Dynamic analysis of a sexually transmitted disease model on complex networks*

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(Received 3 August 2012; revised manuscript received 26 September 2012)

In this paper, a sexually transmitted disease model is proposed on complex networks, where contacts between humans are treated as a scale-free social network. There are three groups in our model, which are dangerous male, non-dangerous male, and female. By mathematical analysis, we obtain the basic reproduction number for the existence of endemic equilibrium and study the effects of various immunization schemes about different groups. Furthermore, numerical simulations are undertaken to verify more conclusions.

Keywords: sexually transmitted diseases, basic reproduction number, complex networks, immunization

PACS: 02.50.−r, 05.90.+m **DOI:** 10.1088/1674-1056/22/3/030207

1. Introduction

Recently, the importance of mathematic models on modeling and controlling epidemic diseases has been given a lot of attention. For devising effective strategies to minimize the destruction caused by infectious diseases, many mathematical models[1–3] have been built to both understand the observed mechanisms of infectious diseases and predict the consequences of the introduction of public health interventions to control the spread of diseases. The susceptible–infected–susceptible (SIS) and the susceptible–infected–recovered/removed (SIR) model are always convenient mathematical models to describe the fundamental mechanism of diseases.[4] For the SIS model, each individual can exist in two states: susceptible and infected. At each stage, the susceptible individual who contacts an infected neighbor will be infected with rate λ. Meanwhile, the infected individuals may recover and become susceptible individuals with a rate γ. For the SIR model, once an infected individual recovers, then the individual will not be infected again.

Traditional models are based on the hypothesis that all individuals undergo homogeneous random mixing, namely, all individuals are equally likely to contact each other, and therefore, if infected, are equally likely to infect susceptible members of the population, which is rarely observed in reality. Much effort has been made to explore more realistic mathematical models for the transmission dynamics of infectious diseases.[5–7] In 1999, Barabási and Albert introduced a new (BA) model of complex networks: scale-free networks.[8] In a scale-free network, the probability of a node which has k linking to other nodes is distributed according to the power law $p(k) = Cf(k)k^{-r}$ $(r \in (2,3])$, where $f(k)$ is the function of k. Many real complex systems have been shown to be scale-free networks, such as the World Wide Web (WWW). Moreover, many epidemic diseases occur in communities which also exhibit characteristics consistent with a scale-free network. For instance, human contact shows scale-free characteristics.[9] Then, some epidemic models on scale-free networks come to birth,[10–13] which are more realistic.

A sexually transmitted disease model on complex networks has been studied by Liu,[13] who believed that the infected rate only related to the number of contacts of an individual, which means that an individual who has many neighborhoods is more likely to be infected. However, in many cases, the infected rate is affected by many other factors. In particular, for sexually transmitted diseases, an individual may have many sexual partners, but may contact his neighbor frequently.[14,15] Furthermore, an individual can contact his sexual partners with or without protection. In this paper, we propose a sexually transmitted disease model with three groups, which are dangerous male, non-dangerous male, and female, in order to describe the spread of sexual disease more realistically and find effective methods for disease control. A dangerous male refers to a male who has many sexual contacts or does not use protection when they have sexual contacts. A non-dangerous male refers to a male who has few sexual contacts or uses protection when he has a sexual contact.

In Section 2, we describe the epidemic model on a complex network and discuss the existence of equilibria and basic reproduction numbers. Section 3 is devoted to giving numerical simulations. In Section 4, we discuss and compare the immunization strategies. In Section 5, we discuss the results.

*Project supported by the National Natural Science Foundation of China (Grant No. 10901145), the Natural Science Foundation of Shanxi Province, China (Grant Nos. 2009011005-1 and 2012011002-1), and the Top Young Academic Leaders of Higher Learning Institutions of Shanxi Province, China.

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2. Model

2.1. Model description

Our model will be proposed in this section. Firstly, the nodes on our network are divided into three types which are dangerous male, not dangerous male and female. For each type of node, they are divided into susceptible individuals and infected individuals. Then we can draw the transfer diagram, which is shown in Fig. 1.

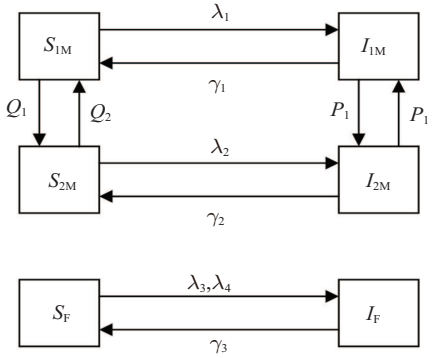


Fig. 1. State-transition rules of the contact process.

The S_{1M} represents susceptible individuals of dangerous males, while S_{2M} represents susceptible individuals of non-dangerous males, and susceptible individuals of females are denoted by S_F. Similarly, I_{1M}, I_{2M}, and I_F represent infected individuals of dangerous males, infected individuals of non-dangerous males, and infected individuals of females, respectively.

