

Dynamical Analysis of a Delayed SIQS Epidemic Model on Scale-free Networks*

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Abstract In this paper, we establish a novel delayed SIQS epidemic model on scale-free networks, where time delay represents the average quarantine period. Through mathematical analysis, we present the basic reproduction number R_0 . Then, we provide the global asymptotical stability of the disease-free equilibrium and the local asymptotical stability of the endemic equilibrium. Finally, we perform numerical simulations to verify the correctness of the main results and analyze the sensitivity of parameters. Our research shows that when $R_0 > 1$, lengthening the quarantine period can slow the spread of the disease and reduce the number of infected individuals.

Keywords Epidemic model, Network, Quarantine period, Stability.

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1. Introduction

Infectious diseases have always been the great enemy of human health. Through out the history, the epidemics of infectious diseases have brought great disasters to human survival and national economy. To study the propagation dynamics and curb strategies of infectious diseases, the mathematical compartmental model is an important method [1].

In recent years, with the development of global transportation network [4], human behavior and social interpersonal communication are heterogeneous. Thus, it is necessary and reasonable to construct epidemic models on complex networks. In the field of complex networks, a research [2] showed that many networks, such as contact networks, have the property of being scale-free. For example, the distribution of connectivity degrees follows the power-law $p(k) = Ck^{-\gamma}$ ($2 < \gamma \leq 3$). In 2001, Pastor-Satorras et al. first denied an SIS model on scale-free networks, and concluded the absence of an epidemic threshold on a wide range of scale-free networks [22]. Moreno et al. studied an SIR epidemic model on two complex networks [20]. These results have attracted more researchers to study this field [3, 8–10, 12, 13, 15–18, 25, 27, 28].

After the disease breaks out, how to control the spread of disease effectively is an important issue. One effective method is quarantine, which has long been widely used to control the spread of disease. By cutting off contact between individuals,

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epidemics are often brought under control. In 2014, Li et al. proposed an SIQRS epidemic model on scale-free networks and proved the stability of disease-free equilibrium and the permanence of the disease [13]. Then, Huang et al. investigated a novel SIQRS epidemic model with demographics and analyzed the global epidemic behavior [8]. In 2019, Li et al. introduced an SIQS model on complex networks with birth and death mechanism, and introduced its optimal control [12]. Chen et al. established two epidemic models including variable population size, degree-related imperfect vaccination and quarantine on scale-free networks and proved the global stability of disease-free and endemic equilibrium [3].

To reflect the propagation process of the epidemic more realistically, many researchers analyzed delayed compartment models on complex networks. Time delays in the models represent average infectious period of the disease [15, 16, 28], average incubation period [9, 25] and the immunity period of recovery [17]. However, few researchers studied the effects of quarantine period on the spread of diseases. In this paper, we focus on the effectiveness of quarantine period on epidemics and construct a novel delayed SIQS epidemic model on scale-free networks.

In the delayed SIQS model, infected individuals may be quarantined and treated for a period of time, and become full susceptibility to the infection again. The primary purpose of this paper is to investigate the dynamical behavior of this model. In Section 2, we present the delayed SIQS model on scale-free networks. In Section 3, we define the basic reproduction number and analyze the dynamical behavior. In Section 4, we give numerical simulations to demonstrate the main results. In Section 5, we summarize this work.

2. Model

Let us make following assumptions:

(H1) Each node on the network represents an individual. All nodes on the networks can be classified into one of three categories: susceptible(S), infected(I) and quarantined(Q).

(H2) A susceptible node can be infected by contact with every infected node.

(H3) Every infected node will become susceptible to infection again after a period of quarantine.

(H4) Similar to [11, 12], we suppose the total nodes on the network is a constant. For example, the number of birth nodes is equal to the number of natural death nodes.

(H5) Every new birth node is susceptible.

Based on above assumptions, we consider a delayed SIQS epidemic model on scale-free networks. Let $S_k(t)$, $I_k(t)$ and $Q_k(t)$ represent the relative density of susceptible nodes, infected nodes and quarantined nodes respectively with degree k ($k = 1, 2, \dots, n$) at time t . The SIQS epidemic model is as follows:

$$\begin{cases} \frac{dS_k(t)}{dt} = \mu - \lambda(k)S_k(t)\Theta(t) + \gamma I_k(t - \tau)e^{-\mu\tau} - \mu S_k(t), \\ \frac{dI_k(t)}{dt} = \lambda(k)S_k(t)\Theta(t) - (\mu + \gamma)I_k(t), \\ \frac{dQ_k(t)}{dt} = \gamma I_k(t) - \gamma I_k(t - \tau)e^{-\mu\tau} - \mu Q_k(t), \end{cases} \quad (2.1)$$

where parameters $\lambda(k)$, γ , and μ are positive. The $\lambda(k)$ (such as λk [23], $\lambda c(k)$ [21]) is the correlated rate when susceptible nodes come into contact with infected nodes. γ is the rate when infective nodes move into quarantine. μ denotes the birth rate, which is equal to the per capita natural mortality rate. τ represents the average quarantine period, which refers to the period during which an infected node becomes a susceptible node after quarantine. $\Theta(t)$ denotes the probability rate that any link points to an infected node, and

$$\Theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)I_k(t). \tag{2.2}$$

Here, $\langle k \rangle$ is the average degree of the network, i.e., $\langle k \rangle = \sum_{k=1}^n kp(k)$. $\varphi(k) = ak^\alpha / (1 + bk^\alpha)$ [26] denotes the infectivity of a node with degree k , where $0 \leq \alpha \leq 1$, $a > 0$ and $b \geq 0$. If $b \neq 0$, $\varphi(k)$ is monotonically increasing with k , and it has an upper bound, i.e., $\lim_{k \rightarrow +\infty} \varphi(k) = a/b$.

