Chapter 2

# The Dynamics Analysis of Two Delayed Epidemic Spreading Models with Latent Period on Heterogeneous Network

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Additional information is available at the end of the chapter

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**Abstract**

Two novel delayed epidemic spreading models with latent period on scale-free network are presented. The formula of the basic reproductive number and the analysis of dynamical behaviors for the models are presented. Meanwhile, numerical simulations are given to verify the main results.

**Keywords:** epidemic spreading, scale-free network, basic reproductive number, time delay, stability

## 1. Introduction

Following the seminal work on scale-free network, in which the probability of $p(k)$ for any node with $k$ links to other nodes is distributed according to the power law $p(k) = Ck^{-\gamma}$ $(2 < \gamma \leq 3)$, suggested by Barabási and Albert [1], the researches of complex network have attracted more and more interests. It was found that many relevant networks, for instance, the internet, the World Wide Web (WWW), the patterns of human sexual contacts, biology network, transportation infrastructure, etc., exhibit power-law or "scale-free" degree distributions.

The dynamical behaviors of epidemic diseases have been studied for a long time. The epidemic spreading process on network is primarily dominated by two factors: one is the macroscopic topology of the underlying network, and the other is the microscopic infection scheme, which includes properties of disease, infection pattern, individual differences, infectivity of individuals, etc. The traditional epidemic dynamics is based on homogeneous network, and the infectivity rate is equally likely over all links [2]. However, the real disease transmission network exhibits scale-free properties, and the spreading of epidemic disease (e.g., computer

virus spreading, epidemic disease between human beings) on heterogeneous network, i.e., scale-free network, has been studied by many researchers [3–27].

A handful of existing works address the complex behavior of epidemic spreading using compartmental differential equations [2, 15]. Comparing with the ordinary differential equation models, more realistic models should be retarded functional differential equation models which can include some of the past states of these systems. Time delay plays an important role in the process of the epidemic spreading, for instance, the latent period of the infectious diseases or computer virus, the infection period of infective members, and the immunity period of the recovered individuals can be represented by time delays [2]. Recently, some researchers discussed the epidemic spreading model with time delays [7, 15–17]. Susceptible-Infected-Removed (*SIR*) model is a basic and important epidemic model, Zou and Wu discussed a delayed SIR model without birth and death [15], and Wang and Wang et al. discussed a delayed SIR model with birth rate and death rate [16]. However, it is suitable to divide the nodes being considered into disjoint classes of susceptible, exposed, infective, and recovered nodes in modeling disease transmission [2, 10], i.e., a susceptive node first through an incubation period (and it is said to become exposed) after infection and before becoming infectious. For example, the latent period of epidemic cholera is about 1–3 days, hepatitis B virus 100 days, measles 10–11 days, chincough 7–10 days, diphtheria 2–4 days, scarlatina 2–5 days, poliomyelitis 7–14 days, and so on [25]; the resulting model is Susceptible-Exposed-Infected-Removed (*SEIR*) model. In addition, some diseases confer temporary immunity, and the recovered nodes cycle back into the susceptive class after an immune period; the resulting model is Susceptible-Exposed-Infected-Removed-susceptible (*SEIRS*) model.

In this paper, we will present a suitable *SEIR* model with time delay and a suitable *SEIRS* model with time delay on heterogeneous network by using functional differential equation to investigate the dynamical behaviors of epidemic spreading.

The rest of this paper is organized as follows: In Section 2, the *SEIRS* model with time delay on scale-free network is discussed. The *SEIRS* model with time delay on scale-free network is discussed in Section 3. Finally, the main conclusions of this work are summarized in Section 4.

## 2. Analysis of the *SEIR* model with time delay

### 2.1. The *SEIR* model

Suppose that the size of the network is a constant $N$ and the degree of each node is time invariant during the period of epidemic spreading, $p(k)$ denotes the degree distribution of the network. We classify all the nodes in the network into n groups such that the nodes in the same group have the same degree. That is, each node in the $k$th group has the same connectivity ($k = m, m+1, \cdots, n$). Let $S_k(t), E_k(t), I_k(t)$ and $R_k(t)$ be the relative density of susceptible nodes, exposed nodes, infected nodes, and recovered nodes of connectivity $k$ at time $t$, respectively, where $k = m, m+1, \cdots, n$ ($m$ and $n$ are the minimum and maximum degree in network topology) and $n$ is related to the network age, measured as the number of nodes $N$ [3]:

$$n = mN^{1/(\gamma-1)}. \tag{1}$$

Let $\tau$ be the latent period of the disease, i.e., each exposed node becomes an infected node after $\tau$. The relative density $S_k(t)$, $E_k(t)$, $I_k(t)$ and $R_k(t)$, at the mean-field level, satisfy the following set of coupled different equations when $t>0$ [15, 16]:

$$\begin{cases} \dot{S}_k(t) &= -\lambda(k)S_k(t)\Theta(t), \\ \dot{E}_k(t) &= \lambda(k)S_k(t)\Theta(t) - \lambda(k)S_k(t-\tau)\Theta(t-\tau), \\ \dot{I}_k(t) &= \lambda(k)S_k(t-\tau)\Theta(t-\tau) - \mu I_k(t), \\ \dot{R}_k(t) &= \mu I_k(t) \end{cases} \tag{2}$$

with the normalization condition

$$S_k(t) + E_k(t) + I_k(t) + R_k(t) = 1 \tag{3}$$

holds due to the fact that the number of total nodes with degree $k$ is a constant $p(k)N$ during the period of epidemic spreading. Where $\lambda(k)$ is the degree-dependent infection rate such as $\lambda(k)=\lambda k$ [3] and $\lambda C(k)$ [4], $\mu$ is the recovery rate of the infected nodes; $\Theta(t)$ represents the probability that any given link points to an infected node. Assuming that the network has no degree correlations [7, 16, 22], we have

$$\Theta(t) = \frac{1}{\langle k\rangle}\sum_{k=m}^{n}\phi(k)p(k)I_k(t) \tag{4}$$

in which $\langle k\rangle = \sum_k p(k)k$ stands for the average node degree and $\phi(k)$ means the occupied edges which can transmit the disease (i.e., represents the infectivity of infected nodes) [22]; they have many different forms, such as $\varphi(k)=A$ in [5], $\varphi(k)=ak^{\alpha}/(1+bk^{\alpha})$, $0<\alpha<1$ in [7], and so on. Here, we point out that the delay $\tau$ in the model (2) in this paper is different from one in the model (2)–(4) in [15]. The incubation period $\tau$ in the model in [15] is another kind of time period, during which the infectious agents develop in the vector and the infected vector becomes infectious after that time.

Note that we obtain from the third equation of system (2) that

$$E_k(t) = \lambda(k)\int_{t-\tau}^{t} S_k(s)\Theta(s)ds \tag{5}$$

and the normalization condition becomes the following mathematical form

$$S_k(t) + \lambda(k)\int_{t-\tau}^{t} S_k(s)\Theta(s)ds + I_k(t) + R_k(t) = 1. \tag{6}$$

The initial conditions of system (2) are

$$S_k(\theta) = \varphi_k(\theta), I_k(\theta) = \Psi_k(\theta), R_k(t) = \zeta_k(\theta), \theta\in[-\tau, 0] \tag{7}$$

and satisfy $S_k(0) + \lambda(k)\int_{-\tau}^{0} S_k(s)\Theta(s)ds + I_k(0) + R_k(0) = 1$ which guarantees the normalization condition holds. And $\Phi_k = (\phi_k(\theta), \Psi_k(\theta), \zeta(\theta), k = m, m+1, \cdots, n-m+1) \in C$ are nonnegative continuous on $[-\tau, 0]$, $\phi_k(0) > 0, \Psi_k(0) > 0$, and $\zeta(\theta) = 0$ for $\theta = 0$. $C$ denotes the Banach space $C([-\tau, 0], R^{3(n-m+1)})$ with the norm, where $|f(\theta)|_\tau = \sup_{-\tau \le \theta \le 0} |f(\theta)|$. $\|\omega\| = \left(\sum_{i=m}^{n} \left(|\Psi_i(\theta)|_\tau^2 + |\phi_i(\theta)|_\tau^2 + |\zeta_i(\theta)|_\tau^2\right)\right)^{1/2}$.

### 2.2. The main results for the model

In this section, we first discuss the final size relation of solutions for system (2).

