Global stability of a network-based SIRS epidemic model with nonmonotone incidence rate

Lijun Liu\textsuperscript{1}, \textsuperscript{*} \hspace{1em} Xiaodan Wei\textsuperscript{2}, \hspace{1em} Naimin Zhang\textsuperscript{3}

1. Department of Mathematics, Dalian Nationalities University, Dalian 116600, China
2. College of Computer Science, Dalian Nationalities University, Dalian 116600, China
3. School of Mathematics and Information Science, Wenzhou University, Wenzhou 325035, China

Abstract

This paper studies the dynamics of a network-based SIRS epidemic model with vaccination and a nonmonotone incidence rate. This type of nonlinear incidence can be used to describe the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals on heterogeneous networks is getting larger. Using the analytical method, epidemic threshold $R_0$ is obtained. When $R_0$ is less than one, we prove the disease-free equilibrium is globally asymptotically stable and the disease dies out, while $R_0$ is greater than one, there exists a unique endemic equilibrium. By constructing a suitable Lyapunov function, we also prove the endemic equilibrium is globally asymptotically stable if the inhibitory factor $\alpha$ is sufficiently large. Numerical experiments are also given to support the theoretical results. It is shown both theoretically and numerically a larger $\alpha$ can accelerate the extinction of the disease and reduce the level of disease.

Keywords: SIRS Epidemic model; Complex networks; Global stability; Nonmonotone incidence.

1 Introduction

Mathematical models which describe the dynamics of infectious diseases have played a crucial role in the disease control in epidemiological aspect. In the traditional epidemiology, it is commonly assumed that individuals mix uniformly and all hosts have identical rates of disease-causing contacts. This over-simplified assumption makes the analysis tractable but not realistic \cite{1}. Virtually, the interpersonal contact underlying disease transmission can be thought of expanding a complex network, where relations (edges) join individuals (nodes) who interact with each other \cite{10,14}. Therefore, the disease transmission should be modeled over complex networks. Related studies indicated that networks structures have profound impacts on the spreading dynamics \cite{13,16,18,26}.

One of the pioneer works in this area was done by Pastor-Satorras and Vespignani \cite{14,15}, where they first succeeded in studying susceptible-infectious-susceptible (SIS) epidemic model on scale-free networks by large-scale simulations. The most striking result

\textsuperscript{*}Corresponding author.

E-mail address: manopt@163.com (L. Liu)
is that they found the absence of the epidemic threshold in these networks. That is, the threshold approaches zero in the limit of a large number of edges and nodes, and even a quite small infectious rate can produce a major epidemic outbreak, for which the rigorous proof was given by Wang and Dai \cite{17} by a monotone iterative technique. Moreno et al. \cite{11} studied susceptible-infectious-recovered (SIR) epidemic model on scale-free complex population networks. They show that the large connectivity fluctuations usually found in these networks strengthen considerably the incidence of epidemic outbreaks. These outstanding results have inspired a great number of related works (see Refs. \cite{8,9,20,25} and the references therein).

It is well-known that the spread of many human diseases can be prevented or reduced by vaccination of the susceptible individuals. Chen and Sun \cite{3} firstly succeeded in studying optimal control of an susceptible-infectious-recovered-susceptible (SIRS) epidemic model with vaccination on heterogeneous networks. They showed that if the percentage of vaccination of the susceptible is smaller than the recovered rate, the diseases may persist on heterogeneous networks.

We shall emphasize that the incidence rate plays an important role in guaranteeing that the model can give a reasonable approximative description for the disease dynamics. However, in most existing epidemic models on complex networks, the incidence rate is usually assumed to be bilinear function based on the mass action law for infection. In fact, there are several reasons for using nonlinear incidence rates, even nonmonotone incidence function \cite{12,21,24}. In practical situations, the number of effective contacts between infective individuals and susceptible individuals usually decreases at high infective levels due to the quarantine of infective individuals or due to the protection measures by the susceptible individuals. Very recently, for modelling such a psychological behavior on complex networks, Li \cite{6} studied the dynamics of a network-based SIS epidemic model with nonmonotone incidence rate,

\[
\begin{align*}
\frac{dS_k(t)}{dt} &= -\lambda k S_k(t) g(\Theta) + I_k(t), \\
\frac{dI_k(t)}{dt} &= \lambda k S_k(t) g(\Theta) - I_k(t), \quad k = 1, 2, \cdots, n,
\end{align*}
\]

where \(\lambda > 0\) is the transmission rate when susceptible individuals contact with infectious. \(S_k(t)\) and \(I_k(t)\) denote the relative densities of susceptible and infectious with degree \(k\) at time \(t\) on the complex networks with maximum degree \(n\). The connectivity of nodes on the network is assumed to be uncorrelated, thus, we have \(\Theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k P(k) I_k(t)\) with \(\langle k \rangle = \sum_{k=1}^{n} k P(k)\) is the average degree of the network and \(P(i)\) is the connectivity distribution.

The function \(g(\Theta)\) is introduced in SIS model (1.1) to reflect the psychological behavior mentioned above, which leads to nonlinear incidence rate defined by

\[
\lambda k S_k(t) g(\Theta) := \lambda k S_k(t) \frac{\Theta}{1 + \alpha \Theta^2},
\]

where \(\alpha \geq 0\) is a parameter.

When \(\alpha = 0\), then the nonlinear incidence rate (1.2) becomes the bilinear one. Hence, the SIS model with (1.2) can be seen a generalization of the existing SIS model. Meanwhile, if \(\alpha\) is large enough (e.g., \(\alpha > 2\)), the function \(g\) becomes a nonmonotone function. The biological meaning is that at high infective risk (i.e., when \(\Theta\) is sufficiently large), the incidence rate may decrease as \(\Theta\) increases because individuals become more careful and tend to reduce
their contacts with other ones. In this sense, we call the parameter \( \alpha \) inhibitory factor from the behavioral change of the susceptible individuals.

