

Research Article

Modeling and Analysis of Epidemic Diffusion within Small-World Network

Ming Liu and Yihong Xiao

Department of Management Science and Engineering, Nanjing University of Science and Technology, Jiangsu 210094, China

Correspondence should be addressed to Ming Liu, liumingseu@126.com

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To depict the rule of epidemic diffusion, two different models, the Susceptible-Exposure-Infected-Recovered-Susceptible (SEIRS) model and the Susceptible-Exposure-Infected-Quarantine-Recovered-Susceptible (SEIQRS) model, are proposed and analyzed within small-world network in this paper. Firstly, the epidemic diffusion models are constructed with mean-field theory, and condition for the occurrence of disease diffusion is explored. Then, the existence and global stability of the disease-free equilibrium and the endemic equilibrium for these two complex epidemic systems are proved by differential equations knowledge and Routh-Hurwitz theory. At last, a numerical example which includes key parameters analysis and critical topic discussion is presented to test how well the proposed two models may be applied in practice. These works may provide some guidelines for decision makers when coping with epidemic diffusion controlling problems.

1. Introduction

Disastrous epidemics such as SARS and H1N1 can significantly impact people's life. The outbreak of infections in Europe last year is another recent example. The infection, from a strain of *Escherichia coli*, can lead to kidney failure and death and is difficult to treat with antibiotics. It is now widely recognized that a large-scale epidemic diffusion can conceivably cause many deaths and more people of permanent sequelae, which presents a severe challenge to a local or regional health-care systems.

After an epidemic outbreak, public officials are faced with many critical and complex issues, the most important of which is to make certain how the epidemic diffuses. Actually, many recent research efforts have been devoted to understanding the prevention and control of epidemics, such as those of Wein et al. [1], Wein et al. [2], Craft et al. [3], Kaplan et al. [4, 5], and Matsuura et al. [6]. A very recent research by Shi and Dong [7] formulates and discusses

models for the spread of infectious diseases with variable population sizes and vaccinations on the susceptible individuals. Various mathematical models, such as susceptible-infected (SI), susceptible-infected-recovered (SIR), susceptible-infected-susceptible (SIS), susceptible-infected-recovered-susceptible (SIRS), susceptible-exposure-infective-recovered (SEIR), and susceptible-infective-quarantine-recovered-susceptible (SIQRS), are proposed to analyze and study the general characteristics of epidemic. It is worth mentioning that the major purpose of these papers is to compare the performance of the following two strategies, the traced vaccination (TV) strategy and the mass vaccination (MV) strategy. Furthermore, epidemic diffusion models they adopted are based on traditional compartment models, while complex topological structure of social contact network has not been considered.

As it is well known, a class of network with a topology interpolating between that of lattices and random graphs is proposed by Watts and Strogatz [8]. In these models, a fraction of the links of the lattice is randomized by connecting nodes, with probability p , with any other node. For a range of p , the network exhibits "small world" behavior, where a local neighborhood (as in lattices) coexists with a short average path length (as in random graphs). Analysis of real networks reveals the existence of small worlds in many interaction networks, including networks of social contacts [9]. Recently, attention has been focused on the impact of network topology on the dynamics of the processes running on it with emphasis on the spreading of infectious diseases. For many infectious diseases, a small-world network on an underlying regular lattice is a suitable simplified model for the contact structure of the host population. It is well known that the contact network plays an important role both in the short-term and in the long-term dynamics of epidemic spread [10]. Thus, one of the major motivations for studying the small world network in this paper is to better understand the structure of social contact network, where there is a natural link between the epidemiological modeling and the science of small world network.

Saramäki and Kaski [11] proposed an SIR model for modeling the spreading process of randomly contagious diseases, such as influenza, based on a dynamic small-world network. Masuda and Konno [12] presented a multistate epidemic process based on a complex network. They analyzed the steady states of various multistate disease propagation models with heterogeneous contact rates. Xu et al. [13] presented a modified SIS model based on complex networks, small world and scale free, to study the spread of an epidemic by considering the effect of time delay. Based on two-dimension small-world networks, a susceptible-infected (SI) model with epidemic alert is proposed by Han [14]. This model indicates that the broadcasting of a timely epidemic alert is helpful and necessary in the control of epidemic spreading, and this is in agreement with the general view of epidemic alert. Stone et al. [15] studied the relative effects of vaccinations and avoidance of infected individuals in a susceptible-infected-recovered (SIR) epidemic model on a dynamic small-world network. They derived the critical mobility required for an outbreak to occur as a function of the disease's infectivity, recovery rate, avoidance rate, and vaccination rate. Hsu and Shih [16] focused on the human-to-human transmission of influenza- and investigated the effects of air travel activities on an influenza pandemic in a small-world network. This study also investigated how the small-world properties of an air transportation network facilitate the spread of influenza around the globe. The results show that, as soon as the influenza is spread to the top 50 global airports, the transmission is greatly accelerated. It is worth mentioning that majority of the existing studies relies on different kinds of differential equations. For instance, first-order partial differential equations are used to integrate the age structures; second-order partial differential equations are suitable when a diffusion term

