamma_2\rho(t)}{1+\gamma_2\rho(t)+\lambda k\Theta(t)+\lambda k\gamma_2\rho(t)\Theta(t)+\gamma_1\gamma_2\rho(t)} \quad (7)$$

Let $\Gamma_1 = \lambda k\Theta(t) + \lambda k\gamma_2\rho(t)\Theta(t) + \gamma_1\gamma_2\rho(t)$,

$\Gamma_2 = 1+\gamma_2\rho(t) + \lambda k\Theta(t) + \lambda k\gamma_2\rho(t)\Theta(t) + \gamma_1\gamma_2\rho(t)$.

One has

$$I_k(t) = \frac{\Gamma_1(\rho,\Theta)}{\Gamma_2(\rho,\Theta)}.$$

Then, one obtains a self-consistency equation as follows:

$$\Theta(t) = \frac{1}{\langle k\rangle}\left\langle k\frac{\Gamma_1(\rho,\Theta)}{\Gamma_2(\rho,\Theta)}\right\rangle \equiv \Gamma(\Theta(t)) \quad (8)$$

Obviously, $\Theta(t) = 0$ is a trivial solution of (8). If there is another solution $\Theta(t) > 0$, we must have

$$\left.\frac{d\Gamma}{d\Theta}\right|_{\Theta=0} > 1.$$

Then, we compute that

$$\frac{d\Gamma}{d\Theta} = \frac{1}{\langle k\rangle}\left\langle k\frac{(\frac{\partial\Gamma_1}{\partial\rho}\frac{d\rho}{d\Theta}+\frac{\partial\Gamma_1}{\partial\Theta})\Gamma_2 - (\frac{\partial\Gamma_2}{\partial\rho}\frac{d\rho}{d\Theta}+\frac{\partial\Gamma_2}{\partial\Theta})\Gamma_1}{\Gamma_2^2}\right\rangle \quad (9)$$

That is

$$\left.\frac{d\Gamma}{d\Theta}\right|_{\Theta=0} = \frac{\lambda\gamma_1\gamma_2\langle k\rangle^2}{(\lambda\langle k^2\rangle - \langle k\rangle)(\gamma_1\gamma_2 - 1)} > 1 \quad (10)$$

Let λ_c be the minimum value of λ satisfying the above inequality. Then,

$$\lambda_c = \frac{(1-\gamma_1\gamma_2)\langle k\rangle}{\gamma_1\gamma_2(\langle k\rangle^2 - \langle k^2\rangle) + \langle k^2\rangle}.$$

In [12], we derived an epidemic threshold for the standard SIS model as $\lambda_c' = \frac{\langle k\rangle}{\langle k^2\rangle}$. It is easy to see that $\lambda_c < \lambda'$ that is, when an infective medium is added to the standard SIS model, the infected individuals are more likely to reach some stationary density. In [11], similarly, one can obtain

$$\lambda_c' = \frac{(1-\gamma_1\gamma_2)\langle k\rangle}{\langle k^2\rangle}.$$

According to the Jessen inequality, $\langle k^2\rangle > \langle k\rangle^2$ is always valid, then it is easy to check that $\lambda_c > \lambda_c''$, this means that the epidemic propagation on the modified model is much harder to outbreak than that for the model discussed in [12](see Fig.2).

III. The Effect of Vaccination Strategies

Vaccination is very powerful in controlling the disease. In this section, we will discuss the impact of various immunization schemes to give some effective strategies to control the disease on various complex networks.

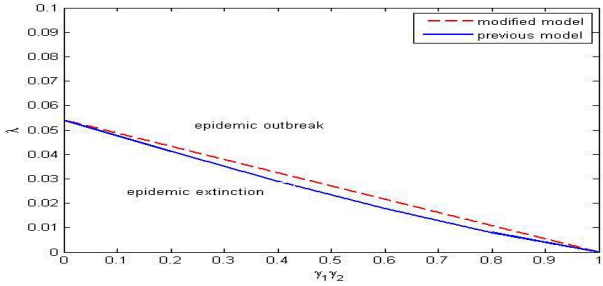

Fig. 2 This simulation is performed on BA networks with size $N = 105$ and the average degree of the network $\langle k\rangle = 6$.

