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Modelling the spread of sexually transmitted diseases on scale-free networks

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In this paper a new model for the spread of sexually transmitted diseases (STDs) is presented. The dynamic behaviors of the model on a heterogenous scale-free (SF) network are considered, where the absence of a threshold on the SF network is demonstrated, and the stability of the disease-free equilibrium is obtained. Three immunization strategies, uniform immunization, proportional immunization and targeted immunization, are applied in this model. Analytical and simulated results are given to show that the proportional immunization strategy in the model is effective on SF networks.

Keywords: epidemic modelling, scale-free networks, immunization, STDs spreading
PACC: 0250, 0590

1. Introduction

The study of epidemic models on complex networks has attracted a lot of attention and interest.[1−10] Within these studies, individuals are modeled as nodes, and possible contacts between individuals are linked by edges. It has been shown that there is an epidemic threshold on the homogeneous Watts–Strogatz (WS) small-world network model, while the epidemic model on SF networks has no epidemic threshold; infections can be proliferated, whatever small infection rate they have. This result disproves the threshold theory in epidemiology. In preventing and controlling infectious diseases, different kinds of immunization strategies have been presented. In WS homogeneous networks, by introducing uniform immunization, proportional immunization and targeted immunization, the eradication of infections could be easily achieved. In SF networks, random immunization where all nodes are immunized or vaccinated with an equal but finite probability becomes inefficient. Instead, targeted immunization where hubs are immunized preferentially proves to be efficient.[3,11]

Generally speaking, most SIS-like models only have one type of node-individuals, and the epidemic can only be transmitted by individual contacts. While in many human infectious diseases, such as malaria, yellow fever, and dengue fever, transmission is mediated by other hosts, such as mosquitoes. In these diseases, direct human-human, or mosquito-mosquito infection is absent, and the distinct populations transmit diseases between each other. Recently, there has been some related research on complex networks, for example, with heterogeneous susceptibility,[12] with infective media,[13] even with two species or pathogens spreading.[14,15] On the control of epidemic models on complex networks, Li *et al* have investigated local area control of virus spreading;[16] Chen and his co-workers have studied a dynamic epidemic control model on uncorrelated complex networks.[17] These studies have extended and detailed epidemic models on complex networks.

The spread of STDs, such as gonorrhea, chlamydia, syphilis and, of course, AIDS, is a major health problem in both developed and developing countries. STDs have certain characteristics which are different from other infections, such as measles or SARS. One difference is that they are mainly restricted to the sexually active community so the assumption of uniform mixing in the whole population is not really justified. It has been shown that networks of human sexual partners are scale-free,[18,19] that is the distribution of the number of contacts over all individuals in the network has a long upper tail which exhibits power-law behavior. Another difference is that STDs induce little or no

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acquired immunity following an infection. In Ref.[20], Zheng and Ergün have studied coupled growing networks, where they mentioned that the STDs model can be studied using two different coupled growing networks. In fact, we can put the different sex individuals into the same network, thus it is easy to analyze the dynamics of the model. To study such a spreading characteristic, this paper extends the standard model to qualitatively understand and describe the mechanism of epidemic spreading based on biological means. Furthermore, by applying three immunization strategies (uniform immunization, proportional immunization and targeted immunization) to the STDs model, it is shown that the main features and theoretical results obtained here are different from those in general SIS models.[2−4]

The organization of this paper is as follows: Section 2 derives the mean-field epidemic spreading model of a SF network, in particular, the 'Barabasi–Albert (BA)' model with connectivity $P(k) \sim k^{-3}$ is obtained, and the existence of the steady-state solution (endemic equilibrium) and the stability of the disease-free equilibrium are analyzed. Section 3 discusses the effect of three immunization strategies on the STDs model. Finally, a brief discussion is given in Section 4.