Inspired by the works of [3] and [6], in this paper we propose the following SIRS epidemic model with vaccination and the nonmonotone incidence rate as follows,

\[
\begin{align*}
\frac{dS_k(t)}{dt} &= -\lambda kS_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)} + \delta R_k(t) - \mu S_k(t), \\
\frac{dI_k(t)}{dt} &= \lambda kS_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)} - \gamma I_k(t), \\
\frac{dR_k(t)}{dt} &= \gamma I_k(t) - \delta R_k(t) + \mu S_k(t), \quad k = 1, 2, \ldots, n.
\end{align*}
\]  

(1.3)

where \( S_k(t), I_k(t), R_k(t) \) denote the relative densities of susceptible nodes, infectious nodes and recovered nodes with degree \( k \) respectively. \( \delta > 0 \) represents the rate of immunization-lost for recovered nodes; \( \gamma > 0 \) represents the recovery rate of infected nodes; \( \mu > 0 \) represents the vaccination percentage for the susceptible nodes.

Among the existing epidemic models on complex networks, there has far been relatively little research into network epidemic models with nonlinear incidence rate. In [6], the author successfully proposed the SIS model with nonmonotone incidence rate and obtained the epidemic threshold. It was proved that if the transmission rate is below the threshold, the disease-free equilibrium is globally asymptotically stable, otherwise the endemic equilibrium is permanent. In our recent paper [19], the global stability and attractivity of the endemic equilibrium of system (1.1) is rigourously proved. When \( \alpha = 0 \), the proposed model (1.3) can be simplified to the model proposed by Chen and Sun [3]. In this paper, the global stability of disease-free equilibrium as well as the endemic equilibrium is rigourously proved without additional assumptions on the constants, which improves the existing results.

The remainder of this paper is organized as follows. In Section 2, we show that the solutions of system (1.3) are positive and the epidemic threshold is obtained. In Section 3, we study the global stability of the disease-free equilibrium as well as the endemic equilibrium. In Section 4, numerical experiments are given to illustrate the theoretical results. Finally, conclusions are drawn in Section 5.

## 2 Positivity of solutions and the epidemic threshold

From a practical perspective, the initial conditions for system (1.3) satisfy

\[
I_k(0) \geq 0, \quad R_k(0) \geq 0, \quad S_k(0) = 1 - I_k(0) - R_k(0) > 0, \quad k = 1, 2, \ldots, n, \quad \Theta(0) > 0.
\]  

(2.1)

Note that \( \frac{d}{dt}(S_k(t) + I_k(t) + R_k(t)) = 0 \) and \( S_k(t) + I_k(t) + R_k(t) = 1 \) for all \( t \geq 0 \) and for all \( k = 1, 2, \ldots, n \). So the system (1.3) becomes the following form:

\[
\begin{align*}
\frac{dS_k(t)}{dt} &= -\lambda kS_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)} + \delta(1 - S_k(t) - I_k(t)) - \mu S_k(t), \\
\frac{dI_k(t)}{dt} &= \lambda kS_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)} - \gamma I_k(t), \quad k = 1, 2, \ldots, n.
\end{align*}
\]  

(2.2)

As system (2.2) is equivalent to the system (1.3), from now on we only consider the dynamics of (2.2).

First we establish the positivity of solutions of system (2.2) in the following lemma.
Lemma 2.1. Let \((S_1, I_1, \ldots, S_n, I_n)\) be the solution of SIRS system \((2.2)\) with initial conditions \((2.1)\). Then for \(k = 1, 2, \ldots, n\), we have \(0 < S_k(t) < 1\), \(0 < I_k(t) < 1\), and \(0 < \Theta(t) < 1\) for all \(t > 0\).

The proof is shown in Appendix A.

Now we are going to compute all biologically feasible equilibria \((S_k, I_k)\) admitted by system \((2.2)\).

First, it is easy to see the disease-free equilibrium of \((2.2)\) is \(E^0 = (\frac{\delta}{\delta + \mu}, 0, \ldots, \frac{\delta}{\delta + \mu}, 0)\).

In order to calculate the epidemic equilibrium \(E^*(S_1^*, I_1^*, \ldots, S_n^*, I_n^*)\). Let \(S_k'(t) = 0\) and \(I_k'(t) = 0\). It follows from \((2.2)\) that

\[
\begin{cases}
S_k = \frac{\gamma \delta + \alpha \delta \gamma \Theta^2}{\gamma (\delta + \mu) + \lambda (\delta + \gamma) k \Theta + \alpha \gamma (\delta + \mu) \Theta^2}, \\
I_k = \frac{\lambda \delta k \Theta}{\gamma (\delta + \mu) + \lambda (\delta + \gamma) k \Theta + \alpha \gamma (\delta + \mu) \Theta^2},
\end{cases}
\tag{2.3}
\]

where \(\Theta = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} i P(i) I_i\). From \((2.3)\), we obtain the self-consistency equation of the form

\[
f(\Theta) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{\lambda \delta k^2 P(k)}{\gamma (\delta + \mu) + \lambda (\delta + \gamma) k \Theta + \alpha \gamma (\delta + \mu) \Theta^2}.
\tag{2.4}
\]

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

\[
\frac{\lambda \delta \langle k^2 \rangle}{\gamma (\delta + \mu) \langle k \rangle} > 1,
\tag{2.5}
\]

where \(\langle k^2 \rangle = \sum_{k=1}^{n} k^2 P(k)\). These analyses lead to the following result.

