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AN SIRS MODEL WITH DIFFERENTIAL SUSCEPTIBILITY AND INFECTIVITY ON UNCORRELATED NETWORKS

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Abstract. We propose and study a model for sexually transmitted infections on uncorrelated networks, where both differential susceptibility and infectivity are considered. We first establish the spreading threshold, which exists even in the infinite networks. Moreover, it is possible to have backward bifurcation. Then for bounded hard-cutoff networks, the stability of the disease-free equilibrium and the permanence of infection are analyzed. Finally, the effects of two immunization strategies are compared. It turns out that, generally, the targeted immunization is better than the proportional immunization.

1. **Introduction.** The term STI (sexually transmitted infection) is now commonly used in place of STD (sexually transmitted disease) as it is more encompassing. STIs are infections that are spread primarily through person-to-person sexual contacts. Despite concerted efforts to control STIs worldwide, they still remain a major public health problem in all population groups and social strata. Over the past two decades, agents such as Chlamydia trachomatis and hepatitis B have been newly recognized as important sexually transmitted pathogens, whereas others such as the genital herpesvirus and Neisseria gonorrhoeae have increased in prevalence.

In order to better understand the epidemiology of STIs, researchers have developed a number of deterministic models. For example, models on complex networks have been extensively studied in recent years (see the review paper by Dorogovtsev et al. [4]). In network models, the population is represented as a graph, where individuals are nodes and partnerships are the edges connecting the nodes. These

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models can describe the fact that different individuals have different sexual behavior.

For simplicity, the networks involved in most study are uncorrelated. Let $P(k)$ be the probability of a randomly chosen node having a degree k. A network is uncorrelated if the conditional probability for a node with degree k to connect a node with degree h, $P(h|k)$, satisfies $P(h|k) = hP(h)/\langle k\rangle$, where $\langle k\rangle = \sum_k kP(k)$ is the average number of edges that a node has or is the first moment of degree k. It is always assumed that $\langle k\rangle < \infty$.

Moreover, the network of sexual partners in humans is claimed to be a scale-free network. In a scale-free network, $P(k)$ follows a power-law distribution, $P(k) \sim k^{-2-\gamma}$, with $0 < \gamma \leq 1$. Data from national sex surveys [12, 22] provide quantitative information on the number of sexual partners, that is, 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 scale-free distributions [12, 17]. As a result, a lot of attention has been paid to epidemic models on uncorrelated scale-free networks (see, for example, [5, 13, 18, 21, 27, 28, 29, 30] and the references therein).

As we know, generic variation of susceptible individuals may lead to their differentiation of susceptibility on infection [23]. The efficacy of available vaccinations for infectious diseases is not perfect. Vaccinated individuals may still contract the disease and the susceptibility varies from individuals to individuals. Moreover, existing studies support that there is variability also in the infectivity among infected individuals (see, for example, [1, 3, 8] and the references therein). Though much has been done to describe such phenomena with multigroup SIR models (to name a few, see [3, 7, 8, 9, 14]), little has been done in the context of complex networks.

Only recently, Lou and Ruggeri [13] studied the dynamics of STIs with differential infectivity by a multiple SIRS model on scale-free networks. They showed that, for an infinite scale-free network, the epidemic processes do not possess an epidemic threshold like the results in SIS and SIR models [16, 18]. A threshold phenomenon means that there is a threshold such that above it there is an epidemic outbreak while below it there is not [15, pp. 321]. In [16, 18], only the existence of equilibria is studied. However, realistic systems are just made up by a finite number of individuals. When a bounded hard-cutoff network is considered, the models can be regarded as multigroup models [10, 24]. Consequently, Lou and Ruggeri also discussed the stability of equilibria and the permanence of infection on a bounded hard-cutoff scale-free network, which does not possess any node with degree larger than k_c [18].

The purpose of this paper is to further study the spreading of STIs on uncorrelated networks. We propose an SIRS model with differential susceptibility and infectivity on uncorrelated networks. Roughly speaking, we shall divide the individuals into individuals with lower susceptibility, higher susceptibility, lower infectivity, higher infectivity, and recovered. Thus we shall get an $S_1S_2I_1I_2RS$ model for STIs.

The remaining of this paper is organized as follows. We first propose the model in Section 2. Section 3 is devoted to the existence of equilibria, followed by the stability of the disease-free equilibrium and the permanence of infection in the context of bounded hard-cutoff networks. 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 [13]. Then in Section 4, we discuss and compare the effect of two immunization strategies. Numerical simulations are provided to support the obtained results in Section 5, where the sensitivity analysis of the basic reproduction number on model parameters is also carried out. The paper concludes with a brief discussion.

We should mention that the presentation of this paper is parallel to that of Lou and Ruggeri [13]. However, in [13], only scale-free networks are considered and the infectivity of a node is proportional to its degree. As we will see, for the general case, some different results can be obtained. Such results include the threshold dynamics even in the infinite uncorrelated networks and back forward bifurcation. Moreover, some approaches like those for the permanence of infection are quite different from those of Lou and Ruggeri [13]

2. **The model.** The purpose of this section is to describe our model on STIs with differential susceptibility and infectivity on uncorrelated networks.

We consider a population with two types of susceptible individuals (one with higher susceptibility while the other with lower susceptibility) and two types of infected individuals (one with smaller infection rate β_1 while the other with larger infection rate β_2). Let S_{1k}, S_{2k}, I_{1k}, I_{2k}, and R_k represent the relative densities of nodes of degree k with higher susceptibility, lower susceptibility, smaller infectivity, higher infectivity, and recovered, respectively. We then have the normalization condition,

$$S_{1k} + S_{2k} + I_{1k} + I_{2k} + R_k = 1.$$

The mean-field theory can be used to derive the following deterministic model

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

where

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

System (1), combined with (2) and the initial conditions $S_{1k}(0) = S_{1k}^0$, $S_{2k}(0) = S_{2k}^0$, $I_{1k}(0) = I_{1k}^0$, $I_{2k}(0) = I_{2k}^0$, and $R_k(0) = 1 - S_{1k}^0 - S_{2k}^0 - I_{1k}^0 - I_{2k}^0$, completely defines the $S_1S_2I_1I_2RS$ model on an uncorrelated network with degree distribution $P(k)$. We only consider the situation that $k \geq 1$ since the probability that a node of connectivity k is connected to an isolated node is zero.