It is easy to know that system (2) only has a disease-free equilibrium set

$$M_0 = \left\{\left(\widehat{S}, \widehat{E}, \widehat{I}, \widehat{R}\right) | E_k = I_k = 0, S_k + R_k = 1, R_k, S_k \ge 0, k = m, m+1, \cdots, n\right\} \tag{8}$$

in which $\widehat{S} = (S_m, S_{m+1}, \cdots, S_n)$, $\widehat{E} = (E_m, E_{m+1}, \cdots, E_n)$, $\widehat{I} = (I_m, I_{m+1}, \cdots, I_n)$, $\widehat{R} = (R_m, R_{m+1}, \cdots, R_n)$.

Supposing $f(t)$ is an arbitrary nonnegative continuous function $f(t)$, we adopt the following convention:

$$f(+\infty) = \lim_{t \to +\infty} f(t) \tag{9}$$

and we obtain from the last equation of system (2) that

$$R_k(+\infty) - R_k(0) = \mu \int_0^{+\infty} I_k(s)ds. \tag{10}$$

According to the last equation of system (2), $R_k(t)$ is increasing and bounded above by 1, and it has a limit as $t \to +\infty$. Thus, the left-hand side of (10) is finite due to boundedness of $R_k(+\infty)$, and $R_k(0)$ exits, i.e., $0 < \int_0^{+\infty} I_k(u)du < +\infty$. Since $I_k(t)$ is smooth nonnegative function, we know $I_k(+\infty) = 0$, i.e., $\lim_{t \to +\infty} I_k(t) = 0$.

Furthermore, we have from (5) that

$$0 \le E_k(t) = \lambda(k)\int_{t-\tau}^{t} S_k(s)\Theta(s)ds \le \lambda(k)\int_{t-\tau}^{t} \Theta(s)ds, \tag{11}$$

In addition, by using mean value theorem for integrals, we have

$$\int_{t-\tau}^{t} \Theta(s)ds = \Theta(\xi)\tau, \ (t - \tau \le \xi \le t). \tag{12}$$

We obtain from $I_k(+\infty) = 0$ that $\lim_{\xi \to +\infty} \Theta(\xi)\tau = 0$ and then $\lim_{t \to +\infty} E_k(t) = 0$. Hence, $M_0$ is globally attractive [27].

In addition, it follows from (2) that

$$\dot{S}_k(t) + \dot{E}_k(t) + \dot{I}_k(t) = -\mu I_k(t). \tag{13}$$

Integrating (13) from 0 to +∞, we obtain that

$$S_k(0) - S_k(+\infty) + E_k(0) - E_k(+\infty) + I_k(0) - I_k(+\infty) = \mu \int_0^{+\infty} I_k(s)ds. \tag{14}$$

Noting that $I_k(+\infty) = 0$, $E_k(0) = E_k(+\infty) = 0$, and $S_k(+\infty)$ exists due to existence of $\int_0^{+\infty} I_k(s)ds$, we have from (14) that

$$\int_0^{+\infty} I_k(s)ds = \frac{1}{\mu}(S_k(0) + I_k(0) - S_k(+\infty)). \tag{15}$$

Additionally, integrating the first equation of system (2) from 0 to + ∞, we have

$$\ln \frac{S_k(0)}{S_k(\infty)} = \frac{\lambda(k)}{\langle k \rangle} \sum_k \phi(k)p(k) \int_0^{+\infty} I_k(s)ds. \tag{16}$$

Substituting (15) into (16), we obtain that

$$\ln \frac{S_k(0)}{S_k(+\infty)} = \frac{\lambda(k)}{\langle k \rangle} \sum_k \phi(k)p(k) \frac{1}{\mu}(S_k(0) + I_k(0) - S_k(+\infty)). \tag{17}$$

Because there are only several infective nodes at the beginning of disease spreading, we take $S_k(0) \approx 1$ and obtain from (17) that

$$\ln S_k(+\infty) = \frac{\lambda(k)}{\mu\langle k \rangle} \sum_k \phi(k)p(k)(S_k(+\infty) - 1). \tag{18}$$

Consequently,

$$R_k(\infty) = 1 - S_k(+\infty). \tag{19}$$

Hence, we have the following result.

**Theorem 2.1.** *The equilibrium set* $M_0 = \left\{ \left( \widehat{S}, \widehat{E}, \widehat{I}, \widehat{R} \right) | E_k = I_k = 0, S_k + R_k = 1, k = 1, 2, \cdots, n \right\}$ *of system* (2) *is globally attractive, i.e.,* $\lim_{t \to +\infty} I_k(t) = 0$, $\lim_{t \to \infty} E_k(t) = 0$. *And* $R_k(+\infty)$, $S_k(+\infty)$ *are given by formulas* (18) *and* (19).

Note that it is impossible for every susceptible to be infected. Supposing $S_k(+\infty) = 0$, we know from (16) that

$$+\infty = \frac{\lambda(k)}{\mu\langle k \rangle} \sum_k \phi(k)p(k) \tag{20}$$

Obviously, Eq. (20) does not hold, i.e., $S_k(+\infty) = 0$. Similar results were obtained in the early literature [19].

Secondly, we discuss the basic reproductive number of model (2). The basic reproductive number is an important conception; it represents the average number of secondary infections

infected by an individual of infective during the whole course of disease in the case that all the members of the population are susceptible [2].

**Theorem 2.2.** *For system* (2),

$$R_0 = \frac{\langle \lambda(k)\phi(k) \rangle}{\mu \langle k \rangle} \tag{21}$$

*is the basic reproductive number for system* (2).

**Proof.** Note that $\sum_{k=m}^{n} \varphi(k)p(k)I_k(t)$ may be considered as the force of infection [15] and $\Theta(t)$ may be considered as the average force of infection. Letting $\Theta(t)$ be an auxiliary function and computing its time derivative along the solution of (2), we get

$$\begin{aligned} \frac{d\Theta(t)}{dt} &= \frac{1}{\langle k \rangle} \sum_k \phi(k)p(k)\dot{I}_k(t) \\ &= \frac{1}{\langle k \rangle} \sum_k \phi(k)p(k)\big(\lambda(k)S_k(t-\tau)\Theta(t-\tau) - \mu I_k(t)\big) \\ &= \Theta(t-\tau)\frac{1}{\langle k \rangle} \sum_k \lambda(k)\phi(k)p(k)S_k(t-\tau) - \mu\Theta(t). \end{aligned} \tag{22}$$

We have

$$\left.\frac{d\Theta(t)}{dt}\right|_{t=0} = \Theta(-\tau)\frac{1}{\langle k \rangle} \sum_k \lambda(k)\phi(k)p(k)S_k(-\tau) - \mu\Theta(0)). \tag{23}$$

Since each exposed node becomes infected node after $\tau$, $I_k(-\tau) = I_k(0)$. It follows that $\Theta(-\tau) = \Theta(0)$. Meanwhile, $S_k(-\tau) \approx 1$. Hence, we have from (23) that

$$\left.\frac{d\Theta(t)}{dt}\right|_{t=0} = \mu\left(\frac{1}{\mu\langle k \rangle} \sum_k \lambda(k)\phi(k)p(k) - 1\right)\Theta(0) = \mu(R_0 - 1)\Theta(0). \tag{24}$$

If $R_0 > 1$, $\left.\frac{d\Theta(t)}{dt}\right|_{t=0} > 0$, which means that $\Theta(t)$ increases at the beginning of the epidemic and there exists at least one outbreak.