The meaning for each parameter is given below. At each time step, a susceptible dangerous male individual is infected with probability λ_1 and a susceptible non-dangerous male individual is infected with probability λ_2 if it is connected to an infected female individual. The probability of a susceptible female individual being infected is λ_3 if she contacts an infected dangerous male, and the probability is λ_4 if she contacts an infected non-dangerous male. The probability of infected individuals becoming susceptible individuals is γ_1 for dangerous males, γ_2 for non-dangerous males, and γ_3 for female individuals. The probability of an infected dangerous male becoming an infected non-dangerous male is P_1, and the probability of an infected non-dangerous male becoming an infected dangerous male is P_2. For this susceptible situation, the rate from S_{1M} to S_{2M} is Q_1, and the rate from S_{2M} to S_{1M} is Q_2. The network we study in this paper is a scale-free network, and we obtain a model with the transfer diagram from Fig. 1 as follows:

$$\begin{aligned}
\frac{\mathrm{d}S_{1M,k}}{\mathrm{d}t} &= \gamma_1 I_{1M,k} - \lambda_1 k S_{1M,k}\Theta_3 - Q_1 S_{1M,k} + Q_2 S_{2M,k},\\
\frac{\mathrm{d}S_{2M,k}}{\mathrm{d}t} &= \gamma_2 I_{2M,k} - \lambda_2 k S_{2M,k}\Theta_3 + Q_1 S_{1M,k} - Q_2 S_{2M,k},\\
\frac{\mathrm{d}S_{F,k}}{\mathrm{d}t} &= \gamma_3 I_{F,k} - \lambda_3 k S_{F,k}\Theta_1 - \lambda_4 k S_{F,k}\Theta_2,\\
\frac{\mathrm{d}I_{1M,k}}{\mathrm{d}t} &= \lambda_1 k S_{1M,k}\Theta_3 - \gamma_1 I_{1M,k} - P_1 I_{1M,k} + P_2 I_{2M,k},\\
\frac{\mathrm{d}I_{2M,k}}{\mathrm{d}t} &= \lambda_2 k S_{2M,k}\Theta_3 - \gamma_2 I_{2M,k} + P_1 I_{1M,k} - P_2 I_{2M,k},\\
\frac{\mathrm{d}I_{F,k}}{\mathrm{d}t} &= \lambda_3 k S_{F,k}\Theta_1 + \lambda_4 k S_{F,k}\Theta_2 - \gamma_3 I_{F,k},
\end{aligned} \tag{1}$$

where $S_{1M,k}$ denotes the density of susceptible dangerous male individuals for degree k in the network. The situation is similar for $S_{2M,k}$, $S_{F,k}$, $I_{1M,k}$, $I_{2M,k}$, and $I_{F,k}$. Furthermore, in order to facilitate the study of disease spread, it is assumed that the total densities of nodes which are male and female are fixed, which implies that

$$\begin{aligned}
S_{1M,k} + S_{2M,k} + I_{1M,k} + I_{2M,k} &= \Lambda,\\
S_{F,k} + I_{F,k} &= 1 - \Lambda,
\end{aligned} \tag{2}$$

where Λ is a constant. The probability $0 \le \Theta_1, \Theta_2, \Theta_3 \le 1$ describes a link pointing to an infected dangerous male, an infected non-dangerous male, and an infected female. According to Refs. [5] and [6], Θ_1, Θ_2, and Θ_3 satisfy

$$\begin{aligned}
\Theta_1 &= \frac{1}{\langle k\rangle}\sum_k k I_{1M,k} p(k),\\
\Theta_2 &= \frac{1}{\langle k\rangle}\sum_k k I_{2M,k} p(k),\\
\Theta_3 &= \frac{1}{\langle k\rangle}\sum_k k I_{F,k} p(k),
\end{aligned} \tag{3}$$

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

2.2. Equilibria and basic reproduction number

Firstly, we can obtain the disease-free equilibrium of model (1) with the left side of model (1) as zero.

$$E^0 = \left(\frac{\Lambda Q_2}{Q_1+Q_2}, \frac{\Lambda Q_1}{Q_1+Q_2}, 1-\Lambda, 0, 0, 0\right). \tag{4}$$

Note that only the compartments $I_{1M,k}$, $I_{2M,k}$, and $I_{F,k}$ are involved in the calculation of the so-called basic reproduction number R_0, which is defined by Diekmann, Heesterbeek, and Metz.[16] According to the concepts of next generation matrix and solving methods for R_0 in Refs. [17] and [18], we can compute the basic reproduction number for model (1)

$$R_0 = \frac{\langle k^2\rangle}{\langle k\rangle}\sqrt{\frac{(1-\Lambda)\Lambda\kappa}{(Q_1+Q_2)(\gamma_1\gamma_2+P_1\gamma_2+P_2\gamma_1)\gamma_3}}, \tag{5}$$

where ρ represents the spectral radius of a matrix. Next we discuss the existence of the endemic equilibrium. The endemic equilibrium must satisfy the equilibrium status of Eqs. (1) and (2), then we can derive

$$\begin{aligned}
S_{1M,k} &= \frac{\Lambda}{\mathrm{H}}\left(k\lambda_2\gamma_1 P_2\Theta_3 + \gamma_1 P_2 Q_2 + \gamma_1\gamma_2 Q_2 + \gamma_2 P_1 Q_2\right),\\
S_{2M,k} &= \frac{\Lambda}{\mathrm{H}}\left(k\lambda_1\gamma_2 P_1\Theta_3 + \gamma_2 P_1 Q_1 + \gamma_1\gamma_2 Q_1 + \gamma_1 P_2 Q_1\right),\\
S_{F,k} &= (1-\Lambda)\frac{\gamma_3}{k(\lambda_3\Theta_1+\lambda_4\Theta_2)+\gamma_3},
\end{aligned}$$