Suppose that the initial condition of the system (2.1) takes the form

$$S_k(\eta) = \phi_1^k(\eta), \quad I_k(\eta) = \phi_2^k(\eta), \quad Q_k(\eta) = \phi_3^k(\eta), \tag{2.3}$$

where

$$\phi_i^k(\eta) \geq 0, \quad (\eta \in [-\tau, 0], i = 1, 2, 3, k = 1, 2, \dots, n).$$

By the theory of functional differential equations [7], system (2.1) has a unique solution $(S_k(t), I_k(t), Q_k(t))$ satisfying the initial conditions (2.3). It also proves that when $t \geq 0$, the solutions of the system (2.1) with the above initial conditions are all positive.

3. Dynamical behavior of the model

In this section, we will give our main conclusions.

Denote

$$R_0 = \frac{\langle \lambda(k)\varphi(k) \rangle}{(\mu + \gamma)\langle k \rangle}, \tag{3.1}$$

where $\langle \lambda(k)\varphi(k) \rangle = \sum_{k=1}^n \lambda(k)\varphi(k)p(k)$ and $\langle k \rangle = \sum_{k=1}^n kp(k)$.

Theorem 3.1. *Consider system (2.1), we have the following assertions.*

- (1) *System (2.1) always has a disease-free equilibrium E^0 , where $E^0 = (S_1^0, I_1^0, Q_1^0, S_2^0, I_2^0, Q_2^0, \dots, S_n^0, I_n^0, Q_n^0)$, in which $S_k^0 = 1, I_k^0 = 0, Q_k^0 = 0, k = 1, 2, \dots, n$.*
- (2) *System (2.1) has a unique endemic equilibrium E^* when $R_0 > 1$, where $E^* = (S_1^*, I_1^*, Q_1^*, S_2^*, I_2^*, Q_2^*, \dots, S_n^*, I_n^*, Q_n^*)$.*

Proof. We can find that

$$\Omega = \{(S_1, I_1, Q_1, \dots, S_n, I_n, Q_n) \in \mathbb{R}_+^{3n} : 0 \leq S_k, I_k, Q_k \leq 1, k = 1, 2, \dots, n\}$$

is a positively invariant set for system (2.1). It is clear that E^0 is always an equilibrium of system (2.1). Then, the equilibrium E^* of system (2.1) satisfies

$$\begin{cases} \mu - \lambda(k)S_k^*\Theta^* + \gamma I_k^*e^{-\mu\tau} - \mu S_k^* = 0, \\ \lambda(k)S_k^*\Theta^* - (\mu + \gamma)I_k^* = 0, \\ \gamma I_k^* - \gamma I_k^*e^{-\mu\tau} - \mu Q_k^* = 0, \end{cases} \quad (3.2)$$

where

$$\Theta^* = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)I_k^*. \quad (3.3)$$

Solving the three equations of (3.2), it yields that

$$I_k^* = \frac{\lambda(k)\Theta^*\mu}{\lambda(k)\Theta^*(\mu + \gamma - \gamma e^{-\mu\tau}) + \mu(\mu + \gamma)}. \quad (3.4)$$

Substituting I_k^* into (3.3), we can obtain the self-consistency equation about Θ^* as follows:

$$\Theta^* = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k) \frac{\lambda(k)\Theta^*\mu}{\lambda(k)\Theta^*(\mu + \gamma - \gamma e^{-\mu\tau}) + \mu(\mu + \gamma)}. \quad (3.5)$$

Evidently, $\Theta^* = 0$ is a solution to (3.5), and it follows that the virus-free equilibrium E_0 of system (2.1) always exists. To ensure (3.5) has a nontrivial solution, we take $\Theta^* > 0$. Then, we divide both sides of (3.5) by Θ^* ,

$$1 = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k) \frac{\lambda(k)\mu}{\lambda(k)\Theta^*(\mu + \gamma - \gamma e^{-\mu\tau}) + \mu(\mu + \gamma)}. \quad (3.6)$$

Let

$$f(\Theta^*) = 1 - \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k) \frac{\lambda(k)\mu}{\lambda(k)\Theta^*(\mu + \gamma - \gamma e^{-\mu\tau}) + \mu(\mu + \gamma)}.$$

Note that

$$f'(\Theta^*) = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k) \frac{\lambda(k)(\mu + \gamma - \gamma e^{-\mu\tau})}{[\lambda(k)\Theta^*(\mu + \gamma - \gamma e^{-\mu\tau}) + \mu(\mu + \gamma)]^2} > 0$$

and

$$\lim_{\Theta \rightarrow +\infty} f(\Theta) = 1.$$

The equation $f(\Theta) = 0$ has a unique non-trivial solution if and only if

$$f(0) = 1 - \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k) \frac{\lambda(k)}{\mu + \gamma} = 1 - R_0 < 0.$$

We conclude that (3.6) has a unique positive solution Θ^* in the interval $(0, 1)$ when $R_0 > 1$. That is to say, system (2.1) has a unique endemic equilibrium E^* , when $R_0 > 1$.

Remark 3.1. The basic reproduction number R_0 is an important conception in the transmission of diseases, which represents the average number of secondary infectious infected by an individual of infections during whose whole course of disease in the case that all the members of the population are susceptible [19]. We explain the biological meaning of R_0 (3.1) as follows. For the primary infected case, the average period spent in an infectious stats is $\mu + \gamma$. During this period, the average number of the susceptible infected by the primary infected case is $\sum_{k=1}^n \frac{\lambda(k)\varphi(k)p(k)}{\langle k \rangle}$. In addition, we get that R_0 decreases with the increase of birth rate μ and quarantine rate γ , and increases with the increase of correlated infection rate $\lambda(k)$.