Meanwhile, if $R_0 \le 1$, we obtain from (24) that $\left.\frac{d\Theta(t)}{dt}\right|_{t=0} \le 0$. Let $t^* = \sup\{T \ge 0 : \Theta(t)$ decreases on $[0, T]\}$. Then, it follows from the above discussion that $T \ge 0$. We will prove that $T = +\infty$. Note that we obtain from the first equation of system (2) that

$$S_k(t) = S_k(0)\mathrm{e}^{-\lambda(k)\Psi(t)} \tag{25}$$

in which $\Psi(t) = \frac{1}{\langle k \rangle}\sum_k \int_0^t \varphi(k)p(k)I_k(u)du$. Hence, it follows from Eqs. (22) and (25) that

$$\frac{d\Theta(t)}{dt} = \Theta(t-\tau)\frac{1}{\langle k \rangle} \sum_k \lambda(k)\phi(k)p(k)S_k(0)\mathrm{e}^{-\lambda(k)\Psi(t-\tau)} - \mu\Theta(t). \tag{26}$$

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By way of contradiction, supposing that $T<+\infty$, then we have $\frac{d}{dt}\Theta(t^*)=0$, and there exists a $t_1\in(t^*, t^*+\tau]$ such that $\frac{d}{dt}\Theta(t_1)>0$. It follows that there is a $t_2\in[t^*, t_1)$ such that $\frac{d}{dt}\Theta(t_2)=0$ and $\Theta(t_2)<\Theta(t_1)$. Note that $\Theta(t_2-\tau)\geq\Theta(t_1-\tau)$. It follows from (25) that

$$0<\frac{d}{dt}\Theta(t_1)\leq\frac{d}{dt}\Theta(t_2)=0, \tag{27}$$

which is a contradiction. Hence, $\Theta(t)$ decreases on $[0,+\infty)$, and there is no one outbreak when $R_0\leq 1$. Hence, $R_0$ is the basic reproductive number for system (2).

It follows from Theorems 2.1 and 2.2 that $R_0$ is the basic reproductive number for system (2), which is irrelative to $\tau$. There exists at least one outbreak for the spreading of epidemic if $R_0>1$, and there is no outbreak if $R_0\leq 1$. Whether or not there exists one outbreak for the spreading of epidemic, $\lim_{t\to+\infty}I_k(t)=0$ due to global attractivity of $M_0$.

Besides, if we let $\tau=0$, $\varphi(k)=k$, $\lambda(k)=\lambda k$, $\mu=1$, the model (2) reduces to the model in [9]. Furthermore, the basic reproductive number for system (2) is $R_0=(\lambda\langle k^2\rangle)/(\langle k\rangle)$, which is identical with the results that the epidemic threshold $\lambda_c=(\lambda\langle k\rangle)/(\langle k^2\rangle)$ in [9]. And, $R_0$ is always more than unity when $N$ is large enough [3, 7], and it means the lack of any basic reproductive number. This result is consistent with the results in epidemic dynamics on heterogeneous network [3, 10].

### 2.3. Numerical simulation for the model

Now, we present numerical simulations to support the results obtained in previous sections and analyze the effect of time delay on behaviors of disease spreading.

The degree distribution of scale-free network is $p(k)=Ck^{-\gamma}$, and $C$ satisfies $\sum_{k=1}^{n}p(k)=1$. Here, we set the maximum degree $n=100$ and the minimum degree $m=1$. Consider system (2), let $\varphi(k)=ak^{\alpha}/(1+b^{\alpha})$ in which $a=0.5, \alpha=0.75, b=0.02$ and $\lambda(k)=\lambda k$, and let $\gamma=2.5$. **Figures 1–4** show the dynamic behaviors of system (2) with the initial functions satisfying condition (7).

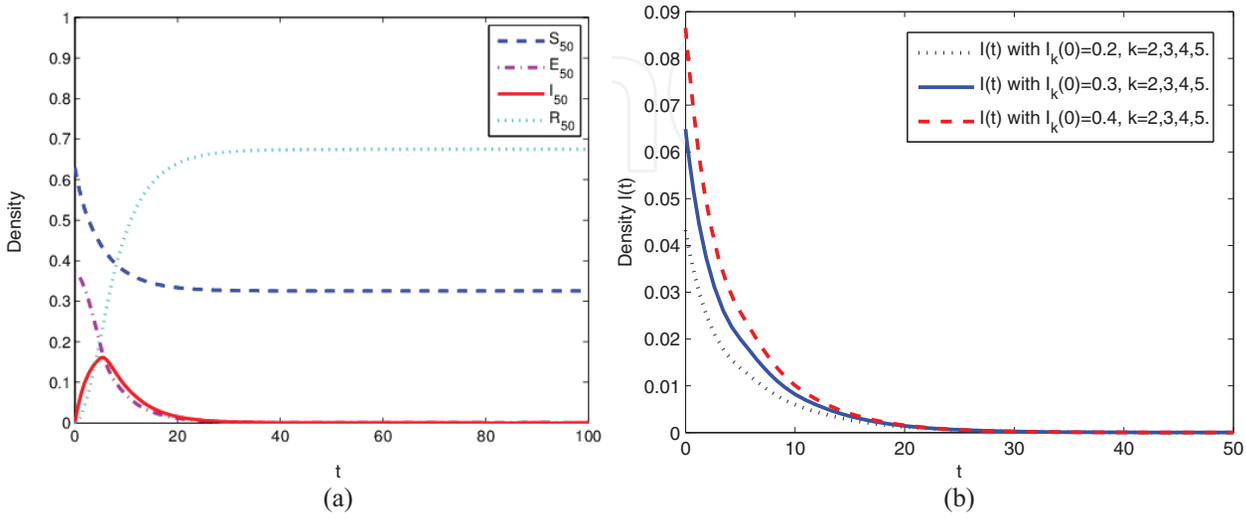


**Figure 1.** (a) The time evolutions for system (2) with $\lambda=0.5, \mu=0.4, \tau=5$ and $I_k(0)=0.2, k=2, 3, 4, 5, I_k(0)=0$ for the other $k$ and $R_0=0.4006$. (b) The time evolutions for system (2) with different initial values, $\lambda=0.4, \mu=0.4, \tau=5$ and $R_0=0.4006$.

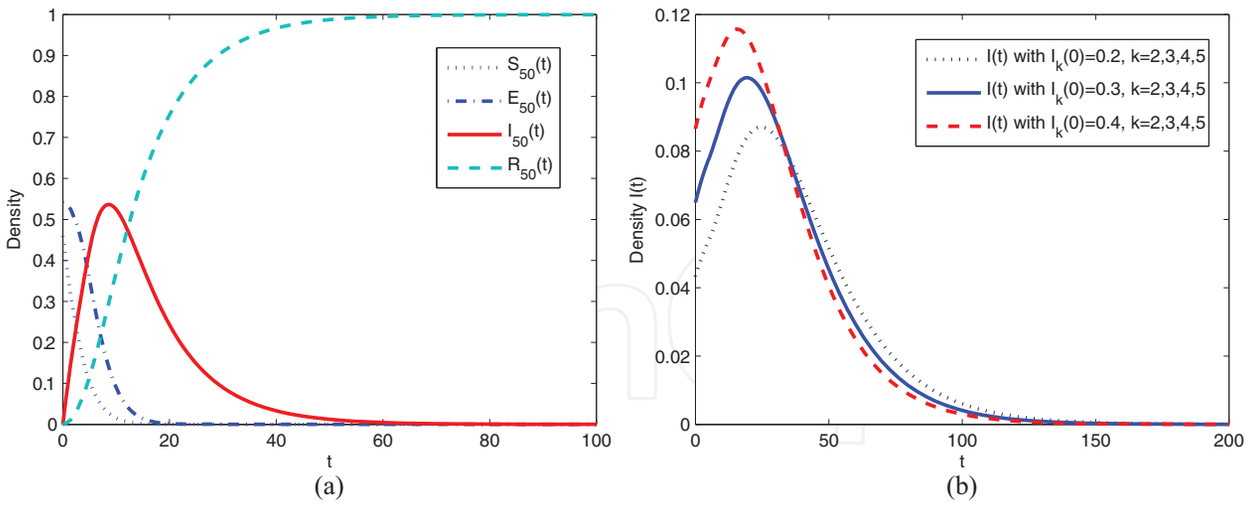


**Figure 2.** (a) The time evolutions for system (2) with $\lambda = 1$, $\mu = 0.1$, $\tau = 5$ and $I_k(0) = 0.2$, $k = 2, 3, 4, 5$, $I_k(0) = 0$ for the other $k$ and $R_0 = 3.2051$. (b) The time evolutions for system (2) with different initial values, $\lambda = 1$, $\mu = 0.1$, $\tau = 5$ and $R_0 = 3.2051$.

