Dynamics of a network-based SIS epidemic model with nonmonotone incidence rate

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Abstract

This paper studies the dynamics of a network-based SIS epidemic model with nonmonotone incidence rate. This type of nonlinear incidence can be used to describe the psychological effect of certain diseases spread in a contact network at high infective levels. We first find a threshold value for the transmission rate. This value completely determines the dynamics of the model and interestingly, the threshold is not dependent on the functional form of the nonlinear incidence rate. Furthermore, if the transmission rate is less than or equal to the threshold value, the disease will die out. Otherwise, it will be permanent. Numerical experiments are given to illustrate the theoretical results. We also consider the effect of the nonlinear incidence on the epidemic dynamics.

Keywords: Complex network, epidemic model, nonlinear incidence, psychological effect, permanence

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1. Introduction

To better predict and control the spread of infectious diseases, mathematical modeling of infectious disease dynamics has been extensively studied for a long time (see the review paper by Hethcote [2]). At the early stages, studies on various epidemic models mainly focus on the homogeneous mixing assumption, that is, each susceptible individual within a population has the same probability to contact with an infected one. However, the effect of contact heterogeneity should be incorporated into consideration in reality because there might exist some members who could transmit infection to many other members of the population. Therefore, the disease transmission should be modeled over complex networks. In recent years, the study of epidemic model in complex networks has attracted a lot of attention due to its theoretical interest [1, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 15, 18, 19, 20, 21, 22, 23, 24, 25].

In mathematical terms, a complex network is composed by a set of nodes and edges. Each node represents an individual in its corresponding epidemiological state, and each edge between two nodes stands for an interaction that may allow disease transmission. For instance, as one consider the so-called SIS model, all the nodes in a given network can be firstly classified into n groups such that the nodes in the same group have equal degree. That is, each node in the k-th group has degree k for $k = 1, 2, \cdots, n$. In addition, according to the spreading of SIS process, each node within the network has one of the two epidemiological states: susceptible and infected. Let $s_k(t)$ and $\rho_k(t)$ be respectively the densities of susceptible and infected nodes in the k-th group at time t, and let $N_k(t) := s_k(t) + \rho_k(t)$ for all $t \geq 0$ and $k = 1, 2, \cdots, n$. With these notations, the dynamics of the SIS model in a given network can be formulated as the following system of ODEs:

$$\begin{cases} s_k'(t) = -\lambda k s_k(t)\Theta(t) + \gamma\rho_k(t), \\ \rho_k'(t) = \lambda k s_k(t)\Theta(t) - \gamma\rho_k(t), \qquad k = 1, 2, \cdots, n, \end{cases} \tag{1}$$

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where $\lambda > 0$ is the transmission rate, $\gamma > 0$ is the recovery rate of infected nodes, and without loss of generality, one can assume that $\gamma = 1$. The dynamics of the n groups of SIS subsystems are coupled through the function $\Theta(t)$, which is the probability that a given edge connected to an infected node. For simplicity, the connectivity of nodes in this network is assumed to be uncorrelated, so

$$\Theta(t) = \frac{1}{\langle k \rangle} \sum_{h=1}^{n} hP(h)\rho_h(t).$$

Here, $P(h) > 0$ is the probability that a node has degree h and thus $\sum_{h=1}^{n} P(h) = 1$; $\langle k \rangle = \sum_{h=1}^{n} hP(h)$ denotes the mean degree of the network. Since $N_k'(t) = 0$ for all $t > 0$ and for all k, this implies that $N_k(t)$ is a constant. By introducing the normalization condition, namely, $s_k(0) + \rho_k(0) = 1$ for all k, (1) becomes the following system

$$\rho_k'(t) = \lambda k(1 - \rho_k(t))\Theta(t) - \rho_k(t), \qquad k = 1, 2, \cdots, n. \tag{2}$$

Pastor-Satorras and Vespignani [11, 12] studied system (2) in scale-free networks and showed that there exists an epidemic threshold, that is, $\lambda_c := \langle k \rangle / \langle k^2 \rangle$ with $\langle k^2 \rangle = \sum_{h=1}^{n} h^2 P(h)$, for the transmission rate λ. If $\lambda < \lambda_c$, then the disease will disappear. Otherwise, the infection spreads and becomes endemic. Besides, they also showed that even if the transmission rate is vanishingly small, the disease can spread and persist in the infinite size limit. Also, the SIR model in scale-free network was studied in [9], the authors concluded that this model exhibits the same absence of epidemic threshold. Besides, other network-based epidemic models, such as SIRS [5], SEIRS [7], SIQRS [6], and a generalized model [25] have been formulated and analyzed. Either the attractivity of the equilibria or the permanence of the models has been reported in these works. For the diseases transmitted by a vector, network-based models with infective media are also investigated in [14, 18].

We shall emphasize that for most of the homogeneous mixing models in the literature, the incidence rates are usually assumed to be a bilinear function based on the mass action law for infection. Such type of incidence rate is also frequently used in network epidemic models. More precisely, in most of the above-mentioned network models, the incidence rate is usually given by $\lambda k s_k(t)\Theta(t)$. However, it has been pointed out that a nonlinear incidence rate seems more reasonable than the bilinear incidence rate [17]. For instance, in practical situation, the incidence rate would decrease at high infective levels (i.e., Θ is large) because of the protection measures by the susceptible individuals or due to the quarantine of infected ones. Therefore, linear incidence rate cannot reflect such behavioral change of individuals in the network and a new incidence rate should be taken into consideration to respond to such psychological or inhibit effect. Motivated by Xiao and Ruan [17], in this paper we will use a nonlinear incidence rate defined by

