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Effect of infection age on an SIS epidemic model on complex networks

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Abstract In this paper, based on an SIS model, we construct an epidemic model with infection age to investigate the disease transmission on complex networks. By analyzing the characteristic equations associated with the equilibria, we obtain the basic reproduction number R_0. It is shown that if $R_0 < 1$ then the disease-free equilibrium is globally asymptotically stable while if $R_0 > 1$ then there is a unique endemic equilibrium, which is asymptotically stable. Our investigation indicates that if the maximal degree of the network is large enough then the endemic equilibrium always exists. Sensitivity analysis on the basic reproduction number R_0 in terms of the parameters is carried out to illustrate their effects on the disease transmission and to develop appropriate control strategies.

Keywords Epidemic model · Infection age · Complex network · Global stability

Mathematics Subject Classification 34K20 · 92D30

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1 Introduction

As indicated by the epidemic history, infectious diseases have taken the lives of millions of people and hence have been a major threat to the health of human being. It is crucial to understand the transmission of these diseases and to develop appropriate control strategies. Mathematical modeling, as an effective approach to capture the key points of disease spreading, has been widely used to study the control of infectious diseases. The earliest recorded such models are dated back to 200 years ago (Bernoulli 1766). Recent epidemic study has been primarily aimed to discover the dynamical mechanisms of the disease transmission and to predict current and future epidemic prevalence. Based upon these investigations, proper prevention measures for the disease can be developed and be provided to Centers for Disease Control (CDC) or health agencies.

The often-used epidemic models are compartmental ones. The first was proposed by Kermack and McKendrick (1927, 1932, 1933) to study the transmission of Black Death. Based on this model, a prediction of the outbreak behaviours of the disease was presented. Since then, compartmental epidemic modeling and threshold theory have been widely used in the study of disease transmission and have become the main methods. One fundamental assumption underlying the compartmental modeling is that all individuals are uniformly mixed and hence have homogenous contacts. This assumption is reasonable in modeling communicable diseases such as influenza and middle east respiratory syndrome and is also suitable for some social environments. However, with the evolution of the society, behaviours of the human being and disease transmissions become heterogeneous. Therefore, using compartmental epidemic modeling alone is not sufficient to describe the characteristics of disease spreading.

In order to capture the heterogeneities in the mechanism of disease transmission, the idea of complex network is introduced to the compartmental epidemic modeling. That is to say, all nodes (or vertices) in a complex network are classified according to their epidemic status. Since contacts of individuals in a society can be precisely described with complex networks and such contacts have significant influences on the transmission of infectious diseases, incorporating complex networks into the epidemic modeling can yield biologically feasible epidemic models. Most epidemic models on complex networks are based on the common SIR, SIS, and SIRS compartmental models. Results obtained from such models provide not only precise description of the current epidemic situation but also useful predictions on the future epidemic outbreaks. Epidemic models with network structure first appeared in the 1980s and have been attracting the interest of many researchers since then (to name a few, see Fu et al. 2013; Pastor-Satorras and Vespignani 2001a, b; Wang and Dai 2008, 2009; Wang et al. 2012a, 2013; Wang and Jin 2013; Zhang and Fu 2009; Zhang et al. 2013). SIS and SIR epidemic models on scale-free networks were investigated in Fu et al. (2013), Wang et al. (2012a, 2013), Wang and Jin (2013), Zhang and Fu (2009), Zhang et al. (2013). Pastor-Satorras and Vespignani (2001a, b) studied disease transmission by an SIS epidemic model on heterogeneous networks. Their conclusions were later rigorously proved by Wang and Dai (2008, 2009) using mathematical analysis. Wang and Jin (2013) proposed an SIS epidemic model with multiple transmission routes on a scale-free network. They established a threshold result and suggested a promising

way for the control of infectious diseases with multiple routes. Wang et al. (2012a) modified an SIS epidemic by incorporating an infective vector on complex networks. They studied the stability of the equilibria and the effects of various immunization schemes. Moreover, Zhang et al. (2013) constructed a model to investigate the spread of sexually transmitted diseases. By using matrix theory and nonlinear analysis, they derived the global dynamics of the model and discussed the control and spread of such diseases.

Most of the existing epidemic models on complex networks are described by ordinary differential equations. In these models, the effective contact rate or the effective transmission rate is constant. However, this may not be true for diseases like HIV, TB, Chicken Pox, and Diphtheria. In the spreading of these diseases, the effective transmission depends greatly on the infection age (the time passed since an individual was infected) or on physiological age. For instance, in TB and HIV, there exists a long latent period. Furthermore, the latently infected individuals and infectious individuals have different transmission rates. Therefore, it is natural and essential to study the demographical effects. Recently, infection age has been integrated into epidemic models to study this phenomenon (see, for example, Chen et al. 2014; Iannelli 1995; Magal and McCluskey 2013; Magal et al. 2010; Wang et al. 2014; Webb 1985). To the best of our knowledge, not much has been done for epidemic models with infection age on complex networks (Wang et al. 2013).

Based on the above discussion, in this paper, we shall build and analyze an SIS epidemic model on complex networks by incorporating both infection age and behavior epidemiology. The rest of this paper is organized as follows. First, the model is formulated in Sect. 2. Sections 3 and 4 are devoted to the stability analysis of equilibria. We shall obtain the basic reproduction number and establish a threshold dynamics. In Sect. 5, we carry out the sensitivity analysis of the basic reproduction number with respect to the model parameters and provide numerical simulations to demonstrate our theoretic results. The paper concludes with a short discussion.

2 The model formulation

In classic compartmental SIS models, the population is divided into two classes: the susceptibles (S) and the infected (I). A simple SIS model without death is described by the following system of ordinary differential equations,

$$\begin{cases} \frac{dS}{dt} = -k\sigma\beta S\frac{I}{S+I} + \gamma I, \\ \frac{dI}{dt} = k\sigma\beta S\frac{I}{S+I} - \gamma I. \end{cases} \tag{2.1}$$

Here k is the average contacts per time unit, σ is the effective exposure rate of a susceptible to the infected individuals (which varies according to behavior change, prevention strategies, and so on), β is the transmission rate, and γ is the recovery rate of the infected ones. In Eq. 2.1, the standard incidence rate is used.

With the structure of a complex network and infection age, we further subdivide the population as follows. Let n be the maximal degree of the complex network. For

$k \in \mathbb{N}_n \triangleq \{1, 2, \ldots, n\}$, let $S_k(t)$ be the number of susceptible vertices of degree k at time t, and $I_k(t, a)$ stand for the density of infected vertices of degree k at time t and with infection age a. Then we can modify Eq. 2.1 to obtain the following SIS model with infection age on complex networks,

$$\begin{cases} \dfrac{dS_k(t)}{dt} = -k\sigma S_k(t)\Theta(I(t,\cdot)) + \displaystyle\int_0^\infty \gamma(a) I_k(t,a)da, \\ \dfrac{\partial I_k(t,a)}{\partial t} + \dfrac{\partial I_k(t,a)}{\partial a} = -\gamma(a) I_k(t,a), \\ I_k(t,0) = k\sigma S_k(t)\Theta(I(t,\cdot)), \end{cases} \quad k \in \mathbb{N}_n, \tag{2.2}$$

where $\gamma(a)$ is the recovery rate at infection age a from infected class to susceptible class, $\Theta(I(t,\cdot)) = (\sum_{k=1}^n k \int_0^\infty \beta(a) I_k(t,a)da)/(\sum_{k=1}^n k N_k(t))$ denotes the infection force with $N_k(t) = S_k(t) + \int_0^\infty I_k(t,a)da$ being the total number of vertices with degree k at time t and $I(t,\cdot) = (I_1(t,\cdot), I_2(t,\cdot), \ldots, I_n(t,\cdot))$, and $\beta(a)$ is the transmission rate at infection age a. Let $\mathbb{R}_+ = [0, \infty)$. We assume that $\gamma, \beta \in L^\infty(\mathbb{R}_+)$such that β is nondecreasing and

$$\lim_{t\to\infty} \left\| \frac{\pi(t+\cdot)}{\pi(\cdot)} \right\|_\infty = 0, \tag{2.3}$$

where

$$\pi(a) = e^{-\int_0^a \gamma(s)ds} \qquad \text{for } a \in \mathbb{R}_+$$

is the probability of the infected individual still staying in the infected compartment. Condition Eq. 2.3 automatically holds if there exist $a_0 \geq 0$ and $\gamma_0 > 0$ such that $\gamma(a) \geq \gamma_0$ for $a \geq a_0$.