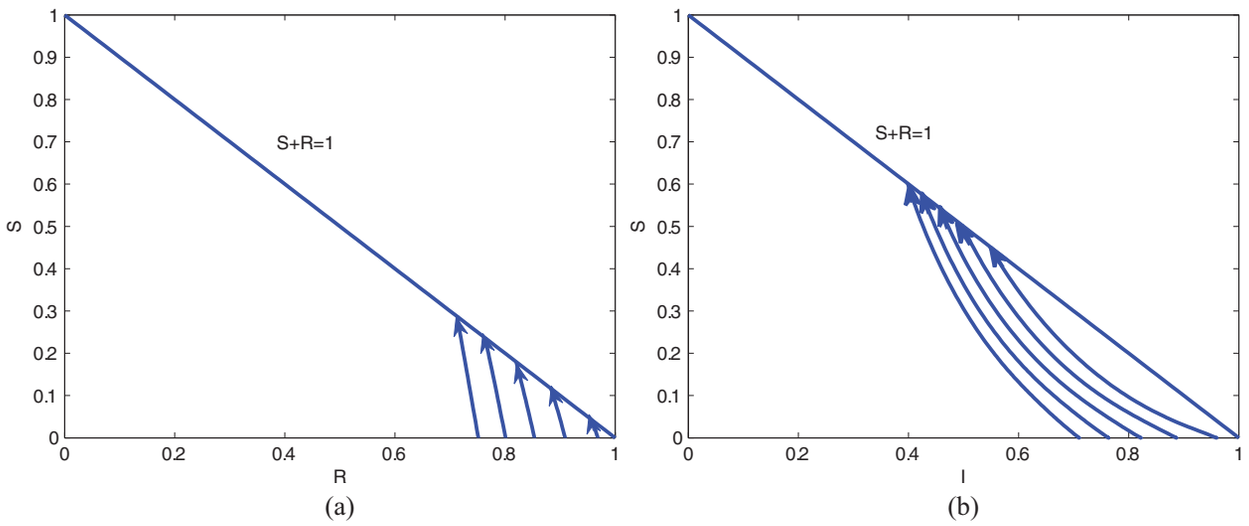


**Figure 3.** (a) Phase trajectories on SR-plane of system (2) with different initial values, $\lambda = 0.5$, $\mu = 0.4$, $\tau = 5$ and $R_0 = 0.4006$. (b) Phase trajectories on SR-plane of system (2) with different initial values, $\lambda = 1$, $\mu = 0.1$, $\tau = 5$ and $R_0 = 3.2051$.

Denote that

$$S(t) = \sum_{k=m}^{n} p(k)S_k(t), I(t) = \sum_{k=m}^{n} p(k)I_k(t), R(t) = \sum_{k=m}^{n} p(k)R_k(t). \tag{28}$$

They are the relative average density of susceptible nodes, exposed nodes, infected nodes, and recovered nodes at time $t$, respectively.

First, **Figures 1** and **2** show that the infection eventually disappears, whatever $R_0 < 1$ or not, and the outbreak of disease spreading appears when $R_0 > 1$ and the outbreak of disease spreading does not appear when $R_0 \leq 1$. Meanwhile, **Figure 3** shows that phase trajectories on SR-plane of system (2) with different initial values tend to be $S(t) + R(t) = 1$, i.e., $\sum_k p(k)(S_k(t) + R_k(t)) = 1$, which is consistent with the fact that the equilibrium $M_0$ is globally attractive. The numerical simulation results are identical with Theorems 2.1–2.2.

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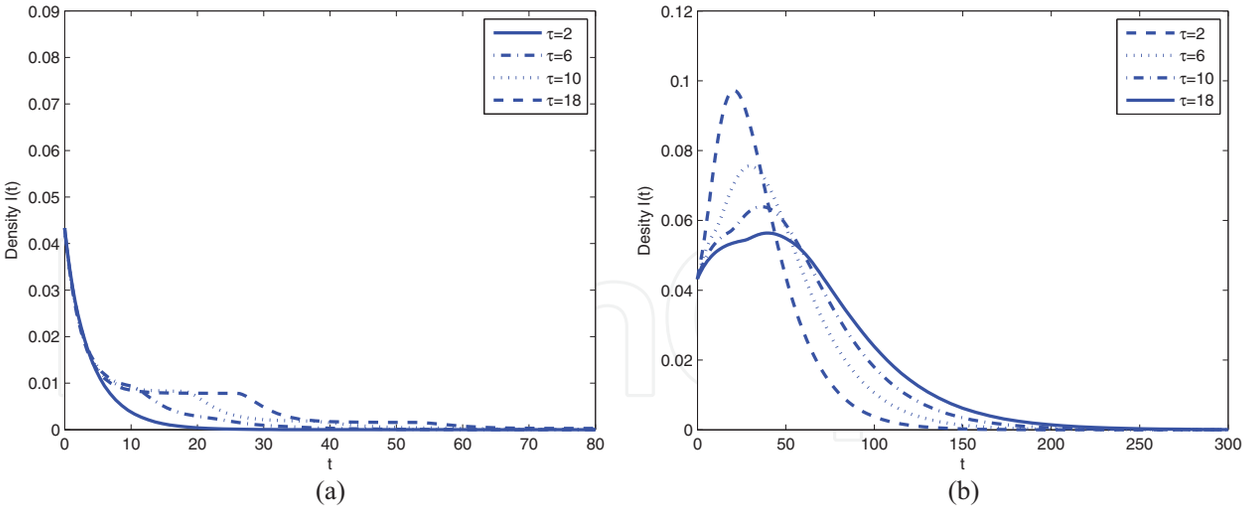


**Figure 4.** (a) The time evolutions of the average relative density $I(t)$ for system (2) with different $\tau$ as well as $I_k(0) = 0.2, k = 2, 3, 4, 5, I_k(0) = 0$ for the other $k$, $\lambda = 0.5, \mu = 0.4$ and $R_0 = 0.4006$. (b) The time evolutions of the average relative $I(t)$ for system (2) with different $\tau$ as well as $I_k(0) = 0.2, k = 2, 3, 4, 5, I_k(0) = 0$ for the other $k$, $\lambda = 1, \mu = 0.1$ and $R_0 = 3.2051$.

Second, time delay $\tau$ has no effects on the basic reproductive number $R_0$ according to (21), but it has much impact on the of process of the disease; the slower the relative density of infected nodes converges to zero, the larger $\tau$ gets, i.e., time delay may slow down the speed of disappearing the disease spreading on network. Meanwhile, time delay may effectively reduce the peak value of the relative density of infected nodes when $R_0 > 1$. Thus, the delay cannot be ignored.

At last, we know from **Figure 5** that time evolutions of the average force of infection for system (2) is consistent with time evolutions of the average relative density $I(t)$. However, there is only one outbreak, which is different from the phenomenon in [15].

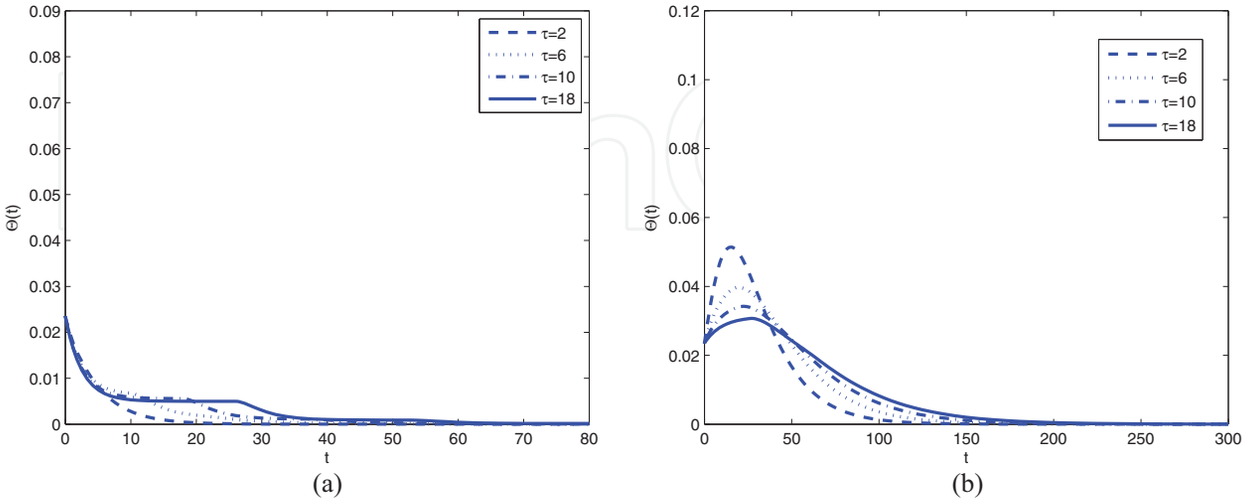


**Figure 5.** (a) The time evolutions of the average force of infection $\Theta(t)$ for system (2) with different $\tau$ as well as with $I_k(0) = 0.2, k = 2, 3, 4, 5, I_k(0) = 0$ for the other $k$, $\lambda = 0.5, \mu = 0.4$ and $R_0 = 0.4006$. (b) The time evolutions of the average force of infection $\Theta(t)$ for system (2) with different $\tau$ as well as $I_k(0) = 0.2, k = 2, 3, 4, 5, I_k(0) = 0$ for the other $k$, $\lambda = 1, \mu = 0.1$ and $R_0 = 3.2051$.