$$I_{1\mathrm{M},k} = \frac{\Lambda}{\mathrm{H}} k(k\lambda_1\lambda_2 P_2\Theta_3 + \lambda_1 Q_2\gamma_2 + \lambda_1 P_2 Q_2 + \lambda_2 Q_1 P_2)\Theta_3, \quad (6)$$

$$I_{2\mathrm{M},k} = \frac{\Lambda}{\mathrm{H}} k(k\lambda_1\lambda_2 P_1\Theta_3 + \lambda_2 Q_1\gamma_1 + \lambda_2 P_1 Q_1 S_{\mathrm{F},k} + \lambda_1 Q_2 P_1)\Theta_3, \quad (7)$$

$$I_{\mathrm{F},k} = (1-\Lambda)\frac{k(\lambda_3\Theta_1 + \lambda_4\Theta_2)}{k(\lambda_3\Theta_1 + \lambda_4\Theta_2) + \gamma_3}, \quad (8)$$

where

$$\mathrm{H} = [(\gamma_2 P_1 + \gamma_2 Q_2 + P_2 Q_2 + P_1 Q_2)\lambda_1 + (P_2 Q_1 + P_2\gamma_1 + P_1 Q_1 + \gamma_1 Q_1)\lambda_2]k\Theta_3 + (Q_1 + Q_2)(\gamma_1\gamma_2 + \gamma_1 P_2 + \gamma_2 P_1) + k^2\lambda_1\lambda_2(P_1 + P_2)\Theta_3^2.$$

Based on the expressions (3) and (8), one can obtain

$$g(\Theta_3) = \frac{(1-\Lambda)}{\langle k\rangle}\sum_k kp(k)\frac{k(\lambda_3\Theta_1 + \lambda_4\Theta_2)}{k(\lambda_3\Theta_1 + \lambda_4\Theta_2) + \gamma_3} = \frac{(1-\Lambda)}{\langle k\rangle}\sum_k kp(k)\left(1 - \frac{\gamma_3}{k(\lambda_3\Theta_1 + \lambda_4\Theta_2) + \gamma_3}\right). \quad (9)$$

It is obvious that $\Theta_3 = 0$ is a solution of the self-consistency equation. Θ_i $(i = 1,2,3)$ are all equal to zero or non-zero. Next,

$$g(1) < \frac{(1-\Lambda)}{\langle k\rangle}\sum_k kp(k) < 1.$$

The condition we are interested in is that a nontrivial solution of the self-consistency equation exists. To allow a nontrivial solution Θ $(\Theta \in (0,1])$, the inequality must satisfy

$$\left.\frac{\mathrm{d}g(\Theta_3)}{\mathrm{d}\Theta_3}\right|_{\Theta_3=0} = R_0^2 > 1.$$

At this point, we can have the following theorem: if and only if $R_0 > 1$, there exists an endemic equilibrium.

3. Numerical simulations and sensitivity analysis

In this section, we perform numerical simulations with the model (1) to verify our result and some sensitivity analysis in terms of the model parameters. Our simulations are based on the BA network.

Firstly, we follow the algorithm developed by Barabasi and Albert[8] to generate a network. Suppose the initial network has 2 nodes. In every step we add 2 nodes into the network. The final network size is $N = 1100$. The degree distribution $p(k)$ is given in Fig. 2, where $p'(k) = (\eta-1)\rho^{\eta-1}k^{-\eta}$ for $\rho = 2$ and $\eta = 3$. From Fig. 2, it can be seen that $p(k) \sim p'(k)$, which can be calculated to be $\langle k\rangle = 3.9746$, and $\langle k^2\rangle = 46.1801$.

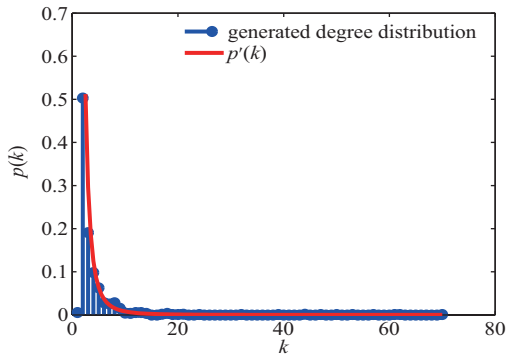


Fig. 2. (color online) The degree distribution of network N.

