CHAPTER 4 NUMERICAL EXPERIMENTS

The dynamics of the models (3.5), (3.6) and (3.24) are illustrated in this chapter. All the numerical results were performed on a desktop computer with 2.00 GHz dual core processer and 2 GB Ram. The software uses MATLAB R2008b running under windows 7. This chapter is organized as follows. Section 4.1, the dynamics of the models (3.5), (3.6) and (3.24) under the conditions of the basic reproductive numbers, $\mathcal{R}_0$ and $\mathcal{R}_{0\gamma}$, are discussed to illustrate the various theoretical results presented in Chapter 3, and to analyze the effect of transport–related infection on its outbreak. In section 4.2, the model is applied to study the outbreak of SARS in Hongkong, and the dynamics of the model with parameter of SARS are discussed.

4.1 Experiment 1: Numerical Solutions of the Models

Firstly, the dynamics of model (3.6) are studies. This model neglects the movement of individuals, that is, $\alpha_1 = 0$ (the rate of individuals in one city leave to another city). The model (3.6) is simulated with parameter values in Table 4.1, and the initial conditions are used: $S(0) = 2$, $E(0) = 1$, $I(0) = 1$ and $R(0) = 0$. To illustrate the typical behaviors of all individuals at steady–states as a function of $\mathcal{R}_0$, the transmission rate within a city, β, is set to be 0.6 and 0.95. These give $\mathcal{R}_0 = 0.72 < 1$ and $\mathcal{R}_0 = 1.14 > 1$, respectively. The results are shown in Figures 4.1–4.2. Figure 4.1 shows the numerical solutions of model (3.6) converge to disease–free equilibrium, P_0, whenever $\mathcal{R}_0 < 1$, and to endemic equilibrium,P_*, in (3.15) – (3.18) if $\mathcal{R}_0 > 1$, (see Figure 4.2). These results are in line with Theorems 3.1 and 3.2, respectively. It is observed that susceptible individuals increases to five, whereas exposed, infected and recovered individuals decline exponentially to zero (see Figure 4.1). This implies that the disease dies out when $\mathcal{R}_0 < 1$. When $\mathcal{R}_0 > 1$, they approach an endemic equilibrium: $S^* = 4.219$, $E^* = 0.317$, $I^* = 0.19$ and $R^* = 0.083$, as shown in Figure 4.2. This means that the disease become endemic when $\mathcal{R}_0 > 1$.

Table 4.1 Description and value of parameters for the models (3.5), (3.6) and (3.24)

Parameters	**Descriptions**	**Values**	**References**
a	Recruitment rate (by birth and by immigration)	1	[10]
b	Natural death rate	0.2	[10]
c	Rate that exposed individuals become infected individuals	0.3	[10]
d	Transfer rate from infected individuals to recovered individuals	0.1	[22]
e	Mortality rate for infected individuals	0.4	[10]
α_2	Rate that recovered individuals become susceptible individuals	0.03	[22]
α_1	Rate that individuals of city i leave to city $j (j \neq i)$	0.9	[10]
β	Transmission rate	$0 \leq \beta \leq 1$	assumed
γ	Transport-related transmission rate	$0 \leq \gamma \leq 1$	assumed

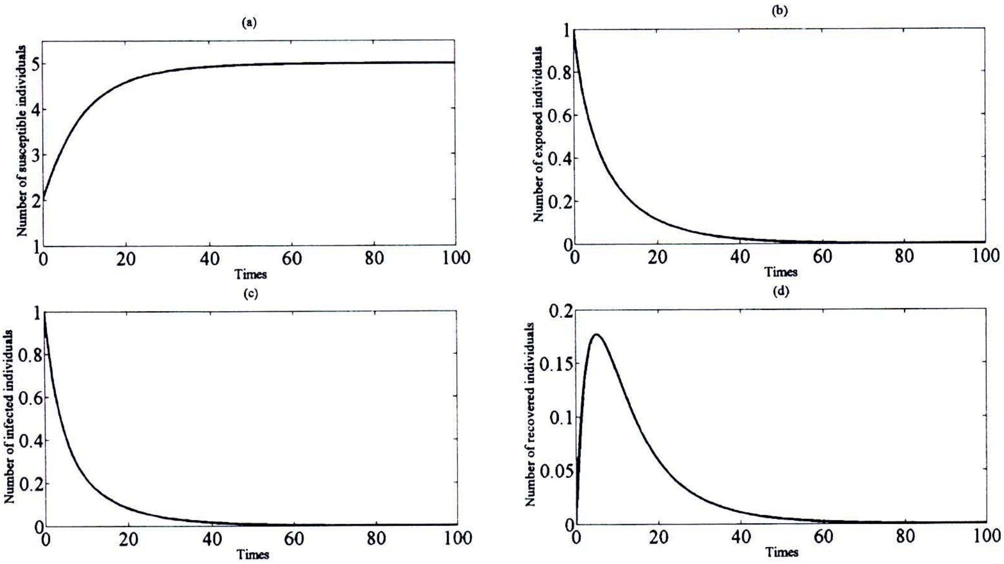

Figure 4.1 Simulations of the model (3.6) showing the number of all individuals as a function of time using the parameter values in Table 4.1 with $\beta = 0.6$ and $\mathcal{R}_0 = 0.72 < 1$.

