Research article

Dynamical analysis of a network-based SIR model with saturated incidence rate and nonlinear recovery rate: an edge-compartmental approach

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Abstract: A new network-based SIR epidemic model with saturated incidence rate and nonlinear recovery rate is proposed. We adopt an edge-compartmental approach to rewrite the system as a degree-edge-mixed model. The explicit formula of the basic reproduction number R_0 is obtained by renewal equation and Laplace transformation. We find that $R_0 < 1$ is not enough to ensure global asymptotic stability of the disease-free equilibrium, and when $R_0 > 1$, the system can exist multiple endemic equilibria. When the number of hospital beds is small enough, the system will undergo backward bifurcation at $R_0 = 1$. Moreover, it is proved that the stability of feasible endemic equilibrium is determined by signs of tangent slopes of the epidemic curve. Finally, the theoretical results are verified by numerical simulations. This study suggests that maintaining sufficient hospital beds is crucial for the control of infectious diseases.

Keywords: edge-compartmental approach; network; SIR model; backward bifurcation; saturated incidence rate; nonlinear recovery rate

1. Introduction

In the modeling of infectious diseases, incidence rate and recovery rate are two important factors that affect the dynamical behavior of the models. The bilinear incidence rate βSI are frequently used to describe the epidemic transmission process, where β is the transmission rate, S and I represent the numbers of susceptible and infective respectively. But, it is unrealistic when the number of infective individuals is large. To study the cholera epidemic spread in Bari in 1973, Capasso and Serio [1]

proposed a saturated incidence rate:

$$\frac{\beta S I}{1 + \alpha I},\tag{1.1}$$

where βI describes the infection force of the disease, $\frac{1}{1+\alpha I}$ measures the inhibition effect from the behavioral change of the susceptible individuals as the number of infected gradually increases.

In classical epidemic models, the recovery rate is usually assumed to be proportional to the number of infected, which means that medical resources are plentiful for the infectious diseases [2]. In fact, when diseases break out, medical resources tend to be scarce [3–5]. In order to study the influence of hospital beds on infectious diseases, the following nonlinear recovery rate function [5] is proposed by Shan and Zhu:

$$\gamma(b, I) = \gamma_0 + \frac{b(\gamma_1 - \gamma_0)}{b + I},\tag{1.2}$$

where γ_0 and γ_1 are the minimum and maximum per-capita recovery rates respectively, b denotes the number of hospital beds. An SIR model with (1.2) is studied, and it is found that backward bifurcation, saddle-node bifurcation, Hopf bifurcation and cusp type of Bogdanov-Takens bifurcation of codimension 3 will happen with different values of b [5].

Cui et al. studied an SIR epidemic model (system (1.3)) with saturate incidence rate and nonlinear recovery rate [6]. It is proved that when the number of hospital beds is small enough, system (1.3) can take place backward bifurcation, saddle-node bifurcation and Hopf bifurcation.

$$\begin{aligned}
S'(t) &= \Lambda - \frac{\beta S I}{1 + \alpha I} - \mu S,\\
I'(t) &= \frac{\beta S I}{1 + \alpha I} - \mu I - \epsilon I - \gamma(b, I)I,\\
R'(t) &= \gamma(b, I)I - \mu R,
\end{aligned}\tag{1.3}$$

where $S(t), I(t), R(t)$ are the numbers of susceptible, infected, recovered at time t respectively, $\gamma(b, I)$ is defined in (1.2). Besides, Λ is the birth rate; the interpretations of β and α are the same as in [1]; μ denotes the natural death rate; ϵ represents the disease-induced death rate.

To describe the heterogeneity of contact, complex networks are therefore incorporated into the epidemic models. Li and Yousef studied a network-based SIR epidemic model with saturated treatment function [7]. Huang and Li studied the complex dynamical properties of a network-based SIS epidemic model with saturated treatment function [8]. A condition which can determine the direction of bifurcation at $R_0 = 1$ is derived in [7, 8]. Wang and Yang [9] employed an edge-compartmental approach which can reduce the dimension of a mean-field vector-borne model to research the global dynamics of the system. Wang and Yang [10] proposed a degree-edge-mixed SIS model and used a novel geometric method to completely investigate the stability of feasible equilibrium. Based on the SIR model [7], Yang et al. adopted the edge-compartmental approach to prove the existence of backward bifurcation and deeply analyzed the local stability of each endemic equilibrium [11].

To our knowledge, few people combined saturated incidence rate and nonlinear recovery rate to discuss the dynamics of network-based epidemic models. So the research of this paper is still valuable, the highlights are summarized as follows:

• A new network-based SIR epidemic model is established and then reduced to a degree-edge-mixed model by an edge-compartmental approach;

• A necessary and sufficient condition which determines the existence of backward bifurcation at $R_0 = 1$ is derived;

• The stability of the feasible endemic equilibrium is proved by a geometric approach.

We know that time delay exists in the epidemic spreading, Jiang and Zhou [12] studied the influence of time delay on epidemic spreading under limited resources. They found that time delay can induce first-order, continuous and hybrid phases and a small resources amount can effectively control the spread of infectious diseases if the delay exceeds a threshold. The stability of nonlinear systems with time delay has been studied in the literature [13–17]. Yang et al. obtained the existence of a positive periodic solution for neutral-type integral differential equation arising in epidemic model with time-varying delay [18]. Of course, we do not consider the delay factor in our paper, which will be a worthy of attention and research work.

The structure of this paper is as follows. In Section 2, a new SIR epidemic model is proposed on complex networks. Besides, the dimension of the system is reduced by the edge-compartmental method. In Section 3, the existence and stability of the disease-free equilibrium and endemic equilibrium are studied. The results of numerical simulations are given in Section 4. Lastly, conclusions and discussions are presented in Section 5.

2. Model description

In this section, we extend model (1.3) to the network. Furthermore, disease-induced death rate is not considered and the susceptible individuals will not be infected once vaccinated. The other parameters are exactly the same as in system (1.3) and we show the interpretation of the parameters in Table 1 for better visualization. All the nodes are classified into n groups and the nodes in the same group have the same degree. Suppose that $S_k(t), I_k(t), R_k(t)$ be the densities of susceptible, infected, recovered with degree k at time t respectively. So, the specific mean-field equations of network-based SIR model are as follows:

$$
\begin{aligned}
S_k'(t) &= \Lambda - \frac{\beta k S_k(t)\theta(t)}{1 + \alpha\theta(t)} - \mu S_k(t) - \eta S_k(t), \\
I_k'(t) &= \frac{\beta k S_k(t)\theta(t)}{1 + \alpha\theta(t)} - \mu I_k(t) - \left(\gamma_0 + \frac{b(\gamma_1 - \gamma_0)}{b + \theta(t)}\right)I_k(t), \\
R_k'(t) &= \left(\gamma_0 + \frac{b(\gamma_1 - \gamma_0)}{b + \theta(t)}\right)I_k(t) - \mu R_k(t) + \eta S_k(t),
\end{aligned}
\tag{2.1}
$$

with $\theta(t)$ is the probability that any given edge is connected to an infected node, assume the network is uncorrelated, then

$$
\theta(t) = \frac{1}{\langle k \rangle}\sum_{k=1}^{n} k p(k) I_k(t),
$$

where $p(k)$ is the probability that a node is connected to k other nodes and $\langle k \rangle = \sum_{k=1}^{n} k p(k)$ denotes the mean degree of the network. It is supposed that newborns are balanced by deaths, that is $\Lambda = \mu$.

