Modelling of Epidemics with a Generalized Nonlinear Incidence on Complex Networks

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Abstract. In this paper the spreading of epidemic model on complex networks with a generalized nonlinear incidence rate is presented. Firstly the SIS model on homogeneous networks with nonlinear incidence rate is considered, and the existence conditions about the disease-free equilibrium and the endemic equilibrium are given. And then the model on heterogenous scale-free (SF) networks is considered, where the absence of the threshold on SF networks with nonlinear incidence is demonstrated. At last the stability of the disease-free equilibrium on SF networks is obtained. From this paper, it is shown that, while the number of the equilibria is indeed different from the corresponding model with linear incidence rate, the basic reproductive number, which determinate whether the disease is spreading or not, is independent of the functional form of the nonlinear incidence rate.

Keywords: Epidemics, Complex networks, Nonlinear incidence, The basic reproductive number.

1 Introduction

The study of epidemic model on complex networks has attracted a lot of attention and interest [1, 2, 3, 4, 5, 6, 7, 8, 9]. Within these studies, individuals are modeled as nodes, and possible contacts between individuals are linked by edges. At the early stages, people mainly studied the spreading of epidemic diseases on homogeneous networks. At a mean-field level, the equation describing the time evolution of the average density of infected individuals $\rho(t)$ is

$$\frac{d\rho(t)}{dt} = -\rho(t) + \lambda < k > \rho(t)(1 - \rho(t)). \tag{1}$$

The most significant and general results is the existence of a nonzero epidemic threshold $\lambda_c = 1/ < k >$: when $\lambda > \lambda_c$, the disease will break out and persist; while when $\lambda < \lambda_c$, the disease will gradually die out.

In 1999, Barabási and Albert addressed a new model for complex network: the scale-free network [10]. In scale-free networks, the probability $P(k)$ that any node

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has k links to other nodes is distributed according to a power law $P(k) = Ck^{-\gamma}$, with $2 < \gamma \leq 3$. Suppose $\rho_k(t)$ is the density of the infected nodes with given degree k, and the mean-field equation is:

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k(1 - \rho_k(t))\Theta(\rho(t)), \tag{2}$$

where $\Theta(\rho(t))$ stands for the probability that an edge emanating from a node of degree k points to an infected site. We can know that in SF networks with connectivity exponent $2 < \gamma \leq 3$, for which $< k^2 > \to \infty$, we have $\lambda_c = 0$. This fact implies that for any positive value of λ the infective individuals can pervade the system with a finite prevalence, in a sufficiently large network. This result disproves the threshold theory in epidemiology.

In modeling of communicable diseases, the incidence rate is consider to play a key role in ensuring that the model does indeed give a reasonable description of the disease dynamics. In most classical disease models, the incidence rate is assumed to be mass action incidence with bilinear interactions given by βIS [11], where β is the probability of transmission per contact, and S and I represent the susceptible and infected individuals, respectively. It can be seen that in the above homogeneous network and scale-free network, the incidence rate are all bilinear functions.

However there are several nonlinear incidence rates to be proposed by many researchers: Yorke & London [12] for a time-dependent infection rate; Capasso & Serio [13] for a saturated incidence rate and Liu et al. [14, 15] for the effect of behavioral changes on the incidence of infection. Recently Alexander & Moghadas [16] have given a general nonlinear function $f(I; v) \in C^3(R)$, for $I, v \geq 0$, which satisfies the following general assumptions:

$(A1^{'}) : f(0, v) = f(I, 0) = 0;$
$(A2^{'}) : \partial f/\partial I > 0$ for $I > 0;$
$(A3^{'}) : \partial^2 f/\partial I^2 \leq 0$ for $I > 0.$

For most examples that appear in the literature (Moghadas & Alexander [17]; Derrick & van den Driessche, [18]; Ruan & Wang [19]; van den Driessche & Watmough [20]; Xiao et al. [21]), are all satisfied these assumptions.

In fact there are some results about different infectivities on complex networks recently. Zhou et al. [22, 23] presented the identical infectivity for every node regardless of their different degrees, and they obtain the epidemic threshold $\lambda_c = 1/A$, where A is the constant infectivity of each node. Fu et al. [24] considered the epidemics on scale-free networks with piecewise linear infectivity and immunization. In above papers the different infectivities are all considered according to the different degrees of nodes. In fact, we can consider the infectivities from another way. Here we call it the nonlinear incidence rate. The aim of this paper is to extend and analyze a simple SIS model on complex networks, with a generalized nonlinear incidence rate which satisfies a few general conditions. We hope that the results presented in this paper will give an extent insight into the spread of the disease on networks.