2. The STDs model

2.1. The model and the existence of equilibria

In this paper, there are two types of nodes in a network, one is composed of N male individuals and the other is composed of Ω female individuals. All nodes can only exist in one of the two states, susceptible or infected. And the disease transmission is described in the following way: At each time step, a susceptible male individual is infected with probability λ_1 if it is connected to an infected female individual, and infected individuals are cured and become susceptible individuals again with probability δ (without loss of generality, we set $\delta = 1$). On the other hand, each susceptible female individual is infected with probability λ_2 just like susceptible male individuals. Thus, all nodes in this network run stochastically through susceptible-infected-susceptible cycles. Here, the possibility of node removal due to death will not be taken into account. So we get the following STDs model:

$$\begin{aligned} \dot{x}_k(t) &= -x_k(t) + \lambda_1 k(1 - x_k(t))\Theta_y(t), \\ \dot{y}_k(t) &= -y_k(t) + \lambda_2 k(1 - y_k(t))\Theta_x(t), \end{aligned} \tag{1}$$

where $x_k(t)$ $(y_k(t))$ denotes the density of infected male (female) individuals with connectivity k in the networks, the probability $0 \leq \Theta_x(t)(\Theta_y(t)) \leq 1$ describes a link pointing to an infected male (female) individual, which satisfies

$$\begin{aligned} \Theta_x(t) &= \frac{1}{\langle k\rangle_x}\sum_k k x_k P_x(k), \\ \Theta_y(t) &= \frac{1}{\langle k\rangle_y}\sum_k k y_k P_y(k), \end{aligned} \tag{2}$$

where $\langle k\rangle_x = \sum_k kP_x(k), \langle k\rangle_y = \sum_k kP_y(k)$, $P_x(k)$ $(P_y(k))$ is the connectivity distribution of the male (female) individuals.

In the steady state

$$\begin{aligned} \dot{x}_k(t) &= 0, \\ \dot{y}_k(t) &= 0, \end{aligned} \tag{3}$$

one has

$$\begin{aligned} -x_k + \lambda_1 k(1 - x_k)\Theta_y &= 0, \\ -y_k + \lambda_2 k(1 - y_k)\Theta_x &= 0. \end{aligned} \tag{4}$$

Substituting Eq.(2) into Eq.(4), it is easy to see that model (1) has a disease-free equilibrium: $E_0 = (0, \ldots, 0)$. As far as the possibility of spreading is concerned, the following lemma holds.

Lemma 1 If and only if

$$\lambda_1\lambda_2\frac{\langle k^2\rangle_x\langle k^2\rangle_y}{\langle k\rangle_x\langle k\rangle_y} > 1,$$

there exists a unique endemic equilibrium $E^* = (x_1^*, \ldots .x_N^*, y_1^*, \ldots, y_\Omega^*)$.

Proof From Eq.(4), we can get

$$\begin{aligned} x_k &= \frac{\lambda_1 k\Theta_y}{1 + \lambda_1 k\Theta_y}, \\ y_k &= \frac{\lambda_2 k\Theta_x}{1 + \lambda_2 k\Theta_x}. \end{aligned} \tag{5}$$

And substituting them into Eq.(2), we get

$$\begin{aligned} \Theta_x &= \frac{1}{\langle k\rangle_x}\sum_k \frac{\lambda_1 k^2 P_x(k)\Theta_y}{1 + \lambda_1 k\Theta_y} \doteq f(\Theta_y), \\ \Theta_y &= \frac{1}{\langle k\rangle_y}\sum_k \frac{\lambda_2 k^2 P_y(k)\Theta_x}{1 + \lambda_2 k\Theta_x} \doteq g(\Theta_x). \end{aligned} \tag{6}$$

Thus

$$\begin{aligned} \Theta_x &= f(g(\Theta_x)), \\ \Theta_y &= g(f(\Theta_y)). \end{aligned} \tag{7}$$

For the first equation of Eq.(7), obviously, $\Theta_x = 0$ is a solution of Eq.(7), which corresponds to the disease-free equilibrium, and it is easy to verify that $f(g(\Theta_x))$ is a convex and increasing function, and $f(g(\Theta_x))|_{\Theta_x=1} < 1$, so if there is another solution $0 < \Theta_x < 1$, it must satisfy

$$\frac{\mathrm{d}f(g(\Theta_x))}{\mathrm{d}\Theta_x}\bigg|_{\Theta_x=0} > 1,$$

that is

$$\lambda_1\lambda_2\frac{\langle k^2\rangle_x\langle k^2\rangle_y}{\langle k\rangle_x\langle k\rangle_y} > 1,$$

where $\langle k^2\rangle_x = \sum_k k^2 P_x(k), \langle k^2\rangle_y = \sum_k k^2 P_y(k)$. In a similar way, we can find that the same condition for the second equation of Eq.(7) has a positive solution $0 < \Theta_y < 1$. Through Eq.(5) the unique endemic equilibrium E^* is determined.