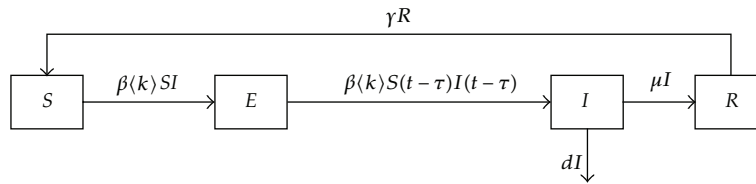


Figure 1: Framework of Model I.

exists; and integral differential equations or differential equations are often used when time delay or delay factors are considered [17].

In this paper, two different models (SEIRS, SEIQRS) based on small-world network are formulated for the spread of infectious diseases. The existence and global stability of the disease-free equilibrium and the endemic equilibrium for these two epidemic systems are proved by differential equations knowledge and Routh-Hurwitz theory. A numerical example, which includes key parameters analysis and critical topic discussion (e.g., medicine resources demand forecasting) is presented to test how well the proposed two models may be applied in practice.

The remainder of the paper is organized as follows. Section 2 presents the SEIRS model considering small-world network effect. Section 3 introduces the SEIQRS model considering small world network effect. Section 4 shows the numerical experiment and discussions. Finally, Section 5 proposes the conclusions.

2. SEIRS Model in Small-World Network (Model I)

2.1. Basic Introduction

For the compartment model of epidemic diffusion in a mature theory, herein, we omit the verbose introduction of the framework process. Readers who interest in such a topic can find more nutrient in [7, 11, 13, 14]. In this section, we consider the situation that infected person will not be quarantined, and we divide people in epidemic area into four groups: susceptible people (S), exposed people (E), infected people (I), and recovered people (R). A survey by Tham [18] shows that part of recovered people who are discharged from the healthcare department will be reinfected again. Thus, considering the small-world network of the social contact, the structure of susceptible-exposure-infective-recovered-susceptible (SEIRS) model is shown as Figure 1.

Notations used in the following sections are specified as follows:

- N : population size in epidemic area,
- $S(t)$: number of susceptible people, $s(t) = S(t)/N$,
- $E(t)$: number of exposed people, $e(t) = E(t)/N$,
- $I(t)$: number of infected people, $i(t) = I(t)/N$,
- $R(t)$: number of recovered people, $r(t) = R(t)/N$,
- $\langle k \rangle$: average degree distribution of small world network,
- β : propagation coefficient of the epidemic,
- γ : reinfected rate of recovered people,

μ : recovered rate,

τ : incubation period of the epidemic,

d : death rate of infected people.

Intuitively, we have the first two equations:

$$\begin{aligned} S(t) + E(t) + I(t) + R(t) &= N, \\ s(t) + e(i) + i(t) + r(t) &= 1. \end{aligned} \quad (2.1)$$

Based on mean-field theory [19], the time-based parameter $s(t)$ meets the following equation from time t to $t + \Delta t$:

$$s(t + \Delta t) - s(t) = -\beta\langle k \rangle s(t)i(t)\Delta t + \gamma r(t)\Delta t. \quad (2.2)$$

Thus, we get

$$\frac{s(t + \Delta t) - s(t)}{\Delta t} = -\beta\langle k \rangle s(t)i(t) + \gamma r(t). \quad (2.3)$$

It can be rewritten as

$$\frac{ds(t)}{dt} = -\beta\langle k \rangle s(t)i(t) + \gamma r(t). \quad (2.4)$$