The organization of this paper is as follows. In the next section, we give the SIS model with nonlinear incidence on homogeneous networks, derive the conditions for the existence of the disease-free equilibrium and the endemic equilibria. In Section 3 we give the model with nonlinear incidence on scale-free networks. In this section the existence conditions for the equilibria and the stability of the disease-free equilibrium are derived. At last a brief conclusion and discussion are given in Section 4.

2 The SIS Model with Nonlinear Incidence on Homogeneous Networks

The SIS model with nonlinear incidence on homogeneous networks is as follows:

$$\frac{d\rho(t)}{dt} = -\rho(t) + \lambda < k > (1 + U(\rho, \alpha))\rho(t)(1 - \rho(t)), \tag{3}$$

where $U(\rho, \alpha)$ satisfies:

$(A1) : U(0, \alpha) = U(\rho, 0) = 0;$
$(A2) : \partial U/\partial\rho > 0$ for $\rho > 0;$
$(A3) : \partial^2 U/\partial\rho^2 \leq 0$ for $\rho > 0.$

The assumption $(A1)$ reflects the fact that, for ρ or α small, the incidence of infection approximates the commonly used mass action form, while for large enough ρ and α, the nonlinear term $U(\rho, \alpha)$ dominates. Therefore, α may be considered as a parameter measuring departure from mass action, and it reduces the infection rate to $\lambda < k > (1 - \rho(t))\rho(t)$ when $\alpha = 0$. The assumption $(A2)$ reflects that the incidence of infection will increase with the increasing of the density of infective individuals. The assumption $(A3)$ shows that the increasing is slow.

Obviously $\rho(t) = 0$ is the disease-free equilibrium of (3). Defining

$$R_0 = \lambda < k > . \tag{4}$$

It is easy to know $\rho(t) = 0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Here R_0 is called the basic reproductive number on homogeneous networks, defined as the the relative density of secondary cases that one infected individual will cause through the duration of the infectious period [11]. In order to establish the conditions for the existence of other equilibria, called endemic equilibria, of the model in the presence of the disease we need analysis the fixed points of the equation:

$$\rho = 1 - \frac{1}{\lambda < k > (1 + U(\rho, \alpha))} \equiv \phi(\rho, \alpha). \tag{5}$$

Note that (4), $\phi(\rho, \alpha) = 1 - \frac{1}{R_0(1+U(\rho,\alpha))}$. The endemic equilibria of the model correspond to the fixed points of $\phi(\rho, \alpha) = \rho$. The functional form of ϕ determines the number of these equilibria. It can be seen that ϕ has the following properties:

(1) : $\phi_0 = \phi(0, \alpha) = 1 - 1/R_0$;
(2) : $\phi_1 = \phi(1, \alpha) < 1$;
(3) : $\frac{\partial \phi}{\partial \rho} = \frac{\partial U/\partial \rho}{R_0(1+U)^2} > 0$ for $\rho > 0$;
(4) : $\frac{\partial^2 \phi}{\partial \rho^2} = \frac{(\partial^2 \phi/\partial \rho^2)(1+U) - 2(\partial U/\partial \rho)^2}{(1+U)^3} < 0$ for $\rho > 0$.

From the properties of function ϕ, we have the following theorem:

Theorem 1

(1) If $R_0 > 1$, the model (3) has a unique endemic equilibrium.

(2) If $R_0 = 1$, the model (3) has a unique endemic equilibrium if $\frac{\partial U}{\partial \rho}\Big|_{\rho=0} > 1$.

(3) If $R_0 < 1$, the model (3) may have no, one or two endemic equilibria.