The meanings for the parameters and terms of (1) are as follows.

1. The parameter δ represents the rate of immunization-lost for recovered individuals. Recovered individuals become susceptible after time span $1/\delta$. For those recovered individuals who lose immunization, a portion q of them enters S_{1k} and the other portion enters S_{2k}.
2. The severeness of the infection is represented by $\Theta(t)$. Here the function $\psi(k)$ in $\Theta(t)$ denotes the infectivity of a node with degree k. Two commonly used forms are $\psi(k) = \alpha k$ [18] and $\psi(k) = A$ [26]. Parameters β_1 and β_2 are the STI transmission rates for groups I_{1k} and I_{2k}, respectively. The parameter ε represents the chance of S_{2k} being infected compared with S_{1k}.

3. The parameter ξ represents the transfer rate from the group of higher susceptibility to the group of lower susceptibility. This is possible, for example, individuals without self-protection awareness may have strong self-protection awareness due to the information they get about the spreading of STIs.
4. The parameter p is the portion of the individuals, who lose susceptibility because of infection, enters I_{1k}.
5. The parameter η is the recovery rate of infected individuals, *i.e.*, infected individuals recover from STIs after time span $1/\eta$.

All the parameters in (1) are non-negative. According to their biological meanings, we have $\beta_2 > \beta_1 > 0$, $\delta > 0$, $\xi > 0$, $\eta > 0$, $p \in (0,1)$, $q \in [0,1/2]$, and $\varepsilon \in (0,1)$. If $p = 1$ is allowed then eventually there is only the infected group I_{1k} while if $p = 0$ is allowed then eventually there is only the infected group I_{2k}. Moreover, if $\varepsilon = 1$ is allowed then there is no susceptibility difference among susceptible individuals and (1) reduces to the model studied by Lou and Ruggeri [13].

3. Some results.

3.1. The threshold R_0.

In this subsection, we discuss the existence of equilibria of (1).

Theorem 3.1. (i) *System* (1) *always has a disease-free equilibrium* $E^0 = (0,1,0,0,0)$.

(ii) *Suppose that* $(\delta+\eta)\xi\varepsilon \geq q\delta\eta(1-\varepsilon)$. *Then* (1) *has an endemic equilibrium if and only if* $R_0 > 1$ *and in this case there is only one endemic equilibrium, where*

$$R_0 = \frac{\varepsilon[p\beta_1 + (1-p)\beta_2]}{\eta}\frac{\langle k\psi(k)\rangle}{\langle k\rangle}. \tag{3}$$

(iii) *Suppose that* $(\delta+\eta)\xi\varepsilon < q\delta\eta(1-\varepsilon)$. *Then* (1) *has a unique endemic equilibrium if* $R_0 \geq 1$.

Proof. Statement (i) is obvious.

Now, assume that $E^k = (S_{1k}, S_{2k}, I_{1k}, I_{2k}, R_k)$ is an endemic equilibrium of (1), that is, there exists a $k_0 \geq 1$ such that at least one of I_{1k_0} and I_{2k_0} is not zero. Setting the right hand sides of the equations in (1) to be zero yields

$$\begin{aligned}
q\delta R_k - kS_{1k}\Theta - \xi S_{1k} &= 0, &(4)\\
(1-q)\delta R_k + \xi S_{1k} - k\varepsilon S_{2k}\Theta &= 0, &(5)\\
pk[S_{1k} + \varepsilon S_{2k}]\Theta - \eta I_{1k} &= 0, &(6)\\
(1-p)k[S_{1k} + \varepsilon S_{2k}]\Theta - \eta I_{2k} &= 0, &(7)\\
\eta(I_{1k} + I_{2k}) - \delta R_k &= 0, &(8)
\end{aligned}$$

where

$$\Theta = \frac{1}{\langle k\rangle}\sum_{k=1}^{\infty}\psi(k)P(k)[\beta_1 I_{1k} + \beta_2 I_{2k}]. \tag{9}$$

It follows that $\Theta \neq 0$ otherwise we can easily obtain that $I_{1k} = I_{2k} = R_k = S_{1k} = 0$ and this is a contradiction to the assumption that the equilibrium is endemic. It is easy to see from (6) and (7) that

$$I_{1k} = \frac{p}{1-p}I_{2k}. \tag{10}$$

Substituting this into (8) gives us

$$R_k = \frac{\eta(I_{1k} + I_{2k})}{\delta} = \frac{\eta}{\delta(1-p)} I_{2k}. \tag{11}$$

This, combined with (4), produces

$$S_{1k} = \frac{q\delta R_k}{\xi + k\Theta} = \frac{q\eta}{(1-p)(\xi + k\Theta)} I_{2k}. \tag{12}$$

Adding (4) and (5) gives us $\delta R_k - kS_{1k}\Theta - k\varepsilon S_{2k}\Theta = 0$. This, combined with (11) and (12), yields

$$S_{2k} = \frac{\delta R_k - kS_{1k}\Theta}{k\varepsilon\Theta} = \frac{\eta(\xi + k\Theta - kq\Theta)}{(1-p)k\varepsilon\Theta(\xi + k\Theta)} I_{2k}. \tag{13}$$

Recall that $1 = S_{1k} + S_{2k} + I_{1k} + I_{2k} + R_k$. Substituting (10)–(13) into this identity, we obtain

$$I_{2k} = \frac{\delta(1-p)k\varepsilon\Theta(\xi + k\Theta)}{(\delta+\eta)\varepsilon(k\Theta)^2 + [\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta) + \delta\eta\xi}. \tag{14}$$

Substituting (10) and (14) into (9) gives us

$$\Theta = \frac{\delta\varepsilon\Theta[p\beta_1 + (1-p)\beta_2]}{\langle k\rangle} \sum_{k=1}^{\infty} k\psi(k)P(k)\Delta(k,\Theta),$$

where $\Delta(k,\Theta) = \frac{\xi + k\Theta}{(\delta+\eta)\varepsilon(k\Theta)^2 + [\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta) + \delta\eta\xi}$. Since $\Theta \neq 0$, it follows that Θ satisfies

$$g(\Theta) := 1 - \frac{\delta\varepsilon[p\beta_1 + (1-p)\beta_2]}{\langle k\rangle} \sum_{k=1}^{\infty} k\psi(k)P(k)\Delta(k,\Theta) = 0. \tag{15}$$

