Chapter 2
Epidemic Dynamics Modeling and Analysis

Disastrous epidemic such as SARS, H1N1, or smallpox released by some terrorists can significantly affect people's life. The outbreak of infections in Europe in 2011 is another example. The infection, from a strain of *Escherichia coli*, can lead to kidney failure and death and is difficult to treat with antibiotics. A recent example of epidemic outbreak was the 2014–2015 Ebola pandemic in West Africa, which infected approximately 28,610 individuals and approximately 11,300 lives were lost in Guinea, Liberia, and Sierra Leone. It is now widely recognized that a large-scale epidemic diffusion can conceivably cause many deaths and more people of permanent sequela, which presents a severe challenge to the local or regional health-care systems. When an epidemic outbreaks, public officials face with many critical and complex issues, the most important of which is to make certain how the epidemic diffuses. This is the focus of this chapter.

2.1 Epidemic Dynamics in Anti-bioterrorism System

2.1.1 Introduction

Bioterrorism is the intentional use of harmful biological substances or germs to cause widespread illness and fear. It is designed to cause immediate damage and release dangerous substances into the air and surrounding environment. Because it would not usually be signaled by an explosion or other obvious cause, a biological attack may not be recognized immediately and may take local health care workers time to discover that a disease is spreading in a particular area.

Over the past few years, the world has been growing increasingly concerned about the threat that bioterrorists pose to societies, especially after the September 11 attacks and the fatal delivery of anthrax via the US Mail in 2001. Henderson [1] pointed out that the two most feared biological agents in a terrorist attack were smallpox and anthrax. Radosavljević and Jakovljević [2] proposed that biological

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attacks can cause an epidemic of infectious disease. Thus, epidemiological triangle chain models can be used to present these types of epidemic. Bouzianas [3] presented that the deliberate dissemination of Bacillus anthracis spores via the US mail system in 2001 confirms their potential use as a biological weapon for mass human casualties. This dramatically highlights the need for specific medical countermeasures to enable the authorities to protect individuals from a future bioterrorism attack.

Actually, many recent research efforts have been devoted to understanding the prevention and control of epidemics, such as those of Wein et al. [4], Wein et al. [5], Craft et al. [6], Kaplan et al. [7, 8], Mu and Shen [9], Hiroyuki et al. [10], Tadahiro et al. [11], Michael et al. [12]. Various mathematical models have been proposed to analyze and study the general characteristics of each epidemic, such as SI, SIR, SIS, SIRS, SEI, SEIR, and others. It is worth mentioning that the major purpose of these articles is to compare the performance of the following two strategies, the traced vaccination (TV) strategy and the mass vaccination (MV) strategy. Furthermore, the epidemic diffusion models which they adopted are based on the traditional compartment model, while the complex topological structure of the social contact network is not considered.

As is well known, a class of network with a topology interpolating between that of lattices and random graphs is proposed by Watts and Strogatz [13]. 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 [14]. 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 in both the short term and the long term dynamics of epidemic spread [15]. Thus, one of the major motivations for studying the complex network in this work is to better understand the structure of social contact network, because there is a natural link between the epidemiological modeling and the science of complex network.

Jari and Kimmo [16] propose an SIR model for modeling the spreading process of randomly contagious diseases, such as influenza, based on a dynamic small-world network. A study by Masuda and Konno [17] presents a multi-state epidemic process based on a complex network. They analyze the steady states of various multi-state disease propagation models with heterogeneous contact rates. In many models, heterogeneity simply decreases epidemic thresholds. Xu et al. [18] present 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 [19]. This model indicates that the broadcasting of a timely epidemic alert is helpful and necessary in the control of epidemic spreading, and is in agreement with the general view of epidemic alert. Shi et al. [20] propose a new

susceptible-infected-susceptible (SIS) model with infective medium. The dynamic behaviors of the model on a homogeneous network and on a heterogeneous scale-free network are considered respectively. Furthermore, it is shown that the immune density of nodes depends not only on the infectivity between individual persons, but also on the infectivity between persons and mosquitoes. Zhang and Fu [21] study the spreading of epidemics on scale-free networks with infectivity which is nonlinear in the connectivity of nodes. The result shows that nonlinear infectivity is more appropriate than a constant or a linear one. With unit recovery rate and nonlinear irrational infectivity, the epidemic threshold is always positive.

As mentioned in Craft et al. [6], deterrence is not a reliable strategy to against the terrorists, and it is difficult to get the biological agents out of the hands of terrorists before they attack. Our security against a biological attack rests largely on consequence management, i.e., how to ensure the availability and supply of emergency resource so that the loss of life can be minimized and the efficiency of each rescue can be maximized? Considering the relationship between an unexpected bioterror attack and the associated emergency logistics decisions, Liu and Zhao [22] focus on how to deliver emergency resources to the epidemic areas when a bioterror attack is suffered, and propose a mixed-collaborative distribution model for the emergency resources distribution based on the epidemic diffusion rule. A very recent research effort by Wang et al. [23] constructs a multi-objective stochastic programming model with time-varying demand for the emergency logistics network based on epidemic diffusion rule. 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 exists; and integral differential equations or differential equations are often used when time delay or delay factors are considered.

2.1.2 SIQRS Epidemic Diffusion Model

(1) ***Modeling assumptions and notations specification***

To facilitate the model formulation in the following section, three assumptions are specified as follows:

(1) Once a bioterror attack is suffered, the epidemic area can be isolated from other areas to avoid the spread of the disease.
(2) Natural birth and death coefficient of the population in the epidemic area are not considered.
(3) Epidemic diffusion will not be disrupted by itself, which means the infection rate is a constant.

In this section, we consider the situation that epidemic diffusion without incubation period. Notations used in the following model are specified as follows (Table 2.1).

Table 2.1 Parameters specification

Parameters	Specification
N	The whole nodes in the affected area
$S(t)$	Susceptible nodes in the affected area which may become infected. $s(t) = S(t)/N$ represents its density
$I(t)$	Infected nodes in the affected area which are infective with strong infectivity, but have not yet been quarantined. $i(t) = I(t)/N$ represents its density
$Q(t)$	Quarantined nodes in the affected area which have been infected, and have been quarantined. $q(t) = Q(t)/N$ represents its density
$R(t)$	Recovered nodes in the affected area which have recovered from the disease. $r(t) = R(t)/N$ represents its density
$<k>$	Average degree distribution of the network
β	Infection rate of the biological epidemic
γ	Rate of the recovered nodes transform to the susceptible nodes
δ	Rate of the infective nodes which will be found and quarantined
μ	Rate of quarantined nodes transform to the recovered nodes
d_1	Death rate of the infective nodes
d_2	Death rate of the quarantined nodes

Furthermore, $S(t) + I(t) + Q(t) + R(t) = N$, $s(t) + i(t) + q(t) + r(t) = 1$.

(2) ***Model formulation***

Since quarantine is a common response measure when an epidemic outbreaks, here we divide people in the epidemic area into four groups: susceptible people (S), infected people (I), quarantined people (Q) and recovered people (R). The survey by Tham [24] shows that some of the recovered people who are discharged from the emergency department will be re-infected again. Thus, epidemic diffusion model in this section can be illustrated as Fig. 2.1.

For epidemic diffusion, models based on a small-world network match the actual social network much better. A great deal of attention has been paid to studying these models. Therefore, based on the mean-filed theory [25], the time-based parameter $s(t)$ meets the following equation from time t to $t + \Delta t$:

$$s(t + \Delta t) - s(t) = -\beta <k> s(t) i(t) \Delta t + \gamma r(t) \Delta t. \tag{2.1}$$

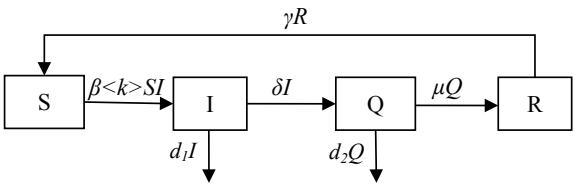


Fig. 2.1 Framework of SIQRS model

Thus, we get:

$$\frac{ds(t)}{dt} = -\beta<k>s(t)i(t) + \gamma r(t). \tag{2.2}$$

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

$$\frac{di(t)}{dt} = \beta<k>s(t)i(t) - d_1 i(t) - \delta i(t). \tag{2.3}$$

$$\frac{dq(t)}{dt} = \delta i(t) - d_2 q(t) - \mu q(t). \tag{2.4}$$

$$\frac{dr(t)}{dt} = \mu q(t) - \gamma r(t). \tag{2.5}$$

Thus, the following SIQRS epidemic diffusion model can be formulated:

$$\begin{cases} \frac{ds(t)}{dt} = -\beta<k>s(t)i(t) + \gamma r(t) \\ \frac{di(t)}{dt} = \beta<k>s(t)i(t) - d_1 i(t) - \delta i(t) \\ \frac{dq(t)}{dt} = \delta i(t) - d_2 q(t) - \mu q(t) \\ \frac{dr(t)}{dt} = \mu q(t) - \gamma r(t) \end{cases}. \tag{2.6}$$

Here, β, $<k>$, γ, δ, μ, d_1, $d_2 > 0$. Initial conditions for this epidemic diffusion model are demonstrated as follows:

$$i(0) = i_0 \ll 1,\ s(0) = s_0 = 1 - i_0,\ q(0) = r(0) = 0.$$

(3) ***Analysis of the epidemic diffusion model***

As is well known, $i(0) = i_0 \ll 1$ and $s(0) = s_0 = 1 - i_0$, are initial percentage of infected people and susceptible people in the population, respectively. Obviously, when wide spread of the epidemic takes place, the following condition should be satisfied:

$$\left.\frac{di(t)}{dt}\right|_{t=0} > 0. \tag{2.7}$$

Considering Eq. (2.3), we have:

$$s_0 > \frac{d_1 + \delta}{\beta<k>}. \tag{2.8}$$

Equation (2.8) means that epidemic diffusion will take place when s_0 meets the above condition. Generally, it is difficult to get the analytic solution for Eq. (2.6). Thus, we consider the stable state of Eq. (2.6). As $s(t) + i(t) + q(t) + r(t) = 1$, and considering Eqs. (2.2), (2.3) and (2.4), then we have:

$$\begin{cases} \frac{ds(t)}{dt} = -\beta<k>s(t)i(t) + \gamma[1 - s(t) - i(t) - q(t)] \\ \frac{di(t)}{dt} = \beta<k>s(t)i(t) - (d_1 + \delta)i(t) \\ \frac{dq(t)}{dt} = \delta i(t) - (d_2 + \mu)q(t) \end{cases}. \tag{2.9}$$

Let $\frac{ds(t)}{dt} = 0$, $\frac{di(t)}{dt} = 0$ and $\frac{dq(t)}{dt} = 0$, we can get an obvious equilibrium point for the epidemic diffusion model I as follows:

$$P_1 = (s,\ i,\ q) = (1,\ 0,\ 0). \tag{2.10}$$

Equation (2.10) shows that both the number of infected people and the number of quarantined people are equal to zero, which indicates that epidemic diffusion in the disaster area does not happen. All people in the area are susceptible at last. Thus, we refer to this as the disease-free equilibrium point.

