J. Math. Biol. (2017) 74:1263–1298
DOI 10.1007/s00285-016-1057-6

Mathematical Biology

Epidemic spreading on adaptively weighted scale-free networks

Mengfeng Sun[1] · Haifeng Zhang[2] · Huiyan Kang[3] · Guanghu Zhu[4] · Xinchu Fu[1]

Received: 8 April 2015 / Published online: 17 September 2016

Abstract We introduce three modified SIS models on scale-free networks that take into account variable population size, nonlinear infectivity, adaptive weights, behavior inertia and time delay, so as to better characterize the actual spread of epidemics. We develop new mathematical methods and techniques to study the dynamics of the models, including the basic reproduction number, and the global asymptotic stability of the disease-free and endemic equilibria. We show the disease-free equilibrium cannot undergo a Hopf bifurcation. We further analyze the effects of local information of diseases and various immunization schemes on epidemic dynamics. We also perform some stochastic network simulations which yield quantitative agreement with the deterministic mean-field approach.

Keywords Scale-free network · Adaptive weight · Behavior inertia · Time delay · Immunization · The basic reproduction number

Mathematics Subject Classification 92B05 · 34D23

✉ Xinchu Fu
xcfu@shu.edu.cn

Mengfeng Sun
1173068801@qq.com

1 Department of Mathematics, Shanghai University, Shanghai 200444, China

2 School of Mathematical Science, Anhui University, Hefei 230039, China

3 School of Mathematics and Physics, Changzhou University, Changzhou 213016, China

4 School of Mathematics and Computing Science, Guilin University of Electronic Technology, Guilin 541004, China

1 Introduction

Since Barabási and Albert (1999) proposed a scale-free network model, in which the degree distribution follows a power-law distribution, scientists have found that a multitude of real networks (e.g., the Internet, biological and social networks, etc.) exhibit scale-free properties (Albert and Barabási 2002). Particularly, the epidemic spreading systems can be modeled on networks, in which nodes stand for individuals and links (edges) connecting two nodes indicate the interactions between them. Researchers have gradually focused on the spread of epidemics on complex networks and obtained a lot of useful and insightful results (Pastor-Satorras and Vespignani 2001a, b).

The dynamical behavior of epidemics has been studied for a long time, and coupled systems of nonlinear differential equations on networks have been used to model the spread of epidemics in heterogeneous populations (Brauer and Castillo-Chavez 2001; Thieme 2003). Previous studies suggest that both the properties of diseases and the network topology determine the dynamical behavior of the spread of epidemics, such as the absence of an epidemic threshold on scale-free networks (Pastor-Satorras and Vespignani 2001b; Moreno et al. 2002), and the hierarchical spreading patterns of epidemic outbreaks (Barthélemy et al. 2005).

Two fundamental epidemic models (SIS and SIR) have been widely studied (Ball et al. 1997; Kuperman and Abramson 2001; May and Lloyd 2001; Newman 2002; Eguiluz and Klemm 2002). But there are some inappropriate assumptions in some existing SIS models to simulate the real spread of epidemics, as listed and discussed in the following:

(i) Constant population size. If the births are not balanced by the deaths, or if there are large amount of disease-related deaths, this assumption may be unreasonable. So it is better to take the factors of birth and death rates into account. For example, in Sanz et al. (2010), an epidemic model with constant birth and death rates was investigated to discuss the dynamics of Tuberculosis-like infection.

(ii) Constant birth rate of empty nodes with different degrees. In Liu et al. (2004), it was suggested that empty nodes with different degrees give birth to individuals with a certain rate, which may be not reasonable in the real situations.

(iii) Fixed transmission rate. In real systems, the transmission rate λ may be different among individuals. Furthermore, this rate λ on a given link is usually treated as a function of the degrees of two connecting nodes in Joo and Lebowitz (2004) and Olinky and Stone (2004), which means that the transmission rates of two opposite directions on the same link are equivalent. In order to make the transmission rate accord better with realistic cases, here, we take the effects of the weights of links and the strength of nodes into account, which are extremely important on weighted networks (Barrat et al. 2004a, b, c; Wu et al. 2006). Particularly, for epidemic systems, the weight can indicate the extent of the contact between individuals. The larger the weight of the link connecting two nodes is, the more intensively the two nodes communicate.

(iv) The same values of infectivity and node degree. In classical SIS model, it is assumed that each infected individual can contact all of its acquaintances (neighbors) at every moment of time, i.e., the infectivity of each infected node equals

to its degree. But in reality, an individual cannot contact all his acquaintances at every moment of time, especially when he/she is ill. Existing studies have proposed different formulas to estimate the infectivity $\varphi(k)$ of nodes with degree k, such as $\varphi(k) = k$ in Moreno et al. (2002) and Pastor-Satorras and Vespignani (2002), $\varphi(k) = A$ (a constant) in Yang et al. (2007), $\varphi(k) = \min(\alpha k, A)$ in Fu et al. (2008), $\varphi(k) = ak^{\alpha}/(1 + bk^{\alpha})$, $0 \leq \alpha \leq 1, a > 0, b \geq 0$ in Zhang and Fu (2009).

(v) Time delay of infection on homogeneous networks. Time delay plays an important role in propagation process of epidemics, and temporal delay in epidemic models makes them more realistic by allowing to describe the effects of disease latency period or immunity period (Beretta and Kuang 2001; Beretta and Takeuchi 1995). In 1973, Cooke presented an SIS model with time delay on homogeneous networks (Ma and Li 2009; Cooke and Yorke 1973). However, very little attention has been paid to time delay on heterogeneous networks (Xia et al. 2013; Xu et al. 2006).

(vi) Constant weights. Weight distribution largely impacts the epidemic spreading taking place on networks. Recently, epidemic spreading on weighted networks has also been discussed in Britton et al. (2011), Rattana et al. (2013), Deijfen (2011) and Yang and Zhou (2012). Britton et al. (2011) introduced a random graph model with prescribed degree distribution and degree dependent edge weights and studied limiting properties of such a network as well as properties of an epidemic spreading on the network. Rattana et al. (2013) studied SIS and SIR epidemic models on undirected, weighted networks by deriving pairwise-type approximate models coupled with individual-based network simulation and considered two different types of theoretical/synthetic weighted network models. Deijfen (2011) studied a model for epidemic spread on weighted graphs with prescribed degree distribution and analyzed a version of the acquaintance vaccination strategy where vertices are chosen randomly and neighbors of these vertices with large edge weights are vaccinated. Yang and Zhou (2012) proposed an edge-based mean-field method to study a susceptible-infected-susceptible model on regular random networks with different kinds of weight distributions and showed that the more homogeneous weight distribution leads to higher epidemic prevalence.

In this paper, we modify the above unreasonable hypotheses and investigate an SIS epidemic model with variable population size, nonlinear infectivity and time delay on undirected, adaptively weighted scale-free networks. In order to better characterize the actual spread of epidemic diseases, we propose the concept of adaptive weights. Then, we investigate the epidemic threshold and propagation dynamics of models, and analyze the influence of weights, the effects of local information of diseases, as well as various immunization schemes on epidemic dynamics.

The rest of this paper is organized as follows: In Sect. 2 we describe stochastic evolution mechanism of an adaptive model and formulate three different models based on mean field theory. In Sect. 3 we calculate the epidemic thresholds of these models, and make a comparison among them. By constructing corresponding Lyapunov functions, we analyze the global stability of the disease-free and endemic equilibria of

an adaptively weighed model. In Sect. 4 we analyze the impacts of local information of diseases on epidemic dynamics. Different immunization and treatment strategies are considered in Sect. 5. In Sect. 6, some numerical simulations are carried out to illustrate and complement the analytical results. Finally, a brief discussion is given in Sect. 7 to conclude the paper.

2 Description and formation of epidemic models

2.1 Stochastic model

Let us consider a population of N individuals whose connections to each other form a network. Each node of the network is empty or occupied by at most one individual. The nodes are enumerated with index $i = 1, 2, \ldots, N$. The degree k_i of a node i is a number of links between node i and other nodes.

We divide all nodes into three categories: susceptible (S), infected (I) and empty (E), that is, each node may have one of the three states: empty state, healthy individual occupation and infected individual occupation.

Birth $E \to S/I$: Each empty node i randomly selects a neighbor at each time step. If the neighbor is susceptible, then the empty node i will give birth to a new susceptible node with the birth rate b; if the neighbor is infected, then the empty node i will give birth to a new infected node with the same rate. Due to the physiological limitation, it is assumed that each non-empty node generates the same birth contacts A at each time step.

Death $S/I \to E$: All susceptible and infected nodes die with the death rate d at each time step. If a non-empty node dies, there is an empty node left.

Infection $S \to I$: Initially, all nodes are in susceptible state, to start the spreading process, a few nodes are chosen as the infected nodes. Each infected node i contacts each of its susceptible neighbors with the probability $\frac{\varphi(k_i)}{k_i}$ at each time step, where $\varphi(k_i)$ is the infectivity of the infected node i. If an infected node i has contact with one of its susceptible neighbors, then this susceptible neighbor j will be infected by the infected node i with the probability λ_{ij}, where λ_{ij} represents the transmission rate from the infected node i to susceptible node j. The specific form of λ_{ij} is discussed below.

Recovery $I \to S$: All infected nodes can be cured and become susceptible with the rate γ at each time step.

In the above expressions, the parameters b, d, A, λ_{ij} and γ are all non-negative.

2.2 Mean-field models

Based on the above assumptions, we consider an SIS model on a scale-free network with the degree distribution $P(k) = ck^{-r}$. Suppose that $S_k(t)$ and $I_k(t)$ are the densities of the healthy and infected nodes with given degree k at time t, respectively. Then we have the following mean-field equations:

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1-S_k(t)-I_k(t)]\sum_i \frac{A}{i}P(i|k)S_i(t) - dS_k(t) - kS_k(t)\Theta_k(t) + \gamma I_k(t), \\ \frac{dI_k(t)}{dt} = bk[1-S_k(t)-I_k(t)]\sum_i \frac{A}{i}P(i|k)I_i(t) + kS_k(t)\Theta_k(t) - (d+\gamma)I_k(t), \end{cases} \tag{2.1}$$

where $\Theta_k(t)$ stands for the probability that the infection transmits through a link which emanates from a node with degree k and points to an infected node, namely,

$$\Theta_k(t) = \sum_i P(i|k)\frac{\varphi(i)}{i}\lambda_{ik}I_i(t), \tag{2.2}$$

where $P(i|k)$ is the probability that a node with degree k points to a node with degree i, $\varphi(i)$ is the infectivity of infected nodes with degree i, and λ_{ik} is the transmission rate from infected nodes with degree i to susceptible nodes with degree k. Without loss of generality, we set $A = 1$ in system (2.1).

In uncorrelated networks, it is shown that $P(i|k) = iP(i)/\langle k\rangle$, which implies that the probability that a node with degree k points to a node with degree i is proportional to degree i and the degree distribution $P(i)$. Here, $\langle k\rangle$ is the normalization factor.

Note that $\varphi(k) = ak^\alpha/(1+bk^\alpha)(0 \le \alpha \le 1, a > 0, b \ge 0)$, Zhang and Fu (2009) may be more suitable than $\varphi(k) = k$, Moreno et al. (2002) and Pastor-Satorras and Vespignani (2002), $\varphi(k) = A$ (a constant), Yang et al. (2007), or $\varphi(k) = \min(\alpha k, A)$, Fu et al. (2008), since an infected individual cannot contact all his acquaintances at every moment of time, Moreno et al. (2002) and Pastor-Satorras and Vespignani (2002), and the heterogeneous infectivity of nodes with different degrees is not considered adequately, Yang et al. (2007) and Fu et al. (2008). For system (2.1), we define

$$\varphi(k) = k^\alpha, \quad 0 < \alpha \le 1,$$

which means that every infected individual can establish contact with its k^α neighbors at every moment of time. It is obvious that the infectivity of nodes with degree k grows nonlinearly as k increases. The exponent α dominates the infectivity of infected nodes with different degrees. The value of α can be adjusted to make the contacts fit practical situations better.

2.2.1 SIS model on networks with fixed weights

Different from the previous studies, here, we mainly consider an SIS model on weighted networks. Among varieties of weighted patterns on complex networks, making full use of nodes' degrees to express the weights of links is very important. The weight between two nodes with degree i and j can be represented by a function of their degrees (Barrat et al. 2004a, b, c; Macdonald et al. 2005), such as $w_{ij} = w_0(ij)^\beta = w_0 i^\beta j^\beta$, where the basic parameter w_0 and the exponent β depend on the particular complex networks [e.g., in the scientist collaboration networks $\beta = 0$ (Barrat et al. 2004a), in the Escherichia coli matabolic network $\beta = 0.5$, in the US airport network $\beta = 0.8$ (Macdonald et al. 2005)]. Accordingly, a node with

degree i also can be measured by weights, i.e., the strength (weight) of a node with degree i, which can be obtained by summing the weights of the links that connect it. If the strength (weight) of a node is large, then the node is powerful and important in networks.

Let Ω_k denote the strength (weight) of a node with degree k, then we have

$$\Omega_k = k \sum_i P(i|k) w_{ik}.$$

In this paper, we focus on uncorrelated networks, in which the conditional probability $P(i|k)$ satisfies $P(i|k) = iP(i)/\langle k \rangle$. For simplicity, we set $w_0 = 1$ and use a general weight function as $w_{ij} = g(i)g(j)$ where $g(i) = i^{\beta}$. Thus, we obtain

$$\Omega_k = kg(k) \frac{\langle kg(k) \rangle}{\langle k \rangle}.$$

Here, for each node with degree i, we fix a total transmission rate given by λi. The transmission rate on links from nodes with degree i to nodes with degree k will be redistributed by the proportion of the weight of the link over the strength of the nodes with degree i, i.e., the transmission rate λ_{ik} can be defined as follows (Chu et al. 2011):

$$\lambda_{ik} = \lambda i \frac{w_{ik}}{\Omega_i} = \frac{\lambda g(k) \langle k \rangle}{\langle kg(k) \rangle}. \tag{2.3}$$

Substituting (2.3) into (2.2), we obtain

$$\Theta_k(t) = \frac{\lambda g(k)}{\langle kg(k) \rangle} \sum_i \varphi(i) P(i) I_i(t), \tag{2.4}$$

which corresponds to the SIS model on networks with fixed weights.

