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Spreading dynamics of a SIQRS epidemic model on scale-free networks

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ABSTRACT

In order to investigate the influence of heterogeneity of the underlying networks and quarantine strategy on epidemic spreading, a SIQRS epidemic model on the scale-free networks is presented. Using the mean field theory the spreading dynamics of the virus is analyzed. The spreading critical threshold and equilibria are derived. Theoretical results indicate that the critical threshold value is significantly dependent on the topology of the underlying networks and quarantine rate. The existence of equilibria is determined by threshold value. The stability of disease-free equilibrium and the permanence of the disease are proved. Numerical simulations confirmed the analytical results.

1. Introduction

Human beings are still at the risk of the outbreak of infectious diseases now even after the development of modern medicine and the appearance of antibiotics [1–4]. What's worse, more and more frequent contacts among people, owing to the global transportation network, make the new emerging outbreak spread worldwide rapidly before adequate supplies of vaccine could be manufactured and distributed [5]. Therefore, the study of infection dynamics is of dramatic significance, which not only plays an important role in planning, implementing, evaluating various prevention and control programs but also contributes to identifying the crucial data that should be collected in order to make forecasts and estimate the uncertainty in forecasts [6,7]. The majority of classical mathematical models in epidemiology are based on a compartmentalization of individuals according to their disease status, which generates two standard models: the SIR (susceptible-infected-recovered) and the SIS (susceptible-infected-susceptible). The SIR model is appropriate for the diseases that lead to permanent immunity and people are never infected by that disease again, such as the parotitis, measles and SARS, etc. The SIS model is different from the SIR model in that the infected nodes will not obtain lifelong immunity and can return to the susceptible state after recovering, such as the influenza and gonorrhea. Early classical representations of infectious disease dynamics assumed that the population was large and homogeneously mixed such that deterministic equations with simple frequency-dependent transmission were appropriate. These simple models were subsequently extended in ways towards making them more realistic. Some such extensions were for example to allow for spatial structure that the topology could be constrained by the geographical embedding [8,9].

Another generalization of the initial simple deterministic epidemic model is to incorporate the complex topology of social interactions. As is shown in the field of complex networks, the scale-free property, i.e., the degree distribution follows a

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power-law form, is an essential discovery in social networks [10,11]. For further understanding of epidemic behaviors in real world, many epidemic models have been generalized to adopt the scale-free property of social networks. In networks, vertices represent individual people and edges represent the relationship of interest. As for epidemic behavior, that relationship is contact to transmit the disease. Recently, starting with the works by Pastor-Satorras and Vespignani [12,13], there has been a burst of activity on investigating the impacts of the network topology on disease spreading. The SIS epidemic spreading on scale-free networks with degree correlations has also been considered [14,15]. Barthelemy et al. observed that epidemic propagation follows a precise hierarchical dynamics in scale-free networks [16]. Loecher and Kadtke also confirmed and expanded upon the details of the hierarchical propagation leading to a considerably enhanced predictability for the order of infected nodes [17].

Additionally, some researchers, with the ultimate goal to contain infection, try to figure out the optimal control strategy [18–20]. One intervention procedure to control the spread of infectious diseases is quarantining the symptomatic individuals, which has been used for preventing human disease, such as smallpox and tuberculosis, as well as animal diseases [21–23]. However, the effects of quarantine on infectious diseases on scale-free network have not been well considered. In this paper, we focus on the (SIQRS) (susceptible-infected-quarantined-recovered-susceptible) epidemic model on contact networks that takes into account the heterogeneity due to the connectivity distribution. In the (SIQRS) epidemic model, infected individuals may be quarantined and develop transient immunity following infection and the recovered individuals, with the immunity waning, eventually return to full susceptibility to the disease. One purpose of this paper is to determine the threshold that governs whether or not an epidemic with few initial infective individuals can become established and the proportion of the population who are ultimately infected by such an epidemic. As the analytical and numerical results show, the network topology plays a significant role in the epidemic threshold and the endemic size of infection diminishes with the increase of quarantined proportion.