$$\lambda k s_k(t) g(\Theta) := \lambda k s_k(t) \frac{\Theta}{1 + \alpha\Theta^2}, \tag{3}$$

where $\alpha \geq 0$ is a parameter and we are mainly concerned with a network-based SIS model which is governed by the following system of ODEs:

$$\begin{cases} s_k'(t) = -\lambda k s_k(t) g(\Theta(t)) + \rho_k(t), \\ \rho_k'(t) = \lambda k s_k(t) g(\Theta(t)) - \rho_k(t), \qquad k = 1, 2, \cdots, n, \end{cases} \tag{4}$$

where the parameters and variables are the same as aforementioned. We remark here that when $\alpha = 0$, the nonlinear incidence rate becomes the bilinear one. Hence system (4) can be simplified to system (2). The graphs of the function $g(\Theta)$ with different values of α are plotted in Fig. 1. As one can see in Fig. 1, if α is large enough (e.g., $\alpha \geq 2$), the function g becomes a nonmonotone function. The biological meaning is that at high infective risk (i.e., when Θ is sufficiently large), the incidence rate may decrease as Θ increases because individuals become more careful and tend to reduce their contacts with other ones.

On the whole there has thus far been relatively little research into network epidemic models with nonlinear incidence rate. The work most closely related to ours is that of Zhang and Sun [22], which studied an SIS model with a feedback mechanism as well as the birth and death rates. The incidence rate proposed in that work is $\lambda k s_k(t)(1 - \alpha\Theta(t))\Theta(t)$ and the authors derived the corresponding basic reproduction number. Furthermore, they

proved that if the basic reproduction number is less than one, then the disease-free equilibrium is globally asymptotically stable. Conversely, if the basic reproduction number is larger than one, then the endemic equilibrium is locally asymptotically stable. However, the global behavior about endemic status is still unsolved. In this paper, we will study the global dynamics of the system (4). We first obtain the epidemic threshold λ_c, see (9) below, and then prove that if the transmission rate $\lambda \leq \lambda_c$, then the disease-free equilibrium is globally attractive. Moreover, it is indeed globally asymptotically stable if $\lambda < \lambda_c$. But, if $\lambda > \lambda_c$, then the disease-free equilibrium becomes unstable; meanwhile, there exists uniquely a positive endemic equilibrium. Besides, we also show that the disease will be permanent when $\lambda > \lambda_c$. Numerical examples with a finite size of scale-free network will be proposed to support the theoretical analysis.

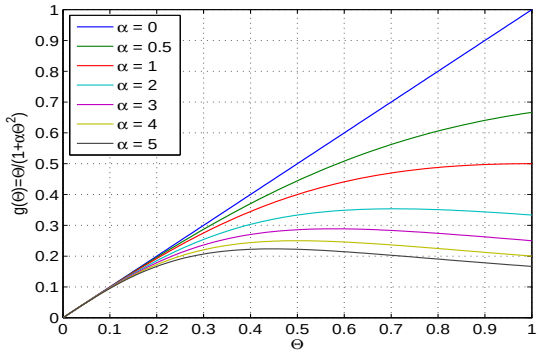

Fig. 1. The graphs of incidence function $g(\Theta)$ with various values of α.

The remainder of this paper is organized as follows. In Section 2, we show that the solutions of system (4) are positive and the epidemic threshold is obtained. In Section 3, we study the stability of the disease-free equilibrium and analyze the permanence of the disease. In Section 4, numerical experiments are given to illustrate the theoretical results. Finally, conclusions and future works are drawn in Section 5.

2. Positivity of solutions and the epidemic threshold

In this section, we will show that the solutions of system (4) with some feasible initial conditions, see (5) below, is positive and the epidemic threshold λ_c is obtained.

From a practical perspective, the initial conditions for system (4) satisfy

$$0 \leq s_k(0), \rho_k(0) \leq 1, \quad s_k(0) + \rho_k(0) = 1, \quad k = 1, 2, \cdots, n, \quad \Theta(0) > 0. \tag{5}$$

Note that $N_k'(t) = 0$ and $s_k(0) + \rho_k(0) = 1$ for $k = 1, 2, \cdots, n$ lead to $s_k(t) + \rho_k(t) = 1$ for all $t \geq 0$ and for all $k = 1, 2, \cdots, n$. Thus, system (4) becomes the following form:

$$\rho_k'(t) = \lambda k(1 - \rho_k(t)) \frac{\Theta(t)}{1 + \alpha\Theta^2(t)} - \rho_k(t), \qquad k = 1, 2, \cdots, n. \tag{6}$$

We now establish the positivity of solutions in the following lemma.

Lemma 2.1. *Let $(s_1, \rho_1, \cdots, s_n, \rho_n)$ be the solution of SIS system (4) with initial conditions (5). Then for $k = 1, 2, \cdots, n$, we have $0 < s_k(t) < 1$, $0 < \rho_k(t) < 1$, and $0 < \Theta(t) < 1$ for all $t > 0$.*

Proof. We first claim that $\rho_k(t) < 1$ for all $k = 1, 2, \cdots, n$ and for all $t > 0$. Note that $\rho_k(0) \leq 1$. Using system (6) and the continuity of $\rho_k(t)$, one can find a small $\delta > 0$ such that $\rho_k(t) < 1$ for $t \in (0, \delta)$. Now we want to show that $\rho_k(t) < 1$ for all $t > 0$. If not, we can find $t_0 \geq \delta > 0$ so that $\rho_k(t_0) = 1$ and $\rho_k(t) < 1$ for $t \in (0, t_0)$. From (6), we have $\rho_k'(t_0) = -1 < 0$ which implies that there exists $\bar{t} \in (0, t_0)$ such that $\rho_k(\bar{t}) > 1$. This leads to a contradiction. Therefore, $\rho_k(t) < 1$ for all k and for all $t > 0$.