Remark 1 (i) Let

$$R = \lambda_1\lambda_2\frac{\langle k^2\rangle_x\langle k^2\rangle_y}{\langle k\rangle_x\langle k\rangle_y}. \tag{8}$$

In mathematical biology, R is called the basic productive number, which determines whether the disease is transmittable or not.

(ii) If we consider

$$P_x(k) = P_y(k) = Ck^{-\upsilon}\delta_{k,k_c}(k_c \geq 2).$$

The network is homogeneous, $\langle k\rangle_x = \langle k\rangle_y = k_c$, $\langle k^2\rangle_x = \langle k^2\rangle_y = k_c^2$, so

$$R = \lambda_1\lambda_2 k_c^2. \tag{9}$$

When $R < 1$, the disease-free equilibrium is locally asymptotically stable, while for $R > 1$, there is an endemic equilibrium. Moreover, R is an increasing function of λ_1, λ_2 and k_c. In order to make $R < 1$, we need to decrease λ_1, λ_2 and k_c.

(iii) By using a continuous k approximation that allows a practical substitution of a series with integrals,[21] the full connectivity distribution can be obtained as $P_x(k) = P_y(k) = 2m^2/k^3$, where m is the minimum number of connections at each individual node, and $\langle k\rangle_x = \int_m^\infty kP_x(k) = 2m$, $\langle k\rangle_y = \int_m^\infty kP_y(k) = 2m$. Furthermore, here $\langle k^2\rangle_x \simeq 2m^2\ln(K_{xc}/m), K_{xc} \to \infty$, $\langle k^2\rangle_y \simeq 2m^2\ln(K_{yc}/m)$, $K_{yc} \to \infty$. Substituting them into Eq.(8) yields

$$R \simeq \lambda_1\lambda_2 m^2\ln(K_{xc}/m)\ln(K_{yc}/m). \tag{10}$$

If we consider the maximum connectivity K_{xc} to N by $K_{xc} \simeq mN^{1/2}$, K_{yc} to Ω by $K_{yc} \simeq m\Omega^{1/2}$,[22] so

$$R \simeq \frac{1}{4}\lambda_1\lambda_2 m^2\ln(N)\ln(\Omega). \tag{11}$$

2.2. The stability of the disease-free equilibrium

In this subsection we consider the stability of the disease-free equilibrium. The Jacobin matrix at disease-free equilibrium is

$$J = \begin{pmatrix} A & B \\ C & D \end{pmatrix}, \tag{12}$$

where

$$A = \begin{pmatrix} -1 & 0 & \cdots & 0 \\ 0 & -1 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & -1 \end{pmatrix}_{N\times N},$$

$$B = \frac{\lambda_1}{\langle k\rangle_y}\left(\begin{pmatrix} 1 \\ 2 \\ \vdots \\ N \end{pmatrix}\begin{pmatrix} 1\times P_y(1) & 2\times P_y(2) & \cdots & \Omega\times P_y(\Omega) \end{pmatrix}\right)_{N\times\Omega},$$

$$C = \frac{\lambda_2}{\langle k\rangle_x}\left(\begin{pmatrix} 1 \\ 2 \\ \vdots \\ \Omega \end{pmatrix}\begin{pmatrix} 1\times P_x(1) & 2\times P_x(2) & \cdots & N\times P_x(N) \end{pmatrix}\right)_{\Omega\times N},$$

$$D = \begin{pmatrix} -1 & 0 & \cdots & 0 \\ 0 & -1 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & -1 \end{pmatrix}_{\Omega\times\Omega}.$$

The matrix J has $N+\Omega-1$ eigenvalues equal to -1: $\mu_1, \cdots = \mu_{N+\Omega-1} = -1$, the $(N+\Omega)$-th is

$$\mu_{N+\Omega} = -1 + \lambda_1\lambda_2 \frac{\langle k^2\rangle_x \langle k^2\rangle_y}{\langle k\rangle_x \langle k\rangle_y}. \tag{13}$$

Having established these premises, we may immediately demonstrate the following theorem.