The initial condition of Eq. 2.2 is

$$(S_0, I_0) = (S_{10}, S_{20}, \ldots, S_{n0}, I_{10}, I_{20}, \ldots, I_{n0}) \in (\mathbb{R}_+)^n \times (L^1_+(\mathbb{R}_+))^n,$$

where

$$L^1_+(\mathbb{R}_+) = \{\varphi \in L^1(\mathbb{R}_+) | \varphi(a) \in \mathbb{R}_+ \text{ for } a \in \mathbb{R}_+\}.$$

Suppose that Eq. 2.2 also satisfies the coupling condition

$$I_{k0}(0) = k\sigma S_{k0}\Theta(I_0), \qquad k \in \mathbb{N}_n.$$

Then it follows from (Iannelli 1995; Webb 1985) that Eq. 2.2 is well-posed. Moreover, it is not difficult to show that the solution exists on $\mathbb{R}_+$, which is nonnegative and $I_k(t,\cdot) \in L^1_+(\mathbb{R}_+)$ for $k \in \mathbb{N}_n$ and $t \in \mathbb{R}_+$. For such solutions, one can easily see that

$$\frac{dN_k(t)}{dt} = -k\sigma S_k(t)\Theta(I(t,\cdot)) + \int_0^\infty \gamma(a) I_k(t,a)da + \int_0^\infty \frac{\partial I_k(t,a)}{\partial t} da$$

$$
\begin{aligned}
&= -k\sigma S_k(t)\Theta(I(t,\cdot)) + \int_0^\infty \gamma(a) I_k(t,a)da \\
&\quad - \int_0^\infty \left[\frac{\partial I_k(t,a)}{\partial a} + \gamma(a) I_k(t,a)\right] da \\
&= -k\sigma S_k(t)\Theta(I(t,\cdot)) - I_k(t,\infty) + I_k(t,0) \\
&= 0,
\end{aligned}
$$

which implies that $N_k(t)$ is a constant for $k \in \mathbb{N}_n$. Denote $N = \sum_{k=1}^n N_k$. Let $p(k) = \frac{N_k}{N}$, $s_k(t) = \frac{S_k(t)}{N_k}$, and $i_k(t,a) = \frac{I_k(t,a)}{N_k}$ for $k \in \mathbb{N}_n$. Dividing both sides of Eq. 2.2 by N_k gives us

$$
\begin{cases}
\dfrac{ds_k(t)}{dt} = -k\sigma s_k(t)\Theta(i(t,\cdot)) + \displaystyle\int_0^\infty \gamma(a) i_k(t,a)da, \\
\dfrac{\partial i_k(t,a)}{\partial t} + \dfrac{\partial i_k(t,a)}{\partial a} = -\gamma(a) i_k(t,a), \\
i_k(t,0) = k\sigma s_k(t)\Theta(i(t,\cdot)),
\end{cases} \qquad k \in \mathbb{N}_n, \tag{2.4}
$$

where $\Theta(i(t,\cdot)) = (\sum_{k=1}^n kp(k)\int_0^\infty \beta(a) i_k(t,a)da)/\langle k\rangle$ and $\langle k\rangle = \sum_{k=1}^n kp(k)$. Since $s_k(t) + \int_0^\infty i_k(t,a) = 1$, Eq. 2.4 is equivalent to

$$
\begin{cases}
\dfrac{\partial i_k(t,a)}{\partial t} + \dfrac{\partial i_k(t,a)}{\partial a} = -\gamma(a) i_k(t,a), \\
i_k(t,0) = \sigma k(1 - \int_0^\infty i_k(t,a)da)\Theta(i(t,\cdot)),
\end{cases} \qquad k \in \mathbb{N}_n \tag{2.5}
$$

with the initial condition $i_0 = (i_{10}, i_{20}, \ldots, i_{n0}) \in (L^1_+(\mathbb{R}_+))^n$ such that $\int_0^\infty i_{k0}(a)da \le 1$ for $k \in \mathbb{N}_n$. We mention that Eq. 2.5 is also an extension of the model studied by Wang and Dai (2008). Again, we always assume that Eq. 2.5 satisfies the coupling condition

$$
i_{k0}(0) = \sigma k\left(1 - \int_0^\infty i_{k0}(a)da\right)\Theta(i_0), \qquad k \in \mathbb{N}_n.
$$

Then Eq. 2.5 is well-posed. Moreover, it is not difficult to show the following result.

Proposition 2.1 *The set*

$$
\Omega = \left\{ i = (i_1, i_2, \ldots, i_n) | i_k \in L^1_+(\mathbb{R}_+) \quad \text{and} \quad \int_0^\infty i_k(a)da \le 1 \quad \text{for } k \in \mathbb{N}_n \right\}
$$

is a positively invariant set of Eq. 2.5.

By the model formulation and Proposition 2.1, we only need to consider Eq. 2.5 with initial conditions in Ω.

The following result tells us that if there is initial infection then the infection persists.

Lemma 2.2 *If $i_0 \in \Omega$ satisfies $\Theta(i_0) > 0$ then the solution i of Eq. 2.5 satisfies $\Theta(i(t,\cdot)) > 0$ and $\int_0^\infty i_k(t,a)da > 0$ for $t \in \mathbb{R}_+$ and $k \in \mathbb{N}_n$.*

Proof From the definition of $\Theta(i(t,\cdot))$, we have

$$\begin{aligned}
&\frac{d\Theta(i(t,\cdot))}{dt}\\
&= \langle k\rangle^{-1}\left(\sum_{k=1}^{n} kp(k)\int_0^\infty \beta(a)\frac{\partial i_k(t,a)}{\partial t}da\right)\\
&= \langle k\rangle^{-1}\left(\sum_{k=1}^{n} kp(k)\int_0^\infty \beta(a)\left(-\gamma(a)i_k(t,a)-\frac{\partial i_k(t,a)}{\partial a}\right)da\right)\\
&\geq -\|\gamma\|_\infty\Theta(i(t,\cdot)) - \langle k\rangle^{-1}\left(\sum_{k=1}^{n} kp(k)\int_0^\infty \beta(a)\frac{\partial i_k(t,a)}{\partial a}da\right)\\
&= -\|\gamma\|_\infty\Theta(i(t,\cdot)) - \langle k\rangle^{-1}\left(\sum_{k=1}^{n} kp(k)\left(\beta(a)i_k(t,a)|_{a=0}^{\infty}-\int_0^\infty i_k(t,a)d\beta(a)\right)\right)\\
&\geq -\|\gamma\|_\infty\Theta(i(t,\cdot)) + \langle k\rangle^{-1}\left(\sum_{k=1}^{n} kp(k)\beta(0)i_k(t,0)\right)\\
&\geq -\|\gamma\|_\infty\Theta(i(t,\cdot))\\
&\quad +\sigma\beta(0)\langle k\rangle^{-1}\left(\sum_{k=1}^{n} k^2p(k)\left(1-\int_0^\infty i_k(t,a)da\right)\right)\Theta(i(t,\cdot)).
\end{aligned}$$

This, combined with $\Theta(i_0)>0$, implies that $\Theta(i(t,\cdot)) > \Theta(i_0)e^{-\|\gamma\|_\infty t}$ for $t\in\mathbb{R}_+$.