We proceed to prove that $\rho_k(t) > 0$ for all k and for all $t > 0$. Integrating (6), we have

$$\rho_k(t) = \rho_k(0)e^{-t} + \int_0^t e^{-(t-u)}\lambda k(1-\rho_k(u))\frac{\Theta(u)}{1+\alpha\Theta^2(u)}du. \tag{7}$$

If the assertion would not hold, then there exists an integer $k_1 \in \{1, 2, \cdots, n\}$ and a number $t_1 > 0$ such that $\rho_k(t) > 0$ hold for all k and $t \in (0, t_1)$. This yields $\Theta(t) > 0$ for $t \in [0, t_1)$ and $\rho_{k_1}(t_1) = 0$. However, it follows from (7) that

$$\rho_{k_1}(t_1) = \rho_{k_1}(0)e^{-t_1} + \int_0^{t_1} e^{-(t_1-u)}\lambda k_1(1-\rho_{k_1}(u))\frac{\Theta(u)}{1+\alpha\Theta^2(u)}du > 0,$$

which is apparently a contradiction. Thus, $\rho_k(t) > 0$ for all k and for all $t > 0$. Consequently, we infer that $0 < \rho_k(t) < 1$ for all k and for all $t > 0$.

A similar argument can show that $0 < s_k(t) < 1$ for all k and for all $t > 0$. Since $0 < \rho_k(t) < 1$ for all k and for all $t > 0$, one can easily see that $0 < \Theta(t) < 1$ for all $t > 0$. The proof is completed. □

Now, we are going to compute all biologically feasible equilibria $\rho_k \geq 0$ admitted by system (6) and then show that there exists a threshold value λ_c, which is related to the network structure such that if $\lambda > \lambda_c$, then a unique endemic equilibrium exists as well.

It can easily be seen that there exists a zero equilibrium $\rho_k = 0$ $(k = 1, 2, \cdots, n)$, which is corresponding to the disease-free equilibrium of system (4). Let $\rho_k'(t) = 0$. It follows from (6) that

$$\rho_k = \frac{\lambda k\Theta}{1+\lambda k\Theta+\alpha\Theta^2}, \tag{8}$$

where $\Theta = \langle k\rangle^{-1}\sum_{h=1}^{n} hP(h)\rho_h$. Substituting (8) into Θ, one can obtain an equation of the form $\Theta f(\Theta) = \Theta$, where

$$f(\Theta) \equiv \frac{1}{\langle k\rangle}\sum_{h=1}^{n}\frac{\lambda h^2 P(h)}{1+\lambda h\Theta+\alpha\Theta^2}.$$

Since $f'(\theta) < 0$ and $f(1) < 1$, the equation $\Theta f(\Theta) = \Theta$ has a unique non-trivial solution if and only if $f(0) > 1$, that is,

$$\frac{\lambda\langle k^2\rangle}{\langle k\rangle} > 1.$$

These analysis lead to the following result.

Lemma 2.2. *Define the epidemic threshold*

$$\lambda_c := \frac{\langle k\rangle}{\langle k^2\rangle}. \tag{9}$$

If $\lambda > \lambda_c$, then system (6) admits a unique positive equilibrium ρ_k^* $(k = 1, 2, \cdots, n)$, which corresponds to the endemic equilibrium of system (4) and satisfies

$$\rho_k^* = \frac{\lambda k\Theta^*}{1+\lambda k\Theta^*+\alpha(\Theta^*)^2} \quad \text{and} \quad \Theta^* = \frac{1}{\langle k\rangle}\sum_{h=1}^{n} hP(h)\rho_h^*.$$

Remark. *From Lemma 2.2, we can see that the epidemic threshold is determined in terms of the network structure and this threshold is just the same as that one derived in [11]. In other words, the nonlinear incidence rate does not affect the threshold λ_c. Besides, as the result obtained in [11], the spreading processes of our model do not possess an epidemic threshold in an infinite scale-free network since $\langle k^2\rangle \to \infty$ in this situation.*

3. Stability of the disease-free equilibrium and the permanence of the disease

In this section, the stability of the disease-free equilibrium and the permanence of the disease will be analyzed. Firstly, we consider the local asymptotic stability and then the global attractivity of the disease-free equilibrium. More precisely, we will show that if $\lambda \leq \lambda_c$, then the disease-free equilibrium is globally attractive. Otherwise, it is unstable. Secondly, from a result derived in [3], we will show that system (4) is permanent if $\lambda > \lambda_c$.

The following lemma is introduced to facilitate the stability analysis.

Lemma 3.1. ([16]) *For a real $n \times n$ matrix $A = (a_{ij})$ where $a_{ij} = \delta_{ij}\sigma_i + p_i q_j$ $(p_i, q_j \geq 0, i, j = 1, 2, \cdots, n)$ and δ_{ij} is the Kronecker symbol. The determinant of A is given by*

$$\det(A) = \sigma_1\sigma_2\cdots\sigma_n + p_1q_1\sigma_2\sigma_3\cdots\sigma_n + \sigma_1 p_2 q_2\sigma_3\cdots\sigma_n + \cdots + \sigma_1\sigma_2\cdots\sigma_{n-1}p_nq_n.$$

Specially, if $\sigma_i \neq 0, i = 1, 2, \cdots, n$, then

$$\det(A) = \left(1 + \sum_{i=1}^{n} \frac{p_i q_i}{\sigma_i}\right) \prod_{i=1}^{n} \sigma_i.$$

We now state the results of the local stability of the disease-free equilibrium.