Inspired by the edge-compartmental method [9–11], we can rewrite system (2.1) as the following degree-edge-mixed model:

$$S'_k(t) = \mu - \frac{\beta k S_k(t)\theta(t)}{1 + \alpha\theta(t)} - \mu S_k(t) - \eta S_k(t),$$

$$\theta'(t) = \frac{\beta}{\langle k \rangle} \frac{\theta(t)}{1 + \alpha\theta(t)} \sum_{k=1}^{n} k^2 p(k) S_k(t) - \mu\theta(t) - \Big(\gamma_0 + \frac{b(\gamma_1 - \gamma_0)}{b + \theta(t)}\Big)\theta(t). \quad (2.2)$$

Remark 1. The dimension of degree-edge-mixed model (2.2) is much lower than system (2.1). So the process of analysis presented in this paper will be relatively concise.

Table 1. Definition of parameters.

Parameter	Definition
μ	The birth rate / The death rate
β	Transmission rate of infected individuals
α	The saturated coefficient
b	The number of hospital beds
γ_0	The minimum per-capita recovery rate
γ_1	The maximum per-capita recovery rate
η	Proportion that has been vaccinated

Lemma 1. If $\theta(0) > 0, S_k(0) > 0$, then for all $k \in N$ and $t \in R_+$, we have $S_k(t) > 0, \theta(t) > 0$.

Proof. Firstly, we prove that $\theta(t) > 0$ for all $t > 0$. From the $(n + 1)$th equation of system (2.2), we have $\theta(t) = \theta(0)e^{\int_0^t z(\tau)d\tau}, z(t) = \frac{\beta}{\langle k \rangle(1+\alpha\theta(t))} \sum_{k=1}^{n} k^2 p(k) S_k(t) - \mu - \gamma_0 - \frac{b(\gamma_1-\gamma_0)}{b+\theta(t)}$. Since $\theta(0) > 0$, we can deduce $\theta(t) > 0$ for all $t > 0$.

Note that $S_k(0) > 0$, in view of the continuity of $S_k(t)$, we can find a small $\delta > 0$, such that $S_k(t) > 0$ for $t \in (0, \delta)$. Now we demonstrate that $S_k(t) > 0$ for all $t > 0$. If not, we may assume that there exists $t_0 \geq \delta > 0$, s.t. $S_k(t_0) = 0$ and $S_k(t) > 0$ for $t \in (0, t_0)$. Thus, from the first n equation of system (2.2), we can obtain $S'_k(t_0) = \mu > 0$. This leads to $S_k(t) < 0$ for some $t \in (0, t_0)$, which is apparently a contradiction. Therefore, $S_k(t) > 0$ for all $t > 0$.

Remark 2. From the result of Lemma 1 and the fact $S_k(t) + I_k(t) + R_k(t) = 1$, we can assert that $\Omega := \{(S_1, S_2, \cdots, S_n, \theta) \mid 0 < S_k(t) \leq 1, 0 \leq \theta(t) < 1\}$ is a positive invariant set of system (2.2).

3. Existence and stability of equilibrium

System (2.2) has a unique disease-free equilibrium given by $E_0 = (\frac{\mu}{\mu+\eta}, \frac{\mu}{\mu+\eta}, \cdots, \frac{\mu}{\mu+\eta}, 0) \in R^{n+1}$. Linearizing the $(n + 1)$th equation of system (2.2) at E_0 yields

$$\theta'(t) = \Big(\frac{\beta\mu\langle k^2 \rangle}{(\mu + \eta)\langle k \rangle} - \mu - \gamma_1\Big)\theta(t). \quad (3.1)$$

Solving Eq (3.1) by a constant variation method, we can build the following equation:

$$\theta(t) = \theta(0)e^{-(\mu+\gamma_1)t} + \frac{\beta\mu\langle k^2 \rangle}{(\mu + \eta)\langle k \rangle} \int_0^t e^{(\mu+\gamma_1)(s-t)}\theta(s)ds.$$

Using the approach introduced in [19], we can calculate the basic reproduction number $\boldsymbol{R_0}$ by taking Laplace transform:

$$\boldsymbol{R_0} = \frac{\beta\mu\langle k^2\rangle}{(\mu+\eta)\langle k\rangle}\int_0^{+\infty} e^{-(\mu+\gamma_1)t}dt = \frac{\beta\mu\langle k^2\rangle}{(\mu+\eta)(\mu+\gamma_1)\langle k\rangle},$$

where $\langle k^2\rangle = \sum\limits_{k=1}^n k^2 p(k)$.

3.1. Stability of the disease-free equilibrium

The local and global stability of the disease-free equilibrium E_0 will be proved in this subsection.
Theorem 1. If $\boldsymbol{R_0} < 1$, the disease-free equilibrium E_0 of system (2.2) is locally asymptotically stable, whereas if $\boldsymbol{R_0} > 1$, E_0 is unstable.
Proof. Linearizing system (2.2) at E_0 yields to

$$
\begin{aligned}
S_k'(t) &= (-\mu-\eta)S_k(t) - \frac{\beta k\mu}{\mu+\eta}\theta(t), \\
\theta'(t) &= \Big(\frac{\beta\mu\langle k^2\rangle}{(\mu+\eta)\langle k\rangle} - \mu - \gamma_1\Big)\theta(t).
\end{aligned}
\tag{3.2}
$$

Furthermore, the Jacobian matrix of Eq (3.2) is

$$
J(E_0) = \begin{pmatrix}
-\mu-\eta & 0 & \cdots & 0 & \frac{-\beta\mu}{\mu+\eta} \\
0 & -\mu-\eta & \cdots & 0 & \frac{-2\beta\mu}{\mu+\eta} \\
\vdots & \vdots & \ddots & \vdots & \vdots \\
0 & 0 & \cdots & -\mu-\eta & \frac{-n\beta\mu}{\mu+\eta} \\
0 & 0 & \cdots & 0 & \frac{\beta\mu\langle k^2\rangle}{(\mu+\eta)\langle k\rangle}-\mu-\gamma_1
\end{pmatrix}_{(n+1)\times(n+1)}.
$$

Obviously, $\lambda = -(\mu+\eta)$ is n-multiple negative eigenvalue. So, the stability of E_0 is determined by the following eigenvalue:

$$\lambda = \frac{\beta\mu\langle k^2\rangle}{(\mu+\eta)\langle k\rangle} - \mu - \gamma_1 = (\mu+\gamma_1)(\boldsymbol{R_0}-1).$$

Therefore, all eigenvalues of the Jacobian matrix $J(E_0)$ are negative when $\boldsymbol{R_0} < 1$, and hence, E_0 is locally asymptotically stable. By contrast, if $\boldsymbol{R_0} > 1$, E_0 is unstable.