Furthermore, according to Eq. (2.9), we can get another equilibrium point for the epidemic diffusion system as follows:

$$\begin{aligned} P_2 &= (s, i, q) \\ &= \left(\frac{d_1 + \delta}{\beta<k>}, \frac{\gamma[\beta<k> - (d_1 + \delta)](d_2 + \mu)}{\beta<k>[(d_1 + \delta + \gamma)(d_2 + \mu) + \gamma\delta]}, \frac{\gamma\delta[\beta<k> - (d_1 + \delta)]}{\beta<k>[(d_1 + \delta + \gamma)(d_2 + \mu) + \gamma\delta]}\right). \end{aligned} \tag{2.11}$$

Equation (2.11) shows that when the epidemic diffusion system is stable, a certain amount of infected people and a certain amount of quarantined people exist in the epidemic area. Thus, we refer to this as the endemic equilibrium point.

Lemma 2.1 *Disease-free equilibrium point P_1 in the epidemic diffusion network is stable when $\beta < \frac{d_1+\delta}{<k>}$.*

Proof Considering $P_1 = (s, i, q) = (1, 0, 0)$, we can obtain the Jacobi matrix of Eq. (2.9) as follows:

$$J_{P_1} = \begin{bmatrix} \frac{\partial P_{11}}{\partial s} & \frac{\partial P_{11}}{\partial i} & \frac{\partial P_{11}}{\partial q} \\ \frac{\partial P_{12}}{\partial s} & \frac{\partial P_{12}}{\partial i} & \frac{\partial P_{12}}{\partial q} \\ \frac{\partial P_{13}}{\partial s} & \frac{\partial P_{13}}{\partial i} & \frac{\partial P_{13}}{\partial q} \end{bmatrix} = \begin{bmatrix} -\gamma & -\beta<k> - \gamma & -\gamma \\ 0 & \beta<k> - (d_1 + \delta) & 0 \\ 0 & \delta & -(d_2 + \mu) \end{bmatrix}. \tag{2.12}$$

Here P_{11}, P_{12} and P_{13} represent the three differential equations in Eq. (2.9), respectively. Thus, it is easy to get the secular equation for the Jacobi matrix as follows:

$$(\lambda + \gamma)(\lambda - \beta<k> + d_1 + \delta)(\lambda + d_2 + \mu) = 0. \tag{2.13}$$

Obviously, three characteristic roots for this secular equation are $-\gamma$, $\beta<k> - d_1 - \delta$, and $-d_2 - \mu$. Based on Routh-Hurwiz stability criterion, when $\beta < \frac{d_1+\delta}{<k>}$, real parts of these three characteristic roots will be negative at the same time. Thus, the disease-free equilibrium point $P_1 = (s, i, q) = (1, 0, 0)$ is stable when $\beta < \frac{d_1+\delta}{<k>}$.

Lemma 2.2 *Endemic equilibrium point P_2 in the epidemic diffusion network is stable when $\beta > \frac{d_1+\delta}{<k>}$.*

Proof Similarly as Lemma 2.1, coupling with Eq. (2.11), we can get the Jacobi matrix of Eq. (2.9) again as follows:

$$J_{P_2} = \begin{bmatrix} -\frac{\gamma[\beta<k>-(d_1+\delta)](d_2+\mu)}{[(d_1+\delta+\gamma)(d_2+\mu)+\gamma\delta]} - \gamma & -(d_1+\delta)-\gamma & -\gamma \\ \frac{\gamma[\beta<k>-(d_1+\delta)](d_2+\mu)}{[(d_1+\delta+\gamma)(d_2+\mu)+\gamma\delta]} & 0 & 0 \\ 0 & \delta & -(d_2+\mu) \end{bmatrix}. \tag{2.14}$$

Then, the secular equation for Eq. (2.14) is

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0. \tag{2.15}$$

Here, $a_0 = 1, a_1 = (d_2+\mu)+(A+\gamma), a_2 = (d_2+\mu)(A+\gamma)+(d_1+\delta+\gamma)A$, $a_3 = (d_2+\mu)(d_1+\delta+\gamma)A+\gamma\delta A$, and $A = \frac{\gamma[\beta<k>-(d_1+\delta)](d_2+\mu)}{[(d_1+\delta+\gamma)(d_2+\mu)+\gamma\delta]}$.

Obviously, when $\beta > \frac{d_1+\delta}{<k>}$, we have $A > 0$. Then, we have $a_1 > 0, a_2 > 0, a_3 > 0$. Therefore,

$$\begin{aligned} a_1a_2 - a_0a_3 = {} & (d_2+\mu)(A+\gamma)(d_2+\mu+A+\gamma) \\ & + (d_1+\delta+\gamma)A^2 + \gamma A(d_1+\gamma) > 0 \end{aligned}$$

According to Routh-Hurwiz stability criterion, Eq. (2.11) contains three characteristic roots with negative real part. Thus, the endemic equilibrium point P_2 is stable when $\beta > \frac{d_1+\delta}{<k>}$.

Remark 2.1 From Lemmas 2.1 and 2.2, we have the first conclusion: without consideration of incubation period, threshold of the epidemic diffusion not only depends on topological structure of the small-world network ($<k>$), but also relies on other two key parameters, the quarantined rate (δ) and the death rate of infected people (d_1).

2.1.3 SEIQRS Epidemic Diffusion Model

(1) ***Model formulation***

In this section, we consider the situation that epidemic diffusion with incubation period, and thus, we divide people in the epidemic area into five groups: susceptible people (S), exposed people (E), infected people (I), quarantined people (Q) and recovered people (R). Similarly, epidemic diffusion model in this section can be illustrated as Fig. 2.2.

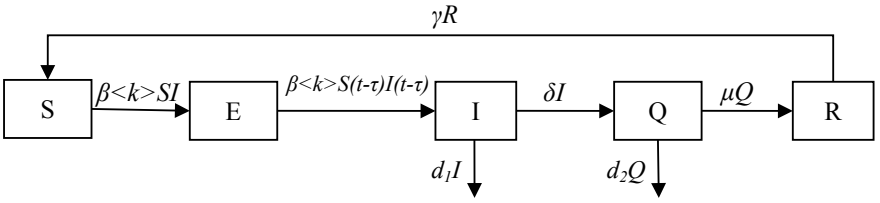


Fig. 2.2 Framework of SEIQRS model

Likewise, the SEIQRS epidemic diffusion model can be formulated as follow:

$$\begin{cases} \frac{ds(t)}{dt} = -\beta<k>s(t)i(t) + \gamma r(t) \\ \frac{de(t)}{dt} = \beta<k>s(t)i(t) - \beta<k>s(t-\tau)i(t-\tau) \\ \frac{di(t)}{dt} = \beta<k>s(t-\tau)i(t-\tau) - d_1 i(t) - \delta i(t) \\ \frac{dq(t)}{dt} = \delta i(t) - d_2 q(t) - \mu q(t) \\ \frac{dr(t)}{dt} = \mu q(t) - \gamma r(t) \end{cases} . \tag{2.16}$$

Here, $E(t)$ stands for the number of exposed people. $e(t) = E(t)/N$. $s(t)+e(t)+i(t)+q(t)+r(t) = 1$. Moreover, β, $<k>$, γ, δ, μ, d_1, d_2,$\tau > 0$. Initial conditions for the epidemic diffusion model are demonstrated as follows:

$$i(0) = i_0 \ll 1,\ e(0) = <k>i(0),\ s(0) = 1 - e_0 - i_0,\ q(0) = r(0) = 0.$$

(2) ***Analysis of the epidemic diffusion model***

Likewise, it is also difficult to get the analytic solution for Eq. (2.16). Thus, we consider the stable state of Eq. (2.16). When the epidemic diffusion system is stable, that means the number of people in each group is unchanged. Then, we have $s(t) = s(t-\tau)$, $i(t) = i(t-\tau)$ and

$$\frac{de(t)}{dt} = \beta<k>s(t)i(t) - \beta<k>s(t-\tau)i(t-\tau) = 0. \tag{2.17}$$

Equation (2.17) means that the number of exposed people is a constant when the epidemic diffusion system is stable. As $s(t) + e(t) + i(t) + q(t) + r(t) = 1$, and considering Eq. (2.16), we have:

$$\begin{cases} \frac{ds(t)}{dt} = -\beta<k>s(t)i(t) + \gamma[1 - s(t) - e(t) - i(t) - q(t)] \\ \frac{di(t)}{dt} = \beta<k>s(t-\tau)i(t-\tau) - (d_1 + \delta)i(t) \\ \frac{dq(t)}{dt} = \delta i(t) - (d_2 + \mu)q(t) \end{cases} . \tag{2.18}$$

Let $\frac{ds(t)}{dt} = 0$, $\frac{di(t)}{dt} = 0$ and $\frac{dq(t)}{dt} = 0$, we get the following two equilibrium points for the SEIQRS epidemic diffusion model:

$$P_3 = (s, i, q) = (1 - e, 0, 0). \tag{2.19}$$

$$P_4 = (s, i, q) = \left(\frac{d_1 + \delta}{\beta <k>}, B, \frac{\delta}{d_2 + \mu} B \right). \tag{2.20}$$

Here, $B = \frac{\gamma[\beta <k>(1-e)-(d_1+\delta)](d_2+\mu)}{\beta <k>[(d_1+\delta+\gamma)(d_2+\mu)+\gamma\delta]}$. Similarly, P_3 is the disease-free equilibrium point and P_4 is the endemic equilibrium point.