Similarly, we have the other three ordinary differential equations as follows:

$$\begin{aligned} \frac{de(t)}{dt} &= \beta\langle k \rangle s(t)i(t) - \beta\langle k \rangle s(t - \tau)i(t - \tau), \\ \frac{di(t)}{dt} &= \beta\langle k \rangle s(t - \tau)i(t - \tau) - di(t) - \mu i(t), \\ \frac{dr(t)}{dt} &= \mu i(t) - \gamma r(t). \end{aligned} \quad (2.5)$$

Thus, the SEIRS epidemic diffusion model which considers small-world network effect can be formulated as follows (Model I):

$$\begin{aligned} \frac{ds(t)}{dt} &= -\beta\langle k \rangle s(t)i(t) + \gamma r(t), \\ \frac{de(t)}{dt} &= \beta\langle k \rangle s(t)i(t) - \beta\langle k \rangle s(t - \tau)i(t - \tau), \\ \frac{di(t)}{dt} &= \beta\langle k \rangle s(t - \tau)i(t - \tau) - di(t) - \mu i(t), \\ \frac{dr(t)}{dt} &= \mu i(t) - \gamma r(t). \end{aligned} \quad (2.6)$$

Here, $\beta, \langle k \rangle, \gamma, \mu, d, \tau > 0$. Initial conditions for this epidemic diffusion model are demonstrated as follows:

$$\begin{aligned} i(0) &= i_0 \ll 1, \\ e(0) &= \langle k \rangle i(0), \\ s(0) &= 1 - e_0 - i_0, \\ r(0) &= 0. \end{aligned} \tag{2.7}$$

2.2. Analysis of the SEIRS Model

As to Model I, while such an epidemic diffusion system is stable, the number of people in different groups will be unchanged. Hence, we have $s(t) = s(t - \tau)$, $i(t) = i(t - \tau)$, and we get

$$\frac{de(t)}{dt} = \beta \langle k \rangle s(t) i(t) - \beta \langle k \rangle s(t - \tau) i(t - \tau) = 0, \tag{2.8}$$

$$\frac{di(t)}{dt} = \beta \langle k \rangle s(t) i(t) - di(t) - \mu i(t). \tag{2.9}$$

Equation (2.8) means that the number of exposed people is constant when epidemic diffusion system is stable. As we all know, if an epidemic is wide spread, it should satisfy the following condition:

$$\left. \frac{di(t)}{dt} \right|_{t=0} > 0. \tag{2.10}$$

Together, this equation with (2.9), we can get

$$s_0 > \frac{d + \mu}{\beta \langle k \rangle}. \tag{2.11}$$

Equation (2.11) shows that the spread of epidemic outbreaks only when s_0 meets the above condition. As $s(t) + e(t) + i(t) + r(t) = 1$, combined with (2.6), we get

$$\begin{aligned} \frac{ds(t)}{dt} &= -\beta \langle k \rangle s(t) i(t) + \gamma(1 - s(t) - e(t) - i(t)), \\ \frac{di(t)}{dt} &= \beta \langle k \rangle s(t) i(t) - di(t) - \mu i(t). \end{aligned} \tag{2.12}$$

Let $ds(t)/dt = 0$ and $di(t)/dt = 0$, we can get an obvious equilibrium point for such an epidemic diffusion model as follows:

$$P_1 = (s, i) = (1, 0). \tag{2.13}$$

As (2.13) shows, the number of infected people is zero, which indicates that spread of epidemics in such an area does not happen. All people are susceptible individuals. Herein, we refer to such a point as the disease-free equilibrium point.

In the other side, according to (2.8), the number of exposed people is constant. Thus, combined with (2.12), we can get another equilibrium result as follows:

$$P_2 = (s, i) = \left(\frac{d + \mu}{\beta \langle k \rangle}, \frac{\gamma [\beta \langle k \rangle (1 - e) - (d + \mu)]}{\beta \langle k \rangle (\gamma + d + \mu)} \right). \quad (2.14)$$

Such a result shows that, when epidemic diffusion system is stable, a certain amount of infected people exist in disaster area. Herein, we refer it as the endemic equilibrium point.