Next, we will prove the global stability of the disease-free equilibrium E_0.
Theorem 2. Denote $\hat{\boldsymbol{R}}_0 = \frac{\beta(b+1)\langle k^2\rangle}{(\mu+b\mu+\gamma_0+b\gamma_1)\langle k\rangle}$, if $\hat{\boldsymbol{R}}_0 \leq 1$, then E_0 is globally asymptotically stable.

Proof. Let a Lyapunov function $V(t) = \theta(t)$, then

$$
\begin{aligned}
V'(t) =& \frac{\beta\theta(t)}{\langle k\rangle(1+\alpha\theta(t))} \sum_{k=1}^{n} k^2 p(k) S_k(t) - \mu\theta(t) - \Big(\gamma_0 + \frac{b(\gamma_1-\gamma_0)}{b+\theta(t)}\Big)\theta(t) \\
\leq& \frac{\beta\langle k^2\rangle}{\langle k\rangle}\theta(t) - \mu\theta(t) - \Big(\gamma_0 + \frac{b(\gamma_1-\gamma_0)}{b+\theta(t)}\Big)\theta(t) \\
=& \Big[\frac{\beta\langle k^2\rangle}{\langle k\rangle} - \mu - \gamma_0 - \frac{b(\gamma_1-\gamma_0)}{b+1} - \frac{b(\gamma_1-\gamma_0)(1-\theta(t))}{(b+1)(b+\theta(t))}\Big]\theta(t) \\
\leq& \Big[\beta\frac{\langle k^2\rangle}{\langle k\rangle} - \mu - \gamma_0 - \frac{b(\gamma_1-\gamma_0)}{b+1}\Big]\theta(t) \\
=& \Big[\mu + \gamma_0 + \frac{b(\gamma_1-\gamma_0)}{b+1}\Big](\hat{\boldsymbol{R}}_0 - 1)\theta(t),
\end{aligned}
$$

if $\hat{\boldsymbol{R}}_0 < 1$, $V'(t) \leq 0$; if $\hat{\boldsymbol{R}}_0 = 1$, then $V'(t) \leq -\frac{b(\gamma_1-\gamma_0)(1-\theta(t))}{(b+1)(b+\theta(t))}\theta(t) \leq 0$. The equality holds if and only if $\theta(t) = 0$. Hence, we can conclude that the disease-free equilibrium E_0 is globally asymptotically stable if $\hat{\boldsymbol{R}}_0 \leq 1$.

Remark 3. Because $\gamma_0 < \gamma_1$, so $\boldsymbol{R}_0 = \frac{\mu(\mu+b\mu+\gamma_0+b\gamma_1)}{(\mu+\eta)(\mu+b\mu+\gamma_1+b\gamma_1)}\hat{\boldsymbol{R}}_0 < \hat{\boldsymbol{R}}_0$.

Remark 4. If $\hat{\boldsymbol{R}}_0 < 1$, that is $\boldsymbol{R}_0 < \frac{\mu(\mu+b\mu+\gamma_0+b\gamma_1)}{(\mu+\eta)(\mu+b\mu+\gamma_1+b\gamma_1)} < 1$, then E_0 is globally asymptotically stable.

Remark 5. It seems that the condition $\boldsymbol{R}_0 < 1$ is not sufficient to ensure the global asymptotic stability of E_0. So system (2.2) may exist backward bifurcation at $\boldsymbol{R}_0 = 1$ which will be proved later.

3.2. Existence of endemic equilibrium

The existence of feasible positive equilibrium will be proved in this subsection.

Theorem 3. If $\boldsymbol{R}_0 > 1$, system (2.2) admits at least an endemic equilibrium.

Proof. Assume that $E^* = (S_k^*, \theta^*), k = 1, 2, \ldots, n$, is an endemic equilibrium of system (2.2), then E^* should satisfy

$$
\begin{aligned}
0 =& \mu - \frac{\beta k S_k^* \theta^*}{1 + \alpha\theta^*} - \mu S_k^* - \eta S_k^*, \\
0 =& \frac{\beta\theta^*}{\langle k\rangle(1+\alpha\theta^*)} \sum_{k=1}^{n} k^2 p(k) S_k^* - \mu\theta^* - \Big(\gamma_0 + \frac{b(\gamma_1-\gamma_0)}{b+\theta^*}\Big)\theta^*.
\end{aligned}
\tag{3.3}
$$

Then we can obtain $S_k^* = \frac{\mu(1+\alpha\theta^*)}{\beta k\theta^* + (\mu+\eta)(1+\alpha\theta^*)}, k = 1, 2, \ldots, n$. Substituting S_k^* into the $(n+1)$th equation of (3.3), then we can get a self-consistency equation:

$$
F(\theta^*) := \mu + \gamma_0 + \frac{b(\gamma_1-\gamma_0)}{b+\theta^*} - \frac{\beta}{\langle k\rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu}{\beta k\theta^* + (\mu+\eta)(1+\alpha\theta^*)} = 0.
\tag{3.4}
$$

Since $\boldsymbol{R}_0 > 1$, $F(0) < 0$, $F(1) > 0$, using the Immediate Value Theorem, we can conclude that Eq (3.4) has at least one positive root in $(0, 1)$. This implies that system (2.2) admits at least an endemic equilibrium.

Remark 6. If $\boldsymbol{R}_0 \leq 1$, then $F(0) \geq 0$, $F(1) > 0$, we can not conclude whether system (2.2) exists endemic equilibrium by the method of the Immediate Value Theorem. However, we already know

that E_0 is not globally asymptotically stable, so system (2.2) may also exist endemic equilibrium when $R_0 \leq 1$. In other words, the system may take place backward bifurcation at $R_0 = 1$ which will be proved in Theorem 4.

Next, we will derive a necessary and sufficient condition which determines the existence of backward bifurcation at $R_0 = 1$.

Theorem 4. System (2.2) exists backward bifurcation at $R_0 = 1$ if and only if $b < \hat{b}$; the system undergoes forward bifurcation if and only if $b > \hat{b}$, where

$$\hat{b} = \frac{\mu(\gamma_1 - \gamma_0)\langle k^2\rangle^2}{(\mu + \gamma_1)[\mu\alpha\langle k^2\rangle^2 + (\mu + \gamma_1)\langle k\rangle\langle k^3\rangle]}, \langle k^3\rangle = \sum_{k=1}^{n} k^3 p(k).$$

Proof. The endemic equilibrium should satisfy Eq (3.4). Replacing β by $\frac{(\mu+\eta)(\mu+\gamma_1)\langle k\rangle R_0}{\mu\langle k^2\rangle}$ and we can obtain the following equation:

$$\mu + \gamma_0 + \frac{b(\gamma_1 - \gamma_0)}{b + \theta^*} - (\mu + \eta)(\mu + \gamma_1)R_0 \sum_{k=1}^{n} k^2 p(k)\frac{\mu}{Q_0} = 0, \tag{3.5}$$

where $Q_0 = (\mu + \eta)(\mu + \gamma_1)\langle k\rangle k\theta^* R_0 + (\mu + \eta)(1 + \alpha\theta^*)\mu\langle k^2\rangle$.

If we keep in mind that θ^* is a function of R_0, then the direction of bifurcation is depended on the sign of $\frac{\partial\theta^*}{\partial R_0}\big|_{(R_0,\theta^*)=(1,0)}$. More exactly, if $\frac{\partial\theta^*}{\partial R_0}\big|_{(R_0,\theta^*)=(1,0)} < 0$, then backward bifurcation occurs at $R_0 = 1$. Conversely, the forward bifurcation happens.