2.2.2 SIS model with behavior inertia on adaptively weighted networks

Taking the reactions of individuals to the spread of diseases into account, the weights of links and the strength of nodes will change as epidemics spread. But Zhu et al. (2013) found that the adaptation of weights cannot change the basic reproduction number. According to reality, the adaptive behavior of individuals should weaken or put off the outbreak of diseases and reduce the basic reproduction number to a certain extent. So, we modify the evolutive way of the weights of links to conform better to the actual situations based on the following assumptions:

1. If we consider individual's reactions in terms of a disease, the weight function $\tilde{g}(k, t)$ will become less and less as the disease prevalence $I(t)$ increases, which implies that there is a negative correlation between the weight function $\tilde{g}(k, t)$ and disease information $I(t)$.

2. Particularly, if an individual has more friends/neighbors, he/she will be more cautious, therefore his/her weight will decay more significantly, which implies that the extent of individual's response to the disease information $I(t)$ is related to the degrees of individuals. And, we use $h(k)$ to express the response extent of the individuals with degree k to the disease information $I(t)$.
3. Individuals will not immediately stop their adaptive behavior when a disease is just disappearing (behavior inertia). For example, individuals will not immediately take off their masks or communicate with their friends/neighbors like before when they realize the fact that the disease has petered out. This implies that when a disease disappears, the weight between individuals will not return to the same weight as before. Furthermore, individuals who have more friends/neighbours lose more weight than individuals who have fewer friends/neighbours if we assume that every individual loses the same weight by each edge. We use $m(k)$ to represent behavior inertia of individuals.

At the beginning of an emerging epidemic, massive news coverage and fast information flow can generate profound psychological impacts on the public, and hence greatly alter individual's behavior and influence the implementation of public intervention and control policies. For example, according to the Chinese Southern Weekend newspaper, the text message "There is a fatal flu in Guangzhou" was sent 126 million times in Guangzhou alone during the 2003 severe acute respiratory syndrome (SARS) outbreak (Tai and Sun 2007), causing people to stay home or wear face masks when going outside. This figure stands in stark contrast to the comparatively low number of 5327 cases recorded in the whole of China (World Health Organization 2005). Furthermore, there were some influential media reports during 2009 novel influenza A (H1N1) (Jones and Salathe 2009; SteelFisher et al. 2010), 2013 H7N9 (Chun-Hai Fung and Wong 2013; Goodwin and Sun 2013) and 2015 Ebola (Fung et al. 2014; Househ 2016).

Recently, several mathematical models have been proposed to investigate media impacts. Existing approaches to modeling the impact of media coverage have focused on how this coverage depends on the number of infected individuals (Cui et al. 2008; Liu et al. 2007; Sun et al. 2011; Xiao et al. 2015), where prototype decreasing functions such as e^{-mI}, $e^{-\alpha_1 E-\alpha_2 I-\alpha_3 H}$ (with hospitalized individuals H, infected individuals I , exposed individuals E and nonnegative constants $m, \alpha_i, i = 1, 2, 3$), $c_1 - c_2 f(I)$ (with constants c_1, c_2) and $e^{-M(I,dI/dt)}$ have been embedded into the incidence rate.

Thus, according to the above assumptions and statements, we can modify the weight function as follows:

$$\tilde{g}(k,t) = g(k) \exp\left(-h(k)I(t) - m(k)\right).$$

Here, we regard the average density of infected individuals $I(t)$ as a kind of information measuring the evolution of epidemics, and individuals usually gain this information from some public channels (such as the newspapers, the Internet and televisions) to update their connection weights.

Response-degree function $h(k)$ is an increasing functions of k, which implies that the reaction of every individual to disease information $I(t)$ is related to its degree. If

an individual has more friends/neighbors, he/she may gain more information about disease through multiple edges, and may make more adaptive behavior to protect himself/herself to reduce his/her weight significantly.

Behavior-inertia function $m(k)$ is also an increasing functions of k, which implies that individuals with more friends/neighbours lose more weight after they experience a disease.

The corresponding λ_{ik} becomes

$$\tilde{\lambda}_{ik} = \frac{\lambda\langle k\rangle g(k)\exp\left(-h(k)I(t) - m(k)\right)}{\langle kg(k)\exp\left(-h(k)I(t) - m(k)\right)\rangle}. \tag{2.5}$$

Substituting (2.5) into (2.2), we have

$$\tilde{\Theta}_k(t) = \frac{\lambda g(k)\exp\left(-h(k)I(t) - m(k)\right)}{\langle kg(k)\exp(-h(k)I(t) - m(k))\rangle}\sum_i \varphi(i)P(i)I_i(t), \tag{2.6}$$

which corresponds to the SIS model with behavior inertia on adaptively weighted networks.

2.2.3 SIS model with behavior inertia and time delay on adaptively weighted networks

The information that individuals obtain is a kind of delayed information due to the following two reasons:

1. The responses of individuals to the information are delayed;
2. The information is not timely released, that is, the release of the information is delayed.

Suppose that the time delay is denoted by a positive constant τ, the corresponding λ_{ik} becomes

$$\bar{\lambda}_{ik} = \frac{\lambda\langle k\rangle g(k)\exp\left(-h(k)I(t-\tau) - m(k)\right)}{\langle kg(k)\exp\left(-h(k)I(t-\tau) - m(k)\right)\rangle}. \tag{2.7}$$

Substituting (2.7) into (2.2), we can obtain $\bar{\Theta}_k(t)$:

$$\bar{\Theta}_k(t) = \frac{\lambda g(k)\exp\left(-h(k)I(t-\tau) - m(k)\right)}{\langle kg(k)\exp\left(-h(k)I(t-\tau) - m(k)\right)\rangle}\sum_i \varphi(i)P(i)I_i(t), \tag{2.8}$$

which corresponds to the SIS model with behavior inertia and time delay on adaptively weighted networks.

3 Analysis of epidemic models

3.1 Preliminaries

Let $A = (a_{kj})$, $B = (b_{kj}) \in R^{n\times n}$ be nonnegative matrices, namely, all of their entries are nonnegative. We say $A \geq B$ if $a_{kj} \geq b_{kj}$ for all k and j, and $A > B$ if $A \geq B$ and $A \neq B$.

Definition 1 For $n > 1$, a matrix $A \in R^{n\times n}$ is reducible if there exists a permutation matrix Q, we have

$$QAQ^T = \begin{pmatrix} A_1 & 0 \\ A_2 & A_3 \end{pmatrix},$$

where A_1 and A_3 are square matrices. Otherwise, A is irreducible.

The following properties of nonnegative matrices are verified in Berman and Plemmons (1979), which will be used to study the dynamic behavior of our models.

R1. If A is nonnegative, then the spectral radius $\rho(A)$ of A is one of its eigenvalues, and A has a nonnegative eigenvector corresponding to $\rho(A)$;

R2. If A is nonnegative and irreducible, then $\rho(A)$ is a simple eigenvalue, and A has a positive eigenvector ω corresponding to $\rho(A)$;

R3. If $0 \leq A \leq B$, then $\rho(A) \leq \rho(B)$. Moreover, if $0 \leq A < B$ and $A + B$ is irreducible, then $\rho(A) < \rho(B)$.

Let $N_k(t) = S_k(t) + I_k(t)$, which is the density of nonempty nodes with degree k at time t. Then, by system (2.1), the evolution of $N_k(t)$ are governed by the following differential equations:

$$\frac{dN_k(t)}{dt} = bk[1 - N_k(t)]\Phi_k(t) - dN_k(t), \tag{3.1}$$

where $\Phi_k(t) = \sum_i N_i(t)P(i)/\langle k\rangle$.

Zhu et al. (2013) drew the following conclusions:

1. When $b < d$, $\lim_{t\to\infty} N_k(t) = 0$, then the equilibrium $N_k^0(0, 0, \ldots, 0)$ of system (3.1) is globally stable. In this case, the population becomes extinct, and there is no other dynamic behaviors any more;
2. When $b > d$, $\lim_{t\to\infty} N_k(t) = N_k^*$ where N_k^* satisfies:

$$N_k^* = \frac{bk\Phi^*}{d + bk\Phi^*}, \quad \Phi^* = \sum_{i=1}^{n} \frac{P(i)}{\langle k\rangle} N_i^*.$$

The analysis of the stability of model (2.4) has been given in Zhu et al. (2013), here, we mainly investigate the stability of models (2.6) and (2.8). Based on the above results, we consider only the case of $b > d$. Since the original system and the limiting system have the same asymptotic dynamical behaviors, to study the stability of models (2.6) and (2.8), we only need to consider their limiting systems under which $N_k^* = S_k(t) + I_k(t)$.

3.2 Epidemic threshold of the model with adaptive weights

The limiting systems of system (2.1) corresponding to models (2.4) and (2.6) are written as follows:

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1 - N_k^*]\sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - S_k(t)\frac{\lambda kg(k)}{\langle kg(k)\rangle}\phi(t) + \gamma[N_k^* - S_k(t)], \\ \frac{dI_k(t)}{dt} = bk[1 - N_k^*]\sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + [N_k^* - I_k(t)]\frac{\lambda kg(k)}{\langle kg(k)\rangle}\phi(t) - (d+\gamma)I_k(t), \end{cases} \tag{3.2}$$

and

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1-N_k^*]\sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - S_k(t)\frac{\lambda kg(k)\exp(-h(k)I(t)-m(k))}{\langle kg(k)\exp(-h(k)I(t)-m(k))\rangle}\phi(t) + \gamma[N_k^* - S_k(t)], \\ \frac{dI_k(t)}{dt} = bk[1 - N_k^*]\sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + [N_k^* - I_k(t)]\frac{\lambda kg(k)\exp(-h(k)I(t)-m(k))}{\langle kg(k)\exp(-h(k)I(t)-m(k))\rangle}\phi(t) - (d+\gamma)I_k(t), \end{cases} \tag{3.3}$$

where $\phi(t) = \sum_i \varphi(i)P(i)I_i(t)$.

Systems (3.2) and (3.3) always have the disease-free equilibrium

$$E_0 = (S_1^0, 0, S_2^0, 0, \ldots, S_n^0, 0),$$

where $S_k^0 = N_k^*, \quad k = 1, 2, \ldots, n$.

In the neighborhood of the disease-free equilibrium E_0 of systems (3.2) and (3.3), the rates of transfer of individuals out of compartments are $V = (d+\gamma)E$ where E is the identity matrix, and the rates of appearance of new infections of systems (3.2) and (3.3) are F and $\tilde{F}$, respectively, then we have

$$F = \begin{pmatrix} X_1P(1) + Y_1\varphi(1)P(1) & X_1P(2) + Y_1\varphi(2)P(2) & \cdots & X_1P(n) + Y_1\varphi(n)P(n) \\ X_2P(1) + Y_2\varphi(1)P(1) & X_2P(2) + Y_2\varphi(2)P(2) & \cdots & X_2P(n) + Y_2\varphi(n)P(n) \\ \vdots & \vdots & \ddots & \vdots \\ X_nP(1) + Y_n\varphi(1)P(1) & X_nP(2) + Y_n\varphi(2)P(2) & \cdots & X_nP(n) + Y_n\varphi(n)P(n) \end{pmatrix},$$

and

$$\tilde{F} = \begin{pmatrix} X_1P(1) + \tilde{Y}_1\varphi(1)P(1) & X_1P(2) + \tilde{Y}_1\varphi(2)P(2) & \cdots & X_1P(n) + \tilde{Y}_1\varphi(n)P(n) \\ X_2P(1) + \tilde{Y}_2\varphi(1)P(1) & X_2P(2) + \tilde{Y}_2\varphi(2)P(2) & \cdots & X_2P(n) + \tilde{Y}_2\varphi(n)P(n) \\ \vdots & \vdots & \ddots & \vdots \\ X_nP(1) + \tilde{Y}_n\varphi(1)P(1) & X_nP(2) + \tilde{Y}_n\varphi(2)P(2) & \cdots & X_nP(n) + \tilde{Y}_n\varphi(n)P(n) \end{pmatrix},$$

where $X_k = bk(1 - N_k^*)/\langle k\rangle$, $Y_k = \lambda kg(k)N_k^*/\langle kg(k)\rangle$ and $\tilde{Y}_k = \lambda kg(k)N_k^* \exp(-m(k))/\langle kg(k)\exp(-m(k))\rangle$.

Obviously, V is a nonsingular M-matrix, and $F, \tilde{F}$ are nonnegative matrices. According to the concepts of next generation matrix and the basic reproduction number given in Van den Driessche and Watmough (2002), the basic reproduction numbers of systems (3.2) and (3.3) equal to

$$R_0 = \rho(FV^{-1}) = \frac{1}{d+\gamma}\rho(F), \tag{3.4}$$

and

$$\tilde{R}_0 = \rho(\tilde{F}V^{-1}) = \frac{1}{d+\gamma}\rho(\tilde{F}), \tag{3.5}$$

where $\rho(A)$ denotes the spectral radius of a matrix A.

Assume that ρ_i and $\tilde{\rho}_i (i = 1, 2, \ldots, n)$ are eigenvalues of the characteristic equation of F and $\tilde{F}$, respectively, then (3.4) and (3.5) can be rewritten as

$$R_0 = \frac{1}{d+\gamma}\max\{|\rho_i|, i = 1, 2, \ldots, n\}, \tag{3.6}$$

and

$$\tilde{R}_0 = \frac{1}{d+\gamma}\max\{|\tilde{\rho}_i|, i = 1, 2, \ldots, n\}, \tag{3.7}$$

where $|\cdot|$ represents the modulus.