Theorem 3.1. *The disease-free equilibrium of system (4) is locally asymptotically stable if $\lambda < \lambda_c$ and it is unstable if $\lambda > \lambda_c$.*

Proof. Here, we consider system (6). The Jacobian matrix evaluated at the zero equilibrium $\rho_k = 0$ $(k = 1, 2, \cdots, n)$ is given by the $n \times n$ matrix

$$A = \begin{pmatrix} -1 + \frac{\lambda\cdot 1\cdot 1\cdot P(1)}{\langle k\rangle} & \frac{\lambda\cdot 1\cdot 2\cdot P(2)}{\langle k\rangle} & \frac{\lambda\cdot 1\cdot 3\cdot P(3)}{\langle k\rangle} & \cdots & \frac{\lambda\cdot 1\cdot (n-1)\cdot P(n-1)}{\langle k\rangle} & \frac{\lambda\cdot 1\cdot n\cdot P(n)}{\langle k\rangle} \\ \frac{\lambda\cdot 2\cdot 1\cdot P(1)}{\langle k\rangle} & -1 + \frac{\lambda\cdot 2\cdot 2\cdot P(2)}{\langle k\rangle} & \frac{\lambda\cdot 2\cdot 3\cdot P(3)}{\langle k\rangle} & \cdots & \frac{\lambda\cdot 2\cdot (n-1)\cdot P(n-1)}{\langle k\rangle} & \frac{\lambda\cdot 2\cdot n\cdot P(n)}{\langle k\rangle} \\ \frac{\lambda\cdot 3\cdot 1\cdot P(1)}{\langle k\rangle} & \frac{\lambda\cdot 3\cdot 2\cdot P(2)}{\langle k\rangle} & -1 + \frac{\lambda\cdot 3\cdot 3\cdot P(3)}{\langle k\rangle} & \cdots & \frac{\lambda\cdot 3\cdot (n-1)\cdot P(n-1)}{\langle k\rangle} & \frac{\lambda\cdot 3\cdot n\cdot P(n)}{\langle k\rangle} \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\ \frac{\lambda\cdot (n-1)\cdot 1\cdot P(1)}{\langle k\rangle} & \frac{\lambda\cdot (n-1)\cdot 2\cdot P(2)}{\langle k\rangle} & \frac{\lambda\cdot (n-1)\cdot 3\cdot P(3)}{\langle k\rangle} & \cdots & -1 + \frac{\lambda\cdot (n-1)\cdot (n-1)\cdot P(n-1)}{\langle k\rangle} & \frac{\lambda\cdot (n-1)\cdot n\cdot P(n)}{\langle k\rangle} \\ \frac{\lambda\cdot n\cdot 1\cdot P(1)}{\langle k\rangle} & \frac{\lambda\cdot n\cdot 2\cdot P(2)}{\langle k\rangle} & \frac{\lambda\cdot n\cdot 3\cdot P(3)}{\langle k\rangle} & \cdots & \frac{\lambda\cdot n\cdot n-1\cdot P(n-1)}{\langle k\rangle} & -1 + \frac{\lambda\cdot n\cdot n\cdot P(n)}{\langle k\rangle} \end{pmatrix}.$$

To assess the eigenvalues of A by Lemma 3.1, let the entries of $A - \mu I$ be $a_{ij} = \delta_{ij}\sigma_i + p_i q_j$, where $\sigma_i = -1 - \mu$, $p_i = \lambda i$, and $q_j = jP(j)/\langle k\rangle$. Therefore, it follows from Lemma 3.1 that the characteristic equation can be expressed as

$$\det(A - \mu I_n) = (-1-\mu)^{n-1}\left(-1 - \mu + \frac{\lambda\langle k^2\rangle}{\langle k\rangle}\right) = 0.$$

It is easy to see that $\mu = -1$ is the negative characteristic root with multiplicity $n - 1$. Thus, the stability of the disease-free equilibrium completely depends on the sign of the root of

$$-1 - \mu + \frac{\lambda\langle k^2\rangle}{\langle k\rangle} = 0.$$

Clearly, $\mu < 0$ if $\lambda < \lambda_c$ and $\mu > 0$ if $\lambda > \lambda_c$. Hence, the disease-free equilibrium is locally asymptotically stable if $\lambda < \lambda_c$ and it is unstable if $\lambda > \lambda_c$. This completes the proof. □

Our task now is to claim that the disease-free equilibrium is indeed globally attractive.

Theorem 3.2. *If $\lambda < \lambda_c$, then the disease-free equilibrium of system (4) is globally asymptotically stable. If $\lambda = \lambda_c$, then it is globally attractive.*

Proof. According to system (6), we have

$$\begin{aligned}\Theta'(t) &= -\Theta(t) + \frac{\lambda}{\langle k\rangle}\sum_{h=1}^{n} h^2 P(h)\,(1-\rho_h(t))\,\frac{\Theta(t)}{1+\alpha\Theta^2(t)},\\ &= \Theta(t)\left(-1 + \frac{1}{1+\alpha\Theta^2(t)}\frac{\lambda\langle k^2\rangle}{\langle k\rangle} - \frac{1}{1+\alpha\Theta^2(t)}\frac{\lambda}{\langle k\rangle}\sum_{h=1}^{n} h^2 P(h)\rho_h(t)\right).\end{aligned}$$

Since $\rho_k(t) > 0$ ($k = 1, 2, \cdots, n$) and $\Theta(t) > 0$ for all $t > 0$, one can derive that

$$\Theta'(t) < \frac{1}{\lambda_c}(\lambda - \lambda_c)\Theta(t), \qquad t > 0.$$

Thus, if $\lambda \le \lambda_c$, then $\Theta'(t) < 0$ and this yields $\lim_{t\to\infty}\Theta(t) = 0$ due to the positivity of $\Theta(t)$. Since $P(k) > 0$ for all $k = 1, 2, \cdots, n$, we have $\lim_{t\to\infty}\rho_k(t) = 0$. This proves that the disease-free equilibrium of system (4) is globally attractive if $\lambda \le \lambda_c$. From Theorem 3.1, we can conclude that the disease-free equilibrium is globally asymptotically stable if $\lambda < \lambda_c$. This completes the proof. □

We have established the stability of the disease-free equilibrium of system (4), and now we want to show that system (4) is indeed permanent if $\lambda > \lambda_c$. To this end, we need the following Lemma.