Proof. It follows from (1)-(4) that $\phi(\rho, \alpha)$ is increasing and concave down on $[0, 1]$. If $R_0 > 1$, then $\phi_0 > 0$. Thus there must exists a unique $\rho^* > 0$, such that $\phi(\rho^*, \alpha) = \rho^*$(Fig.1 (a)). If $R_0 = 1$, then $\phi_0 = 0$ and $\partial\phi/(\partial\rho)(0) > 1 (\leq 1)$ if $\frac{\partial U}{\partial \rho}\Big|_{\rho=0} > 1$. Thus, $\phi(\rho, \alpha) = \rho$ has a unique positive root if $\frac{\partial U}{\partial \rho}\Big|_{\rho=0} > 1$, and no positive root otherwise(Fig.1 (b,c)). Finally suppose $R_0 < 1$, so that $\phi_0 < 0$, there may have no, one, or two positive roots (Fig.2). □

About the conditions under which the model exhibits two endemic equilibria when $R_0 < 1$, we give the following theorem.

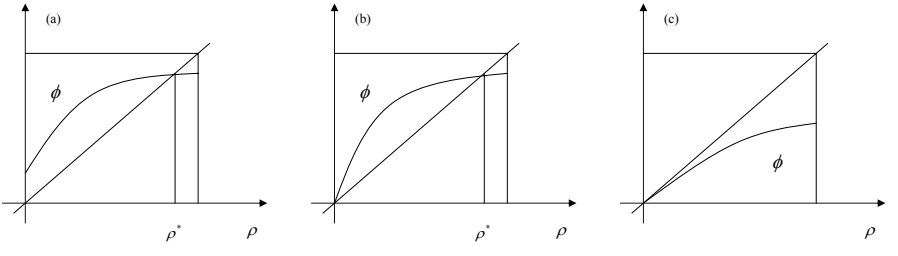

Fig. 1. Phase diagrams of $\phi(\rho, \alpha)$ showing the location of the fixed points for (a)$R_0 > 1$ with a unique fixed point; (b) and (c) for $R_0 = 1$ with a unique fixed point and no fixed point

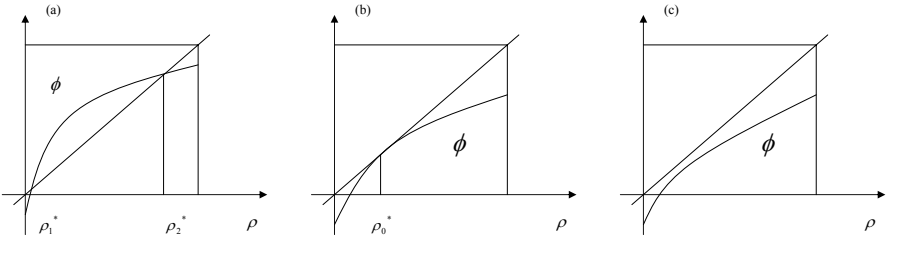

Fig. 2. Phase diagrams of $\phi(\rho, \alpha)$ showing the location of the fixed points for $R_0 < 1$ with two fixed points (a); a unique fixed point (b) and no fixed point (c)

Theorem 2. *If* $\frac{\partial U}{\partial \rho}\Big|_{\rho=0} > 1$ *holds, then there exists a unique* $R^* < 1$, *such that:*

(1) If $R^* < R_0 < 1$, *the model (3) has two endemic equilibria.*
(2) If $R_0 = R^*$ *, the model (3) has a unique endemic equilibrium.*
(3) If $R_0 < R^*$, *the model (3) have no endemic equilibrium.*

Proof. Let $H(\rho, \alpha) = \phi(\rho, \alpha) - \rho$, and suppose that $\frac{\partial U}{\partial \rho}\Big|_{\rho=0} > 1$, which implies $(\partial H/\partial \rho)|_{\rho=0} > 0$, and $\partial^2 H/\partial \rho^2 < 0$ for $0 < \rho < 1$. Hence $\partial H/\partial \rho$ is a monotone decreasing function of ρ, and thus there exists a unique $\rho_0^* > 0$ such that $(\partial H/\partial \rho)|_{\rho=\rho_0^*} = 0$. This implies that H is an increasing function on $(0, \rho_0^*]$ and decreasing on $(\rho_0^*, 1)$. Since $H(0) < 0$ when $R_0 < 1$, and $H(0) = 0$ when $R_0 = 1$, by continuity, it follows that there exists R^* with $R^* < 1$ for which $\partial H/\partial \rho$ has a unique root ρ_0^* with $(\partial H/\partial \rho)|_{\rho=\rho_0^*} = H(\rho_0^*) = 0$. Then for some values of $R_0 \in (R^*, 1)$, H has two roots ρ_1^* and ρ_2^*. Assume that $(\partial \rho/\partial R_0)|_{\rho_1^*} > 0$. Using the expression $\phi(\rho, \alpha) = R_0 + \phi_\rho(\hat{\rho}, \alpha)\rho$ for some $\hat{\rho} \in (0, \rho)$, and noting that $(\partial H/\partial \rho)|_{\rho_1^*} > 1$, we have