Lemma 2.3 *Disease-free equilibrium point P_3 is stable when $\beta < \frac{d_1+\delta}{<k>(1-e)}$.*

Lemma 2.4 *Endemic equilibrium point P_4 is stable when $\beta > \frac{d_1+\delta}{<k>(1-e)}$.*

The proof process of Lemmas 2.3 and 2.4 are similar as introduced in Sect. 2.1.2. Thus, it is trivial to prove Lemmas 2.3 and 2.4.

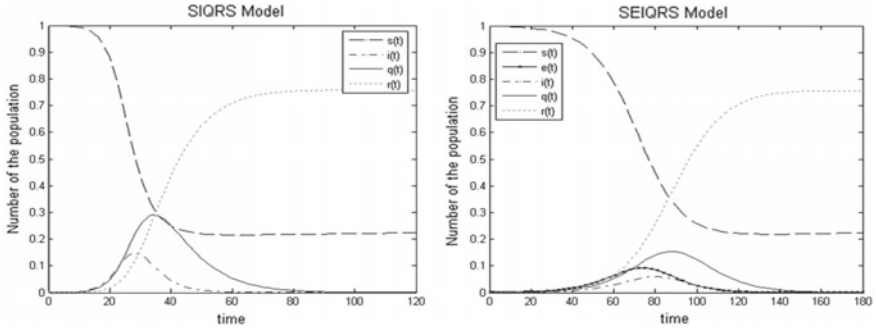
Remark 2.2 From Lemmas 2.3 and 2.4, we get the second conclusion: with the consideration of incubation period, threshold of the epidemic diffusion not only depends on key parameters $<k>$, δ and d_1, but also relies on the number of exposed people when the system is stable.

2.1.4 Computational Experiments and Result Analysis

To test how well the model may be applied in a real world, we exhibit a case study to demonstrate the efficiency of the proposed two different models. To facilitate the calculation process, we assume that a bioterror attack is suffered. The initial values of the parameters in these two epidemic diffusion models are given as follows: $\beta = 10^{-6}$, $<k> = 6$, $\gamma = 2 \times 10^{-4}$, $\delta = 0.3$, $\mu = 0.1$, $d_1 = 5 \times 10^{-3}$, $d_2 = 1 \times 10^{-3}$, $\tau = 5$, $N = 10^5$ and $i(0) = 2 \times 10^{-4}$. We use the MATLAB 7.0 mathematical programming solver to simulate these two models. The tests are performed on an Intel(R) Core(TM) 2 CPU 1.66 GHz with 1.5 GB RAM under Microsoft Windows XP. Figure 2.3 is the numerical simulation of these two epidemic models. The curves respectively represent the different groups of people over time.

From Fig. 2.3, we observe that threshold of the epidemic diffusion exists in both Model I and Model II. Comparing these two Figures, we find the peak of I(t) in Model II appears later than it in Model I. It is worth mentioning that the largest number of infected people in Model II is also smaller than it in Model I. This result is reasonable, because the incubation period is considered in Model II. Thus, the number of infected people in Model II would be divided into two parts. On the other hand, it confirms that incubation time plays an important role in epidemic diffusion network.

During an actual emergency rescue process, the time-based parameter $I(t)$, which represents the number of infected people, is much more concerned. Thus, a short sensitivity analysis of the three key parameters (β, $<k>$ and δ) is conducted in the following.

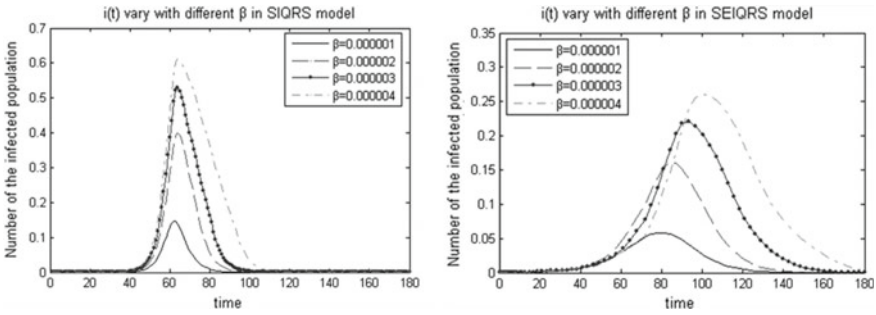


(a) Model I (b) Model II

Fig. 2.3 Diffusion profile of the biological epidemic models

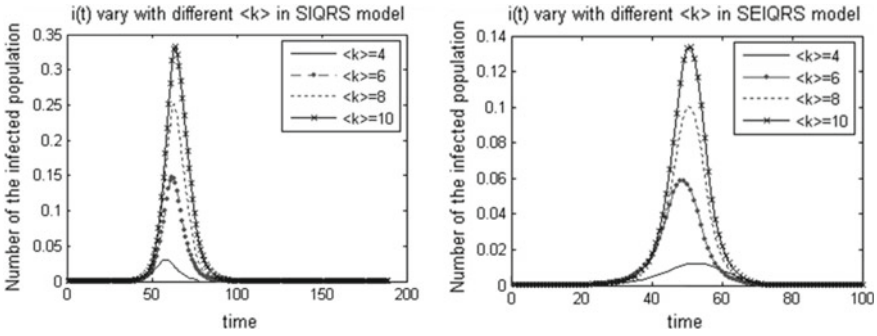
Holding all the other parameters fixed as in the numerical example given above, except that β takes on four different values ranging from 10^{-6} to 4×10^{-6} with an increment of 10^{-6}. Figure 2.4 shows that the number of infected people is changed over time. From this figure, we observe that no matter in Model I or Model II, there almost get no distinguish among these curves in the first 40 days. However, distinguish is obvious in the following days. The larger the initial size of β is, the faster the increments speed is. Note that though initial size of β is varied, peaks of different curves in Model I appear almost at the same time. However, situation in Model II is different. The larger β is, the later the peak appears. This phenomenon enlightens us again that the incubation time is an important factor in an anti-bioterrorism system.

Holding all the other parameters fixed as in the numerical example given above, except that $<k>$ takes on four different values ranging from 4 to 10 with an increment of 2. Figure 2.5 shows that the number of infected people is changed as time goes by. As before, no matter in Model I and Model II, there almost get no distinguish among these curves in the first 40 days. After then, the number of infected people shows a positive proportional to parameter $<k>$. From Fig. 2.5, we get a conclusion that



(a) Model I (b) Model II

Fig. 2.4 Regularity of the infected nodes with different initial size of β

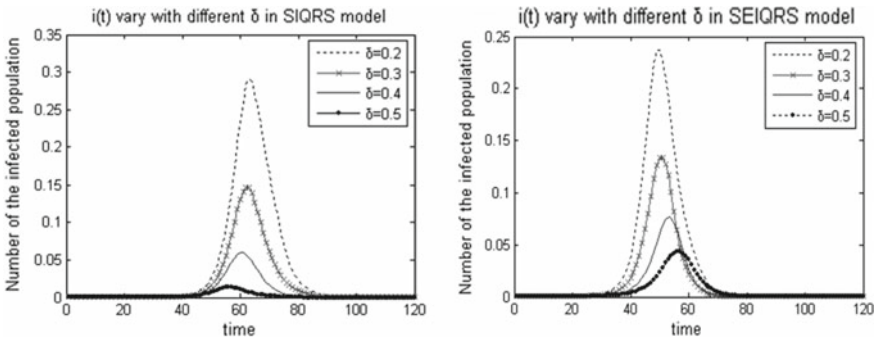


(a) Model I (b) Model II

Fig. 2.5 Regularity of the infected nodes with different initial size of $<k>$

self-quarantine is an effective strategy for controlling the epidemic diffusion. And this is why Chinese government implements a series of strict quarantine measures when SARS outbreaks. Note that peaks of different curves appear almost at the same time in Model II. This is different from Fig. 2.4b.

Similarly, holding all the other parameters fixed as in the numerical example given above, except that δ takes on four different values ranging from 0.2 to 0.5 with an increment of 0.1. Figure 2.6 shows that the number of infected people is changed over time. Exactly same as our expected, we get the similar conclusion as the former two parameters. Moreover, we get the delay phenomenon again as Fig. 2.4b. Figure 2.6 means that to quarantine the infected people as early as possible is also very important during an actual emergency rescue process.