In Fig. 3, the simulations about the solutions are done. The parameters are taken as $\lambda_1 = 0.17$, $\lambda_2 = 0.02$, $\lambda_3 = 0.12$, $\lambda_4 = 0.06$, $\gamma_1 = 0.11$, $\gamma_2 = 0.12$, $\gamma_3 = 0.13$, $\Lambda = 4/5$, $Q_1 = 0.1$, $Q_2 = 0.1$, $P_1 = 0.1$, $P_2 = 0.1$, and the initial values are

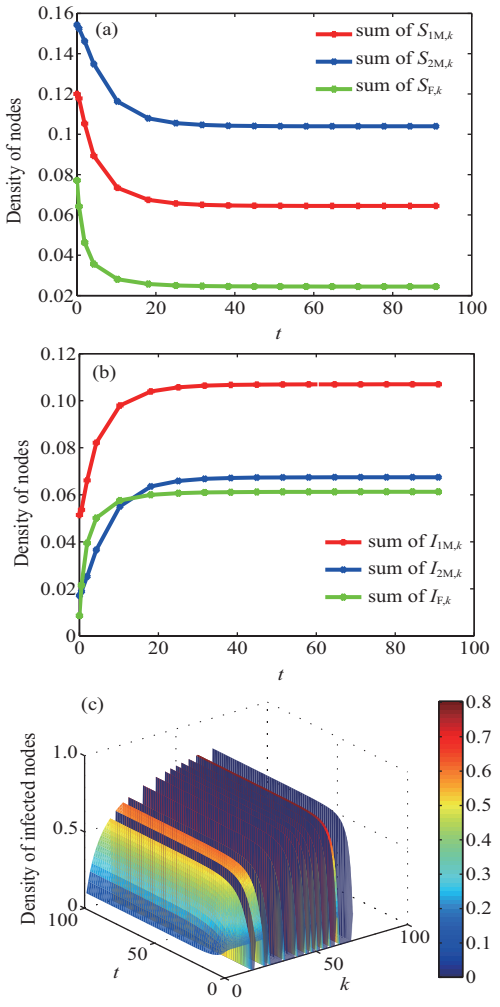


Fig. 3. (color online) Solutions from a numerical simulation when $R_0 = 3.7107$. (a) Simulation solutions for the sum of $S_{1\mathrm{M},k}$, the sum of $S_{2\mathrm{M},k}$, and the sum of $S_{\mathrm{F},k}$ with the time series; (b) simulation solutions about the sum of $I_{1\mathrm{M},k}$, the sum of $I_{2\mathrm{M},k}$, the sum of $I_{\mathrm{F},k}$ with the time series; (c) the density of the infected nodes is regarded as a function of k and time.

$S_{1\mathrm{M},k}(0) = 0.28$, $S_{1\mathrm{M},k}(0) = 0.36$, $S_{\mathrm{F},k}(0) = 0.18$, $I_{1\mathrm{M},k}(0) = 0.12$, $I_{1\mathrm{M},k}(0) = 0.04$, and $I_{\mathrm{F},k}(0) = 0.02$. Then, the reproduc-

tion number is $R_0 = 3.7107$, and we can see that there is an endemic equilibrium when $R_0 > 1$.

In Fig. 4, the parameters and initial values are taken as $\lambda_1 = 0.025$, $\lambda_2 = 0.008$, $\lambda_3 = 0.035$, $\lambda_4 = 0.01$, $\gamma_1 = 0.11$, $\gamma_2 = 0.12$, $\gamma_3 = 0.13$, $\Lambda = 4/5$, $Q_1 = 0.1$, $Q_2 = 0.1$, $P_1 = 0.1$, $P_2 = 0.1$, $S_{1M,k}(0) = 0.12$, $S_{2M,k}(0) = 0.16$, $S_{F,k}(0) = 0.08$, $I_{1M,k}(0) = 0.28$, $I_{2M,k}(0) = 0.24$, and $I_{F,k}(0) = 0.12$. Here $R_0 = 0.6020$ is the reproduction number, and we can see that the disease dies out.

The density of the infected nodes is regarded as a function of time t and parameters λ_1, λ_2, λ_3, and λ_4, which is simulated in Fig. 5, where λ_1, λ_2, λ_3, and λ_4 vary from 0 to 0.2, and the other parameters and initial values are the same as Fig. 3. In Fig. 6, the relationship between the basic reproduction number R_0 and parameters γ_1, γ_2, and γ_3 is shown. Here, γ_1, γ_2, and γ_3 change from 0 to 0.4, and their critical values are calculated to be $\gamma_1^C = 0.187$, $\gamma_2^C = 0.182$, and $\gamma_3^C = 0.139$ for $R_0 = 1$.

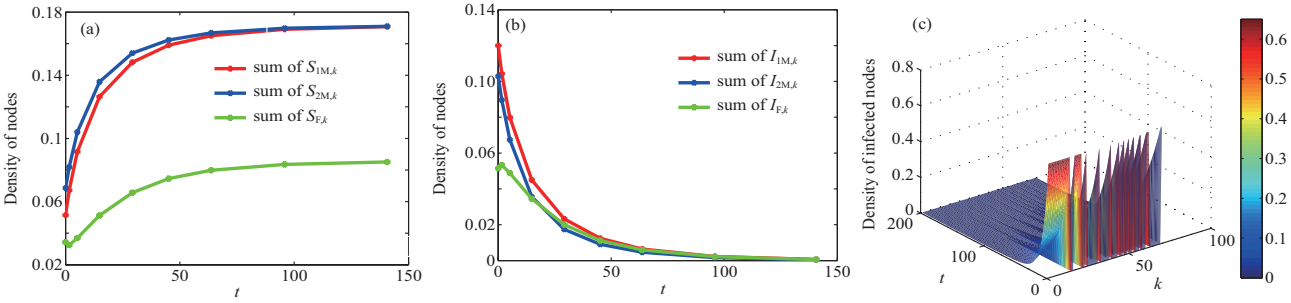


Fig. 4. (color online) Solutions from numerical simulations when $R_0 = 0.6020$. (a) Simulation solutions for the sum of $S_{1M,k}$, the sum of $S_{2M,k}$, and the sum of $S_{F,k}$ with time series; (b) the simulation solutions for the sum of $I_{1M,k}$, the sum of $I_{2M,k}$, and the sum of $I_{F,k}$ with time series; (c) the density of the infected nodes as a function of k and time.