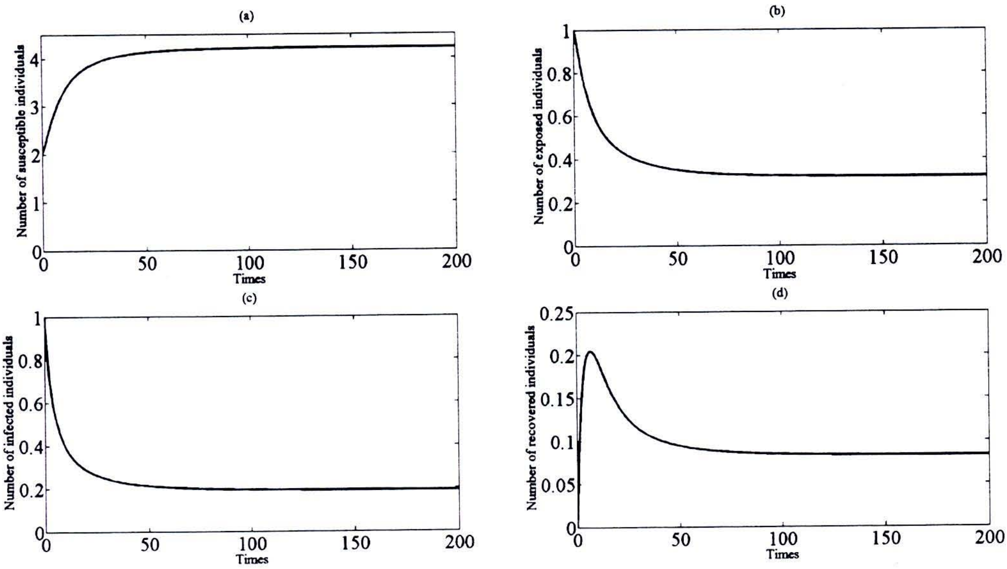

Figure 4.2 Simulations of the model (3.6) showing the number of all individuals as a function of time using the parameter values in Table 4.1 with $\beta = 0.95$ and $\mathcal{R}_0 = 1.14 > 1$.

Next, assume that only susceptible and exposed individuals travel to another city at the same rate α_1 while the infected and recovered individuals are inhibited from travelling to another city. Thus, model (3.6) is extend to model (3.24). To study the dynamics of model (3.24), it is simulated with parameter values given in Table 4.1. For numerical simulation purposes, the transmission rate within a city, β, is set to be 0.6 and 0.95, and the initial conditions are used: $S_1(0) = 2$, $E_1(0) = 1$, $I_1(0) = 1$, $R_1(0) = 0$, $S_2(0) = 2$, $E_2(0) = 0$, $I_2(0) = 0$ and $R_2(0) = 0$. The profiles of susceptible, exposed, infected and recovered individuals at steady–state are depicted in Figures 4.3 – 4.4. Let $\beta = 0.6$, then $\mathcal{R}_0' = 0.72$. It is seen that the simulation results obtained show convergence to the disease–free equilibrium $P_1 = (S^0, 0, 0, 0, S^0, 0, 0, 0) = (5, 0, 0, 0, 5, 0, 0, 0)$ if $\mathcal{R}_0' < 1$, as shown in Figure 4.3. According to Theorem 3.3, the disease–free equilibrium P_1 is locally asymptotically stable whenever $\mathcal{R}_0' < 1$. It interprets that the infected individuals in city 1 decrease while the infected individuals in city 2 appear to be pandemic initially, and are eventually extinct. Therefore, the disease die out separately in two cities if $\mathcal{R}_0' < 1$. When $\beta = 0.95$, then $\mathcal{R}_0' = 1.14$. All solutions of the model (3.24) admit an endemic equilibrium $P_2 = (S^*, E^*, I^*, R^*, S^*, E^*, I^*, R^*) = (4.219, 0.317, 0.19, 0.083, 4.219, 0.317, 0.19, 0.083)$, see Figure 4.4. This confirms that the endemic equilibrium, P_2, is locally asymptotically stable whenever $\mathcal{R}_0' > 1$ (as guaranteed by Theorem 3.4). Moreover, the results have some import implications. First, if the disease have appeared in both cities then the travel of susceptible and exposed individuals does not change the dynamics of disease spreading, and the final size of susceptible, exposed, infected and recovered individuals does not change, see Figure 4.4. Second, if a disease has appeared only in city 1 with $E_1(0) > 0$, $I_1(0) > 0$, $E_2(0) = 0$, $I_2(0) = 0$ and $\mathcal{R}_0' > 1$ (see Figures 4.4(b) – (c)), the travelling of exposed individuals will bring the disease to city 2 and the disease will break out later in city 2 (see Figures 4.4(f) – (g)). On the contrary, if $\mathcal{R}_0' < 1$, there is not the possibility for disease spreading in both cities, as shown in Figures 4.3(b) – (c) and Figures 4.3(f) – (g).