Lemma 2.2. Define the epidemic threshold

\[
R_0 := \frac{\lambda \delta \langle k^2 \rangle}{\gamma (\delta + \mu) \langle k \rangle}.
\]

If \(R_0 > 1\), then system \((2.2)\) admits a unique positive equilibrium \(E^*(S_1^*, I_1^*, \ldots, S_n^*, I_n^*)\), which corresponds to the endemic equilibrium of system \((1.3)\) and satisfies

\[
\begin{cases}
S_k^* = \frac{\gamma \delta + \alpha \delta \gamma (\Theta^*)^2}{\gamma (\delta + \mu) + \lambda (\delta + \gamma) k \Theta^* + \alpha \gamma (\delta + \mu) (\Theta^*)^2}, \\
I_k^* = \frac{\lambda \delta k \Theta^*}{\gamma (\delta + \mu) + \lambda (\delta + \gamma) k \Theta^* + \alpha \gamma (\delta + \mu) (\Theta^*)^2},
\end{cases}
\tag{2.6}
\]

where \(\Theta^* = \langle k \rangle^{-1} \sum_{k=1}^{n} k^2 P(k) I_k^* \in (0, 1)\) is the unique positive root of the equation

\[
\Theta = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{\lambda \delta k^2 P(k) \Theta}{\gamma (\delta + \mu) + \lambda (\delta + \gamma) k \Theta + \alpha \gamma (\delta + \mu) \Theta^2}.
\]

Remark 2.1. From Lemma 2.2, we can see that the epidemic threshold is determined in terms of the network structure and this threshold is same as that derived in Ref. [2]. In other words, the nonlinear incidence rate does not affect the threshold \(R_0\). Besides, as the result obtained in Ref. [14], 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 Global stability analysis

In this section, we investigate the globally asymptotical stability of the disease-free equilibrium $E_0$ as well as the endemic equilibrium $E^*$.  

**Theorem 3.1.** Let $R_0 := \frac{\lambda(\langle k^2 \rangle)}{\gamma(\delta + \mu)(\langle k \rangle)} < 1$. Then the disease-free equilibrium $E^0 = (\frac{\delta}{\delta + \mu}, 0, \ldots, \frac{\delta}{\delta + \mu}, 0)$ of system (2.2) is globally asymptotically stable, i.e., the disease fades out.

**Proof.** We first claim that  

$$
\limsup_{t \to +\infty} S_k(t) = \frac{\delta}{\delta + \mu} := S_0^k. \tag{3.1}
$$

Since by Lemma 2.1 we have $0 < S_k(t), I_k(t) < 1$, and $0 < \Theta(t) < 1$ for all $t > 0$. From the first equation of system (2.2), it follows that  

$$
\frac{dS_k(t)}{dt} \leq \delta - (\delta + \mu)S_k(t). \tag{3.2}
$$

which implies (3.1). So for any $\epsilon > 0$, there holds $S_k(t) \leq \frac{\delta}{\delta + \mu} + \epsilon = S_0^k + \epsilon$ when $t$ is sufficiently large. Thus from the second equation of system (2.2), we have  

$$
\frac{dI_k(t)}{dt} < \lambda k(S_0^k + \epsilon)\Theta(t) - \gamma I_k(t). \tag{3.3}
$$

It then suffices to show the positive solutions of the system  

$$
\frac{dI_k(t)}{dt} = \lambda k(S_0^k + \epsilon)\Theta(t) - \gamma I_k(t) \tag{3.4}
$$

tend to zero at $t$ goes to infinity. Consider the Lyapunov function  

$$
V(t) = \sum_{k=1}^{n} w_k I_k(t), \tag{3.5}
$$

where  

$$
w_k = \frac{kP(k)}{\gamma \langle k \rangle}, \quad k = 1, 2, \ldots, n.
$$

Calculating the derivative of $V(t)$ along the positive solutions of (3.4), we have  

$$
\frac{dV(t)}{dt} = \sum_{k=1}^{n} w_k [\lambda k(S_0^k + \epsilon)\Theta(t) - \gamma I_k(t)],
$$

$$
= \sum_{k=1}^{n} \frac{kP(k)}{\gamma \langle k \rangle} [\lambda k(S_0^k + \epsilon)\Theta(t) - \gamma I_k(t)],
$$

$$
= \Theta(t) \left( R_0 + \frac{\lambda(\langle k^2 \rangle)\epsilon}{\gamma \langle k \rangle} - 1 \right). \tag{3.6}
$$

Since $R_0 < 1$, we can fix an $\epsilon > 0$ small enough such that $R_0 + \frac{\lambda(\langle k^2 \rangle)\epsilon}{\gamma \langle k \rangle} < 1$. That ensures $\frac{dV}{dt} \leq 0$ and $\frac{dV}{dt} = 0$ holds only if $I_k = 0$ for $k = 1, \ldots, n$. Hence the trivial solution of (3.4) is globally asymptotically stable. Further from the nonnegativeness of the positive solution of (2.2) and the the comparison theorem, we complete the proof. \qed
Now our task is to claim the globally asymptotical stability of the endemic equilibrium.

**Theorem 3.2.** Let $R_0 := \frac{\lambda \delta(k^2)}{\gamma(\delta+\mu)(k)} > 1$. If $\alpha \geq \alpha_c := \frac{\lambda^2 n^2 R_0^2}{16 \omega^2}$ or $\alpha \leq \alpha_l := \frac{2 \omega}{\lambda n}$ with constant $\omega > 0$ defined in equation (B.3), then the endemic equilibrium $E^*(S^*_1, I^*_1, \ldots, S^*_n, I^*_n)$ of system (2.2) is globally asymptotically stable.

**Proof.** By Lemma 2.1 we have $0 < S_k(t) < 1, 0 < \Theta(t) < 1$ for all $t > 0$. The first equation of (2.2) can be reformulated as

$$S'_k(t) = \delta - (\delta + \mu)S_k(t) - \delta I_k(t) - \lambda k S_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)}.$$  

Moreover, one can derive from the second equation of (2.2) that

$$\Theta'(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k P(k) I'_k(t)$$

$$= \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k P(k) \left[ \lambda k S_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)} - \gamma I_k(t) \right]$$

$$= -\gamma \Theta(t) + \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{n} k^2 P(k) S_k(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)}.$$  

Consider the following Lyapunov function

$$V(t) = \frac{1}{2} \sum_{k=1}^{n} \left\{ a_k (S_k(t) - S^*_k)^2 + m a_k (R_k(t) - R^*_k)^2 \right\} + \Theta(t) - \Theta^* - \Theta^* \ln \frac{\Theta(t)}{\Theta^*},$$

where

$$R^*_k = 1 - S^*_k - I^*_k, \quad a_k = \frac{k P(k)}{\langle k \rangle S^*_k} = \frac{k P(k)}{\langle k \rangle S^*_k}, \quad k = 1, 2, \ldots, n,$$

and the positive constant $m$ will be determined later.