This article is organized as follows. The establishment of scale-free network model is described in Section 2. In Section 3, the epidemic threshold and the size of endemic prevalence are obtained. Simulation results of the proposed model are shown in Section 4. Finally, we conclude the paper in Section 5.

2. Network model

In this paper, we focus particularly on the Barabási and Albert (BA) model, incorporating two ingredients, namely, growth and preferential attachment. Starting with a small number (m_0) of fully connected vertices, every time step a new vertex is added, with m edges that are connected to an old vertex i with probability $\prod_{k_i} = k_i/\sum_j k_j$, where k_i is the connectivity of the ith vertex. After a long period of evolution, this network organizes itself into a scale-free stationary state, in which the probability that a vertex has kedges following a power law with an exponent, i.e., $P(k) = 2m^2k^{-3}$, $k \geqslant m$. Besides, the mean number and the variance of individual connectivity are $\langle k\rangle = \int_m^\infty kP(k)dk = 2m$, $\langle k^2\rangle = +\infty$, respectively (in the present work we will consider the parameters N = 10,000, m_0 = 3 and m = 3).

3. SIQRS epidemic model on the scale-free networks

3.1. Model formulation

One of the most effective interventions to contain the spread of infectious diseases is to isolate some infectors, in order to reduce infection transmission. In order to investigate the efficiency of quarantine policy, we consider the (SIQRS) model on BA network. The epidemic model has the flow diagram given in Fig. 1 with the following assumptions. Every vertex of the network can only exist in one of the four discrete states, namely, susceptible, infected, quarantined and removed, denoted respectively by S, I, Q, and R, and each link is a connection along which the infection can spread. In the course of disease transmission, a susceptible individual is infected with probability β if it is connected to an infected individual. Susceptible individuals are vaccinated with probability α. Infective individuals recover spontaneously with probability γ or are quarantined with probability σ. Some useful management or antiviral treatment are employed on the quarantined individuals so that they are supposed to leave for the removed state R, in which individuals have temporary immunity, with rate constant ε. However, some removed individuals, due to loss of immunization, join the susceptible individuals again, i.e., moving back to susceptible state, with probability δ.

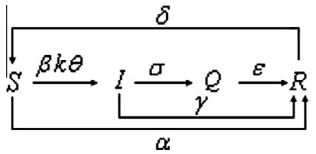

Fig. 1. The flow diagram of the SIQRS model.

For the (SIQRS) model on scale-free networks, taking into account the heterogeneity induced by the presence of vertices with different connectivities, let $S_k(t)$, $I_k(t)$, $Q_k(t)$, $R_k(t)$ be the densities of susceptible, infected, quarantined and removed vertices of connectivity k at time t, respectively. Thus, the dynamic mean-field reaction rate equations can be written as

$$\begin{cases} \frac{dS_k(t)}{dt} = -\beta k S_k(t)\theta(t) - \alpha S_k(t) + \delta R_k(t) \\ \frac{dI_k(t)}{dt} = \beta k S_k(t)\theta(t) - \gamma I_k(t) - \sigma I_k(t) \\ \frac{dQ_k(t)}{dt} = \sigma I_k(t) - \varepsilon Q_k(t) \\ \frac{dR_k(t)}{dt} = \gamma I_k(t) + \varepsilon Q_k(t) + \alpha S_k(t) - \delta R_k(t) \end{cases} \tag{1}$$

The probability $\theta(t)$ describes a link pointing to an infected individual, which satisfies the relation

$$\theta(t) = \frac{\sum_k kP(k)I_k(t)}{\sum_s sP(s)} = \frac{1}{\langle k\rangle}\sum_k kP(k)I_k(t) \tag{2}$$

where $\langle k\rangle$ is the average degree within the network and denotes the normalization factor. And $I(t) = \sum_k P(k)I_k(t)$ is the density of infected individuals in the whole network, $P(k)$ is the connectivity distribution.

3.2. Stability analysis

In this section, we present an analytic solution to the deterministic equations describing the dynamic of the (SIQRS) epidemic spreading process.