Theorem 1 If $R \leq 1$, then the disease-free equilibrium of Eq.(1) is asymptotically stable in the set $[0,1]^{N+\Omega}$, otherwise there exists a unique endemic equilibrium $E^* = (x_1^*, \ldots, x_N^*, y_1^*, \ldots, y_\Omega^*)$.

Remark 2 The biological consequence of this result is that if the epidemic threshold $R = 1$ is not exceeded the disease will disappear. On the contrary if $R > 1$, then there is an endemic solution which is reached independently of the initial state of the disease. In other words, however complex the system may be and whatever the initial state of the male and the female individuals are, whether the disease will be eradicated or not only depends on the basic reproductive number R.

3. Immunization strategies

3.1. Uniform immunization

In this case, for fixed spreading rates λ_1 and λ_2, let g be the density of immune nodes in the network. At the mean-field level, the presence of uniform immunity will effectively reduce the spreading rate λ by a factor $\lambda(1-g)$. Thus we can approximatively use $\lambda_1(1-g)$ to substitute λ_1 and use $\lambda_2(1-g)$ to substitute λ_2, thus the uniform immunization system is as follows:

$$\begin{aligned} \dot{x}_k(t) &= -x_k(t) + \lambda_1(1-g)k(1-x_k(t))\Theta_y(t), \\ \dot{y}_k(t) &= -y_k(t) + \lambda_2(1-g)k(1-y_k(t))\Theta_x(t). \end{aligned} \tag{14}$$

Using the same method as in subsection 2.1, we can see that the stationary condition satisfies

$$\lambda_1\lambda_2(1-g)^2 \frac{\langle k\rangle_x \langle k\rangle_y}{\langle k^2\rangle_x \langle k^2\rangle_y} = 1. \tag{15}$$

So that

$$g = 1 - \sqrt{\frac{\langle k\rangle_x \langle k\rangle_y}{\lambda_1\lambda_2\langle k^2\rangle_x \langle k^2\rangle_y}}. \tag{16}$$

In an infinite SF network, $\langle k^2\rangle_x \to \infty$, $\langle k^2\rangle_y \to \infty$, so $g \to 1$. Hence, uniform immunization is useless. On the other hand, in a finite SF network, in particular the BA model,

$$g \simeq 1 - \frac{4}{\sqrt{\lambda_1\lambda_2} m^2 \ln N \ln \Omega}. \tag{17}$$

For fixed infectivities λ_1 and λ_2, this implies that the larger the network size, the more nodes are needed to be immunized.

As shown in Fig.1, in the STDs model on SF networks, one also has to immunize most male and female individuals with increasing λ_1 and λ_2, it is easy to find that for however small infectivity, we need to immunize most individuals in order to eliminate the epidemic. Hence, the uniform immunization strategy is ineffective for scale-free networks.