Lemma 3.2. ([3]) *Consider the system*

$$\boldsymbol{y}' = A\boldsymbol{y} + N(\boldsymbol{y}), \tag{10}$$

where A is an $n \times n$ matrix and $N(\boldsymbol{y})$ is continuously differentiable in a region $\mathcal{D} \subset \boldsymbol{R}^n$. Assume

(i) the compact convex set $C \subset \mathcal{D}$ is positively invariant with respect to system (10), and $\mathbf{0} \in C$;

(ii) $\lim_{\boldsymbol{y}\to\mathbf{0}} \|N(\boldsymbol{y})\|/\|\boldsymbol{y}\| = 0$;

(iii) there exist $r > 0$ and a (real) eigenvector $\boldsymbol{w}$ of $A^\top$ such that $(\boldsymbol{w}\cdot\boldsymbol{y}) \ge r\|\boldsymbol{y}\|$ for all $\boldsymbol{y} \in C$;

(iv) $(\boldsymbol{w}\cdot N(\boldsymbol{y})) \le 0$ for all $\boldsymbol{y} \in C$;

(v) $\boldsymbol{y} = \mathbf{0}$ is the largest positively invariant set for (10) contained in $H = \{\boldsymbol{y} \in C : (\boldsymbol{w}\cdot N(\boldsymbol{y})) = 0\}$.

Then either $\boldsymbol{y} = \mathbf{0}$ is globally asymptotically stable in C, or for any $\boldsymbol{y}_0 \in C - \{\mathbf{0}\}$ the solution $\phi(t, \boldsymbol{y}_0)$ of (10) satisfies $\liminf_{t\to\infty}\|\phi(t, \boldsymbol{y}_0)\| \ge m$, where $m > 0$, independent of $\boldsymbol{y}_0$. Moreover, there exists a constant solution of (10), $\boldsymbol{y} = \boldsymbol{y}^*, \boldsymbol{y}^* \in C - \{\mathbf{0}\}$.

The following result states the conditions for the permanence of system (4) which relies on the conclusion of Lemma 3.2.

Theorem 3.3. *If $\lambda > \lambda_c$, then system (4) is permanent, that is, there exists a number $\zeta > 0$ such that*

$$\liminf_{t\to\infty}\{\rho_k(t)\}_{k=1}^n \ge \zeta,$$

for any solution of system (4) with $\rho_k(0) > 0$ for some k.

Proof. Let $\boldsymbol{\rho} = (\rho_1, \rho_2, \cdots, \rho_n)^\top$. Then, system (6) can be rewritten as

$$\boldsymbol{\rho}'(t) = A\boldsymbol{\rho}(t) + N(\boldsymbol{\rho}), \tag{11}$$

where $A = (a_{ij})$ is an $n \times n$ real matrix and $a_{ij} = -\delta_{ij} + \lambda ijP(j)/\langle k\rangle$ for $i, j = 1, 2, \cdots, n$ and δ_{ij} is the Kronecker symbol. The nonlinear vector $N(\boldsymbol{\rho}) = (N_1(\boldsymbol{\rho}), N_2(\boldsymbol{\rho}), \cdots, N_n(\boldsymbol{\rho}))^\top$ is given by

$$N_k(\boldsymbol{\rho}) = \frac{-\lambda k\rho_k\Theta - \lambda k\alpha\Theta^3}{1+\alpha\Theta^2}, \quad k = 1, 2, \cdots, n.$$

Denote $S(A) := \max\{\mathrm{Re}(\mu) : \mu \text{ is the eigenvalue of } A\}$, where $\mathrm{Re}(\mu)$ represents the real part of μ. Let

$$\Omega := \{(\rho_1, \rho_2, \cdots, \rho_n) : 0 \le \rho_k \le 1,\ k = 1, 2, \cdots, n\}.$$

It follows from Lemma 2.1 that Ω is positively invariant with respect to (11).

Now, we shall confirm that (11) satisfies the conditions (i)-(v) of Lemma 3.2. Condition (i) holds for (11) by selecting $C = \Omega$. By using the equivalence relations of norms on $\boldsymbol{R}^n$, we can deduce that

$$\frac{\|N(\boldsymbol{\rho})\|}{\|\boldsymbol{\rho}\|} \le \sqrt{M_1\|\boldsymbol{\rho}\|^2 + M_2\|\boldsymbol{\rho}\|^4} \le \|\boldsymbol{\rho}\|\sqrt{M(1 + \|\boldsymbol{\rho}\|^2)},$$

where

$$M_1 = \lambda^2 n^3 \left(\frac{n}{\langle k\rangle}\right)^2 \left(1 + 2\alpha n^2 \left(\frac{n}{\langle k\rangle}\right)^2\right), \quad M_2 = \alpha^2\lambda^2 n^6 \left(\frac{n}{\langle k\rangle}\right)^6, \quad M = \max\{M_1, M_2\}.$$