Theorem 3.2. *If $R_0 < 1$, the disease-free equilibrium E^0 of system (2.1) is globally asymptotically stable for $\forall \tau \geq 0$.*

Proof. Consider the following Lyapunov function

$$V(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)I_k(t).$$

Taking the derivative of $V(t)$ along the solution of system (2.1) and noting that $0 < S_k(t) \leq 1$, we obtain

$$\begin{aligned} \frac{dV(t)}{dt} |_{(2.1)} &= \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)[\lambda(k)S_k(t)\Theta(t) - (\mu + \gamma)I_k(t)] \\ &\leq \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)[\lambda(k)\Theta(t) - (\mu + \gamma)I_k(t)] \\ &= \Theta(t) \left[\frac{\langle \lambda(k)\varphi(k) \rangle}{\langle k \rangle} - (\mu + \gamma) \right]. \end{aligned}$$

Thus, $\frac{dV(t)}{dt} \leq 0$ when $R_0 < 1$, and $\frac{dV(t)}{dt} = 0$ if and only if $I_k(t) = 0$. According to the LaSalle Invariance Principle [6], we conclude that E^0 is globally asymptotically stable when $R_0 < 1$ for $\tau \geq 0$.

Theorem 3.3. *If $R_0 > 1$, the infectious disease is uniformly persistent, i.e., there exists a positive constant ϵ such that $\lim_{t \rightarrow +\infty} \inf I_k(t) > \epsilon$.*

Proof. Denote

$$D = \left\{ (S_k(t), I_k(t), Q_k(t)) \in \Omega : I(t) = \sum_{k=1}^n P(k)I_k(t) > 0 \right\}, \partial D = D/\Omega.$$

Firstly, we prove that D is positively invariant with respect to system (2.1). $(S_k(0), I_k(0), Q_k(0)) \in D$, which means $\Theta(0) > 0$. Then, calculating the derivative of $\Theta(t)$ along the solution of system (2.1), we get

$$\frac{d\Theta(t)}{dt} = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)[\lambda(k)S_k(t)\Theta(t) - (\mu + \gamma)I_k(t)].$$

Thus,

$$\Theta(t) = \Theta(0) \exp \left\{ \frac{1}{\langle k \rangle} \int_0^t \sum_{k=1}^n \varphi(k) p(k) \lambda(k) S_k(\xi) d\xi - (\gamma + \mu)t \right\},$$

which has $\Theta(t) > 0$ for all $t > 0$. It implies that

$$\begin{aligned} \frac{dI(t)}{dt} &= \sum_{k=1}^n p(k) \frac{dI_k(t)}{dt} \\ &= \sum_{k=1}^n p(k) [\lambda(k) S_k(t) \Theta(t) - (\mu + \gamma) I_k(t)] \\ &> -(\mu + \gamma) I_k(t). \end{aligned}$$

Hence, $I(t) > I(0) \exp[-(\mu + \gamma)t] > 0$. Obviously, D is a positive invariant, and system (2.1) is a dissipative system. Then, E_0 is the unique equilibrium of system (2.1) on ∂D , and the ω -limit of system (2.1) on ∂D . E_0 is isolated and acyclic. Lastly, by Theorem 4.6 in [24], the infectious disease will be uniformly persistent, if we prove that

$$W^s(E_0) \cap \partial D = \emptyset, \quad (3.7)$$

where $W^s(E_0)$ denotes the stable manifold of E_0 .

Suppose it is not valid, then there is a solution $(S_k(t), I_k(t), Q_k(t)) \in D$ such that $S_k(t) \rightarrow 1, I_k(t) \rightarrow 0, Q_k(t) \rightarrow 0$ as $t \rightarrow \infty$. Since $R_0 > 1$, we can choose a $\eta > 0$ small enough such that $(1 - \eta)R_0 > 1$. On the other hand, for the above $\eta > 0$, there is a $t_\eta > 0$ such that $1 - \eta \leq S_k(t) \leq 1 + \eta, 0 \leq I_k(t) \leq \eta, 0 \leq Q_k(t) \leq \eta$ for $t > t_\eta$. Denote

$$\Phi(t) = \frac{1}{\gamma + \mu} \Theta(t),$$

and $\Phi(t)$ is clearly bounded function. Taking the derivative of $\Phi(t)$ along solution of system (2.1) for $t > t_\eta$, we come to a conclusion that

$$\begin{aligned} \frac{d\Phi(t)}{dt} &= \frac{1}{\gamma + \mu} \langle k \rangle^{-1} \sum_{k=1}^n \varphi(t) p(k) [\lambda(k) S_k(t) \Theta(t) - (\mu + \gamma) I_k(t)] \\ &\geq \frac{1}{\gamma + \mu} \langle k \rangle^{-1} \sum_{k=1}^n \varphi(t) p(k) [\lambda(k) (1 - \eta) \Theta(t) - (\mu + \gamma) I_k(t)] \\ &= \frac{1}{\gamma + \mu} [\langle k \rangle^{-1} \sum_{k=1}^n \varphi(t) p(k) \lambda(k) (1 - \eta) \Theta(t) - (\mu + \gamma) \Theta(t)] \\ &= (\gamma + \mu) [(1 - \eta) R_0 - 1] \Phi(t). \end{aligned}$$

Hence, $\Phi(t) \geq \Phi(t_\eta) \exp[(\gamma + \mu)[(1 - \eta)R_0 - 1]t]$, we have $\Phi(t) \rightarrow +\infty$ as $t \rightarrow +\infty$. It is contradictory to the boundedness of $\Phi(t)$. This completes the proof.