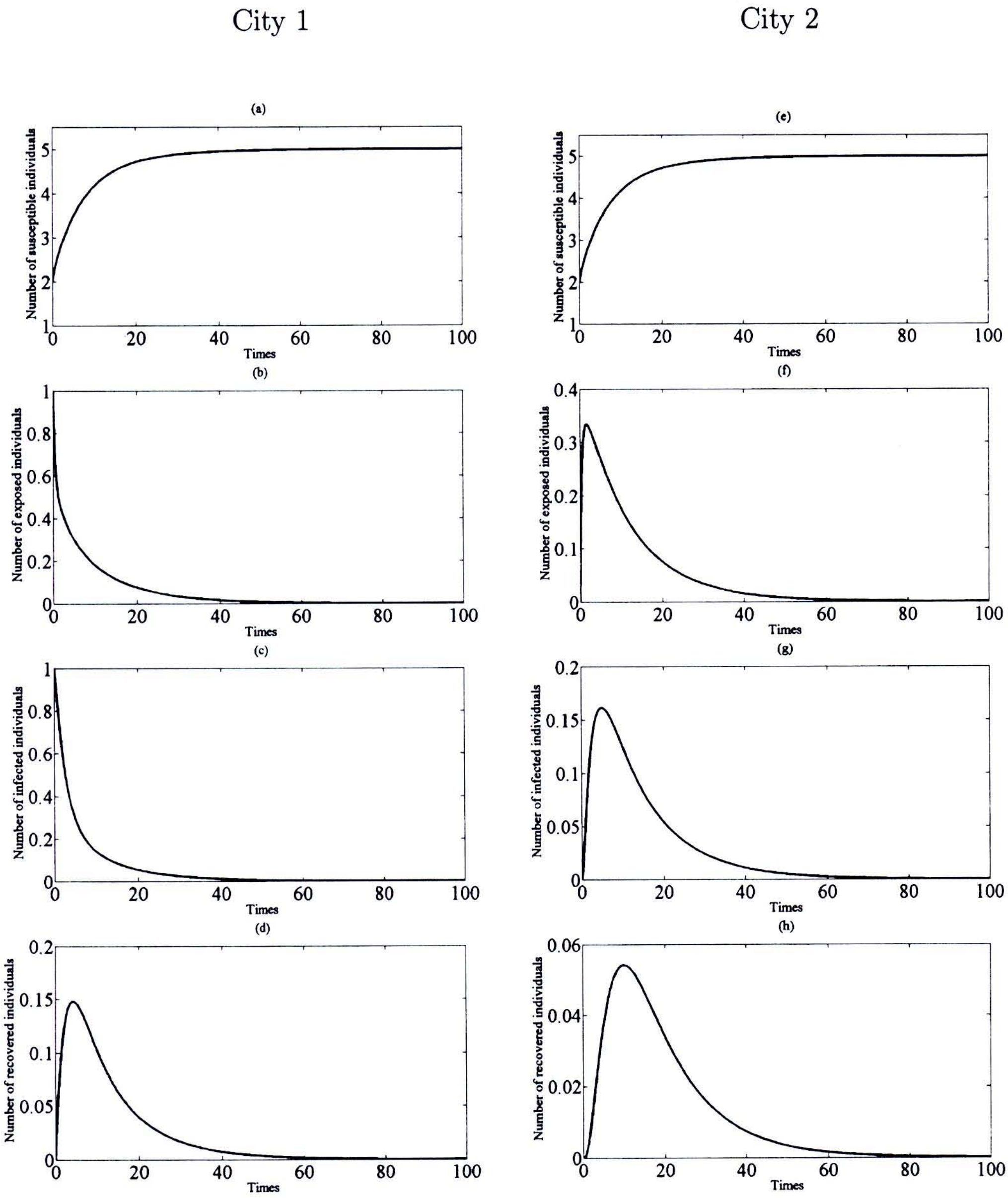

Figure 4.3 Simulations of the model (3.24) showing the number of all individuals in two cities as a function of time using the parameter values in Table 4.1 with $\beta = 0.6$ and $\mathcal{R}_0 = 0.72 < 1$.

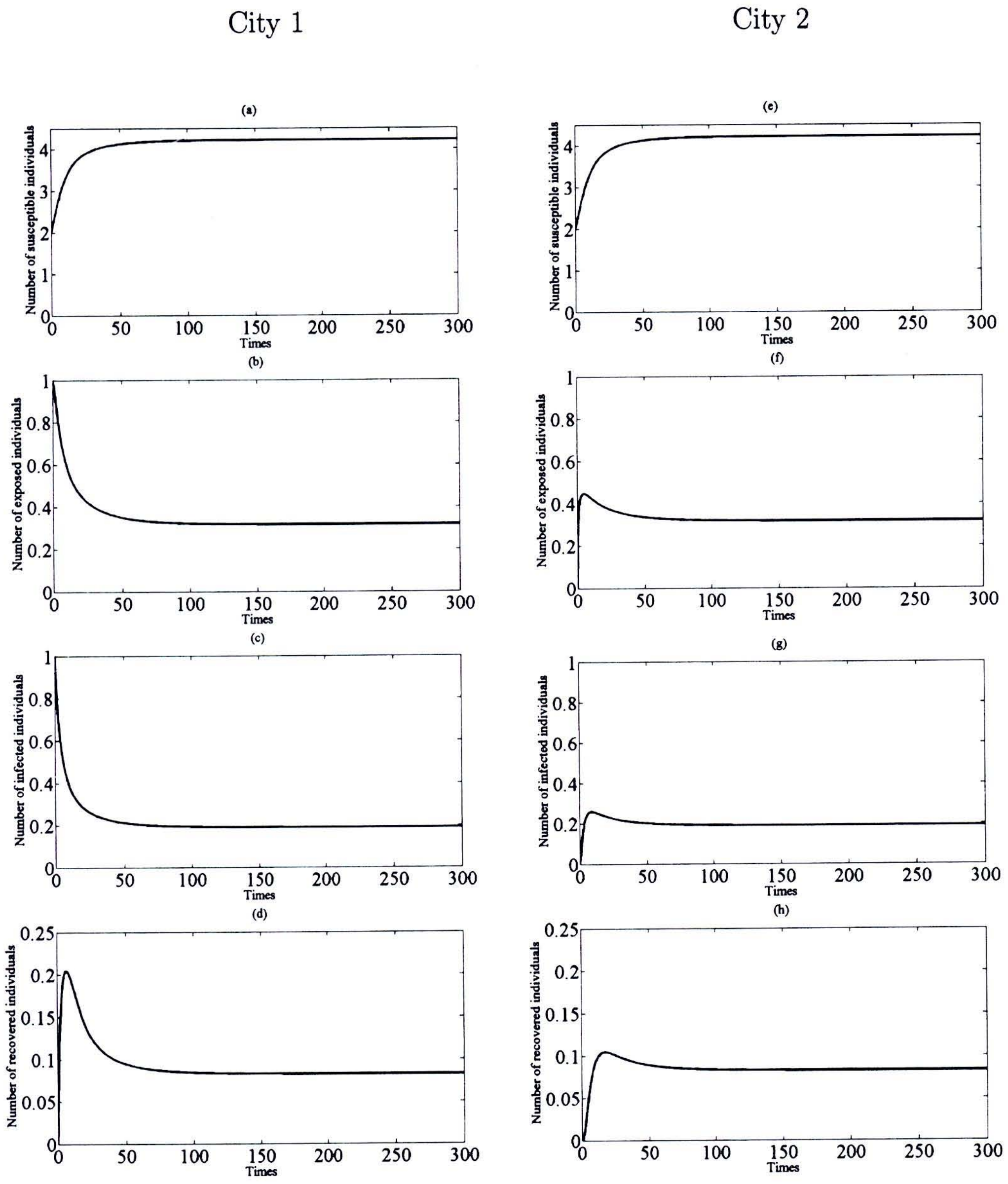

Figure 4.4 Simulations of the model (3.24) showing the number of all individuals in two cities as a function of time using the parameter values in Table 4.1 with $\beta = 0.95$ and $\mathcal{R}_0 = 1.14 > 1$.