## 3. Analysis of the *SEIRS* model with time delays

### 3.1. The *SEIRS* model

Since some diseases confer temporary immunity, the recovered nodes cycle back into the susceptive class after an immune period. Let $\omega$ be furthermore the immune period of the recovered node, and the recovered node cycles back into the susceptive class after an immune period $\omega$. Denote that $\sigma = \max\{\tau, \omega\}$. Based on the model (2), the relative densities $S_k(t)$, $E_k(t)$, $I_k(t)$ and $R_k(t)$, at the mean-field level, satisfy the following set of coupled different equations when $t > 0$:

$$\begin{cases} \dot{S}_k(t) = -\lambda(k)S_k(t)\Theta(t) + \mu I_k(t-\omega), \\ \dot{E}_k(t) = \lambda(k)S_k(t)\Theta(t) - \lambda(k)S_k(t-\tau)\Theta(t-\tau), \\ \dot{I}_k(t) = \lambda(k)S_k(t-\tau)\Theta(t-\tau) - \mu I_k(t), \\ \dot{R}_k(t) = \mu I_k(t) - \mu I_k(t-\omega) \end{cases} \tag{29}$$

with the normalization condition (3).

Furthermore, we obtain from the third equation and the fourth equation of system (29) that

$$E_k(t) = \lambda(k)\int_{t-\tau}^{t} S_k(s)\Theta(s)ds, R_k(t) = \mu\int_{t-\omega}^{t} I_k(s)ds. \tag{30}$$

Hence, the normalization condition becomes the following mathematical form:

$$S_k(t) + \lambda(k)\int_{t-\tau}^{t} S_k(s)\Theta(s)ds + I_k(t) + \mu\int_{t-\omega}^{t} I_k(s)ds = 1. \tag{31}$$

Obviously, if we discuss the dynamical behaviors of system (29), we just need to discuss the following system:

$$\begin{cases} \dot{S}_k(t) = -\lambda(k)S_k(t)\Theta(t) + \mu I_k(t-\omega), \\ \dot{I}_k(t) = \lambda(k)S_k(t-\tau)\Theta(t-\tau) - \mu I_k(t) \end{cases} \tag{32}$$

with the normalization condition (31).

The initial conditions of system (32) are

$$S_k(\theta) = \varphi_k(\theta), I_k(\theta) = \Psi_k(\theta), \theta \in [-\sigma, 0], \tag{33}$$

which satisfy $S_k(0) + \lambda(k)\int_{-\tau}^{0} S_k(s)\Theta(s)ds + I_k(0) + \mu\int_{-\omega}^{0} I_k(s)ds = 1$. Hence, the normalization condition (31) holds. And, $\Phi_k = (\phi_k(\theta), \Psi_k(\theta), k = m, m+1, \cdots, n-m+1) \in C$ are nonnegative continuous on $[-\sigma, 0]$, $\phi_k(0) > 0, \Psi_k(0) > 0$, and $\zeta(\theta) = 0$ for $\theta = 0$. $C$ denotes the Banach space $C([-\sigma, 0], R^{2(n-m+1)})$ with the norm $\|\omega\| = \left(\sum_{i=m}^{n}\left(|\Psi_i(\theta)|_\sigma^2 + |\varphi_i(\theta)|_\sigma^2\right)\right)^{1/2}$, where $|f(\theta)|_\sigma = \sup_{-\tau\le\theta\le 0}|f(\theta)|$.

### 3.2. The main results for the model

Denote that

$$R_0 = \frac{1}{\mu}\frac{\langle \lambda(k)\varphi(k)\rangle}{\langle k\rangle}, \tag{34}$$

where $\langle f(k)\rangle = \sum_{k=m}^{n} f(k)p(k)$ in which $f(k)$ is a function.

**Theorem 3.1.** *System* (32) *always has a disease-free equilibrium* $E_0$ $(1, 1, \cdots, 1, 0, 0, \cdots, 0)$, *and it has a unique endemic equilibrium* $E_* = \left(S_m^*, S_{m+1}^*, \cdots, S_n^*, I_m^*, I_{m+1}^*, \cdots, I_n^*\right)$ *when* $R_0 > 1$.

**Proof.** Obviously, the disease-free equilibrium $E_0$ of system (32) always exists. Now, we discuss the existence of the endemic equilibrium of system (32). Note that the equilibrium $E_*$ should satisfy:

$$\begin{aligned} &-\lambda k S_k^* \Theta^* + \mu I_k^* = 0, \\ &S_k^* + \lambda(k)\tau S_k^* \Theta^* + I_k^* + \omega\mu I_k^* = 1, \end{aligned} \tag{35}$$

where

$$\Theta^* = \frac{1}{\langle k\rangle}\sum_{k'} \phi(k')p(k')I_k^*. \tag{36}$$

We obtain from (35) that

$$I_k^* = \frac{\lambda k \Theta^*}{\mu + \lambda k\left(1 + \mu\tau + \omega\mu\right)\Theta^*}. \tag{37}$$

Substituting it into Eq. (4), we obtain the self-consistency equality:

$$\Theta^* = \frac{\lambda}{\langle k\rangle}\sum_{k=m}^{n} \phi(k)p(k)\frac{\lambda k\Theta^*}{\mu + \lambda k\left(1 + \mu\tau + \omega\mu\right)\Theta^*} = f(\Theta^*). \tag{38}$$

Note that

$$f'(\Theta^*)\big|_{\Theta^*=0} = \frac{\lambda}{\langle k\rangle}\sum_{k=m}^{n} \phi(k)p(k)\frac{\lambda k\mu}{\left(\mu + \lambda k\left(1 + \mu\tau + \omega\mu\right)\Theta^*\right)^2}\bigg|_{\Theta^*=0} = \frac{\lambda\langle k\phi(k)\rangle}{\mu\langle k\rangle} = R_0 \tag{39}$$

and

$$f''(\Theta^*) = \frac{-2\lambda}{\langle k\rangle}\sum_{k=m}^{n} \phi(k)p(k)\frac{\lambda^2 k^2\mu\left(1 + \mu\tau + \omega\mu\right)\Theta^*}{\left(\mu + \lambda k\left(1 + \mu\tau + \omega\mu\right)\Theta^*\right)^3} < 0. \tag{40}$$

Hence, if $R_0 > 1$, Eq. (38) has a unique positive solution. Consequently, system (32) has a unique endemic equilibrium $E_*\left(S_1^*, S_2^*, \cdots, S_n^*, I_1^*, I_2^*, \cdots, I_n^*\right)$ since (35) and (37) hold.