(a) Model I (b) Model II

Fig. 2.6 Regularity of the infected nodes with different initial size of δ

2.2 Epidemic Dynamics Modeling for Influenza

2.2.1 Introduction

The first mathematical model that could be used to describe an influenza epidemic was developed early in the 20th century by Kermack and McKendrick [26]. This model is known as the Susceptible-Infectious-Recovered (SIR) model. To simulate an influenza epidemic, the model is analyzed on a computer and one infected individual (I) is introduced into a closed population where everyone is susceptible (S). Each infected individual (I) transmits influenza, with probability β, to each susceptible individual (S) they encounter. The number of susceptible individuals decreases as the incidence (i.e., the number of individuals infected per unit time) increases. At a certain point the epidemic curve peaks, and subsequently declines, because infected individuals recover and cease to transmit the virus. Only a single influenza epidemic can occur in a closed population because there is no inflow of susceptible individuals. The severity of the epidemic and the initial rate of increase depend upon the value of the Basic Reproduction Number (R_0). R_0 is defined as the average number of new infections that one case generates, in an entirely susceptible population, during the time they are infectious. If $R_0 > 1$ an epidemic will occur and if $R_0 < 1$ the outbreak will die out. The value of R_0 for any specific epidemic can be estimated by fitting the SIR model to incidence data collected during the initial exponential growth phase. The value of R_0 may also be calculated retroactively from the final size of the epidemic. If the SIR model is used, R_0 for influenza is equal to the infectivity/transmissibility of the strain (β) multiplied by the duration of the infectious period. Therefore once the value of R_0 has been obtained, the value of β can be determined.

The SIR model has been used as a basis for all subsequent influenza models. The simplest extension to the SIR model includes demographics; specifically, inflow and outflow of individuals into the population. Analysis of this demographic model shows that influenza epidemics can be expected to cycle, with damped oscillations, and reach a stable endemic level [27]. By modifying the basic SIR model in a variety of ways (e.g., by including seasonality [28, 29]) influenza epidemics can be shown to have sustained cycles. The SIR model has also been extended so that it can be used to represent and/or predict the spatial dynamics of an influenza epidemic. The first spatial-temporal model of influenza was developed in the late 1960s by Rvachev [30]. He connected a series of SIR models in order to construct a network model of linked epidemics. He then modeled the geographic spread of influenza in the former Soviet Union by using travel data to estimate the degree of linkage between epidemics in major cities. In the 1980s, he and his colleagues Baroyan and Longini extended his network model and evaluated the effect of air travel on influenza pandemics [31, 32]. Since then other modeling studies have quantified the importance of air travel on geographic spread [33, 34]. For example, a recent study has modeled the potential for influenza epidemics to move through nine European cities: Amsterdam, Berlin, Budapest, Copenhagen, London, Madrid, Milan, Paris, and Stockholm. The authors estimate that, due to a high degree of connectedness through air travel, it would take

less than a month for an epidemic beginning in any one of these cities to spread to the other eight [33]. Network models have also been use to understand the temporal and spatial synchrony of influenza epidemics within the United States (US) [35].

In this section, SEIRS model based on small-world network is formulated for depicting the spread of infectious diseases. The existence and global stability of the disease-free equilibrium and the endemic equilibrium for the epidemic system is proved by differential equations knowledge and Routh-Hurwiz 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 model may be applied in practice.

2.2.2 SEIRS Model with Small World Network

(1) ***Basic introduction***

For the compartment model of epidemic diffusion is a mature theory, herein we omit the verbose introduction of the framework process. In this section, we consider the situation that infected person will not be quarantined, and 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 [24] shows that part of recovered people who are discharged from the healthcare department will be re-infected again. Thus, considering the small world network of the social contact, the structure of Susceptible–Exposure–Infective–Recovered–Susceptible (SEIRS) model is shown as Fig. 2.7.

Notations used in 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, $r(t) = R(t)/N$.
$<k>$	Average degree distribution of small world network.
β	Propagation coefficient of the epidemic.
γ	Re-infected rate of recovered people.

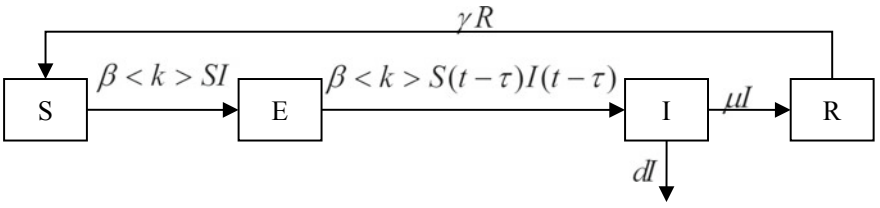


Fig. 2.7 Framework of SEIRS model

μ Recovered rate.
τ Incubation period of the epidemic.
d Death rate of infected people.

Intuitively, we have the first two equations:

$$S(t) + E(t) + I(t) + R(t) = N \tag{2.21}$$

$$s(t) + e(i) + i(t) + r(t) = 1 \tag{2.22}$$

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

$$s(t + \Delta t) - s(t) = -\beta <k> s(t) i(t) \Delta t + \gamma r(t) \Delta t. \tag{2.23}$$

Thus, we get:

$$\frac{s(t + \Delta t) - s(t)}{\Delta t} = -\beta <k> s(t) i(t) + \gamma r(t). \tag{2.24}$$

It can be rewritten as:

$$\frac{ds(t)}{dt} = -\beta <k> s(t) i(t) + \gamma r(t). \tag{2.25}$$

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

$$\frac{de(t)}{dt} = \beta <k> s(t) i(t) - \beta <k> s(t - \tau) i(t - \tau) \tag{2.26}$$

$$\frac{di(t)}{dt} = \beta <k> s(t - \tau) i(t - \tau) - di(t) - \mu i(t) \tag{2.27}$$

$$\frac{dr(t)}{dt} = \mu i(t) - \gamma r(t) \tag{2.28}$$

Thus, the SEIRS epidemic diffusion model which considers small world network effect can be formulated as follows:

$$\begin{cases} \frac{ds(t)}{dt} = -\beta <k> s(t) i(t) + \gamma r(t) \\ \frac{de(t)}{dt} = \beta <k> s(t) i(t) - \beta <k> s(t - \tau) i(t - \tau) \\ \frac{di(t)}{dt} = \beta <k> s(t - \tau) i(t - \tau) - di(t) - \mu i(t) \\ \frac{dr(t)}{dt} = \mu i(t) - \gamma r(t) \end{cases}. \tag{2.29}$$

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

$$\begin{cases} i(0) = i_0 \ll 1 \\ e(0) = <k>i(0) \\ s(0) = 1 - e_0 - i_0 \\ r(0) = 0 \end{cases} \tag{2.30}$$

(2) ***Analysis of the SEIRS model***

As to SEIRS model, while such an epidemic diffusion system is stable, 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:

$$\frac{de(t)}{dt} = \beta <k> s(t)i(t) - \beta <k> s(t-\tau)i(t-\tau) = 0. \tag{2.31}$$

$$\frac{di(t)}{dt} = \beta <k> s(t)i(t) - di(t) - \mu i(t) \tag{2.32}$$

Equation (2.31) means that number of exposed people is a constant when epidemic diffusion system is stable. As we all know, if an epidemic is wide spread, it should satisfy the following condition:

$$\left.\frac{di(t)}{dt}\right|_{t=0} > 0. \tag{2.33}$$

Together this equation with Eq. (2.32), we can get:

$$s_0 > \frac{d+\mu}{\beta <k>}. \tag{2.33}$$

Equation (2.33) shows that the spread of epidemic outbreaks only when s_0 meets the above condition. As $s(t) + e(t) + i(t) + r(t) = 1$, and combine with Eq. (2.29), we get:

$$\begin{cases} \frac{ds(t)}{dt} = -\beta <k> s(t)i(t) + \gamma(1 - s(t) - e(t) - i(t)) \\ \frac{di(t)}{dt} = \beta <k> s(t)i(t) - di(t) - \mu i(t) \end{cases}. \tag{2.34}$$

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

$$P_1 = (s, i) = (1, 0). \tag{2.35}$$

As Eq. (2.35) shows, number of infected people is zero, which indicates that spread of epidemic in such an area does not happened. All people are susceptible individuals. Herein, we refer to such a point as the disease-free equilibrium point.

On the other side, according to Eq. (2.31), number of exposed people is a constant. Thus, combine with Eq. (2.34), we can get another equilibrium result as follows:

$$P_2 = (s, i) = \left(\frac{d+\mu}{\beta<k>}, \frac{\gamma[\beta<k>(1-e) - (d+\mu)]}{\beta<k>(\gamma+d+\mu)} \right) \tag{2.36}$$

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 2.5 *Disease-free equilibrium point P_1 is stable only when $\beta < \frac{d+\mu}{<k>}$.*

Proof As $P_1 = (s, i) = (1, 0)$, we can obtain the Jacobi matrix of Eq. (2.34) as follows:

$$J_{P_1} = \begin{bmatrix} \frac{\partial \Pi_1}{\partial s} & \frac{\partial \Pi_1}{\partial i} \\ \frac{\partial \Pi_2}{\partial s} & \frac{\partial \Pi_2}{\partial i} \end{bmatrix} = \begin{bmatrix} -\gamma & -\beta<k> - \gamma \\ 0 & \beta<k> - d - \mu \end{bmatrix}. \tag{2.37}$$

Here, Π_1 and Π_2 are the two differential equations in Eq. (2.34). The secular equation for the Jacobi matrix is:

$$(\lambda + \gamma)(\lambda - \beta<k> + d + \mu) = 0. \tag{2.38}$$

It is easy to get the two characteristic roots for this secular equation, which are $-\gamma$ and $\beta<k> - d - \mu$. Based on Routh-Hurwiz stability criterion, when $\beta < \frac{d+\mu}{<k>}$, 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 $\beta < \frac{d+\mu}{<k>}$.