A. Uniform immunization on complex networks

Uniform immunization strategy is the simplest immunization scheme [14], so we use it as a typical methodology to compare its different effects in the modified model here and in the model of [11]. Consider the following model:

$$\begin{cases} \dot{I}_k(t) = -I_k(t) + \lambda(1-g)k[1-I_k(t)]\Theta(t) + \gamma_1[1-I_k(t)]\upsilon(t) \\ \dot{\upsilon}(t) = -\upsilon(t) + \gamma_2[1-\upsilon(t)]\rho(t) \end{cases} \quad (11)$$

Here, g is the density of immune nodes on the network. Similarly to Section 2.1, one can obtain

$$\lambda_{c1} = \frac{[1-\gamma_1\gamma_2(1-g)]\langle k\rangle}{(1-g)^2\gamma_1\gamma_2(\langle k\rangle^2 - \langle k^2\rangle) + (1-g)\langle k^2\rangle}$$

In [11], the following uniform immunization system is studied:

$$\begin{cases} \dot{I}_k(t) = -I_k(t) + \lambda(1-g)k[1-I_k(t)]\Theta(\rho(t)) + \gamma_1(1-g)[1-I_k(t)]\upsilon(t) \\ \dot{\upsilon}(t) = -\upsilon(t) + \gamma_2[1-\upsilon(t)]\Theta(\rho(t)) \end{cases}$$

For the above system, similarly to Section 2.1, one has

$$\lambda_{c2} = \frac{[1-\gamma_1\gamma_2(1-g)]\langle k\rangle}{(1-g)\langle k^2\rangle}$$

Then, it is easy to obtain $\lambda_{c1} > \lambda_{c2}$, therefore clearly, the uniform immunization used in the modified model is more efficient than that used in the model presented in [11] (see Fig. 3).

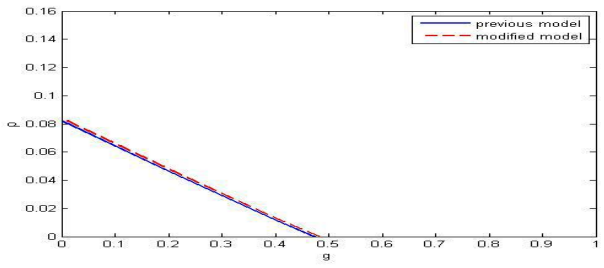

Fig. 3 The change between the density of infected individuals ρ and the density of immune nodes on the network g, and this simulation is performed on BA networks with size $N = 105$, the average degree of the network $\langle k \rangle = 6, \lambda = 0.1, \lambda_1 = 0.4$ and $\lambda_2 = 0.5$.

B. Targeted immunization on complex networks

While uniform immunization schemes are effective in networks with well-defined immunization thresholds, it is still important to consider the effect of targeted immunization on the models. The strategy here is to immune the most highly connected nodes, i.e. the ones that more likely spread the disease. For the network, we introduce an upper threshold k_t, and all nodes with connectivity $k > k_t$ are immune. Calculating a complex network with the continuous k-approxim--ation shows that the density of immunized nodes is related to the connectivity threshold, as

$$g = 1 - \int_m^{k_t} P(k)dk = m^2 k_t^{-2}$$

In an SF network, the contact between individuals relates to individuals' connectivity k, but a mosquito will bite a person without selectivity. That is, the infections among individuals are associated with the connectivity distribution, yet the transmission between persons and mosquitoes is only determined by the infectivities λ_1 and λ_2. Thus, the dynamic mean-field reaction rate equations can be written as

$$\begin{cases} \dot{\rho}_k(t) = -\rho_k(t) + \lambda k[1-\rho_k(t)]\Theta(\rho(t)) + \gamma_1[1-\rho_k(t)]\upsilon(t) \\ \dot{\upsilon}(t) = -\upsilon(t) + \gamma_2[1-\upsilon(t)]\Theta(\rho(t)) \end{cases} \quad (12)$$

where the probability $0 \le \Theta(\rho(t)) \le 1$ describes a link pointing to an infected individual, which satisfies the relation

$$\Theta(\rho(t)) = \sum_k \frac{kP(k)\rho_k}{\sum_s sP(s)} \quad (13)$$

and $\rho(t) = \sum_k P(k)\rho_k$ is the density of infected individuals in the whole network, $P(k)$ is the connectivity distribution.

