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THE ANALYSIS OF SEXUALLY TRANSMITTED DISEASES WITH DEMOGRAPHICS ON SCALE-FREE NETWORK†

MAOXING LIU* AND YUNLI ZHANG

Abstract. In this paper we consider a model with demographics for sexually transmitted diseases (STDs) spread on scale-free networks. We derive the epidemic threshold, which is depend on the birth rate, the natural death rate and other parameters. The absence of a threshold in infinite scale-free network is proved. For a hard cut off scale-free network, we also analyze the stability of disease-free equilibrium and the persistence of STDs infection. Two immunization schemes, proportional scheme and targeted vaccination, are studied and compared. We find that targeted strategy is more effective on scale-free networks.

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1. Introduction

The dynamical behaviors of epidemic diseases have been studied for a long time, SIS and SIR are the two important and fundamental epidemic models [1]. Epidemic spreading can be thought of as occurring on complex networks where the nodes of the network represent individuals and the links represent various interactions among those individuals. In general, networks can be characterized by the connectivity of their nodes. The degree k of a node is defined as the number of links connected to the node. The degree distribution of a network $P(k)$ is defined as the probability of a randomly chosen node to have a degree k. Many networks such as social networks, the Internet and the World Wide Web (WWW) have been found to be scale-free networks [5, 6, 7, 8, 9], meaning that the degree distribution follows a power-law $P(k) \sim k^{-2-\gamma}$, with $0 < \gamma \leq 1$.

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Models of epidemic spreading on networks have been studied extensively in recent years [3]. For instance, a very important example of scale-free networks is found in the web of human sexual contacts [4]. Data from national sex surveys [4, 10] provide quantitative information on the number of sexual partners, i.e., the degree k, of an individual. The respondents are asked to provide information on sexual attitudes such as the number of sex partners they have had in the last 12 months or in their entire life. It turns out that the number of heterosexual partners reported from different populations is well described by power-law distribution [2, 11].

Since STDs can spread through scale-free networks, many mathematical models on this topic have been studied [12, 16, 23, 24]. R. Pastor-Satorras et al. stated that, in infinite scale-free networks, epidemic processes do not possess an epidemic threshold [14]. The absence of an intrinsic epidemic threshold has been found in both the susceptible-infected-susceptible (SIS) model [14] and the susceptible-infected-removed (SIR) model in infinite scale-free networks [15]. The above results normally only prove the existence of epidemic equilibrium, but from a mathematical aspect, such SI_1I_2RS models on a bounded network can be viewed as multiple $SIRS$ models [16, 17]. In this way, the stabilities of equilibria also can be proved [17]. Because of some infectious disease with the longer spreading time, demographics such as birth, death etc, should be considered in the epidemic models. Liu consider the spread of epidemic diseases with birth and death on networks [19].

Based on the above results, in this paper, we consider a population with two types of susceptible individuals and two types of infected individuals: One proportion of susceptible individuals has weakly self-protection awareness, so the rate of being infected is bigger than other susceptible individuals; One proportion of infected individuals who potentially has a small infection rate since they use such as condoms to protect their partners (for most STDs, although a condom can reduce the chances of the transmission of these virus or bacterium if it covers the affected areas, it is not entirely effective. A condom may not cover all of the sores or rashes in the affected areas, and direct skin contact may give rise to transmission [18]); The other proportion who do not have any protection (we say they have high-risk sexual behaviors) and potentially have a large infection rate.

Thus we get an $S_1S_2I_1I_2RS$ STDs model on a scale-free network. Our model relies on the following rough description of individuals in the population. Namely, each node of the graph represents an individual and each link is a connection along which the STDs can spread. We suppose each susceptible (healthy) node is infected with rate β_1 or β_2 if it is connected to one or more infected nodes. Infected nodes are cured with rate η and recovered nodes again become susceptible with rate δ. We derive that epidemic processes of our model do not possess an epidemic threshold, like in model SIS and SIR [14, 15], in infinite scale-free network. Since realistic systems are actually made up by a

finite number of individuals, this finite population introduces a maximum connectivity, depending on the size of the network [20]. In this paper, we also discuss the stability of equilibria and the permanent of infection on a bounded hard-cutoff scale-free network. Since a finite network has the effect of restoring a boundary in the connectivity fluctuations, in this way it produces an effective non-zero threshold [23, 24]. By applying two immunization strategies (proportional immunization, targeted immunization) to the STDs model, it is shown that the second strategy has an overwhelming advantage compare with the first one. Finally, we prove the benefit that divides the susceptible individuals into two subgroup according to sexual values, and divides the infected individuals into two subgroup according to condom using or not. For a given immunization rate, we can get the suitable condom using rate to control STDs spread on networks.

The organized of this paper is as follows. In the next section, we will describe the epidemic model on networks with birth and death. Section 3.1 is devoted to the threshold for the STDs spread on scale-free network. We analyze the stability of disease-free equilibrium and the persistence of STDs infection in section 3.2. In Section 4, we discuss and compare the effect of two immunization strategies. The paper ends with a conclusion and discussion in Section 5.