Afterwards, we will give a detailed derivation of $\tilde{R}_0$ in (3.7). The associated characteristic equation of $\tilde{F}$ is $|\tilde{\rho}E - \tilde{F}| = 0$, and its specific form is as follows:

$$\begin{vmatrix} \tilde{\rho} - [X_1P(1) + \tilde{Y}_1\varphi(1)P(1)] & -[X_1P(2) + \tilde{Y}_1\varphi(2)P(2)] & \cdots & -[X_1P(n) + \tilde{Y}_1\varphi(n)P(n)] \\ -[X_2P(1) + \tilde{Y}_2\varphi(1)P(1)] & \tilde{\rho} - [X_2P(2) + \tilde{Y}_2\varphi(2)P(2)] & \cdots & -[X_2P(n) + \tilde{Y}_2\varphi(n)P(n)] \\ \vdots & \vdots & \ddots & \vdots \\ -[X_nP(1) + \tilde{Y}_n\varphi(1)P(1)] & -[X_nP(2) + \tilde{Y}_n\varphi(2)P(2)] & \cdots & \tilde{\rho} - [X_nP(n) + \tilde{Y}_n\varphi(n)P(n)] \end{vmatrix} = 0.$$

By the properties of determinants, we can simplify the above characteristic equation as follows:

$$\begin{vmatrix} \sum_k X_kP(k) - \tilde{\rho} & \sum_k \tilde{Y}_kP(k) & 0 & \cdots & 0 \\ \sum_k X_kP(k)(\varphi(1) - \varphi(k)) - \tilde{\rho}\varphi(1) & \sum_k \tilde{Y}_kP(k)(\varphi(1) - \varphi(k)) + \tilde{\rho} & 0 & \cdots & 0 \\ 0 & 0 & \tilde{\rho} & \cdots & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & \cdots & \tilde{\rho} \end{vmatrix} = 0.$$

Thus, the characteristic equation of $\tilde{F}$ equals to

$$\tilde{\rho}^{n-2}\left\{\tilde{\rho}^2 - \left[\sum_k X_kP(k) + \sum_k \tilde{Y}_k\varphi(k)P(k)\right]\tilde{\rho} + \sum_k X_kP(k)\sum_k \tilde{Y}_k\varphi(k)P(k) - \sum_k X_k\varphi(k)P(k)\sum_k \tilde{Y}_kP(k)\right\} = 0.$$

It can be further simplified into the following form:

$$\tilde{\rho}^{n-2}\left\{\tilde{\rho}^2-\left[\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle\right]\tilde{\rho}+\langle X_k\rangle\langle\tilde{Y}_k\varphi(k)\rangle-\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle\right\}=0.$$

Hence, $\tilde{\rho}=0$ is the trivial root and other nontrivial roots satisfy the following equation:

$$\tilde{\rho}^2-\left[\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle\right]\tilde{\rho}+\langle X_k\rangle\langle\tilde{Y}_k\varphi(k)\rangle-\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle=0. \tag{3.8}$$

Therefore, the solutions of (3.8) are

$$\tilde{\rho}_{1,2}=\frac{\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle\pm\sqrt{(\langle X_k\rangle-\langle\tilde{Y}_k\varphi(k)\rangle)^2+4\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle}}{2}.$$

According to the definition of $\tilde{R}_0$ in (3.7), we obtain

$$\tilde{R}_0=\frac{1}{d+\gamma}\max\{|\tilde{\rho}_i|,i=1,2,\ldots,n\}=\frac{\tilde{\rho}_2}{d+\gamma}, \tag{3.9}$$

where $\tilde{\rho}_2=\frac{\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle+\sqrt{(\langle X_k\rangle-\langle\tilde{Y}_k\varphi(k)\rangle)^2+4\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle}}{2}$.

Similar to the derivation of $\tilde{R}_0$, we have the expression of R_0 in (3.6)

$$R_0=\frac{1}{d+\gamma}\max\{|\rho_i|,i=1,2,\ldots,n\}=\frac{\rho_2}{d+\gamma}, \tag{3.10}$$

where $\rho_2=\frac{\langle X_k\rangle+\langle Y_k\varphi(k)\rangle+\sqrt{(\langle X_k\rangle-\langle Y_k\varphi(k)\rangle)^2+4\langle X_k\varphi(k)\rangle\langle Y_k\rangle}}{2}$.

Next, we compare the basic reproduction numbers of models (2.4) and (2.6). For arbitrary elements f_{kj}, $\tilde{f}_{kj}$, $k,j=1,2,\ldots,n$ in F and $\tilde{F}$, we have

$$\begin{aligned}
f_{kj}-\tilde{f}_{kj}&=[X_kP(j)+Y_k\varphi(j)P(j)]-[X_kP(j)+\tilde{Y}_k\varphi(j)P(j)]\\
&=[Y_k-\tilde{Y}_k]\varphi(j)P(j)\\
&=\left[\lambda kg(k)N_k^*/\langle kg(k)\rangle-\lambda kg(k)N_k^*\exp(-m(k))/\langle kg(k)\exp(-m(k))\rangle\right]\\
&\quad\varphi(j)P(j)\\
&=\lambda kg(k)N_k^*\left[\frac{1}{\langle kg(k)\rangle}-\frac{\exp(-m(k))}{\langle kg(k)\exp(-m(k))\rangle}\right]\varphi(j)P(j).
\end{aligned}$$

Note that

$$\frac{1}{\langle kg(k)\rangle}-\frac{\exp(-m(k)}{\langle kg(k)\exp(-m(k))\rangle}\geq 0.$$

Hence, $f_{kj}-\tilde{f}_{kj}\geq 0$, $k,j=1,2,\ldots,n$, which means $\tilde{F}\leq F$. Moreover, $F,\tilde{F}$ are nonnegative matrices and $\tilde{F}\neq F$, so $0\leq\tilde{F}<F$. For arbitrary element

$f_{kj}+\tilde{f}_{kj},\ k,\ j=1,2,\ldots,n$ in $F+\tilde{F}$, $f_{kj}+\tilde{f}_{kj}>0$, i.e., there is no zero elements in $F+\tilde{F}$, so $F+\tilde{F}$ is irreducible. Then, we can obtain $\rho(\tilde{F})<\rho(F)$ by R3 of Sect. 3.1. Furthermore, we obtain $\tilde{R}_0<R_0$, which implies that the adaptation of weights can lower the basic reproduction number.

Remark 1 If the disease cannot be transmitted by contact but just by congenital infection, i.e., $\lambda=0$, then the basic reproduction numbers R_0 and $\tilde{R}_0$ become

$$R_{01}=\tilde{R}_{01}=\frac{b\left(\langle k\rangle-\langle kN_k^*\rangle\right)}{(d+\gamma)\langle k\rangle},$$

which only depend on the birth and death rates, the recovery rate, and the degree distribution of the network.

Remark 2 If the effects of birth and death are ignored, i.e., $b=d=0$, $N_k=1$, then the basic reproduction numbers R_0 and $\tilde{R}_0$ are simplified to

$$R_{02}=\frac{\lambda\langle kg(k)\varphi(k)\rangle}{\gamma\langle kg(k)\rangle},\quad \tilde{R}_{02}=\frac{\lambda\langle kg(k)\exp(-m(k))\varphi(k)\rangle}{\gamma\langle kg(k)\exp(-m(k))\rangle}.$$

It can be observed that the infectivity function $\varphi(k)$ has a stronger effect than the weight function $g(k)$ and behavior-inertia function $m(k)$ on R_{02} and $\tilde{R}_{02}$. Further, we list the following two cases:

(H1) If the infectivity function $\varphi(k)$ is the same for each degree, i.e., $\varphi(k)=w$, implying that each node establishes contact with its w neighbors per unit time, then the basic reproduction numbers become $\lambda w/\gamma$, which is exactly the epidemic threshold for regular networks. In this case, the epidemic threshold has nothing to do with the weight function and the network structure;

(H2) If the weights are not considered, i.e., $g(k)$, $\tilde{g}(k,t)$ is a constant, then the basic reproduction numbers become $\lambda\langle k\varphi(k)\rangle/\gamma\langle k\rangle$; further, if $\varphi(k)=k$, then the reproduction number becomes $\lambda\langle k^2\rangle/\gamma\langle k\rangle$, which is the same as the classical results in Pastor-Satorras and Vespignani (2001b), Moreno et al. (2002) and Barthélemy et al. (2005).

Remark 3 If the infectivity function $\varphi(k)$ is the same for each degree, i.e., $\varphi(k)=w$, it follows from (3.9) and (3.10) that the basic reproduction numbers R_0 and $\tilde{R}_0$ become

$$\begin{aligned}R_{03}&=\frac{b(\langle k\rangle-\langle kN_k^*\rangle)}{(d+\gamma)\langle k\rangle}+\frac{\lambda w\langle kg(k)N_k^*\rangle}{(d+\gamma)\langle kg(k)\rangle},\\ \tilde{R}_{03}&=\frac{b(\langle k\rangle-\langle kN_k^*\rangle)}{(d+\gamma)\langle k\rangle}+\frac{\lambda w\langle kg(k)\exp(-m(k))N_k^*\rangle}{(d+\gamma)\langle kg(k)\exp(-m(k))\rangle}.\end{aligned}$$

The first and second terms in the expressions of R_{03} and $\tilde{R}_{03}$ are contributed by congenital infection (from parents) and contact infection (from linking neighbors), respectively.

3.3 Epidemic threshold of the model with adaptive weights and time delay

We obtain the following limiting system of system (2.1) in the case of $\Theta_k(t) = \bar{\Theta}_k(t)$ corresponding to model (2.8) when $t > \tau$:

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1 - N_k^*]\sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - S_k(t)\frac{\lambda kg(k)\exp(-h(k)I(t-\tau)-m(k))}{\langle kg(k)\exp(-h(k)I(t-\tau)-m(k))\rangle}\phi(t) + \gamma[N_k^* - S_k(t)], \\ \frac{dI_k(t)}{dt} = bk[1 - N_k^*]\sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + [N_k^* - I_k(t)]\frac{\lambda kg(k)\exp(-h(k)I(t-\tau)-m(k))}{\langle kg(k)\exp(-h(k)I(t-\tau)-m(k))\rangle}\phi(t) - (d+\gamma)I_k(t), \end{cases} \tag{3.11}$$

where $\phi(t) = \sum_i \varphi(i)P(i)I_i(t)$.

Initial conditions of model (2.8) satisfy the following SIS differential equation model on fixedly weighted networks without time delay and adaptation:

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)]\sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - S_k(t)\frac{\lambda kg(k)}{\langle kg(k)\rangle}\phi(t) + \gamma I_k(t), \\ \frac{dI_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)]\sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + S_k(t)\frac{\lambda kg(k)}{\langle kg(k)\rangle}\phi(t) - (d+\gamma)I_k(t). \end{cases} \tag{3.12}$$

This is a suitable assumption since there is no information about diseases although infections occur during the time $0 \le t \le \tau$. Furthermore, individuals will not make adaptive reactions to disease information.

Denote $H_k(I) = \frac{\exp(-h(k)I(t)-m(k))}{\langle kg(k)\exp(-h(k)I(t)-m(k))\rangle}$, by imposing steady state $\dot{I}_k(t) = 0$ in (3.11), we get the stationary solution

$$I_k = \frac{X_k I + \lambda kg(k)N_k^* H_k(I)\phi}{d + \gamma + \lambda kg(k)H_k(I)\phi}. \tag{3.13}$$

Substituting (3.13) into $\phi(t) = \sum_i \varphi(i)P(i)I_i(t)$, we obtain a self-consistency equation of ϕ:

$$\phi = \sum_{i=1}^{n} \varphi(i)P(i)\frac{X_i I + \lambda ig(i)N_i^* H_i(I)\phi}{d + \gamma + \lambda ig(i)H_i(I)\phi} = L(\phi). \tag{3.14}$$

Obviously, $\phi = 0$ is a solution of Eq. (3.14). If there is another positive solution ($\phi \in (0, 1]$), the following inequality must be satisfied:

$$\left.\frac{dL(\phi)}{d\phi}\right|_{\phi=0} > 1. \tag{3.15}$$

Substituting (3.13) into $I(t) = \sum_i P(i)I_i(t)$, we have

$$I = \sum_{i=1}^{n} P(i)\frac{X_i I + \lambda ig(i)N_i^* H_i(I)\phi}{d + \gamma + \lambda ig(i)H_i(I)\phi}. \tag{3.16}$$

According to Eq. (3.16), we get

$$\left.\frac{dI}{d\phi}\right|_{\phi=0} = \frac{\langle Y_k\rangle}{d+\gamma-\langle X_k\rangle}. \tag{3.17}$$