Lemma 2.1. *Disease-free equilibrium point P_1 is stable only when $\beta < (d + \mu) / \langle k \rangle$.*

Proof. As $P_1 = (s, i) = (1, 0)$, we can obtain the Jacobi matrix of (2.12) as follows:

$$J_{P_1} = \begin{bmatrix} \frac{\partial \Pi_1}{\partial s} & \frac{\partial \Pi_1}{\partial i} \\ \frac{\partial \Pi_2}{\partial s} & \frac{\partial \Pi_2}{\partial i} \end{bmatrix} = \begin{bmatrix} -\gamma & -\beta \langle k \rangle - \gamma \\ 0 & \beta \langle k \rangle - d - \mu \end{bmatrix}. \quad (2.15)$$

Here, Π_1 and Π_2 are the two differential equations in (2.12). The secular equation for the Jacobi matrix is

$$(\lambda + \gamma)(\lambda - \beta \langle k \rangle + d + \mu) = 0. \quad (2.16)$$

It is easy to get the two characteristic roots for this secular equation, which are $-\gamma$ and $\beta \langle k \rangle - d - \mu$. Based on Routh-Hurwitz stability criterion, when $\beta < (d + \mu) / \langle k \rangle$, real parts of the two characteristic roots are negative. Thus, the disease-free equilibrium point $P_1 = (s, i) = (1, 0)$ is stable only when $\beta < (d + \mu) / \langle k \rangle$. \square

Lemma 2.2. *Endemic equilibrium point P_2 is stable only when $\beta > (d + \mu) / \langle k \rangle (1 - e)$.*

Proof. Similarly as Lemma 2.1, coupling with (2.14), the Jacobi matrix of (2.12) can be rewritten as follows:

$$J_{P_2} = \begin{bmatrix} \frac{\gamma [-\beta \langle k \rangle (1 - e) - \gamma]}{(\gamma + d + \mu)} & -d - \mu - \gamma \\ \frac{\gamma [\beta \langle k \rangle (1 - e) - (d + \mu)]}{(\gamma + d + \mu)} & 0 \end{bmatrix}. \quad (2.17)$$

The secular equation for (2.17) can be expressed as follows:

$$a\lambda^2 + b\lambda + c = 0. \quad (2.18)$$

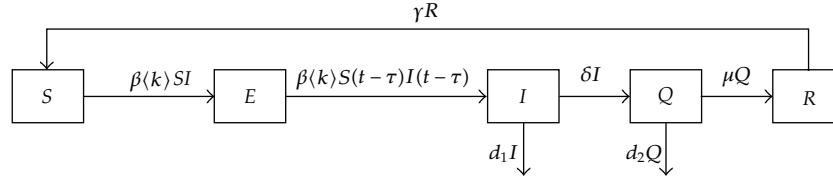


Figure 2: Framework of Model II.

Herein, $a = 1$, $b = \gamma[\beta\langle k \rangle(1 - e) + \gamma]/(\gamma + d + \mu)$, and $c = \gamma[\beta\langle k \rangle(1 - e) - (d + \mu)]$. Based on the quadratic equation theory, such a secular equation contains two characteristic roots λ_1 and λ_2 , and satisfies

$$\lambda_1 + \lambda_2 = -\frac{b}{a} = -\frac{\gamma[\beta\langle k \rangle(1 - e) + \gamma]}{(\gamma + d + \mu)} < 0, \quad (2.19)$$

$$\lambda_1 \cdot \lambda_2 = \frac{c}{a} = \gamma[\beta\langle k \rangle(1 - e) - (d + \mu)]. \quad (2.20)$$

According to Routh-Hurwitz stability criterion, if we want to get two negative characteristic roots λ_1 and λ_2 again, the (2.20) should be constant greater than zero, which means that $\beta > (d + \mu)/\langle k \rangle(1 - e)$ should be satisfied. Thus, only when $\beta > (d + \mu)/\langle k \rangle(1 - e)$, the endemic equilibrium point P_2 is stable. \square

3. SEIQRS Model Considering Small-World Network Effect (Model II)

3.1. Basic Introduction

In this section, the quarantine measure are considered in disaster area, and people in epidemic area are divided into five groups here: susceptible people (S), exposed people (E), infected people (I), quarantined people (Q), and recovered people (R). Framework of epidemic diffusion in this section is illustrated as Figure 2.