Note that

$$\frac{\partial\Delta(k,\Theta)}{\partial\Theta} = -\frac{k[(\delta+\eta)\varepsilon(k\Theta)^2 + 2(\delta+\eta)\xi\varepsilon k\Theta + \xi[(\delta+\eta)\xi\varepsilon - q\eta\delta(1-\varepsilon)]}{\{(\delta+\eta)\varepsilon(k\Theta)^2 + [\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta) + \delta\eta\xi\}^2}.$$

We first prove (ii). It follows from $(\delta+\eta)\xi\varepsilon \geq q\delta\eta(1-\varepsilon)$ that $\frac{\partial\Delta(k,\Theta)}{\partial\Theta} < 0$ for all $k \geq 1$ and $\Theta > 0$. Then

$$\frac{dg(\Theta)}{d\Theta} > 0 \qquad \text{for } \Theta > 0.$$

Note $g(0) = 1 - R_0$ and $\lim_{\Theta\to\infty} g(\Theta) = 1$. Equation (15) has a positive solution if and only if $R_0 > 1$ and in this case the solution is unique, which will produce a unique endemic equilibrium of (1).

We now come to prove (iii). We rewrite $\frac{\partial\Delta(k,\Theta)}{\partial\Theta}$ as

$$\frac{\partial\Delta(k,\Theta)}{\partial\Theta} = -k - \frac{k\{[(\delta+\eta)\xi\varepsilon + q\delta\eta(1-\varepsilon) - \delta\eta]k\Theta + \xi[(\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon) - \delta\eta]\}}{\{(\delta+\eta)\varepsilon(k\Theta)^2 + [\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta) + \delta\eta\xi\}^2}.$$

Notice that

$$\begin{aligned}
(\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon) - \delta\eta &< (\delta+\eta)\xi\varepsilon + q\delta\eta(1-\varepsilon) - \delta\eta \\
&< 2q\delta\eta(1-\varepsilon) - \delta\eta \\
&\leq \delta\eta(1-\varepsilon) - \delta\eta \qquad (\text{as } q \leq 1/2) \\
&= -\delta\eta\varepsilon < 0
\end{aligned}$$

and

$$\begin{aligned}
& [(\delta+\eta)\xi\varepsilon + q\delta\eta(1-\varepsilon) - \delta\eta]\delta\eta\xi \\
& -2\xi[(\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon) - \delta\eta][\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)] \\
> \; & [(\delta+\eta)\xi\varepsilon + q\delta\eta(1-\varepsilon) - \delta\eta]\delta\eta\xi \\
& -\xi[(\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon) - \delta\eta][\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)] \\
= \; & \xi[q(\delta\eta)^2(1-\varepsilon) - q^2(\delta\eta)^2(1-\varepsilon)^2 + 2(\delta+\eta)q\delta\eta\xi\varepsilon(1-\varepsilon) \\
& +(\delta+\eta)\delta\eta\xi\varepsilon - (\delta+\eta)^2(\xi\varepsilon)^2] \\
> \; & \xi[q(\delta\eta)^2(1-\varepsilon) - q^2(\delta\eta)^2(1-\varepsilon)^2 + 2(\delta+\eta)^2(\xi\varepsilon)^2 \\
& +(\delta+\eta)\delta\eta\xi\varepsilon - (\delta+\eta)^2(\xi\varepsilon)^2] \qquad (\text{as } (\delta+\eta)\xi\varepsilon < q\delta\eta(1-\varepsilon)) \\
> \; & 0.
\end{aligned}$$

We can easily check that $\frac{\partial^2\Delta(k,\Theta)}{\partial\Theta^2} < 0$ for all $k \geq 1$ and $\Theta > 0$. It follows that $\frac{d^2g(\Theta)}{d\Theta^2} > 0$ for all $k \geq 1$ and $\Theta > 0$. Recall that $g(0) = 1 - R_0$, $\lim_{\Theta\to\infty} g(\Theta) = 1$, and $\frac{dg(0)}{d\Theta} < 0$. Therefore, if $R_0 \geq 1$ then (15) has a unique positive solution. This finishes the proof. □

Let us end this subsection with some remarks.

Remark 1. (i) In Theorem 3.1, we derived the reproduction number R_0 for general complex networks and general infectivity functions. From the expression of R_0 in (3), it is possible to have epidemic threshold in some networks. For example if $\psi(k) = A$ is a constant, then we have an epidemic threshold $R_0 = \frac{A\varepsilon[p\beta_1+(1-p)\beta_2]}{\eta}$. However, in a scale-free network with $\psi(k) = \alpha k$, $R_0 = \infty$ and hence the epidemic processes of our model do not possess an epidemic threshold as in the model of Lou and Ruggeri [13].

(ii) The condition $(\delta+\eta)\xi\varepsilon \geq q\delta\eta(1-\varepsilon)$ is not very restrictive. For example if q is very small, that is, most of the recovered with immunization loss enter the class of susceptible with lower susceptibility, then this condition may easily hold.

(iii) Suppose that there is an epidemic threshold. When $(\delta+\eta)\xi\varepsilon < q\delta\eta(1-\varepsilon)$ and $R_0 < 1$, system (1) can have at most two endemic equilibria since $g(\Theta)$ is convex. As shown below, each situation can occur. For this purpose, let us fix all parameters except η. For convenience, we denote $g(\Theta)$ as $g(\Theta,\eta)$. If $\eta = \eta_0 := \frac{\varepsilon[p\beta_1+(1-p)\beta_2]\langle k\psi(k)\rangle}{\langle k\rangle}$ then $R_0 = 1$. From the proof of Theorem 3.1 we know that there exists $\Theta_0 > 0$ such that $g(\Theta_0,\eta_0) = \min_{\Theta\in[0,\infty)} g(\Theta,\eta_0) < 0$. By the continuity of $g(\Theta,\eta)$ in (Θ,η), there exists a $\zeta \in (0,\eta_0)$ such that $g(\Theta_0,\eta) < g(\Theta_0,\eta_0)/2 < 0$ if $\eta \in (\eta_0-\zeta,\eta_0+\zeta)$. On the other hand, note that

$$\begin{aligned}
\Delta(k,\Theta) &= \frac{\xi}{(\delta+\eta)\varepsilon(k\Theta)^2 + [\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta) + \delta\eta\xi} \\
&\quad + \frac{k\Theta}{(\delta+\eta)\varepsilon(k\Theta)^2 + [\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta) + \delta\eta\xi} \\
&\leq \frac{\xi}{\delta\eta\xi} + \frac{k\Theta}{[\delta\eta + (\delta+\eta)\xi\varepsilon - q\delta\eta(1-\varepsilon)](k\Theta)} \\
&< \frac{1}{\delta\eta} + \frac{1}{(1-q)\delta\eta} = \frac{2-q}{(1-q)\delta\eta}.
\end{aligned}$$