2. A multiple $SIRS$ model

Let S_{1k}, S_{2k}, I_{1k}, I_{2k} and R_k represent the relative densities of nodes of degree k. They also denote the densities of the susceptible with weakly self-protection awareness, the susceptible with strongly self-protection awareness, the infectious with low infectivity, the infectious with high infectivity and the recovered respectively. So that the total population size is

$$N(t) = S_{1k}(t) + S_{2k}(t) + I_{1k}(t) + I_{2k}(t) + R_k(t). \tag{1}$$

On the network each site of N is empty or occupied by only one individual. We give each site a number: 0, 1, 2, 3, 4 or 5. Alternatively we can interpret the six states as 0 : vacant, 1 : a healthy individual with weakly self-protection occupation, 2 : a healthy individual with strongly self-protection occupation, 3 : an infected individual with low infection rate occupation, 4 : an infected individual with high infection rate occupation, 5 : a recover individual occupation. The state of the system at time t can be described by a set of numbers $0, 1, 2, 3, 4, 5$. That means if the system is in the state A and the site $x \in N$, then $A_t(x) \in \{0, 1, 2, 3, 4, 5\}$. Each site can change its state with a certain rate. A empty site can give new individual to healthy with strongly self-protection at the rate b (about the new individual the protection from the family). The state-transition rules of the contact process are schematically shown in Fig. 1.

Then we have the following dynamics model:

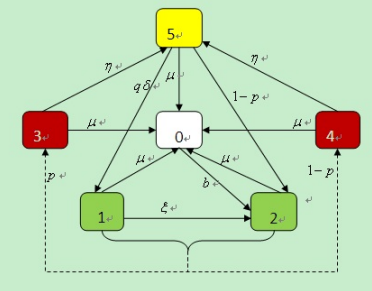

FIGURE 1. The state-transition rules of the multiple $SIRS$ model.

$$\begin{cases} \frac{dS_{1k}(t)}{dt} = q\delta R_k(t) - kS_{1k}(t)\Theta(t) - (\xi+\mu)S_{1k}(t), \\ \frac{dS_{2k}(t)}{dt} = b[1 - S_{1k}(t) - S_{2k}(t) - I_{1k}(t) - I_{2k}(t) - R_k(t)] \\ \qquad +(1-q)\delta R_k(t) + \xi S_{1k}(t) - k\varepsilon S_{2k}(t)\Theta(t) - \mu S_{2k}(t), \\ \frac{dI_{1k}(t)}{dt} = pk[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - (\eta+\mu)I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} = (1-p)k[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - (\eta+\mu)I_{2k}(t), \\ \frac{dR_k(t)}{dt} = \eta I_{1k}(t) + \eta I_{2k}(t) - (\delta+\mu)R_k(t). \end{cases} \tag{2}$$

where $\Theta(t)$ is defined as

$$\Theta(t) = \frac{1}{\langle k\rangle}\sum_{k=1}^{\infty}\psi(k)P(k)[\beta_1 I_{1k}(t) + \beta_2 I_{2k}(t)]. \tag{3}$$

In this paper, we suppose that the connectivity of nodes on this network is uncorrelated. In the case of an uncorrelated random network, the probability that a link points to a node of connectivity s is independent of the connectivity k of the node from which the link is emanating. The meanings for each parameter or item of system (2) are:

Infected nodes are cured with rate η and recovered nodes again become susceptible with rate δ. Parameter ε represents the rate of the lost of self-protection awareness for the susceptible with strongly self-protection awareness. Parameter ξ represents the transfer rate from the susceptible who has weakly self-protection awareness to the one with strongly self-protection awareness, based on the spreading of the information about the sexually transmitted diseases. Parameter μ represents the rate of natural death.

In the first and second equation, $kS_{1k}(t)\Theta(t)$ and $k\varepsilon S_{2k}(t)\Theta(t)$ represent the lost of the weakly self-protection and the strongly self-protection awareness susceptible individuals respectively because of infection, which is proportional to the connectivity k, the densities of the weakly self-protection healthy nodes S_{1k} and the strongly self-protection awareness healthy nodes S_{2k} respectively, infected nodes I_{1k} and I_{2k}. Factors $\frac{\psi(k)P(k)}{\langle k\rangle}$ in $\Theta(t)$ represents the expectation that any given link emanating from a node of connectivity k points to an infected

node. Parameters β_1 and β_2 in $\Theta(t)$ are the STDs transmission rates for each sexual behavior of subgroups I_{1k} and I_{2k} respectively.

In the third and fourth equations, parameter p is the rate of the usage of condom. Suppose that after being infected by STDs, there are p proportion infected individuals begin to control their behavior, such as using condom to protect their partners, but the other $1-p$ proportion individuals still keep their high risk behavior. Here $q\delta$ means the rate from immunization-lost for recovered individuals to the unawareness of self-protection susceptible compartment, but the other $(1-q)\delta$ proportion immunization-lost for recovered individuals enter into the awareness of strongly self-protection susceptible compartment. And according to their biological meanings, we have $\beta_2 > \beta_1 > 0$, $\delta > 0$, $\xi > 0$, $\varepsilon > 0$, $\mu > 0$, $\eta > 0$ and $0 \le p, q \le 1$.