Calculating the derivative of $V(t)$ along the positive solution of (2.2), we have

$$V' = \sum_{k=1}^{n} a_k (S_k - S^*_k) S'_k + \sum_{k=1}^{n} m a_k (R_k - R^*_k) R'_k + \frac{\Theta - \Theta^*}{\Theta} \Theta' =: V_1 + V_2 + V_3.$$  

Using (3.8) and the identity $1 = \frac{\lambda}{\gamma \langle k \rangle} \sum_{k=1}^{n} k^2 P(k) S^*_k$, we obtain

$$V_3 = \gamma (\Theta - \Theta^*) \left[ -1 + \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 P(k) S_k}{1 + \alpha \Theta^2} \right]$$

$$= \Theta - \Theta^* \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 P(k)}{1 + \alpha \Theta^2} \left( \frac{S_k}{1 + \alpha \Theta^2} - \frac{S^*_k}{1 + \alpha (\Theta^*)^2} \right)$$

$$= \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{n} k^2 P(k) \left[ \frac{(S_k - S^*_k)(\Theta - \Theta^*)}{1 + \alpha (\Theta^*)^2} - \frac{\alpha S_k (\Theta - \Theta^*)(\Theta^2 - (\Theta^*)^2)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} \right].$$
Using the last equation of (1.3) and the identity $\gamma I_k^* - \delta R_k^* + \mu S_k^* = 0$, we have

$$V_2 = \sum_{k=1}^{n} m_k(R_k - R_k^*) (\gamma I_k - \delta R_k + \mu S_k)$$

$$= \sum_{k=1}^{n} m_k(R_k - R_k^*) \left[ \gamma(I_k - I_k^*) - \delta(R_k - R_k^*) + \mu(S_k - S_k^*) \right]$$

$$= \sum_{k=1}^{n} m_k(R_k - R_k^*) \left[ -\gamma(R_k - R_k^*) - \gamma(S_k - S_k^*) - \delta(R_k - R_k^*) + \mu(S_k - S_k^*) \right]$$

$$= -\sum_{k=1}^{n} (\gamma + \delta)m_k(R_k - R_k^*)^2 - \sum_{k=1}^{n} (\gamma - \mu)m_k(R_k - R_k^*)(S_k - S_k^*).$$

Using (3.7) and the identity $\delta = (\delta + \mu)S_k^* + \delta I_k^* + \lambda k S_k^* \frac{\Theta^*}{1 + \alpha(\Theta^*)^2}$, we have

$$V_1 = \sum_{k=1}^{n} a_k(S_k - S_k^*) \left( \delta - (\delta + \mu)S_k - \delta I_k - \lambda k S_k \frac{\Theta}{1 + \alpha(\Theta^2)} \right)$$

$$= \sum_{k=1}^{n} a_k(S_k - S_k^*) \left\{ - (\delta + \mu)(S_k - S_k^*) - \delta(I_k - I_k^*) + \lambda k \left[ \frac{S_k^* \Theta^*}{1 + \alpha(\Theta^2)^2} - \frac{S_k \Theta}{1 + \alpha(\Theta^2)} \right] \right\}$$

$$= \sum_{k=1}^{n} a_k(S_k - S_k^*) \left\{ - \mu(S_k - S_k^*) + \delta(R_k - R_k^*) + \lambda k \left[ \frac{S_k^* \Theta^*}{1 + \alpha(\Theta^2)^2} - \frac{S_k \Theta}{1 + \alpha(\Theta^2)} \right] \right\}$$

$$= -\sum_{k=1}^{n} \mu a_k(S_k - S_k^*)^2 + \sum_{k=1}^{n} \delta a_k(S_k - S_k^*)(R_k - R_k^*)$$

$$- \frac{\Theta}{1 + \alpha(\Theta^2)^2} \sum_{k=1}^{n} \lambda k a_k(S_k - S_k^*)^2 - \sum_{k=1}^{n} \lambda k a_k S_k^* \frac{\Theta^*}{1 + \alpha(\Theta^*^2)^2} (S_k - S_k^*) (\Theta - \Theta^*)$$

$$+ \sum_{k=1}^{n} \alpha \lambda k a_k S_k^* (S_k - S_k^*) \frac{\Theta^2 - (\Theta^*)^2}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)}.$$

Note that the final term of right hand side of the above equality can be written as

$$\sum_{k=1}^{n} \alpha \lambda k a_k S_k^* (S_k - S_k^*) \frac{\Theta^2 - (\Theta^*)^2}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)}$$

$$= \sum_{k=1}^{n} \alpha \lambda k a_k S_k^* (S_k - S_k^*) \left[ \frac{(\Theta - \Theta^*)(\Theta^2 - (\Theta^*)^2)}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)} + \frac{\Theta^*(\Theta^2 - (\Theta^*)^2)}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)} \right]$$

$$= \sum_{k=1}^{n} \alpha \lambda k a_k S_k^* (S_k - S_k^*) \frac{(\Theta - \Theta^*)(\Theta^2 - (\Theta^*)^2)}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)} - \sum_{k=1}^{n} \frac{\alpha \lambda k a_k S_k^* (S_k - S_k^*) (\Theta + \Theta^*)}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)} (\Theta - \Theta^*)^2$$

$$+ \sum_{k=1}^{n} \frac{\alpha \lambda k a_k S_k^* (S_k - S_k^*) (\Theta + \Theta^*)}{(1 + \alpha(\Theta^2)^2)(1 + \alpha(\Theta^2)^2)} (S_k - S_k^*) (\Theta - \Theta^*).$$
Substituting it into (3.12) yields

\[
V_1 = -\sum_{k=1}^{n} \mu a_k (S_k - S_k^*)^2 + \sum_{k=1}^{n} \delta a_k (S_k - S_k^*) (R_k - R_k^*)
\]
\[
- \frac{\Theta}{1 + \alpha \Theta^2} \sum_{k=1}^{n} \lambda k a_k (S_k - S_k^*)^2
\]
\[
+ \sum_{k=1}^{n} \frac{\alpha \lambda k a_k S_k^* \Theta^* (\Theta + \Theta^*)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} (S_k - S_k^*) (\Theta - \Theta^*)
\]
\[
- \sum_{k=1}^{n} \frac{\alpha \lambda k a_k (S_k^*)^2 (\Theta + \Theta^*)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} (\Theta - \Theta^*)^2
\]
\[
- \sum_{k=1}^{n} \lambda k a_k S_k^* \left[ \frac{(S_k - S_k^*)(\Theta - \Theta^*)}{1 + \alpha (\Theta^*)^2} - \frac{\alpha S_k (\Theta - \Theta^*)(\Theta^2 - (\Theta^*)^2)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} \right].
\]