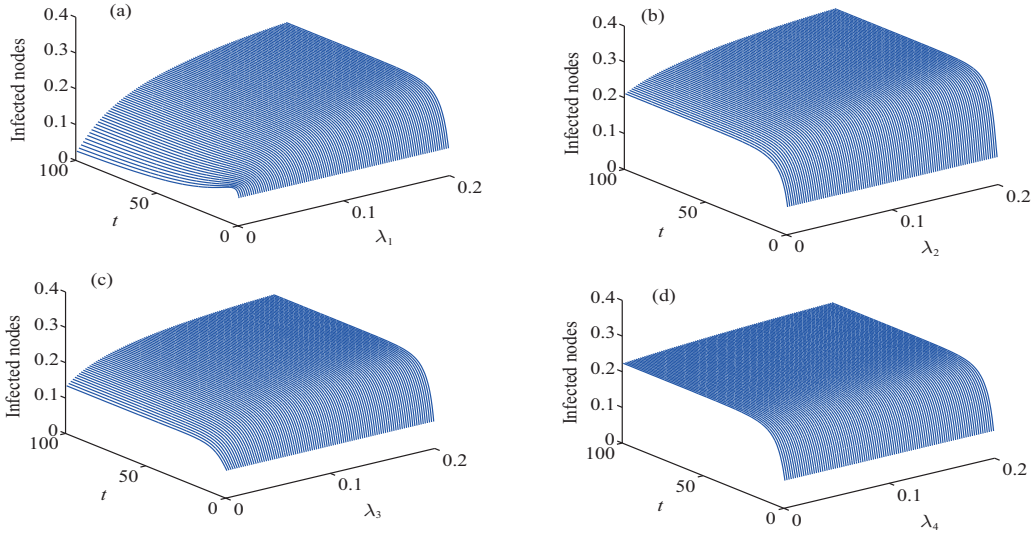


Fig. 5. (color online) Density of the infected nodes as a function of time and parameters λ_1, λ_2, λ_3, and λ_4. (a) Simulation result of parameter λ_1; (b) simulation result of parameter λ_2; (c) simulation result of parameter λ_3; (d) simulation result of parameter λ_4.

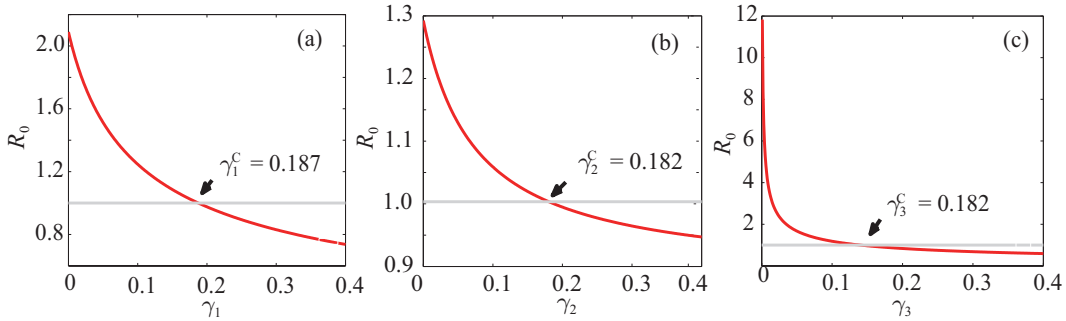


Fig. 6. (color online) Relationship between the basic reproduction number R_0 and parameters γ_1, γ_2, and γ_3. (a) Simulation results of R_0 and γ_1; (b) simulation results of R_0 and γ_2; (c) simulation results of R_0 and γ_3.

4. Effect of vaccination strategies

In this section, we compare the effect of different immunization schemes for different groups in the cases of Barabási and Albert network.

4.1. Proportional immunization

Proportional immunization strategy is the simplest immunization scheme. Fu,[19] Pastor-Satorras,[20] and Madar[21] discussed it with their model for disease control. Let us use σ_1, σ_2, and σ_3 to denote the immunization rate for $S_{1\mathrm{M},k}$, $S_{1\mathrm{M},k}$, and $S_{\mathrm{F},k}$, where $k = 1,2,\ldots n$. Then the model (1) becomes