It follows that $g(\Theta,\eta) > 1-\frac{2-q}{1-q}R_0$. This implies that $g(\Theta,\eta) > 0$ if $R_0 < \frac{1-q}{2-q}$ or equivalently if $\eta > \eta_2 := \frac{\varepsilon(2-q)[p\beta_1+(1-p)\beta_2]\langle k\psi(k)\rangle}{(1-q)\langle k\rangle} (> \eta_0)$. Notice that $g(\Theta,\eta)$ is increasing in η and convex in Θ. From the above discussion, we can easily see that there exists an $\eta_1 \in (\eta_0,\eta_2)$ such that

$$\min_{\Theta\in[0,\infty)} g(\Theta,\eta) \begin{cases} > 0, & \text{if } \eta > \eta_1, \\ = 0, & \text{if } \eta = \eta_1, \\ < 0, & \text{if } \eta \in (\eta_0,\eta_1). \end{cases}$$

Define $R_1 = \frac{\varepsilon[p\beta_1+(1-p)\beta_2]\langle k\psi(k)\rangle}{\eta_1\langle k\rangle}$. Then we have **a backward bifurcation**. Precisely, *suppose that* $(\delta+\eta)\xi\varepsilon < q\delta\eta(1-\varepsilon)$. *Then there exists an* $R_1 \in (0,1)$ *such that*

(a) *System* (1) *always has a disease-free equilibrium.*
(b) *If* $R_0 \geq 1$ *then* (1) *has a unique endemic equilibrium.*
(c) *If* $R_0 < R_1$ *then* (1) *has no endemic equilibrium; if* $R_0 = R_1$ *it has a unique equilibrium; if* $R_1 < R_0 < 1$ *then it has two endemic equilibria.*

We mention that with a slight modification similar result of backward bifurcation can be shown if we fix all parameters except one. We conjecture that this is true in the general case. However, it is not easy to prove this so far.

3.2. **Stability of the disease-free equilibrium and permanence of STIs on a bounded hard-cutoff network.** Real systems are actually made up by a finite number of individuals. This finite population introduces a maximum connectivity k_c. Consequently, we restrict our attention to a bounded hard-cutoff network [19]. In this case, the involved summation is from 1 to k_c and we also know that there is a threshold defined by (3).

Theorem 3.2. (i) *The disease-free equilibrium* E^0 *is locally asymptotically stable when* $R_0 < 1$ *and unstable when* $R_0 > 1$.

(ii) *Suppose* $R_0 > 1$. *Then* (1) *is permanent of infection, that is, there exists a* $\gamma > 0$ *such that*

$$\liminf_{t\to\infty} I_{ij}(t) > \gamma, \qquad i = 1,2, j = 1,2,\ldots,k_c,$$

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

Proof. Since $S_{1k} + S_{2k} + I_{1k} + I_{2k} + R_k = 1$, we only need to study the stability of the trivial equilibrium of

$$\begin{cases} \frac{dS_{1k}(t)}{dt} &= q\delta R_k(t) - kS_{1k}(t)\Theta(t) - \xi S_{1k}(t), \\ \frac{dI_{1k}(t)}{dt} &= pk[S_{1k}(t) + \varepsilon(1 - S_{1k}(t) - I_{1k}(t) - I_{2k}(t) - R_k(t))]\Theta(t) - \eta I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} &= (1-p)k[S_{1k}(t) + \varepsilon(1 - S_{1k}(t) - I_{1k}(t) - I_{2k}(t) - R_k(t))]\Theta(t) - \eta I_{2k}(t), \\ \frac{dR_k(t)}{dt} &= \eta[I_{1k}(t) + I_{2k}(t)] - \delta R_k(t), \end{cases} \tag{16}$$

$k = 1, 2, \ldots, k_c$. Denote $q_j = \frac{\psi(j)P(j)}{\langle k\rangle}$ for $1 \leq j \leq k_c$. The Jacobian matrix of the trivial equilibrium of (16), which is a $4k_c \times 4k_c$ matrix, is

$$J = (A_{ij})_{k_c\times k_c},$$

where

$$A_{ij} = \begin{pmatrix} 0 & -iS_{1i}q_j\alpha\beta_1 & -iS_{1i}q_j\alpha\beta_2 & 0 \\ 0 & ip(S_{1i}+\varepsilon S_{2i})q_j\alpha\beta_1 & ip(S_{1i}+\varepsilon S_{2i})q_j\alpha\beta_2 & 0 \\ 0 & i(1-p)(S_{1i}+\varepsilon S_{2i})q_j\alpha\beta_1 & i(1-p)(S_{1i}+\varepsilon S_{2i})q_j\alpha\beta_2 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$
$$+\delta_{ij}\begin{pmatrix} -\xi & 0 & 0 & q\delta \\ 0 & -\eta & 0 & 0 \\ 0 & 0 & -\eta & 0 \\ 0 & \eta & \eta & -\delta \end{pmatrix}$$

with $\delta_{ij} = \begin{cases} 1 & \text{if } i = j, \\ 0 & \text{if } i \neq j. \end{cases}$ Using mathematical induction, we obtain the characteristic equation of J, $g_1 \cdot g_2 = 0$, where

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

and

$$g_2 = \lambda+\eta-\varepsilon[p\beta_1+(1-p)\beta_2]\frac{\langle k\psi(k)\rangle}{\langle k\rangle}.$$

It follows that the trivial equilibrium of (16) and hence the disease-free equilibrium E^0 of (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. This proves (i).