Theorem 1. Let $\rho = \frac{\delta\beta}{(\alpha+\delta)(\gamma+\sigma)}\frac{\langle k^2\rangle}{\langle k\rangle}$. There always exists a disease-free equilibrium $E_0(\delta/\delta+\alpha, 0, 0, \alpha/\delta+\alpha)$ and when $\rho > 1$, then system (1) has a unique endemic equilibrium $E_+(S_k^\infty, I_k^\infty, Q_k^\infty, R_k^\infty)$.

Proof. To get the equilibrium solution $E_+(S_k^\infty, I_k^\infty, Q_k^\infty, R_k^\infty)$, we need to make the right side of system (1) equal to zero. Then the equilibrium $E_+(S_k^\infty, I_k^\infty, Q_k^\infty, R_k^\infty)$ should satisfy

$$\begin{cases} -\beta k S_k^\infty \theta^\infty - \alpha S_k^\infty + \delta R_k^\infty = 0 \\ \beta k S_k^\infty \theta^\infty - \gamma I_k^\infty - \sigma I_k^\infty = 0z \\ \sigma I_k^\infty - \varepsilon Q_k^\infty = 0 \\ \gamma I_k^\infty + \varepsilon Q_k^\infty + \alpha S_k^\infty - \delta R_k^\infty = 0 \end{cases}$$

where $\theta^\infty = \frac{1}{\langle k\rangle}\sum_k kP(k)I_k^\infty$. One has

$$\begin{cases} S_k^\infty = \frac{(\gamma+\sigma)}{\beta k\theta^\infty} I_k^\infty \\ Q_k^\infty = \frac{\sigma}{\varepsilon} I_k^\infty \\ R_k^\infty = \frac{(\beta k\theta^\infty+\alpha)(\gamma+\sigma)}{\delta\beta k\theta^\infty} I_k^\infty \end{cases}. \tag{3}$$

According to the following normalization condition for all k:

$$S_k^\infty + I_k^\infty + Q_k^\infty + R_k^\infty = 1,$$

we can obtain

$$I_k^\infty = \frac{\beta k\delta\theta^\infty}{\beta k\theta^\infty[\delta(1+\sigma/\sigma\varepsilon\varepsilon) + (\gamma+\sigma)] + (\gamma+\sigma)(\alpha+\delta)}. \tag{4}$$

Inserting Eq. (4) into Eq. (2), we obtain the following equation

$$\theta^\infty = \frac{\sum_k kP(k)I_k^\infty}{k} = \frac{\sum_k k^2P(k)}{k}\frac{\beta\delta\theta^\infty}{\beta k\theta^\infty[\delta(1+\sigma/\varepsilon) + (\gamma+\sigma)] + (\gamma+\sigma)(\alpha+\delta)} \triangleq f(\theta^\infty). \tag{5}$$

Obviously, $\theta^\infty = 0$ is a solution of Eq. (5), i.e., $f(\theta^\infty) = 0$. To ensure Eq. (5) have a nontrivial solution, i.e. $0 < \theta^\infty \leqslant 1$, the following conditions must be satisfied

$$\left.\frac{df(\theta^\infty)}{d\theta^\infty}\right|_{\theta^\infty=0} > 1 \text{ and } f(1) \leqslant 1.$$

So, we have

$$\frac{\delta\beta}{(\alpha+\delta)(\gamma+\sigma)}\frac{\langle k^2\rangle}{\langle k\rangle} > 1.$$

Let $\rho = \frac{\delta\beta}{(\alpha+\delta)(\gamma+\sigma)}\frac{\langle k^2\rangle}{\langle k\rangle}$, a nontrivial solution exists if and only if $\rho > 1$.

The epidemic threshold can be defined by

$$\beta_c = \left(1+\frac{\alpha}{\delta}\right)(\gamma+\sigma)\frac{\langle k\rangle}{\langle k^2\rangle} \tag{6}$$

Namely, the epidemic incidence will die out if $\beta < \beta_c$, and it will break out if $\beta > \beta_c$.