Lemma 2.6 *Endemic equilibrium point P_2 is stable only when $\beta > \frac{d+\mu}{<k>(1-e)}$.*

Proof Similarly as Lemma 2.1, coupling with Eq. (2.31), the Jacobi matrix of Eq. (2.29) can be rewritten as follow:

$$J_{P_2} = \begin{bmatrix} \frac{\gamma[-\beta<k>(1-e)-\gamma]}{(\gamma+d+\mu)} & -d-\mu-\gamma \\ \frac{\gamma[\beta<k>(1-e)-(d+\mu)]}{(\gamma+d+\mu)} & 0 \end{bmatrix}. \tag{2.39}$$

The secular equation for Eq. (2.39) can be expressed as follows:

$$a\lambda^2 + b\lambda + c = 0. \tag{2.40}$$

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

$$\lambda_1 + \lambda_2 = -\frac{b}{a} = -\frac{\gamma[\beta<k>(1-e) + \gamma]}{(\gamma+d+\mu)} < 0. \tag{2.41}$$

$$\lambda_1 \cdot \lambda_2 = \frac{c}{a} = \gamma[\beta <k>(1-e) - (d+\mu)]. \tag{2.42}$$

According to Routh-Hurwiz stability criterion, if we want to get two negative characteristic roots λ_1 and λ_2 again, the Eq. (2.42) should be constant greater than zero, which means, $\beta > \frac{d+\mu}{<k>(1-e)}$ should be satisfied. Thus, only when $\beta > \frac{d+\mu}{<k>(1-e)}$, the endemic equilibrium point P_2 is stable.

Remark 2.3 From these two 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 $<k>$, recovered rate μ, death rate of infected people d, also number of exposed people when the system is stable.

2.2.3 *Emergency Demand Base on Epidemic Diffusion Model*

In this section, 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 people infected, the more resources demanded. Thus, it can be rewritten as:

$$D(t) \propto f[I(t)]. \tag{2.43}$$

We assume that each infected person should be cured for a certain time (the cure cycle), e.g. 10 days, and during these days he/she needs for medicine presents a law of decreasing. Hence, the total demand of medicine resources for each infected/quarantined person is:

$$\psi = \int_0^c \varphi(t)dt, \tag{2.44}$$

where $\varphi(t)$ is a decreasing function in the First Quartile. c is the cure cycle. To the SEIRS model, the average demand for medicine resources in time t can be formulated as follows:

$$D_I(t) = \frac{I(t) \cdot \psi}{c} = \frac{N}{c} \int_0^c \varphi(t)dt \int_0^t \beta <k> s(t-\tau)i(t-\tau) - di(t) - \mu i(t)dt. \tag{2.45}$$

Hence we get that:

$$\frac{dD_I(t)}{dt} = \Lambda[\beta < k > s(t-\tau)i(t-\tau) - di(t) - \mu i(t)], \tag{2.46}$$

where Λ is a constant.

2.2.4 Numerical Test

In this section, we take a numerical simulation to test how well the proposed model may be applied in practice. The initial values of relative parameters in the proposed epidemic diffusion models are given as follows: $\beta = 2 \times 10^{-5}$, $< k > = 6$, $\gamma = 2 \times 10^{-4}$, $\delta = 0.3$, $\mu = 0.2$, $d = d_1 = 5 \times 10^{-3}$, $d_2 = 1 \times 10^{-3}$, $\tau = 5$ (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 the propose model. The tests are performed on an Intel(R) Core(TM) i3 CPU 2.4 GHz with 2 GB RAM under Microsoft Windows XP. Figure 2.8 is the numerical simulation of the smallpox epidemic models. The curves respectively represent the different groups of people over time.

From this Figure we can find that there is a threshold value of epidemic diffusion. The rush of the infected curve in such a Model is around on the 32–33 day. Based on the above theory analysis, we find that some factors, such as β and $< k >$, are key parameters in epidemic diffusion system. Herein, we present a short sensitivity analysis for them. Holding all the other parameters fixed as in the numerical example given above, except that β takes on four different values ranging from $\beta = 2 \times 10^{-5}$ to

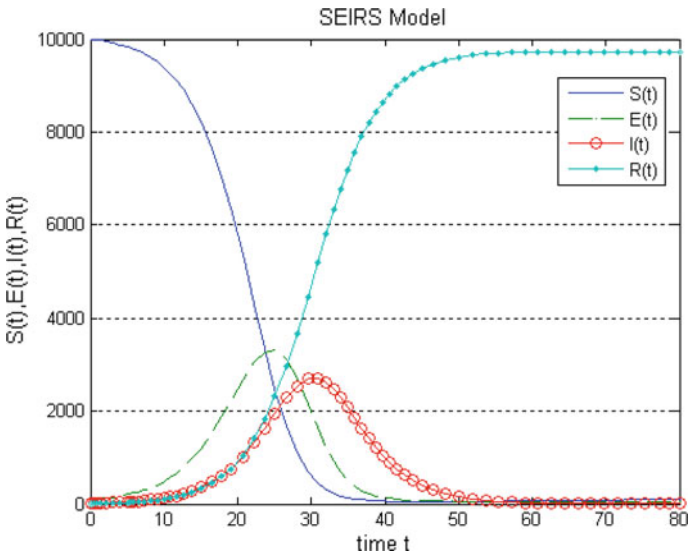


Fig. 2.8 Numerical simulation for SEIRS model

8×10^{-5} with an increment of 2×10^{-5}, Fig. 2.9 shows that number of infected people changed over time. From this figure, we observe that there almost get no distinguish among these curves in the first 15–20 days. However, distinguish is obvious in the following days. The larger initial size of β is, the faster increments speed is. From this figure we know that propagation coefficient controlling is a very important and effective method to prevent the smallpox epidemic diffusion.

Holding all the other parameters fixed as in the numerical example given above, except that $<k>$ takes on four different values ranging from 4 to 10 with an increment of 2. Figure 2.10 shows that number of infected people is changed as time goes by. As before, number of infected people shows a positive proportional to such a parameter $<k>$. From this figure, we know that self-quarantine and decreasing the contact with people around is an effective strategy for controlling epidemic diffusion. Hence, during the SARS period, governments implement a series of strict quarantine measures.

To facilitate the process in the following section, here we given that $\Lambda = 1$ directly. Such an operation will not affect the final compare result. According to Eq. (2.27), holding all the parameters fixed as in the above numerical example, we can get the demand for medicine resources by the proposed model, which are shown in Fig. 2.11.

Form this figure, we can decompose the entire smallpox emergency rescue process into three mutually correlated stages, and we present three corresponding controlling strategies for them.

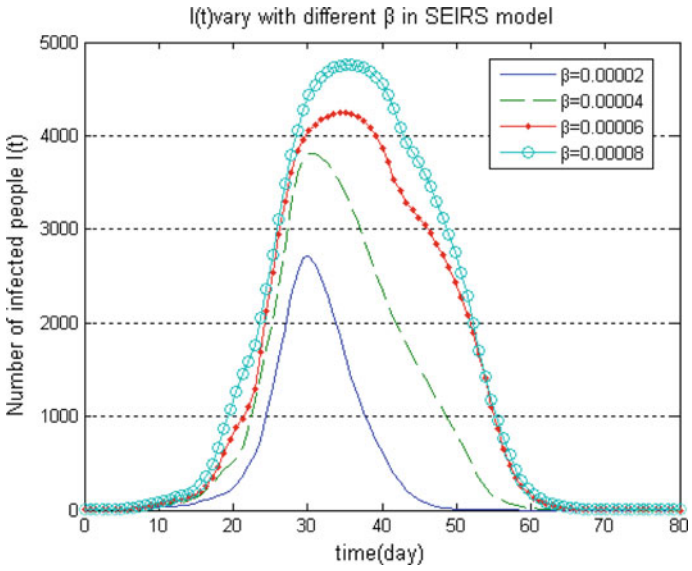


Fig. 2.9 Number of *I(t)* with different β

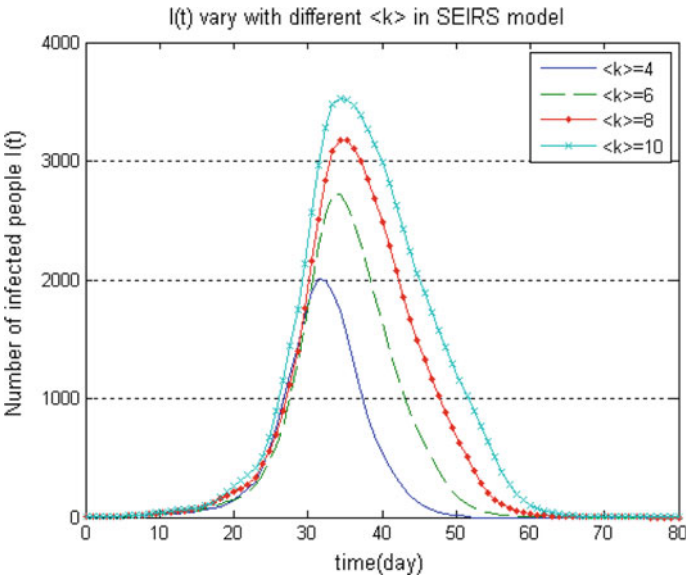


Fig. 2.10 Number of *I(t)* with different $<k>$

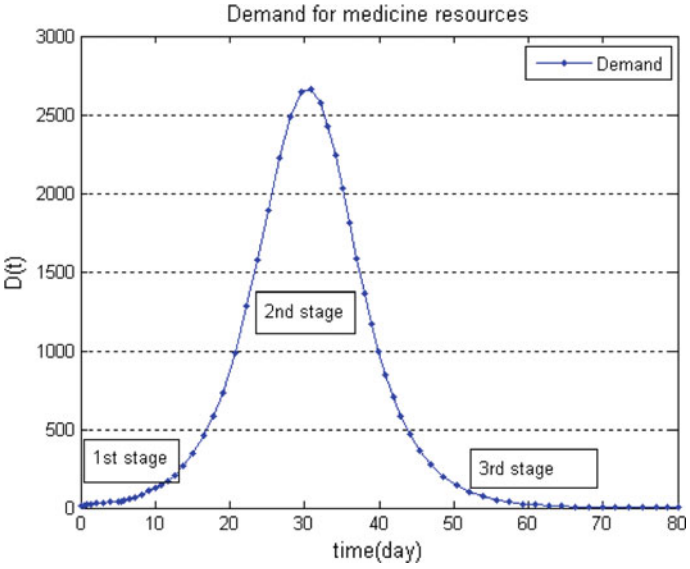


Fig. 2.11 Demand for medicine resources

(1) 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 emergency rescue time. The demand for medicine resources during this period keeps in low level. Hence, medicine resources inventory in local health departments should be distributed to the infected people's hands as quickly as possible.
(2) If we miss the opportunities in the first stage, epidemic would cause a widespread diffusion, which makes us face to the second stage. In such a stage (e.g. 15–70 days), demand for medicine resources is dynamic and time-varying. Thus, resources distribution program in such a stage should also be varied over time.
(3) At the third stage (e.g. 70-more days), epidemic diffusion goes to be stable, and demand for medicine resources shows decreasing. Hence, we can replenish the inventory of medicine resources for these local health departments, and allocate some other medicine resources to the remaining infected areas, simultaneously.