Theorem 3.4. *If $R_0 > 1$, the endemic equilibrium E^* of system (2.1) is locally asymptotically stable for $\forall \tau \geq 0$.*

Proof. Since $Q_k(t) = 1 - S_k(t) - I_k(t)$, it is ample to discuss the following system,

$$\begin{cases} \frac{dS_k(t)}{dt} = \mu - \lambda(k)S_k(t)\Theta(t) + \gamma I_k(t - \tau) - \mu S_k(t), \\ \frac{dI_k(t)}{dt} = \lambda(k)S_k(t)\Theta(t) - (\mu + \gamma)I_k(t). \end{cases} \tag{3.8}$$

Let $x_k(t) = S_k(t) - S_k^*$, $y_k(t) = I_k(t) - I_k^*$. The linear system of system (3.8) at (S_k^*, I_k^*) is as follows:

$$\begin{cases} \frac{dx_k(t)}{dt} = -(\mu + \lambda(k)\Theta^*)x_k(t) - \lambda(k)S_k^*\theta(t) + \gamma y_k(t - \tau), \\ \frac{dy_k(t)}{dt} = \lambda(k)\Theta^*x_k(t) + \lambda(k)S_k^*\theta(t) - (\mu + \gamma)y_k(t), \end{cases}, \tag{3.9}$$

where $\theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^n \varphi(k)p(k)y_k(t)$.

Denote $b(k) = \frac{1}{\langle k \rangle} \varphi(k)p(k)$, and we can obtain the following equation from (3.2) and (3.9):

$$\begin{aligned} \frac{d\theta(t)}{dt} &= \sum_{k=1}^n b(k)[\lambda(k)\Theta^*x_k(t) + \lambda(k)S_k^*\theta(t) - (\mu + \gamma)y_k(t)] \\ &= \sum_{k=1}^n b(k)\lambda(k)\Theta^*x_k(t) \\ &= \Theta^* \sum_{k=1}^n b(k)\lambda(k)x_k(t). \end{aligned}$$

Analyze the following linear system:

$$\frac{d}{dt} \begin{pmatrix} \theta(t) \\ x_1(t) \\ y_1(t) \\ \vdots \\ x_n(t) \\ y_n(t) \end{pmatrix} = M \begin{pmatrix} \theta(t) \\ x_1(t) \\ y_1(t) \\ \vdots \\ x_n(t) \\ y_n(t) \end{pmatrix} + N \begin{pmatrix} \theta(t - \tau) \\ x_1(t - \tau) \\ y_1(t - \tau) \\ \vdots \\ x_n(t - \tau) \\ y_n(t - \tau) \end{pmatrix}. \tag{3.10}$$

Let ρ represent the eigenvalue of the characteristic equation of system (3.10), and ρ satisfies the following equation

$$f(\rho, \tau) = \det |\rho I - M - Ne^{-\rho\tau}| = 0. \tag{3.11}$$

Denote $\bar{M} = M + Ne^{-\rho\tau}$, where \bar{M} is a $(2n + 1) \times (2n + 1)$ matrix, and

$$\bar{M} = \begin{bmatrix} 0 & \Theta^*b(1)\lambda(1) & 0 & \cdots & \Theta^*b(k)\lambda(k) & 0 & \cdots & \Theta^*b(n)\lambda(n) & 0 \\ -\lambda(1)S_1^* & h_1(1) & h_2 \cdots & 0 & 0 \cdots & 0 & 0 & 0 & 0 \\ \lambda(1)S_1^* & h_3(1) & h_4 \cdots & 0 & 0 \cdots & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \vdots \ddots & \vdots & \vdots \ddots & \vdots & \vdots & \vdots & \vdots \\ -\lambda(k)S_k^* & 0 & 0 \cdots & h_1(k) & h_2 \cdots & 0 & 0 & 0 & 0 \\ \lambda(k)S_k^* & 0 & 0 \cdots & h_3(k) & h_4 \cdots & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \vdots \ddots & \vdots & \vdots \ddots & \vdots & \vdots & \vdots & \vdots \\ -\lambda(n)S_n^* & 0 & 0 \cdots & 0 & 0 \cdots & h_1(n) & h_2 & h_3 & h_4 \\ \lambda(n)S_n^* & 0 & 0 \cdots & 0 & 0 \cdots & h_3(n) & h_4 & h_5 & h_6 \end{bmatrix},$$

where $h_1(k) = -(\lambda(k)\Theta^* + \mu)$, $h_2 = \gamma e^{-\rho\tau}$, $h_3(k) = \lambda(k)\Theta^*$, $h_4 = -(\gamma + \mu)$. In order not to change the eigenvalue of the matrix, we apply the similarity transformation to the matrix \bar{M} . Specifically, the $2k$ th row is added to the $(2k + 1)$ th row, the $(2k + 1)$ th column multiplied by -1 is added to the $2k$ th column, $k = 1, 2, \dots, n$. Hence matrix \bar{M} becomes

$$\bar{M}_1 = \begin{bmatrix} 0 & \Theta^*b(1)\lambda(1) & 0 & \cdots & \Theta^*b(k)\lambda(k) & 0 & \cdots & \Theta^*b(n)\lambda(n) & 0 \\ -\lambda(1)S_1^* & \bar{h}_1(1) & \bar{h}_2 \cdots & 0 & 0 \cdots & 0 & 0 & 0 & 0 \\ 0 & \bar{h}_3 & \bar{h}_4 \cdots & 0 & 0 \cdots & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \vdots \ddots & \vdots & \vdots \ddots & \vdots & \vdots & \vdots & \vdots \\ -\lambda(k)S_k^* & 0 & 0 \cdots & \bar{h}_1(k) & \bar{h}_2 \cdots & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 \cdots & \bar{h}_3 & \bar{h}_4 \cdots & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \vdots \ddots & \vdots & \vdots \ddots & \vdots & \vdots & \vdots & \vdots \\ \lambda(n)S_n^* & 0 & 0 \cdots & 0 & 0 \cdots & \bar{h}_1(n) & \bar{h}_2 & \bar{h}_3 & \bar{h}_4 \\ 0 & 0 & 0 \cdots & 0 & 0 \cdots & \bar{h}_3 & \bar{h}_4 & \bar{h}_5 & \bar{h}_6 \end{bmatrix},$$