Substitute the nontrivial solution of (5) into (4), we can get I_k^∞. By (3) and (4), we can easily obtain

$$0 < S_k^\infty < 1, \quad 0 < I_k^\infty < 1, \quad 0 < Q_k^\infty < 1, \quad 0 < R_k^\infty < 1.$$

Therefore, the equilibrium $E_+(S_k^\infty, I_k^\infty, Q_k^\infty, R_k^\infty)$ is well-defined. Hence, when $\rho > 1$, one and only one endemic equilibrium $E_+(S_k^\infty, I_k^\infty, Q_k^\infty, R_k^\infty)$ of system (1) exists. This completes the proof.

Now, consider a BA model. As a matter of fact, the mean number of individual degree and the degree distribution of the BA network satisfy

$$\begin{cases} \langle k\rangle = \int_m^\infty kP(k) = 2m \\ P(k) = \frac{2m^2}{k^3} \end{cases}$$

where m is the minimum individual degree in the network. Inserting this into Eq. (5), the following can be obtained by integrating with respect to the connectivity degree k:

$$\frac{1}{m} = \int_m^\infty \frac{\beta\delta}{k}\{\beta k\theta^\infty[\delta(1+\sigma/\varepsilon)+(\gamma+\sigma)]+(\gamma+\sigma)(\alpha+\delta)\}dk$$

which yields

$$\theta^\infty = \frac{(\gamma+\sigma)(\alpha+\delta)}{m\beta[\delta(1+\sigma/\varepsilon)+(\gamma+\sigma)]\left(e^{\frac{(\gamma+\sigma)(\alpha+\delta)}{m\beta\delta}}-1\right)} \tag{7}$$

By Eq. (4) and Eq. (7), we obtain the density of infected individuals in the steady (epidemic) state of the whole network.

$$I^\infty = \sum_k P(k)I_k^\infty = \frac{2\left[m\beta\delta\left(e^{\frac{(\gamma+\sigma)(\alpha+\delta)}{m\beta\delta}}-1\right)-(\gamma+\sigma)(\alpha+\delta)\right]}{m\beta\left(e^{\frac{(\gamma+\sigma)(\alpha+\delta)}{m\beta\delta}}-1\right)^2[\delta(1+\sigma/\varepsilon)+(\gamma+\sigma)]} \qquad \square$$

Remark. The epidemic threshold is obtained by Eq. (6), which means that the epidemic threshold depends on the fluctuations of the degree distribution and the quarantined rate σ. When σ increases, the corresponding threshold β_c rises and I^∞ decreases significantly, which shows that increasing the quarantined rate helps contain the epidemic. When $\sigma = 0$ and $\alpha = 0$, the model yields the standard SIRS epidemic with the valid threshold $\lambda_c = \frac{\beta_c}{\gamma} = \frac{\langle k\rangle}{\langle k^2\rangle}$, which consists with [12], only correlated with network topology. Apparently, in the eco-limits of infinite network size the nodes grows to infinity, i.e. $N \to \infty$, then $\langle k^2\rangle \to \infty$, so the absence of an epidemic threshold, i.e. $\lambda_c \to 0$, is observed.

Theorem 2. The disease-free equilibrium E_0 of the system (1) is locally asymptotically stable when $\rho < 1$ and unstable when $\rho > 1$. When $\rho > 1$, the disease is permanent on the scale-free networks, i.e., there exists a $\varsigma > 1$, such that

$$\liminf_{t\to\infty} I(t) = \liminf_{t\to\infty}\sum_k P(k)I_k(t) > \varsigma.$$

Proof. We rewrite the system (1) as

$$\begin{cases} \frac{dI_k(t)}{dt} = \frac{\beta k}{\langle k\rangle}(1-I_k(t)-Q_k(t)-R_k(t))\sum_k kP(k)I_k(t) - \gamma I_k(t) - \sigma I_k(t) \\ \frac{dQ_k(t)}{dt} = \sigma I_k(t) - \varepsilon Q_k(t) \\ \frac{dR_k(t)}{dt} = \gamma I_k(t) + \varepsilon Q_k(t) + \alpha(1-I_k(t)-Q_k(t)-R_k(t)) - \delta R_k(t) \end{cases} \tag{8}$$