Therefore, inequality (3.15) can be further expanded

$$\begin{aligned}
\left.\frac{dL(\phi)}{d\phi}\right|_{\phi=0} &= \sum_{i=1}^{n}\varphi(i)P(i)\left\{\frac{\left[X_i\frac{dI}{d\phi}+\lambda ig(i)N_i^*\left(\frac{dH_i(I)}{dI}\frac{dI}{d\phi}\phi+H_i(I)\right)\right](d+\gamma+\lambda ig(i)H_i(I)\phi)}{(d+\gamma+\lambda ig(i)H_i(I)\phi)^2}\right.\\
&\quad\left.-\frac{(X_iI+\lambda ig(i)N_i^*H_i(I)\phi)\lambda ig(i)\left(\frac{dH_i(I)}{dI}\frac{dI}{d\phi}\phi+H_i(I)\right)}{(d+\gamma+\lambda ig(i)H_i(I)\phi)^2}\right\}\Big|_{\phi=0}\\
&=\sum_{i=1}^{n}\varphi(i)P(i)\frac{\left[X_i\frac{dI}{d\phi}+\lambda ig(i)N_i^*\left(\frac{dH_i(I)}{dI}\frac{dI}{d\phi}\phi+H_i(I)\right)\right](d+\gamma+\lambda ig(i)H_i(I)\phi)}{(d+\gamma+\lambda ig(i)H_i(I)\phi)^2}\Big|_{\phi=0}\\
&=\sum_{i=1}^{n}\varphi(i)P(i)\frac{\left(X_i\frac{dI}{d\phi}+\lambda ig(i)N_i^*H_i(I)\right)|_{\phi=0}}{d+\gamma}\\
&=\sum_{i=1}^{n}\varphi(i)P(i)\frac{X_i\frac{\langle Y_k\rangle}{d+\gamma-\langle X_k\rangle}+Y_i}{d+\gamma}\\
&=\frac{\langle X_k\varphi(k)\rangle\langle Y_k\rangle+(d+\gamma-\langle X_k\rangle)\langle Y_k\varphi(k)\rangle}{(d+\gamma)(d+\gamma-\langle X_k\rangle)}\\
&>1.
\end{aligned}$$

The above inequality can be further simplified as

$$(d+\gamma)^2-\left[\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle\right](d+\gamma)+\langle X_k\rangle\langle\tilde{Y}_k\varphi(k)\rangle-\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle<0. \tag{3.18}$$

Based on inequality (3.18), we have

$$d+\gamma<\frac{\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle+\sqrt{(\langle X_k\rangle-\langle\tilde{Y}_k\varphi(k)\rangle)^2+4\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle}}{2}=\tilde{\rho}_2. \tag{3.19}$$

From inequality (3.19), we get the basic reproduction number of model (2.8):

$$\bar{R}_0=\frac{\tilde{\rho}_2}{d+\gamma}=\tilde{R}_0.$$

This implies that time delay will not change the basic reproduction number of model (2.6). Since $\tilde{R}_0<R_0$, we can obtain $\bar{R}_0=\tilde{R}_0<R_0$.

Based on the above analysis, we have the following theorem:

Theorem 3.1 *Model (2.8) has a disease-free equilibrium* $E_0=(S_1^0,0,S_2^0,0,\ldots,S_n^0,0)$, *where* $S_k^0=N_k^*$, $k=1,2,\ldots,n$. *If* $\bar{R}_0>1$, *model (2.8) has a positive equilibrium* $E^*=(S_1^*,I_1^*,S_2^*,I_2^*,\ldots,S_n^*,I_n^*)$, *which satisfies*

$$I_k^* = \frac{X_k I^* + \lambda k g(k) N_k^* H_k(I^*)\phi^*}{d+\gamma+\lambda k g(k) H_k(I^*)\phi^*}, \quad S_k^* = N_k^* - I_k^*,$$
$$\phi^* = \sum_{i=1}^{n} \varphi(i) P(i) I_i^*, \quad k = 1, 2, \ldots, n.$$

3.4 Stability analysis for the adaptively weighted model

The basic reproduction number is a critical value to determine whether epidemics prevail or not. The biological significance of the basic reproduction number is that if $\tilde{R}_0 < 1$, the disease dies out; while if $\tilde{R}_0 > 1$, the disease may become endemic (Van den Driessche and Watmough 2002; Diekmann et al. 1990). According to the Theorem 2 in Van den Driessche and Watmough (2002), we obtain the following stability results.

Theorem 3.2 (1) *If $\tilde{R}_0 < 1$, the disease-free equilibrium E_0 of system (3.3) is locally asymptotically stable,*
(2) *If $\tilde{R}_0 > 1$, the disease-free equilibrium E_0 is unstable, where $\tilde{R}_0$ is defined by (3.5).*

Denote $\Lambda_k = bk[1 - N_k^*]\sum_i N_i^* P(i)/\langle k\rangle + \gamma N_k^*$, $\alpha_{kj} = bk[1 - N_k^*]P(j)/\langle k\rangle$, $\beta_{kj} = \lambda k g(k)\varphi(j)P(j)$, $f_j(I_j) = I_j$, $f_{kj}(S_k, I_j, I) = \frac{\exp(-h(k)I(t)-m(k))}{\langle kg(k)\exp(-h(k)I(t)-m(k))\rangle} S_k I_j$, then system (3.3) can be rewritten as follows:

$$\begin{cases} \frac{dS_k(t)}{dt} = \Lambda_k - (d+\gamma)S_k(t) - \sum_j \alpha_{kj} f_j(I_j) - \sum_j \beta_{kj} f_{kj}(S_k, I_j, I) \\ \qquad\quad = \Lambda_k - (d+\gamma)S_k(t) - \sum_j \beta_{kj}\left(\frac{\alpha_{kj}}{\beta_{kj}} f_j(I_j) + f_{kj}(S_k, I_j, I)\right), \\ \frac{dI_k(t)}{dt} = \sum_j \alpha_{kj} f_j(I_j) + \sum_j \beta_{kj} f_{kj}(S_k, I_j, I) - (d+\gamma)I_k(t) \\ \qquad\quad = \sum_j \beta_{kj}\left(\frac{\alpha_{kj}}{\beta_{kj}} f_j(I_j) + f_{kj}(S_k, I_j, I)\right) - (d+\gamma)I_k(t). \end{cases} \tag{3.20}$$

Therefore, omega limit sets of system (3.20) are contained in the following bounded region

$$\Gamma = \left\{(S_1, I_1, \ldots, S_n, I_n) \in R_+^{2n} \middle| S_k \le \frac{\Lambda_k}{d+\gamma}, \ S_k + I_k = N_k^*, k = 1, 2, \ldots, n\right\}. \tag{3.21}$$

It can be verified that Γ in (3.21) is positively invariant with respect to system (3.20). Let Γ° denote the interior of Γ, and the disease-free equilibrium E_0 of system (3.20) is on the boundary of Γ.

Proposition 3.3 *Assume that the matrices $A = (\alpha_{kj})$ and $B = (\beta_{kj})$ are irreducible. Then the following results hold:*

1. *If $\tilde{R}_0 \le 1$, then E_0 is the unique equilibrium of system (3.3) and it is globally stable in Γ;*
2. *If $\tilde{R}_0 > 1$, then E_0 is unstable and system (3.3) is uniformly persistent.*

Proof Let $I = (I_1, I_2, \ldots, I_n) \in R^n$ and $I_0 = (0, 0, \ldots, 0)$. The second equation of system (3.3) can be rewritten as a vector equation as follows:

$$\dot{I}(t) = M(I)I - (d + \gamma)I.$$

where $m_{kj} = X_k P(j) + Z_k \varphi(j) P(j)$,
$Z_k = \lambda k g(k)[N_k^* - I_k(t)] \frac{\exp(-h(k)I(t)-m(k))}{\langle kg(k)\exp(-h(k)I(t)-m(k))\rangle}$.

Then $M_0 = M(I_0) = \tilde{F}$. By (3.5), we can obtain $\tilde{R}_0 = \frac{1}{d+\gamma}\rho(\tilde{F}) = \frac{1}{d+\gamma}\rho(M_0)$.

For $1 \le k \le n$, we have $0 \le M(I) \le M(I_0) = M_0$, and if $I \ne I_0$, then $M(I) < M(I_0)$. Since A, B are irreducible, we know $M(I)$ and M_0 are irreducible. Furthermore, $M(I) + M_0$ is irreducible, thus we obtain $\rho(M(I)) < \rho(M_0)$ if $I \ne I_0$ by R3 of Sect. 3.1.

If $\tilde{R}_0 = \frac{1}{d+\gamma}\rho(M_0) \le 1$ and $I \ne I_0$, then $\frac{1}{d+\gamma}\rho(M(I)) < 1$, and $M(I)I-(d+\gamma)I$ has only the trivial solution $I = I_0$. Thus E_0 is the only equilibrium of system (3.3) in Γ if $\tilde{R}_0 \le 1$.

Let $\omega = (\omega_1, \omega_2, \ldots, \omega_n)$ be a left eigenvector of M_0 corresponding to $\rho(M_0)$, i.e.,

$$\omega\rho(M_0) = \omega M_0.$$

Since $\tilde{F}$ is irreducible, which implies that M_0 is irreducible, we know $\omega_i > 0$ for $i = 1, 2, \ldots, n$, by R2 of Sect. 3.1.

Considering the following Lyapunov function:

$$L(t) = \sum_{k=1}^{n} \omega_k I_k.$$

Calculating the derivative of $L(t)$ along the solution of system (3.3), we have

$$\begin{aligned}\frac{dL(t)}{dt} &= \sum_{k=1}^{n} \omega_k \dot{I}_k = \omega[M(I)I - (d+\gamma)I] \\ &\le \omega[M_0 I - (d+\gamma)I] = \omega M_0 I - (d+\gamma)\omega I \\ &= \rho(M_0)\omega I - (d+\gamma)\omega I = (d+\gamma)\tilde{R}_0\omega I - (d+\gamma)\omega I \\ &= (d+\gamma)(\tilde{R}_0 - 1)\omega I.\end{aligned}$$

If $\tilde{R}_0 < 1$, then $\frac{dL(t)}{dt} < 0$, which means $I = I_0$. If $\tilde{R}_0 = 1$, then $\frac{dL(t)}{dt} = 0$, which implies $\omega M(I)I = (d+\gamma)\omega I$. If $I \ne I_0$, then $\omega M(I) < \omega M_0 = \omega\rho(M_0) = (d+\gamma)\omega$. Thus $\omega M(I)I = (d+\gamma)\omega I$ has only the trivial solution $I = I_0$. Therefore, $\frac{dL(t)}{dt} = 0 \Leftrightarrow I = I_0$ if $\tilde{R}_0 \le 1$. It can be verified that the only compact invariant subset of the set where $\frac{dL(t)}{dt} = 0$ is the singleton $\{E_0\}$. By LaSalle's Invariance Principle (LaSalle 1976), E_0 is globally asymptotically stable in Γ if $\tilde{R}_0 \le 1$.

If $\tilde{R}_0 > 1$ and $I \ne I_0$, we know that $\omega M_0 - \omega = (d+\gamma)(\tilde{R}_0 - 1)\omega > 0$, and thus $\frac{dL(t)}{dt} = \omega[M(I)I-(d+\gamma)I] > 0$ in a neighborhood of E_0 in Γ°, by continuity. This

implies that E_0 is unstable. Using a uniform persistence result from Freedman et al. (1994) and a similar argument as in the proof of Proposition 3.3 in Li et al. (1999), we can show that, when $\tilde{R}_0 > 1$, the instability of E_0 implies the uniform persistence of system (3.3). This completes the proof of Proposition 3.3. □

Corollary 3.4 *Assume that the matrices $A = (\alpha_{kj})$ and $B = (\beta_{kj})$ are irreducible. If $\tilde{R}_0 > 1$, then system (3.3) has at least one endemic equilibrium.*

Denote by

$$E^* = (S_1^*, I_1^*, S_2^*, I_2^*, \ldots, S_n^*, I_n^*)$$

the endemic equilibrium, where $S_k^*, I_k^* > 0$ for $k = 1, 2, \ldots, n$. We have the following main results on the uniqueness and global stability of E^* when $\tilde{R}_0 > 1$. Our results will be stated for system (3.20), and can be translated straightforwardly to system (3.3).

Theorem 3.5 *Assume that the matrices $A = (\alpha_{kj})$ and $B = (\beta_{kj})$ are irreducible, and $f_j(I_j)$ and $f_{kj}(S_k, I_j, I)$ satisfy the following conditions:*

$$\begin{aligned} &0 < \lim_{I_j \to 0^+} \frac{f_j(I_j) + f_{kj}(S_k, I_j, I)}{I_j} \leq +\infty, \quad 0 < S_k \leq S_k^0, \quad \textit{and} \\ &\begin{cases} \left(f_j(I_j)S_k^* - f_j(I_j^*)S_k\right)\left(\frac{f_j(I_j)S_k^*}{I_j} - \frac{f_j(I_j^*)S_k}{I_j^*}\right) \leq 0, \\ \left(f_{kj}(S_k^*, I_j, I) - f_{kj}(S_k^*, I_j^*, I^*)\right)\left(\frac{f_{kj}(S_k^*, I_j, I)}{I_j} - \frac{f_{kj}(S_k^*, I_j^*, I^*)}{I_j^*}\right) \leq 0. \end{cases} \end{aligned} \tag{3.22}$$

If $\tilde{R}_0 > 1$, and $S_k, I_j > 0$, then there exists a unique endemic equilibrium E^* for system (3.20), and E^* is globally asymptotically stable in Γ°.

Proof From Corollary 3.4, we know that the endemic equilibrium E^* exists if $\tilde{R}_0 > 1$. We prove that E^* is globally asymptotically stable in Γ°. In this case, the endemic equilibrium is unique.

We consider a Lyapunov function for a single-group model in Korobeinikov (2007) as follows:

$$V_k = S_k - S_k^* \ln S_k + I_k - I_k^* \ln I_k.$$

Next, we verify that V_k satisfies the assumptions of Theorem 3.1 in Li and Shuai (2010).