**Theorem 3.2.** *If $R_0 \le 1$, the disease-free equilibrium $E_0$ of system* (32) *is globally attractive.*

**Proof.** We define a Lyapunov function $V(t)$ as

$$V(t) = \frac{1}{2}\Theta^2(t) + \gamma \int_{t-\tau}^{t} \Theta^2(\mu)d\mu, \tag{41}$$

where $\gamma$ is a constant to be determined. Let $G = \{\phi : \dot{V}(\phi) = 0\}$, and $M$ is the largest set in $G$ which is invariant with respect to system (32). Clearly, $M$ is not empty since $E_0 \in M$. Calculating the derivative of $V(t)$ along the solution of (32), we get

$$\begin{aligned}
\dot{V}(t)\big|_{(3.3)} &= \Theta(t)\left[\frac{1}{\lambda(k)}\rangle \sum_k \varphi(k)p(k)\big(-\lambda k S_k(t-\tau)\Theta(t-\tau) - \mu\Theta(t)\big)\right] + \gamma\Theta^2(t) - \gamma\Theta^2(t-\tau) \\
&\le \Theta(t)\left[\frac{1}{\langle k\rangle}\langle\lambda(k)\phi(k)\rangle\Theta(t-\tau) - \mu\Theta(t)\right] + \gamma\Theta^2(t) - \gamma\Theta^2(t-\tau) \\
&\le \frac{1}{2\langle k\rangle}\langle\lambda(k)\phi(k)\rangle\Theta^2(t) + \frac{1}{2\langle k\rangle}\langle\lambda(k)\phi(k)\rangle\Theta^2(t-\tau) - \mu\Theta^2(t) + \gamma\Theta^2(t) - \gamma\Theta^2(t-\tau) \\
&= \left(\frac{1}{2\langle k\rangle}\langle\lambda(k)\phi(k)\rangle - \mu + \gamma\right)\Theta^2(t) + \left(\frac{1}{2\langle k\rangle}\langle\lambda(k)\phi(k)\rangle - \gamma\right)\Theta^2(t-\tau).
\end{aligned} \tag{42}$$

Note that $R_0 \le 1$ implies $\frac{1}{\langle k\rangle}\langle\lambda(k)\varphi(k)\rangle \le \mu < 0$; if we let $\gamma = \frac{1}{2\langle k\rangle}\langle\lambda(k)\varphi(k)\rangle$, we have from (42) that

$$\dot{V}(t)\big|_{(3.3)} \le \left(\frac{1}{\langle k\rangle}\langle\lambda(k)\phi(k)\rangle - \mu\right)\Theta^2(t) \le 0. \tag{43}$$

It follows from $S_k(t) + E_k(t) + I_k(t) + R_k(t) = 1$ that $M = E_0$. Therefore, by the LaSalle invariance principle [24], the disease-free equilibrium $E_0$ is globally attractive.

**Lemma 3.1.** [28] *Consider the following equation:*

$$\dot{x}(t) = a_1 x(t-\tau) - a_2 x(t), \tag{44}$$

where $a_1, a_2, \tau > 0$; $x(t) > 0$ for $-\tau \le t \le 0$. We have

**i.** if $a_1 < a_2$, then $\lim_{t \to +\infty} x(t) = 0$,

**ii.** if $a_1 > a_2$, then $\lim_{t \to +\infty} x(t) = +\infty$.

**Lemma 3.2.** ([29], p 273–280) *Let $X$ be a complete metric space, $X = X^0 \cup \partial X^0$, where $\partial X^0$, assumed to be nonempty, is the boundary of $X^0$. Assume the $C^0$ – semigroup $T(t)$ on $X$ satisfies $T(x) : X^0 \to X^0$, $T(x) : \partial X^0 \to \partial X^0$ and*

**i.** there is a $t_0$ such that $T(t)$ is compact for $t > t_0$.

**ii.** $T(t)$ is point dissipative in $X$.

**iii.** $\tilde{A}_\partial$ is isolated and has an acyclic covering $M$.

Then, $T(t)$ is uniformly persistent if and only if, for each $M_i \in M$,

$$W^s(M_i) \cap X^0 = \varnothing, \tag{45}$$

where $\tilde{A}_\partial = \bigcup_{x \in A_\partial} \omega(x)$, $\omega(x)$ is the omega limit set of $T(x)$ through $x$, and $A_\partial$ is global attractor of $T_\partial(t)$ in $\partial X^0$ in which $T_\partial(t) = T(t)|_{\partial X^0}$.

**Theorem 3.3.** *For system* (32), *if* $R_0 > 1$, *the disease is uniformly persistent, i.e., there exists a positive constant* $\varepsilon$ *such that* $\lim_{t \to +\infty} \inf I(t) > \varepsilon$, *where* $I(t) = \sum_{k=m}^{n} \phi(k) p(k) I_k(t)$.

**Proof.** Denote that

$$X = \left\{ \left(\overline{S}, \overline{\Psi}\right) : \Psi_k(\theta) \geq 0, for\ \ all\ \ \theta \in [-\zeta, 0], k = m, m+1, \cdots, n \right\}, \tag{46}$$

$$X^0 = \left\{ \left(\overline{S}, \overline{\Psi}\right) : \Psi_k(\theta) > 0, for\ \ some\ \ \theta \in [-\zeta, 0], k = m, m+1, \cdots, n \right\}, \tag{47}$$

and, consequently,

$$\partial X^0 = X / X^0 = \left\{ \left(\overline{S}, \overline{\Psi}\right) : \Psi_i(\theta) = 0, for\ \ all\ \ \theta \in [-\sigma, 0], i \in \{m, m+1, \cdots, n\} \right\}, \tag{48}$$

where $\left(\overline{S}, \overline{\Psi}\right) = (S_m, S_{m+1}, \cdots, S_n, \Psi_m, \Psi_{m+1}, \cdots, \Psi_n)$.

Let $(S_m(t), I_m(t), \cdots, S_n, I_n(t)) = (S_m(t, \omega), I_m(t, \omega), \cdots, S_n(t, \omega), I_n(t, \omega))$ be the solution of (32) with initial function $\omega = (\zeta_m(\theta), \Psi_m(\theta), \cdots, \Psi_n(\theta), \phi_n(\theta))$ and

$T(t)(\omega)(\theta) = (S_m(t+\theta, \omega), I_m(t+\theta, \omega), \cdots, S_n(t+\theta, \omega), I_n(t+\theta, \omega))$, $\theta \in [-\sigma, 0]$. Obviously, $X$ and $X^0$ are positively invariant sets for $T(t)$. $T(t)$ is completely continuous for $t > 0$. Also, it follows from $0 < S_k(t), I_k(t) \leq 1$ for $t > 0$ that $T(t)$ is point dissipative. $E_0$ is the unique equilibrium of system (32) on $\partial X^0$, and it is globally stable on $\partial X^0$, $\tilde{A}_\partial = \{E_0\}$, while $E_0$ is isolated and acyclic.

Finally, the proof will be done if we prove $W^s(E_0) \cap X^0 = \varnothing$, where $W^s(E_0)$ is the stable manifold of $E_0$. Suppose it is not true, then there exists a solution $\left(\overline{S}, \overline{I}\right)$ in $X^0$ such that

$$\lim_{t \to +\infty} \inf S_k(t) = 1, \ \lim_{t \to +\infty} \inf I_k(t) = 0, k = 1, 2, \cdots, n. \tag{49}$$

Since $R_0 > 1$, we may choose $0 < \eta < 1$ such that $\alpha = \eta(\lambda(k)\tau\langle\varphi(k)\rangle + 1 + \mu\omega)$ satisfies $(1 - \alpha)R_0 > 1$. At the same time, there exists a $t_1 > \tau$ such that $I_k(t) < \eta$ for $t > t_1$ due to $\lim_{t \to +\infty} \inf I_k = 0$.

When $t > t_1$, we obtain from (32) that

$$S_k(t) = 1 - \left( I_k(t) + \lambda(k) \int_{t-\tau}^{t} S_k(s)\Theta(s)ds + \mu \int_{t-\omega}^{t} I_k(s)ds \right) \geq 1 - \left(\eta + \lambda(k)\tau\eta\langle\phi(k)\rangle + \mu\omega\eta\right) = 1 - \alpha. \tag{50}$$

On the other hand, for $t > t_1$ we have from (4) and (50) that

$$\begin{aligned}\dot{\Theta}(t) &= \frac{1}{\langle k\rangle}\sum_{k=m}^{n}\phi(k)p(k)\dot{I}_k(t)\\ &= \frac{1}{\langle k\rangle}\sum_{k=m}^{n}\phi(k)p(k)\left[\lambda(k)S_k(t-\tau)\Theta(t-\tau)-\mu I_k(t)\right]\\ &\geq (1-\alpha)\frac{\left\langle\lambda(k)\phi(k)\right\rangle}{\langle k\rangle}\Theta(t-\tau))-\mu\Theta(t)\end{aligned} \tag{51}$$

Note that $(1-\alpha)R_0>1$, and it follows with $(1-\alpha)\frac{\langle\lambda(k)\varphi(k)\rangle}{\langle k\rangle}>\mu$. Hence, we obtain from (51) that $\lim_{t\to+\infty}\Theta(t)=+\infty$ according to Lemma 3.1 contradicts $\lim_{t\to+\infty}\Theta(t)=0$ due to $\lim_{t\to+\infty}I_k(t)=0$. Then, $W^s(E_0)\cap X^0=\varnothing$.