Next, taking the derivative of Eq (3.5) associated with R_0 by the implicit function theorem, then yields to the following equation:

$$\frac{-b(\gamma_1 - \gamma_0)\frac{\partial\theta^*}{\partial R_0}}{(b + \theta^*)^2} + (\mu + \eta)(\mu + \gamma_1)\Big[\sum_{k=1}^{n} \frac{k^2 p(k)(R_0 Q_1 - \mu Q_0)}{Q_0^2}\Big] = 0, \tag{3.6}$$

where $Q_1 = \mu(\mu + \eta)[(\mu + \gamma_1)\langle k\rangle k\theta^* + (\mu + \gamma_1)\langle k\rangle k\frac{\partial\theta^*}{\partial R_0}R_0 + \mu\alpha\langle k^2\rangle\frac{\partial\theta^*}{\partial R_0}]$.

Substituting $(R_0, \theta^*) = (1, 0)$ into Eq (3.6), we can obtain the following equation by simple calculation and arrangement:

$$\frac{\partial\theta^*}{\partial R_0}\Big|_{(R_0,\theta^*)=(1,0)} = \frac{b\mu(\mu + \gamma_1)\langle k^2\rangle^2}{b(\mu + \gamma_1)^2\langle k\rangle\langle k^3\rangle + b\mu\alpha(\mu + \gamma_1)\langle k^2\rangle^2 - \mu(\gamma_1 - \gamma_0)\langle k^2\rangle^2}.$$

Thus if

$$\frac{\partial\theta^*}{\partial R_0}\Big|_{(R_0,\theta^*)=(1,0)} < 0 \Leftrightarrow b < \frac{\mu(\gamma_1 - \gamma_0)\langle k^2\rangle^2}{(\mu + \gamma_1)[\mu\alpha\langle k^2\rangle^2 + (\mu + \gamma_1)\langle k\rangle\langle k^3\rangle]}.$$

The same holds true also for the other proposition.

Remark 7. It is a fact that the number of hospital beds does play a key role in determining whether there is a backward bifurcation at $R_0 = 1$. More precisely, if b is small enough to satisfy the result of Theorem 4, backward bifurcation will occur. In this case, there will also exist endemic equilibrium even if $R_0 < 1$ (see Figure 2(b)). That is to say, $R_0 < 1$ is not sufficient to eradicate the diseases from the population, which is unfavorable to the control of infectious diseases.

Otherwise, when b is big enough, forward bifurcation will take place at $\boldsymbol{R_0} = 1$. In this case, E_0 will be globally asymptotically stable when $\boldsymbol{R_0} < 1$. This also shows the existence of the threshold for the number of hospital beds. When the relevant departments provide enough hospital beds such that $b > \hat{b}$, infectious diseases can be completely eliminated if $\boldsymbol{R_0} < 1$.

From the above discussions, we can summarize the following results.

Corollary 1. For system (2.2),

(1) if $\boldsymbol{R_0} < 1$ and

  (a) $b < \hat{b}$, there exists two endemic equilibria;

  (b) $b > \hat{b}$, there is no endemic equilibrium.

(2) when $\boldsymbol{R_0} = 1$, if $b < \hat{b}$, then there exists a unique endemic equilibrium; otherwise, there is no endemic equilibrium.

(3) if $\boldsymbol{R_0} > 1$, the endemic equilibrium always exist (see Theorem 3) and the number of endemic equilibrium is associated with the value of b (see Figure 3).

3.3. Stability of endemic equilibrium

In this subsection, we will use a geometric approach to prove the local stability of the endemic equilibrium. Linearizing system (2.2) at E^* yields

$$
\begin{aligned}
S'_k(t) =& \left(-\mu - \eta - \frac{\beta k \theta^*}{1 + \alpha \theta^*} \right) S_k(t) - \frac{\beta k S_k^*}{(1 + \alpha \theta^*)^2} \theta(t), \\
\theta'(t) =& \frac{\beta \theta^*}{\langle k \rangle (1 + \alpha \theta^*)} \sum_{k=1}^{n} k^2 p(k) S_k(t) + \left(\frac{\beta}{\langle k \rangle (1 + \alpha \theta^*)^2} \sum_{k=1}^{n} k^2 p(k) S_k^* - \mu - \gamma_0 - \frac{b^2(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} \right) \theta(t).
\end{aligned}
\tag{3.7}
$$

And hence the characteristic equation can be expressed as follows:

$$
\begin{aligned}
& \left(\lambda + \mu + \eta + \frac{\beta k \theta^*}{1 + \alpha \theta^*} \right) x_k + \frac{\beta k S_k^*}{(1 + \alpha \theta^*)^2} y = 0, \\
& -\frac{\beta \theta^*}{\langle k \rangle (1 + \alpha \theta^*)} \sum_{k=1}^{n} k^2 p(k) x_k + \left(\lambda + \mu + \gamma_0 + \frac{b^2(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} - \frac{\beta}{\langle k \rangle (1 + \alpha \theta^*)^2} \sum_{k=1}^{n} k^2 p(k) S_k^* \right) y = 0,
\end{aligned}
\tag{3.8}
$$

where (x_k, y) is the eigenvector that correspond to eigenvalue λ. From the first equation of (3.8), we can obtain

$$
x_k = \frac{-\beta k S_k^* y}{\beta k \theta^* (1 + \alpha \theta^*) + (\lambda + \mu + \eta)(1 + \alpha \theta^*)^2}, k = 1, 2, \ldots, n.
$$

Substituting x_k into the second equation of (3.8) leads to

$$
H(\lambda) y = 0,
$$

where $H(\lambda) = \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^3 p(k) \beta S_k^* \theta^*}{\beta k \theta^* (1 + \alpha \theta^*)^2 + (\lambda + \mu + \eta)(1 + \alpha \theta^*)^3} + \lambda + \mu + \gamma_0 + \frac{b^2(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} - \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k) S_k^*}{(1 + \alpha \theta^*)^2}$.