Thus,

$$\lim_{\boldsymbol{\rho}\to\mathbf{0}} \frac{\|N(\boldsymbol{\rho})\|}{\|\boldsymbol{\rho}\|} \le \lim_{\boldsymbol{\rho}\to\mathbf{0}} \|\boldsymbol{\rho}\|\sqrt{M(1 + \|\boldsymbol{\rho}\|^2)} = 0$$

and condition (ii) follows. For condition (iii), notice that $A^\top$ is irreducible and $a_{ji} > 0$ whenever $j \ne i$, then by the Perron-Frobenius Theorem, there exists an eigenvector $\boldsymbol{w} = (w_1, w_2, \cdots, w_n)^\top$ of $A^\top$ such that $w_i > 0$ for all $i = 1, 2, \cdots, n$, and the corresponding eigenvalue is $S(A^\top) = S(A) = (\lambda/\lambda_c - 1)$. If we let $r = \min_{1\le i\le n} w_i > 0$, then for any $\boldsymbol{\rho} \in C$ one can obtain $(\boldsymbol{w}\cdot\boldsymbol{\rho}) \ge r\sum_{k=1}^n \rho_k \ge r\|\boldsymbol{\rho}\|$. Condition (iv) is clearly satisfied due to $N_k(\boldsymbol{\rho}) \le 0$ for all $k = 1, 2, \cdots, n$. To examine condition (v), we set $H = \{\boldsymbol{\rho} \in C : (\boldsymbol{w}\cdot N(\boldsymbol{\rho})) = 0\}$. If $\boldsymbol{\rho} \in H$, then we have

$$\sum_{k=1}^n w_k(\lambda k\rho_k\Theta + \lambda k\alpha\Theta^3) = 0.$$

Since each term of the sum is nonnegative, we can conclude that each term is equal to 0, which implies that $\boldsymbol{\rho} = \mathbf{0}$. Therefore, the only invariant set with respect to (11) contained in H is $\boldsymbol{\rho} = \mathbf{0}$, so condition (v) is satisfied. If $\lambda > \lambda_c$, then $\boldsymbol{\rho} = \mathbf{0}$ is an unstable equilibrium of (11) and hence the result of this theorem follows by Lemma 3.2. This completes the proof. □

4. Numerical experiments

In this section, we will give some numerical simulations to illustrate the theoretical analysis. Firstly, we choose $\alpha = 5$ to make sure that the nonlinear function $g(\Theta)$ is a nonmonotone function (cf. Fig. 1). The considered network architecture is a finite scale-free network which contains 500 nodes and has the degree distribution $P(k) = \beta k^{-3}$, where the constant β is chosen to keep $\sum_{k=1}^{500} P(k) = 1$. Then one can verify that the epidemic threshold $\lambda_c = 0.2419$.

Example 4.1. We first consider the stability of the disease-free equilibrium Thus, it follows from Theorem 3.1 that if the transmission rate $\lambda \le \lambda_c$, then the disease-free equilibrium is globally attractive and Fig. 2 demonstrates this result. As one can see, the infected individuals indeed disappear eventually. Moreover, two remarkable findings are highlighted here: firstly, the smaller transmission rate is, the faster the infected individuals disappear. Secondly, one can also observe that the larger the degree is, the larger the outbreak level will be. Besides, Theorem 3.2 indicates that if $\lambda < \lambda_c$, the disease-free equilibrium is indeed globally asymptotically stable. To illustrate this result, we fix $\lambda = 0.2$ and choose 10 different initial values to plot the time evolution of $\rho_{100}(t)$ and $\rho_{300}(t)$ in Fig. 3. Obviously, all the trajectories converge to the trivial equilibrium and this could support the global stability of the disease-free equilibrium.

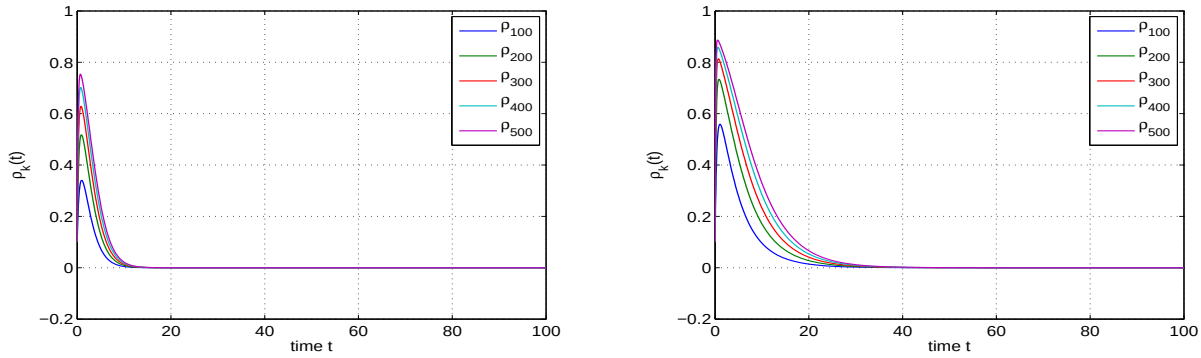

Fig. 2. The time series and trajectories of system (6) with (left) $\lambda = 0.1$; (right) $\lambda = 0.2$. The initial values are given by $\rho_k(0) = 0.1$ for $k = 100, 200, 300, 400, 500$.