Finally, the relations among two the basic reproductions numbers, $\mathcal{R}_0$ and $\mathcal{R}_{0\gamma}$, are given by

$$\mathcal{R}_0 = \frac{\beta\, c}{(b+c)\,(e+d)} \quad \text{and} \quad \mathcal{R}_{0\gamma} = \mathcal{R}_0 + \frac{\gamma \alpha_1 c}{(b+c)(e+d)}. \tag{4.1}$$

Equation (4.1) verifies that $\mathcal{R}_{0\gamma} > \mathcal{R}_0$ and $\mathcal{R}_{0\gamma}$ depends on $\mathcal{R}_0$ and transport–related infection rate, γ. It is found that, if $\beta = 0.6$ with the parameters b, c, d, e, α_1 given in Table 4.1 then $\mathcal{R}_0 < 1$ and $\mathcal{R}_{0\gamma} < 1$ whenever $0 < \gamma < 7/27$. Conversely, if $7/27 < \gamma \leq 1$ then $\mathcal{R}_{0\gamma} > 1$. When $\beta = 0.95$ then $\mathcal{R}_0 > 1$. This implies that $\mathcal{R}_{0\gamma} > 1$ for all $\gamma > 0$. Thus, this experiment discusses the dynamics of disease transmission by solving model (3.5) with various values of β and γ: $\beta = 0.6, 0.95$ and $\gamma = 0.09, 0.2, 1$, whilst retaining the same values of the other parameters. In all computations, the initial conditions are taken to be $S_1(0) = 2$, $E_1(0) = 2$, $I_1(0) = 2$, $R_1(0) = 2$, $S_2(0) = 1$, $E_2(0) = 1$, $I_2(0) = 1$, $R_2(0) = 1$.

When $\mathcal{R}_0 < 1$ and $\mathcal{R}_{0\gamma} < 1$, the parameters β and γ are chosen to be $\beta = 0.6$ and $\gamma = 0.09, 0.2$, respectively. The profiles of susceptible, exposed, infected and recovered individuals, as depicted in Figures 4.5 – 4.6, reveal that the numerical solutions of model (3.5) converge to disease–free equilibrium, P_1, whenever $\mathcal{R}_{0\gamma} < 1$ (as guaranteed by Lemma 3.2). This study suggests that the transport–related infection may not lead to the disease becoming endemic when $\mathcal{R}_0 < 1$ and $\mathcal{R}_{0\gamma} < 1$ for small γ.

For $\mathcal{R}_{0\gamma} > 1$, the values of $\gamma = 1$, $\beta = 0.6$ and $\gamma = 1$, $\beta = 0.95$ give $\mathcal{R}_0 = 0.72$, $\mathcal{R}_{0\gamma} = 1.8$ and $\mathcal{R}_0 = 1.14$, $\mathcal{R}_{0\gamma} = 2.22$, respectively. This lead to study the dynamics of model (3.5) in the cases $\mathcal{R}_0 < 1 < \mathcal{R}_{0\gamma}$, and $1 < \mathcal{R}_0 < \mathcal{R}_{0\gamma}$. In all experiments, the number of all individuals asymptotically approach to endemic equilibrium for $\mathcal{R}_{0\gamma} > 1$, see Figures 4.7 – 4.8. These simulation results are guaranteed by Theorem 3.5 . Therefore, the results suggest that if there is transport – related infection such that $\mathcal{R}_{0\gamma} > 1$, then the disease is endemic in two cities.