Next, we show the permanence. The argument is similar to that of Lemma 3.5 of De Leenheer and Smith [11], which is based on Theorem 4.6 of Thieme [25]. For simplicity, we denote

$$x = (S_{11}, S_{21}, I_{11}, I_{21}, R_1, \cdots, S_{1k_c}, S_{2k_c}, I_{1k_c}, I_{2k_c}, R_{k_c})$$

to be the state variable of (1) and $x(t)$ represents a solution of (1). Define

$$X = \{x \in \mathbb{R}_+^{5k_c} : S_{1k}+S_{2k}+I_{1k}+I_{2k}+R_k = 1, k = 1,2,\ldots,k_c\},$$
$$X_0 = \left\{x \in X : \sum_k \psi(k)P(k)I_{1k} > 0 \text{ or } \sum_k \psi(k)P(k)I_{2k} > 0\right\},$$

and

$$\partial X_0 = X \backslash X_0.$$

In the following, we show that (1) is uniformly persistent with respect to $(X_0, \partial X_0)$.

Obviously, X is positively invariant with respect to (1). We can also see that X_0 is positively invariant with respect to (1) since

$$\frac{d}{dt}(\sum_k \psi(k)P(k)I_{ik}(t)) \geq -\eta\sum_k \psi(k)P(k)I_{ik}(t), \qquad i = 1,2.$$

Furthermore, there exists a compact set B in which all solutions of (1) initiating in X will enter and remain there.

Denote

$$M_0 = \{x(0) \in \partial X_0 : x(t) \in \partial X_0, t \geq 0\}$$

and

$$\Omega = \bigcup\{\omega(x(t)) : x(0) \in M_0\}.$$

Restricting (1) on M_0 gives

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

Clearly, system (17) has a unique equilibrium E^0. It is easy to see from the first, third, fourth and fifth equations of (17) that all $S_{1k}(t)$, $I_{1k}(t)$, $I_{2k}(t)$, and $R_k(t)$ tend to zero as $t \to \infty$. Then $S_{2k}(t) \to 1$ as $t \to \infty$ since $S_{1k}(t) + S_{2k}(t) + I_{1k}(t) + I_{2k}(t) + R_k(t) = 1$. Therefore, $\Omega = E^0$.

Note that E^0 is a covering of Ω, which is isolated and is acyclic. To finish the proof, it suffices to show that E^0 is a weak repeller for X_0, that is

$$\limsup_{t\to\infty} \operatorname{dist}(x(t), E^0) > 0$$

where $x(t)$ is any arbitrary solution of (1) with $x(0) \in X_0$. We only need to prove $W^s(E^0) \bigcap X_0 = \emptyset$, where $W^s(E^0)$ is the stable manifold of E^0. By way of contradiction, suppose this is not true. Then there exists a solution $x(t) \in X_0$ such that $S_{1k}(t) \to 0$, $S_{2k}(t) \to 1$, $I_{1k}(t) \to 0$, $I_{2k}(t) \to 0$, and $R_k(t) \to 0$ as $t \to \infty$. Since $R_0 = \frac{\varepsilon[p\beta_1+(1-p)\beta_2]}{\eta} \frac{\langle k\psi(k)\rangle}{\langle k\rangle} > 1$, we can choose $\nu > 0$ such that $(1-\nu)R_0 > 1$. For such $\nu > 0$, there exists a $T > 0$ such that, for $t \geq T$, $0 \leq S_{1k}(t) < \nu$, $1 - \nu < S_{2k}(t) \leq 1$, $0 \leq I_{1k}(t) < \nu$, $0 \leq I_{2k}(t) < \nu$, and $0 \leq R_k(t) < \nu$. Let

$$V(t) = \sum_{k=1}^{k_c} \psi(k)P(k)(\beta_1 I_{1k}(t) + \beta_2 I_{2k}(t)).$$

In fact, $V(t) = \langle k\rangle\Theta(t)$. Then, for $t \geq T$, we have

$$\begin{aligned} \frac{dV(t)}{dt} &= \sum_{k=1}^{k_c} \psi(k)P(k)\left[\beta_1 \frac{dI_{1k}(t)}{dt} + \beta_2 \frac{dI_{2k}(t)}{dt}\right] \\ &= \sum_{k=1}^{k_c} \beta_1\psi(k)P(k)\left[pk(S_{1k}(t) + \varepsilon S_{2k}(t))\frac{V(t)}{\langle k\rangle} - \eta I_{1k}(t)\right] \\ &\quad + \sum_{k=1}^{k_c} \beta_2\psi(k)P(k)\left[(1-p)k(S_{1k}(t) + \varepsilon S_{2k}(t))\frac{V(t)}{\langle k\rangle} - \eta I_{2k}(t)\right] \\ &\geq \left[\frac{\varepsilon(1-\nu)[\beta_1 p + \beta_2(1-p)]\langle k\psi(k)\rangle}{\langle k\rangle} - \eta\right] V(t) \\ &= \eta[(1-\nu)R_0 - 1]V(t). \end{aligned}$$

Since $(1-\nu)R_0 > 1$, it follows immediately that $V(t) \to \infty$ as $t \to \infty$, which contradicts with the fact that $V(t)$ is bounded. This completes the proof. □

4. **Immunization strategies.** Vaccination is very helpful in controlling vaccine preventable diseases [20, 29]. In this section we discuss (1) with two immunization schemes: the proportional immunization and the targeted immunization.

4.1. **Proportional immunization.** Let γ be the immunization rate, $0 < \gamma < 1$. Then (1) becomes

$$\begin{cases} \frac{dS_{1k}(t)}{dt} &= q\delta R_k(t) - k(1-\gamma)S_{1k}(t)\Theta(t) - \xi S_{1k}(t), \\ \frac{dS_{2k}(t)}{dt} &= (1-q)\delta R_k(t) + \xi S_{1k}(t) - k\varepsilon(1-\gamma)S_{2k}(t)\Theta(t), \\ \frac{dI_{1k}(t)}{dt} &= pk(1-\gamma)[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - \eta I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} &= (1-p)k(1-\gamma)[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - \eta I_{2k}(t), \\ \frac{dR_k(t)}{dt} &= \eta[I_{1k}(t) + I_{2k}(t)] - \delta R_k(t). \end{cases} \tag{18}$$

Similar arguments as those in Section 3 give us the epidemic threshold

$$\hat{R}_0 = (1-\gamma)R_0. \tag{19}$$

To further the discussion, we assume that $(\delta+\eta)\xi\varepsilon \geq q\eta\delta(1-\varepsilon)$. Then similar arguments as those in the proof of Theorem 3.1 yields the following result.