(3.13)

Since \(a_k = \frac{k^{P(k)}}{(k)S_k^*}\), we find the final term of (3.13) is equal to \(-V_3\). Then combining (3.11), it follows that

\[
V' = -\sum_{k=1}^{n} a_k F_k(m) - \frac{\Theta}{1 + \alpha \Theta^2} \sum_{k=1}^{n} \lambda k a_k (S_k - S_k^*)^2
\]
\[
+ \sum_{k=1}^{n} \frac{\alpha \lambda k a_k S_k^* \Theta^* (\Theta + \Theta^*)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} (S_k - S_k^*) (\Theta - \Theta^*)
\]
\[
- \sum_{k=1}^{n} \frac{\alpha \lambda k a_k (S_k^*)^2 (\Theta + \Theta^*)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} (\Theta - \Theta^*)^2,
\]

(3.14)

where

\[
F_k(m) := \mu (S_k - S_k^*)^2 + [(\gamma - \mu)m - \delta] (R_k - R_k^*) (S_k - S_k^*) + (\gamma + \delta)m (R_k - R_k^*)^2.
\]

(3.15)

In the Appendix B, we show there exists \(m^* > 0\) such that

\[
F_k(m^*) \geq \omega(m^*) (S_k - S_k^*)^2 + \nu(m^*) (R_k - R_k^*)^2,
\]

(3.16)

where \(\omega(m^*), \nu(m^*) > 0\) are two constants shown in (B.5) and (B.6). For simplicity, in the following, we denote \(\omega(m^*)\) by \(\omega\), \(\nu(m^*)\) by \(\nu\) respectively.
Substituting (3.16) into (3.14), we have

\[ V' \leq -\sum_{k=1}^{n} \omega a_k (S_k - S_k^*)^2 - \sum_{k=1}^{n} \nu a_k (R_k - R_k^*)^2 - \frac{\Theta}{1 + \alpha \Theta^2} \sum_{k=1}^{n} \lambda k a_k (S_k - S_k^*)^2 + \sum_{k=1}^{n} \alpha \lambda k a_k S_k^*(\Theta + \Theta^*) (S_k - S_k^*) (\Theta - \Theta^*) \]

\[ + \sum_{k=1}^{n} \frac{\alpha \lambda k a_k S_k^*(\Theta + \Theta^*)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} (S_k - S_k^*) (\Theta - \Theta^*) \]

\[ - \sum_{k=1}^{n} \frac{\alpha \lambda k a_k (S_k^*)^2(\Theta + \Theta^*)}{(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} (\Theta - \Theta^*)^2 \]

\[ = - \frac{\Theta}{1 + \alpha \Theta^2} \sum_{k=1}^{n} \lambda k a_k (S_k - S_k^*)^2 - \sum_{k=1}^{n} \nu a_k (R_k - R_k^*)^2 \]

\[ - \sum_{k=1}^{n} \omega a_k \left\{ X_k^2 - \frac{\alpha \lambda k S_k^*(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} X_k Y + \frac{\alpha \lambda k (S_k^*)^2(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} Y^2 \right\} \tag{3.17} \]

where

\[ X_k = S_k - S_k^*, \quad Y = \Theta - \Theta^*. \tag{3.18} \]

Below we prove that

\[ X_k^2 - \frac{\alpha \lambda k S_k^*(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} X_k Y + \frac{\alpha \lambda k (S_k^*)^2(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} Y^2 \geq 0. \tag{3.19} \]

For this purpose, it suffices to show the following

\[ \Delta := \left[ \frac{\alpha \lambda k S_k^*(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} \right]^2 - 4 \frac{\alpha \lambda k (S_k^*)^2(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} \]

\[ = \frac{\alpha \lambda k (S_k^*)^2(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} \left[ \frac{\alpha \lambda k (\Theta^*)^2(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} - 4 \right] \leq 0. \tag{3.20} \]

Notice that $0 \leq \Theta, \Theta^* \leq 1$. Then when $\alpha \leq \frac{2\omega}{\lambda n}$, obviously we have

\[ \frac{\alpha \lambda k (\Theta^*)^2(\Theta + \Theta^*)}{\omega(1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2)} - 4 \leq \frac{2\alpha \lambda n}{\omega} - 4 \leq 0, \tag{3.21} \]

which implies (3.20).

On the other hand, by the inequalities $a^2 + b^2 \geq \frac{1}{2} (a + b)^2$ and $a^2 + b^2 \geq 2ab$, we have

\[ (1 + \alpha (\Theta^*)^2)(1 + \alpha \Theta^2) = 1 + \alpha \left[ \Theta^2 + (\Theta^*)^2 \right] + \alpha^2 \Theta^2 (\Theta^*)^2 \]

\[ \geq 1 + \alpha \left[ \Theta^2 + (\Theta^*)^2 \right] \]

\[ \geq 1 + \frac{\alpha}{2} (\Theta + \Theta^*)^2, \tag{3.22} \]
and
\[
I_k^* \leq \frac{\lambda \delta k \Theta^*}{\gamma(\delta + \mu)(1 + \alpha(\Theta^*)^2)} \leq \frac{\lambda \delta k}{2\sqrt{\alpha} \gamma(\delta + \mu)}, \quad k = 1, 2, \cdots, n.
\]

It follows from (3.23) that
\[
\Theta^* = \langle k \rangle^{-1} \sum_{k=1}^{n} k P(k) I_k^* \leq \frac{\lambda \delta}{2\sqrt{\alpha} \gamma(\delta + \mu)} \langle k \rangle \sum_{k=1}^{n} k^2 P(k) = \frac{R_0}{2 \sqrt{\alpha}}.
\]