Hence, the infection is uniformly persistent according to Lemma 3.2, i.e., there exists a positive constant $\varepsilon$ such that $\lim_{t\to+\infty}\inf I_k>\varepsilon$ and, consequently, $\lim_{t\to+\infty}\inf I(t)>\sum_{k=m}^{n}p(k)\varepsilon=\varepsilon$. This completes the proof.

In addition, Liu and Zhang discussed a simple *SEIRS* model without delay in [25], and the basic productive number for the model in [25] is $\lambda A/\gamma$, which is consistent with $R_0$ for the model (32) in which $\phi(k)=A$ in this paper.

### 3.3. Numerical simulations for the model

Now, we present the results of numerical simulations. The degree distribution of the scale-free network is $p(k)=Ck^{-\gamma}$, and $C$ satisfies $\sum_{k=1}^{n}p(k)=1$. Here, we set still the maximum degree $n=100$ and the minimum degree $m=1$.

Consider system (32). Let $\varphi(k=ak^{\alpha}/(1+b^{\alpha})$ in which $a=0.5$, $\alpha=0.75$, $b=0.02$ and $\lambda(k)=\lambda k$, and let $\gamma=2.5$ and $\mu=0.06$. **Figures 1–4** show that the dynamic behaviors of system (32) with the initial functions satisfy condition (33) in which $I_k(s)=0.45, k=2,3,4,5$ for $s\in[-\sigma,0]$ and $I_k=0$, $k\neq2,3,4,5$.

Although $R_0$ is irrelative to $\tau$ and $\omega$. **Figures 6** and **7** show that both the delay $\tau$ and $\omega$ have certain influence on the relative density of the infected nodes when $R_0<1$, for example, the faster the relative density of infected nodes converges to zero, the larger $\omega$ gets or the smaller $\tau$ gets. In addition, **Figures 6** and **7** show that the average relative density of the infected nodes $I(t)$ monotonically decreases to zero, whereas the relative density of infected nodes of connectivity $k$ always breaks out first and then decreases to zero; the reason of the phenomenon appears that the spreading network is a scale-free one. Note that **Figures 8** and **9** show that the delay $\tau$ and $\omega$ have much impact on the steady state of density of the infected nodes when $R_0>1$, the density of infected decreases as the delay $\tau$ and $\omega$ increase, which is consistent with the formula (37). We also know from (37) that $I(t)\to0$ as $\omega\to+\infty$ or $\tau\to+\infty$.

Especially, system (29) reduces to the following *SIRS* model [26]:

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$$\begin{cases} \dot{S}_k(t) = -\lambda(k)S_k(t)\Theta(t) + \mu I_k(t-\omega), \\ \dot{I}_k(t) = \lambda(k)S_k(t)\Theta(t) - \mu I_k(t), \\ \dot{R}_k(t) = \mu I_k(t) - \mu I_k(t-\omega). \end{cases} \tag{52}$$

with the normalization condition

$$S_k(t) + I_k(t) + R_k(t) = 1. \tag{53}$$

**Figure 10** shows that when $R_0 > 1$, the quarantine delay $\omega$ can impact the density of infected nodes at the stationary state, and raising the quarantine period will suppress the viruses when $\omega$ is not large enough, which coincides with formula (37). Moreover, there exists periodic oscillation near the endemic equilibrium when $\omega$ is large enough. This is an interesting phenomenon which means that a bifurcation may appear.

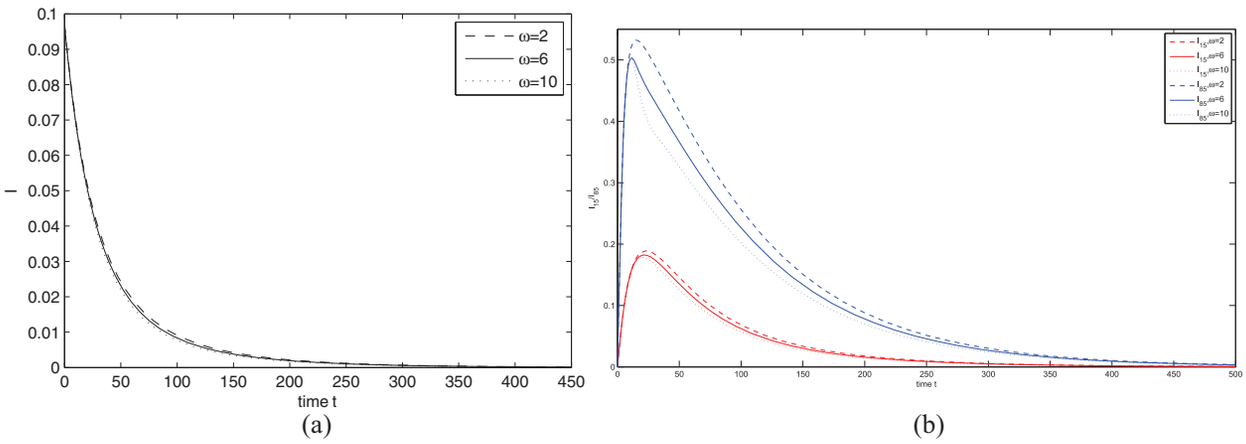


**Figure 6.** (a) Evolutions of $I(t)$ for system (32) with $\tau = 3$, $\lambda(k) = 0.03k$, and $R_0 = 0.8037$. (b) Evolutions of $I_{15}$ and $I_{85}$ for system (32) with $\tau = 3$, $\lambda(k) = 0.03k$, and $R_0 = 0.8037$.

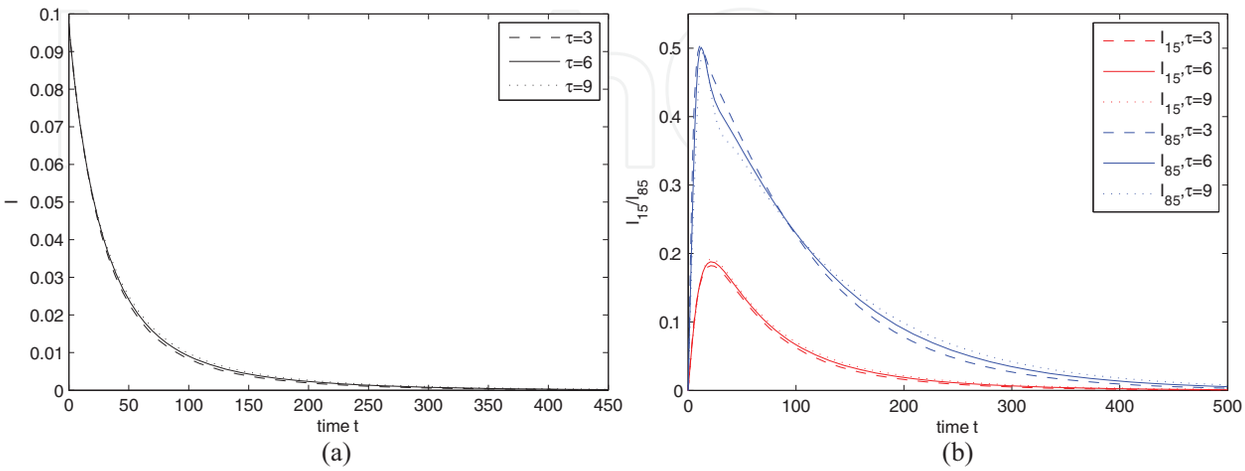


**Figure 7.** (a) Evolutions of $I(t)$ for system (32) with $\omega = 6$, $\lambda(k) = 0.03k$, and $R_0 = 0.8037$. (b) Evolutions of $I_{15}$ and $I_{85}$ for system (32) with $\omega = 6$, $\lambda(k) = 0.03k$, and $R_0 = 0.8037$.