If $y = 0$, then $\lambda = -\mu - \eta - \frac{\beta k \theta^*}{1 + \alpha \theta^*} < 0$. Otherwise, when $y \neq 0$, then $H(\lambda) = 0$. Since

$$\frac{\beta k S_k^* \theta^*}{1 + \alpha \theta^*} = \mu - \mu S_k^* - \eta S_k^*,$$

$$S_k^* = \frac{\mu(1 + \alpha \theta^*)}{\beta k \theta^* + (\mu + \eta)(1 + \alpha \theta^*)}, \tag{3.9}$$

$$\frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{S_k^*}{1 + \alpha \theta^*} = \mu + \gamma_0 + \frac{b(\gamma_1 - \gamma_0)}{b + \theta^*},$$

and hence

$$\begin{aligned}
H(\lambda) =& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)(\mu - \mu S_k^* - \eta S_k^*)}{\beta k \theta^*(1 + \alpha \theta^*) + (\lambda + \mu + \eta)(1 + \alpha \theta^*)^2} + \lambda + \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k) S_k^*}{1 + \alpha \theta^*} \\
&- \frac{b(\gamma_1 - \gamma_0)\theta^*}{(b + \theta^*)^2} - \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)\mu}{\beta k \theta^*(1 + \alpha \theta^*) + (\mu + \eta)(1 + \alpha \theta^*)^2} \\
=& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)(\mu - \mu S_k^* - \eta S_k^*)}{\beta k \theta^*(1 + \alpha \theta^*) + (\lambda + \mu + \eta)(1 + \alpha \theta^*)^2} + \lambda - \frac{b(\gamma_1 - \gamma_0)\theta^*}{(b + \theta^*)^2} \\
&+ \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)\mu}{\beta k \theta^* + (\mu + \eta)(1 + \alpha \theta^*)} - \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)\mu}{\beta k \theta^*(1 + \alpha \theta^*) + (\mu + \eta)(1 + \alpha \theta^*)^2} \\
=& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)(\mu - \mu S_k^* - \eta S_k^*)}{\beta k \theta^*(1 + \alpha \theta^*) + (\lambda + \mu + \eta)(1 + \alpha \theta^*)^2} + \lambda - \frac{b(\gamma_1 - \gamma_0)\theta^*}{(b + \theta^*)^2} \\
&+ \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 p(k)\mu \alpha \theta^*}{\beta k \theta^*(1 + \alpha \theta^*) + (\mu + \eta)(1 + \alpha \theta^*)^2}.
\end{aligned} \tag{3.10}$$

Besides, taking the derivative of F associated with θ^*, we can have:

$$\begin{aligned}
F'(\theta^*) =& -\frac{b(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} + \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu(\beta k + (\mu + \eta)\alpha)}{(\beta k \theta^* + (\mu + \eta)(1 + \alpha \theta^*))^2} \\
=& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu(\frac{\beta k S_k^* \theta^*}{1 + \alpha \theta^*} + (\mu + \eta)\alpha \frac{S_k^* \theta^*}{1 + \alpha \theta^*})}{(\beta k \theta^* + (\mu + \eta)(1 + \alpha \theta^*))^2 \frac{S_k^* \theta^*}{1 + \alpha \theta^*}} - \frac{b(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} \\
=& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu - \mu S_k^* - \eta S_k^* + (\mu + \eta)\alpha \frac{S_k^* \theta^*}{1 + \alpha \theta^*}}{\theta^*(\beta k \theta^* + (\mu + \eta)(1 + \alpha \theta^*))} - \frac{b(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} \\
=& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{(\mu - \mu S_k^* - \eta S_k^*)(1 + \alpha \theta^*) + (\mu + \eta)\alpha S_k^* \theta^*}{\theta^*(\beta k \theta^*(1 + \alpha \theta^*) + (\mu + \eta)(1 + \alpha \theta^*)^2)} - \frac{b(\gamma_1 - \gamma_0)}{(b + \theta^*)^2} \\
=& \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu - \mu S_k^* - \eta S_k^* + \mu \alpha \theta^*}{\theta^*(\beta k \theta^*(1 + \alpha \theta^*) + (\mu + \eta)(1 + \alpha \theta^*)^2)} - \frac{b(\gamma_1 - \gamma_0)}{(b + \theta^*)^2}.
\end{aligned} \tag{3.11}$$

Therefore, we can obtain that

$$H(0) = \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu - \mu S_k^* - \eta S_k^* + \mu \alpha \theta^*}{\beta k \theta^*(1 + \alpha \theta^*) + (\mu + \eta)(1 + \alpha \theta^*)^2} - \frac{b(\gamma_1 - \gamma_0)\theta^*}{(b + \theta^*)^2} = \theta^* F'(\theta^*). \tag{3.12}$$

Theorem 5. When $F'(\theta^*) < 0$, then E^* is unstable; when $F'(\theta^*) > 0$, then E^* is locally asymptotically stable.

Proof. When $F'(\theta^*) < 0$, since $H(0) = \theta^* F'(\theta^*) < 0$, $\lim\limits_{\lambda \to +\infty} H(\lambda) = +\infty$, by the Intermediate Value Theorem, we can conclude that the equation $H(\lambda) = 0$ has at least one positive real solution, so E^* is unstable.

When $F'(\theta^*) > 0$, we rewrite the equation $H(\lambda) = 0$ as the following equation:

$$
\begin{aligned}
\widehat{H}(\lambda) = &\frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k)(\mu - \mu S_k^* - \eta S_k^*) \prod_{i \neq k}^{n} L(\lambda, i, \theta^*) \\
&+ \frac{\beta}{\langle k \rangle} \sum_{k=1}^{n} k^2 p(k) \frac{\mu \alpha \theta^*}{\beta k \theta^*(1 + \alpha\theta^*) + (\mu + \eta)(1 + \alpha\theta^*)^2} \prod_{k=1}^{n} L(\lambda, k, \theta^*) \\
&+ \left(\lambda - \frac{b(\gamma_1 - \gamma_0)\theta^*}{(b + \theta^*)^2} \right) \prod_{k=1}^{n} L(\lambda, k, \theta^*) = 0,
\end{aligned}
\tag{3.13}
$$

where $L(\lambda, k, \theta^*) = \beta k \theta^*(1 + \alpha\theta^*) + (\lambda + \mu + \eta)(1 + \alpha\theta^*)^2$. Suppose Eq (3.13) has a solution λ^* with $Re\lambda^* \geq 0$, then since

$$
|\widehat{H}(\lambda^*)| \geq \widehat{H}(Re\lambda^*) \geq \widehat{H}(0) = \prod_{k=1}^{n} L(0, k, \theta^*) H(0) = \prod_{k=1}^{n} L(0, k, \theta^*) \theta^* F'(\theta^*),
$$

if $F'(\theta^*) > 0$, we can conclude that $|\widehat{H}(\lambda^*)| > 0$, this contradicts with the fact $\widehat{H}(\lambda^*) = 0$. So Eq (3.13) has no solution with nonnegative real parts. Therefore, when $F'(\theta^*) > 0$, E^* is locally asymptotically stable.

Corollary 2. If $R_0 < 1$ and $b < \hat{b}$, system (2.2) exists two endemic equilibria, the one with smaller value is unstable; the other one with larger value is locally asymptotically stable.

Proof. If $R_0 < 1$ and $b < \hat{b}$, there exists two endemic equilibria (see Corollary 1). For the endemic equilibrium with smaller value, since $F'(\theta^*) < 0$ (see Figure 2(b)), then E^* is unstable; and for the endemic equilibrium with larger value, since $F'(\theta^*) > 0$, so E^* is locally asymptotically stable.