Likewise, the SEIQRS epidemic diffusion model which considers small-world network effect can be formulated as follow (Model II):

$$\begin{aligned} \frac{ds(t)}{dt} &= -\beta\langle k \rangle s(t)i(t) + \gamma r(t), \\ \frac{de(t)}{dt} &= \beta\langle k \rangle s(t)i(t) - \beta\langle k \rangle s(t-\tau)i(t-\tau), \\ \frac{di(t)}{dt} &= \beta\langle k \rangle s(t-\tau)i(t-\tau) - d_1 i(t) - \delta i(t), \\ \frac{dq(t)}{dt} &= \delta i(t) - d_2 q(t) - \mu q(t), \\ \frac{dr(t)}{dt} &= \mu q(t) - \gamma r(t). \end{aligned} \quad (3.1)$$

Here, $Q(t)$ stands for number of quarantined people, $q(t) = Q(t)/N$. $s(t) + e(t) + i(t) + q(t) + r(t) = 1$. d_1 is death rate of infected people, d_2 is death rate of quarantined people, and δ is quarantine rate of infected people. Moreover, $\beta, \langle k \rangle, \gamma, \delta, \mu, d_1, d_2, \tau > 0$. Initial conditions for Model II are presented as follows:

$$\begin{aligned} i(0) &= i_0 \ll 1, \\ e(0) &= \langle k \rangle i(0), \\ s(0) &= 1 - e_0 - i_0, \\ q(0) &= 0, \\ r(0) &= 0. \end{aligned} \tag{3.2}$$

3.2. Analysis of the Epidemic Diffusion Model

Similar as Section 2.2, combined with (2.8) and (3.2), the (3.1) can be converted as follows:

$$\begin{aligned} \frac{ds(t)}{dt} &= -\beta \langle k \rangle s(t)i(t) + \gamma [1 - s(t) - e(t) - i(t) - q(t)], \\ \frac{di(t)}{dt} &= \beta \langle k \rangle s(t)i(t) - (d_1 + \delta)i(t), \\ \frac{dq(t)}{dt} &= \delta i(t) - (d_2 + \mu)q(t). \end{aligned} \tag{3.3}$$

Let $ds(t)/dt = 0$, $di(t)/dt = 0$ and $dq(t)/dt = 0$, two equilibrium points for Model II can be solved and shown as follows:

$$\begin{aligned} P_3 &= (s, i, q) = (1 - e, 0, 0), \\ P_4 &= (s, i, q) = \left(\frac{d_1 + \delta}{\beta \langle k \rangle}, B, \frac{\delta}{d_2 + \mu} B \right). \end{aligned} \tag{3.4}$$

Herein, $B = \gamma [\beta \langle k \rangle (1 - e) - (d_1 + \delta)] (d_2 + \mu) / \beta \langle k \rangle [(d_1 + \delta + \gamma)(d_2 + \mu) + \gamma \delta]$, P_3 is the disease-free equilibrium point, and P_4 is the endemic equilibrium point.

Lemma 3.1. Disease-free equilibrium point P_3 is stable only when $\beta < (d_1 + \delta) / \langle k \rangle (1 - e)$.

Lemma 3.2. Endemic equilibrium point P_4 is stable only when $\beta > (d_1 + \delta) / \langle k \rangle (1 - e)$.

The proof procedure for Lemmas 3.1 and 3.2 is similar as in Section 2. Thus, it is trivial to do the work again.

Remark 3.3. From the above four lemmas, we can get the first conclusion that threshold of the epidemic diffusion depends on some key parameters, such as average degree distribution of the small-world network, recovered rate, death rate of infected people, and also a number of exposed people when the system is stable.

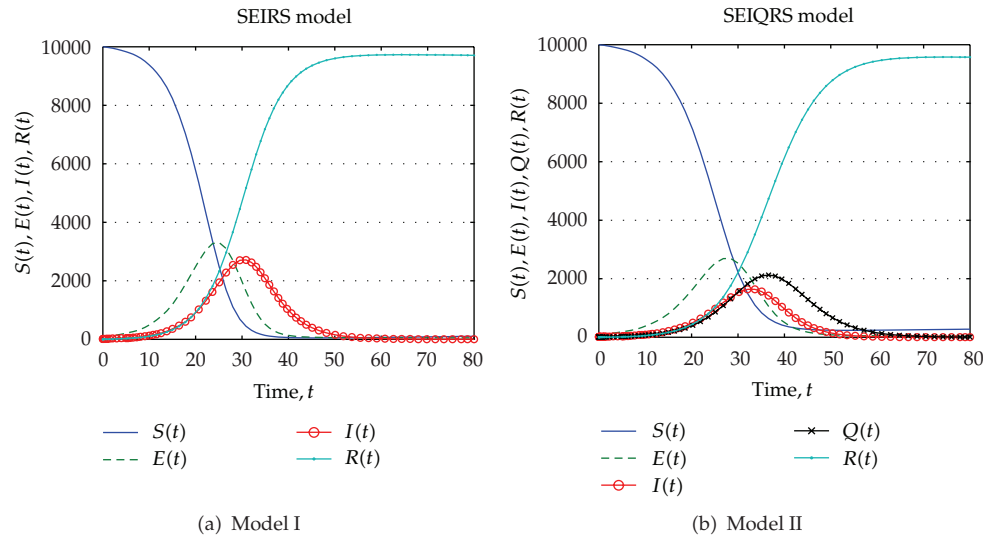


Figure 3: Numerical simulation for Model I and Model II.