Now we give some explanations to symbols in $\Theta(t)$. Here $\langle k \rangle$ is the average degree of the network, which can be understood as the first moment of degree k: $\langle k \rangle = \sum_k kP(k)$. $P(k)$ is the degree distribution, i.e., the probability that a randomly chosen node within the network has degree k. Function $\psi(k)$ denotes the infectivity of a node with degree k. In [13], the authors suppose that the infectivity $\psi(k)$ of each node (each nodes potential infection-activity) with degree k is $\psi(k) = \alpha k$, where α is a positive constant, $0 < \alpha \le 1$. Then they get the epidemic threshold $\lambda_c = 0$ for sufficiently large networks. In [18], the authors suppose the infectivity $\psi(k)$ of a node with degree k is a constant A, which means every node has the same infectivity, no matter its degree. In this case, $\lambda_c = \frac{1}{A}$ is a positive threshold which is independent of the topology. But for STDs spread, different kind of nodes, such as a sex workers and a normal woman, they of course have different numbers of sexual contacts in one time step. For this reason, we think that $\psi(k) = \alpha k$ is much more suitable than a constant A one for each node of degree k.

Since the probability that a node of connectivity k is connected to an isolated node is zero, so we only consider the situation that $k \ge 1$ in our paper. So system (2), combined with (3) and the initial conditions $S_{1k}(0) = S_{1k}^0$, $I_{1k}(0) = I_{1k}^0$, $I_{2k}(0) = I_{2k}^0$, $R_k(0) = R_k^0$, and $S_{2k}(0) = \frac{b}{b+\mu} - S_{1k}^0 - I_{1k}^0 - I_{2k}^0 - R_k^0$, completely define the $S_1S_2I_1I_2RS$ model on an uncorrelated network with degree distribution $P(k)$.

3. Some results

For each k, adding five equations in (2) gives

$$\frac{dN_k(t)}{dt} = b - (b+\mu)N_k(t).$$

Hence $\limsup_{t\to\infty}(S_{1k} + S_{2k} + I_{1k} + I_{2k} + R_k) \le \frac{b}{b+\mu}$. Therefore, omega limit sets of system (2) are contained in the following bounded region in the non-negative cone of $R^{5\kappa_c}$:

$$C = \{(S_{11}, S_{21}, I_{11}, I_{21}, R_1, \ldots, S_{1\kappa_c}, S_{2\kappa_c}, I_{1\kappa_c}, I_{2\kappa_c}, R_{\kappa_c}), S_{1k} \ge 0, S_{2k} \ge 0,$$

$$I_{1k} \geq 0, I_{2k} \geq 0, R_k \geq 0, S_{1k} + S_{2k} + I_{1k} + I_{2k} + R_k \leq b/(b+\mu)\},$$

and κ_c is the maximum number of contact each individual. It can be verified that region C is positively invariant. Consequently, the dynamics of the model would be considered in C.

3.1. The threshold R_0 on infinite scale-free network. In this subsection, we discuss the existence of the epidemic equilibrium solution of system (2). We have the following theorem.

Theorem 3.1. *Define*

$$R_0 = \frac{\alpha b\varepsilon[p\beta_1 + (1-p)\beta_2]}{(\eta+\mu)(b+\mu)} \frac{\langle k^2\rangle}{\langle k\rangle}, \tag{4}$$

there always exists a disease-free equilibrium solution $E^0 = (0, \frac{b}{b+\mu}, 0, 0, 0)$ for system (2), and when $R_0 > 1$ there exists one and only one epidemic equilibrium.

Proof. Since S_{1k}, S_{2k}, I_{1k}, I_{2k} and R_k represent the relative densities of nodes of degree k and we consider the death rate and the birth rate of each node, so these variables obey that

$$S_{1k}(t) + S_{2k}(t) + I_{1k}(t) + I_{2k}(t) + R_k(t) = \frac{b}{b+\mu}. \tag{5}$$

To get the epidemic solution , we need to impose the right side of system (2) to be zero. Then any equilibrium $(S_{1k}(\infty), S_{2k}(\infty), I_{1k}(\infty), I_{2k}(\infty), R_k(\infty))$ should satisfy

$$I_{2k}(\infty) = \frac{(1-p)k\varepsilon b\Theta(\infty)[\xi+\mu+k\Theta(\infty)](\delta+\mu)}{\triangle}, \tag{6}$$

where $\triangle = (b+\mu)(\delta+\mu)(\eta+\mu)(\xi+\mu) + (b+\mu)[\varepsilon(\delta+\eta+\mu)][k\Theta(\infty)]^2 + (b+\mu)[(\eta+\mu)(\delta+\mu) + \varepsilon(\xi+\mu)(\eta+\delta+\mu) - q\eta\delta(1-\varepsilon)]k\Theta(\infty)$, and

$$I_{1k}(\infty) = \frac{p}{1-p} I_{2k}(\infty), \tag{7}$$

$$R_k(\infty) = \frac{\eta}{\delta+\mu}[I_{1k}(\infty) + I_{2k}(\infty)], \tag{8}$$

$$S_{1k}(\infty) = \frac{q\delta}{\xi+\mu+k\Theta(\infty)} R_k(\infty), \tag{9}$$

$$S_{2k}(\infty) = \frac{b}{b+\mu} - S_{1k}(\infty) - I_{1k}(\infty) - I_{2k}(\infty) - R_k(\infty). \tag{10}$$