2.3 Epidemic Dynamics Considering Population Migration

2.3.1 Introduction

As mentioned in Rachaniotis et al. [36], a serious epidemic is a problem that tests the ability of a nation to effectively protect its population, to reduce human loss and to rapidly recover. Sometime such a problem may acquire worldwide dimensions. For example, during the period from November 2002 to August 2003, 8422 people in 29 countries were infected with SARS, 916 of them were dead at last for the effective medical resources appeared late. Other diseases, such as HIV, H1N1 can also cause significant numbers of direct infectious disease deaths. Epidemic diffusion is a typical complex dynamic system problem in Gao et al. [37], for we don't know what kind of epidemic outbreaks, when it outbreaks, and how it diffuses. Generally, 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 so that the rescue operation efficiency maximized.

Traditionally, analytical works on epidemic diffusion are concentrated on the compartmental epidemic models of ordinary differential equations [38–42]. In these models, the total population is divided into several independence classes and each class of individuals is closed into a compartment. The sizes of the compartments are large enough and the mixing of members is homogeneous. In other words, the models based on the differential equations are always under the assumption of both homogeneous infectivity and homogeneous connectivity of each individual. However, the traditional models do not consider the population migration among different compartments.

The other stream of related research to our work is on the epidemic diffusion with population migration. For instance, Hethcote [43] proposed that deterministic communicable disease models were initial value problems for a system of ordinary

differential equations, and thus he considered the asymptotic stability for the equilibrium points for models involving temporary immunity, disease-related fatalities, carriers, migration, dissimilar interacting groups, and transmission by vectors. In his work, both susceptible individuals and infected individuals in each population could migrate (only equal rates were considered), which led to different equilibriums. Another model that considers two interacting populations undergoing SIS dynamics was presented in Kribs-Zaleta and Velasco-Hernandex [44]. The authors considered that the two groups may have different values for model parameters especially those dealing with vaccination. Liebovitch and Schwartz [45] proposed that classical disease models always use a mass action term as the interaction between infected and susceptible people in separate patches and they derived the equations when this interaction is a migration of people between patches. Sani and Kroese [46] formulated various mathematical control problems for HIV spread in mobile heterosexual populations. They applied the cross-entropy method to solve these highly multi-modal and non-linear optimization problems, and demonstrated the effectiveness of the method via a range of experiments and illustrated how the form of the optimal control function depends on the mathematical model used for the HIV spread. Yang et al. [47] considered SIR and SIS epidemic models with bilinear incidence and migration between two patches, where infected individuals cannot migrate from one patch to another due to medical screening. They found the thresholds classifying the global dynamics of the models in terms of the model parameters, and they obtained the global asymptotical stability of the disease free and the disease endemic equilibrium. Wolkewitz and Schumacher [48] pointed out that the main limitation of the compartmental models is that several parameters are based on uncertain expert guesses (default values) and are not estimated from the study data. Lee et al. [49] extended the SEIR model to incorporate population migration between cities and investigated the effectiveness of travel restrictions as a control against the spread of influenza.

As a continued work, this section presents an SIS epidemic model with population migration between two cities. We consider unequal migration rates for these two populations and only susceptible individuals can migrate, which is different from the whole existing works.

2.3.2 Epidemic Model with Population Migration

As the compartment model of epidemic diffusion is a mature theory, herein we omit the verbose introduction of the framework process. In this section, we divide people in epidemic areas into two groups: susceptible individuals (S) and infected individuals (I). The transfer diagram of individuals in the epidemic areas can be illustrated as Fig. 2.12.

To smooth the formulation progress of the SIS epidemic diffusion model in the following subsections, some assumptions and parameters are specified as follows:

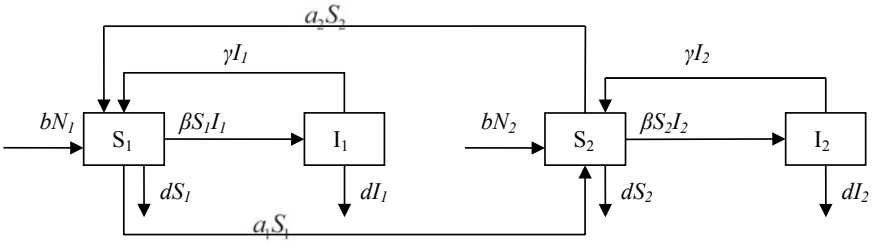


Fig. 2.12 The transfer diagram of SIS model with population migration

(1) The susceptible individuals and the infected individuals in city i at time t are denoted as $S_i(t)$ and $I_i(t)$, respectively. Thus, the total individuals in city i is $N_i(t) = S_i(t) + I_i(t), i = 1, 2$.
(2) b and d are the natural birth rate and the natural death rate, respectively. γ is the recovery rate. β is the propagation coefficient. To facilitate the process in the following sections, we assume that $b = d$. Moreover, disease-related death rate is not considered in this work.
(3) Only the susceptible individuals can migrate in this paper. a_i represents the migrating-out rate of susceptible individuals in city i ($a_i > 0$ for $i = 1, 2$ and $a_1 \neq a_2$).
(4) Using the notation N to represent the total number of the population in these two cities $N = N_1 + N_2$. Note that N is a constant.

Hence, the ordinary differential equations for the SIS epidemic diffusion model can be formulated as:

$$\begin{cases} \frac{dS_1}{dt} = bN_1 - a_1S_1 + a_2S_2 - dS_1 - \beta S_1 I_1 + \gamma I_1 \\ \frac{dI_1}{dt} = \beta S_1 I_1 - \gamma I_1 - dI_1 \\ \frac{dS_2}{dt} = bN_2 + a_1S_1 - a_2S_2 - dS_2 - \beta S_2 I_2 + \gamma I_2 \\ \frac{dI_2}{dt} = \beta S_2 I_2 - \gamma I_2 - dI_2 \end{cases}. \tag{2.47}$$

ODE (2.47) describes the following dynamics of epidemic diffusion among the population groups. (1) The change rate of the susceptible population in both city 1 and city 2 are determined by the entry population, the exiting population, and the losing population who actually gets exposed to the disease and thus is counted towards the class of infected population. The last one is in proportion to the propagation coefficient β, and both of the current mass of the susceptible individuals and the current mass of the infected individuals. (2) The change rate of the infected population is determined by the difference between the entering population, those of the susceptible population who get sick, the exiting population, and the losing population. All parameters β, b, γ, a_1, a_2 are positive and initial conditions for the model are demonstrated as follows:

$$I_1(0) = i_1^0 \ll N,\ I_2(0) = i_2^0 \ll N,\ S_1(0) = s_1^0,\ S_2(0) = N - s_1^0 - i_1^0 - i_2^0. \tag{2.48}$$

2.3.3 *Model Analysis*

(1) **Condition of the epidemic diffusion**

As shown in above, $I_1(0) = i_1^0 \ll N$, $I_2(0) = i_2^0 \ll N$, $S_1(0) = s_1^0$ and $S_2(0) = N - s_1^0 - i_1^0 - i_2^0$ are initial conditions for the proposed model, which symbolize the initial number of susceptible and infected individuals. Then, it is easy to obtain the initial condition for epidemic diffusion, which should satisfy the following premise:

$$\frac{dI_1}{dt}|_{t=0} > 0 \text{ or } \frac{dI_2}{dt}|_{t=0} > 0. \tag{2.49}$$

Taking it into Eq. (2.47), we can obtain the initial condition of the susceptible individuals in city 1 and city 2:

$$s_1^0 > \frac{b+\gamma}{\beta} \text{ or } s_2^0 < N - i_1^0 - i_2^0 - \frac{b+\gamma}{\beta}. \tag{2.50}$$

Equation (2.50) shows that the spread of epidemic only when s_1^0 and s_2^0 meet the above initial conditions.

(2) **Existence of the system equilibrium solution**

Generally, it is difficult to obtain the analytic solution of the Eq. (2.47). To analyze the epidemic diffusion, we consider the stable state of Eq. (2.47). Considering that $b = d$ and expunging S_2, Eq. (2.47) can be rewritten as:

$$\begin{cases} \frac{dS_1}{dt} = -a_1 S_1 + a_2(N - S_1 - I_1 - I_2) - \beta S_1 I_1 + (b+\gamma) I_1 \\ \frac{dI_1}{dt} = \beta S_1 I_1 - (b+\gamma) I_1 \\ \frac{dI_2}{dt} = \beta(N - S_1 - I_1 - I_2) I_2 - (b+\gamma) I_2 \end{cases}. \tag{2.51}$$

Let $\frac{dI_1}{dt} = 0$, we can get $I_1 = 0$ or $S_1 = \frac{b+\gamma}{\beta}$. Similarly, let $\frac{dI_2}{dt} = 0$, we can obtain $I_2 = 0$ or $S_1 + I_1 + I_2 = N - \frac{b+\gamma}{\beta}$. With the partial derivative $\frac{dS_1}{dt} = 0$, $\frac{dI_1}{dt} = 0$ and $\frac{dI_2}{dt} = 0$, we can obtain one equilibrium point of the SIS epidemic diffusion system intuitively when $I_1 = 0$ and $I_2 = 0$:

$$P_1 = (S_1,\ I_1,\ I_2) = \left(\frac{a_2}{a_1 + a_2} N,\ 0,\ 0\right)). \tag{2.52}$$

From Eq. (2.52) we can see that both number of infected individuals in city 1 and city 2 are zero, which indicate that epidemic diffusion in these two cities does not happened, and all the individuals in these two cities are susceptible individuals at last. Herein, we call it disease-free equilibrium point.