4. Numerical Simulation and Discussion

4.1. Numerical Experiment

In this section, we take a numerical simulation to test how well the proposed two models may be applied in practice. The initial values of parameters in the proposed epidemic diffusion models are acquired from [20] and by interviews with public health care administrative personnel, which are given as follows: $\beta = 2 \times 10^{-5}$, $\langle k \rangle = 6$, $\gamma = 2 \times 10^{-4}$, $\delta = 0.3$, $\mu = 0.2$, $d = d_1 = 5 \times 10^{-3}$, $d_2 = 1 \times 10^{-3}$, $\tau = 5$ (day), $N = 10^4$, and $i(0) = 1 \times 10^{-3}$. We use MATLAB 7.0 mathematical solver together with Runge-Kutta method to simulate these two models. The tests are performed on an Intel(R) Core(TM) i3 CPU 2.4 GHz with 2 GB RAM under Microsoft Windows XP. Figure 3 is the numerical simulation of these two epidemic models. The curves respectively represent the different groups of people over time.

As Figure 3 shows, both infected curves $I(t)$ in Model I and Model II exhibit similar trends, namely, the number of infected people will first increase along with the spreading the epidemic, and then it will decrease after the epidemic is brought under control. Threshold value of epidemic diffusion exists in both Model I and Model II. The rush of $I(t)$ is around on the 32-33 day. It seems that the quarantine measure does not work in our example. On the contrary, by comparing Figure 3(a) with Figure 3(b), one can observe that the number of infected people in Model II is way below it in Model I, suggesting that the quarantine measure will significantly reduce the infectivity in the disaster area. Similarly, trend can be found in $E(t)$ curve. We conclude all these result owe to the quarantine measure in Model II.

Based on the theory analysis in Sections 2 and 3, one can observe that some factors, such as β and $\langle k \rangle$, are key parameters in epidemic diffusion system. Herein, we present a short sensitivity analysis for them. Holding all the other parameters fixed as given in the numerical example, except that β takes on four different values ranging from $\beta = 2 \times 10^{-5}$ to 8×10^{-5} with an increment of 2×10^{-5} , Figure 4 shows that number of infected people changed over time. As Figure 4 shows, one can observe that, no matter in Model I or Model II, there

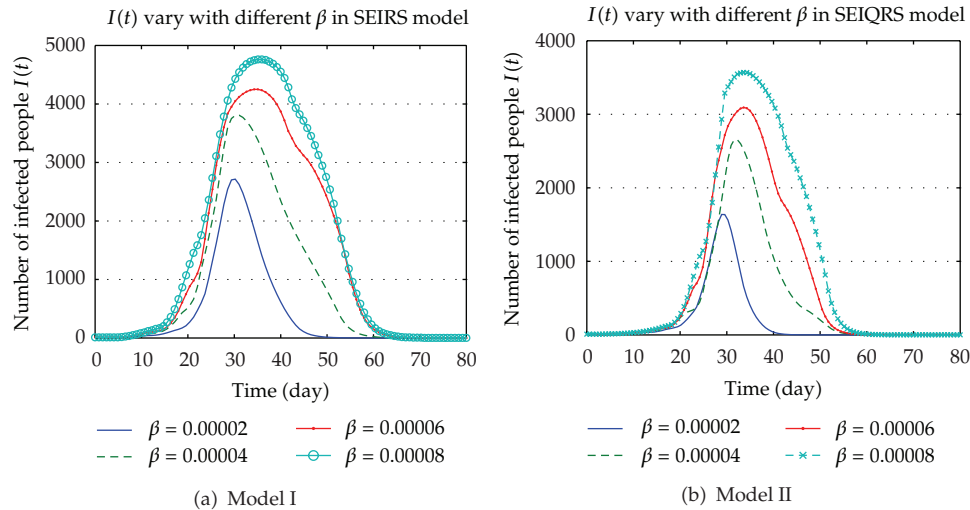


Figure 4: Number of infected people with different β .

almost have no differences among these curves in the first 15–20 days. However, differences is obvious in the following days. The larger initial size of β is, the faster increments speed is. Figure 4 tells us that controlling the propagation coefficient is an effective way to prevent the spread of the epidemic. By comparison with Figures 4(a) and 4(b), a number of infected people in Model II is also way below it in Model I, such result enlightens us again that the quarantine measure is an important factor in epidemic controlling.