The Jacobian matrix of system (8) at $\{(0,0,\alpha/\delta+\alpha)\}$ is a $3k_{\max}\times 3k_{\max}$ as follows

$$J = \begin{bmatrix} A_1 & B_{12} & B_{13} & \cdots & B_{1k_{\max}} \\ B_{21} & A_2 & B_{23} & \cdots & B_{2k_{\max}} \\ \vdots & & \ddots & & \vdots \\ B_{k_{\max}1} & B_{k_{\max}2} & B_{k_{\max}3} & \cdots & A_{k_{\max}} \end{bmatrix},$$

where

$$A_j = \begin{bmatrix} \frac{\beta j^2}{\langle k\rangle}P(j) - \gamma - \sigma & 0 & 0 \\ 0 & -\varepsilon & 0 \\ \gamma - \alpha & \varepsilon - \alpha & -\alpha - \delta \end{bmatrix}$$

$$B_{ij} = \begin{bmatrix} \frac{\beta ij}{\langle k\rangle}P(j) & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

By mathematical induction method, the characteristic polynomial can be calculated as follows

$$(z+\varepsilon)^{k_{\max}}(z+\alpha+\delta)^{k_{\max}-1}(z+\gamma+\sigma)^{k_{\max}-1}\left((z+\alpha+\delta)(z+\gamma+\sigma) - \beta\frac{P(1)+2^2P(2)+\cdots+k_{\max}^2P(k_{\max})}{\langle k\rangle}\right) = 0$$

Note that

$$\left\langle k^2\right\rangle = P(1)+2^2P(2)+\cdots+k_{\max}^2P(k_{\max})$$

The stability of E_0 is only dependent on

$$(z+\alpha+\delta)(z+\gamma+\sigma) - \beta\frac{\left\langle k^2\right\rangle}{\langle k\rangle} = 0$$

Hence, E_0 is locally asymptotically stable if $\rho < 1$ and unstable if $\rho > 1$.

Next, we discuss the permanence of the disease. We will use the result of Thieme in Theorem 4.6 [24] to prove it. Define
$X = \{(S_1, I_1, Q_1, R_1, \ldots, S_{K_{max}}, I_{K_{max}}, Q_{K_{max}}, R_{K_{max}}) : S_k, I_k, Q_k, R_k \geq 0 \text{ and } S_k + I_k + Q_k + R_k = 1, k = 1, K_{max}$
$X_0 = \{(S_1, I_1, Q_1, R_1, \ldots, S_{k_{\max}}, I_{k_{\max}}, Q_{k_{\max}}, R_{k_{\max}}) \in X : \sum_k P(k)I_k > 0\}$, $\partial X_0 = X \setminus X_0$.

In the following, we will show that (1) is uniformly persistent with respect to $(X_0, \partial X_0)$.

Obviously, X is positively invariant with respect to system (1). If $S_k(0) \geqslant 0, \sum_k P(k)I_k > 0, Q_k(0) \geqslant 0$ and $R_k(0) \geqslant 0$ for $k = 1, \cdots, k_{\max}$, then $S_k(0) \geqslant 0, \sum_k P(k)I_k > 0, Q_k(0) \geqslant 0$ and $R_k(0) \geqslant 0$ for all $t > 0$. Since $\left(\sum_k P(k)I_k(t)\right)' \geqslant -(\gamma+\sigma)\sum_k P(k)I_k(t)$ and $\sum_k P(k)I_k(0) > 0$, we have $\sum_k P(k)I_k(t) \geqslant \sum_k P(k)I_k(0)e^{-(\gamma+\sigma)t} > 0$. Thus, X_0 is also positively invariant. Furthermore, there exists a compact set B in which all solutions of (1) initiated in X will enter and remain forever after. The compactness condition (C4.2) in Thieme [24] is easily verified for this set B. Denote

$$M_\partial = \{(S_1(0), I_1(0), Q_1(0), R_1(0), \ldots, S_{k_{\max}}(0), I_{k_{\max}}(0), Q_{k_{\max}}(0), R_{k_{\max}}(0)) : (S_1(t), I_1(t), Q_1(t), R_1(t), \ldots, S_{k_{\max}}(t), I_{k_{\max}}(t), Q_{k_{\max}}(t), R_{k_{\max}}(t)) \in \partial X_0, t \geqslant 0\}.$$