When $I_1 = 0$ and $S_1 + I_1 + I_2 = N - \frac{b+\gamma}{\beta}$, we can obtain the second equilibrium point of the SIS epidemic diffusion system:

$$P_2 = (S_1,\ I_1,\ I_2) = \left(\frac{a_2}{a_1} \cdot \frac{b+\gamma}{\beta}, 0,\ N - \frac{a_1 + a_2}{a_1} \cdot \frac{b+\gamma}{\beta}\right). \tag{2.53}$$

From Eq. (2.53), when the SIS epidemic diffusion system is stable, the number of infected individuals in city 1 is zero, and some infected individuals in city 2 still exist. In this condition, we call it the endemic equilibrium point.

Likewise, when $S_1 = \frac{b+\gamma}{\beta}$ and $I_2 = 0$, we can obtain the third equilibrium point of the SIS epidemic diffusion system:

$$P_3 = (S_1,\ I_1,\ I_2) = \left(\frac{b+\gamma}{\beta},\ N - \frac{a_1 + a_2}{a_2} \cdot \frac{b+\gamma}{\beta}, 0\right). \tag{2.54}$$

In line with the above work, when the SIS epidemic diffusion system is stable, the number of infected individuals in city 2 is zero, and some infected individuals in city 1 still exist. So it is called endemic equilibrium point as well.

It is worth mentioning that when $S_1 = \frac{b+\gamma}{\beta}$ and $S_1 + I_1 + I_2 = N - \frac{b+\gamma}{\beta}$, there is $\frac{dS_1}{dt} = (a_2 - a_1) \cdot \frac{b+\gamma}{\beta} \neq 0$ for that $a_1 \neq a_2$. That means, under the conditions of $S_1 = \frac{b+\gamma}{\beta}$ and $S_1 + I_1 + I_2 = N - \frac{b+\gamma}{\beta}$, there is no solution for the simultaneous Equations $\frac{dS_1}{dt} = 0$, $\frac{dI_1}{dt} = 0$ and $\frac{dI_2}{dt} = 0$.

(3) **Stability of the system equilibrium solution**

Lemma 2.7 *Disease-free equilibrium point P_1 in the SIS epidemic diffusion system is stable only when $\beta < \min\{\frac{(a_1+a_2)(b+\gamma)}{a_1 N}, \frac{(a_1+a_2)(b+\gamma)}{a_2 N}\}$.*

Proof Let $P = \frac{dS_1}{dt}$, $Q = \frac{dI_1}{dt}$ and $R = \frac{dI_2}{dt}$, the Jacobi matrix of Eq. (2.51) can be obtained as follows:

$$\begin{aligned} J &= \begin{pmatrix} \frac{\partial P}{\partial S_1} & \frac{\partial P}{\partial I_1} & \frac{\partial P}{\partial I_2} \\ \frac{\partial Q}{\partial S_1} & \frac{\partial Q}{\partial I_1} & \frac{\partial Q}{\partial I_2} \\ \frac{\partial R}{\partial S_1} & \frac{\partial R}{\partial I_1} & \frac{\partial R}{\partial I_2} \end{pmatrix} \\ &= \begin{pmatrix} -a_1 - a_2 - \beta I_1 & b + \gamma - a_2 - \beta S_1 & -a_2 \\ \beta I_1 & \beta S_1 - (b+\gamma) & 0 \\ -\beta I_2 & -\beta I_2 & \beta(N - S_1 - I_1 - 2I_2) - (b+\gamma) \end{pmatrix}. \end{aligned} \tag{2.55}$$

For $P_1 = (S_1,\ I_1,\ I_2) = \left(\frac{a_2}{a_1+a_2}N,\ 0,\ 0\right)$, the Jacobi matrix J can be rewritten as follows:

$$J_{P_1} = \begin{pmatrix} -a_1 - a_2 & b+\gamma-a_2-\frac{a_2N}{a_1+a_2}\beta & -a_2 \\ 0 & \frac{a_2N}{a_1+a_2}\beta-(b+\gamma) & 0 \\ 0 & 0 & \frac{a_1N}{a_1+a_2}\beta-(b+\gamma) \end{pmatrix}.$$

According to the Jacobi matrix J_{P_1}, it is easy to obtain the secular equation of Eq. (2.51):

$$(\lambda+a_1+a_2)(\lambda+b+\gamma-\frac{a_2N}{a_1+a_2}\beta)(\lambda+b+\gamma-\frac{a_1N}{a_1+a_2}\beta)=0. \tag{2.56}$$

The three latent roots of this secular equation are $-a_1-a_2$, $\frac{a_2N}{a_1+a_2}\beta-b-\gamma$ and $\frac{a_1N}{a_1+a_2}\beta-b-\gamma$. Based on Routh-Hurwiz stability criterion, only when $\beta < \frac{(a_1+a_2)(b+\gamma)}{a_1N}$ and $\beta < \frac{(a_1+a_2)(b+\gamma)}{a_2N}$, three latent roots of the secular equation would have negative real part, simultaneously, and then $P_1 = (S_1,\ I_1,\ I_2) = \left(\frac{a_2}{a_1+a_2}N,\ 0,\ 0\right)$ is the stable solution of the differential equations.

Lemma 2.8 *Endemic equilibrium point P_2 in the SIS epidemic diffusion system is stable only when $a_2 < a_1$ and $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_1N}$.*

Proof As far as we concerned, if the endemic equilibrium point $P_2 = (S_1, I_1, I_2) = \left(\frac{a_2}{a_1}\cdot\frac{b+\gamma}{\beta}, 0,\ N-\frac{a_1+a_2}{a_1}\cdot\frac{b+\gamma}{\beta}\right)$ exists, it should satisfy condition $I_2 > 0$ firstly. Namely, the propagation coefficient β should satisfy $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_1N}$. Then, similar as Lemma 2.7, we can obtain the Jacobi matrix for P_2 as follows:

$$J_{P_2} = \begin{pmatrix} -a_1-a_2 & b+\gamma-a_2-\frac{a_2}{a_1}(b+\gamma) & -a_2 \\ 0 & \frac{a_2}{a_1}(b+\gamma)-(b+\gamma) & 0 \\ \frac{a_1+a_2}{a_1}(b+\gamma)-\beta N & \frac{a_1+a_2}{a_1}(b+\gamma)-\beta N & \frac{a_1+a_2}{a_1}(b+\gamma)-\beta N \end{pmatrix}.$$

According to the Jacobi matrix J_{P_2}, we can obtain the secular equation of Eq. (2.51) again:

$$[\lambda+b+\gamma-\frac{a_2}{a_1}(b+\gamma)](\lambda^2+A_1\lambda+A_0)=0. \tag{2.57}$$

where $A_0 = a_1[\beta N - \frac{a_1+a_2}{a_1}(b+\gamma)]$ and $A_1 = a_1+a_2+\beta N-\frac{a_1+a_2}{a_1}(b+\gamma)$. Obviously, one of the latent roots of Eq. (2.57) is $\lambda_1^* = \frac{a_2-a_1}{a_1}(b+\gamma)$. Only when $a_2 < a_1$, the latent root λ_1^* is negative. On the other hand, when $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_1N}$, there is $A_0 > 0$ and $A_1 > 0$. Based on Routh-Hurwiz stability criterion, the other

two latent roots of Eq. (2.57) will be with negative real part. Therefore, P_2 is the stable solution of the simultaneous differential equations only when $a_2 < a_1$ and $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_1 N}$.

Lemma 2.9 *Endemic equilibrium point P_3 in the SIS epidemic diffusion system is stable only when $a_1 < a_2$ and $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_2 N}$.*

Proof Similarly as Lemma 2.8, if the endemic equilibrium point $P_3 = (S_1, I_1, I_2) = \left(\frac{b+\gamma}{\beta}, N - \frac{a_1+a_2}{a_2} \cdot \frac{b+\gamma}{\beta}, 0\right)$ is exist, it should satisfy condition $I_1 > 0$. That is, the propagation coefficient β should satisfy $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_2 N}$. Then, we can obtain the Jacobi matrix for P_3 as follows:

$$J_{P_3} = \begin{pmatrix} -a_1 - a_2 - \beta N + \frac{a_1+a_2}{a_2}(b+\gamma) & -a_2 & -a_2 \\ \beta N - \frac{a_1+a_2}{a_2}(b+\gamma) & 0 & 0 \\ 0 & 0 & \frac{a_1-a_2}{a_2}(b+\gamma) \end{pmatrix}.$$

Again, according to the Jacobi matrix J_{P_3}, we can obtain the secular equation of Eq. (2.51):

$$[\lambda - \frac{a_1 - a_2}{a_2}(b+\gamma)](\lambda^2 + B_1\lambda + B_0) = 0. \tag{2.58}$$

where $B_0 = a_2[\beta N - \frac{a_1+a_2}{a_2}(b+\gamma)]$ and $B_1 = a_1 + a_2 + \beta N - \frac{a_1+a_2}{a_2}(b+\gamma)$. Obviously, one of the latent roots of Eq. (2.58) is $\lambda_1^* = \frac{a_1-a_2}{a_2}(b+\gamma)$. Only when $a_1 < a_2$, the latent root λ_1^* is negative. On the other hand, when $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_2 N}$, there is $B_0 > 0$ and $B_1 > 0$. Based on Routh-Hurwiz stability criterion, the other two latent roots of Eq. (2.58) will be with negative real part. Therefore, P_3 is the stable solution of the simultaneous differential equations only when $a_1 < a_2$ and $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_2 N}$.