$$\begin{cases} \dfrac{\mathrm{d}S_{1\mathrm{M},k}}{\mathrm{d}t} = \gamma_1 I_{1\mathrm{M},k} - \lambda_1(1-\sigma_1)kS_{1\mathrm{M},k}\Theta_3 \\ \qquad\qquad - Q_1 S_{1\mathrm{M},k} + Q_2 S_{2\mathrm{M},k}, \\ \dfrac{\mathrm{d}S_{2\mathrm{M},k}}{dt} = \gamma_2 I_{2\mathrm{M},k} - \lambda_2(1-\sigma_2)kS_{2\mathrm{M},k}\Theta_3 \\ \qquad\qquad + Q_1 S_{1\mathrm{M},k} - Q_2 S_{2\mathrm{M},k}, \\ \dfrac{dS_{\mathrm{F},k}}{\mathrm{d}t} = \gamma_3 I_{\mathrm{F},k} - \lambda_3(1-\sigma_3)kS_{\mathrm{F},k}\Theta_1 \\ \qquad\qquad - \lambda_4(1-\sigma_3)kS_{\mathrm{F},k}\Theta_2, \\ \dfrac{\mathrm{d}I_{1\mathrm{M},k}}{\mathrm{d}t} = \lambda_1(1-\sigma_1)kS_{1\mathrm{M},k}\Theta_3 - \gamma_1 I_{1\mathrm{M},k} \\ \qquad\qquad - P_1 I_{1\mathrm{M},k} + P_2 I_{2\mathrm{M},k}, \\ \dfrac{\mathrm{d}I_{2\mathrm{M},k}}{\mathrm{d}t} = \lambda_2(1-\sigma_2)kS_{2\mathrm{M},k}\Theta_3 - \gamma_2 I_{2\mathrm{M},k} \\ \qquad\qquad + P_1 I_{1\mathrm{M},k} - P_2 I_{2\mathrm{M},k}, \\ \dfrac{\mathrm{d}I_{\mathrm{F},k}}{\mathrm{d}t} = \lambda_3(1-\sigma_3)kS_{\mathrm{F},k}\Theta_1 + \lambda_4 k(1-\sigma_3)S_{\mathrm{F},k}\Theta_2 \\ \qquad\qquad - \gamma_3 I_{\mathrm{F},k}, \end{cases}$$

Using the same method introduced in Section 2.2, we then calculate the reproductive number to obtain

$$\tilde{R}_0 = \frac{\langle k^2\rangle}{\langle k\rangle}\sqrt{\frac{(1-\Lambda)\Lambda\tilde{\kappa}}{(Q_1+Q_2)(\gamma_1\gamma_2+P_1\gamma_2+P_2\gamma_1)\gamma_3}}$$

where

$$\begin{aligned}\tilde{\kappa} \triangleq\ & (1-\sigma_3)(\lambda_3(Q_2\gamma_2\lambda_1(1-\sigma_1)+P_2Q_2\lambda_1(1-\sigma_1) \\ & + P_2Q_1\lambda_2(1-\sigma_2))+\lambda_4(Q_1\gamma_1\lambda_2(1-\sigma_2) \\ & + P_1Q_1\lambda_2(1-\sigma_2)+P_1Q_2\lambda_1(1-\sigma_1))).\end{aligned}$$

Comparing the expressions of $\tilde{R}_0$ and R_0, we can find that $\tilde{R}_0 = R_0$ if $\sigma_1 = 0$, $\sigma_2 = 0$, and $\sigma_3 = 0$, which means that there is no immunization; for $0 < \sigma_i < 1$ $(i = 1,2,3)$, $\tilde{R}_0 < R_0$, that is, the immunization scheme is effective; if $\sigma_1 \to 1$, $\sigma_2 \to 1$ or $\sigma_3 \to 1$, then $\tilde{R}_0 \to 0$, namely, in the case of a full immunization, it would be impossible for the epidemic to spread in the network.

Figure 7 shows the comparison of immunization for different groups, where the parameters are chosen as $\lambda_1 = 0.09$, $\lambda_2 = 0.008$, $\lambda_3 = 0.07$, $\lambda_4 = 0.005$, $\gamma_1 = 0.11$, $\gamma_2 = 0.12$, $\gamma_3 = 0.13$, $\Lambda = 4/5$, $Q_1 = 0.1$, $Q_2 = 0.1$, $P_1 = 0.1$, and $P_2 = 0.1$. From this figure, one can find that the effects of immunization for $S_{1\mathrm{M},k}$ and $S_{\mathrm{F},k}$ are better than the effects of immunization for $S_{2\mathrm{M},k}$.

4.2. Targeted immunization

Another immunization strategy is targeted immunization. Fu[19] and Pastor-Satorras[20] have mentioned targeted immunization and found that it is effective. The strategy is to immunize the most highly connected nodes, i.e., the ones that are more likely to spread the disease. For the network, we introduce lower and upper thresholds α_1 and α_2, such that if $k > \alpha_2$, all nodes with connectivity k are immunized, while if $\alpha_1 < k \le \alpha_2$, c_k $(0 < c_k \le 1)$ portion will be immunized, and c_k is defined as the fraction of individuals to be immunized, i.e., we define the immunization rate $\delta_{i,k}$ for different susceptible groups, $\delta_{1,k}$ for $S_{1\mathrm{M},k}$, $\delta_{2,k}$ for $S_{1\mathrm{M},k}$, and $\delta_{3,k}$ for $S_{\mathrm{F},k}$.

$$\delta_{i,k} = \begin{cases} 1, & (k > \alpha_2), \\ c_{i,k}, & (\alpha_1 < k \le \alpha_2,\ \ i = 1,2,3), \\ 0, & (k \le \alpha_1), \end{cases}$$