For simplicity, we omit the symbol ∞ in the following. Substitute (6) and (7) in (3), we obtain a self-consistency equation as follows:

$$\Theta = f_1(\Theta) f_2(\Theta) \equiv f(\Theta), \tag{11}$$

where $f_1(\Theta) = \frac{1}{\langle k\rangle} \sum_{k=1}^{\infty} \alpha k^2 P(k) \frac{p\beta_1 + (1-p)\beta_2}{1-p}$, $f_2(\Theta) = \frac{(1-p)b\varepsilon\Theta(\xi+\mu+k\Theta)(\delta+\mu)}{g(\Theta)}$ and $g(\Theta) = (b+\mu)(\delta+\mu)(\eta+\mu)(\xi+\mu) + (b+\mu)[\varepsilon(\delta+\eta+\mu)](k\Theta)^2 + [(\eta+\mu)(\delta+$

$\mu) + \varepsilon(\xi + \mu)(\eta + \mu + \delta) - q\eta\delta(1 - \varepsilon)](b + \mu)k\Theta$. Obviously $\Theta = 0$ is a solution of (11), i.e., $f(0) = 0$. So if a non-trivial solution exists, it should satisfy

$$\frac{df(\Theta)}{d\Theta}|_{\Theta=0} > 1,$$

that is,

$$\frac{\alpha b\varepsilon[p\beta_1 + (1-p)\beta_2]}{(\eta+\mu)(b+\mu)}\frac{\langle k^2\rangle}{\langle k\rangle} > 1,$$

which yields the critical epidemic threshold R_0 given in (4). So when $R_0 > 1$, one and only one epidemic equilibrium solution of system (2) exists. This finishes the proof. □

Clearly, for an infinite scale-free networks (in which situation $\langle k^2\rangle \to \infty$), the epidemic processes of our model do not possess an epidemic threshold, below which diseases cannot produce a major epidemic outbreak, like the results of standard SIS model and SIR model [14, 15].

3.2. The stability and the persistence. Real systems are actually made up by a finite number of individuals. This finite population introduces a maximum connectivity k_c . In this section, we will discuss the stability of equilibria for a hard cutoff scale-free network [20]. First we recall a theorem by Lajmanovich and York [16] that will be useful as a lemma in the following.

Lemma3.2 (Lajmanovich and York). *Consider the system*

$$\frac{dy}{dt} = Ay + N(y), \tag{12}$$

where A is an $n \times n$ matrix and $N(y)$ is continuously differentiable in a region $D \subset R^n$. Assume
1. *the compact convex set $C \subset D$ is positively invariant with respect to the system (12), and $0 \in C$;*
2. $\lim\limits_{y\to 0} \|N(y)\|/\|y\| = 0$;
3. *there exist $r > 0$ and a (real) eigenvector ω of A^T such that $\omega \cdot y \geq r\|y\|$ for all $y \in C$;*
4. $\omega \cdot N(y) \leq 0$ *for all $y \in C$;*
5. *$y = 0$ is the largest positively invariant set [for (12)] contained in $H = y \in C \mid (\omega \cdot N(y)) = 0$. Then either $y = 0$ is globally asymptotically stable in C, or for any $y_0 \in C - \{0\}$ the solution $\phi(t, y_0)$ of (12) satisfies $\liminf\limits_{t\to\infty} \|\phi(t, y_0)\| \geq m$, where $m > 0$, independent of y_0. Moreover, there exists a constant solution of (12), $y = k$, $k \in C - \{0\}$.*

About the stability of the disease-free equilibrium we have the following theorem.

Theorem 3.2. *If $R_0 < 1$, the disease-free equilibrium solution is globally asymptotically stable; If $R_0 > 1$, one and only one epidemic equilibrium solution of system (2) exists, and system (2) is permanent of infection, i.e., there exists an $\varepsilon > 0$, such that*

$$\lim_{t\to\infty} \inf\{I_{1k}(t), I_{2k}(t)\}_{k=1}^{\kappa_c} > \varepsilon,$$

for any solution of (2) with $S_{1k}(0) > 0$, $S_{2k}(0) > 0$, $I_{1k}(0) > 0$ (or $I_{2k} > 0$ or both hold) and $R_k(0) \geq 0$.