where $\bar{h}_1(k) = -(\lambda(k)\Theta^* + \mu + \gamma e^{-\rho\tau})$, $\bar{h}_2 = \gamma e^{-\rho\tau}$, $\bar{h}_3 = \gamma(1 - e^{-\rho\tau})$, $\bar{h}_4 = -\mu + \gamma(e^{-\rho\tau} - 1)$.

Considering the characteristic root of matrix \bar{M}_1 , the characteristic equation (3.11) becomes

$$f(\rho, \tau) = \rho \prod_{k=1}^n A_k(\rho) + \sum_{k=1}^n B_k(\rho) \prod_{l \neq k}^n A_l(\rho) = 0, \tag{3.12}$$

where

$$A_k(\rho) = \begin{vmatrix} \lambda(k)\Theta^* + \mu + \gamma e^{-\rho\tau} + \rho & -\gamma e^{-\rho\tau} \\ \gamma(e^{-\rho\tau} - 1) & \mu + \gamma(1 - e^{-\rho\tau}) + \rho \end{vmatrix},$$

$$B_k(\rho) = \Theta^*b(k)\lambda(k)[(\lambda(k)S_i^*)(\mu + \gamma(1 - e^{-\rho\tau}) + \rho)], \quad i = 1, 2, \dots, n.$$

(I) When $\tau = 0$, $e^{-\rho\tau} = 1$,

$$A_k(\rho) = (\lambda(k)\Theta^* + \mu + \gamma + \rho)(\mu + \rho).$$

Obviously, the real parts of the eigenvalues of the corresponding matrix to $A_k(\rho)$ are negative. Then, we mark that $\rho_k = \lambda(k)\Theta^* + \mu + \gamma$, and $A_k(\rho)$ becomes

$$A_k(\rho) = (\rho + \rho_k)(\rho + \mu).$$

Then,

$$\begin{aligned} B_k(\rho) &= \Theta^*b(k)\lambda(k)[(\lambda(k)S_k^*)(\rho + \mu)] \\ &= C_k(\rho + \mu), \end{aligned}$$

where $C_k = \Theta^*b(k)\lambda(k)^2S_k^*$, and

$$\begin{aligned} f(\rho, 0) &= \rho \prod_{k=1}^n A_k(\rho) + \sum_{k=1}^n B_k(\rho) \prod_{l \neq k}^n A_l(\rho) \\ &= \rho \prod_{k=1}^n (\rho + \rho_k)(\rho + \mu) + (\rho + \mu) \sum_{k=1}^n C_k \prod_{l \neq k}^n (\rho + \rho_l)(\rho + \mu) \tag{3.13} \\ &= (\rho + \mu)^n \left[\rho \prod_{k=1}^n (\rho + \rho_k) + \sum_{k=1}^n C_k \prod_{l \neq k}^n (\rho + \rho_l) \right]. \end{aligned}$$

Consider the different forms of $\lambda(k)$. Firstly, assume that all $\lambda(k)$ are respectively unequal, and ρ_k are also mutually unequal. From equation (3.13), we obtain that n eigenvalues are equal to $-\mu$, and others satisfy the following equation

$$\rho \prod_{k=1}^n (\rho + \rho_k) + \sum_{k=1}^n C_k \prod_{l \neq k}^n (\rho + \rho_l) = 0. \tag{3.14}$$

Noting that equation (3.14) has the same form as [27] equation (3.4), we can refer to the method of proof which is presented in [27] Theorem 3.3]. We obtain that the real parts of all roots of equation (3.14) are negative.

Secondly, without loss of generality, we assume that m_1 functions $\lambda(k)$ are mutually unequal, m_2 functions $\lambda(k)$ have one equal value and m_3 functions $\lambda(k)$ have another equal value, where $n = m_1 + m_2 + m_3$. Then, we reorder $\lambda(k)$ as $\tilde{\lambda}(k)$: when $k = 1, 2, \dots, m_1, m_1 + 1, m_1 + 2, \dots, m_1 + m_2$, $\tilde{\lambda}(k)$ are mutually unequal; when $k = m_1 + 3, m_1 + 4, \dots, m_1 + m_2 + 1, \dots, m_1 + m_2 + m_3$, $\tilde{\lambda}(k) = \tilde{\lambda}(m_1 + 1)$; when $k = m_1 + m_2 + 2, m_1 + m_2 + 3, \dots, n$, $\tilde{\lambda}(k) = \tilde{\lambda}(m_1 + 2)$. C_k changes order with $\tilde{\lambda}(k)$ to become \tilde{C}_k . Denote $\tilde{\rho}_k = \tilde{\lambda}(k)\Theta^* + \mu + \gamma$, and equation(3.13) becomes

$$f(\rho, 0) = (\rho + \mu)^n (\rho + \tilde{\rho}_{m_1+1})^{m_2-1} (\rho + \tilde{\rho}_{m_1+2})^{m_3-1} \left[\rho \prod_{k=1}^{m_1+2} (\rho + \tilde{\rho}_k) + \sum_{k=1}^n \tilde{C}_k \prod_{l \neq k}^{m_1+2} (\rho + \tilde{\rho}_l) \right]. \tag{3.15}$$