Now, for $k\in\mathbb{N}_n$, it follows from Eq. 2.5 that

$$\begin{aligned}
\frac{d\int_0^\infty i_k(t,a)da}{dt} &= \int_0^\infty \frac{\partial i_k(t,a)}{\partial t}da\\
&= -\int_0^\infty\left(\gamma(a)i_k(t,a)+\frac{\partial i_k(t,a)}{\partial a}\right)da\\
&\geq -\|\gamma\|_\infty\int_0^\infty i_k(t,a)da + i_k(t,0)\\
&= -\|\gamma\|_\infty\int_0^\infty i_k(t,a)da + \sigma k\left(1-\int_0^\infty i_k(t,a)da\right)\Theta(i(t,\cdot))\\
&= -(\|\gamma\|_\infty+\sigma k\Theta(i(t,\cdot)))\int_0^\infty i_k(t,a)da + \sigma k\Theta(i(t,\cdot)).
\end{aligned}$$

As $\Theta(i(t,\cdot)) > \Theta(i_0)e^{-\|\gamma\|_\infty t}$ for $t\in\mathbb{R}_+$, it follows that $\int_0^\infty i_k(t,a)da > 0$ for $t>0$. This completes the proof. □

The following result will be used in establishing the stability of equilibria.

Lemma 2.3 (Proposition 3.1, Iannelli 1995) *Suppose* $h : \mathbb{R}_+ \to \mathbb{R}_+$ *is a bounded function and* $k \in L^1(\mathbb{R}_+)$. *Then*

$$\limsup_{t\to\infty} \int_0^t k(\theta)h(t-\theta)d\theta \le \|k\|_1 \left(\limsup_{t\to\infty} h(t)\right).$$

Under the assumptions of Lemma 2.3,one can easily get

$$\liminf_{t\to\infty} \int_0^t k(\theta)h(t-\theta)d\theta \ge \|k\|_1 \left(\liminf_{t\to\infty} h(t)\right).$$

This will be used in the proof of Eq. 4.5 in Lemma 4.3.

3 Global asymptotic stability of the disease-free equilibrium

Obviously, $E_0 = (0, 0, \ldots, 0) \in \Omega$ is an equilibrium of Eq. 2.5, called the disease-free equilibrium.

Theorem 3.1 *The disease-free equilibrium* E_0 *of Eq. 2.5 is locally asymptotically stable if* $R_0 < 1$ *and it is unstable if* $R_0 > 1$, *where*

$$R_0 = \sigma K \frac{\langle k^2\rangle}{\langle k\rangle} \tag{3.1}$$

with $K = \int_0^\infty \beta(a)\pi(a)da$ *and* $\langle k^2\rangle = \sum_{k=1}^n k^2 p(k)$.

Proof The linearized system of Eq. 2.5 around the disease-free equilibrium E_0 is

$$\begin{cases} \dfrac{\partial i_k(t,a)}{\partial t} + \dfrac{\partial i_k(t,a)}{\partial a} = -\gamma(a) i_k(t,a), \\ i_k(t,0) = \sigma k \Theta(i(t,\cdot)), \end{cases} \qquad k \in \mathbb{N}_n. \tag{3.2}$$

where $i(t,\cdot) \triangleq (i_1(t,\cdot), i_2(t,\cdot), \ldots, i_n(t,\cdot))$. Substitute $i_k(t,a) = i_k(a)e^{\lambda t}$ into Eq. 3.2 to get

$$\begin{cases} \dfrac{di_k(a)}{da} = -(\lambda + \gamma(a))i_k(a), \\ i_k(0) \triangleq i_{k0} = \sigma k \Theta(i(\cdot)), \end{cases} \qquad k \in \mathbb{N}_n.$$

It follows that

$$i_{k0} = \frac{\sigma}{\langle k\rangle} k \sum_{l=1}^n l p(l) \int_0^\infty i_{l0}\beta(a)\pi(a)e^{-\lambda a}da, \qquad k \in \mathbb{N}_n,$$

or

$$\left(I - \frac{\sigma}{\langle k\rangle}(ijp(j)\widehat{K}(\lambda))_{n\times n}\right)(i_{10}, i_{20}, \ldots, i_{n0})^T = (0, 0, \ldots, 0)^T,$$

where $\widehat{K}(\lambda) = \int_0^\infty \beta(a)e^{-\lambda a}\pi(a)da$. Then the characteristic equation at E_0 is

$$\left| I - \frac{\sigma}{\langle k \rangle}(ijp(j)\widehat{K}(\lambda))_{n\times n} \right| = 0.$$

After some row and column manipulations, we can get the determinant, which is

$$1 - \frac{\sigma\langle k^2 \rangle}{\langle k \rangle}\widehat{K}(\lambda) = 0. \tag{3.3}$$

We know that E_0 is locally asymptotically stable if all the roots of Eq. 3.3 have negative real parts and it is unstable if Eq. 3.3 has at least one root with positive real part. First, suppose $R_0 < 1$. We claim that all roots of Eq. 3.3 have negative real parts. By way of contradiction, suppose that Eq. 3.3 has a root λ_0 with $\mathrm{Re}(\lambda_0) \geq 0$. Then it follows from $1 - \frac{\sigma\langle k^2 \rangle}{\langle k \rangle}\widehat{K}(\lambda_0) = 0$ that

$$1 = \left| \frac{\sigma\langle k^2 \rangle}{\langle k \rangle}\widehat{K}(\lambda_0) \right| \leq \frac{\sigma\langle k^2 \rangle}{\langle k \rangle}K = R_0,$$

a contradiction. This proves the claim and hence E_0 is locally asymptotically stable if $R_0 < 1$. Now, suppose $R_0 > 1$. In this case, we have

$$1 - \frac{\sigma\langle k^2 \rangle}{\langle k \rangle}\widehat{K}(0) = 1 - R_0 < 0$$

and

$$\lim_{\lambda\to\infty}\left(1 - \frac{\sigma\langle k^2 \rangle}{\langle k \rangle}\widehat{K}(\lambda)\right) = 1.$$

By the Intermediate Value Theorem, Eq. 3.3 has at least one positive root. Therefore, E_0 is unstable if $R_0 > 1$. This completes the proof. □

In epidemiology, R_0 called the basic reproduction number, which is the average number of newly infected individuals produced by introducing an infectious individual to a total susceptible population. For our model, $\beta(a)\pi(a)$ is the transmission ability of an infected individual still staying in the infectious compartment at infection age a and hence K is the total transmission ability of an infected individual during the infectious period. Then σK is the effective transmission ability. Note that $\frac{\langle k^2 \rangle}{\langle k \rangle}$ is the total contacts by an infected individual in the network consisting of susceptible individuals only. Therefore, the definition of R_0 in Eq. 3.1 agrees with the biological interpretation of the basic reproduction number.

In fact, when E_0 is locally stable it is also globally stable, which we will prove now.