Proof. From (5) we know that system (2) can be rewritten as

$$\begin{cases} \frac{dS_{1k}(t)}{dt} = q\delta R_k(t) - kS_{1k}(t)\Theta(t) - (\xi+\mu)S_{1k}(t), \\ \frac{dI_{1k}(t)}{dt} = pk[S_{1k}(t)\Theta(t) + \varepsilon(\frac{b}{b+\mu} - S_{1k}(t) - I_{1k}(t) - I_{2k}(t) - R_k(t)) \\ \qquad \Theta(t)] - (\eta+\mu)I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} = (1-p)k[S_{1k}(t)\Theta(t) + \varepsilon(\frac{b}{b+\mu} - S_{1k}(t) - I_{1k}(t) - I_{2k}(t) \\ \qquad -R_k(t))\Theta(t)] - (\eta+\mu)I_{2k}(t), \\ \frac{dR_k(t)}{dt} = \eta I_{1k}(t) + \eta I_{2k}(t) - (\delta+\mu)R_k(t). \end{cases} \tag{13}$$

In the following, we discuss the dynamic of (13) in the subspace

$$C_1 = \{(S_{11}, I_{11}, I_{21}, R_1, \ldots, S_{1\kappa_c}, I_{1\kappa_c}, I_{2\kappa_c}, R_{\kappa_c}),$$

$$S_{1k} \geq 0, I_{1k} \geq 0, I_{2k} \geq 0, R_k \geq 0, S_{1k} + I_{1k} + I_{2k} + R_k \leq b/(b+\mu)\}.$$

For simplicity, let $\frac{sP(s)}{\langle k\rangle} \equiv q_s$. The Jacobian matrix of the disease-free equilibrium of system (13) which is a $4\kappa_c \times 4\kappa_c$ matrix can be written as follows:

$$J = \begin{pmatrix} A & \cdots & B \\ \vdots & \ddots & \vdots \\ C & \cdots & D \end{pmatrix},$$

where

$$A = \begin{pmatrix} -(\xi+\mu) & -S_{11}\alpha\beta_1 q_1 & -S_{11}\alpha\beta_2 q_1 & q\delta \\ 0 & a_{22} & a_{23} & 0 \\ 0 & a_{32} & a_{33} & 0 \\ 0 & \eta & \eta & -(\delta+\mu) \end{pmatrix},$$

where $a_{22} = p\alpha\beta_1 q_1[(1-\varepsilon)S_{11}(t) + \frac{b\varepsilon}{b+\mu}] - (\eta+\mu)$, $a_{23} = p\alpha\beta_2 q_1[(1-\varepsilon)S_{11}(t) + \frac{b\varepsilon}{b+\mu}]$, $a_{32} = \alpha(1-p)\beta_1 q_1[(1-\varepsilon)S_{11}(t) + \frac{b\varepsilon}{b+\mu}]$, $a_{33} = \alpha(1-p)\beta_2 q_1[(1-\varepsilon)S_{11}(t) + \frac{b\varepsilon}{b+\mu}] - (\eta+\mu)$, and

$$B = \begin{pmatrix} 0 & -S_{11}\alpha q_{\kappa_c}\beta_1 & -S_{11}\alpha q_{\kappa_c}\beta_2 & 0 \\ 0 & b_{22} & b_{23} & 0 \\ 0 & b_{32} & b_{33} & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

where $b_{22} = p\alpha[(1-\varepsilon)S_{11} + \frac{b\varepsilon}{b+\mu}]q_{\kappa_c}\beta_1$, $b_{23} = p\alpha[(1-\varepsilon)S_{11} + \frac{b\varepsilon}{b+\mu}]q_{\kappa_c}\beta_2$, $b_{32} = (1-p)\alpha q_{\kappa_c}\beta_1[(1-\varepsilon)S_{11} + \frac{b\varepsilon}{b+\mu}]$, $b_{33} = (1-p)\alpha q_{\kappa_c}\beta_2[(1-\varepsilon)S_{11} + \frac{b\varepsilon}{b+\mu}]$, and

$$C = \begin{pmatrix} 0 & -\kappa_c S_{1\kappa_c}\alpha q_{\kappa_c}\beta_1 & -\kappa_c S_{1\kappa_c}\alpha q_{\kappa_c}\beta_2 & 0 \\ 0 & c_{22} & c_{23} & 0 \\ 0 & c_{32} & c_{33} & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

where $c_{22} = \kappa_c p[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}]\alpha q_1\beta_1$, $c_{23} = \kappa_c p[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}]\alpha q_1\beta_2$, $c_{32} = \kappa_c(1-p)\alpha q_1\beta_1[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}]$, $c_{33} = \kappa_c(1-p)\alpha q_1\beta_2[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}]$, and

$$D = \begin{pmatrix} -(\xi+\mu) & -\kappa_c S_{1\kappa_c}\alpha q_{\kappa_c}\beta_1 & -\kappa_c S_{1\kappa_c}\alpha q_{\kappa_c}\beta_2 & q\delta \\ 0 & d_{22} & d_{23} & 0 \\ 0 & d_{32} & d_{33} & 0 \\ 0 & \eta & \eta & -(\delta+\mu) \end{pmatrix},$$