It holds all the other parameters are fixed as given in the numerical example, except that $\langle k \rangle$ takes on four different values ranging from 4 to 10 with an increment of 2. Figure 5 shows that the number of infected people is changed over time. Similarly, no matter in Model I or in Model II, a number of infected people shows a positive proportional to the parameter $\langle k \rangle$. Figure 5 transfers an important information, that is, self-quarantine and decreasing the contact with people around are effective strategies for controlling epidemic diffusion. Such a conclusion explains that why Chinese government implements a series of strict quarantine measures during the SARS period.

4.2. A Critical Topic Discussion

The purpose of dynamic analysis of epidemic diffusion models considering small-world network effect in this paper is to depict the epidemic diffusion rule and provide guidance for the emergency management practice. One critical topic of emergency management practice is to forecast the medicine resources demand. However, it is often difficult to predict the actual demand based on historical data (for many events, the historical data may not even exist). Moreover, before 2003, when SARS hit Asia, such an operation in some parts of China has always been done unsystematically (based on the decision maker's experience), which leads to stock-out or surplus phenomenon happening occasionally.

Based on dynamic analysis of epidemic diffusion models in the above sections, herein, we are going to discuss how to forecast the time-varying demand in disaster area. Let $D(t)$ represents demand for medicine resources in disaster area at time t . Obviously, the more

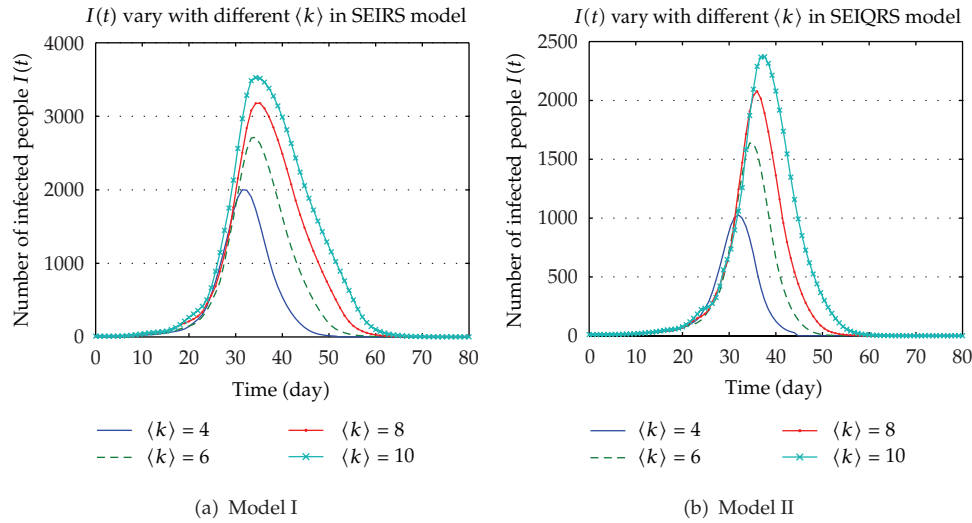


Figure 5: Number of infected people with different $\langle k \rangle$.

people infected, the more resources would be demanded, which can be rewritten as

$$D(t) \propto f[I(t) + Q(t)]. \tag{4.1}$$

Furthermore, each infected person or quarantined person should be cured for certain time (the cure cycle), for example, 30 days. During the cure cycle, demand of medicine resources for each person may shows a property of nonlinearity. Here, we use a function $\varphi(t)$ to represent it. Hence, the total demand of medicine resources for each infected/quarantined person is

$$\varphi = \int_0^c \varphi(t) dt, \tag{4.2}$$

where c is the cure cycle.