Using the equilibrium equations

$$\Lambda_k = (d + \gamma)S_k^*(t) + \sum_j \alpha_{kj} f_j(I_j^*) + \sum_j \beta_{kj} f_{kj}(S_k^*, I_j^*, I^*),$$

and

$$(d + \gamma)I_k^* = \sum_j \alpha_{kj} f_j(I_j^*) + \sum_j \beta_{kj} f_{kj}(S_k^*, I_j^*, I^*),$$

we obtain

$$\begin{aligned}
\frac{dV_k}{dt} &= \frac{dS_k}{dt} - \frac{S_k^*}{S_k}\frac{dS_k}{dt} + \frac{dI_k}{dt} - \frac{I_k^*}{I_k}\frac{dI_k}{dt} \\
&= \Lambda_k - (d+\gamma)S_k(t) - \sum_j \alpha_{kj} f_j(I_j) - \sum_j \beta_{kj} f_{kj}(S_k, I_j, I) \\
&\quad - \Lambda_k \frac{S_k^*}{S_k} + (d+\gamma)S_k^* + \frac{S_k^*}{S_k}\sum_j \alpha_{kj} f_j(I_j) + \frac{S_k^*}{S_k}\sum_j \beta_{kj} f_{kj}(S_k, I_j, I) \\
&\quad + \sum_j \alpha_{kj} f_j(I_j) + \sum_j \beta_{kj} f_{kj}(S_k, I_j, I) \\
&\quad - \frac{I_k}{I_k^*}\left[\sum_j \alpha_{kj} f_j(I_j^*) + \sum_j \beta_{kj} f_{kj}(S_k^*, I_j^*, I^*)\right] \\
&\quad - \frac{I_k^*}{I_k}\sum_j \alpha_{kj} f_j(I_j) - \frac{I_k^*}{I_k}\sum_j \beta_{kj} f_{kj}(S_k, I_j, I) + (d+\gamma)I_k^*(t) \\
&= -(d+\gamma)S_k^*\left(\frac{S_k^*}{S_k} + \frac{S_k}{S_k^*} - 2\right) \\
&\quad + \sum_j \alpha_{kj} f_j(I_j^*)\left(2 - \frac{S_k^*}{S_k} + \frac{f_j(I_j)S_k^*}{f_j(I_j^*)S_k} - \frac{f_j(I_j)I_k^*}{f_j(I_j^*)I_k} - \frac{I_k}{I_k^*}\right) \\
&\quad + \sum_j \beta_{kj} f_{kj}(S_k^*, I_j^*, I^*) \\
&\qquad \times \left(2 - \frac{S_k^*}{S_k} + \frac{f_{kj}(S_k^*, I_j, I)}{f_{kj}(S_k^*, I_j^*, I^*)} - \frac{f_{kj}(S_k, I_j, I)I_k^*}{f_{kj}(S_k^*, I_j^*, I^*)I_k} - \frac{I_k}{I_k^*}\right) \\
&= -(d+\gamma)S_k^*\left(\frac{S_k^*}{S_k} + \frac{S_k}{S_k^*} - 2\right) \\
&\quad + \sum_j \beta_{kj} f_{kj}(S_k^*, I_j^*, I^*) \\
&\qquad \times \left[\frac{\alpha_{kj} f_j(I_j^*)}{\beta_{kj} f_{kj}(S_k^*, I_j^*, I^*)}\left(2 - \frac{S_k^*}{S_k} + \frac{f_j(I_j)S_k^*}{f_j(I_j^*)S_k} - \frac{f_j(I_j)I_k^*}{f_j(I_j^*)I_k} - \frac{I_k}{I_k^*}\right)\right. \\
&\qquad \left. + \left(2 - \frac{S_k^*}{S_k} + \frac{f_{kj}(S_k^*, I_j, I)}{f_{kj}(S_k^*, I_j^*, I^*)} - \frac{f_{kj}(S_k, I_j, I)I_k^*}{f_{kj}(S_k^*, I_j^*, I^*)I_k} - \frac{I_k}{I_k^*}\right)\right].
\end{aligned}$$

Since

$$\frac{S_k^*}{S_k} + \frac{S_k}{S_k^*} - 2 \geq 0,$$

we have

$$-(d+\gamma)S_k^*\left(\frac{S_k^*}{S_k}+\frac{S_k}{S_k^*}-2\right)\le 0, \tag{3.23}$$

and the equality holds if and only if $S_k = S_k^*$.

Let $a_{kj} = \beta_{kj} f_{kj}(S_k^*, I_j^*, I^*)$, $G_k(I_k) = -\frac{I_k}{I_k^*} + \ln\frac{I_k}{I_k^*}$, $\hat{F}_{kj}(S_k, I_j) = 2 - \frac{S_k^*}{S_k} + \frac{f_j(I_j)S_k^*}{f_j(I_j^*)S_k} - \frac{f_j(I_j)I_k^*}{f_j(I_j^*)I_k} - \frac{I_k}{I_k^*}$, $\check{F}_{kj}(S_k, I_j, I) = 2 - \frac{S_k^*}{S_k} + \frac{f_{kj}(S_k^*, I_j, I)}{f_{kj}(S_k^*, I_j^*, I^*)} - \frac{f_{kj}(S_k, I_j, I)I_k^*}{f_{kj}(S_k^*, I_j^*, I^*)I_k} - \frac{I_k}{I_k^*}$, and we have

$$F_{kj}(S_k, I_j, I) = \frac{\alpha_{kj} f_j(I_j^*)}{\beta_{kj} f_{kj}(S_k^*, I_j^*, I^*)}\hat{F}_{kj}(S_k, I_j) + \check{F}_{kj}(S_k, I_j, I).$$

Then, by (3.23), we have

$$\frac{dV_k}{dt} \le \sum_{j=1}^{n} a_{kj} F_{kj}(S_k, I_j, I).$$

Let $\Phi(a) = 1 - a + \ln a$. Then $\Phi(a) \le 0$ for $a > 0$ and equality holds only at $a = 1$. Furthermore,

$$\begin{aligned}\hat{F}_{kj}(S_k, I_j) &= G_k(I_k) - G_j(I_j) + \Phi\left(\frac{S_k^*}{S_k}\right) + \Phi\left(\frac{I_j f_j(I_j^*)S_k}{I_j^* f_j(I_j)S_k^*}\right) + \Phi\left(\frac{f_j(I_j)I_k^*}{f_j(I_j^*)I_k}\right)\\ &\quad + \left(\frac{f_j(I_j)S_k^*}{f_j(I_j^*)S_k} - 1\right)\left(1 - \frac{I_j f_j(I_j^*)S_k}{I_j^* f_j(I_j)S_k^*}\right)\\ &\le G_k(I_k) - G_j(I_j) + \left(\frac{f_j(I_j)S_k^*}{f_j(I_j^*)S_k} - 1\right)\left(1 - \frac{I_j f_j(I_j^*)S_k}{I_j^* f_j(I_j)S_k^*}\right),\end{aligned}$$

and

$$\begin{aligned}\check{F}_{kj}(S_k, I_j, I) &= G_k(I_k) - G_j(I_j) + \Phi\left(\frac{S_k^*}{S_k}\right)\\ &\quad + \Phi\left(\frac{I_j f_{kj}(S_k^*, I_j^*, I^*)}{I_j^* f_{kj}(S_k^*, I_j, I)}\right) + \Phi\left(\frac{f_{kj}(S_k^*, I_j, I)I_k^*}{f_{kj}(S_k^*, I_j^*, I^*)I_k}\right)\\ &\quad + \left(\frac{f_{kj}(S_k^*, I_j, I)}{f_{kj}(S_k^*, I_j^*, I^*)} - 1\right)\left(1 - \frac{I_j f_{kj}(S_k^*, I_j^*, I^*)}{I_j^* f_{kj}(S_k^*, I_j, I)}\right)\\ &\le G_k(I_k) - G_j(I_j) + \left(\frac{f_{kj}(S_k^*, I_j, I)}{f_{kj}(S_k^*, I_j^*, I^*)} - 1\right)\\ &\quad \times \left(1 - \frac{I_j f_{kj}(S_k^*, I_j^*, I^*)}{I_j^* f_{kj}(S_k^*, I_j, I)}\right).\end{aligned}$$

Thus

$$F_{kj}(S_k, I_j, I) \le \left(\frac{\alpha_{kj} f_j(I_j^*)}{\beta_{kj} f_{kj}(S_k^*, I_j^*, I^*)} + 1\right)\big(G_k(I_k) - G_j(I_j)\big) + \frac{\alpha_{kj} f_j(I_j^*)}{\beta_{kj} f_{kj}(S_k^*, I_j^*, I^*)}\left(\frac{f_j(I_j)S_k^*}{f_j(I_j^*)S_k} - 1\right)\left(1 - \frac{I_j f_j(I_j^*)S_k}{I_j^* f_j(I_j)S_k^*}\right) + \left(\frac{f_{kj}(S_k^*, I_j, I)}{f_{kj}(S_k^*, I_j^*, I^*)} - 1\right)\left(1 - \frac{I_j f_{kj}(S_k^*, I_j^*, I^*)}{I_j^* f_{kj}(S_k^*, I_j, I)}\right).$$

Under condition (3.22), we can show that V_k, F_{kj}, G_k, a_{kj} satisfy the assumptions of Theorem 3.1 and Corollary 3.3 in Li and Shuai (2010). Therefore, the function $V = \sum_{k=1}^{n} c_k V_k$ as defined in Theorem 3.1 in Li and Shuai (2010) is a Lyapunov function for system (3.20), namely, $\frac{dV}{dt} \le 0$ for $(S_1, I_1, \ldots, S_n, I_n) \in \Gamma^\circ$. It can be verified similarly as in Section 4 of Li and Shuai (2010) that the only compact invariant set where $\frac{dV}{dt} = 0$ is the singleton $\{E^*\}$. By the LaSalle's Invariance Principle (LaSalle 1976), E^* is globally asymptotically stable in Γ°. This completes the proof of Theorem 3.5. □

4 The effects of local information of diseases on epidemic dynamics

In general, with the progress of epidemics, infected individuals are not evenly distributed, then individuals may be more concerned about quite a few seriously infected areas, and take corresponding actions to protect themselves based on the information of diseases in these areas (local information). Below we discuss the effects of local information of diseases, namely, the average densities of infected individuals in a local area, on epidemic dynamics.

When a disease occurs in a human population, if the disease spreads locally, then the vast majority of infected individuals with degree k are distributed in a serious disaster area, we use $l(k)$ to describe the nonuniform of the distribution of the infected individuals with degree k, which is associated with the degree k of infected individuals. The larger degree k of infected individuals, the larger value of $l(k)$, which implies that the nonuniform of the distribution of infected individuals with large degree is more obvious than the nonuniform of the distribution of infected individuals with small degree. If $l(k) > 1$, then the local area is a high incidence area; if $l(k) < 1$, then the local area is a low incidence area; if $l(k) = 1$, then the local area is a uniform incidence area. Similarly, we can also use $l = \sum_k l(k)P(k)$ to characterize the incidence degree of a local area.

Denote by U, U^*, D_k and $\breve{I}_k$ the set consisting of all individuals in the investigated area (universal set), the set consisting of all individuals in a heavily infected area (subset), the set consisting of individuals with degree k in the investigated area, and the set consisting of infected individuals with degree k in the investigated area, respectively. $P(U)$ represents the probability of $x \in U$ for a randomly chosen individual x; $P(D_k)$ represents the probability of $x \in D_k$ for a randomly chosen individual x; $P(\breve{I}_k)$ rep-

resents the probability of $x \in \breve{I}_k$ for a randomly chosen individual x; I_k represents the densities of infected individuals with degree k, namely, the probability of $x \in \breve{I}_k$ for a randomly chosen individual x. $P(U^*|D_k)$ represents the probability of $x \in U^*$ for a randomly chosen individual x in D_k, similarly, $P(U^*|\breve{I}_k)$ represents the probability of $x \in U^*$ for a randomly chosen individual x in $\breve{I}_k$. $P(D_k|U^*)$ represents the probability of $x \in D_k$ for a randomly chosen individual x in U^*; $P(\breve{I}_k|U^*)$ represents the probability of $x \in \breve{I}_k$ for a randomly chosen individual x in U^*.

According to the above formulas and symbols, we have $P(D_k) = P(k)$, $P(\breve{I}_k) = I_k$, $P(U) = 1$.

For an uncorrelated network, we assume that individuals with degree k are evenly distributed in U and the vast majority of infected individuals with degree k are distributed in U^*. Thus we have

$$P(U^*|D_k) = \frac{P(U^*)}{P(U)} = P(U^*),$$
$$P(U^*|\breve{I}_k) = l(k)\frac{P(U^*)}{P(U)} = l(k)P(U^*),$$

where $l(k) \geq 1$.

By using Bayes' formula, we have

$$P(D_k|U^*) = \frac{P(D_k)P(U^*|D_k)}{P(U^*)} = P(k),$$
$$P(\breve{I}_k|U^*) = \frac{P(\breve{I}_k)P(U^*|\breve{I}_k)}{P(U^*)} = l(k)I_k.$$

Then, the average density of infected individuals in a serious disaster area, i.e., the local information of diseases is

$$\tilde{I} = \sum_k P(D_k|U^*)P(\breve{I}_k|U^*) = \sum_k l(k)P(k)I_k.$$

In addition, the average density of infected individuals in the investigated area, i.e., the global information of diseases is

$$I = \sum_k P(D_k|U)P(\breve{I}_k|U) = \sum_k P(D_k)P(\breve{I}_k) = \sum_k P(k)I_k.$$

Since $l(k) \geq 1$, we have $\tilde{I} \geq I$. Now, we can replace I with $\tilde{I}$ in the adaptive weight function, so we can compute and analyze the basic reproduction numbers of models (2.6) and (2.8) with respect to the local information of diseases by a similar discussion as that in Sect. 3.2. Interestingly, we will find that the local information of diseases cannot change the basic reproduction numbers of models (2.6) and (2.8) with respect to the global information of diseases. However, the local information of diseases will influence more significantly the progress of both models (2.6) and (2.8) than the global information of diseases.

5 Immunization and treatment strategies

The appropriate immunization and treatment strategies are very important for the prevention and control of infectious diseases. In this section, we discuss the SIS model (2.6) on an adaptively weighted scale-free network with various immunization schemes (Fu et al. 2008) and treatment of infected individuals through contact tracing and isolation (Xiao et al. 2015).