Theorem 3.2 *When $R_0 < 1$, the disease-free equilibrium E_0 of Eq. 2.5 is globally asymptotically stable.*

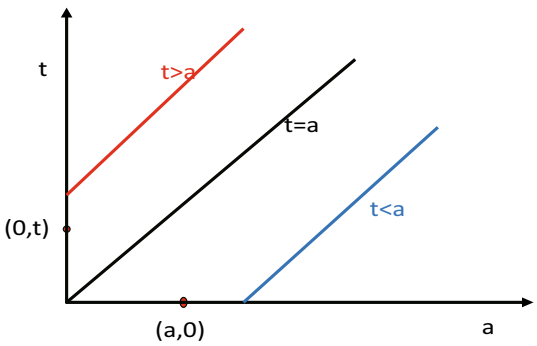


Fig. 1 Characteristic line. *Red solid line* is $t > a$; *black line* is $t = a$; *blue line* is $t < a$ (colour figure online)

Proof For any solution i with $i_0 \in \Omega$, integrating Eq. 2.5 along the characteristic line $t - a = c$ (a constant) gives us

$$i_k(a + c, a) = i_k(a + \xi, a)e^{-\int_\xi^a \gamma(s)ds}$$

where ξ is determine by the signal of c (see Fig. 1),

$$\xi = \begin{cases} 0, & \text{if } c > 0, \\ -c, & \text{otherwise.} \end{cases}$$

Therefore,

$$i_k(t, a) = \begin{cases} B_k(t - a)\pi(a), & t \geq a, \\ i_{k0}(a - t)\frac{\pi(a)}{\pi(a-t)}, & t < a, \end{cases} \qquad k \in \mathbb{N}_n, \tag{3.4}$$

where $B_k = i_k(t, 0)$. It follows from Proposition 2.1 that B_k is nonnegative and bounded for $k \in \mathbb{N}_n$. Substitute Eq. 3.4 into Eq. 2.5 to obtain

$$B_k(t) \leq \sigma k\langle k\rangle^{-1}\left[\sum_{l=1}^{n} lp(l)\left(\int_0^t \beta(a)B_l(t - a)\pi(a)da + \int_t^\infty \beta(a)i_{l0}(a - t)\frac{\pi(a)}{\pi(a - t)}da\right)\right]$$

for $k \in \mathbb{N}_n$. For simplicity of notation, let $B^\infty = (\limsup_{t\to\infty} B_k(t))_{1\times n}$. Note that

$$\int_t^\infty \beta(a)i_{l0}(t - a)\frac{\pi(a)}{\pi(a - t)}da = \int_0^\infty \beta(a + t)i_{l0}(a)\frac{\pi(a + t)}{\pi(a)}da \leq \|\beta\|_\infty \|i_{l0}\|_1 \left\|\frac{\pi(t + \cdot)}{\pi(\cdot)}\right\|_\infty.$$

This, together with Eq. 2.3 and Lemma 2.3, produces

$$B^{\infty} \leq AB^{\infty}, \tag{3.5}$$

where $A = (\sigma\langle k\rangle^{-1} Kijp(j))_{n\times n}$. It is easy to check that $\rho(A) = R_0 < 1$, where $\rho(A)$ is the spectral radius of the matrix A. Hence $B^{\infty} = 0$ by Eq. 3.5. Then the result follows directly from Eq. 3.4. □

4 Existence and stability of the endemic equilibrium

By Theorem 3.1, if $R_0 > 1$ then the disease-free equilibrium of Eq. 2.5 is unstable. In this section, we consider the endemic equilibria. Let $E^* = i^* = (i_1^*, i_2^*, \ldots, i_n^*) \in \Omega$ be an endemic equilibrium of Eq. 2.5. Then, for $k \in \mathbb{N}_n$, $i_k^* \neq 0$ and

$$\begin{cases} \dfrac{di_k^*(a)}{da} = -\gamma(a) i_k^*(a), \\ i_k^*(0) \triangleq B_k^* = \sigma k(1 - \int_0^{\infty} i_k^*(a)da)\Theta(i^*). \end{cases} \tag{4.1}$$

Solving Eq. 4.1 yields $i_k^*(a) = B_k^* \pi(a)$. We substitute it into the expression of B_k^* to get

$$B_k^* = \frac{\sigma k\langle k\rangle^{-1} K\hat{B}^*}{1 + \sigma k\langle k\rangle^{-1} K_1 K\hat{B}^*}, \qquad k \in \mathbb{N}_n, \tag{4.2}$$

where $K_1 = \int_0^{\infty} \pi(a)da$ and $\hat{B}^* = \sum_{k=1}^{n} kp(k)B_k^*$. It follows that

$$\hat{B}^* = \frac{\sigma K}{\langle k\rangle} \sum_{k=1}^{n} \frac{k^2 p(k)\hat{B}^*}{1 + \sigma k\langle k\rangle^{-1} K_1 K\hat{B}^*} \triangleq f(\hat{B}^*). \tag{4.3}$$

One can see that $f'(\hat{B}^*) > 0$ with $f'(0) = R_0 > 1$ and $f''(\hat{B}^*) < 0$. It follows that Eq. 4.3 has a unique positive solution and this solution combined with Eq. 4.2 and Eq. 4.1 gives the existence of a unique endemic equilibrium. In summary, we have proved the following result.

Theorem 4.1 *Suppose $R_0 > 1$. Then Eq. 2.5 has a unique endemic equilibrium $E^* = (i_1^*, i_2^*, \ldots, i_n^*)$, which is in Ω.*

To establish the stability of the endemic equilibrium E^*, we need the following two results. The first result is about the uniform strong ρ-persistence of the disease. The proof is based on the theory of uniform persistence (Thieme 2000). Though the arguments are tedious, they are standard (see, for example, Browne and Pilyugin 2013). Thus we omit the detail here.

Lemma 4.2 *Suppose $R_0 > 1$. Then the disease is uniformly strongly ρ-persistent, that is, there is an $\eta > 0$ such that if $i_0 \in \Omega$ satisfies $\Theta(i_0) > 0$ then the solution i of Eq. 2.5 satisfies $\liminf_{t\to\infty} \Theta(i(t, \cdot)) \geq \eta$.*

The next result tells us that, for some quantities related to solutions of Eq. 2.5, one can get smaller (respectively, bigger) upper bounds (respectively, lower bounds) from existing bounds.

Lemma 4.3 *For $k \in \mathbb{N}_n$, suppose that there exists $0 \le m_k \le M_k$ such that* $\limsup_{t\to\infty} B_k(t) \le M_k$ *and* $m_k \le \liminf_{t\to\infty} B_k(t)$. *Then*

$$\limsup_{t\to\infty} B_k(t) \le \frac{\sigma k \langle k\rangle^{-1} K \sum_{l=1}^{n} lp(l)M_l}{1+\sigma k \langle k\rangle^{-1} K_1 K \sum_{l=1}^{n} lp(l)M_l} \tag{4.4}$$

and

$$\liminf_{t\to\infty} B_k(t) \ge \frac{\sigma k \langle k\rangle^{-1} K \sum_{l=1}^{n} lp(l)m_l}{1+\sigma k \langle k\rangle^{-1} K_1 K \sum_{l=1}^{n} lp(l)m_l} \tag{4.5}$$

for $k \in \mathbb{N}_n$.