In this strategy, g_k is defined as the fraction of immune individuals with a given connectivity k. Suppose the condition for the proportional immunization is

$$\lambda k(1-g) = \lambda_1 = constant$$

Then, system (11) yields

$$\begin{cases} \dot{\rho}_k(t) = -\rho_k(t) + \lambda_1[1-\rho_k(t)]\Theta(\rho(t)) + \dfrac{\lambda_1}{\lambda_k}[1-\rho_k(t)]\upsilon(t) \\ \dot{\upsilon}(t) = -\upsilon(t) + \gamma_2[1-\upsilon(t)]\Theta(\rho(t)) \end{cases} \quad (14)$$

Then, system (14) yields

$$\begin{cases} \dot{\rho}_k(t) = -\rho_k(t) + \lambda_1\left(1-m^2k_t^{-2}\right)k[1-\rho_k(t)]\Theta(\rho(t)) + \gamma_1\left(1-m^2k_t^{-2}\right)[1-\rho(t)]\upsilon(t) \\ \dot{\upsilon}(t) = -\upsilon(t) + \gamma_2[1-\upsilon(t)]\Theta(\rho(t)). \end{cases} \quad (15)$$

In the steady (epidemic) state, ρ is just a function of λ, λ_1 and λ_2, thus the probability Θ becomes an implicit function of the spreading rates, λ_1 and λ_2. By imposing the stationarity condition

$$\begin{cases} \dot{\rho}_k(t) = 0, \\ \dot{\upsilon}(t) = 0, \end{cases}$$

one has

$$\begin{cases} -\rho_k + \lambda\left[1-m^2k_t^{-2}\right]k[1-\rho_k]\Theta + \gamma_1\left[1-m^2k_t^{-2}\right][1-\rho_k]\upsilon = 0 \\ -\upsilon + \gamma_2[1-\upsilon]\Theta = 0 \end{cases} \quad (16)$$

The second equation of (16) yields

$$\upsilon = \frac{\gamma_2\Theta}{1+\gamma_2\Theta},$$

and substituting it into the first equation of system (16) gives

$$\rho_k = \frac{\lambda k\left(1-m^2k_t^{-2}\right)\Theta + \gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right) + \lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta^2}{1+\gamma_2\Theta + \lambda k\left(1-m^2k_t^{-2}\right)\Theta + \gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right) + \lambda k\gamma_2\Theta^2}$$

By relation (13) and $\langle k \rangle = \sum_s sP(s)$, we have

$$\Theta = \sum_k \frac{kP(k)\rho_k}{\sum_s sP(s)}$$

$$= \frac{1}{\langle k \rangle}\sum_k kP(k)\frac{\lambda k\left(1-m^2k_t^{-2}\right)\Theta + \gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right) + \lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta^2}{1+\gamma_2\Theta + \lambda k\left(1-m^2k_t^{-2}\right)\Theta + \gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right) + \lambda k\gamma_2\Theta^2} \quad (17)$$

Obviously, $\Theta = 0$ is a solution of (17). If there is another solution $0 < \Theta \le 1$, it must satisfy

$$\frac{d}{d\Theta}\left(\frac{1}{\langle k\rangle}\sum_k kP(k)\frac{\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta^2}{1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2}\right)\Bigg|_{\Theta=0}$$

$\geq 1,$

that is,

$$\frac{1}{\langle k\rangle}\sum_k kP(k)\left\{\frac{\left[\lambda k\left(1-m^2k_t^{-2}\right)+\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)+2\lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta\right]}{\left[1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2\right]^2}\right.$$

$$\left.-\frac{\left[\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta^2\right]\left[\gamma_2+\lambda k\left(1-m^2k_t^{-2}\right)+\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)+2\lambda k\gamma_2\right]}{\left[1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2\right]^2}\right\}\Bigg|_{\Theta=0}$$