We obtain that equation (3.15) has n eigenvalues equaling to $-\mu$, $m_2 - 1$ eigenvalues equaling to $-\tilde{\rho}_{m_1+1}$, $m_3 - 1$ eigenvalues equaling to $-\tilde{\rho}_{m_1+2}$, and other eigenvalues satisfying the following equation

$$\rho \prod_{k=1}^{m_1+2} (\rho + \tilde{\rho}_k) + \sum_{k=1}^n \tilde{C}_k \prod_{l \neq k}^{m_1+2} (\rho + \tilde{\rho}_l) = 0. \tag{3.16}$$

Since equation (3.16) has the same expression with equation (3.14), we can solve it with the same mathematical proof. Namely, the real parts of roots of equation (3.16) are negative, and other similar circumstances can be also proved. Thus, the real parts of eigenvalues of $f(\rho, 0) = 0$ are negative.

(II) When $\tau > 0$, assume that equation (3.12) has a purely imaginary root $\rho = \omega i (\omega > 0)$.

$$\begin{aligned} A_k(\omega i) &= -\omega^2 + i(\lambda(k)\Theta^* + 2\mu + \gamma)\omega + (\lambda(k)\Theta^* + \mu)(\mu + \gamma) - \\ &\quad \lambda(k)\Theta^*\gamma(\cos(\omega\tau) - i\sin(\omega\tau)) \\ &= 0. \end{aligned}$$

Separate real and imaginary parts of $A_k(\omega i)$,

$$\begin{cases} \lambda(k)\Theta^*\gamma\cos(\omega\tau) = -\omega^2 + (\lambda(k)\Theta^* + \mu)(\mu + \gamma), \\ \lambda(k)\Theta^*\gamma\sin(\omega\tau) = -(\lambda(k)\Theta^* + 2\mu + \gamma)\omega. \end{cases}$$

Square and add two equations,

$$[\lambda(k)\Theta^*\gamma]^2 = [\omega^2 + (\lambda(k)\Theta^* + \mu)^2][\omega^2 + (\mu + \gamma)^2].$$

Since $\omega^2 > 0$, this equation has no root, i.e., equation $A_k(\rho) = 0$ has no purely imaginary roots.

Then, we separate real and imaginary parts of $B_k(\omega i)$,

$$\begin{cases} \gamma\cos(\omega\tau) = \mu + \gamma, \\ \gamma\sin(\omega\tau) = -\omega. \end{cases}$$

Namely,

$$\gamma^2 = (\mu + \gamma)^2 + \omega^2,$$

Since $\omega^2 > 0$, this equation has no root, i.e., equation $B_k(\rho) = 0$ has no purely imaginary roots.

Moreover, we study equation (3.12),

$$\begin{aligned} f(\omega i, \tau) &= \omega i \prod_{k=1}^n A_k(\omega i) + \sum_{k=1}^n B_k(\omega i) \prod_{l \neq k}^n A_l(\omega i) \\ &= \left(\sum_{k=1}^n \frac{B_k(\omega i)}{A_k(\omega i)} + \omega i \right) \prod_{k=1}^n A_k(\omega i). \end{aligned}$$

We mention that equation $\frac{B_k(\omega i)}{A_k(\omega i)}$ has real parts, i.e., there are no purely imaginary roots such that $\left(\sum_{k=1}^n \frac{B_k(\omega i)}{A_k(\omega i)} + \omega i \right) = 0$. Thus, combining that equation $A_k(\rho) = 0$ has no purely imaginary roots, we obtain that there are not purely imaginary roots satisfying equation (3.12).

Therefore, on the one hand, the real parts of all roots of the equation $f(\rho, 0) = 0$ are negative. On the other hand, equation (3.12) has no purely imaginary roots. According to [[6], Chapter 8], the real parts of eigenvalues of the characteristic equation (3.12) are negative. Namely, the endemic equilibrium E^* of system (2.1) is locally asymptotically stable for $\forall \tau \geq 0$.

4. Numerical simulations

In this section, we present some numerical simulations to verify the correctness of our main result. The simulations are based on a heterogeneous scale-free network, in which the degree distribution follows a low law distribution. The degree distribution $p(k) = Ck^{-\gamma}$ and $\gamma = 2.8$ [5], where the constant C satisfies $\sum_{k=1}^n p(k) = 1$.

We assume the network is finite, the minimal degree of nodes is 1 and the maximal degree of nodes n is equal to 100. The nonlinear infectivity $\varphi(k) = ak^\alpha / (1 + bk^\alpha)$, in which $a = 0.5$, $\alpha = 0.75$, $b = 0.02$. The correlated infection rate $\lambda(k) = \lambda k$. Denote

$$I(t) = \sum_{k=1}^n p(k)I_k(t),$$

in which $I(t)$ represents the average density of the infected nodes. Similarly, $S(t)$ and $Q(t)$ represent the average density of the susceptible and quarantine nodes respectively. The initial values are $I_k(s) = 0.01$, $k = 5, 6, 7, 8, 9$ and $I_k(s) = 0$, $k \neq 5, 6, 7, 8, 9$ where $s \in [-\tau, 0]$ and $Q_k(s) = 0$, where $s \in [-\tau, 0]$.