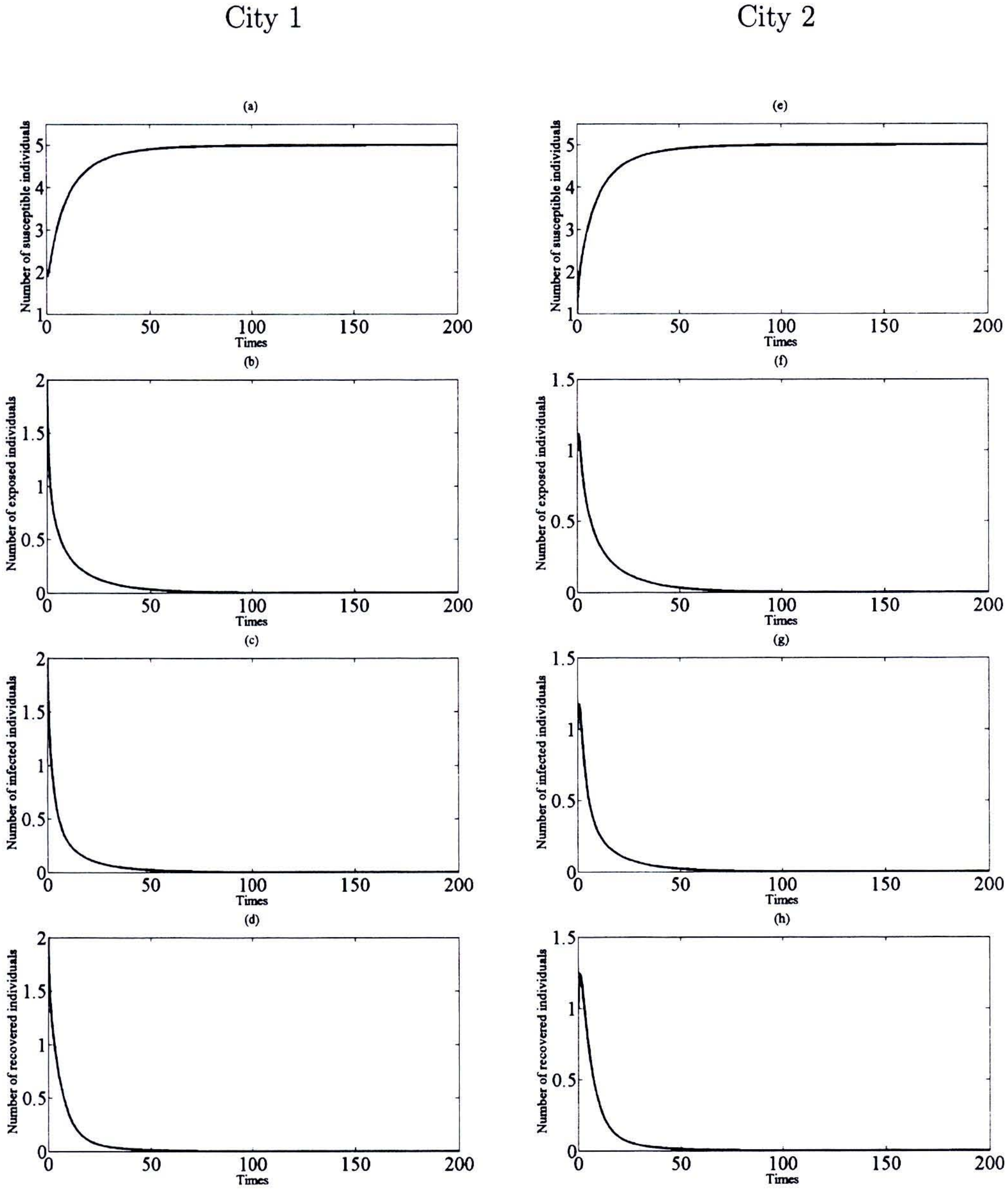

Figure 4.5 Simulations of the model (3.5) showing the number of all individuals in two cities as a function of time using the parameter values in Table 4.1 with $\beta = 0.6$, $\gamma = 0.09$, $\mathcal{R}_0 = 0.72 < 1$ and $\mathcal{R}_{0\gamma} = 0.82 < 1$.

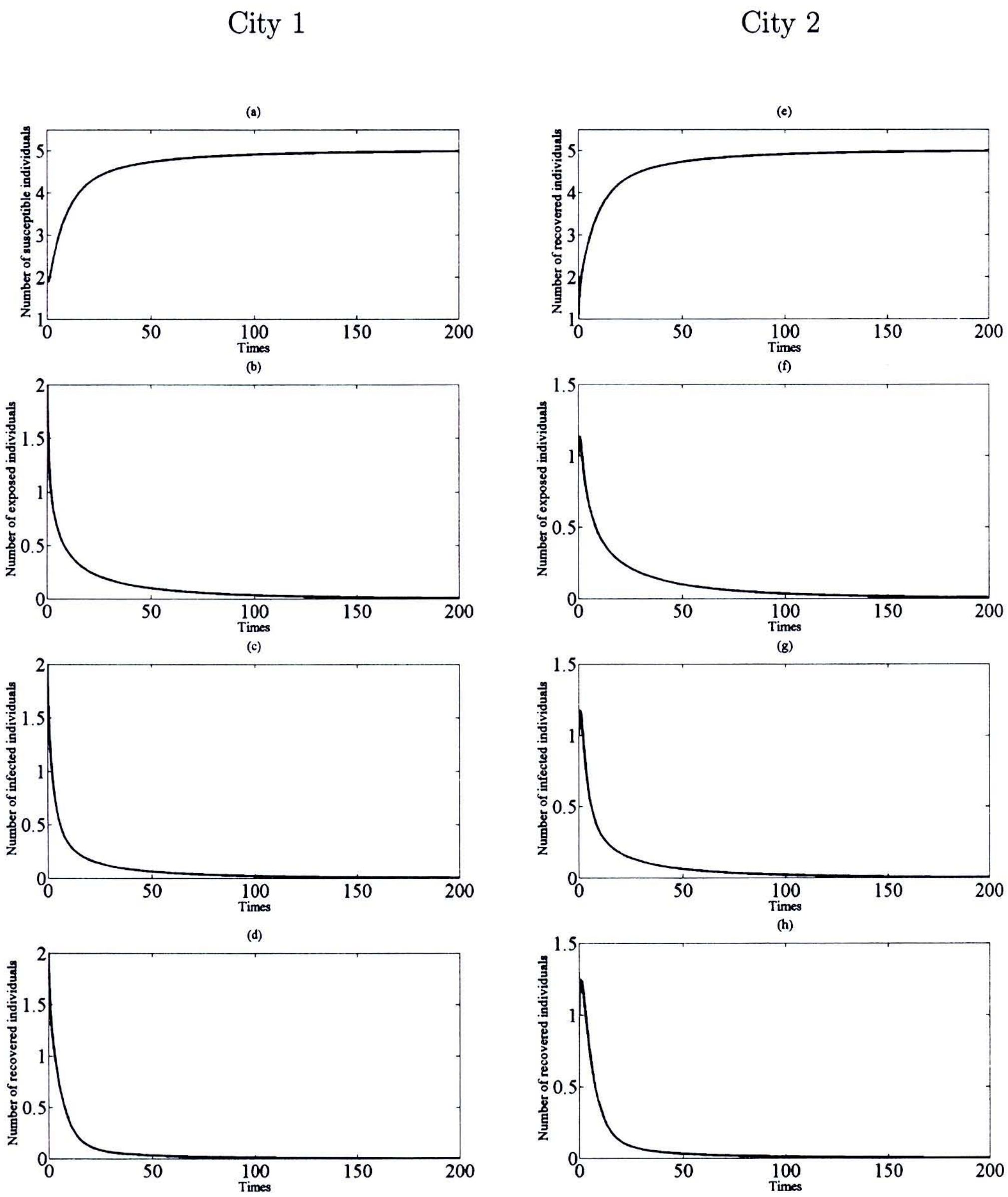

Figure 4.6 Simulations of the model (3.5) showing the number of all individuals in two cities as a function of time using the parameter values in Table 4.1 with $\beta = 0.6$, $\gamma = 0.2$, $\mathcal{R}_0 = 0.72 < 1$ and $\mathcal{R}_{0\gamma} = 0.936 < 1$.