Proof For $k \in \mathbb{N}_n$,

$$\begin{aligned} B_k(t) = \sigma k\langle k\rangle^{-1} &\left(1-\int_0^t B_k(t-a)\pi(a)da - \int_t^\infty i_{k0}(a-t)\frac{\pi(a)}{\pi(a-t)}da\right) \\ &\times\left(\sum_{l=1}^{n} lp(l)\left(\int_0^t \beta(a)B_l(t-a)\pi(a)da + \int_t^\infty \beta(a)i_{l0}(a-t)\frac{\pi(a)}{\pi(a-t)}da\right)\right). \end{aligned} \tag{4.6}$$

Note that, for $k \in \mathbb{N}_n$, similar arguments as in the proof of Theorem 3.2 produce

$$\lim_{t\to\infty}\int_t^\infty i_{k0}(a-t)\frac{\pi(a)}{\pi(a-t)}da = 0$$

and hence

$$\lim_{t\to\infty}\int_t^\infty \beta(a)i_{k0}(t-a)\frac{\pi(a)}{\pi(a-t)}da = 0.$$

In the following we only prove Eq. 4.4 since Eq. 4.5 can be proved similarly.

Since $\limsup_{t\to\infty} B_k(t) \le M_k$ for all $k \in \mathbb{N}_n$, it follows from Lemma 2.3 that

$$\begin{aligned} &\limsup_{t\to\infty}\left(\sum_{l=1}^{n} lp(l)\left(\int_0^t \beta(a)B_l(t-a)\pi(a)da + \int_t^\infty \beta(a)i_{l0}(a-t)\frac{\pi(a)}{\pi(a-t)}da\right)\right) \\ &\le \sum_{l=1}^{n} lp(l)M_l K. \end{aligned}$$

Let $k_0 \in \mathbb{N}_n$. On the other hand, for $\varepsilon > 0$, there exists $T \ge 0$ such that

$$\mathrm{B}_{k_0}(t) \ge \limsup_{t\to\infty} B_{k_0}(t) - \varepsilon \quad \text{for } t \ge T.$$

Then, for $t \geq T$,

$$\int_0^t B_{k_0}(t-a)\pi(a)da \geq \int_0^{t-T} B_{k_0}(t-a)\pi(a)da \geq \left[\limsup_{t\to\infty} B_{k_0}(t) - \varepsilon\right]\int_0^{t-T} \pi(a)da.$$

These, combined with Eq. 4.6, give

$$B_{k_0}(t) \leq \sigma k_0\langle k\rangle\left(1 - \left[\limsup_{t\to\infty} B_{k_0}(t) - \varepsilon\right]\int_0^{t-T}\pi(a)da\right)\left(\sum_{l=1}^{n} lp(l)M_l K\right).$$

Taking lim sup on both sides of the above inequality yields

$$\limsup_{t\to\infty} B_{k_0}(t) \leq \sigma k_0\langle k\rangle\left(1 - K_1\left[\limsup_{t\to\infty} B_{k_0}(t) - \varepsilon\right]\right)\left(\sum_{l=1}^{n} lp(l)M_l K\right).$$

As ε is arbitrary, we obtain

$$\limsup_{t\to\infty} B_{k_0}(t) \leq \sigma k_0\langle k\rangle^{-1}\left(1 - K_1 \limsup_{t\to\infty} B_{k_0}(t)\right)\left(\sum_{l=1}^{n} lp(l)M_l K\right),$$

which immediately implies Eq. 4.4. □

Now we are ready to prove the main result of this section.

Theorem 4.4 *Suppose that $R_0 > 1$. If $i_0 \in \Omega$ satisfying*

$$\sum_{k=1}^{n} kp(k)\int_0^{\infty}\beta(a)i_{k0}(a)da > 0,$$

then the solution i satisfies $\lim_{t\to\infty} i(t) = E^*$.

Proof By Eq. 3.4, we only need to show that $\lim_{t\to\infty} B_k(t) = B_k^*$ for $k \in \mathbb{N}_n$. The arguments below are similar to those in Wang and Dai (2008).

First, we show that $\limsup_{t\to\infty} B_k(t) \leq B_k^*$ for $k \in \mathbb{N}_n$. Clearly, it follows from Propositions 2.1 that $\Theta(i(t,\cdot)) \leq \|\beta\|_\infty$ for $t \in \mathbb{R}_+$. Then $B_k(t) \leq \sigma k\|\beta\|_\infty$ for $t \in \mathbb{R}_+$ and hence by Lemma 4.3 we have $\limsup_{t\to\infty} B_k(t) \leq 1/K_1 \triangleq u_k^{(1)}$ for $k \in \mathbb{N}_n$. For $m \in \mathbb{N}$ and $k \in \mathbb{N}_n$, define

$$u_k^{(m+1)} = \frac{\sigma k\langle k\rangle^{-1} K\sum_{l=1}^{n} lp(l)u_l^{(m)}}{1 + \sigma k\langle k\rangle^{-1} K_1 K\sum_{l=1}^{n} lp(l)u_l^{(m)}}.$$

By Lemma 4.3, we have

$$\limsup_{t\to\infty} B_k(t) \le u_k^{(m)} \quad \text{for all } k \in \mathbb{N}_n \quad \text{and} \quad m \in \mathbb{N}.$$

Moreover, $u_k^{(2)} \le u_k^{(1)}$ for $k \in \mathbb{N}_n$ and hence by induction we have

$$u_k^{(m+1)} \le u_k^{(m)} \quad \text{for all } k \in \mathbb{N}_n \quad \text{and} \quad m \in \mathbb{N}.$$

Let $u_k = \lim_{m\to\infty} u_k^{(m)}$ for $k \in \mathbb{N}_n$. Then

$$\limsup_{t\to\infty} B_k(t) \le u_k \quad \text{and} \quad u_k = \frac{\sigma k \langle k\rangle^{-1} K \sum_{l=1}^n lp(l)u_l}{1+\sigma k\langle k\rangle^{-1} K_1 K \sum_{l=1}^n lp(l)u_l}$$

for $k \in \mathbb{N}_n$. It follows that $f(\sum_{k=1}^n kp(k)u_k) = \sum_{k=1}^n kp(k)u_k$. This implies that $\sum_{k=1}^n kp(k)u_k = \hat{B}^*$ or 0. Therefore, we have $\limsup_{t\to\infty} B_k(t) \le B_k^*$ for $k \in \mathbb{N}_n$.

Next, we show that $\liminf_{t\to\infty} B_k(t) \ge B_k^*$ for $k \in \mathbb{N}_n$. By Lemma 4.2, there exists $\eta > 0$ such that $\liminf_{t\to\infty} \Theta(i(t,\cdot)) \ge \eta$. Also, for $k \in \mathbb{N}_n$, since $\limsup_{t\to\infty} B_k(t) \le B_k^*$, we see that

$$\begin{aligned}\limsup_{t\to\infty} \int_0^\infty i_k(t,a)da &= \limsup_{t\to\infty}\left[\int_0^t B_k(t-a)\pi(a)da \right.\\ &\qquad \left. + \int_t^\infty i_{k0}(a-t)\frac{\pi(a)}{\pi(a-t)}da\right]\\ &\le \int_0^\infty i_k^*(a)da < 1.\end{aligned}$$

Then we can get

$$\begin{aligned}\liminf_{t\to\infty} B_k(t) &\ge \sigma k\left(1-\limsup_{t\to\infty}\int_0^\infty i_k(t,a)da\right)\liminf_{t\to\infty}\Theta(i(t,\cdot))\\ &\ge \sigma k\eta\left(1-\int_0^\infty i_k^*(a)da\right) > 0.\end{aligned}$$