5.1 Proportional immunization

Denote the immunization rate by δ, $0 < \delta < 1$, then system (2.1) becomes

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)] \sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - (1-\delta)kS_k(t)\Theta_k(t) + \gamma I_k(t), \\ \frac{dI_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)] \sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + (1-\delta)kS_k(t)\Theta_k(t) - (d+\gamma)I_k(t). \end{cases} \tag{5.1}$$

By a similar discussion as in Sect. 3.2, the basic reproduction number $\tilde{R}_0$ can be shown that

$$\tilde{R}_0^P = \frac{1}{d+\gamma} \max\{|\tilde{\rho}_{ip}|, i = 1, 2, \ldots, n\} = \frac{\tilde{\rho}_{2p}}{d+\gamma}, \tag{5.2}$$

where $\tilde{\rho}_{2p} = \frac{\langle X_k\rangle + (1-\delta)\langle \tilde{Y}_k\varphi(k)\rangle + \sqrt{(\langle X_k\rangle - (1-\delta)\langle \tilde{Y}_k\varphi(k)\rangle)^2 + 4(1-\delta)\langle X_k\varphi(k)\rangle\langle \tilde{Y}_k\rangle}}{2}$.

Note that in (5.2), if $\delta = 0$, which means no immunization scheme is implemented, then $\tilde{R}_0^P = \tilde{R}_0$; if $0 < \delta < 1$, then $\tilde{R}_0^P < \tilde{R}_0$, implying that the immunization scheme is more effective; while if $\delta = 1$, we obtain $\tilde{R}_0 = \frac{b(\langle k\rangle - \langle kN_k^*\rangle)}{(d+\gamma)\langle k\rangle}$. That is to say, in the case of a full immunization, the spread of epidemic diseases in the network is only contributed by congenital infection (from parents).

5.2 Targeted immunization

We can devise a targeted immunization scheme (Fu et al. 2008; Pastor-Satorras and Vespignani 2002). Introduce an upper threshold κ, such that all nodes with connectivity $k > \kappa$ are immunized, i.e., we defined the immunization rate δ_k by

$$\delta_k = \begin{cases} 1, & k \geq \kappa, \\ c, & k = \kappa, \\ 0, & k < \kappa, \end{cases} \tag{5.3}$$

where $0 < c \leq 1$, and $\sum_k \delta_k P(k) = \bar{\delta}$, where $\bar{\delta}$ is the average immunization rate.

The system (2.1) can be written as follows:

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)] \sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - (1-\delta_k)kS_k(t)\Theta_k(t) + \gamma I_k(t), \\ \frac{dI_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)] \sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + (1-\delta_k)kS_k(t)\Theta_k(t) - (d+\gamma)I_k(t). \end{cases} \tag{5.4}$$

By a similar discussion as in Sect. 3.2, the basic reproduction number $\tilde{R}_0$ can be shown that

$$\tilde{R}_0^T = \frac{1}{d+\gamma} \max\{|\tilde{\rho}_{it}|, i = 1, 2, \ldots, n\} = \frac{\tilde{\rho}_{2t}}{d+\gamma}, \tag{5.5}$$

where $\tilde{\rho}_{2t} = \frac{\langle X_k\rangle + \langle(1-\delta_k)\tilde{Y}_k\varphi(k)\rangle + \sqrt{(\langle X_k\rangle - \langle(1-\delta_k)\tilde{Y}_k\varphi(k)\rangle)^2 + 4\langle X_k\varphi(k)\rangle\langle(1-\delta_k)\tilde{Y}_k\rangle}}{2}$.

It is clear that $\tilde{R}_0^T < \tilde{R}_0$, for the convenience of comparison, we consider the case of infectivity function $\varphi(k) = w$. By (5.5), the basic reproduction number $\tilde{R}_0^T$ can be expressed as

$$\tilde{R}_0^{T\star} = \frac{\langle X_k\rangle}{d+\gamma} + \frac{w\left(\langle\tilde{Y}_k\rangle - \langle\delta_k\tilde{Y}_k\rangle\right)}{d+\gamma}.$$

Note that $\langle\delta_k\tilde{Y}_k\rangle = \bar{\delta}\langle\tilde{Y}_k\rangle + \delta'$, where $\delta' = \langle(\delta_k - \bar{\delta})(\tilde{Y}_k - \langle\tilde{Y}_k\rangle)\rangle$ is the covariance of δ_k and $\tilde{Y}_k$.

As discussed in a targeted immunization scheme (Fu et al. 2008), by similar argument we can obtain

$$\tilde{R}_0^{T\star} - \frac{\langle X_k\rangle}{d+\gamma} < \frac{1-\bar{\delta}}{1-\delta}\left(\tilde{R}_0^{P\star} - \frac{\langle X_k\rangle}{d+\gamma}\right).$$

If we set $\delta = \bar{\delta}$, then

$$\tilde{R}_0^{T\star} < \tilde{R}_0^{P\star},$$

which means that the targeted immunization scheme is more efficient than the proportional scheme for the same average immunization rate when $\varphi(k) = w$.

5.3 Acquaintance immunization

Choose a random fraction p of the N nodes, the probability that a particular node with k contacts is selected for immunization is $kP(k)/(N\langle k\rangle)$ (Fu et al. 2008; Dorogovtsev et al. 2008; Cohen et al. 2000), therefore $\delta_k = \frac{p}{\langle k\rangle}kP(k)$, so the basic reproduction number for this immunization scheme with $\varphi(k) = w$ is determined by

$$\begin{aligned}\tilde{R}_0^{Aq\star} - \frac{\langle X_k\rangle}{d+\gamma} &= w\left(\langle\tilde{Y}_k\rangle - \frac{p}{\langle k\rangle}\langle kP(k)\tilde{Y}_k\rangle\right)\\ &= \frac{\langle\tilde{Y}_k\rangle - \frac{p}{\langle k\rangle}\langle kP(k)\tilde{Y}_k\rangle}{(1-\bar{\delta})\langle\tilde{Y}_k\rangle - \delta'}\left(\tilde{R}_0^{T\star} - \frac{\langle X_k\rangle}{d+\gamma}\right).\end{aligned}$$

Note that

$$\begin{aligned}(1-\bar{\delta})\langle\tilde{Y}_k\rangle - \delta' &= (1-\bar{\delta})\langle\tilde{Y}_k\rangle - \langle(\delta_k - \bar{\delta})(\tilde{Y}_k - \langle\tilde{Y}_k\rangle)\rangle\\ &> (1-\bar{\delta})\langle\tilde{Y}_k\rangle - \langle(1-\bar{\delta})(\tilde{Y}_k - \langle\tilde{Y}_k\rangle)\rangle\end{aligned}$$

$$
\begin{aligned}
&= (1-\bar{\delta})\langle\tilde{Y}_k\rangle - [\langle(1-\bar{\delta})\tilde{Y}_k\rangle - \langle(1-\bar{\delta})\langle\tilde{Y}_k\rangle\rangle] \\
&> (1-\bar{\delta})\langle\tilde{Y}_k\rangle - \langle(1-\bar{\delta})\tilde{Y}_k\rangle \\
&= (1-\bar{\delta})\langle\tilde{Y}_k\rangle - (1-\bar{\delta})\langle\tilde{Y}_k\rangle \\
&= 0,
\end{aligned}
$$

and

$$
\langle\tilde{Y}_k\rangle - \frac{p}{\langle k\rangle}\langle kP(k)\tilde{Y}_k\rangle = \langle\tilde{Y}_k\rangle - \frac{cp}{\langle k\rangle}\langle k^{1-r}\tilde{Y}_k\rangle.
$$

If $c < \frac{\langle k\rangle}{p}$, then $\langle\tilde{Y}_k\rangle - \frac{p}{\langle k\rangle}\langle kP(k)\tilde{Y}_k\rangle > 0$. Thus we have $\tilde{R}_0^{Aq\star} - \frac{\langle X_k\rangle}{d+\gamma} = \Upsilon\left(\tilde{R}_0^{T\star} - \frac{\langle X_k\rangle}{d+\gamma}\right)$, where Υ is a positive constant. This means that the acquaintance immunization scheme is comparable to the targeted immunization scheme in effectiveness without congenital infection.

5.4 Active immunization

We now discuss a new immunization scheme proposed by Fu et al. (2008): choose an infected node and immunize its neighbors whose degree $k \geq \kappa$. The system (2.1) becomes:

$$
\begin{cases}
\frac{dS_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)]\sum_i \frac{P(i)}{\langle k\rangle} S_i(t) - dS_k(t) - kS_k(t)\Theta_k(t) + (\gamma + \bar{\delta}_k)I_k(t), \\
\frac{dI_k(t)}{dt} = bk[1 - S_k(t) - I_k(t)]\sum_i \frac{P(i)}{\langle k\rangle} I_i(t) + kS_k(t)\Theta_k(t) - (d + \gamma + \bar{\delta}_k)I_k(t),
\end{cases}
\tag{5.6}
$$

where $\bar{\delta}_k = \sum_k \frac{kP(k)}{\langle k\rangle}\delta_k$ and δ_k is defined in (5.3).

Similar to the discussion in Sect. 3.2, the basic reproduction number $\tilde{R}_0$ can be shown that

$$
\tilde{R}_0^{At} = \frac{1}{d+\gamma+\bar{\delta}_k}\max\{|\tilde{\rho}_i|, i = 1, 2, \ldots, n\} = \frac{\tilde{\rho}_2}{d+\gamma+\bar{\delta}_k}, \tag{5.7}
$$

where $\tilde{\rho}_2 = \frac{\langle X_k\rangle + \langle\tilde{Y}_k\varphi(k)\rangle + \sqrt{(\langle X_k\rangle - \langle\tilde{Y}_k\varphi(k)\rangle)^2 + 4\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle}}{2}$. Therefore, we have

$$
\tilde{R}_0^{At} = \frac{d+\gamma}{d+\gamma+\bar{\delta}_k}\tilde{R}_0 < \tilde{R}_0.
$$

That is to say, the immunization scheme we propose here is indeed effective. The lower κ is, the greater $\bar{\delta}_k$ is, and the more effective the immunization scheme is.

5.5 Treatment of infected individuals through contact tracing and isolation

We reclassify the usual susceptible (S_k) and infected (I_k) compartments of the SIS model (2.6) on an adaptively weighted scale-free network, to include the quarantined

susceptible (S_k^q) and isolated infected (I_k^q) compartments. We assume that the contact tracing rate is fixed (denoted by the constant c_T). In addition to the existing dynamical processes of the adaptively weighted model (2.6), with contact tracing, a proportion, q, of individuals with degree k who have had contact with isolated infected individuals is quarantined. The quarantined individuals can either move to compartment I_k^q or S_k^q, depending on whether they are infected or not. The non-isolated infected individuals can be detected and then isolated at a rate of δ_I. The quarantined susceptible individuals can be released into the wider community at a rate of δ_S. We denote by γ_q the recovery rate of isolated infected individuals. The SIS model (2.6) with contact tracing and isolation on an adaptively weighted scale-free network can be modified as

$$\begin{cases} \frac{dS_k(t)}{dt} = bk[1-N_k(t)-N_k^q(t)]\sum_i \frac{A}{i}P(i|k)S_i(t) - kS_k(t)\Theta_k(t) - qc_Tk S_k(t)\Psi_k(t) - dS_k(t) \\ \qquad +\delta_S S_k^q(t) + \gamma I_k(t) + \gamma_q I_k^q(t), \\ \frac{dI_k(t)}{dt} = bk[1-N_k(t)-N_k^q(t)]\sum_i \frac{A}{i}P(i|k)I_i(t) + kS_k(t)\Theta_k(t) - qc_Tk I_k(t)\Psi_k(t) \\ \qquad -(d+\gamma+\delta_I)I_k(t), \\ \frac{dS_k^q(t)}{dt} = bk[1-N_k(t)-N_k^q(t)]\sum_i \frac{A}{i}P(i|k)S_i^q(t) + qc_Tk S_k(t)\Psi_k(t) - (\delta_S+d)S_k^q(t), \\ \frac{dI_k^q(t)}{dt} = bk[1-N_k(t)-N_k^q(t)]\sum_i \frac{A}{i}P(i|k)I_i^q(t) + qc_Tk I_k(t)\Psi_k(t) + \delta_I I_k(t) - (d+\gamma_q)I_k^q(t), \end{cases} \tag{5.8}$$

where $N_k(t) = S_k(t)+I_k(t)$, $N_k^q(t) = S_k^q(t)+I_k^q(t)$, $\Theta_k(t) = \sum_i P(i|k)\frac{\varphi(i)}{i}\lambda_{ik}I_i(t)$, $\Psi_k(t) = \sum_i P(i|k)I_i^q(t)$.

The contact tracing model (5.8) includes adaptive weight and some interventions (such as quarantine, isolation and treatment), mainly focussing on the contact tracing and quarantine of individuals who have had contacts with isolated infected individuals. In contact tracing model (5.8), contact tracing is driven by the number of infected individuals in isolation. Further, we not only trace the non-isolated susceptible individuals that are in contact with isolated infected individuals, but also trace the non-isolated infected individuals who have contacts with isolated infected individuals. Meanwhile, the contact tracing function $\Psi_k(t)$ is included in the non-isolated susceptible population (S_k), non-isolated infected population (I_k), isolated susceptible population (S_k^q) and isolated infected population (I_k^q).

Similar to the analysis in Sect. 3.2, we calculate the basic reproduction number $\tilde{R}_0^C$ by using the next generation matrix as follows:

$$\tilde{R}_0^C = \max\left\{\frac{\tilde{\rho}_{2c}}{d+\gamma+\delta_I}, \frac{\langle X_k\rangle}{d+\delta_S}, \frac{\langle X_k\rangle}{d+\gamma_q}\right\}, \tag{5.9}$$

where $\tilde{\rho}_{2c} = \frac{\langle X_k\rangle+\langle\tilde{Y}_k\varphi(k)\rangle+\sqrt{(\langle X_k\rangle-\langle\tilde{Y}_k\varphi(k)\rangle)^2+4\langle X_k\varphi(k)\rangle\langle\tilde{Y}_k\rangle}}{2}$.