City 1 City 2

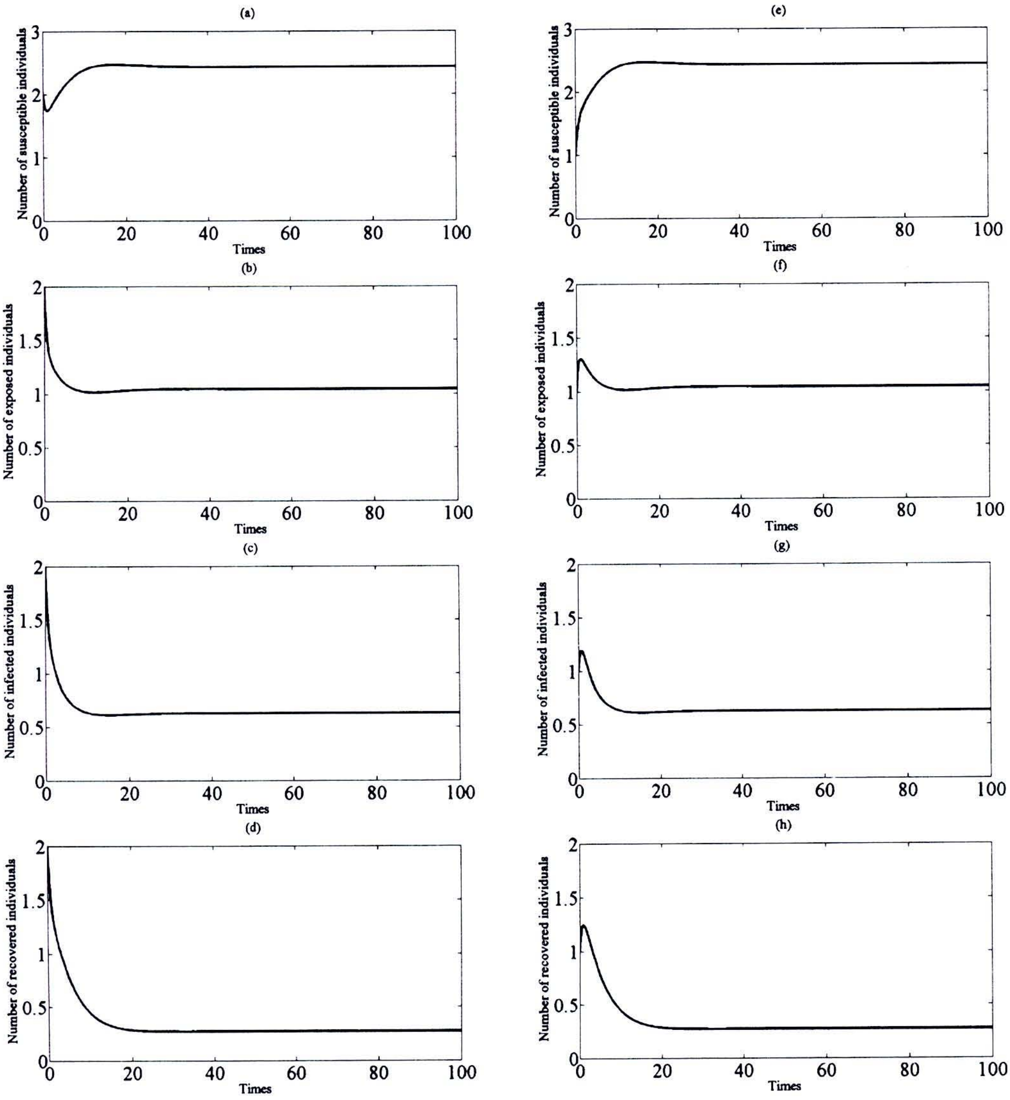

Figure 4.7 Simulations of the model (3.5) showing the number of all individuals in two cities as a function of time using the parameter values in Table 4.1 with $\beta = 0.6$, $\gamma = 1$, $\mathcal{R}_0 = 0.72 < 1$ and $\mathcal{R}_{0\gamma} = 1.8 > 1$.