where $d_{22} = p\kappa_c\alpha\beta_1 q_{\kappa_c}[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}] - (\eta+\mu)$, $d_{23} = \kappa_c p[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}]\alpha q_{\kappa_c}\beta_2$, $d_{32} = \kappa_c(1-p)[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}]\alpha q_{\kappa_c}\beta_1$, $d_{33} = (1-p)\kappa_c\alpha\beta_2 q_{\kappa_c}[(1-\varepsilon)S_{1\kappa_c} + \frac{b\varepsilon}{b+\mu}] - (\eta+\mu)$. Using mathematical induction method we can calculate that, the polynomial equation of the disease-free equilibrium is $g_1 \cdot g_2 = 0$, where

$$g_1 = (\lambda+\xi+\mu)^{\kappa_c}(\lambda+\delta+\mu)^{\kappa_c}(\lambda+\eta+\mu)^{2\kappa_c-1},$$

$$g_2 = \lambda+\eta+\mu-\alpha[p\beta_1+(1-p)\beta_2]\cdot\{\frac{1^2P(1)}{\langle k\rangle}[(1-\varepsilon)S_{11}+\frac{b\varepsilon}{b+\mu}]$$

$$+\frac{2^2P(2)}{\langle k\rangle}[(1-\varepsilon)S_{12}+\frac{b\varepsilon}{b+\mu}]+\cdots+\frac{\kappa_c^2P(\kappa_c)}{\langle k\rangle}[(1-\varepsilon)S_{1\kappa_c}+\frac{b\varepsilon}{b+\mu}]\}.$$

So there exists a unique positive eigenvalue λ of J if and only if $R_0 > 1$, under which, the unique epidemic equilibrium exists. Otherwise all real-valued eigenvalues of J are negative. According to the Perron-Frobenius theorem, this implies that the maximal of the real parts of all eigenvalues of J is positive if and only if $R_0 > 1$. From Lemma 3.2 we finish the proof of this theorem. □

4. Immunization strategies

Vaccination is very helpful in controlling vaccine-preventable disease [14, 21]. In this section we discuss system (2) on a scale-free network with two immunization schemes: the proportional immunization and the targeted immunization.

4.1. Proportional immunization. In this case, for fixed spreading rates β_1, β_2, let γ be the immunization rate, $0 < \gamma < 1$. At the mean-filed level, the presence of proportional immunity will effectively reduce the spreading rate. Thus we can approximatively use $\beta_1(1-\gamma)$ to substitute β_1 and use $\beta_2(1-\gamma)$

to substitute β_2, then system (2) becomes

$$\begin{cases} \frac{dS_{1k}(t)}{dt} = q\delta R_k(t) - k(1-\gamma)S_{1k}(t)\Theta(t) - (\xi+\mu)S_{1k}(t), \\ \frac{dS_{2k}(t)}{dt} = b[1 - S_{1k}(t) - S_{2k}(t) - I_{1k}(t) - I_{2k}(t) - R_k(t)] + (1-q)\delta R_k(t) \\ \qquad +\xi S_{1k}(t) - k(1-\gamma)\varepsilon S_{2k}(t)\Theta(t) - \mu S_{2k}(t), \\ \frac{dI_{1k}(t)}{dt} = p(1-\gamma)k[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - (\eta+\mu)I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} = (1-p)(1-\gamma)k[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - (\eta+\mu)I_{2k}(t), \\ \frac{dR_k(t)}{dt} = \eta I_{1k}(t) + \eta I_{2k}(t) - (\delta+\mu)R_k(t). \end{cases} \tag{14}$$

Using the method in subsection 3.1, we can get that

$$\begin{aligned} I_{2k}(\infty) &= \frac{k\varepsilon b(1-p)(1-\gamma)\Theta(\infty)(\mu+\delta)[\xi+\mu+k(1-\gamma)\Theta(\infty)]}{\triangle_1}, \\ I_{1k}(\infty) &= \frac{p}{1-p}I_{2k}(\infty), \\ R_k(\infty) &= \frac{\eta}{\delta+\mu}[I_{1k}(\infty) + I_{2k}(\infty)], \\ S_{1k}(\infty) &= \frac{q\delta}{\xi+\mu+k(1-\gamma)\Theta(\infty)}R_k(\infty), \\ S_{2k}(\infty) &= \frac{b}{b+u} - S_{1k}(\infty) - I_{1k}(\infty) - I_{2k}(\infty) - R_k(\infty), \end{aligned}$$