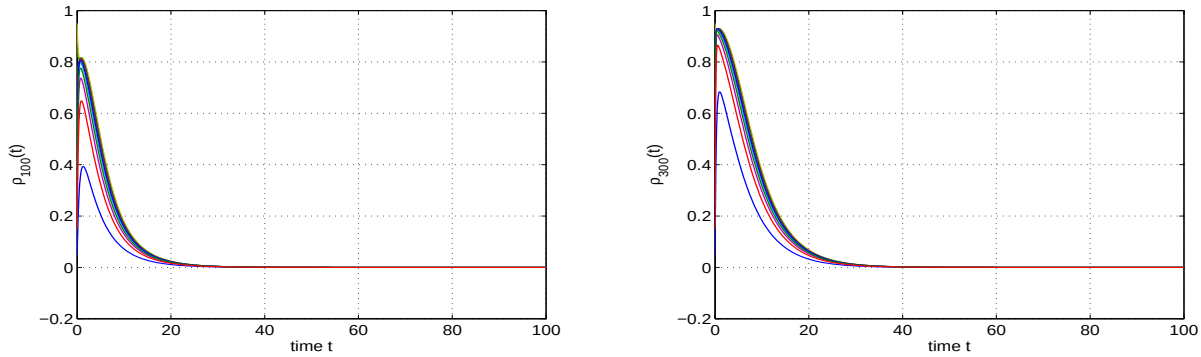

Fig. 3. The time evolution of $\rho_{100}(t)$ and $\rho_{300}(t)$ with $\lambda = 0.2$ and 10 different initial values.

Example 4.2. We now consider the case for $\lambda > \lambda_c$. Theorem 3.3 indicates that if $\lambda > \lambda_c$, the disease is permanent and Fig. 4 confirms this result. We can observe that not only the disease persists but also the density of each infected individual tends to a positive steady state. We can also point out two noteworthy findings: firstly, the larger the degree is, the larger value of the steady state will be. Secondly, we can see that the smaller the transmission rate is, the lower endemic level will be. Since the density of each infected individual converges to a positive constant, we now fix $\lambda = 0.3$ and use 10 different initial values to plot waveforms of $\rho_{100}(t)$ and $\rho_{300}(t)$ in Fig. 5. It seems probable that the endemic equilibrium is globally asymptotically stable, though the rigorous analysis does not present in this paper.

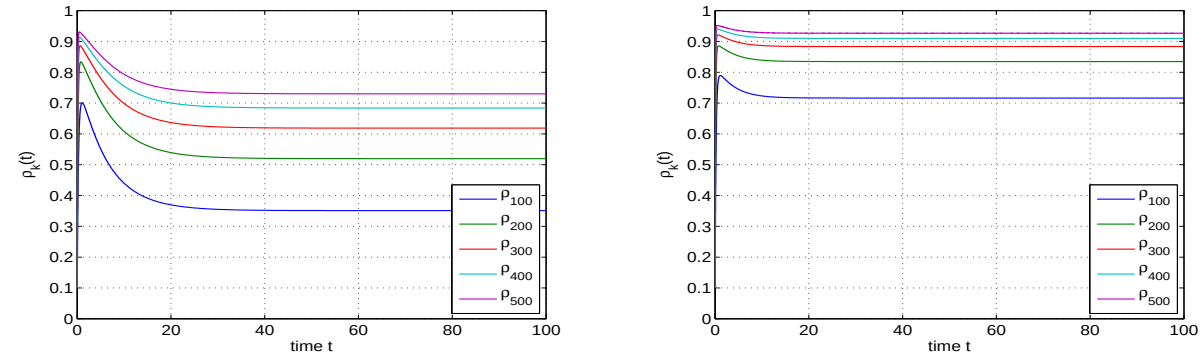

Fig. 4. The time series and trajectories of system (6) with (left) $\lambda = 0.3$; (right) $\lambda = 0.4$. The initial values are given by $\rho_k(0) = 0.1$ for $k = 100, 200, 300, 400, 500$.

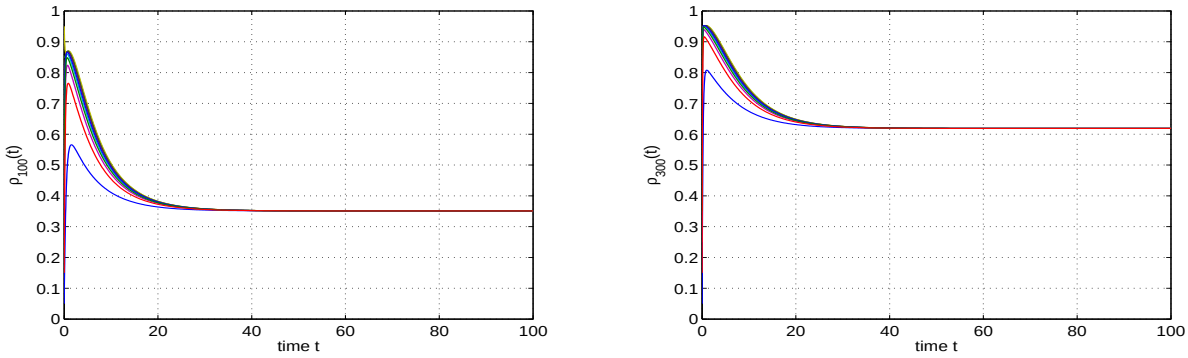

Fig. 5. The time evolution of $\rho_{100}(t)$ and $\rho_{300}(t)$ with $\lambda = 0.3$ and 10 different initial values.

Example 4.3. In the final example, we consider the effect of the parameter α on the epidemic dynamics. Even though the epidemic threshold λ_c does not depends on α explicitly, Fig. 6 highlights an interesting discovery that when the disease is endemic, the densities of the infected nodes decrease as α increases. This suggests that the total of infection $\rho_T(t) \equiv \sum_{k=1}^{500} P(k)\rho_k(t)$ decreases as α increases (cf. Fig. 7).