Note that the basic reproduction number $\tilde{R}_0^C$ of the model (5.8) is the same as $\tilde{R}_0$ in the absence of contact tracing, quarantine, isolation and treatment. With contact tracing, if $\delta_I = 0$, then we have $\tilde{R}_0^C \geq \frac{\tilde{\rho}_{2c}}{d+\gamma} = \tilde{R}_0$, implying that treatment of infected individuals through contact tracing and isolation is indeed more effective.

6 Numerical simulations

We perform some numerical simulations to complement the theoretical results, and to understand the effects of parameters on the spread of epidemics, so as to find better control strategies. Here, we mainly use models (2.6) and (2.8) to simulate the evolutive process of epidemics.

We consider a BA (Barabási-Albert) scale-free (preferential attachment) network (Barabási and Albert 1999) with the degree distribution $P(k) \sim k^{-3}$ and the network size $N = 500$. This network evolves from initial network with size $m_0 = 3$ and we add each new node with $m = 3$ new edges. The average degree of the generation network is $\langle k \rangle = 6$ and its maximum degree is $n = 40$. Initially, each node and each edge is assigned a weight according to the weight function $g(k)$. As the disease progresses, the weights of the nodes and edges are updated based on the weight function $\tilde{g}(k, t)$. Let $\varphi(k) = k^{\alpha}$, $g(k) = k^{\beta}$, $h(k) = k^{\mu}$ and $m(k) = k^{\sigma}$, where α, β, μ and σ are all positive constants. It should be noted that $\beta = 0$ means no weight, $\beta \neq 0$, $\mu = 0$ and $\sigma = 0$ mean fixed weights, and $\beta \neq 0$, $\mu \neq 0$ and $\sigma \neq 0$ mean adaptive weights.

Firstly, we show the comparison of the mean-field approach and Monte Carlo stochastic simulations for the prediction of the average fraction of infected individuals at time t, $I(t)$, in model (2.6), see Fig. 1. This SIS dynamic process on adaptively weighted networks proceeds with parallel updating. To minimise random fluctuation caused by the initial conditions, we make average of $I(t)$ over 20 realizations at each time step for different initial infectious nodes. From Fig. 1, we can observe that the agreement between the numerical results from the two approaches is very good and the average accuracy of the mean-field approach is within 3 %, which implies that the analysis based on the mean-field approach is very effective. Thus, in the figures below, our numerical results are mainly obtained from the mean-field approach.

Let $\gamma = 0.2$ in Figs. 2, 3, 4 and 5 and other parameters α, b, d are set as $\alpha = 1$, $b = 0.2$ and $d = 0.1$ in Figs. 3, 4 and 5. All the following simulations (except Fig. 2a) are based on the generated BA network with the power exponent $r = 3$.

Fig. 2(a) illustrates that the basic reproduction number $\tilde{R}_0(\bar{R}_0)$ on the adaptively weighted network is lower than R_0 on the fixedly weighted network, but $\tilde{R}_0(\bar{R}_0)$ and R_0 increase as the weight exponent β increases and decrease as the power-law exponent r increases. Since r indicates the heterogeneity of the network, the more heterogeneous the network is, the larger the basic reproduction numbers $\tilde{R}_0(\bar{R}_0)$ and R_0 are.

From Fig. 2b, with the increase of the infectivity exponent α and weight exponent β, the basic reproduction numbers $\tilde{R}_0(\bar{R}_0)$ and R_0 also increase, but the infectivity exponent α contributes much more impacts on them.

From Fig. 3, we can observe that the initial conditions have almost no influence on the stationary fraction of infected individuals. If $R_0, \tilde{R}_0, \bar{R}_0 < 1$, no matter how many infected individuals initially exist, the disease eventually dies out quickly, which implies that the disease-free equilibrium is globally asymptotically stable; If $R_0, \tilde{R}_0, \bar{R}_0 > 1$, the disease persists on a unique positive state, which implies that there is a stable endemic equilibrium. Moreover, the fixed weight makes the disease rapidly reach a peak of outbreak, while the adaptive weight and time delay cause the disease to rapidly drop and then experience a valley and a low peak, the greater the

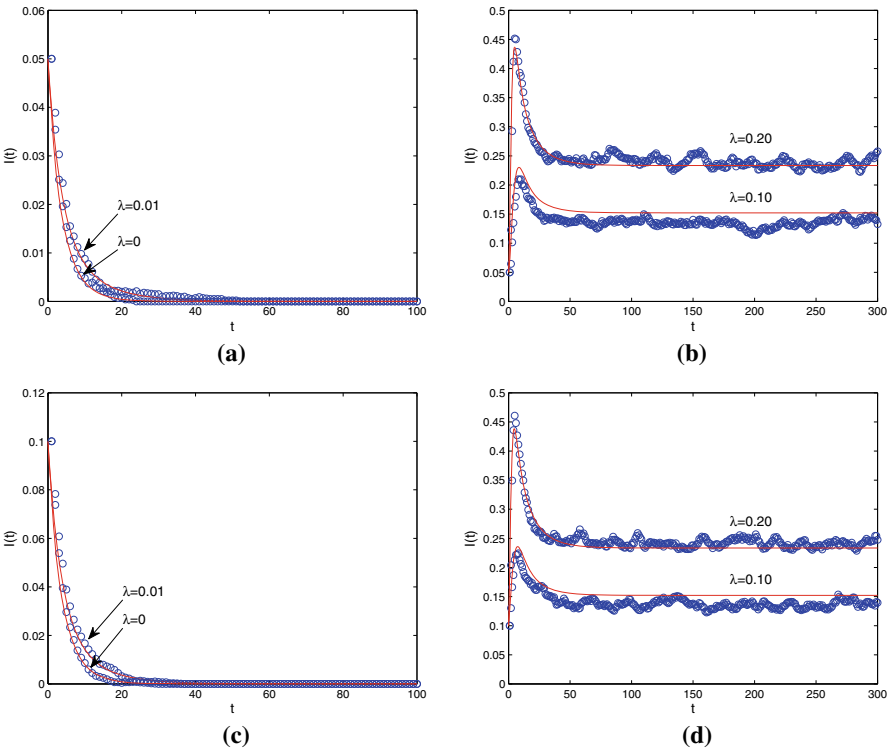

Fig. 1 (Color online) The comparison of Monte Carlo stochastic simulations (*blue circle*) and the mean-field approach (*red line*) for adaptively weighted model (2.6). The parameter values are set as follows: $\alpha = 1, \beta = 1, \mu = 1, \sigma = 0.06, b = 0.2, d = 0.1, \gamma = 0.2$. **a**, **c** The initial fraction of infected nodes is set 0.05, $\lambda = 0$ ($\tilde{R}_0 = 0.33$), $\lambda = 0.01$ ($\tilde{R}_0 = 0.41$); **b**, **d** the initial fraction of infected nodes is set 0.10, $\lambda = 0.10$ ($\tilde{R}_0 = 2.57$), $\lambda = 0.20$ ($\tilde{R}_0 = 5.08$). The Monte Carlo stochastic simulations and the mean-field approach are averaged by 20 realizations

degree of nodes is, the more pronounced this phenomenon becomes. With respect to the adaptive weight, the adaptive weight with time delay obviously reduces the outbreak scale of the disease, but will not change the final steady state.

From Fig. 4a, b, we can see that I_k with larger degree k experience several oscillations before they reach the steady levels. In the case of the larger adaptivity exponent μ, I_k with larger degree k does not mean that they can reach higher endemic levels. The larger the adaptivity exponent μ is, the lower steady levels I_k with larger degree k reach. Moreover, the larger adaptivity exponent μ effectively suppresses the outbreak of the disease. From Fig. 4a, c, d, the greater the inertia exponent σ is, the lower the greatest outbreak scale that I_k with larger degree k reach is. In addition, the greater inertia exponent σ makes the basic reproduction number $\tilde{R}_0$ become smaller and I_k with larger degree k reach the lower steady levels. From Fig. 4e, f, time delay will put off the outbreak of the disease, but will not change the steady levels I_k reach. The greater the time delay τ, the lower the valleys I_k with larger degree k experience.

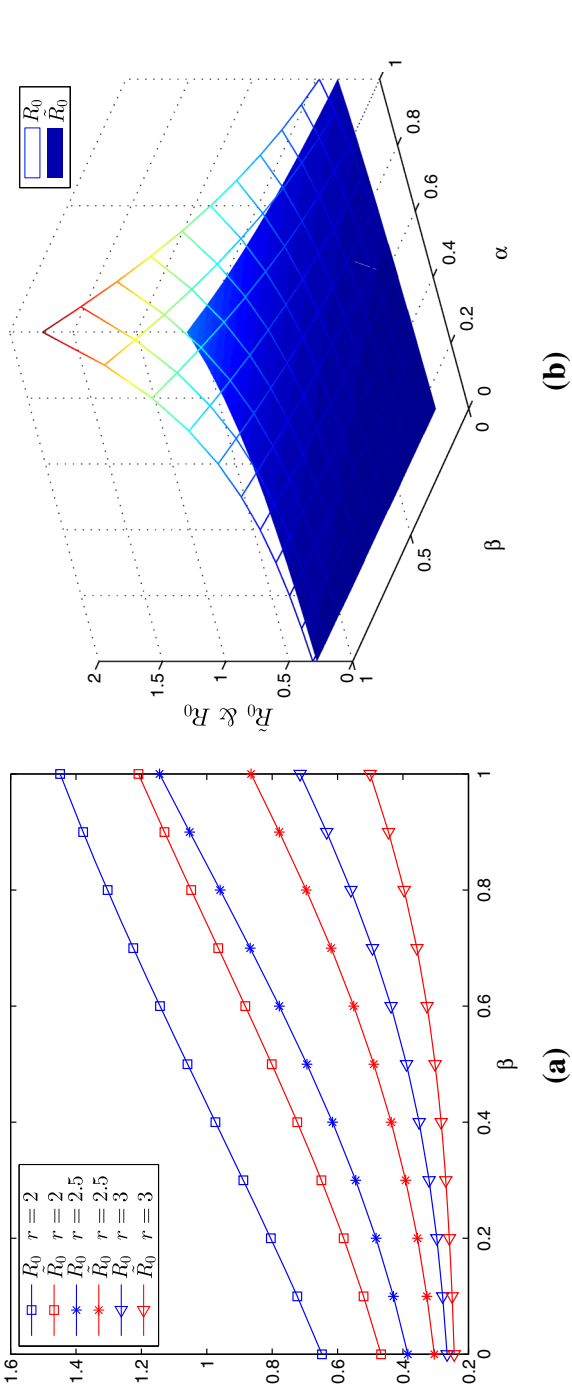

Fig. 2 (Color online) The combined influence of parameters on $\tilde{R}_0$ and R_0: $b = 0.08$, $d = 0.04$. **a** $\tilde{R}_0$ and R_0 in terms of the weight exponent β and power-law exponent r: $\lambda = 0.02$, $\alpha = 1$, $\sigma = 0.2$. **b** $\tilde{R}_0$ and R_0 in terms of the infectivity exponent α and weight exponent β: $\lambda = 0.05$, $r = 3$, $\sigma = 0.4$

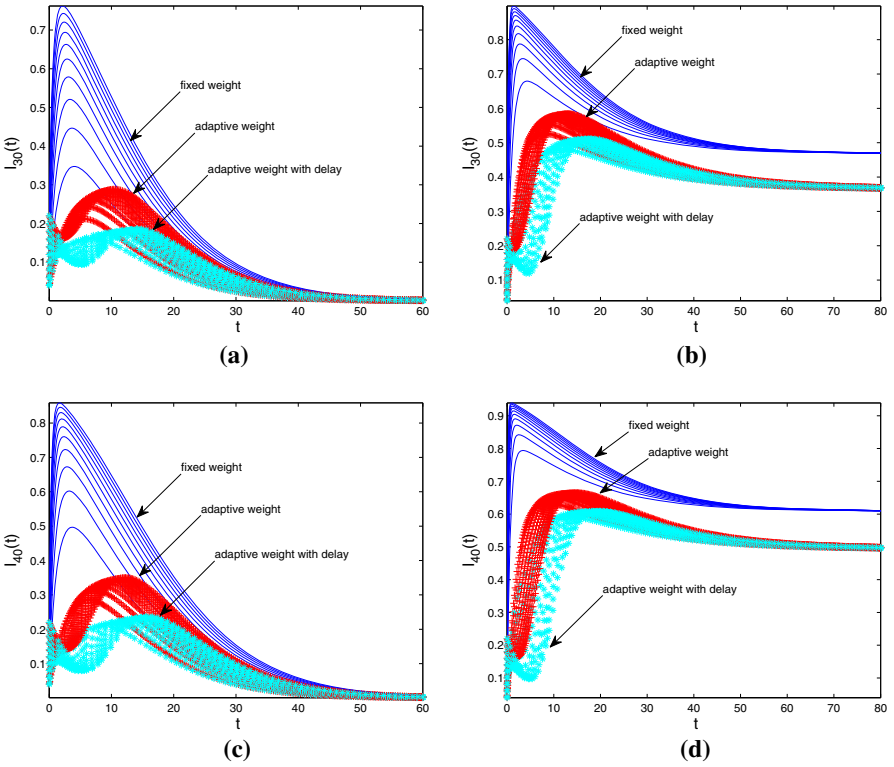

Fig. 3 (Color online) The influence of initial conditions on the density of infected individuals in models (2.4), (2.6) and (2.8), $\beta = 1, \mu = 1, \sigma = 0.06$. **a**, **c** $\lambda = 0.02$, $R_0 = 0.63$, $\tilde{R}_0 = \bar{R}_0 = 0.59$; **b**, **d** $\lambda = 0.05$, $R_0 = 1.43$, $\tilde{R}_0 = \bar{R}_0 = 1.32$

From Fig. 5a, we notice that the fixed weight triggers the outbreak of the disease, the larger adaptivity exponent μ and inertia exponent σ make the average density of infected individuals I reach lower steady levels. That is to say, the adaptivity of weights and behavior inertia can suppress epidemic diseases globally to a low level, which is more effective if the adaptivity exponent μ and inertia exponent σ are bigger. In some cases ($\beta = 1, \mu = 1, \sigma = 0.5$), the adaptivity of weights and behavior inertia can even prevent the disease from growing into an endemic. From Fig. 5b, time delay will make the average density I of infected individuals experience several oscillations before they reach the steady levels, which is more obvious if τ is larger, and put off the time the average density I of infected individuals reach common steady levels.