4. Simulations

Next, numerical simulations will be presented to validate the previous theoretical results. Our simulations are based on the scale-free network with $p(k) = \xi k^{-2.5}, k = 1, 2, \ldots, 100$, and the constant ξ is chosen so that the equation $\sum_{k=1}^{100} p(k) = 1$ holds. For better visualization, we present the values of the parameters in Table 2.
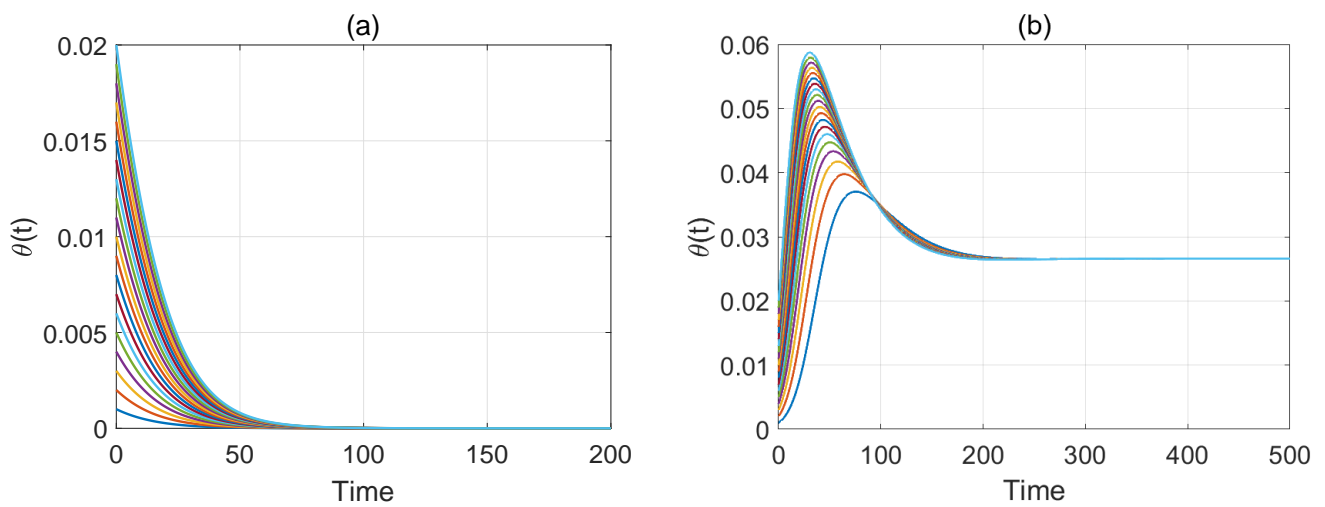
Figure 1. The time evolutions of $\theta(t)$ with different initial conditions $\theta(0) = 0.001 \times i, i = 1, 2, \ldots, 20$. (a) $\hat{R}_0 = 0.8933 < 1$. (b) $R_0 = 1.5008 > 1$.

Table 2. Parameter values of numerical simulations.

Parameter	Figure 1(a)	Figure 1(b)	Figure 2(a)	Figure 2(b)	Figure 3	Figure 4(a)	Figure 4(b)
μ	0.02	0.02	0.01	0.01	0.01	0.02	0.02
β	0.006	0.03	0.009	—	—	0.03	0.03
α	10	10	10	10	10	0/3/6/9/12	10
b	0.03	0.03	0.003	0.003/0.005/0.007 /0.009/0.011	0.0165/0.017 /0.0175/0.018	0.03	0/0.003/0.006 /0.009/0.012
γ_0	0.03	0.03	0.005	0.005	0.005	0.03	0.03
γ_1	0.09	0.09	0.08	0.08	0.08	0.09	0.09
η	0.008	0.008	0.008	0.008	0.008	0.008	0.008

Firstly, we verify the stability of the disease-free equilibrium E_0. According to the selected parameter values, we can calculate $R_0 = 0.3002, \hat{R}_0 = 0.8933$. It is known that E_0 is globally asymptotically stable from the result of Theorem 2. As seen in Figure 1(a), $\lim_{t \to +\infty} \theta(t) = 0$. Therefore, this does support the global stability of E_0.

In Figure 1(b), we can obtain $R_0 = 1.5008 > 1$ and hence model (2.2) exists endemic equilibrium. Observing the simulation results, we can see that all trajectories move towards to a positive constant and the unique endemic equilibrium could be stable. However, when $R_0 > 1$, model (2.2) can exhibit more complex dynamics (see Figure 3).

Secondly, we will verify that system (2.2) may exist endemic equilibrium when $R_0 < 1$. The parameter values as shown in Table 2, since $R_0 = 0.4280 < 1, \hat{R}_0 = 4.5545 > 1$, therefore E_0 is not globally asymptotically stable from the result of Theorem 2. From Figure 2(a), it can be observed that the trajectories partially converge to zero or partially move towards to a positive level which corresponds to different initial conditions. Therefore, the initial infectious density must be controlled to a lower level for the control of the diseases. The result of the co-existence of two locally
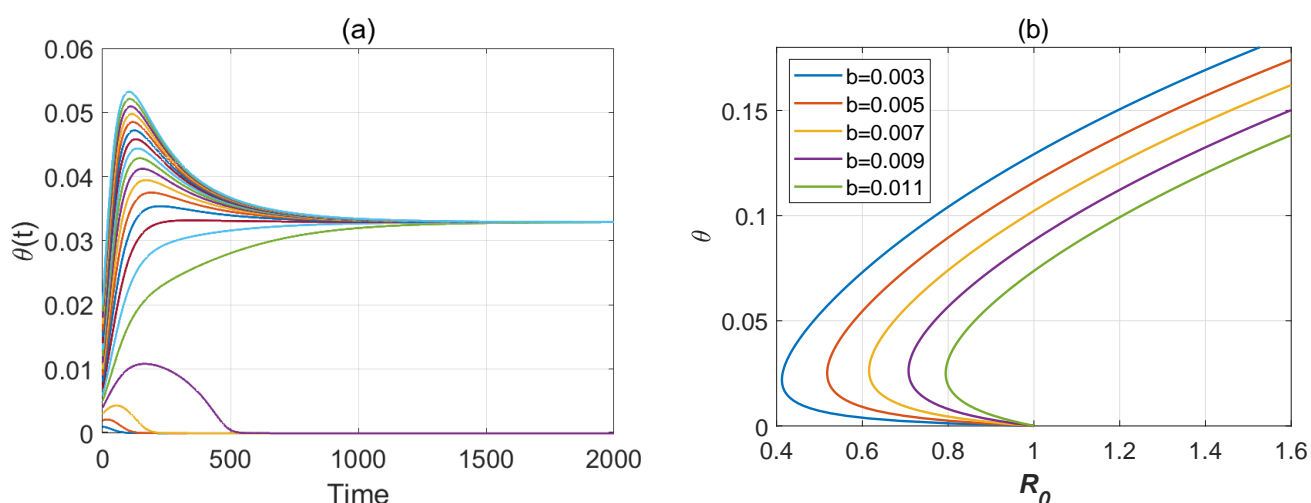
Figure 2. (a) The time evolutions of $\theta(t)$ with different initial conditions $\theta(0) = 0.001 \times i, i = 1, 2, \ldots, 20$. (b) The backward bifurcation diagrams in the $\boldsymbol{R_0}\theta$-plane with different values of b.

asymptotically stable equilibria is named as the bistable phenomenon.

Different values of b are taken to show the bifurcation diagrams. In Figure 2(b), backward bifurcation occurs at $\boldsymbol{R_0} = 1$ when $b < \hat{b} = 0.016$. Besides, the larger the value of b, the smaller the depth of backward bifurcation. In Figure 3, the parameters are the same as those given in Figure 2, except for the value of b. It can be seen that forward bifurcation occurs at $\boldsymbol{R_0} = 1$ for each subgraph. The reason is that although the values of b are different, they are all bigger than $\hat{b}$. Hence, the numerical simulation results are consistent with the theoretical results of Theorem 4.