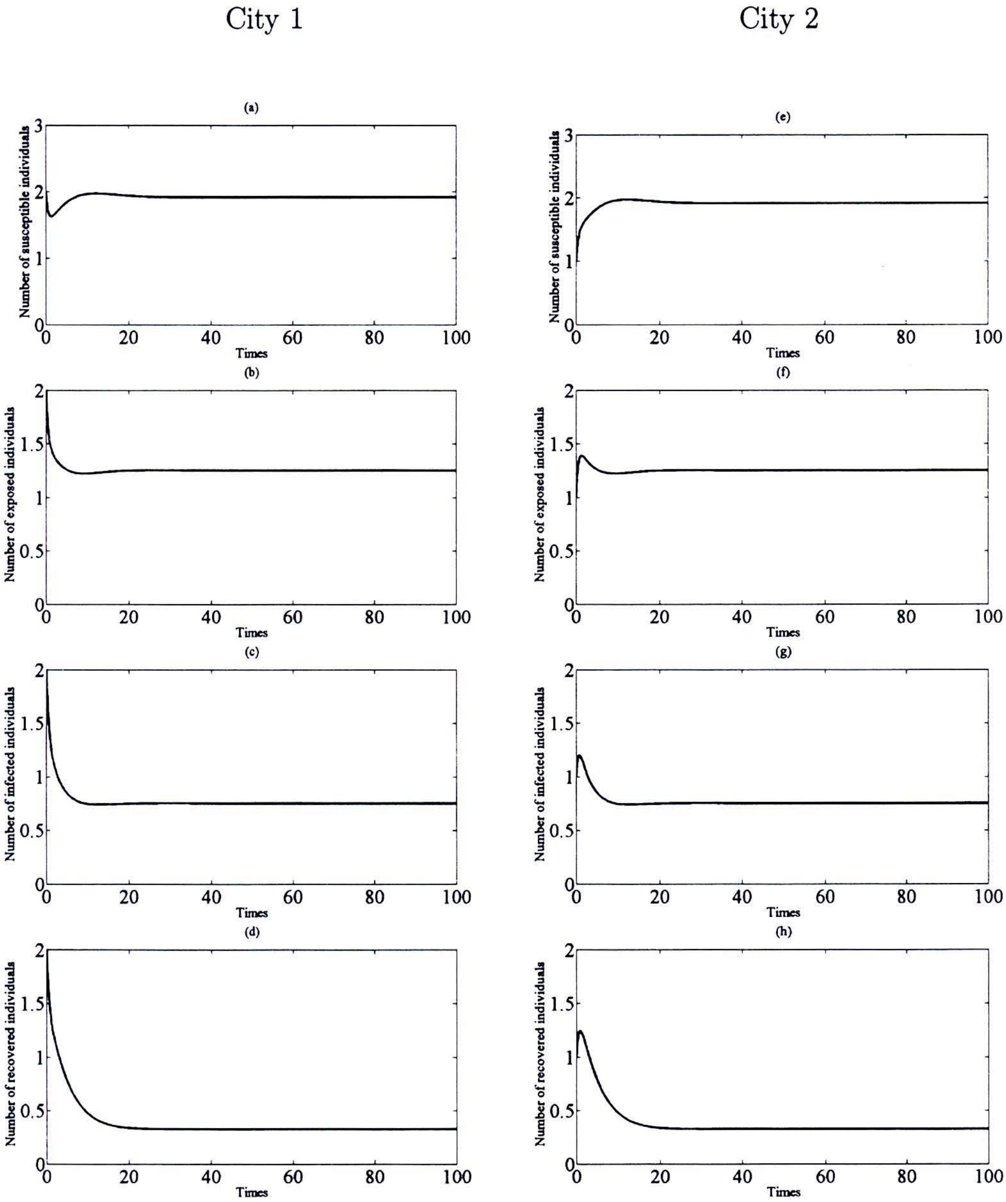

Figure 4.8 Simulations of the model (3.5) showing the number of all individuals in two cities as a function of time using the parameter values in Table 4.1 with $\beta = 0.95$, $\gamma = 1$, $\mathcal{R}_0 = 1.14 > 1$ and $\mathcal{R}_{0\gamma} = 2.22 > 1$.

As above results, it can be concluded that if the disease is endemic in both isolated cities, then transport–related infection will surely lead to the disease becoming endemic. When the two isolated cities are disease–free, transport–related infection may also have the possibility to lead to the disease becoming endemic. In addition, to see clearly the effect of transport–related infection, the relations among the two reproduction number,$\mathcal{R}_0$ and $\mathcal{R}_{0\gamma}$, are considered. By (4.1), it is found that $\mathcal{R}_{0\gamma} > \mathcal{R}_0$ for $\gamma > 0$, and $\mathcal{R}_{0\gamma} = \mathcal{R}_0$ for $\gamma = 0$. Since $\dfrac{\partial \mathcal{R}_{0\gamma}}{\partial \gamma} = \dfrac{\alpha_1}{(b+c)(e+d)} > 0$ for all $\gamma > 0$, it implies that $\mathcal{R}_{0\gamma}$ increases with the increase of γ. Consider the coexistence steady state $P^*_\gamma(S^*_\gamma, E^*_\gamma, I^*_\gamma, R^*_\gamma, S^*_\gamma, E^*_\gamma, I^*_\gamma, R^*_\gamma)$ of model (3.5) given by

$$\begin{aligned}
S^*_\gamma &= \frac{a\left\{(b+\alpha_2)\left(c+d+e\right)+cd\right\}}{\Omega_\gamma},\\
E^*_\gamma &= \frac{a\left(\mathcal{R}_{0\gamma}-1\right)\left(b+\alpha_2\right)\left(e+d\right)}{\Omega_\gamma},\\
I^*_\gamma &= \frac{ac\left(\mathcal{R}_{0\gamma}-1\right)\left(b+\alpha_2\right)}{\Omega_\gamma},\\
R^*_\gamma &= \frac{acd\left(\mathcal{R}_{0\gamma}-1\right)\left(b+\alpha_2\right)}{\Omega_\gamma},
\end{aligned}$$

with

$$\begin{aligned}
\Omega_\gamma &= (\mathcal{R}_{0\gamma}-1)(b(be+cd+d\alpha_2+e\alpha_2+bd+ce)+ce\alpha_2)\\
&\quad +b(bc+c\alpha_2+be+cd+d\alpha_2+e\alpha_2+bd),
\end{aligned}$$

as a function of γ, it is clear that $S^*_\gamma \longrightarrow S^*$, $E^*_\gamma \longrightarrow E^*$, $I^*_\gamma \longrightarrow I^*$, $R^*_\gamma \longrightarrow R^*$ as $\gamma \longrightarrow 0$. Comparing coexistence steady state values of susceptible, exposed, infected and recovered individuals in the case of $\gamma = 0$ with those of $\gamma > 0$, respectively, give $S^*_\gamma < S^*$, $E^*_\gamma > E^*$, $I^*_\gamma > I^*$ and $R^*_\gamma > R^*$ for $\gamma > 0$ because of

$$\begin{aligned}
\frac{\partial S^*_\gamma}{\partial \gamma} &= -\frac{a[(b+\alpha_2)(c+d+e)+cd]\partial \mathcal{R}_{0\gamma}/\partial\gamma}{\Omega^2_\gamma} < 0,\\
\frac{\partial E^*_\gamma}{\partial \gamma} &= \frac{a\Lambda(b+\alpha_2)(e+d)\partial \mathcal{R}_{0\gamma}/\partial\gamma}{\Omega^2_\gamma} > 0,\\
\frac{\partial I^*_\gamma}{\partial \gamma} &= \frac{ac\Lambda(b+\alpha_2)\partial \mathcal{R}_{0\gamma}/\partial\gamma}{\Omega^2_\gamma} > 0,\\
\frac{\partial R^*_\gamma}{\partial \gamma} &= \frac{ac\Lambda(b+\alpha_2)\partial \mathcal{R}_{0\gamma}/\partial\gamma}{\Omega^2_\gamma} > 0,
\end{aligned}$$

with $\Lambda = b(bc+c\alpha_2+be+cd+d\alpha_2+e\alpha_2+bd)$. It also found that $S^*_\gamma = S^*$, $E^*_\gamma = E^*$, $I^*_\gamma = I^*$ and $R^*_\gamma = R^*$ when $\gamma = 0$. This implies that, at steady–state, the total number of susceptible individuals in the both cities decreases with the increase of γ, while the total number of exposed, infected and recovered individuals increase with the increase of γ.