Substituting (3.22) and (3.24) into (3.20) and using the assumption \( \alpha \geq \lambda^2 n^2 (R_0)^2 \), we obtain
\[
\alpha \lambda k (\Theta^*)^2 (\Theta + \Theta^*) \omega (1 + \alpha(\Theta^*)^2) - 4 \leq \frac{2 \lambda k \Theta^*}{\omega} - 4 \leq \frac{\lambda n R_0}{\omega \sqrt{\alpha}} - 4 \leq 0,
\]
which implies (3.20). Thus (3.19) holds when \( \alpha \geq \alpha_c \) or \( \alpha \leq \alpha_l \), so it follows from (3.17) that
\[
\frac{dV}{dt} \leq - \frac{\Theta}{1 + \alpha \Theta^2} \sum_{k=1}^{n} \lambda k a_k (S_k - S_k^*)^2 - \sum_{k=1}^{n} \nu \delta a_k (R_k - R_k^*)^2 \leq 0.
\]

Also \( \frac{dV}{dt} = 0 \) if and only if \( S_k = S_k^* \) and \( R_k = R_k^* \) for \( k = 1, 2, \cdots, n \). According to the LaSalle’s invariant principle [5], \((S_1^*, R_1^*, \ldots, S_n^*, R_n^*)\) is globally asymptotically stable, so is \((S_1^*, I_1^*, \ldots, S_n^*, I_n^*)\). The proof is completed.

Remark 3.1. From Theorem 3.2 we can see under the condition \( R_0 > 1 \), the endemic equilibrium is globally asymptotically stable if \( \alpha \geq \alpha_c \) or \( \alpha \leq \alpha_l \). In the section of numerical experiment, we find that it seems that in the case of \( R_0 > 1 \), for any \( \alpha > 0 \) the system (2.2) is globally attractive. Also it is worth noting when \( \alpha = 0 \), the proposed system (1.3) can be seen as a generalization to the model proposed in Chen and Sun [3]. The global stability of the endemic equilibrium is proved under no assumptions on the vaccination percentage \( \mu \) and the recovered rate \( \gamma \), which improves the results obtained in [3]. Although the parameter \( \alpha > 0 \) does not affect the epidemic threshold, we can show easily that
\[
\limsup_{t \to \infty} I_k(t) < \lambda k \frac{\sqrt{\alpha}}{2 \gamma},
\]
which together with equation (3.23), we can see that a larger \( \alpha \) can accelerate the extinction of the disease and reduce the level of disease, which is further verified in the numerical results.

4 Numerical results

In this section, we will give some numerical simulations to illustrate the theoretical findings. Our simulations are based on the BA network with maximum degree \( n = 500 \). The degree distribution \( P(k) = ck^{-m} \), and the constant \( c \) satisfying \( \sum_{k=1}^{n} P(k) = 1 \).
In all of the experiments below, we fix $m = 3, \lambda = 0.01$ and $\delta = 0.02$. In Fig.1 we choose $\gamma = 0.01$ and $\mu = 0.2$. By simple computations, we can derive $R_0 \approx 0.3759 < 1$. The time series of infected nodes with degree $k = 100, 200, \cdots, 500$ are shown in Fig.1(a) ($\alpha = 0.1$) and Fig.1(b) ($\alpha = 10$). The initial values are $S_k(0) = 0.3, I_k(0) = 0.1, R_k(0) = 0.6$ for any degree $k$. It can be seen that the disease eventually becomes extinct when $R_0 < 1$, which implies that the disease-free equilibrium is stable. Meanwhile it should be noted that, although for both choices of $\alpha > 0$, the density of the infected node with a bigger degree experiences a peak before it leads to the disease-free equilibrium, the larger $\alpha$ is, the lower the peak level is, which means that large $\alpha$ can effectively degrade the epidemic peak when it breaks out initially. To show the global stability of the disease-free equilibrium, we choose 10 different initial values to plot the time series of $I_{400}(t)$ in Fig.2. Obviously all trajectories converge to the trivial equilibrium and this supports the global stability of disease-free equilibrium. Also as is shown in Fig.2(b) when $R_0 < 1$, the larger $\alpha > 0$ is, the faster the disease dies out.

In Fig.3 and Fig.4, we choose $\mu = 0.001$ and $\gamma = 0.01$. Thus we have $\mu < \gamma$ and $R_0 \approx 3.9377 > 1$. Accordingly, the parameters in Theorem 3.2 are computed as $\omega = 0.0004, \alpha_l = 0.0002, \alpha_c \approx 1.3 \times 10^8$. So in this case we choose $\alpha = 10^{-4} < \alpha_l$ and $\alpha = 10^9 > \alpha_c$ respectively. As can be see from Fig.3 and Fig.5 in the case of $R_0 > 1$ the disease will persist on a positive steady level. Fig.4 and Fig.6 display the evolutions of $I_{400}(t)$ for a set of initial conditions. In both cases, as can be seen from Fig.3 to Fig.4, the endemic equilibrium $E^*$ is globally asymptotically stable whenever $R_0 > 1$ and $\alpha > \alpha_c$ or $\alpha < \alpha_l$, which is in agreement with Theorem 3.2. Again we can see in Fig.3 and Fig.5 when the disease is endemic, the densities of the infected nodes decreases as $\alpha$ increases, which means that a larger $\alpha$ can accelerate the extinction of the disease and reduce the level of disease.

When $R_0 > 1$, Theorem 3.2 shows the endemic equilibrium $E^*$ is globally asymptotically stable when $\alpha$ is sufficiently large or small enough. However, as is shown in Fig.7 for any
Figure 2: The time series of $I_{400}(t)$ with 10 different initial values and $R_0 < 1$.

Figure 3: The time series of infected nodes in (2.2) with $R_0 > 1$ and $\mu > \gamma$. 
Figure 4: The time series of $I_{400}(t)$ with 10 different initial values when $R_0 > 1$ and $\mu > \gamma$.

Figure 5: The time series of infected nodes in (2.2) with $R_0 > 1$ and $\mu < \gamma$. 
Figure 6: The time series of $I_{400}(t)$ with 10 different initial values when $R_0 > 1$ and $\mu < \gamma$.