where $\triangle_1 = (b+\mu)(\eta+\mu)(\delta+\mu)(\xi+\mu) + k(b+\mu)(1-\gamma)\Theta(\infty)[(\eta+\mu)(\delta+\mu) - (1-\varepsilon)q\delta\eta + \varepsilon(\xi+\mu)(\delta+\mu+\eta)] + (b+\mu)[\varepsilon(\mu+\delta+\eta)][k(1-\gamma)\Theta(\infty)]^2$, and $\Theta = \bar{f}_1(\Theta)\bar{f}_2(\Theta) \equiv \bar{f}(\Theta)$, $\bar{f}_1(\Theta) = \frac{1}{\langle k\rangle}\sum\limits_{k=1}^{\infty}\frac{\alpha kP(k)[p\beta_1+(1-p)\beta_2]}{1-p}$, $\bar{f}_2(\Theta) = \frac{k\varepsilon b(1-p)(1-\gamma)(\mu+\delta)\Theta[\xi+\mu+k(1-\gamma)\Theta]}{\bar{g}(\Theta)}$, where $\bar{g}(\Theta) = (b+\mu)(\eta+\mu)(\delta+\mu)(\xi+\mu) + (b+\mu)[\varepsilon(\mu+\delta+\eta)][k(1-\gamma)\Theta]^2 + (b+\mu)[(\eta+\mu)(\delta+\mu) - (1-\varepsilon)q\delta\eta + \varepsilon(\xi+\mu)(\delta+\mu+\eta)]k(1-\gamma)\Theta$. By arguments similar to those in Section 3, the epidemic threshold $\hat{R}_0$ is determined by the following inequality:

$$\frac{d\bar{f}(\Theta)}{d\Theta}\mid_{\Theta=0} > 1. \tag{15}$$

Therefore, it can be shown that

$$\hat{R_0} = \frac{\alpha\varepsilon b(1-\gamma)[p\beta_1 + (1-p)\beta_2]}{(\eta+\mu)(b+\mu)}\frac{\langle k^2\rangle}{\langle k\rangle},$$

that is,

$$\hat{R_0} = (1-\gamma)R_0. \tag{16}$$

Note that in (16), define

$$\gamma_c = 1 - \frac{1}{R_0} = 1 - \frac{1}{\Pi}\cdot\frac{\langle k\rangle}{\langle k^2\rangle},$$

where

$$\Pi = \frac{\alpha b\varepsilon[p\beta_1 + (1-p)\beta_2]}{(b+\mu)(\eta+\mu)}.$$

From the above analysis, we can see when $\gamma > \gamma_c$ (we have $\hat{R_0} < 1$), then the epidemic cannot spread in the network. When $\gamma = 0$, i.e., if no immunization were done, then $\hat{R_0} = R_0 > 1$; when $0 < \gamma < \gamma_c$, i.e., $1 < \hat{R_0} < R_0$, that is, the immunization scheme is effective, but not so effective to control the spread of STDs on networks.

4.2. Targeted immunization. While proportional immunization schemes are effective, there may be more efficient schemes due to the heterogeneous nature of scale-free networks: they are robust to random attacks, but fragile to selective attacks. Accordingly, we can devise a targeted immunization scheme [22]. First, we introduce an upper threshold k_t, and all nodes with connectivity $k > k_t$ are immunized. So the immunization rate γ_k can be defined as

$$\gamma_k = \begin{cases} 1, & k > k_t, \\ c, & k = k_t, \\ 0, & k < k_t. \end{cases}$$

where $0 < c \leq 1$, and $\sum_k \gamma_k P(k) = \bar{\gamma}$, where $\bar{\gamma}$ is the average immunization rate. Then the epidemic dynamic model is

$$\begin{cases} \frac{dS_{1k}(t)}{dt} = q\delta R_k(t) - k(1-\gamma_k)S_{1k}(t)\Theta(t) - (\xi+\mu)S_{1k}(t), \\ \frac{dS_{2k}(t)}{dt} = b[1 - S_{1k}(t) - S_{2k}(t) - I_{1k}(t) - I_{2k}(t) - R_k(t)] + (1-q)\delta R_k(t) \\ \qquad +\xi S_{1k}(t) - k(1-\gamma_k)\varepsilon S_{2k}(t)\Theta(t) - \mu S_{2k}(t), \\ \frac{dI_{1k}(t)}{dt} = p(1-\gamma_k)k[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - (\eta+\mu)I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} = (1-p)(1-\gamma_k)k[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - (\eta+\mu)I_{2k}(t), \\ \frac{dR_k(t)}{dt} = \eta I_{1k}(t) + \eta I_{2k}(t) - (\delta+\mu)R_k(t). \end{cases} \tag{17}$$

which leads to

$$\Theta = \hat{f}_1(\Theta)\hat{f}_2(\Theta) \equiv \hat{f}(\Theta),$$

where

$$\hat{f}_1(\Theta) = \frac{1}{\langle k\rangle}\sum_{k=1}^{\infty}\frac{\alpha k P(k)[p\beta_1 + (1-p)\beta_2]}{1-p},$$

$$\hat{f}_2(\Theta) = \frac{k\varepsilon b(1-p)(1-\gamma_k)\Theta(\mu+\delta)[\xi+\mu+k(1-\gamma_k)\Theta]}{\hat{g}(\Theta)},$$

where $\hat{g}(\Theta) = (b+\mu)(\eta+\mu)(\delta+\mu)(\xi+\mu) + (b+\mu)[\varepsilon(\mu+\delta+\eta)][k(1-\gamma_k)\Theta]^2 + (b+\mu)[(\eta+\mu)(\delta+\mu) - (1-\varepsilon)q\delta\eta + \varepsilon(\xi+\mu)(\delta+\mu+\eta)]k(1-\gamma_k)\Theta$. The epidemic threshold is determined by the following inequality:

$$\frac{d\hat{f}(\Theta)}{d\Theta}|_{\Theta=0} = \frac{\alpha b\varepsilon[p\beta_1 + (1-p)\beta_2]}{(b+\mu)(\eta+\mu)}\frac{\langle k^2\rangle - \langle\gamma_k k^2\rangle}{\langle k\rangle} > 1.$$