$\geq 1,$

Let λ_c be the minimum value of λ satisfying the above inequality. Then

$$\frac{\lambda_c\sum_k k^2\left(1-m^2k_t^{-2}\right)P(k)+\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)\sum_k kP(k)}{\langle k\rangle}=1,$$

that is,

$$\lambda_c\left(1-m^2k_t^{-2}\right)P(k)\frac{\langle k\rangle}{k}+\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)=1.$$

Hence,

$$\lambda_c=\frac{(1-\gamma_1\gamma_2)\langle k\rangle}{\left(1-m^2k_t^{-2}\right)\langle k^2\rangle} \quad (18)$$

Where $\langle k^2\rangle=\sum_k k^2P(k).$

Now, consider a BA model. By using a continuous k approximation that allows a practical substitution of series with integrals [13],[23], the full connectivity distribution can be obtained as $P(k)=2m^2/k^3$, where m is the minimum number of connections at each individual node, and $\langle k\rangle=\int_m^\infty kP(k)dk=2m.$ Clearly $\langle k^2\rangle\approx 2m^2\ln(K_c/m),\ K_c\to\infty.$ Submitting them into (18) yields

$$\lambda_c\approx\frac{1-\gamma_1\gamma_2}{m\left(1-m^2k_t^{-2}\right)\ln(K_c/m)}.$$

In order to consider the dependence of network size N on λ_c, we have to relate the maximum connectivity K_c to N. The relation is given by $K_c\approx mN^{\frac{1}{2}}$ [20], so

$$\lambda_c=\frac{\langle k\rangle(1-\gamma_1\gamma_2)}{\left(1-m^2k_t^{-2}\right)\langle k^2\rangle}\approx\frac{2(1-\gamma_1\gamma_2)}{m\left(1-m^2k_t^{-2}\right)\ln N}.$$

Next, when $\lambda>\lambda_c$, the stationary state ρ can be obtained as follows: Integrating (17) gives

$$\frac{1}{m}=\int_m^\infty\frac{\lambda k\left(1-m^2k_t^{-2}\right)+\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta}{k^2\left[1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2\right]}dk$$

$$=\int_m^\infty\left(\frac{A}{k}+\frac{B}{k^2}+\frac{C}{1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2}\right)dk$$

$$=A\left[\ln\frac{1}{\lambda\left(1-m^2k_t^{-2}\right)\Theta+\gamma_2\Theta^2}-\ln\frac{m}{1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda m\left(1-m^2k_t^{-2}\right)\Theta+\lambda m\gamma_2\Theta^2}\right]+\frac{B}{m}$$

$$=A\ln\frac{1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda m\left(1-m^2k_t^{-2}\right)\Theta+\lambda m\gamma_2\Theta^2}{\lambda m\Theta\left[\left(1-m^2k_t^{-2}\right)+\gamma_2\Theta\right]}+\frac{B}{m} \quad (19)$$

Where

$$A=\frac{\lambda\left(1-m^2k_t^{-2}\right)\left(1+\gamma_2\Theta\right)^2}{\left[1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)\right]^2}$$

$$B=\frac{\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)}{1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)}$$

$$C=-\frac{\lambda^2\left(1-m^2k_t^{-2}\right)\Theta\left(1+\gamma_2\Theta\right)^3}{\left[1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)\right]^2}$$

Hence, Θ is a solution to the following algebraic equation

$$\ln\left\{1+\frac{1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)}{\lambda m\Theta\left[\left(1-m^2k_t^{-2}\right)+\gamma_2\Theta\right]}\right\}$$

$$=\frac{\left[1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)-\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)\right]\left[1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)\right]}{\lambda m\left(1-m^2k_t^{-2}\right)\left(1+\gamma_2\Theta\right)^2} \quad (20)$$

Obviously, this equation has one and only one solution, because the rhs of (20) is smaller than the lhs when $\Theta=0$ and the rhs of (20) is larger than the lhs when $\Theta=1$. At the same time, the rhs of (20) is a monotonously increasing function of Θ and the lhs of (20) is a monotonously decreasing function of Θ. In order to obtain an explicit expression of ρ, the following lemma is useful.