Theorem 4.1. *Assume that $(\delta+\eta)\xi\varepsilon \geq q\delta\eta(1-\varepsilon)$. The following statements hold.*

(i) *System (18) always has a disease-free equilibrium.*
(ii) *System (18) has no endemic equilibrium if $\hat{R}_0 < 1$; otherwise, it has a unique endemic equilibrium.*

Now, assume that $R_0 \in [1, \infty)$, that is, there is an outbreak of the disease. Define

$$\gamma_c = 1 - \frac{1}{R_0}.$$

By Theorem 4.1 and (19), we can draw the following conclusions.

(i) If $\gamma \in (\gamma_c, 1]$ then $\hat{R}_0 < 1$ and hence the epidemic cannot spread in the network.
(ii) If $\gamma \in (0, \gamma_c)$ then $\hat{R}_0 \in (1, R_0)$. Though the immunization scheme is effective, it is not effective enough to control the spread of STIs.

4.2. **Targeted immunization.** While proportional immunization schemes are effective, there may be more efficient schemes due to the heterogeneous nature of complex networks: they are robust to random attacks, but fragile to selective attacks. Accordingly, we can devise a targeted immunization scheme [6], that is, the immunization rate γ depends on the degree k, denoted as $\gamma(k)$. Modifying (18) accordingly gives us

$$\begin{cases} \frac{dS_{1k}(t)}{dt} &= q\delta R_k(t) - k(1-\gamma(k))S_{1k}(t)\Theta(t) - \xi S_{1k}(t), \\ \frac{dS_{2k}(t)}{dt} &= (1-q)\delta R_k(t) + \xi S_{1k}(t) - k\varepsilon(1-\gamma(k))S_{2k}(t)\Theta(t), \\ \frac{dI_{1k}(t)}{dt} &= pk(1-\gamma(k))[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - \eta I_{1k}(t), \\ \frac{dI_{2k}(t)}{dt} &= (1-p)k(1-\gamma(k))[S_{1k}(t) + \varepsilon S_{2k}(t)]\Theta(t) - \eta I_{2k}(t), \\ \frac{dR_k(t)}{dt} &= \eta[I_{1k}(t) + I_{2k}(t)] - \delta R_k(t). \end{cases} \tag{20}$$

Again, with similar arguments as before, we can obtain the endemic threshold

$$\check{R}_0 = \frac{\varepsilon[p\beta_1 + (1-p)\beta_2]}{\eta} \frac{\langle k\psi(k)(1-\gamma(k))\rangle}{\langle k\rangle}. \tag{21}$$

It follows from (21) that (20) can possess an epidemic threshold even in the case where (1) does not. Recall that when (1) does not possess an epidemic threshold neither does (18). From the view of threshold, targeted immunization is better than the proportional immunization. Furthermore, let us assume that (1) has an

epidemic threshold. Obviously, $\check{R}_0 < R_0$, which implies that targeted immunization can be effective in controlling the spread of STIs. On the other hand, note that

$$\langle k\psi(k)(1-\gamma(k))\rangle = \langle k\psi(k)\rangle - \langle k\psi(k)\rangle\overline{\gamma} + \langle k\psi(k)(\overline{\gamma}-\gamma(k))\rangle,$$

where $\overline{\gamma} = \langle\gamma(k)\rangle$ is the average immunization rate. If $\langle k\psi(k)(\overline{\gamma}-\gamma(k))\rangle < 0$ then

$$\check{R}_0 \le (1-\overline{\gamma})R_0$$

and hence with the same immunization rate, $\gamma = \overline{\gamma}$, the targeted immunization is more efficient than the proportional immunization.

5. **Numerical simulations and sensitivity analysis.** In this section, we not only present some numerical simulations to support the above-obtained theoretical results but also perform some sensitivity analysis of the basic reproduction number R_0 in terms of the model parameters. The simulations are based on a BA network with $\zeta = 2$, $m = 3$, and the maximal degree $k_c = 1000$. Hence $P(k) = (\zeta - 1)m^{\zeta-1}k^{-\zeta} = 3k^{-2}$. For details on generating BA networks, see, for instance, Barabási and Albert [2]. We also assume $\psi(k) = k$.

First, take $\beta_1 = 0.002$, $\beta_2 = 0.01$, $\xi = 0.01$, $\varepsilon = 0.9$, $\eta = 0.01$, $\delta = 0.1$, $p = 0.4$, and $q = 0.2$. Then $R_0 = 1.9417 > 1$. By Theorem 3.2, the disease will persist without immunization. Fig 1 shows the times series of some representative nodes

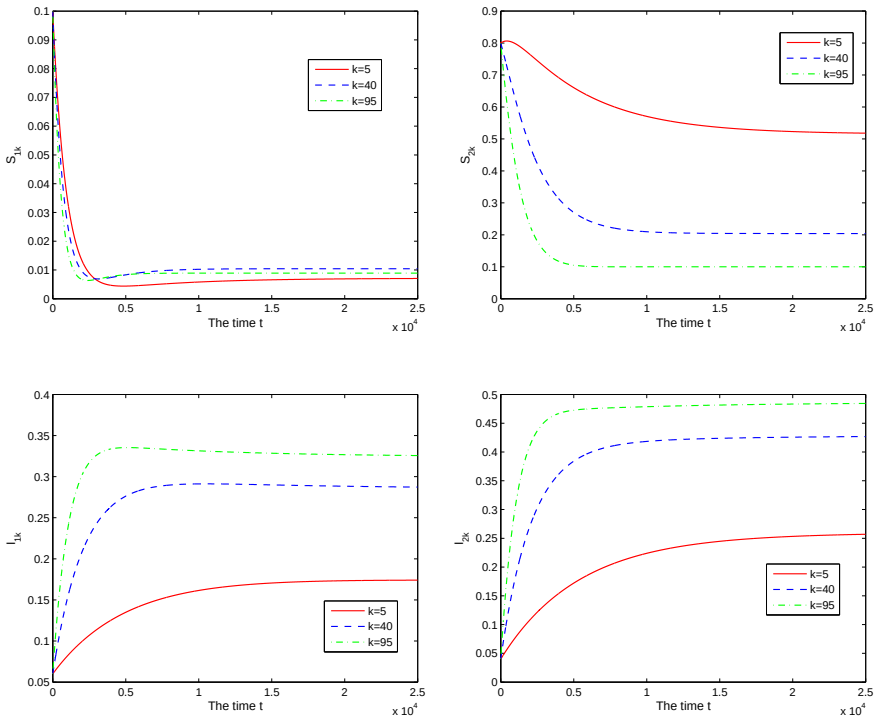

FIGURE 1. The time series of (1) for the representative nodes with degrees 5, 40, and 95

with degrees 5, 40, and 95. Obviously, the prevalence rate of I_{2k} is higher than that of I_{1k}. Interestingly, the difference between $k = 40$ and $k = 95$ is not very big.