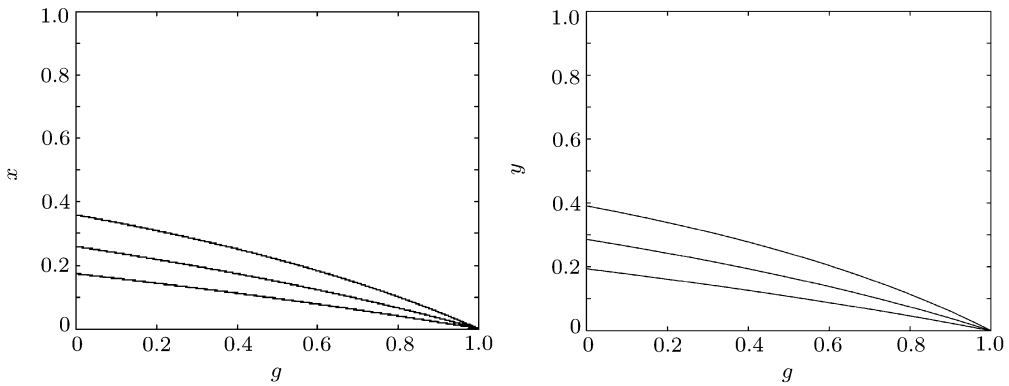


Fig.1. Densities of infected male and female individuals as functions of the density g of immune nodes in a scale-free network. Infected rates $\lambda_1 = \lambda_2 = 0.3, 0.5, 0.8$ (from bottom to up), with $m = 2$, $N = 2000$, $\Omega = 1500$.

3.2. Proportional immunization

Although uniform immunization is ineffective on SF networks, it is still meaningful to look specifically for other immunization strategies.[23−25] The next two sections consider proportional immunization and targeted immunization on SF networks, respectively.

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

$$\lambda_1 k(1-g_k)=\lambda_1',$$
$$\lambda_2 k(1-g_k)=\lambda_2',$$

where λ_1', λ_2' are constant. Then, system (1) yields

$$\dot{x}_k(t)=-x_k(t)+\lambda_1'(1-x_k(t))\Theta_y(t),$$
$$\dot{y}_k(t)=-y_k(t)+\lambda_2'(1-y_k(t))\Theta_x(t). \quad (18)$$

Using the method in subsection 2.2, one can see that there is a permanent stationary state which satisfies

$$x_k=\frac{\lambda_1'\Theta_y}{1+\lambda_1'\Theta_y},$$
$$y_k=\frac{\lambda_2'\Theta_x}{1+\lambda_2'\Theta_x}. \quad (19)$$

when

$$R=\lambda_1'\lambda_2'>1. \quad (20)$$

In order to eliminate the spread of the STDs, one can conclude that for a network of nodes with a large connectivity, one should immunize the nodes at high rates, and for a network of nodes with a small connectivity, we only need to immunize them at low rates. In order to make λ_1' and λ_2' constant, more individuals with degree k need to be immune with λ_1 and λ_2 increasing.

Since proportional immunization schemes are effective in networks with well-defined immunization thresholds, and in order to control the epidemic more effectively, we plan to only immunize the male individuals or female individuals with proportional immunization. Without loss of generality, we assume only the female individuals are immunized proportionally, that is

$$\lambda_2 k(1-g_k)=\bar{\lambda}_2,$$

where $\bar{\lambda}_2$ is constant. Then system (1) yields

$$\dot{x}_k(t)=-x_k(t)+\lambda_1 k(1-x_k(t))\Theta_y(t),$$
$$\dot{y}_k(t)=-y_k(t)+\bar{\lambda}_2(1-y_k(t))\Theta_x(t). \quad (21)$$

Using the same method, we can get when

$$R=\lambda_1\bar{\lambda}_2\frac{\langle k^2\rangle_x}{\langle k\rangle_x}>1, \quad (22)$$

the model has a permanent stationary state.

In an infinite SF network, $\langle k^2\rangle_x\to\infty$. Hence, proportional immunization is useless. While in a finite SF network, in particular the BA model,

$$R\simeq\frac{1}{2}m\lambda_1\bar{\lambda}_2\ln(\Omega). \quad (23)$$

This implies that in this case proportional immunization is ineffective. For fixed λ_1 and $\bar{\lambda}_2$, the larger the network size, the more nodes need to be immunized.