Lemma.If

$$=2m^2\left(1-m^2k_t^{-2}\right)\int_m^\infty\frac{\lambda k+\lambda k\gamma_2\Theta+\gamma_1\gamma_2}{k^3\left[1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2\right]}dk$$

then $\int_m^\infty\frac{ax+b}{(cx+d)x^3}dx=\frac{a-c}{md}+\frac{b}{2m^2d}.$

Its proof is given in [24].

Furthermore, we have

$$\rho=\sum_k P(k)\rho_k=\sum_k \frac{2m^2\left[\lambda k\left(1-m^2k_t^{-2}\right)+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\left(1-m^2k_t^{-2}\right)\Theta^2\right]}{k^3\left[1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2\right]}$$

$$=2m^2\left(1-m^2k_t^{-2}\right)\int_m^\infty \frac{\lambda k+\lambda k\gamma_2\Theta+\gamma_1\gamma_2}{k^3\left[1+\gamma_2\Theta+\lambda k\left(1-m^2k_t^{-2}\right)\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)+\lambda k\gamma_2\Theta^2\right]}dk \quad (21)$$

By (19), applying Lemma 1 to (21) yields

$$\rho=\Theta\frac{\left[2m\lambda\left(1-m^2k_t^{-2}\right)(1-\Theta)(1+\gamma_2\Theta)+\gamma_1\gamma_2\left(1-m^2k_t^{-2}\right)\right]}{1+\gamma_2\Theta+\gamma_1\gamma_2\Theta\left(1-m^2k_t^{-2}\right)}$$

where Θ is the solution of (20).

As shown in Fig.4, the modified SIS model on SF networks shows that the epidemic threshold λ_c almost disappears for any fixed γ_1 and γ_2. There is always a stationary state for any $\gamma_1>0$, $\gamma_2>0$ or $\gamma>0$. This characteristic is consistent with the standard SIS model for SF networks. At the same time, one can easily find their difference-modified model relies on the infectivity between different γ_1 and γ_2.

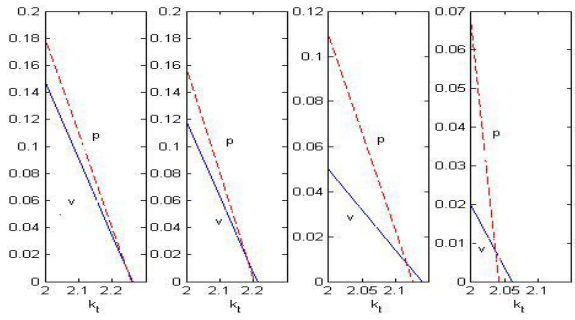

Fig. 4 (Clour online) Densities of infected individuals ρ (red, dash) and v (blue, solid) as functions of the degree k_t of immune nodes in a scale-free network. $m=2$, with infected rates $\lambda_1=\lambda_2=\lambda=1,0.8,0.5,0.3$.

Remark. We can easily find that with the increasing of the infectivities γ_1 and γ_2, the number of infected individuals and that of infected mosquitoes both increase. But the number of infected mosquitoes in SF models will not go to the 0 equilibrium as in the WS models. From a biological point of view, this result means that for any infectivities λ, γ_1 and γ_2, the disease will reach some stationary density either in humans or in vectors, which is consistent with the natural phenomena. On the other hand, it also implies that the real world has a prominent SF network feature.

IV. Conclusions

In this paper, we analyze a modified susceptible-infected-susceptible (SIS) model with an infective vector. In our modified model, infective vector is also incorporated. Through Analysis in this paper, the vector plays an important role in epidemic transmission. The standard SIS model represents a more realistic situation with the vector added into. And the infected individuals are more likely to reach some stationary density. It easily shows that, the uniform immunization strategy in the modified model is more efficient than that used in the previously proposed model. Without doubt, the modified SIS model researched in this paper has some limitations. These and some other related problems will be further studied in the near future.

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