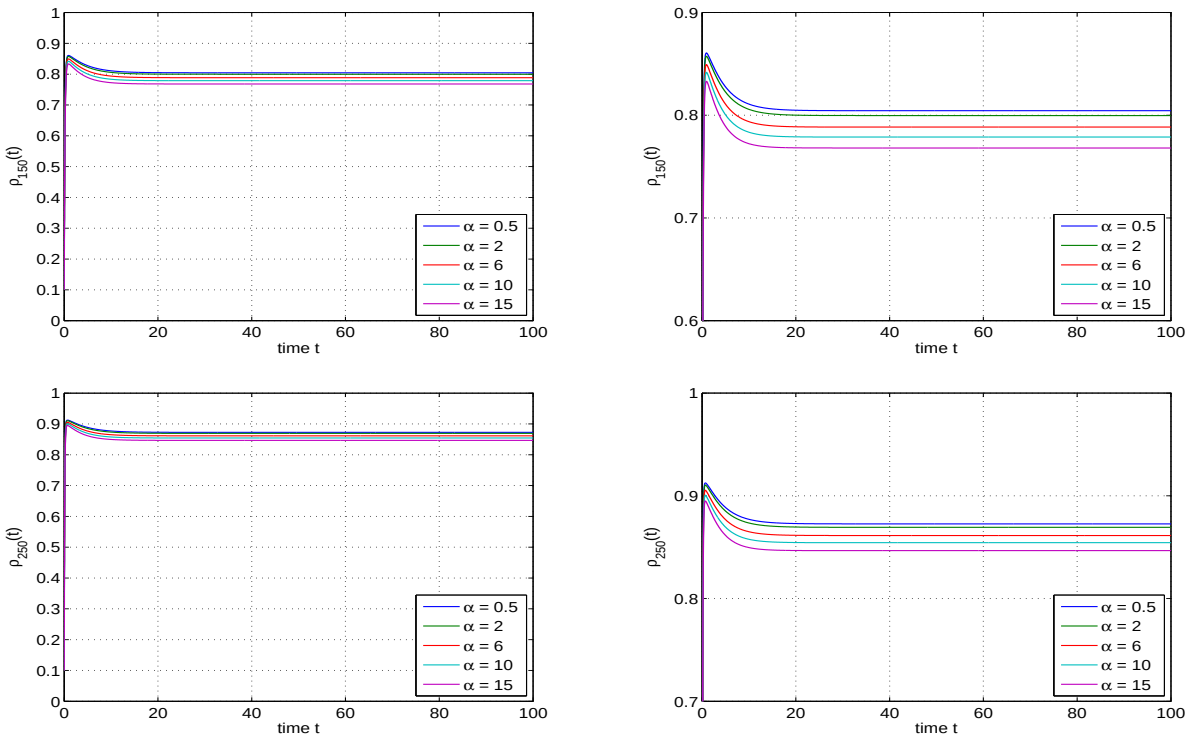

Fig. 6. The time evolution of $\rho_{150}(t)$ and $\rho_{250}(t)$ with $\lambda = 0.4$ and different values of α. The right column contains local amplifications of the left column.

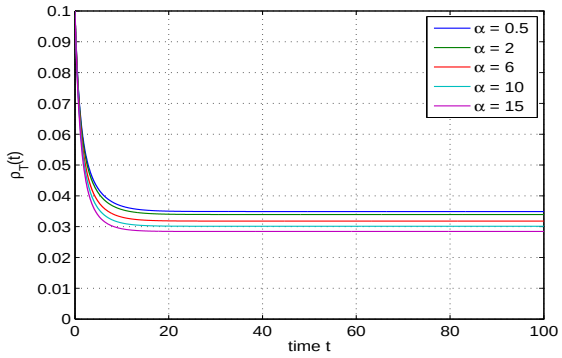

Fig. 7. The time evolution of the total of infection $\rho_T(t)$ with $\lambda = 0.4$ and different values of α.

5. Conclusions

In this paper, we have studied the dynamics of a network-based SIS epidemic model with nonmonotone incidence rate. The nonlinear incidence rate can be used to interpret the psychological effect, namely, the incidence rate would decrease at high infective levels due to the quarantine of infected individuals or the protection measures by the susceptible ones. We have proved that there exists an epidemic threshold λ_c for the transmission rate λ. The threshold determines not only the existence of the endemic equilibrium but also the the global stability of the disease-free equilibrium. More specifically, we have showed that if $\lambda < \lambda_c$ then the disease-free equilibrium is globally asymptotically stable, and as $\lambda = \lambda_c$, it is globally attractive. The biological meaning is that if $\lambda \leq \lambda_c$, the disease will disappear eventually. On the other hand, if $\lambda > \lambda_c$, the disease-free equilibrium becomes unstable; meanwhile, there exists uniquely an endemic equilibrium. In addition, we further show that the disease will be permanent in the network when $\lambda > \lambda_c$.

We have also performed numerical experiments to demonstrate the theoretical results. From the numerical results, we have observed that the endemic equilibrium seems probable to be globally asymptotically stable. However, the detailed analysis of the global stability of the endemic equilibrium remains a challenge problem. Besides, the effect of the parameter α on the epidemic dynamics has been discovered. Numerical simulations indicate that when the disease is endemic, with the increase of the value α, the total of infection will decline. In summary, the studies on network epidemic models with nonlinear incidence rate are still rare. Therefore, studying the spreading dynamics of other network epidemic models with nonlinear incidence and how to control the disease spreading in complex networks will be our future works.

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