Figure 7: The time series of infected nodes in (2.2) with $R_0 > 1$ and $\alpha = 10$.

When $\alpha > 0$ it seems that $E^*$ is indeed attractive when $R_0 > 1$. However, the rigorous proof to this conclusion is still mathematically difficult which will be considered later.

5 Conclusion

In this paper, we have discussed an SIRS epidemic model with vaccination and nonmonotone incidence rate on complex networks. 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 taken by the susceptible ones. Although the parameter $\alpha$ does not affect the epidemic threshold, it plays a role in weakening the spreading of disease, as can be seen in equations (3.23) and (3.26). We have shown by Lyapunov function that without additional conditions on the constants $\mu$ and $\gamma$, the endemic equilibrium of system (1.3) is globally asymptotically stable, thus the disease becomes endemic. The results can be viewed as an important supplement to the result in [3].
Furthermore, numerical simulations are done. As has been seen, the simulations verify the globally asymptotical stability of $E^*$ and controlling effect of the inhibitory factor $\alpha > 0$ on reducing the disease level.

**Acknowledgments**

The research was supported in part by the NSFC (grants 61002039, 61572018, 11571062), the Program for Liaoning Excellent Talents in University (grant LJQ2013124) and the Fundamental Research Fund for the Central Universities (grants DC201502050404, DC201502050202).

**Appendix A  Proof of Lemma 2.1**

Since $S_k(0) > 0$ for $k = 1, 2, \cdots, n$, by continuity, we have that for any $k$, there exists some $t_k > 0$ such that $S_k(t) > 0$ for any $t \in (0, t_k)$. Thus, the set $\mathcal{E}_k := \{\tau > 0; S_k(t) > 0, \forall t \in (0, \tau)\}$ is not empty for any $k = 1, 2, \cdots, n$. Let $\alpha_k = \sup \mathcal{E}_k$ for $k = 1, 2, \cdots, n$. Then $\alpha_k > 0$ for $k = 1, 2, \cdots, n$.

We will show $S_k(t) > 0$ for all $t > 0$ and all $k = 1, 2, \cdots, n$. To prove this, it suffices to show that $\alpha_k = \infty$ for $k = 1, 2, \cdots, n$. Suppose, on the contrary, that $\alpha_m < \infty$ for some $m \in \{1, 2, \cdots, n\}$. Then $S_m(t) > 0$ for all $t \in (0, \alpha_m)$. By continuity, there must have $S_m(\alpha_m) = 0$. In the following, we will show this is not true.

Firstly, we will prove $\Theta(t) > 0$ for all $t > 0$. From the second equation of (2.2), we have

$$
\frac{d\Theta(t)}{dt} = \left[ \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{n} k^2 P(k) \frac{S_k(t)}{1 + \Theta^2(t)} - \gamma \right] \Theta(t) =: X(t)\Theta(t),
$$

which gives

$$
\Theta(t) = \Theta(0) \exp \left\{ \int_{0}^{t} X(s) ds \right\} > 0, \quad \forall t > 0.
$$

Thus, for any $t \in (0, \alpha_m)$, by the second equation of (2.2), we also obtain that

$$
\frac{d}{dt} (\exp\{\gamma t\} I_m(t)) = \lambda m S_m(t) \frac{\Theta(t)}{1 + \alpha \Theta^2(t)} \exp\{\gamma t\} > 0,
$$

which means that $I_m(t) \geq 0$ for all $t \in (0, \alpha_m)$.

Next we will prove that $S_m(t) + I_m(t) < 1$ for all $t \in (0, \alpha_m]$. Adding the two equations in (2.2) yields

$$
\frac{d(S_k + I_k)}{dt} = \delta - (\delta + \mu)(S_k + I_k) + (\mu - \gamma)I_k, \quad k = 1, 2, \cdots, n,
$$

which is equivalent to

$$
\frac{d(S_k + I_k)}{dt} = \delta - (\delta + \gamma)(S_k + I_k) + (\gamma - \mu)I_k, \quad k = 1, 2, \cdots, n.
$$

If $\mu \leq \gamma$, we derive from (A.3) with $k = m$ that

$$
\frac{d(S_m + I_m)}{dt} \leq \delta - (\delta + \mu)(S_m + I_m)
$$

$$
< (\delta + \mu)(1 - S_m - I_m), \quad \forall t \in (0, \alpha_m].
$$
We now show there exists \( m^* \) from (A.4) that 

\[
I_{X} \text{Denote}
\]

Appendix B Proof of equation (3.16)

Denote \( X = R_k - R_k^* \) and \( Y = S_k - S_k^* \). Then following the expression of \( F_k(m) \) in (3.16), we have

\[
F_k(m) = (\gamma + \delta)mX^2 + [(\gamma - \mu)m - \delta]XY + \mu Y^2
\]

\[
= (\gamma + \delta)m \left[ X + \frac{(\gamma - \mu)m - \delta}{2(\gamma + \delta)m}Y \right]^2 + 2\omega(m)Y^2
\]

\[
= \mu \left[ Y + \frac{(\gamma - \mu)m - \delta}{2\mu}X \right]^2 + 2\nu(m)X^2,
\]

where \( \omega(m) = -\frac{\triangle(m)}{8(\gamma + \delta)m} \) and \( \nu(m) = -\frac{\triangle(m)}{8\mu} \) with

\[
\triangle(m) = [(\gamma - \mu)m - \delta]^2 - 4\mu(\gamma + \delta)m
\]

\[
= (\gamma - \mu)^2m^2 - [2\delta(\gamma - \mu) + 4\mu(\gamma + \delta)]m + \delta^2.
\]

We now show there exists \( m^* > 0 \) such that \( \triangle(m^*) < 0 \). We prove this by two parts.

1. If \( \mu = \gamma \), we can choose \( m^* = \frac{\delta}{4\mu} > 0 \). It is easy to see \( \triangle(m^*) = -\gamma\delta < 0 \).