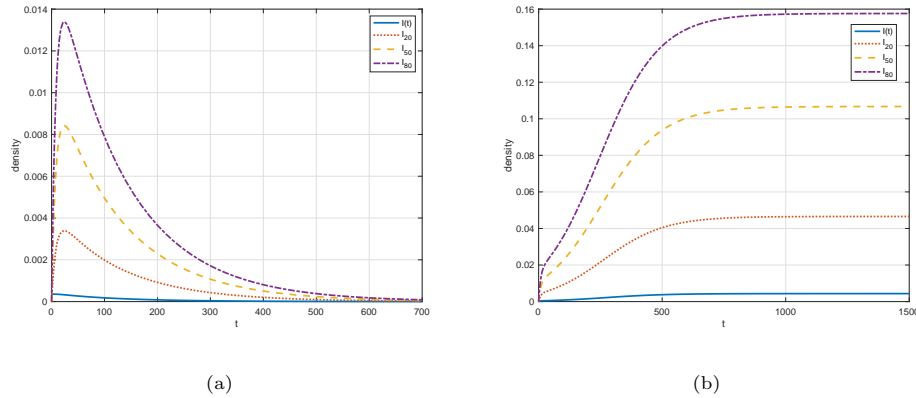


Figure 1. (a) Time evolution of $I(t)$, $I_{20}(t)$, $I_{50}(t)$ and $I_{80}(t)$ when $R_0 \approx 0.9376 < 1$; (b) Time evolution of $I(t)$, $I_{20}(t)$, $I_{50}(t)$ and $I_{80}(t)$ when $R_0 \approx 1.0176 > 1$.

First, we show the stability of the equilibrium. Give the following parameters $\lambda = 0.07$, $\mu = 0.06$, $\gamma = 0.06$, $\tau = 6$, and the basic reproduction number $R_0 \approx 0.9376 < 1$. Figure 1(a) shows the evolution of $I(t)$, $I_{20}(t)$, $I_{50}(t)$ and $I_{80}(t)$. Infected nodes tend to 0 as $t \rightarrow \infty$, which consistent with Theorem 3.2. Choose $\lambda = 0.08$, and the basic reproduction number $R_0 \approx 1.0716 > 1$. Figure 1(b) shows the evolution of $I(t)$, $I_{20}(t)$, $I_{50}(t)$ and $I_{80}(t)$. Infected nodes continues to exist on a unique positive stats, which consistent with Theorem 3.4.

Then, we study the influence of quarantine period τ . Give the following parameters $\lambda = 0.07$, $\mu = 0.06$, $\gamma = 0.06$ and the basic reproduction number $R_0 \approx 0.9376 < 1$. Let τ vary, time evolution of $I(t)$, $S(t)$ and $Q(t)$ are shown in Figure 2(a) 2(b) and 2(c) respectively. Choose $\lambda = 0.2$, and the basic reproduction number $R_0 \approx 2.6789 > 1$. Time evolution of $I(t)$, $S(t)$ and $Q(t)$ with different τ are shown in Figure 2(d), 2(e) and 2(f) respectively. According to these figures, we find that when $R_0 < 1$, time delay τ impact the asymptotic convergence rate

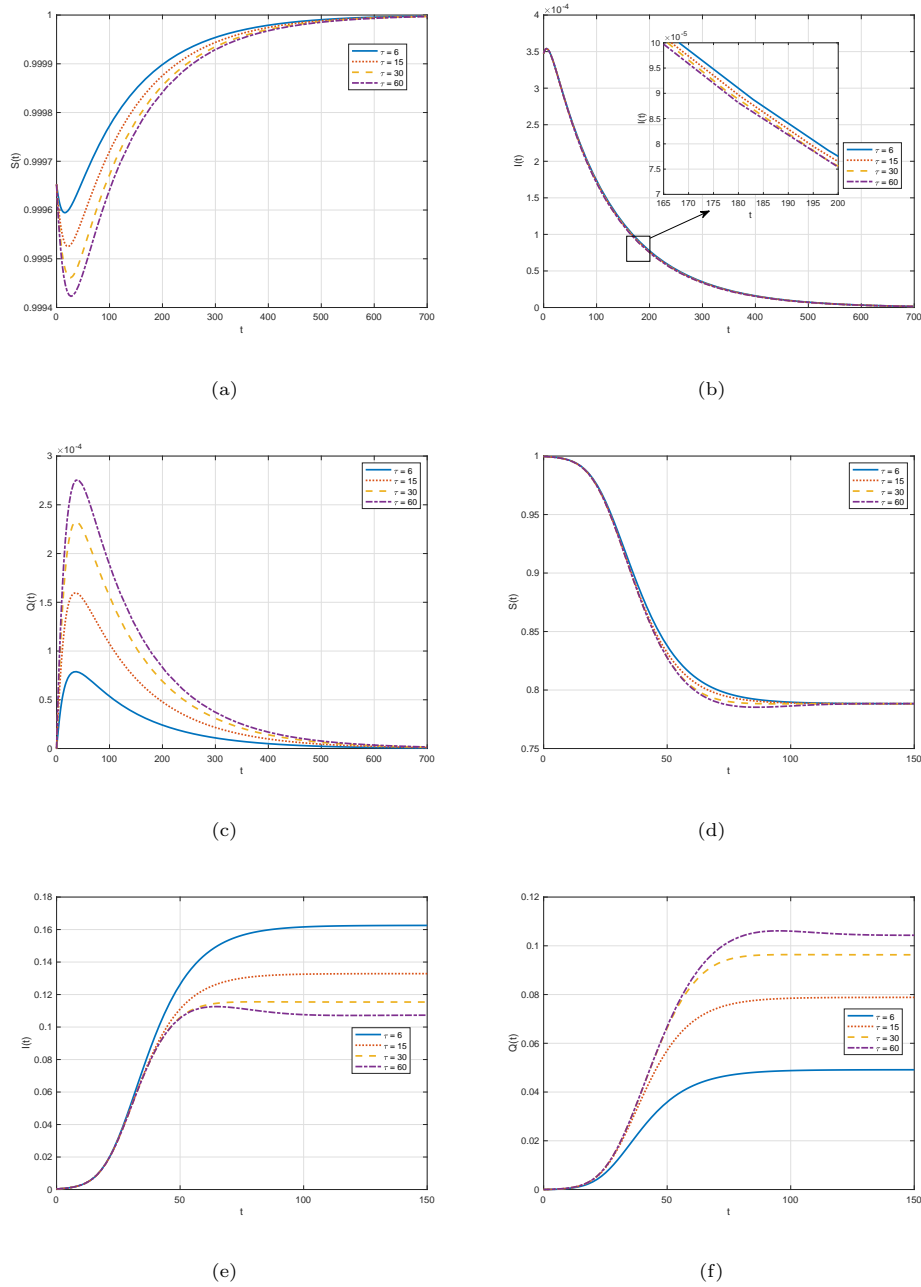
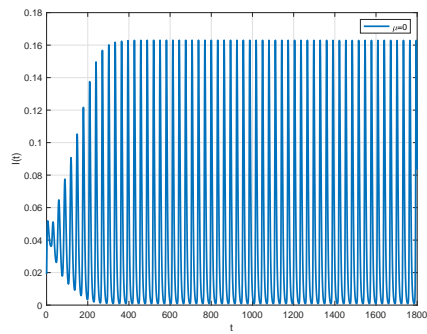
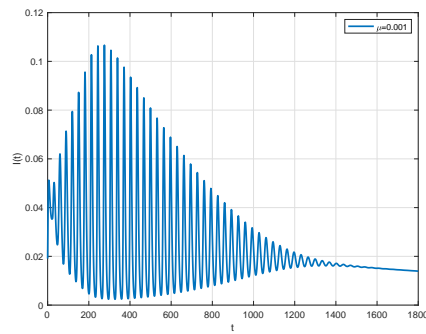