Therefore, we can choose $v_k^{(1)} > 0$ such that

$$\liminf_{t\to\infty} B_k(t) \ge v_k^{(1)} \quad \text{for } k \in \mathbb{N}_n$$

and

$$f\left(\sum_{k=1}^n kp(k)v_k^{(1)}\right) > 0. \tag{4.7}$$

For $k \in \mathbb{N}_n$ and $m \in \mathbb{N}$, define

$$v_k^{(m+1)} = \frac{\sigma k \langle k \rangle^{-1} K \sum_{l=1}^{n} lp(l) v_l^{(m)}}{1 + \sigma k \langle k \rangle^{-1} K_1 K \sum_{l=1}^{n} lp(l) v_l^{(m)}}.$$

By Lemma 4.3, we have

$$\liminf_{t \to \infty} B_k(t) \geq v_k^{(m)} \quad \text{for} \quad k \in \mathbb{N}_n \quad \text{and} \quad m \in \mathbb{N}.$$

Furthermore, Eq. 4.7 implies that

$$\sum_{k=1}^{n} kp(k) v_k^{(2)} > \sum_{k=1}^{n} kp(k) v_k^{(1)}.$$

It follows that $v_k^{(3)} \geq v_k^{(2)}$ for all $k \in \mathbb{N}_n$ and hence by induction $v_k^{(m+1)} > v_k^{(m)}$ for all $k \in \mathbb{N}_n$ and $m \in \mathbb{N}$. Denote $v_k = \lim_{m \to \infty} v_k^{(m)}$ (>0) for $k \in \mathbb{N}_n$. Then we have

$$\liminf_{t \to \infty} B_k(t) \geq v_k \quad \text{and} \quad v_k = \frac{\sigma k \langle k \rangle^{-1} K \sum_{l=1}^{n} lp(l) v_l}{1 + \sigma k \langle k \rangle^{-1} K_1 K \sum_{l=1}^{n} lp(l) v_l}.$$

It is easy to see that $\sum_{k=1}^{n} kp(k) v_k > 0$ and $f(\sum_{k=1}^{n} kp(k) u_k) = \sum_{k=1}^{n} kp(k) u_k$, which imply that $\sum_{k=1}^{n} kp(k) u_k = \hat{B}^*$. Therefore, we can easily get $\liminf_{t \to \infty} B_k(t) \geq B_k^*$ for $k \in \mathbb{N}_n$. This completes the proof. □

5 Sensitivity analysis and simulations

By Theorems 3.2 and 4.4, the reproduction number R_0 is a key threshold for the control of the disease. In this section, we first carry out the sensitivity analysis of R_0 in terms of transmission parameters. For convenience, we set

$$\gamma(a) = \begin{cases} 0, & a \leq \tau \\ \gamma, & a \geq \tau \end{cases} \quad \text{and} \quad \beta(a) = \begin{cases} 0, & a \leq \omega, \\ \beta, & a \geq \omega. \end{cases}$$

In other words, the incubation period is ω and the cure period is τ. Then

$$R_0 = \sigma \beta \frac{e^{\gamma(\tau - \omega)}}{\gamma} \frac{\langle k^2 \rangle}{\langle k \rangle} \quad \text{if} \quad \omega \geq \tau$$

and

$$R_0 = \sigma \beta \frac{\gamma(\tau - \omega) + 1}{\gamma} \frac{\langle k^2 \rangle}{\langle k \rangle} \quad \text{if} \quad \omega < \tau.$$

In either case, one can easily see that $\frac{\partial R_0}{\partial \sigma} > 0$, $\frac{\partial R_0}{\partial \beta} > 0$, $\frac{\partial R_0}{\partial \gamma} < 0$, $\frac{\partial R_0}{\partial \tau} > 0$, and $\frac{\partial R_0}{\partial \omega} < 0$. Therefore, in order to prevent the outbreak of the disease, one can decrease the exposure rate and/or the transmission rate, or shorten the cure period, or improve the cure rate, or lengthen the latent period.

By the expression of R_0, it is easy to see that the topological structure of the complex network plays an important role on controlling the disease spread. Elasticity is a powerful tool to help us to realize it. According to the definition of elasticity, the elasticity of R_0 to the five parameters (σ, β, ω, τ, and γ) are

$$E^{\sigma}_{R_0} = \frac{\sigma}{R_0}\frac{\partial R_0}{\partial \sigma} = 1 \quad \text{and} \quad E^{\beta}_{R_0} = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta} = 1$$

in either case and if $\omega \geq \tau$ then

$$\begin{aligned} E^{\gamma}_{R_0} &= \frac{\gamma}{R_0}\frac{\partial R_0}{\partial \gamma} = (\tau - \omega)\gamma - 1, \\ E^{\tau}_{R_0} &= \frac{\tau}{R_0}\frac{\partial R_0}{\partial \tau} = \gamma\tau, \\ E^{\omega}_{R_0} &= \frac{\omega}{R_0}\frac{\partial R_0}{\partial \omega} = -\gamma\omega \end{aligned}$$

while if $\omega < \tau$ then

$$\begin{aligned} E^{\gamma}_{R_0} &= \frac{\gamma}{R_0}\frac{\partial R_0}{\partial \gamma} = -\frac{1}{\gamma(\tau - \omega) + 1}, \\ E^{\tau}_{R_0} &= \frac{\tau}{R_0}\frac{\partial R_0}{\partial \tau} = \frac{\gamma\tau}{\gamma(\tau - \omega) + 1}, \\ E^{\omega}_{R_0} &= \frac{\omega}{R_0}\frac{\partial R_0}{\partial \omega} = -\frac{\gamma\omega}{\gamma(\tau - \omega) + 1}. \end{aligned}$$

These expressions effects of the parameters on the basic reproduction number R_0 depend on the values chosen. To further study the clear relations among the elasticities of R_0 to the parameters, for simplicity of notation, we rewrite $E^i = |E^i_{R_0}|$, $i \in \{\beta, \sigma, \gamma, \omega, \tau\}$. Then we have the following situations.

When the cure period is longer than the latent period, i.e., $\tau > \omega$, one can obtain (i) for $\omega < \tau < 2\omega$,

$$\begin{cases} E^{\beta} = E^{\sigma} > E^{\gamma} > E^{\tau} > E^{\omega} & \text{if } \gamma < \frac{1}{\tau}, \\ E^{\beta} = E^{\sigma} > E^{\tau} > E^{\gamma} > E^{\omega} & \text{if } \frac{1}{\tau} < \gamma < \frac{1}{\omega}, \\ E^{\tau} > E^{\beta} = E^{\sigma} > E^{\omega} > E^{\gamma} & \text{if } \frac{1}{\omega} < \gamma < \frac{1}{2\omega - \tau}, \\ E^{\tau} > E^{\omega} > E^{\beta} = E^{\sigma} > E^{\gamma} & \text{if } \frac{1}{2\omega - \tau} < \gamma, \end{cases}$$

and (ii) for $\tau > 2\omega$,

$$\begin{cases} E^{\beta} = E^{\sigma} > E^{\gamma} > E^{\tau} > E^{\omega} & \text{if } \gamma < \frac{1}{\tau}, \\ E^{\beta} = E^{\sigma} > E^{\tau} > E^{\gamma} > E^{\omega} & \text{if } \frac{1}{\tau} < \gamma < \frac{1}{\omega}, \\ E^{\tau} > E^{\beta} = E^{\sigma} > E^{\omega} > E^{\gamma} & \text{if } \frac{1}{\omega} < \gamma. \end{cases}$$

One can see that, for example, if $\omega < \tau < 2\omega$ and $\gamma > \frac{1}{2\omega-\tau}$ then the cure period τ has the greatest effect on R_0, followed by the latent period ω, then by the transmission rate β or the exposure rate σ, and the cure rate has the weakest effect on R_0.