Denote

$$\Omega = \cup\{\omega(S_1(0), I_1(0), Q_1(0), R_1(0), \ldots, S_{k_{\max}}(0), I_{k_{\max}}(0), Q_{k_{\max}}(0), R_{k_{\max}}(0)) : (S_1(0), I_1(0), Q_1(0), R_1(0), \ldots, S_{k_{\max}}(0), I_{k_{\max}}(0), Q_{k_{\max}}(0), R_{k_{\max}}(0)) \in X\},$$

where $\omega(S_1(0), I_1(0), Q_1(0), R_1(0), \ldots, S_{k_{\max}}(0), I_{k_{\max}}(0), Q_{k_{\max}}(0), R_{k_{\max}}(0))$ is the omega limit set of the solutions of system (1) starting in $(S_1(0), I_1(0), Q_1(0), R_1(0), \ldots, S_{k_{\max}}(0), I_{k_{\max}}(0), Q_{k_{\max}}(0), R_{k_{\max}}(0))$, Restricting system (1) on M_∂ gives

$$\begin{cases} \frac{dS_k(t)}{dt} = -\alpha S_k(t) + \delta R_k(t) \\ \frac{dI_k(t)}{dt} = -\gamma I_k(t) - \sigma I_k(t) \\ \frac{dQ_k(t)}{dt} = -\varepsilon Q_k(t) \\ \frac{dR_k(t)}{dt} = \varepsilon Q_k(t) + \alpha S_k(t) - \delta R_k(t) \end{cases} \tag{9}$$

It is easy to verify that system (9) has a unique equilibrium E_0 in X. Thus E_0 is the unique equilibrium of system (1) in M_∂. It is easy to check that E_0 is locally asymptotically stable. This implies that E_0 is globally asymptotically stable for (9) is a linear system. Therefore $\Omega = \{E_0\}$. And E_0 is a covering of X, which is isolated and is acyclic (since there exists no solution in M_∂) which links E_0 to itself). Finally, the proof will be done if we show E_0 is a weak repeller for X_0, i.e.,

$$\limsup_{t\to\infty} dist((S_1(t), I_1(t), Q_1(t), R_1(t), \cdots, S_{k_{\max}}(t), I_{k_{\max}}(t), Q_{k_{\max}}(t), R_{k_{\max}}(t)), E_0) > 0,$$

where $(S_1(t), I_1(t), Q_1(t), R_1(t), \cdots, S_{k_{\max}}(t), I_{k_{\max}}(t), Q_{k_{\max}}(t), R_{k_{\max}}(t))$ is an arbitrarily solution with initial value in X_0. By Leenheer and Smith [25], we need only to prove $W^s(E_0) \cap X_0 = \emptyset$ where $W^s(E_0)$ is the stable manifold of E_0. Suppose it is not true, then there exists a solution $(S_1(t), I_1(t), Q_1(t), R_1(t), \ldots, S_{k_{\max}}(t), I_{k_{\max}}(t), Q_{k_{\max}}(t), R_{k_{\max}}(t))$ in X_0, such that

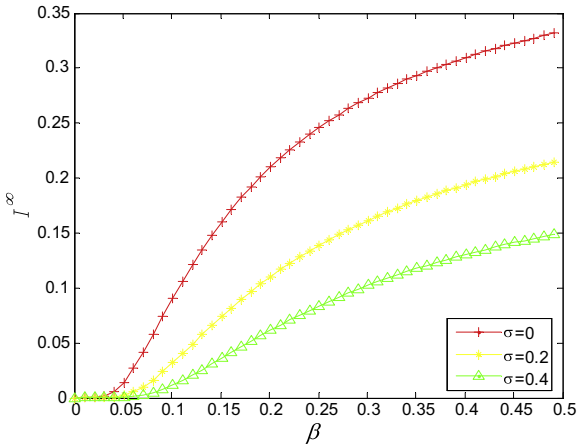

Fig. 2. The relations between I^∞ and β with $\sigma = 0$, $\sigma = 0.2$, $\sigma = 0.4$.