3.3. Targeted immunization

The strategy here is to immune the most highly connected nodes, i.e., the ones that are more likely to spread the disease. First, we introduce an upper threshold k_t, and all nodes with connectivity $k>k_t$ are immunized. Calculating a BA network with the continuous k-approximation shows that the density of immunized nodes is related to the connectivity threshold, as

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

Then the model (1) yields

$$\dot{x}_k(t)=-x_k(t)+\lambda_1 k(1-m^2k_t^{-2})(1-x_k(t))\Theta_y(t),$$
$$\dot{y}_k(t)=-y_k(t)+\lambda_2 k(1-m^2k_t^{-2})(1-y_k(t))\Theta_x(t). \quad (24)$$

It is also easy to verify that there is a permanent stationary state when

$$R=\lambda_1\lambda_2(1-m^2k_t^{-2})^2\frac{\langle k^2\rangle_x\langle k^2\rangle_y}{\langle k\rangle_x\langle k\rangle_y}>1. \quad (25)$$

It can be seen that for however small infectivities λ_1 and λ_2, the basic productive number R is easily more than 1 because $\langle k^2\rangle\to\infty$. This indicates that targeted immunization is impractical for SF networks, which is different from general SIS models where targeted immunization is a good strategy. If we consider a BA network, the above R yields

$$R\simeq\frac{1}{4}\lambda_1\lambda_2m^2(1-m^2k_t^{-2})^2\ln(N)\ln(\Omega), \quad (26)$$

and an upper threshold

$$k_t=m^4\left(1-\frac{2}{m\sqrt{\lambda_1\lambda_2\ln(N)\ln(\Omega)}}\right)^{-2}. \quad (27)$$

This implies that the larger the network size, the more nodes need to be immunized, for fixed infectivities λ_1 and λ_2.

As shown in Fig.2, one also has to immunize most male and female individuals with increasing λ_1 and λ_2, and for however small infectivity, we need to immunize most individuals in order to eliminate the epidemic. Hence, the targeted immunization strategy is ineffective for scale-free networks.

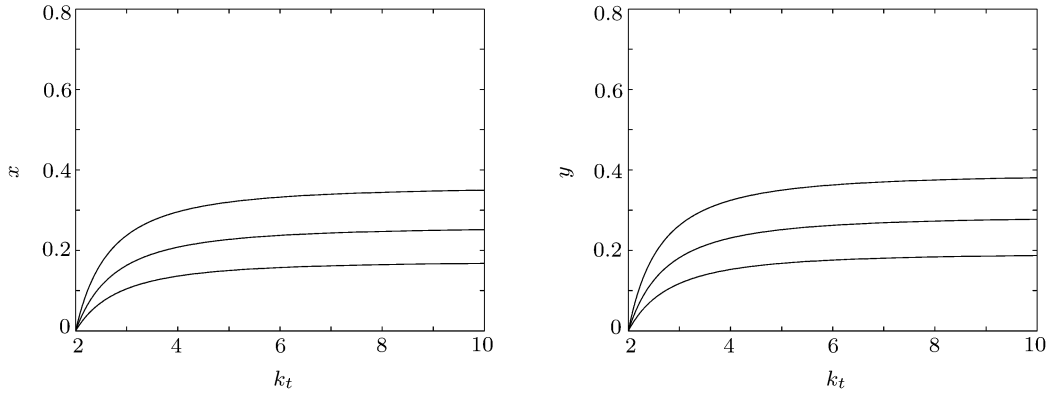


Fig.2. Densities of infected male and female individuals as functions of k_t in a scale-free network. Infected rates $\lambda_1 = \lambda_2 = 0.3$, 0.5, 0.8 (from bottom to up), with $m = 2$, $N = 2000$, $\Omega = 1500$.

4. Conclusion

In this paper, a new model for the spread of sexually transmitted disease on complex networks has been proposed and simulated. Different from the classical epidemic model, in the new model infection between two species (male and female) is taken into account. By applying the uniform, proportional and target immunization strategies to the STDs model, it is concluded that only the proportional immunization strategy is effective on scale-free networks. In the proportional immunization strategy, we consider only the immunization of the male or female individuals, analyses and simulations show that it is ineffective. Thus, in controlling STDs, we should take the proportional immunization strategy, and not only for the male individuals, but also for the female individuals. Unlike the classical SIS model, targeted immunization is ineffective for the STDs model on scale-free networks. Moreover, spreading thresholds of the STDs model are determined, and the stability of the disease-free equilibrium is given, but we have not mentioned the stability of the endemic equilibrium in this paper, which can show the global behavior of the solutions of the model. These and some other related issues will be studied further in the future.

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