Next, we illustrate the effect of ξ on the transmission dynamics. Take the same parameter values above except that of ξ. Fig 2 shows the time series of the node with

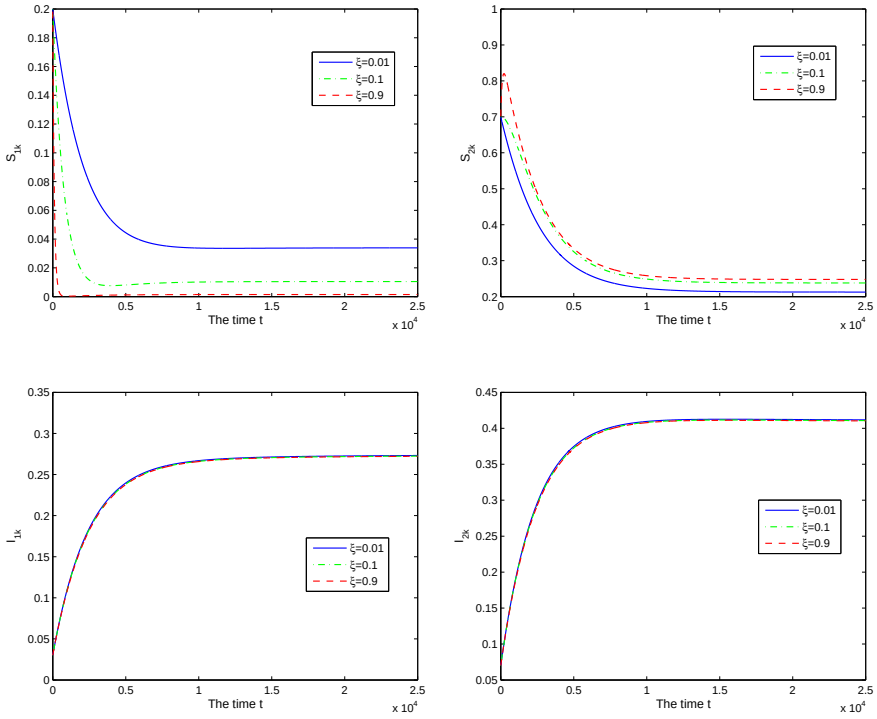

FIGURE 2. The time series of (1) of the node with degree $k = 40$ for $\xi = 0.01$, 0.1, and 0.9 respectively

degree 40 for $\xi = 0.01$, 0.1, and 0.9 respectively. We can see that the relative density of the higher susceptibility decreases with respect to ξ but the relative density of the lower susceptibility increases with respect to ξ. Moreover, ξ does not affect the relative densities of both higher infectivity and lower infectivity. This agrees with our intuition.

The theoretical analysis tells us that the basic reproduction number R_0 is an important quantity to characterize the transmission dynamics. The expression of R_0 clearly indicates that it is an increasing function of the transmission rates β_1 and β_2 and a decreasing function of the recovery rate η when the other parameters are fixed. With the same parameter values in the first case except those of (left) β_1 and η or (right) β_2 and η, Fig.3 indicates the combined impact of the transmission rates and the recovery rate on R_0.

Finally, we consider the effects of immunization with the same parameter values in the first case. We take $\gamma = 0.2$ in the proportional immunization and $\gamma(k) = \begin{cases} 1 & \text{if } k \geq k^* = 10, \\ 0 & \text{if } k \neq 10. \end{cases}$ in the targeted immunization. Fig. 4 depicts re-

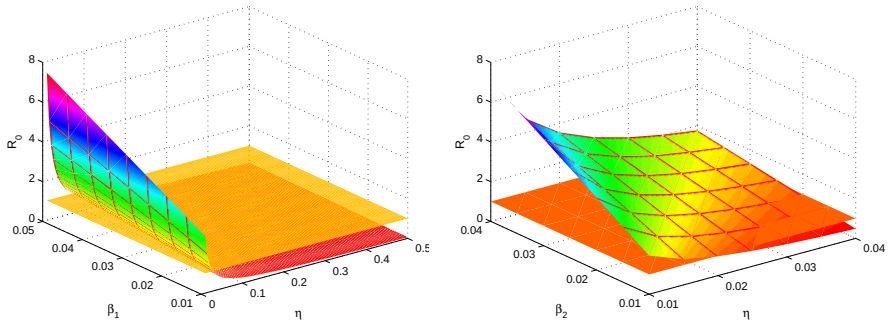

FIGURE 3. Combined impacts of the transmission rates (β_1 and β_2) and the recovery rate η on the basic reproduction number R_0

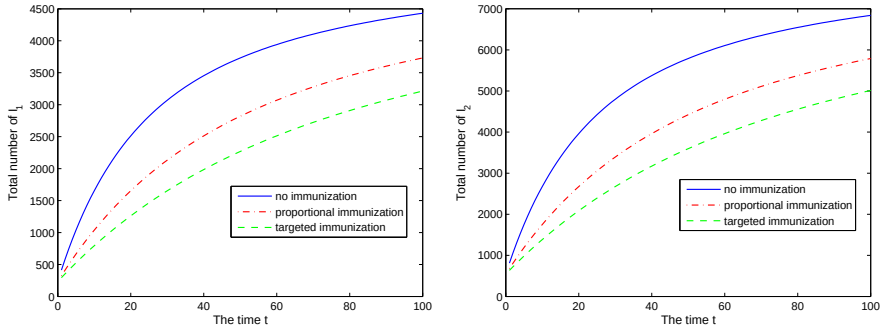

FIGURE 4. The time series of (1) with different immunization strategies

spectively the total numbers I_1 and I_2 of the smaller infectivity and the higher infectivity for the situations of no immunization, proportional immunization, and targeted immunization. Though both proportional immunization and the targeted immunization have the ability to control the disease spread, the targeted immunization seems to have the better effect.