When the cure period is shorter than the latent period, i.e., $\tau < \omega$, one has

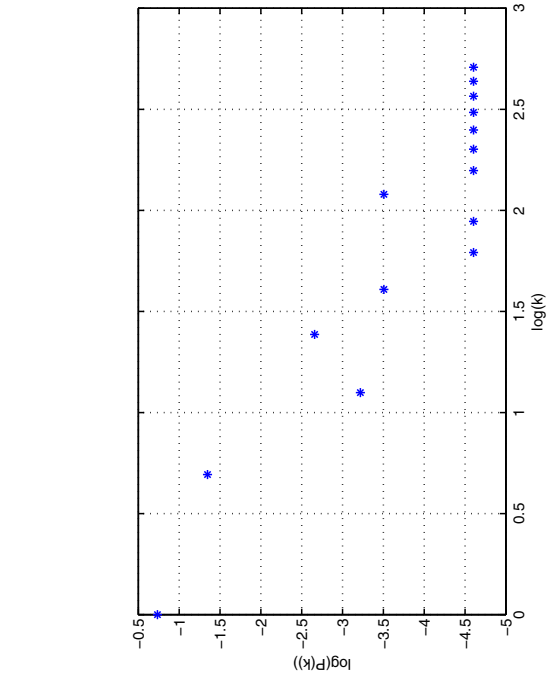
$$\begin{cases} E^{\tau} < E^{\beta} = E^{\sigma} < E^{\gamma} < E^{\omega} & \text{if } 0 < \gamma < \frac{1}{\tau} \\ E^{\beta} = E^{\sigma} < E^{\tau} < E^{\gamma} < E^{\omega} & \text{if } \frac{1}{\tau} < \gamma < \frac{1}{2\tau-\omega} \\ E^{\beta} = E^{\sigma} < E^{\gamma} < E^{\tau} < E^{\omega} & \text{if } 0 < \gamma < \frac{1}{2\tau-\omega} \end{cases}$$

for $\tau < \omega < 2\tau$, and

$$\begin{cases} E^{\tau} < E^{\omega} < E^{\gamma} < E^{\beta} = E^{\sigma} & \text{if } 0 < \gamma < \frac{1}{\omega} \\ E^{\tau} < E^{\beta} = E^{\sigma} < E^{\omega} < E^{\gamma} & \text{if } \frac{1}{\omega} < \gamma < \frac{1}{\tau} \\ E^{\beta} = E^{\sigma} < E^{\tau} < E^{\omega} < E^{\gamma} & \text{if } \gamma > \frac{1}{\tau} \end{cases}$$

for $\omega > 2\tau$. As before, from the above relations, one can easily see which parameters have the most important effect on the basic reproduction number R_0 in each circumstance.

Now, we consider the combined effects of parameters on the basic production number R_0. Based on the preferential algorithm (Pastor-Satorras and Vespignani 2001a, b), we can generate a BA network with $m = 3$ and $N = 100$ by Matlab. The dynamics of the nodes is implemented based on the BA network with 100 nodes (see Fig. 2). The interconnectedness is 2.76 and the minimum degree is 3 while the maximum degree $k_{max} = 95$. Figure 3a shows the effects of the transmission rate β and the cure rate γ with $\tau = 10$, $\omega = 15$, and $\sigma = 0.05$. It says that the basic reproduction number R_0 decreases as the cure rate γ increases and the transmission rate β decreases. Visually the transmission rate β has the greater effect than the cure rate γ. Figure 3b indicates that the transmission rate β has the greater effect on R_0 than the latent period ω, where $\tau = 10$, $\gamma = 2$, and $\sigma = 0.05$. Figure 4a shows the relation of the reproduction number R_0 with the cure period τ and the latent period ω. Here $\beta = 0.08$, $\gamma = 2$, and $\sigma = 0.05$. We see that the cure period has the greater effect than the latent period. With $\beta = 0.08$, $\tau = 10$, $\sigma = 0.05$, Fig. 4b tells us that the basic reproduction number R_0 is larger than 1 even when the cure rate γ is large while the latent period ω is short enough. At the same time if the cure rate is small while the latent period ω is short enough the basic reproduction number R_0 is still larger than 1. The effects of the two parameters γ and ω on R_0 depend on the other parameters.



(a) **(b)**

Fig. 2 A BA scale-free network with 100 nodes and $m = 3$. **a** The network, **b** the evolution of the logarithm of the node probability $p(k)$ versa the logarithm of degree k

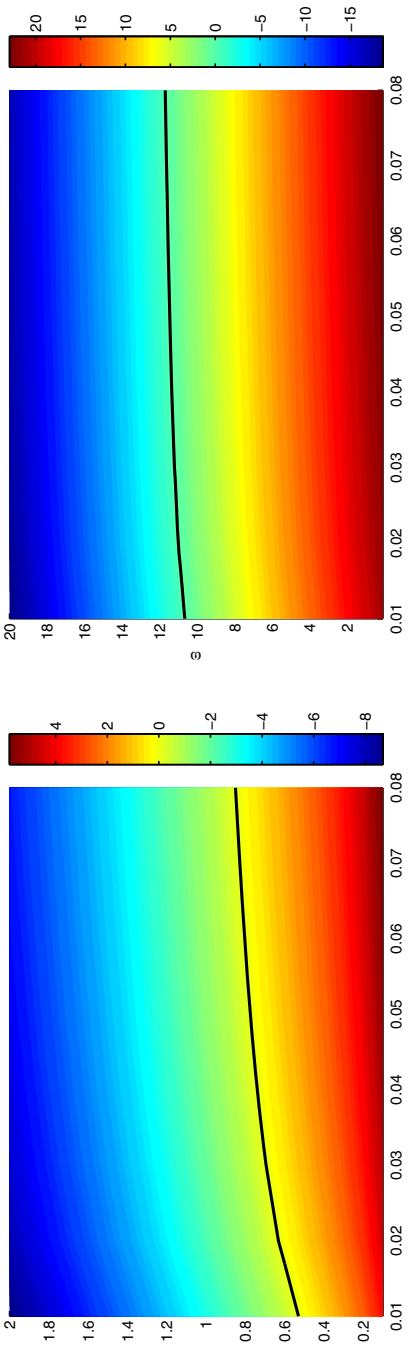


Fig. 3 The logarithm of the basic reproduction number of Eq. 2.5. **a** $\tau = 10$, $\omega = 15$, $\sigma = 0.05$ when $\gamma \in [0.1, 2]$ and $\beta \in [0.01, 0.08]$; **b** $\tau = 10$, $\gamma = 2$, $\sigma = 0.05$ when $\omega \in [8, 15]$ and $\beta \in [0, 0.08]$