Remark 2.4 From Lemmas 2.7, 2.8 and 2.9, we can draw a conclusion that the diffusion threshold of the SIS epidemic diffusion model relies on the migrating-out coefficients of susceptible individuals of the two cities $a_i (i = 1, 2)$, also depends on the three key parameters: the total individuals of the two cities N, the birth rate b and the recovery rate γ.

2.3.4 Numerical Test

In this section, we take a numerical simulation to test how well the proposed model may be applied in practice. The initial values of parameters in the proposed epidemic diffusion model are listed as follows: $\beta = 8 \times 10^{-6}$, $b = 2 \times 10^{-4}$, $\gamma = 0.4$,

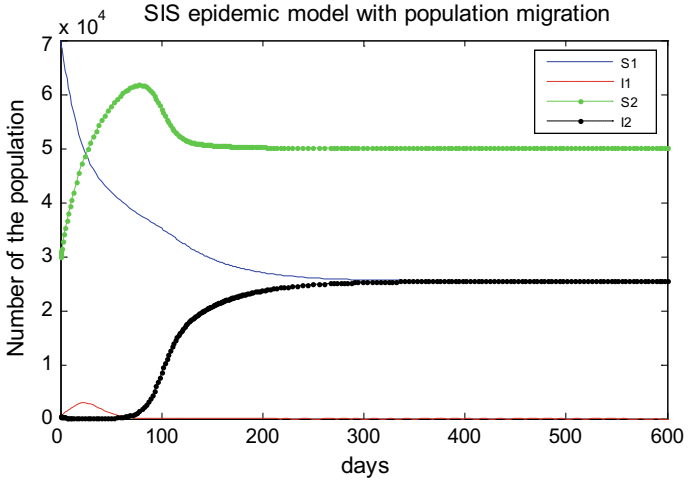
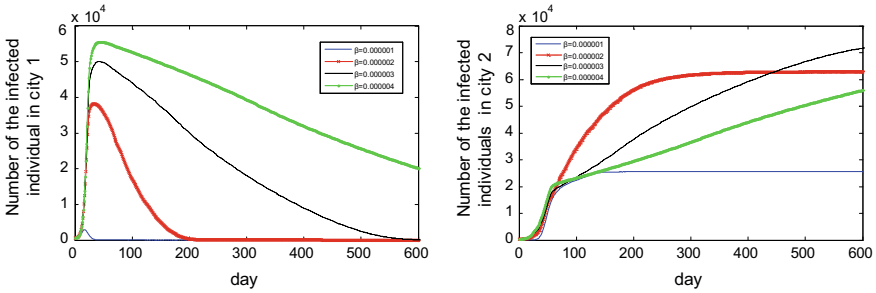


Fig. 2.13 Evolution trajectories of the SIS epidemic model

$a_1 = 0.02$, $a_2 = 0.01$, $N = 10^5$, $S_1(0) = 0.7 \times 10^5$, $I_1(0) = 600$ and $I_2(0) = 400$. We use MATLAB 7.0 mathematical solver together with Runge-Kutta method to simulate the epidemic model. The test is performed on an Intel(R) Core(TM) i3 CPU 2.4 GHz with 2 GB RAM under Microsoft Windows XP. Figure 2.13 is the evolution trajectories of the epidemic model. The curves respectively represent the different groups of people over time in these two cities.

From Fig. 2.13, one can see that the evolution trajectories of the SIS epidemic model with population migration between two cities are complicated. The number of susceptible individuals in city 1 decreases gradually with time increasing, while the number of susceptible individuals in city 2 increases at first and then decreases. On the other hand, the number of infected individuals in city 1 increases at first and then decreases; while the number of infected individuals in city 2 increases gradually with time increasing. However, all the susceptible individuals and infected individuals in city 1 and city 2 tend to the fixed values when time is long enough ($t > 300$). Meanwhile, the limit value of the SIS epidemic diffusion model with population migration between two cities is $Q_1 = (S_1, S_2, I_1, I_2) = (2.5013 \times 10^4,\ 5.0025 \times 10^4,\ 0,\ 2.4961 \times 10^4)$.

In line with the initial values we defined above, we have $a_2 < a_1$ and $\beta > \frac{(a_1+a_2)(b+\gamma)}{a_1 N}$. According to Lemma 2.8, one can get that the number of susceptible and infected individuals will be converged at $Q_2 = (S_1^*, S_2^*, I_1^*, I_2^*) = (2.50125 \times 10^4,\ 5.00245 \times 10^4,\ 0,\ 2.4963 \times 10^4)$. One can see that Q_1 is very close to Q_2, which is not a surprise, as it is consistent with the analytical conclusion in the last section. Once an epidemic outbreak, we are more concerned with the change regularity of the infected individuals in practice. Therefore, in the following subsections, we will discuss the relationship between the key parameters and the number of the infected individuals.

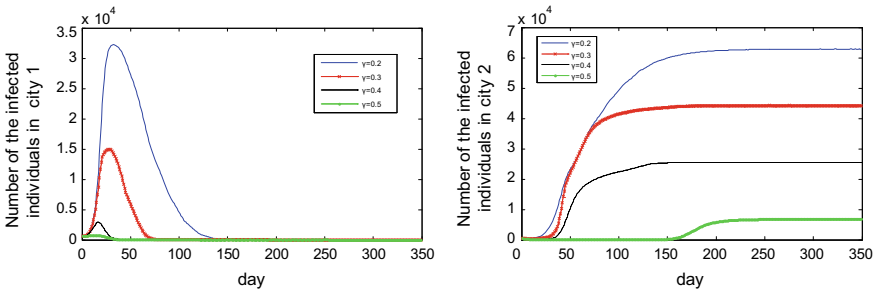


(a) City1 (b) City 2

Fig. 2.14 Number of the infected individuals versus different β

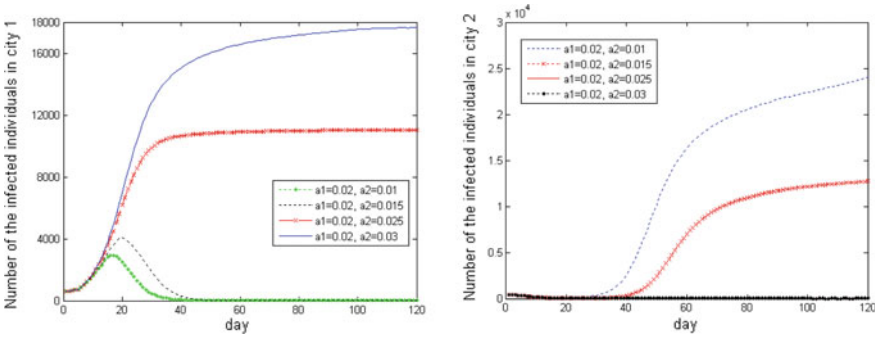
Figure 2.14 demonstrates the change of the number of the infected individuals in both cities with different propagation coefficient. It is easy to know that the evolution trajectories of infected individuals in city 1 are different from that in city 2 for any certain propagation coefficient β. From Fig. 2.14a, we can see that in the first some days, the larger β is, the faster spread of the epidemic in city 1 is. However, from Fig. 2.14b, we can't get the similar conclusion. Number of infected individuals in city 2 is not in direct proportion to the propagation coefficient β. It is worth mentioning that when the number of infected individuals in city 1 reach zero, the number of infected individuals in the other city is still positive when the epidemic diffusion system is stable. This is consistent with the Lemma 2.8. Similarly, if the initial conditions changed, we can also test and verify the other lemmas.

Figure 2.15 demonstrates the change of the number of the infected individuals in both cities with different recovery rate γ. As shown in Fig. 2.15a, when $\gamma = 0.2$, the maximum number of infected individual in city 1 is about 3.3×10^4. When $\gamma = 0.3$, the number is about 1.5×10^4. When $\gamma = 0.4$, the maximum number of infected individual in city 1 is less than 0.5×10^4. It informs us that the larger the recovery rate constant is, the smaller of the maximum number of infected individuals in city 1 is. Similar phenomenon can also be observed from Fig. 2.15b in city 2. Such figure



(a) City1 (b) City 2

Fig. 2.15 Number of the infected individuals versus different γ



(a) City1 (b) City 2

Fig. 2.16 Number of the infected individuals versus different a_1 and a_2

informs us it is important to improve the recovery rate as much as possible when in controlling an epidemic spread.

Figure 2.16 shows the change of the number of infected individuals in both cities with different migrating-out coefficients a_1 and a_2. According to Fig. 2.16, one can observe that no matter in city 1 or in city 2, the evolution trajectories of the infected individuals may generate a serious change when the migrating-out coefficient of susceptible individuals changed. For example, in city 1, when $a_2 < a_1 (a_1 = 0.02,\ a_2 = 0.01$ and $a_1 = 0.02,\ a_2 = 0.015)$, the number of infected individual tends to be zero. However, when $a_1 < a_2$ ($a_1 = 0.02, a_2 = 0.025$ and $a_1 = 0.02, a_2 = 0.03$), the number of infected individuals tends to be a positive constant above 1×10^4. In other words, with the increment of migrating-out coefficient in city 2, the limit number of infected individuals in city 1 may become positive from zero. The larger the migrating-out coefficient in city 2 is, the larger the limit number of infected individuals in city 1 is. Opposite to city 1, when $a_2 < a_1$, the number of infected individuals in city 2 tends to be a positive constant above 1×10^4. When $a_1 < a_2$, the number of infected individuals is very small and tends to be zero at last. It informs us that with the increment of migrating-out coefficient in city 2, the limit number of infected individuals in city 2 may become zero from a positive value. The larger the migrating-out coefficient in city 2 is, the smaller the limit number of infected individuals in city 2 is. To summarize, decreasing the migration population in only one city is not as effective as improving the recovery rate for controlling the epidemic diffusion. However, we can find a trade-off between the migrating-out coefficients in these two cities, and hence can control the infected individuals in both cities at last.

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