At the same time, we can observe that the shapes of the bifurcation curves are different from Figure 3. For example, the endemic equilibrium curve has an "S shape" when $b = 0.017$. That is to say, backward bifurcation takes place at a certain value of $\boldsymbol{R_0}$. And the situation results in the existence of three endemic equilibria. So, the number of endemic equilibrium is uncertain when $\boldsymbol{R_0} > 1$.

Lastly, we will investigate the effect of α and b on diseases transmission through numerical simulations. The parameters in Figure 4 are the same as those in Figure 1(b) and $\boldsymbol{R_0} = 1.5008 > 1$. From Figure 4, we can observe that the larger α or b, the value of $\theta(t)$ at steady state will be smaller, which means that the final density of infected individuals will also be smaller. This indicates that the behavioral change of individuals can indeed suppress the spread of the diseases. Besides, enriching the adequate hospital beds is also important for the control of the diseases.

5. Conclusions and discussion

Many scholars proved that the number of hospital beds plays an important role in the occurrence of bifurcation [5, 6, 20–22], but most of them studied the homogeneously mixed models. In [6], Cui et al. obtained a quadratic equation with respect to I and discussed the existence and classification of equilibrium from the algebraic perspective. The condition for the existence of backward bifurcation is proved by reducing system (1.3) into the center manifold. In this paper, a network-based SIR epidemic
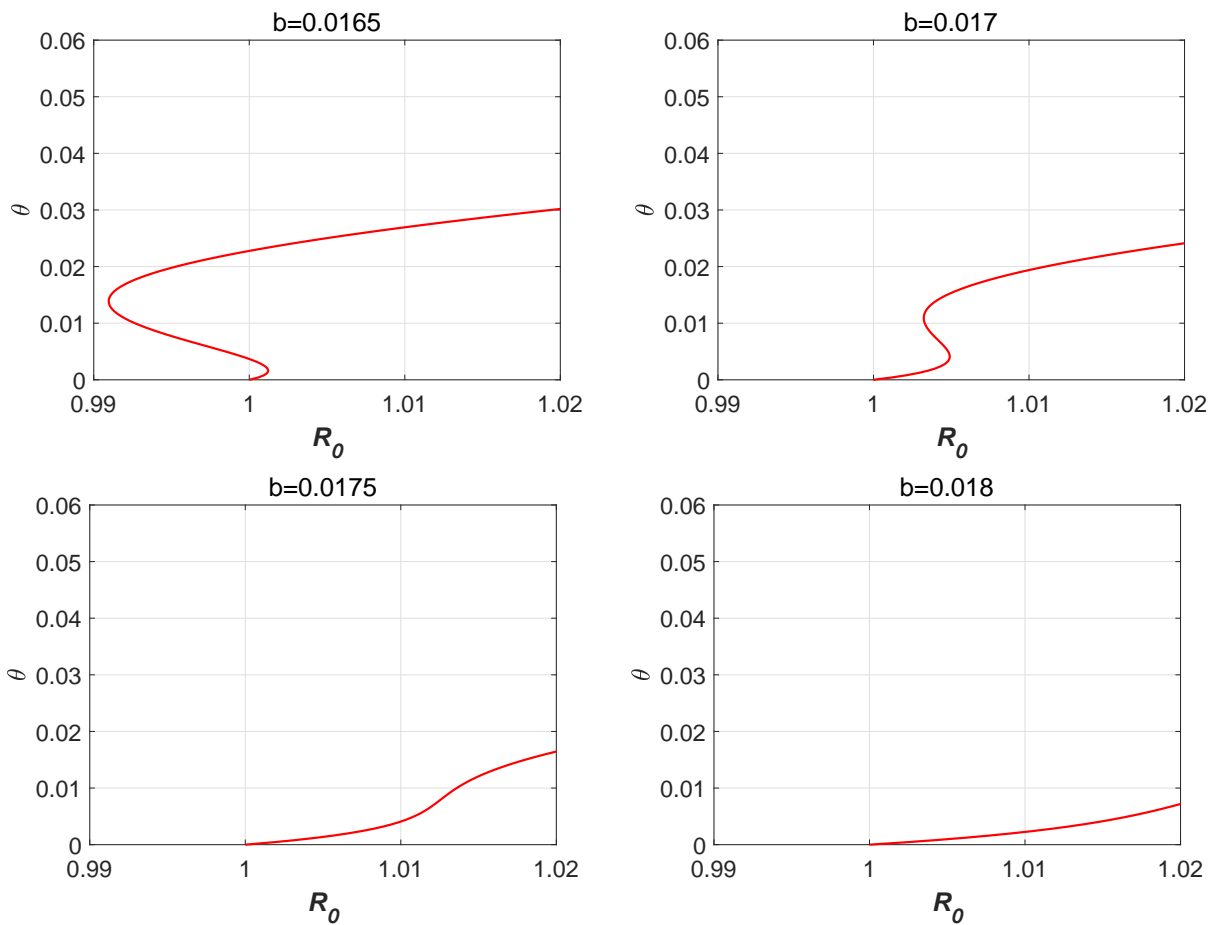
Figure 3. Bifurcation diagrams in the $R_0\theta$-plane with different values of b. Forward bifurcation takes place at $R_0 = 1$ for each subgraph.
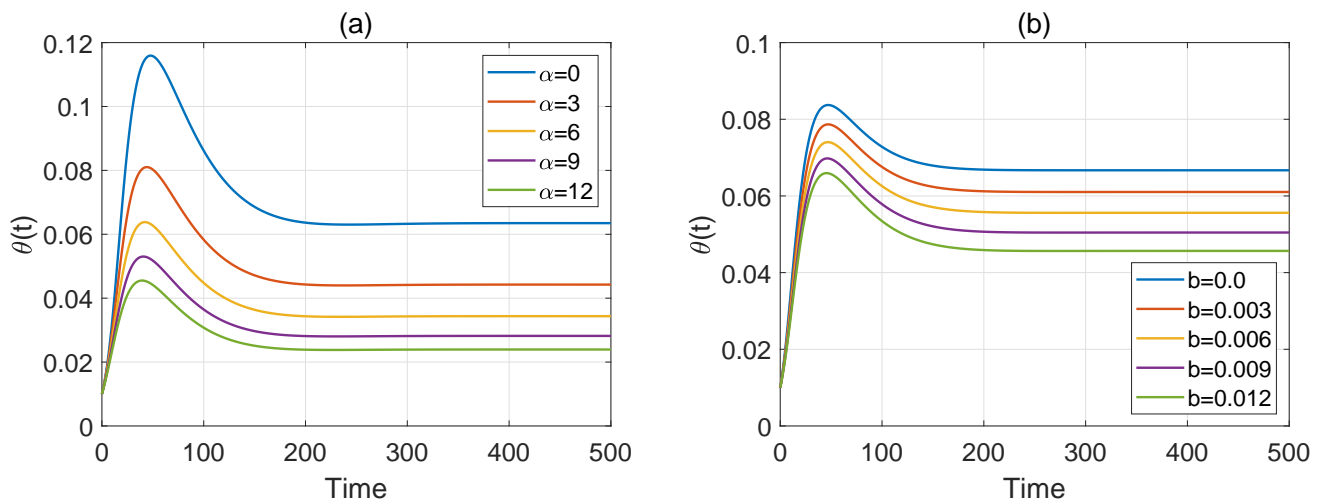