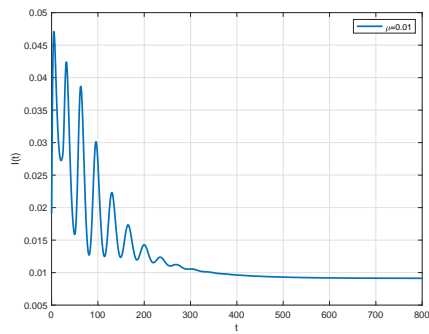
Figure 2. When $R_0 < 1$, time evolution of $S(t)$, $I(t)$ and $Q(t)$ with different τ in (a), (b) and (c) respectively; When $R_0 > 1$, time evolution of $S(t)$, $I(t)$ and $Q(t)$ with different τ in (d), (e) and (f) respectively.



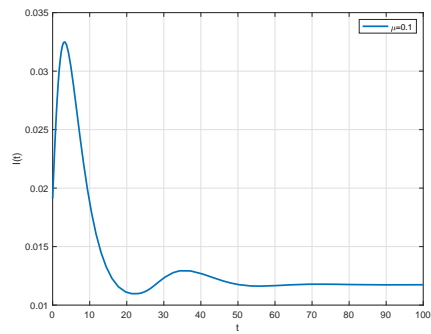
(a)



(b)



(c)



(d)

Figure 3. Time evolution of $I(t)$ with different μ .

of the system, but scale tends to have no disease eventually. When $R_0 > 1$, the different τ not only impact the asymptotic convergence rate of the system, but also the eventually scale of diseases. That is, when $R_0 > 1$, infected nodes decrease with the increase of quarantine period τ , quarantine nodes increase with the increase of τ and susceptible nodes are independent of τ .

Finally, we show an interesting phenomenon about parameter μ . The initial values are $I_k(s) = 0.55$, $k = 2, 3, 4, 5$ and $I_k(s) = 0$, $k \neq 2, 3, 4, 5$ where $s \in [-\tau, 0]$ and $Q_k(s) = 0$ where $s \in [-\tau, 0]$. Provide the following parameters $\lambda = 0.6$, $\gamma = 0.5$, $\tau = 23$. As can be seen in Figure 3(a), bifurcation phenomenon occurs at the endemic equilibrium when $\mu = 0$ and $R_0 \approx 1.9231 > 1$. This is consistent with the conclusion in Reference [14]. In Figure 3(b), Figure 3(c) and Figure 3(d), we take $\mu = 0.001$, $\mu = 0.01$ and $\mu = 0.1$ respectively, and the basic reproduction numbers are $R_0 \approx 1.9192$, $R_0 \approx 1.8854$ and $R_0 \approx 1.6026$ respectively. It is clear that with the increase of μ , the frequency of the oscillation of $I(t)$ decreases, and the convergence rate accelerates.

5. Conclusion

To study the effectiveness of quarantine period on diseases transmission, we discuss a novel delayed SIQS epidemic model on scale-free networks. Based on mathematical proofs, we obtain the formula of basic reproduction number R_0 , which is independent of the average quarantine period τ . When $R_0 < 1$, the infection disappears, i.e., the disease-free equilibrium E_0 is globally asymptotically stable. Otherwise, when $R_0 > 1$, the infectious disease is uniformly persistent, and the endemic equilibrium E^* is locally asymptotically stable. The following numerical simulations verify the correctness of the main result.

The simulation result shows that the quarantine period τ affects the dynamical behavior of system. When $R_0 > 1$, infected individuals decrease as τ increases, quarantine individuals increase as τ increases, and susceptible individuals are independent of quarantine period τ . Therefore, when an epidemic breaks out, appropriately increasing the quarantine period is an effective control strategy.

Interestingly, we also find that system (2.1) has a bifurcation phenomena when there is no birth rate μ . In other words, even if the total number of nodes in the network does not change, the dynamical performance on static networks and dynamic networks are different. We leave it for our future research.

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