6. **Discussion.** In this paper, we propose and study a model on the spread of STIs on uncorrelated networks. One feature of this model is that both differential susceptibility and infectivity are incorporated into it. The model includes the one studied by Lou and Ruggeri [13]. As in Lou and Ruggeri [13], we determine the spreading threshold. The results are more general and richer. For example, threshold may exist in infinite networks. Moreover, backward bifurcation may occur. Then for the bounded hard-cutoff networks, we study the stability of the disease free equilibrium and the permanence of infection. Finally, the effects of two immunization strategies, proportional immunization and targeted immunization, are compared. Generally,

targeted immunization has better effect than proportional immunization. We believe that the idea here can also be applied to study other epidemic diseases, such as HIV etc..

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REFERENCES

[1] M. Artzrouni, Transmission probabilities and reproductive numbers for sexually transmitted infections with variable infectivity: Application to the spread of HIV between low- and high-activity populations, *Mathematical Population Studies,* **16** (2009), 266–287.
[2] A.-L. Barabási and R. Alberta, Emergence of scaling in random networks, *Science,* **286** (1999), 509–512.
[3] B. Bonzi, A. A. Fall, A. Iggidr and G. Sallet, Stability of differential susceptibility and infectivity epidemic models, *J. Math. Biol.,* **62** (2011), 39–64.
[4] S. N. Dorogovtsev, A. V. Goltsev and J. F. F. Mendes, Critical phenomena in complex networks, *Rev. Mod. Phys.,* **80** (2008), 1275–1335.
[5] X. Fu, M. Small, D. M. Walker and H. Zhang, Epidemic dynamics on scale-free networks with piecewise linear infectivity and immunization, *Phys. Rev. E,* **77** (2008), 036113, 8pp.
[6] W.-P. Guo, X. Li and X.-F. Wang, Epidemics and immunization on Euclidean distance preferred small-world networks, *Physica A,* **380** (2007), 684–690.
[7] J. M. Hyman and J. Li, Differential susceptibility epidemic models, *J. Math. Biol.,* **50** (2005), 626–644.
[8] J. M. Hyman and J. Li, Differential susceptibility and infectivity epidemic models, *Math. Biosci. Engrg.,* **3** (2006), 89–100.
[9] J. M. Hyman and J. Li, Epidemic models with differential susceptibility and staged progression and their dynamics, *Math. Biosci. Engrg.,* **6** (2009), 321–332.
[10] A. Lajmanovich and J. A. Yorke, A deterministic model for gonorrhea in a nonhomogeneous population, *Math. Biosci.,* **28** (1976), 221–236.
[11] P. De Leenheer and H. L. Smith, Virus dynamics: A global analysis, *SIAM J. Appl. Math.,* **63** (2003), 1313–1327.
[12] F. Liljeros, C. R. Edling, L. A. N. Amaral, H. E. Stanley and Y. Åberg, The web of human sexual contacts, *Nature,* **411** (2001), 907–908.
[13] J. Lou and T. Ruggeri, The dynamics of spreading and immune strategies of sexually transmitted diseases on scale-free network, *J. Math. Anal. Appl.,* **365** (2010), 210–219.
[14] Z. Ma, J. Liu and J. Li, Stability analysis for differential infectivity epidemic models, *Nonlinear Anal.: RWA,* **4** (2003), 841–856.
[15] J. D. May, *Mathematical Biology I: An Introduction*, 3rd Ed., Springer-Verlag, New York, 2002.
[16] R. M. May and A. L. Lloyd, Infection dynamics on scale-free networks, *Phys. Rev. E,* **64** (2001), 066112.
[17] M. E. J. Newman, The structure and function of complex networks, *SIAM Rev.,* **45** (2003), 167–256.
[18] R. Pastor-Satorras and A. Vespignani, Epidemic spreading in scale-free networks, *Phys. Rev. Lett.,* **86** (2001), 3200–3203.
[19] R. Pastor-Satorras and A. Vespignani, Epidemic dynamics in finite size scale-free networks, *Phys. Rev. E,* **65** (2002), 035108.
[20] R. Pastor-Satorras and A. Vespignani, Immunization of complex networks, *Phys. Rev. E,* **65** (2002), 036104.
[21] W. J. Reed, A stochastic model for the spread of a sexually transmitted disease which results in a scale-free network, *Math. Biosci.,* **201** (2006), 3–14.
[22] A. Schneeberger, C. H. Mercer, S. A. J. Gregson, N. M. Ferguson, C. A. Nyamukapa, R. M. Anderson, A. M. Johmson and G. P. Garnett, Scale-free networks and sexually transmitted diseases: A description of observed patterns of sexual contacts in Britain and Zimbabwe, *Sex. Transm. Dis.,* **31** (2004), 380–387.
[23] A. Smed-Sörensen et al., Differential susceptibility to human immunodeficiency virus type 1 infection of myeloid and Plasmacytoid dendritic cells, *J. Virology,* **79** (2005), 8861–8869.

[24] N. Sugimine and K. Aihara, Stability of an equilibrium state in a multiinfectious-type SIS model on a truncated network, *Artif. Life Robotics,* **11** (2007), 157–161.
[25] H. R. Thieme, Persistence under relaxed point-dissipativity (with application to an endemic model), *SIAM J. Math. Anal.,* **24** (1993), 407–435.
[26] R. Yang et al., Epidemic spreading on heterogeneous networks with identical infectivity, *Phys. Lett. A,* **364** (2007), 189–193.
[27] H. Zhang, L. Chen and J. J. Nieto, A delayed epidemic model with stage-structure and pulses for pest management strategy, *Nonlinear Anal. Real World Appl.,* **9** (2008), 1714–1726.
[28] Z. Zhang and J. Peng, A SIRS epidemic model with infection-age dependence, *J. Math. Anal. Appl.,* **331** (2007), 1396–1414.
[29] Z. Zhang, J. Peng and J. Zhang, Analysis of a bacteria-immunity model with delay quorum sensing, *J. Math. Anal. Appl.,* **340** (2008), 102–115.
[30] S. Zou, J. Wu and Y. Chen, Multiple epidemic waves in delayed susceptible-infected-recovered models on complex networks, *Phys. Rev. E,* **83** (2011), 056121.

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