To Model I, average demand for medicine resources at time t can be formulated as

$$D_I(t) = \frac{I(t) \cdot \varphi}{c} = \frac{N}{c} \int_0^c \varphi(t) dt \int_0^t \beta \langle k \rangle s(t - \tau) i(t - \tau) - di(t) - \mu i(t) dt. \tag{4.3}$$

Hence, we get

$$\frac{dD_I(t)}{dt} = \Lambda [\beta \langle k \rangle s(t - \tau) i(t - \tau) - di(t) - \mu i(t)], \tag{4.4}$$

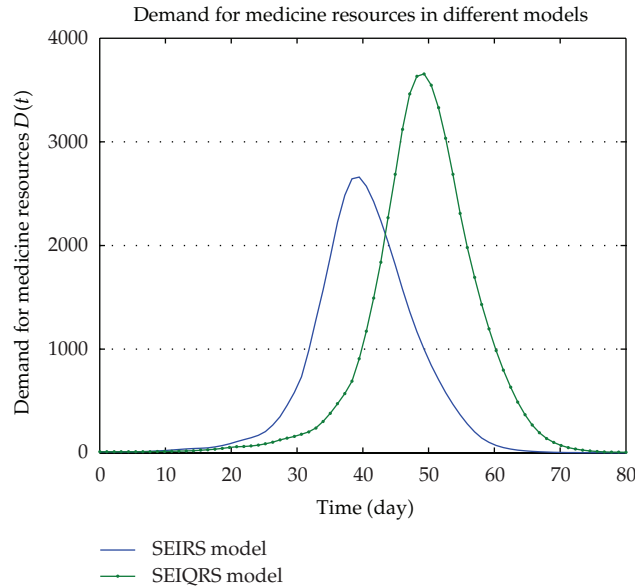


Figure 6: Demand for medicine resources in different models.

where Λ is a constant. Similarly, to Model II, we have

$$\begin{aligned}
 D_{II}(t) &= \frac{[I(t) + Q(t)] \cdot \varphi}{c} \\
 &= \frac{N}{c} \int_0^c \varphi(t) dt \left(\int_0^t \beta(k) s(t-\tau) i(t-\tau) - d_1 i(t) - \delta i(t) dt + \int_0^t \delta i(t) - d_2 q(t) - \mu q(t) dt \right),
 \end{aligned} \tag{4.5}$$

$$\frac{dD_{II}(t)}{dt} = \Lambda [\beta(k) s(t-\tau) i(t-\tau) - d_1 i(t) - d_2 q(t) - \mu q(t)]. \tag{4.6}$$

To depict the variation trend of demand for medicine resources, here, we let $\Lambda = 1$. Such an operation will not affect the final result. According to (4.4) and (4.6), holding all the parameters fixed as given in the above numerical example, one can get the demand for medicine resources by these two models as Figure 6 shows.

As Figure 6 shows, variation trend of demand for medicine resources presents an obvious three different stages. At the first stage (e.g., 0–15 days), epidemic has just outbreak, and it has not yet caused a widespread diffusion. Such a period is the best rescue time. Demand for medicine resources in this period are kept in low level. Hence, medicine resources inventory in local health departments should be distributed to infected people as quickly as possible. If the rescue opportunities in first stage are missed, epidemic will cause a widespread diffusion in the following time, which brings us to the second stage. In such a stage (e.g., 15–70 days), demand for medicine resources is time varying, resources distribution program in such a stage should be varied over time correspondingly. All these changes make the emergency work much more trouble. At the third stage (e.g., 70–more days), demand for medicine resources shows stable again. The inventory of medicine

resources in local health departments should be replenished at this period; meanwhile, a fraction of medicine resources should be allocated to the remaining infected areas.

5. Conclusions

In this paper, we analyze two different epidemic models (SEIRS, SEIQRS) considering small-world network effect, and we prove the global stability of the disease-free equilibrium and the endemic equilibrium for them. A numerical example, which includes key parameters analysis and critical topic discussion, is presented to test how well the proposed two models may be applied in practice. The novelty of our model against the existing works in literature is characterized by the following aspects

(1) While most research on epidemic diffusion studies a compartment model taking no consideration the complexity of social contact network, the models proposed in this paper address the small world network effect in match of the course of an epidemic diffusion.

(2) Based on the theory analysis and numerical simulation, epidemic diffusion rules are depicted, and a series of suggestions for emergency management practice is presented. These methods remarkably outperform the traditional measurements and will be much more suitable for real operations.

As the limitations of the models, they are developed in geographic area where an epidemic disease has been spreading and does not consider possible cross area diffusion between two or more geographic areas. We assume that once an epidemic outbreaks, the government has effective means to separate the epidemic areas so that cross-area spread can be basically prevented. However, this cannot always be guaranteed in reality.

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