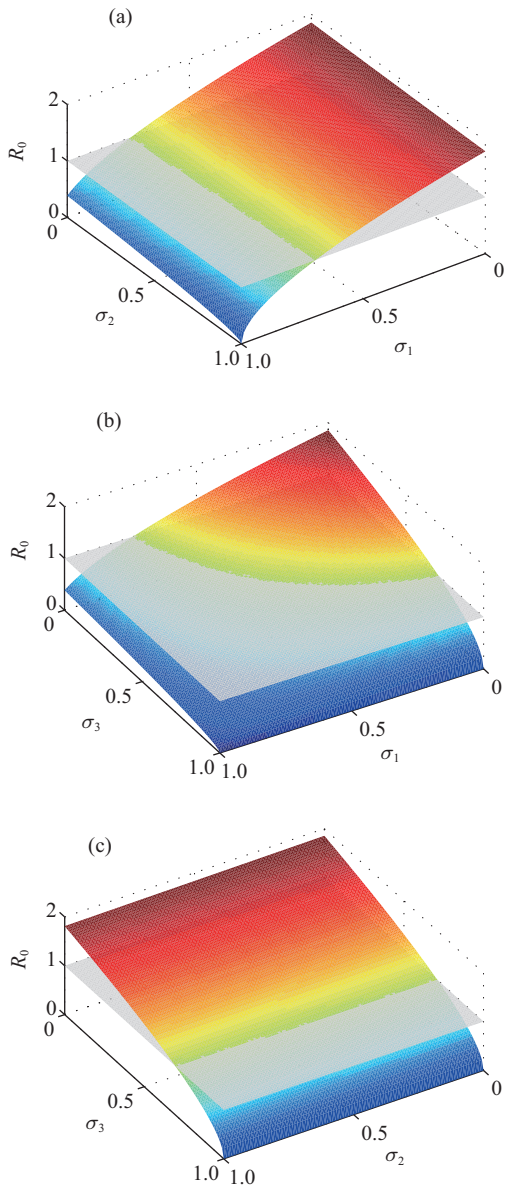


Fig. 7. (color online) The influence of parameters σ_1, σ_2, and σ_3 on $\tilde{R}_0$.

and $\bar{\delta}_i = \sum_k \delta_{i,k} p(k)$ is the average immunization rate. Now the epidemic model (1) becomes

$$\begin{cases} \dfrac{dS_{1M,k}}{dt} = \gamma_1 I_{1M,k} - \lambda_1 (1-\delta_{1,k}) k S_{1M,k} \Theta_3 \\ \qquad - Q_1 S_{1M,k} + Q_2 S_{2M,k}, \\ \dfrac{dS_{2M,k}}{dt} = \gamma_2 I_{2M,k} - \lambda_2 (1-\delta_{2,k}) k S_{2M,k} \Theta_3 \\ \qquad + Q_1 S_{1M,k} - Q_2 S_{2M,k}, \\ \dfrac{dS_{F,k}}{dt} = \gamma_3 I_{F,k} - \lambda_3 (1-\delta_{3,k}) k S_{F,k} \Theta_1 \\ \qquad - \lambda_4 (1-\delta_{3,k}) k S_{F,k} \Theta_2, \\ \dfrac{dI_{1M,k}}{dt} = \lambda_1 (1-\delta_{1,k}) k S_{1M,k} \Theta_3 - \gamma_1 I_{1M,k} \\ \qquad - P_1 I_{1M,k} + P_2 I_{2M,k}, \\ \dfrac{dI_{2M,k}}{dt} = \lambda_2 (1-\delta_{2,k}) k S_{2M,k} \Theta_3 - \gamma_2 I_{2M,k} \\ \qquad + P_1 I_{1M,k} - P_2 I_{2M,k}, \\ \dfrac{dI_{F,k}}{dt} = \lambda_3 (1-\delta_{3,k}) k S_{F,k} \Theta_1 + \lambda_4 (1-\delta_{3,k}) k S_{F,k} \Theta_2 \\ \qquad - \gamma_3 I_{F,k}, \end{cases}$$

Then, it is easy to calculate the basic reproductive number

$$\widehat{R}_0 = \frac{1}{\langle k \rangle} \sqrt{\frac{(1-\Lambda)\Lambda \left(\widehat{\kappa}_1 \widehat{\kappa}_2 + \widehat{\kappa}_3 \widehat{\kappa}_4 \right)}{\gamma_3 (Q_1+Q_2)(\gamma_1\gamma_2 + P_1\gamma_2 + P_2\gamma_1)}},$$

where

$$\begin{aligned} \widehat{\kappa}_1 &= \sum_k k^2 p(k) \lambda_3 (1-\delta_{3,k}), \\ \widehat{\kappa}_2 &= \sum_k k^2 p(k) (Q_2\gamma_2\lambda_1 (1-\delta_{1,k}) + P_2 Q_2 \lambda_1 (1-\delta_{1,k}) \\ &\quad + P_2 Q_1 \lambda_2 (1-\delta_{2,k})), \\ \widehat{\kappa}_3 &= \sum_k k^2 p(k) \lambda_4 (1-\delta_{3,k}), \\ \widehat{\kappa}_4 &= \sum_k k^2 p(k) (Q_1\gamma_1\lambda_2 (1-\delta_{2,k}) + P_1 Q_1 \lambda_2 (1-\delta_{2,k}) \\ &\quad + P_1 Q_2 \lambda_1 (1-\delta_{1,k})). \end{aligned}$$

Note that $\langle k^2 (1-\delta_{i,k}) \rangle = \bar{\delta}_i \langle k^2 \rangle + \delta_i'$, where $\delta_i' = \langle (\delta_{i,k} - \bar{\delta}_i)(k^2 - \langle k^2 \rangle) \rangle$ is the covariance of $\delta_{i,k}$ and k^2. If we let $\sigma_i = \bar{\delta}_i$, then

$$\widehat{R}_0 < \tilde{R}_0,$$

which means the targeted immunization scheme is more efficient than the proportional scheme for the same average immunization rate.