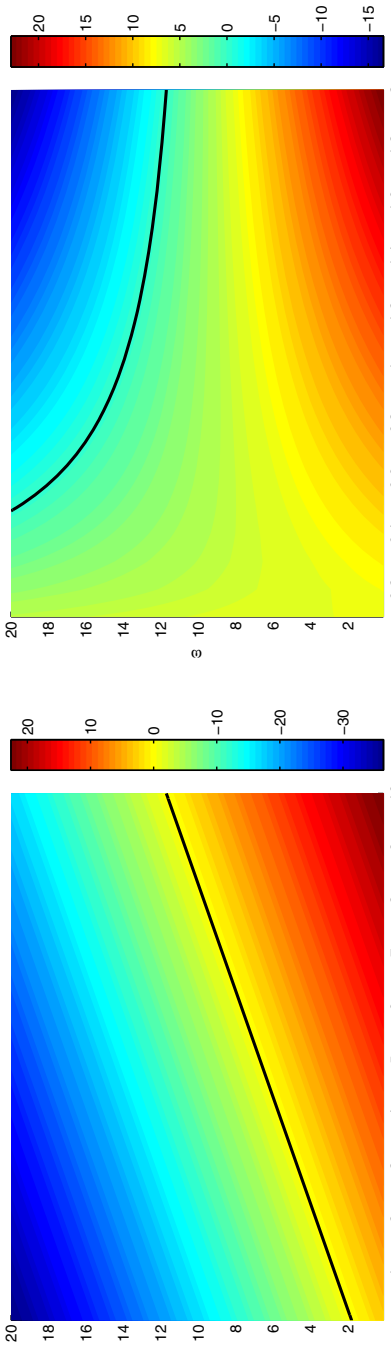


Fig. 4 The logarithm of the basic reproduction number of Eq. 2.5. **a** $\beta = 0.08$, $\gamma = 2$, $\sigma = 0.05$ when $\tau \in [0, 10]$ and $\omega \in [0, 20]$; **b** $\beta = 0.08$, $\tau = 10$, $\sigma = 0.05$ when $\omega \in [0, 20]$ and $\gamma \in [0, 2]$

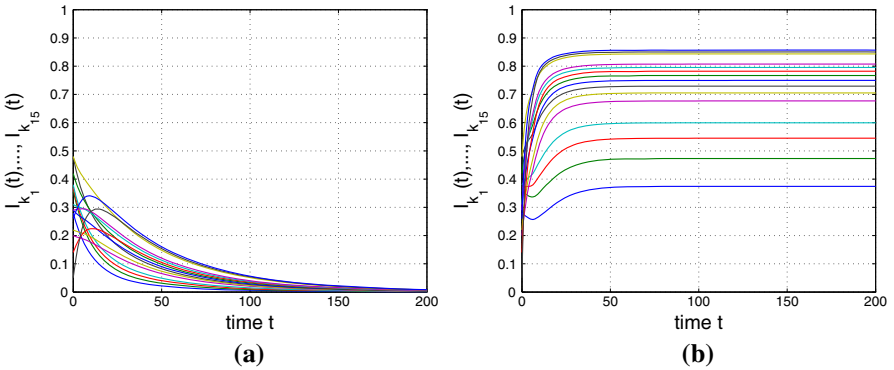


Fig. 5 Stability of the equilibria of Eq. 2.5. **a** The disease-free equilibrium is globally asymptotically stable when $R_0 < 1$. Here $\beta = 0.01$, $\sigma = 1$, $\gamma = 0.1$, and $\tau = \omega = 5$; **b** the endemic equilibrium is globally stable when $R_0 > 1$. Here $\beta = 0.05$, $\sigma = 1$, $\gamma = 0.1$, and $\tau = \omega = 5$

Finally, we illustrate our theoretical results with numerical simulations using the above network. For this purpose, we first take $\sigma = 1$,

$$\beta(a) = \begin{cases} 0, & a \leq 5, \\ 0.01, & a > 5, \end{cases}$$

and

$$\gamma(a) = \begin{cases} 0, & a \leq 5, \\ 0.1, & a > 5. \end{cases}$$

Then $R_0 = 0.7430 < 1$. According to Theorem 3.2, the disease-free equilibrium E_0 is globally stable (see Fig. 5a).

Now, we enlarge the transmission rate by taking

$$\beta(a) = \begin{cases} 0, & a \leq 5, \\ 0.05, & a > 5. \end{cases}$$

Then $R_0 = 3.7150 > 1$. According to Theorem 4.4, the endemic equilibrium E^* is globally asymptotically stable (see Fig 5b).

Since the key parameter for controlling the disease spreading is the basic reproduction number, we conclude this section by discussing the differences between the basic reproduction numbers of homogeneous networks and heterogeneous networks, which are

$$R_0^h = \sigma K \quad \text{(homogeneous networks)},$$
$$R_0 = \sigma K \frac{\langle k^2 \rangle}{\langle k \rangle} \quad \text{(heterogeneous networks)}.$$

Obviously, the substantial difference is that the basic reproduce number mainly depends on the topological structure for heterogeneous networks. The higher the degree distributions of the nodes for the networks, the larger the reproduction number is. This implies that decreasing the degree distributions of the nodes is beneficial to control the disease spread on complex networks.

6 Discussion

Infection age is an important factor in the transmission of infectious diseases such as HIV, TB, and Hand–Foot–Mouth Disease. In this paper, we incorporated infection age into a simple SIS epidemic model. Unlike existing models on complex networks, the state variables in the model depend on the infection age a. From the mathematical point of view, the system is described by a system of ordinary differential equations coupled with partial differential equations rather than by a system only involving ordinary differential equations. Such infinitely dimensional systems are a challenge to study. From the biological point of view, the infection age enhances the intrinsic characters of some diseases.

Roughly speaking, we established a threshold dynamics. When the basic reproduction number $R_0 < 1$, the model has a unique equilibrium, the disease-free equilibrium, which is globally asymptotically stable. When $R_0 > 1$, the disease-free equilibrium loses its stability and there is a unique endemic equilibrium, which is globally stable. These theoretical results are supported by numerical simulations. We also carried out the sensitivity analysis of R_0 to the parameters. Our model is a generalization of some SIS epidemic models on complex networks (Fu et al. 2013; Wang and Dai 2008, 2009). In fact, if we take $\beta(a) = \beta$, $\gamma(a) = \gamma$, and $I_k(t) = \int_0^\infty i_k(t, a)da$, then Eq. 2.5 reduces to

$$\begin{cases} \dfrac{dI_k(t)}{dt} = \sigma\beta k(1 - I_k)\Theta(I_k) - \gamma I_k(t) \\ I_k(0) = I_{k0}. \end{cases} \qquad k \in \mathbb{N}_n \tag{6.1}$$

Eq. 6.1 is the systems discussed in Fu et al. (2013), Wang and Dai (2008, 2009). If we choose the transmission rate and treatment rate as step functions,

$$\beta(a) = \begin{cases} 0, & 0 \le a \le \tau, \\ \beta, & a > \tau, \end{cases} \qquad \gamma(a) = \begin{cases} 0, & 0 \le a \le \tau, \\ \gamma, & a > \tau, \end{cases}$$

and $I_k(t) = \int_\tau^\infty i_k(t, a)da$, then Eq. 2.5 becomes

$$\begin{cases} \dfrac{dI_k(t)}{dt} = \sigma\beta k(1 - I_k)\Theta(I_k(t - \tau)) - \gamma I_k(t), t > \tau, \\ I_k(t) = e^{-\gamma t}\displaystyle\int_{\tau - t}^\infty i_0(s)e^{-\gamma(s-\tau)}ds, t \in (0, \tau], \\ I_k(0) = \displaystyle\int_0^\tau i_k(t, a)da. \end{cases} \qquad k \in \mathbb{N}_n.$$

Hence our results are more general than those in the just mentioned references.

Recently, multi-strain epidemic models on complex networks have been investigated by some authors (see, for example, Wang et al. 2012b; Wu et al. 2011). Obtained results include (a) the existence and local stability of the boundary equilibria, and (b) sufficient conditions on coexistence of strains. It is natural to introduce infection age to these models and then establish similar results or even results on global stability. Moreover, with the introduction of infection age, interesting complex dynamics such as oscillations and chaotic attractors may occur. We leave this to be future work.

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