2. If \( \mu \neq \gamma \), let \( m^* = \frac{\delta + \mu + 2\nu(\gamma + \delta)}{(\gamma - \mu)^2} > 0 \), obviously we have

\[
\triangle(m^*) = -\frac{4\mu\gamma(\gamma + \delta)(\delta + \mu)}{(\gamma - \mu)^2} < 0.
\]
Thus the above claim is proved. So we have $\omega(m^*) > 0$ and $\nu(m^*) > 0$, from which we have

$$F_k(m^*) \geq \omega(m^*)(S_k - S_k^*)^2 + \nu(m^*)(R_k - R_k^*)^2,$$

(B.4)

with

$$\omega(m^*) = -\frac{\Delta(m^*)}{8(\gamma + \delta)m^*},$$

(B.5)

and

$$\nu(m^*) = -\frac{\Delta(m^*)}{8\mu}.$$

(B.6)

References

[1] S. Bansal, B. T. Grenfell, and L. A. Meyers. When individual behaviour matters: homogeneous and network models in epidemiology. *Journal of the Royal Society Interface*, 4(16):879–891, 2007.

[2] H. Chen and J. Sun. Global stability of delay multigroup epidemic models with group mixing and nonlinear incidence rates. *Applied Mathematics and Computation*, 218(8):4391–4400, 2011.

[3] L. Chen and J. Sun. Global stability and optimal control of an sirs epidemic model on heterogeneous networks. *Physica A: Statistical Mechanics and its Applications*, 410:196–204, 2014.

[4] S. Huang, F. Chen, and L. Chen. Global dynamics of a network-based siqrs epidemic model with demographics and vaccination. *Communications in Nonlinear Science and Numerical Simulation*, 43:296–310, 2017.

[5] J. LaSalle. *The Stability of Dynamical Systems*. SIAM, Philadelphia, PA, 1976.

[6] C.-H. Li. Dynamics of a network-based sis epidemic model with nonmonotone incidence rate. *Physica A: Statistical Mechanics and its Applications*, 427:234–243, 2015.

[7] C.-H. Li, C.-C. Tsai, and S.-Y. Yang. Analysis of epidemic spreading of an sirs model in complex heterogeneous networks. *Communications in Nonlinear Science and Numerical Simulation*, 19(4):1042–1054, 2014.

[8] T. Li, Y. Wang, and Z.-H. Guan. Spreading dynamics of a siqrs epidemic model on scale-free networks. *Communications in Nonlinear Science and Numerical Simulation*, 19(3):686–692, 2014.

[9] J. Liu and T. Zhang. Epidemic spreading of an seirs model in scale-free networks. *Communications in Nonlinear Science and Numerical Simulation*, 16(8):3375–3384, 2011.

[10] M. Liu and J. Run. Modelling the spread of sexually transmitted diseases on scale-free networks. *Chinese Physics B*, 18(6):2115–2120, 2009.

[11] Y. Moreno, R. Pastor-Satorras, and A. Vespignani. Epidemic outbreaks in complex heterogeneous networks. *European Physical Journal B*, 26(4):521–529, 2002.
[12] Y. Muroya, Y. Enatsu, and Y. Nakata. Global stability of a delayed SIRS epidemic model with a non-monotonic incidence rate. *Journal of Mathematical Analysis and Applications*, 377(1):1–14, 2011.

[13] M. Newman. Spread of epidemic disease on networks. *Physical Review E - Statistical Physics, Plasmas, Fluids, and Related Interdisciplinary Topics*, 66(1), 2002.

[14] R. Pastor-Satorras and A. Vespignani. Epidemic spreading in scale-free networks. *Physical Review Letters*, 86(14):3200–3203, 2001.

[15] R. Pastor-Satorras and A. Vespignani. Epidemic dynamics in finite size scale-free networks. *Physical Review E - Statistical Physics, Plasmas, Fluids, and Related Interdisciplinary Topics*, 65(3), 2002.

[16] X. Peng, X. Xu, X. Fu, and T. Zhou. Vaccination intervention on epidemic dynamics in networks. *Physical Review E*, 87(2):022813, 2013.

[17] L. Wang and G.-Z. Dai. Global stability of virus spreading in complex heterogeneous networks. *SIAM Journal on Applied Mathematics*, 68(5):1495–1502, 2008.

[18] Y. Wang, Z. Jin, Z. Yang, Z.-K. Zhang, T. Zhou, and G.-Q. Sun. Global analysis of an sis model with an infective vector on complex networks. *Nonlinear Analysis: Real World Applications*, 13(2):543–557, 2012.

[19] X. Wei, L. Liu, and W. Zhou. Global stability and attractivity of a network-based SIS epidemic model with nonmonotone incidence rate. *Physica A: Statistical Mechanics and its Applications*, 469:789–798, 2017.

[20] X. Wei, G. Xu, L. Liu, and W. Zhou. Global stability of endemic equilibrium of an epidemic model with birth and death on complex networks. *Physica A: Statistical Mechanics and its Applications*, 477:78–84, 2017.

[21] D. Xiao and S. Ruan. Global analysis of an epidemic model with nonmonotone incidence rate. *Mathematical Biosciences*, 208(2):419–429, 2007.

[22] W. Yang, C. Sun, and J. Arino. Global analysis for a general epidemiological model with vaccination and varying population. *Journal of Mathematical Analysis and Applications*, 372(1):208–223, 2010.

[23] H. Yuan, G. Liu, and G. Chen. On modeling the crowding and psychological effects in network-virus prevalence with nonlinear epidemic model. *Applied Mathematics and Computation*, 219(5):2387–2397, 2012.

[24] J. Zhang and J. Sun. Stability analysis of an sis epidemic model with feedback mechanism on networks. *Physica A: Statistical Mechanics and its Applications*, 394:24–32, 2014.

[25] G. Zhu, G. Chen, and X. Fu. Effects of active links on epidemic transmission over social networks. *Physica A: Statistical Mechanics and its Applications*, 468:614 – 621, 2017.
[26] G. Zhu, X. Fu, and G. Chen. Global attractivity of a network-based epidemic sis model with nonlinear infectivity. *Communications in Nonlinear Science and Numerical Simulation*, 17(6):2588–2594, 2012.