When we set $c_{i,k} = c_i$ for $i = 1,2,3$, $k = 1,2,\ldots,n$, $\tilde{R}_0$ as a function of α_2 and c_i is sketched in Fig. 8 with the parameters being $\rho = 2$, $\eta = 3$, $n = 100$, $\lambda_1 = 0.09$, $\lambda_2 = 0.008$, $\lambda_3 = 0.07$, $\lambda_4 = 0.005$, $\gamma_1 = 0.11$, $\gamma_2 = 0.12$, $\gamma_3 = 0.13$, $\Lambda = 4/5$, $Q_1 = 0.1$, $Q_2 = 0.1$, $P_1 = 0.1$, $P_2 = 0.1$, and $\alpha_1 = 10$ on the Barabási and Albert network with $p(k) = (\eta-1)\rho^{\eta-1}k^{-\eta}$. One can see from this figure that $\tilde{R}_0$ is an increasing function of α_2 but a decreasing function of c_i. In other words, if c_i is large or α_2 is small, more people receive a vaccination, and consequently the disease can be controlled. Moreover, it can be seen that the effects of immunization for $S_{1M,k}$ and $S_{F,k}$ are better than the effects of immunization for $S_{2M,k}$.

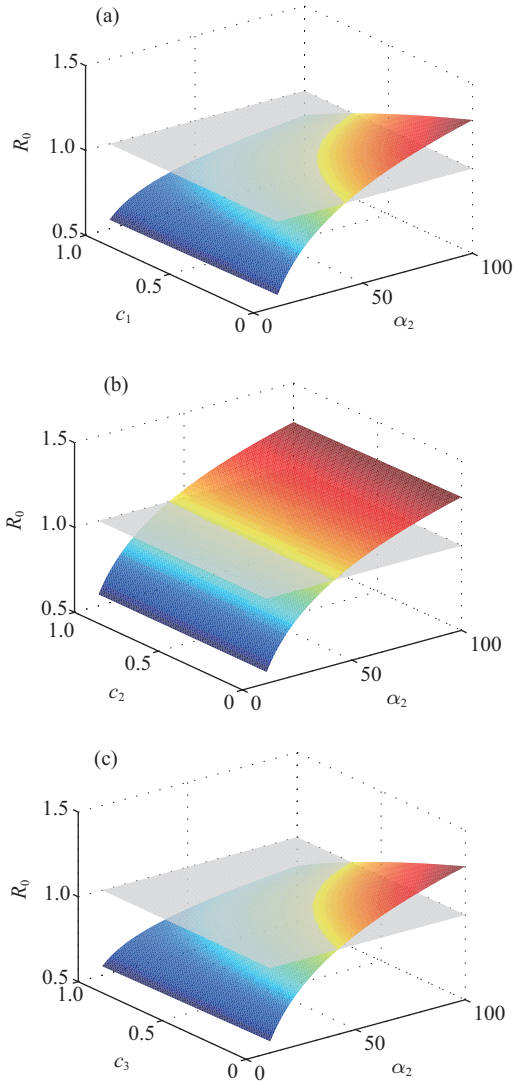


Fig. 8. (color online) The influence of parameters c_1, c_2, and c_3 with α_2 on $\widehat{R}_0$.

5. Discussion

In a previous work,[13] the authors also proposed a sexually transmitted disease model on complex networks, and they held the idea that the male and the female are in different networks, and the model they studied is based on the two BA networks. However, the disease spread depends on the contact between humans. The contact between humans forms the network. If the male and the female are in different networks, it means that they would not contact each other. Furthermore, we propose our sexually transmitted disease model about commercial sex on complex networks, where contacts between humans are treated as a scale-free social network. Moreover, differently from the other disease model on complex networks, we believe that the disease spread is not only related to the degree of the individuals, but also to the different behavior

of individuals. Thus, in view of different sexual behavior of males, there are three groups of people in our model, which are a dangerous male, a non-dangerous male, and a female. A dangerous male represents a male who has many sexual contacts or does not use protection when they have sexual contact. A non-dangerous male represents a male who has few sexual contacts or uses protection when they have sexual contact. A female refers to the sex workers. Then, the basic reproduction number R_0 for the existence of the endemic equilibrium, which numerical simulations confirm, is obtained. Finally, the effects of various immunization schemes for different groups are studied. Through comparing them, we find that the targeted immunization scheme is more efficient than the proportional scheme for the same average immunization rate, and the effects of immunization for $S_{1\mathrm{M},k}$ and $S_{\mathrm{F},k}$ are better than the effects of immunization for $S_{2\mathrm{M},k}$.

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