$$S_k(t) \to \frac{\delta}{\delta+\alpha}, \quad I_k(t) \to 0, Q_k(t) \to 0, \quad R_k(t) \to \frac{\alpha}{\delta+\alpha} ast \to \infty \tag{10}$$

Since $\rho = \frac{\delta\beta}{(\alpha+\delta)(\gamma+\sigma)} \frac{\langle k^2\rangle}{\langle k\rangle} > 1$, we can choose $\eta > 0$ such that

$$\frac{\delta\beta\left(\frac{\delta}{\delta+\alpha} - \eta\right)}{(\alpha+\delta)(\gamma+\sigma)} \frac{\langle k^2\rangle}{\langle k\rangle} > 1.$$

For $\eta > 0$, by (10) there exists a $T > 0$ such that

$$\frac{\delta}{\delta+\alpha} - \eta < S_k(t) < \frac{\delta}{\delta+\alpha} + \eta, \quad 0 \leqslant I_k(t) < \eta, \quad 0 \leqslant Q_k(t) < \eta, \frac{\alpha}{\delta+\alpha} - \eta < Q_k(t) < \frac{\alpha}{\delta+\alpha} + \eta$$

for all $t \geqslant T$ and $k = 1, \cdots, k_{\max}$. Let

$$V(t) = \sum_k \delta k P(k) I_k(t).$$

The derivative of V along the solution is given by

$$\begin{aligned}
\dot{V}(t) &= \sum_k \delta k P(k) \left[\beta k S_k(t) \frac{\sum_k kP(k)I_k(t)}{\langle k\rangle} - (\gamma+\sigma) I_k(t)\right] \geqslant \sum_k P(k) \frac{\delta\beta k^2}{\langle k\rangle} \left(\frac{\delta}{\delta+\alpha} - \eta\right) \sum_k kP(k)I_k(t) - \sum_k \delta k P(k)(\gamma+\sigma) I_k(t) \\
&= \sum_k P(k) k \left[\frac{\delta\beta\langle k^2\rangle}{\langle k\rangle} \left(\frac{\delta}{\delta+\alpha} - \eta\right) - \delta(\gamma+\sigma)\right] I_k(t) = \sum_k P(k) k \left[\frac{\delta\beta\langle k^2\rangle}{\langle k\rangle} \left(\frac{\delta}{\delta+\alpha} - \eta\right) - (\alpha+\delta)(\gamma+\sigma)\right] I_k(t) \\
&> \sum_k P(k) k I_k(t) \geqslant 0
\end{aligned}$$

Hence $V(t) \to \infty$ as $t \to \infty$, which contradicts to the boundedness of $V(t)$. This completes the proof. □

4. Numerical simulations

In this section, we verify the analytical results of the epidemic threshold above through numerical simulations on Barabási and Albert (BA) scale-free network. The parameters of BA networks are N = 10,000, $m = 3$, so $\langle k\rangle = 6$. Here, The parameters are chosen as $\alpha = 0.1, \delta = 0.4, \gamma = 0.5\varepsilon = 0.6$. Fig. 2 shows the relations between I^∞ and the spreading probability β with different values of σ.

Clearly, with changes of σ, different threshold β_c and densities of infected individuals in steady state are observed. We can see that, when σ increases, the corresponding β_c rises and I^∞ decreases significantly. The simulations indicate that the numerical results are well consistent with the theoretical analysis.

5. Conclusion

Considering an efficient strategy in epidemic containment, quarantining the infective individuals, we have proposed a (SIQRS) epidemic model on scale-free networks in this paper. By mean-filed theory, we obtain two epidemiologically

relevant quantities, namely, the threshold for invasion and the endemic prevalence of infection. As results indicate, the epidemic threshold on scale-free networks is virtually correlated with the fluctuations of the degree distribution. Moreover, increasing the quarantining proportion can result in the raise of infection threshold and the decrease of the population finally infected. The study has valuable guiding significance in effectively preventing epidemic spreading.

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