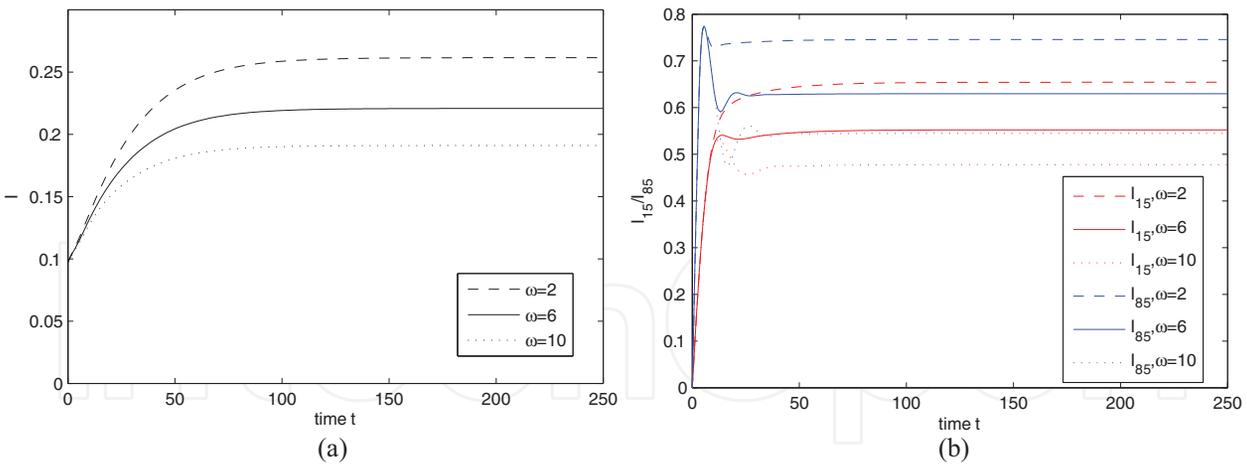


**Figure 8.** (a) Evolutions of $I(t)$ for system (32) with $\tau = 3$, $\lambda(k) = 0.14k$, and $R_0 = 3.7505$. (b) Evolutions of $I_{15}$ and $I_{85}$ for system (32) with $\tau = 3$, $\lambda(k) = 0.14k$, and $R_0 = 3.7505$.

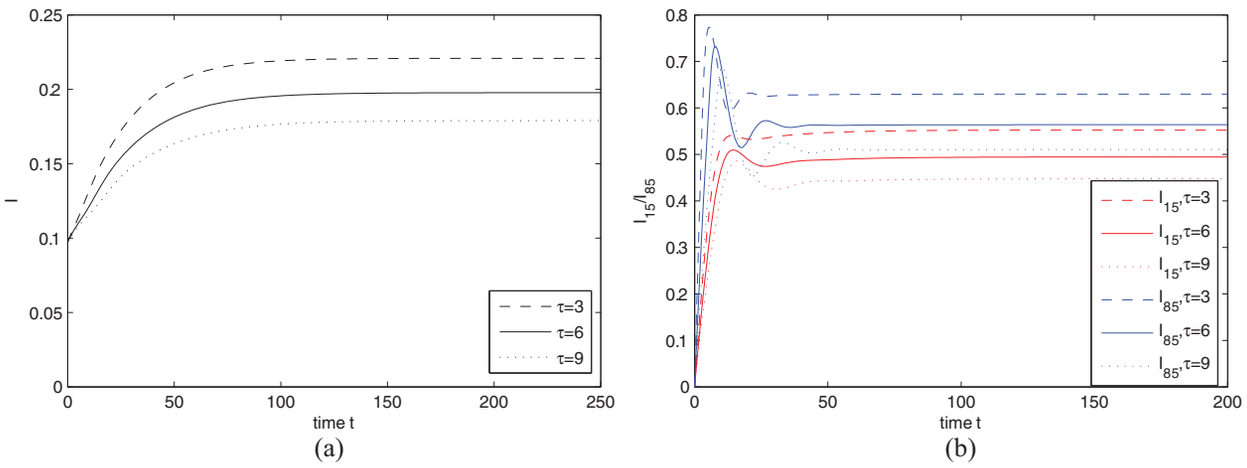


**Figure 9.** (a) Evolutions of $I(t)$ for system (32) with $\omega = 6$, $\lambda(k) = 0.14k$, and $R_0 = 3.7505$. (b) Evolutions of $I_{15}$ and $I_{85}$ for system (32) with $\omega = 6$, $\lambda(k) = 0.14k$, and $R_0 = 3.7505$.

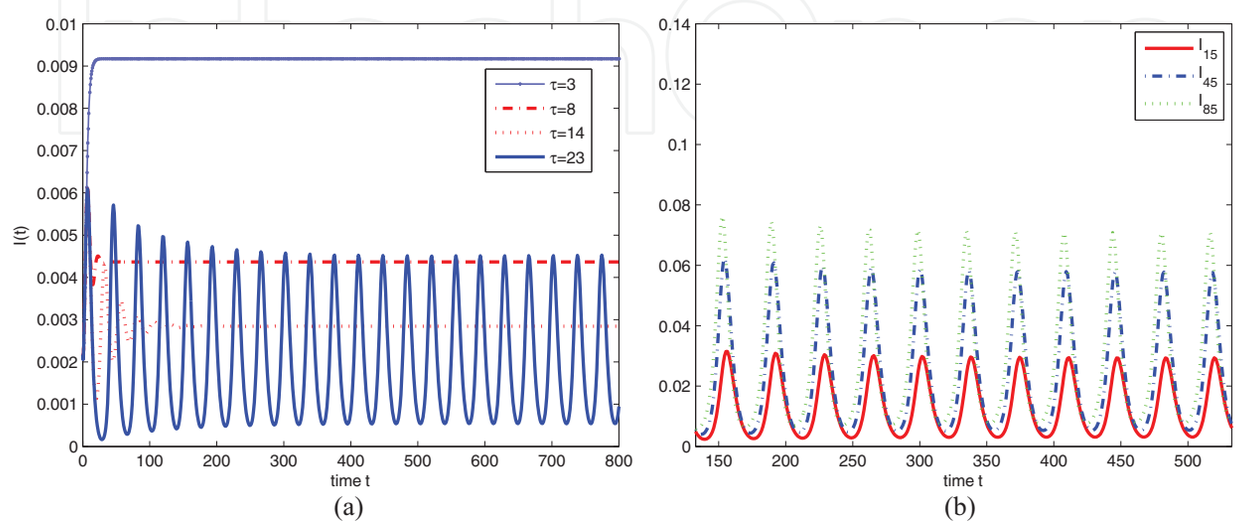


**Figure 10.** (a) Evolutions of $I(t)$, for system (52) with $\omega = 3, 8, 14, 23$, respectively, when $R_0 = 1.3881 > 1$. (b) Evolutions of $I_{15}$, $I_{45}$, $I_{85}$, for system (1) with $\omega = 23$, respectively, when $R_0 = 1.3881 > 1$.

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## 4. Conclusion and discussion

An *SEIR* model with time delay on the scale-free network, which formulated a disease or computer virus transmission with constant latent period, is presented. For *SEIR* model, the basic reproduction number is

$$R_0 = \frac{1}{\mu}\frac{\langle \lambda(k)\phi(k)\rangle}{\langle k\rangle}. \tag{54}$$

When $R_0 \le 1$, there is no outbreak of the disease spreading, and the infection eventually disappears. When $R_0 > 1$, there exists at least one outbreak for the spreading of epidemic, and then $\lim_{t\to+\infty} I_k(t) = 0$ due to global attractivity of $M_0$. If the recovered nodes cycle back into the susceptive class after an immune period, we obtain a *SEIRS* model with two time delays on the scale-free network, which formulated a disease transmission with constant latent and immune periods. For *SEIRS* model, the basic reproduction number is still $R_0$ shown in (54). If $R_0 \le 1$, although the equilibrium $E_0$ is globally stable and the infection eventually disappears, the equilibrium $E_0$ may lose its stability when $R_0 > 1$ and the infection will always exists.

Although $R_0$ is irrelevant to time delays, they influence the dynamical behaviors of the model such as slowing down the speed disappear of disease spreading on network, depressing the density of infected nodes at the stationary state.

In addition, for *SEIRS* model, numerical simulations show that the endemic equilibrium $E_*$ may be globally asymptotically stable under some conditions when $R_0 > 1$ (shown in **Figures 8** and **9**). We would like to mention here that it is interesting but challenging to discuss the stability of equilibrium $E_*$ when $R_0 > 1$.

Furthermore, more and more researchers realize the fundamental role of the stochastic nature of diseases on their dynamics. In order to gain analytical insight into the behavior of the epidemic spreading, we also may extend the models (2) and (29) to the ones with random perturbations, i.e., stochastic differential equation models.

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