Figure 4. The time evolutions of $\theta(t)$ with initial condition $\theta(0) = 0.01$. (a) $\alpha = 3 \times (i - 1)$, (b) $b = 0.003 \times (i - 1), i = 1, 2, \ldots, 5$.

model which is the extension of model (1.3) is proposed. Using the edge-compartmental approach, we reduced the dimension of the system. It is proved that when $\frac{\partial \theta^*}{\partial R_0}\big|_{(R_0,\theta^*)=(1,0)} < 0 \Leftrightarrow b < \hat{b}$, backward bifurcation will occur at $R_0 = 1$. The method is different from the approach in [6]. Moreover, the condition which determines the direction of bifurcation at $R_0 = 1$ is related to the network structure.

It is also possible that system (2.2) exists multiple endemic equilibria when $R_0 > 1$ (see Figure 3). In conclusion, the condition $R_0 < 1$ is not enough to guarantee the global asymptotic stability of E_0, and $R_0 > 1$ does not confirm the uniqueness of endemic equilibrium. In addition, we established a relationship between the local stability of endemic equilibrium and the tangent slope of the epidemic curve. It is observed that the larger α or b can reduce the endemic level (see Figure 4). Therefore, the effect from the individual behavioral change should not be ignored and the hospital beds should be adequately provided in the process of controlling the infectious diseases.

However, whether system (2.2) can exhibit more complex dynamical behavior such as Hopf, saddle-node, transcritical and Bogdanov-Takens bifurcation are not discussed. At present, the bifurcation theory on networks is still less. It will be interesting and important to solve the problem and we leave this for future work.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Acknowledgments

Conflict of interest

The authors declare there is no conflict of interest in this paper.

References

1. V. Capasso, G. Serio, A generalization of the Kermack-McKendrick deterministic epidemic model, *Math. Biosci.*, **42** (1978), 43–61. https://doi.org/10.1016/0025-5564(78)90006-8

2. R. M. Anderson, R. M. May, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, 1991. https://doi.org/10.1093/oso/9780198545996.001.0001

3. W. Wang, S. Ruan, Bifurcations in an epidemic model with constant removal rate of the infectives, *J. Math. Anal. Appl.*, **291** (2015), 775–793. https://doi.org/10.1016/j.jmaa.2003.11.043

4. J. Eckalbar, W. Eckalbar, Dynamics of an epidemic model with quadratic treatment, *Nonlinear Anal. Real.*, **12** (2011), 320–332. https://doi.org/10.1016/j.nonrwa.2010.06.018

5. C. Shan, H. Zhu, Bifurcations and complex dynamics of an SIR model with the impact of the number of hospital beds, *J. Differ. Equations*, **257** (2014), 1662–1688. https://doi.org/10.1016/j.jde.2014.05.030

6. Q. Cui, Z. Qiu, W. Liu, Z. Hu, Complex dynamics of an SIR epidemic model with nonlinear saturate incidence and recovery rate, *Entropy*, **19** (2017), 305–320. https://doi.org/10.3390/e19070305

7. C. Li, A. M. Yousef, Bifurcation analysis of a network-based SIR epidemic model with saturated treatment function, *Chaos*, **29** (2019), 033129. https://doi.org/10.1063/1.5079631

8. Y. Huang, C. Li, Backward bifurcation and stability analysis of a network-based SIS epidemic model with saturated treatment function, *Phys. A*, **527** (2019), 121407. https://doi.org/10.1016/j.physa.2019.121407

9. X. Wang, J. Yang, Dynamical analysis of a mean-field vector-borne diseases model on complex networks: An edge based compartmental approach, *Chaos*, **30** (2020), 013103. https://doi.org/10.1063/1.5116209

10. X. Wang, J. Yang, A bistable phenomena induced by a mean-field SIS epidemic model on complex networks: A geometric approach, *Front. Phys.*, **9** (2021), 681268. https://doi.org/10.3389/fphy.2021.681268

11. J. Yang, X. Duan, X. Li, Complex patterns of an SIR model with a saturation treatment on complex networks: An edge-compartmental approach, *Appl. Math. Lett.*, **123** (2022), 107573. https://doi.org/10.1016/j.aml.2021.107573

12. J. Jiang, T. Zhou, The influence of time delay on epidemic spreading under limited resources, *Phys. A*, **508** (2018), 414–423. https://doi.org/10.1016/j.physa.2018.05.114

13. R. Rao, Z. Lin, X. Ai, J. Wu, Synchronization of epidemic systems with neumann boundary value under delayed impulse, *Mathematics*, **10** (2022), 2064. https://doi.org/10.3390/math10122064

14. Y. Zhao, L. Wang, Practical exponential stability of impulsive stochastic food chain system with time-varying delays, *Mathematics*, **11** (2023), 147. https://doi.org/10.3390/math11010147

15. B. Wang, Q. Zhu, S. Li, Stability analysis of discrete-time semi-Markov jump linear systems with time delay, *IEEE. Trans. Autom. Control*, **68** (2023), 6758–6765. https://doi.org/10.1109/TAC.2023.3240926

16. H. Xu, Q. Zhu, W. Zheng, Exponential stability of stochastic nonlinear delay systems subject to multiple periodic impulses, *IEEE Trans. Autom. Control*, (2023), 1–8. https://doi.org/10.1109/TAC.2023.3335005

17. L. Fan, Q. Zhu, W. Zheng, Stability analysis of switched stochastic nonlinear systems with state-dependent delay, *IEEE Trans. Autom. Control*, (2023), 1–8. https://doi.org/10.1109/TAC.2023.3315672

18. Q. Yang, X. Wang, X. Cheng, B. Du, Y. Zhao, Positive periodic solution for neutral-type integral differential equation arising in epidemic model, *Mathematics*, **11** (2023), 2701. https://doi.org/10.3390/math11122701

19. J. Yang, F. Xu, The computational approach for the basic reproduction number of epidemic models on complex networks, *IEEE Access*, **7** (2019), 26474–26479. https://doi.org/10.1109/ACCESS.2019.2898639

20. A. Abdelrazec, J. Belair, C. Shan, H. Zhu, Modeling the spread and control of dengue with limited public health resources, *Math. Biosci.*, **271** (2016), 136–145. https://doi.org/10.1016/j.mbs.2015.11.004

21. G. Li, Y. Zhang, Dynamic behaviors of a modified SIR model in epidemic diseases using nonlinear incidence and recovery rates, *PLoS ONE*, **12** (2017), e0175789. https://doi.org/10.1371/journal.pone.0175789

22. G. Lan, S. Yuan, B. Song, The impact of hospital resources and environmental perturbations to the dynamics of SIRS model, *J. Franklin Inst.*, **358** (2021), 2405–2433. https://doi.org/10.1016/j.jfranklin.2021.01.015