Therefore, the epidemic threshold

$$\check{R_0} = \frac{\alpha b\varepsilon[p\beta_1 + (1-p)\beta_2]}{(b+\mu)(\eta+\mu)} \cdot \frac{\langle k^2\rangle - \langle\gamma_k k^2\rangle}{\langle k\rangle}.$$

Note that

$$\langle\gamma_k k^2\rangle \quad = \quad \langle\gamma_k\rangle \cdot \langle k^2\rangle + cov(\gamma_k, k^2)$$

$$\begin{aligned} &= \bar{\gamma} \cdot \langle k^2 \rangle + \langle (\gamma_k - \langle \gamma_k \rangle) \cdot (k^2 - \langle k^2 \rangle) \rangle \\ &= \bar{\gamma} \cdot \langle k^2 \rangle + \langle (\gamma_k - \bar{\gamma}) \cdot (k^2 - \langle k^2 \rangle) \rangle. \end{aligned}$$

For appropriate k, $\gamma_k - \bar{\gamma}$ and $k^2 - \langle k^2 \rangle$ have the same signs, except for some $k's$ where $\gamma_k = \bar{\gamma}$ and/or $k^2 = \langle k^2 \rangle$, so $cov(\gamma_k, k^2) > 0$. Obviously, we can get that $\check{R}_0 < R_0$, which means the targeted immunization is effective. We can easily get that $\check{R}_0 < \frac{1-\bar{\gamma}}{1-\gamma}\hat{R}_0$. If we set $0 < \bar{\gamma} = \gamma < 1$, then $\check{R}_0 < \hat{R}_0$, which means the targeted immunization strategy is more efficient than the proportional strategy for the same average immunization rate. When $\check{R}_0 < 1$, STDs can be controlled by the targeted immunization.

Since the usage of condom p is the only controllable parameter in system (2), so as the end of this section, we would like to discuss the effective condom using rate to control SDTs spread on scale-free network for a given immunization rate (i.e., γ or k_t are given). Suppose $R_0 > 1$. Let

$$\tilde{p} = \frac{(b+\mu)(\eta+\mu)\langle k \rangle - \beta_2 \alpha b \varepsilon \langle k^2 \rangle}{\alpha b \varepsilon (\beta_1 - \beta_2)\langle k^2 \rangle},$$

$$\hat{p} = \frac{(b+\mu)(\eta+\mu)\langle k \rangle - \alpha b \varepsilon \beta_2 (1-\gamma)\langle k^2 \rangle}{\alpha b \varepsilon (1-\gamma)(\beta_1 - \beta_2)\langle k^2 \rangle},$$

$$\check{p} = \frac{(b+\mu)(\eta+\mu)\langle k \rangle - \alpha b \varepsilon \beta_2 [\langle k^2 \rangle - \langle \gamma_k k^2 \rangle]}{\alpha b \varepsilon (\beta_1 - \beta_2)[\langle k^2 \rangle - \langle \gamma_k k^2 \rangle]}.$$

When $(1-p) < \tilde{p}$, which means that STDs can be controlled in the network without immunization. When $(1-p) < \hat{p}$, which means that STDs can be controlled in the network under proportional immunization with immunization rate γ. When $(1-p) < \check{p}$, which means that STDs can be controlled in the network under targeted immunization with immunization rate γ_k.

5. Conclusion and Discussion

In this paper, a new model for the spread of the sexually transmitted diseases on complex networks has been proposed. From a mathematical aspect, such $S_1S_2I_1I_2RS$-type models on networks can be viewed as an multi-type $SIRS$ models if the networks possess the bounded degree property. Different from the classical epidemic model, in the new model the susceptible individuals and the infective individuals all divided into two subgroup. The thresholds of the STDs model are determined. We also got the stability of disease-free equilibrium and the persistence of STDs infection. At the same time, we have also discussed proportional and targeted immunization strategies to the STDs model. By comparing the thresholds for different immunization schemes, we have concluded that the targeted immunization strategy is more effective on the scale-free networks. We suggest to use target immunization scheme to decrease the spread of STDs. The $S_1S_2I_1I_2RS$-type model can be used to analysis other epidemic diseases.

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Maoxing Liu received M. Sc. from Xi'an Jiaotong University and Ph.D at Fudan University. Since 2003 he has been at North University of China. His research interests include epidemic dynamical systems and biological networks.

Department of Mathematics, North University of China, Taiyuan 030051, P. R. China.
e-mail: liumaoxing@126.com

Yunli Zhang received M. Sc. from North University of China. Her research interests are epidemic models and complex networks.

Department of Mathematics, North University of China, Taiyuan 030051, P. R. China.
e-mail: zhangyunli5365@sina.com