$$\begin{aligned} \frac{\partial \phi}{\partial R_0}\Big|_{\rho_1^*} &= 1 + \phi_\rho(\hat{\rho}, \alpha)\, \frac{\partial \rho}{\partial R_0}\Big|_{\rho_1^*} > 1 + \phi_\rho(\rho_1^*, \alpha)\, \frac{\partial \rho}{\partial R_0}\Big|_{\rho_1^*} \\ &= 1 + \frac{\partial \phi}{\partial R_0}\Big|_{\rho_1^*} \end{aligned}$$

which is a contradiction. This implies that $(\partial \rho/\partial R_0)|_{\rho_1^*} < 0$ and thus, the number of infected individuals at the low endemic equilibrium reduces as R_0 increases. Similarly, it can be shown that $(\partial \rho/\partial R_0)|_{\rho_2^*} > 0$ which implies that the number of infected individuals at the high endemic equilibrium increases as R_0 increases. Therefore, the quantity R^* for which $(\partial H/\partial \rho)|_{\rho=\rho_0^*} = H(\rho_0^*) = 0$ is unique, and we end this theorem. □

From the above analysis, we can see the dynamic behaviors of the SIS model with nonlinear incidence on homogeneous networks is different from the general bilinear epidemic model. In the general bilinear epidemic model, there have a threshold λ_c, corresponding to the basic reproductive number $R_0 = \lambda < k >$. When $R_0 > 1$, the disease will break out and persist; while when $R_0 < 1$, the disease will gradually die out. However in the nonlinear epidemic model, despite the disease will break out when $R_0 > 1$, the disease will not die out when $R_0 < 1$, a new threshold R^* appear, if $R^* \leq R_0 < 1$, the epidemic will break out and persist, if $R_0 < R^*$, the epidemic will die out.

3 The SIS Model with Nonlinear Incidence on Scale-Free Networks

3.1 The Model and the Existence of Equilibria

In model (2), the probability of last term is proportional to the infection rate λ, the number of connections k, and the probability $\Theta(\rho(t))$ that any given link points to an infected node. yielding

$$\Theta(\rho(t)) = \frac{1}{< k >} \sum_k kP(k)\rho_k(t), \tag{6}$$

the last term can be read as the incidence rate and the term as a linear force of infection. Thus we propose and study here the following model with nonlinear incidence

$$\dot{\rho}_k(t) = -\rho_k(t) + \lambda k(1 - \rho_k(t))(1 + V(\Theta, \mu))\Theta, \tag{7}$$

where $V(\Theta, \mu)$ satisfies:

$(B1) : V(0, \mu) = V(\Theta, 0) = 0;$
$(B2) : \partial V/\partial\Theta > 0$ for $\Theta > 0;$
$(B3) : \partial^2 V/\partial\Theta^2 \leq 0$ for $\Theta > 0.$

We can also note from $(B1)$ that, for Θ and μ small, the bilinear term dominates, while for large enough Θ and μ, the nonlinear term V dominates. Therefore, μ is a parameter measuring departure from mass action, and it reduces the infection rate to $\lambda k(1 - \rho_k(t))\Theta$.

In case of nonlinear infection rate, proceeding as in Section 2 we have that the equilibrium values of Θ are determined by the following relationship:

$$\Theta = \frac{\lambda}{< k >} \sum_k k^2 P(k) \frac{(1 + V(\Theta, \mu))\Theta}{1 + \lambda k(1 + V(\Theta, \mu))\Theta} \doteq \Phi(\Theta). \tag{8}$$

Obviously, $\Theta = 0$ is a solution of (7), which corresponds the disease-free equilibrium $\rho_k(t) = 0, k = 1, 2, \cdots$. As to the stability of the disease-free equilibrium, we will give in the next subsection. In the following we will consider the existence of the positive equilibrium of model (7). We note $V^{'} \doteq \partial V/\partial\Theta$ and $V^{''} \doteq \partial^2 V/\partial\Theta^2$ and it is easy to verify that:

$(5) : \Phi_0 = \Phi(0, \mu) = 0;$
$(6) : \Phi_1 = \Phi(1, \mu) < 1;$
$(7) : \frac{\partial\Phi}{\partial\Theta} = \frac{\lambda}{<k>} \sum_k k^2 P(k) \frac{1+V(\Theta,\mu)+V^{'}(\Theta,\mu)\Theta}{(1+\lambda k(1+V(\Theta,\mu))\Theta)^2} > 0;$
$(8) : \frac{\partial^2\Phi}{\partial\Theta^2} = \frac{\lambda}{<k>} \Sigma_k k^2 P(k) \frac{(2V^{'}+V^{''}\Theta)(1+\lambda k(1+V)\Theta)-\lambda(1+V+\Theta V^{'})^2}{(1+\lambda k(1+V)\Theta)^3}.$

Let

$$F(\Theta) = (2V^{'} + V^{''}\Theta)(1 + \lambda k(1 + V)\Theta) - \lambda(1 + V + \Theta U^{'})^2, \tag{9}$$

if $F(\Theta) < 0$, then $\Phi^{''}(\Theta) < 0$. So if there is another solution $0 < \Theta < 1$, it must satisfy $\left.\frac{\partial\Phi(\Theta)}{\partial\Theta}\right|_{\Theta=0} > 1$, that is $\lambda\frac{<k^2>}{<k>} > 1$, where $< k^2 >= \sum_k k^2 P(k)$.

Let

$$R_1 = \lambda\frac{< k^2 >}{< k >}, \tag{10}$$

where R_1 is called the basic reproductive number on scale-free networks.

If $\frac{\partial^2\Phi}{\partial\Theta^2}$ has variable sign, there may be two or more co-existing solutions, corresponding to unstable and locally stable endemic equilibria of (7). From the view of biology, this implies that the long term time course of the epidemics depends

on its initially observed distribution. For example, considering two initial states located in the two different basins of attraction, they will have very different asymptotic behaviors. It is also interesting to stress that the basic productive number in the models with the nonlinear incidence rate is same to it in the models with the bilinear.

3.2 The Stability of the Disease-Free Equilibrium

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

$$J = \begin{pmatrix} -1 \cdots & 0 \\ \vdots \quad \ddots & \vdots \\ 0 \quad \cdots & -1 \end{pmatrix} + \frac{\lambda}{< k >} \begin{pmatrix} 1 \\ \vdots \\ N \end{pmatrix} \begin{pmatrix} 1 \times P(1) \cdots N \times P(N) \end{pmatrix}.$$

The matrix J has $N-1$ eigenvalues equal to -1: $\mu_1, \cdots = \mu_{N-1} = -1$, the Nth is

$$\mu_N = -1 + \lambda \frac{< k^2 >}{< k >}. \tag{11}$$

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

Theorem 3. *If $R_1 \leq 1$, then the disease-free equilibrium of (2.1) is asymptotically stable in the set $[0,1]^N$, otherwise there exists a unique endemic equilibrium if $F(\Theta) < 0$.*

The biological consequence of this result is that if the epidemic threshold $R_1 = 1$ is not exceeded the disease will disappear. On the contrary if $R_1 > 1$, then there may be 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 individuals are, whether the disease will eradicate or not only depend on the basic reproductive number R_1.

4 Conclusion

In this paper, epidemic models on complex networks with nonlinear incidence rate have been proposed. Different from other incidence rate on complex networks, which is related with the degrees of nodes, in the new model the generalized nonlinear incidence rate is taken into account, which is the generalization of the bilinear incidence rate on complex networks. Firstly the SIS model on homogeneous networks with nonlinear incidence rate is considered, and the existence of the equilibria are given. And then the model on heterogenous scale-free networks are considered, where the absence of the threshold on the SF network is demonstrated, and the stability of the disease-free equilibrium is obtained.

From this paper, it is shown that introducing nonlinear incidence rate makes the model much more complex. However the basic reproductive number is independent of the functional form of the nonlinear incidence rate, while the number

of the equilibria is indeed different from the corresponding model with linear incidence rate. Moreover, the stability of the disease-free equilibrium on complex networks are given, but as to the stability of the endemic equilibria and bifurcations, which can show the global behavior of the solutions of the model, we have not mentioned in this paper. These and some other related issues will be further studied in the future.

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