7 Discussions and conclusions

On social networks, it has been observed that when individuals realize that a contagious disease is in their proximity, they may take adaptive measures to reduce the contact weights (i.e., the intimacy or familiarity between neighbors). The adaptivity of weight signifies that individuals will change their social behavior as the disease develops. As

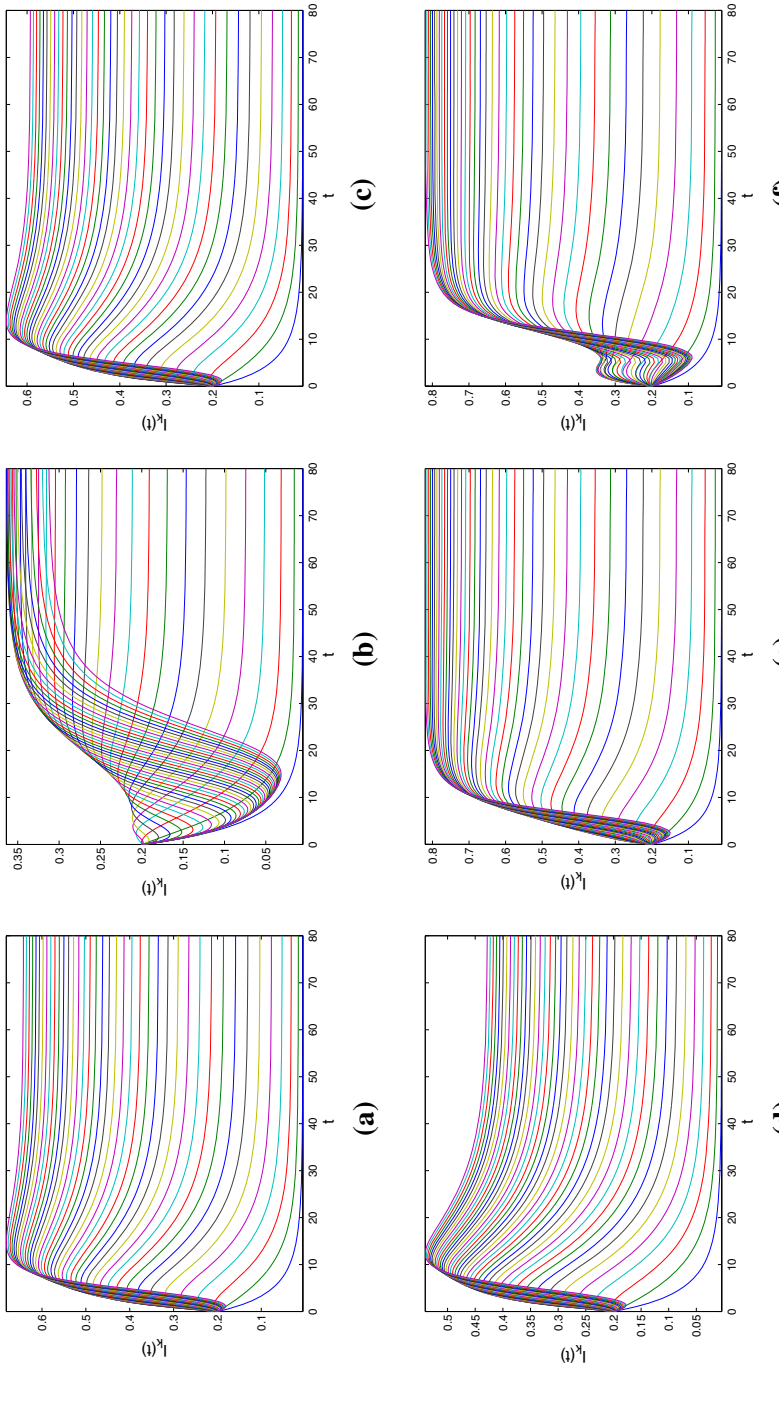

Fig. 4 (Color online) The densities of infected individuals with different degrees in models (2.6) and (2.8). The *lines from bottom to top* are $I_1(t), I_2(t), \ldots, I_{40}(t)$, respectively, $\lambda = 0.1$, $\beta = 1$. **a** $\mu = 1$, $\sigma = 0.06$, $\tilde{R}_0 = 2.57$; **b** $\mu = 1.5$, $\sigma = 0.06$, $\tilde{R}_0 = 2.57$; **c** $\mu = 1$, $\sigma = 0.1$, $\tilde{R}_0 = 2.40$; **d** $\mu = 1$, $\sigma = 0.2$, $\tilde{R}_0 = 1.91$; **e** $\mu = 1$, $\sigma = 0.06$, $\tau = 1$, $\tilde{R}_0 = 2.57$; **f** $\mu = 1$, $\sigma = 0.06$, $\tau = 5$, $\tilde{R}_0 = 2.57$

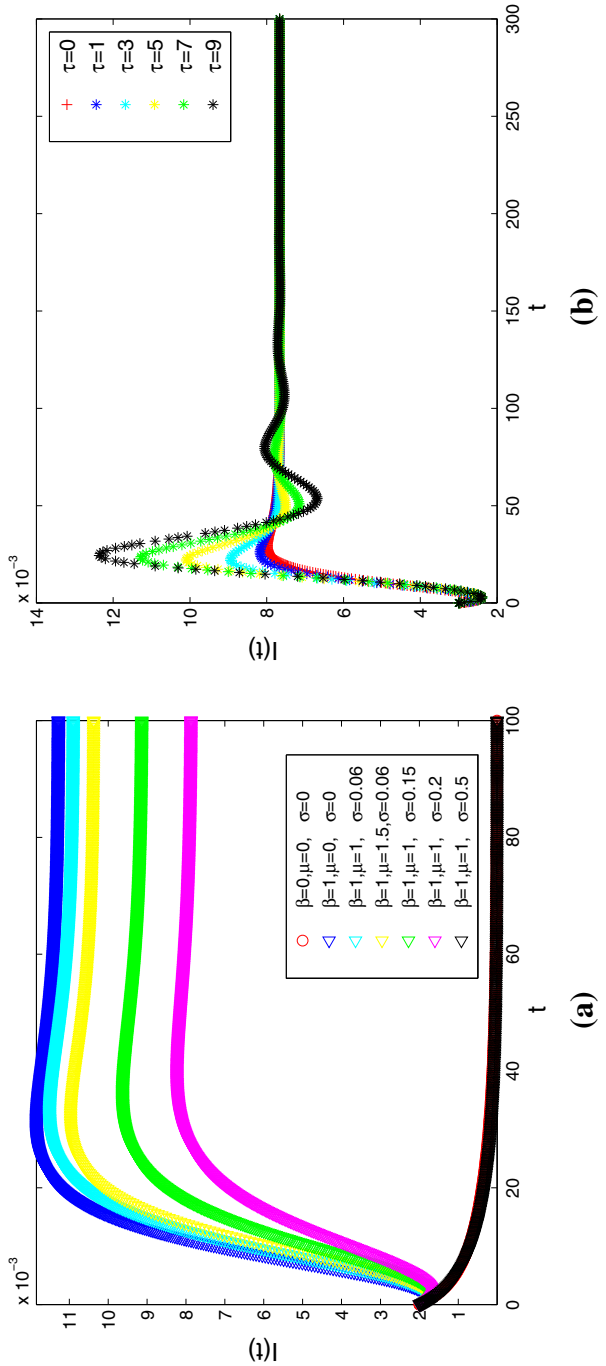

Fig. 5 (Color online) The influence of the adaptive weight, behavior inertia and time delay on the average density of infected individuals, $\lambda = 0.1$. **a** The influence of the adaptive weight and inertia on the average density of infected individuals. **b** The influence of time delay on the average density of infected individuals: $\beta = 1, \mu = 1.9, \sigma = 0.01, \bar{K}_0 = 2.76$

social network structure plays an important role in the process of epidemic spreading, the adaptivity in human behavior should be taken into account when modelling disease progression.

In this paper, we have developed and investigated the modified SIS models with variable population size, nonlinear infectivity and time delay on adaptively weighted scale-free networks. Our models suggest how the presence of a contagious disease induces a change in individuals behavior on weighted networks, and our results show how this could feed back to alter the disease dynamics. Through mean-field analysis, we find that the basic reproduction number is the threshold determining whether the epidemic spreads, which depends on birth rate b, death rate d (natural death), transmission rate λ, cure rate γ, the infectivity function $\varphi(k)$, the weight function $g(k)$, behavior-inertia function $m(k)$, and the structure of the network. Furthermore, compared with the weight function $g(k)$ and behavior-inertia function $m(k)$, the infectivity function $\varphi(k)$ has a dominant impact on the epidemic threshold, which means that controlling the contacts of individuals is more important than the infection ability of the disease itself. We compare the basic reproduction numbers of constantly weighted model, adaptively weighted model and adaptively weighted model with time delay, and find that the adaptivity of weight can reduce the epidemic threshold, but a time-delayed adaptivity of weight cannot change the epidemic threshold with respect to a non-delayed adaptivity of weight.

By constructing suitable Lyapunov functions, we obtain the global stability of the disease-free and the endemic equilibria for the adaptively weighted model. If $\tilde{R}_0 \leq 1$, the disease-free equilibrium E_0 is globally asymptotically stable, which implies that the disease will dies out. If $\tilde{R}_0 > 1$ and condition (3.22) holds, the endemic equilibrium E^* is globally asymptotically stable, therefore the disease persists. From numerical simulations, we can also obtain the global stability of the disease-free and the endemic equilibria for the adaptively weighted model with time delay. Compared with constantly weighted model, we can observe that the adaptivity of weight can induce the disease to decay quickly; particularly, strong adaptivity can suppress the epidemic globally to a low level. The time-delayed adaptivity of weight can put off and weaken the outbreak of the disease to a certain extent, but it cannot change the steady level of the epidemic. Therefore, in disease propagation, individual's adaptive behavior can quickly reduce the incidence, but in order to eliminate the disease, one should weaken the intensity of interaction, especially decrease the frequency of interaction. The adaptive behavior of individuals is beneficial, as it delays outbreak peaks and thus provides a critical response time needed for vaccine production and facility preparation.

The adaptivity of human behavior not only lowers the incidence of a disease, but also in some cases prevents the disease from growing into an endemic. If the adaptive behavior of individuals is not triggered by global information, but instead based on local information that is from some severely affected areas, we will find that the local information of diseases cannot change the basic reproduction numbers of models (2.6) and (2.8) with respect to the global information of diseases. Beyond a critical infection rate, the spread of local information can more significantly slow down the spread of a disease and lower the final incidence than

global information, but it also cannot completely stop it from reaching epidemic proportions.

Moreover, we have also discussed proportional, targeted, acquaintance, and active immunization schemes for the adaptively weighted model. By comparing the thresholds for different immunization schemes, we concluded that the targeted immunization scheme is more efficient than the proportional scheme for the same average immunization rate when $\varphi(k) = w$; the acquaintance immunization scheme is comparable to the targeted immunization scheme in effectiveness without congenital infection; and the effectiveness of the active immunization scheme is also discussed. In Pastor-Satorras and Vespignani (2002), a probability approach is used to calculate epidemic thresholds for random, targeted, and acquaintance immunization schemes, which are critical probability values and can be used to evaluate the fraction of immunized individuals. Here, we use the method proposed in Fu et al. (2008) to give a direct characterization of epidemic thresholds for more immunization schemes, including active immunization, so the thresholds are easier to apply practically. Furthermore, the most effective method is to decrease the infected rate while increasing the cure rate. In order to provide a complete picture of intervention strategies, we consider treatment of infected individuals through contact tracing and isolation, which is a key control measure in the battle against infectious diseases. By applying this strategy, a major outbreak can be significantly reduced or even eliminated at a small additional cost.

Our results are based only on the assumptions that the network structure is fixed and the weights are built on the connected nodes. Only if the disease is easily recognized and information spreads rapidly, while at the same time there is a strong tendency toward protective behavior, adaptive reactions of individuals to a disease can bring the infection rate of a disease down significantly. We do not analyze a specific disease in our model. For a specific disease, there is a great need to analyze the disease through modeling and comparing the epidemic model with real data. We expect that our analysis and simulations presented here may provide some insight into the studying of epidemic dynamics or other related diffusion processes.

It would be interesting to further consider adaptive dynamical behavior on a time-varying network and the corresponding model with double delays. Some further research is necessary and interesting in this field, such as the global asymptotic stability of the disease-free and endemic equilibria for delayed systems. We hope to tackle these questions in the future.

Acknowledgements This research was jointly supported by the NSFC Grants under Grant Nos. 11331009, 11572181 and 61473001, Guangxi Natural Science Foundation Program (2014GXNSFBA118016) and the fund of Education Department of Guangxi (YB2014122). The authors would also like to thank the anonymous referees for their invaluable comments and suggestions.

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