Next, the effect of transport-related infection to the final size of population is discussed. Note that

$$N^*_\gamma = S^*_\gamma + E^*_\gamma + I^*_\gamma + R^*_\gamma = \frac{a[(b+\alpha_2)(c+d+e)+cd]}{\Delta + \aleph}, \tag{4.2}$$

where $\Delta = b(be+cd+d\alpha_2+e\alpha_2+bd+ce)+ce\alpha_2$ and $\aleph = \dfrac{c(b+\alpha_2)(b-e)(e+d)(b+c)}{\beta+\gamma\alpha_1}$.

The partial derivative of N^*_γ with respect to γ is given by

$$\frac{\partial N^*_\gamma}{\partial \gamma} = -\frac{a[(b+\alpha_2)(c+d+e)+cd]}{(\Delta+\aleph)^2}\frac{\partial \aleph}{\partial \gamma}$$

with

$$\frac{\partial \aleph}{\partial \gamma} = \frac{c\alpha_1(b+\alpha_2)(e-b)(e+d)(b+c)}{(\beta+\gamma\alpha_1)^2}.$$

Since $e > b$ then $\dfrac{\partial \aleph}{\partial \gamma} > 0$. It follows that $\dfrac{\partial N^*_\gamma}{\partial \gamma} < 0$. Therefore, $N^*_\gamma < N^*$ for $\gamma > 0$ and $N^*_\gamma = N^*$ for $\gamma = 0$, where $N^* = S^* + E^* + I^* + R^*$. This implies that the final size of populations decreases with the increase of γ.

By the way, it is found that

$$\frac{\partial}{\partial \gamma}\left(\frac{E^*_\gamma + I^*_\gamma + R^*_\gamma}{N^*_\gamma}\right) = \frac{1}{N^{*2}_\gamma}\left[\frac{\partial(E^*_\gamma + I^*_\gamma + R^*_\gamma)}{\partial \gamma}S^*_\gamma - \frac{\partial S^*_\gamma}{\partial \gamma}(E^*_\gamma + I^*_\gamma + R^*_\gamma)\right] > 0,$$

$$\frac{\partial}{\partial \gamma}\left(\frac{S^*_\gamma}{N^*_\gamma}\right) = \frac{1}{N^{*2}_\gamma}\left[\frac{\partial S^*_\gamma}{\partial \gamma}(E^*_\gamma + I^*_\gamma + R^*_\gamma) - S^*_\gamma\frac{\partial(E^*_\gamma + I^*_\gamma + R^*_\gamma)}{\partial \gamma}\right] < 0,$$

because of $\dfrac{\partial S^*_\gamma}{\partial \gamma} < 0$. These imply that the proportion of the total number of exposed, infected and recovered individuals (i.e. the total number of individuals affected by the disease) increases with the increase of γ. On the contrary, the proportion of the susceptible individuals decreases with the increase of γ. Therefore, as above described, it can be suggested that transport–related infection will cause an endemic disease more seriously on spreading disease. Moreover, from these epidemiological implications, it is very essential to strengthen restrictions of passengers once when an infectious disease appears.

4.2 Experiment 2: Effect of Transport–Related Infection to SARS Outbreak in Hongkong

The $SEIRS$ model (3.6) is first applied to study the outbreak of SARS in Hongkong. The parameters used in the model (3.6) are obtained by choosing the most proper parameters (base-case estimates) to let the simulated curve fit the curve of cumulative number due to SARS on 17 March 2003 to 26 April 2003 [27]. After that the model (3.5) is used to study the outbreak of SARS in two cities.

For comparing the predicted total cases and the observed total cases (actual data) by WHO [27], the model (3.6) is modified by adding the cumulative number of SARS cases [28], see schematic diagram in Figure 4.9. From Figure 4.9, the cumulative number of SARS cases is given by

$$C' = kI \tag{4.3}$$

where C denotes cumulative number of SARS cases, and is used to compare with actual data, and k is the rate of progression from infective to diagnosed.

To study the outbreak of SARS in Hongkong, the $SEIRS$ model (3.6) and (4.3) is solved by using the following parameter values:

$$\begin{aligned} &a = 3 \quad \text{day}^{-1}, \quad b = 0.000034 \quad \text{day}^{-1}, \quad c = \frac{1}{6.4} \quad \text{day}^{-1}, \quad d = \frac{1}{4} \quad \text{day}^{-1}, \\ &e = 0.007934 \quad \text{day}^{-1}, \quad \alpha_2 = 0.001 \quad \text{day}^{-1} \quad \text{and} \quad k = \frac{1}{3} \quad \text{day}^{-1}. \end{aligned} \tag{4.4}$$

The values of b, c and d correspond to life expectancy of 80 years [29], an average incubation period of 6.4 days and infectious period of approximately 4 days [30], respectively. The rate of SARS induced mortality is 0.0079 day^{-1} [29]. The rate k is progression from infective to diagnosed and is set to be 1/3 day^{-1} [28]. The natural death rate is 0.000034 day^{-1} [29], then the rate e is 0.007934 day^{-1} (summation of natural death rate and SARS induced mortality rate). The basic reproduction number ($\mathcal{R}_0$) values for SARS is in the range 2.2 to 3.7 [31], then $\mathcal{R}_0$ is selected as 2.7 [31]. Substituting $\mathcal{R}_0 = 2.7$ in (3.13) give the transmission rate

$$\beta = 0.679 \quad \text{day}^{-1}. \tag{4.5}$$

For numerical simulations, the initial conditions are assumed to be $S(0) = 1,100$, $E(0) = 95$, $I(0)$, $R(0) = 0$ and $C(0) = 95$. For $I(0) = 95$ corresponds to number of infectious on 17 March 2003. The numerical results of model (3.6) and (4.3) are shown in Figures 4.10 – 4.12. Figure 4.10 shows that the number of susceptible individuals decrease whereas the number of exposed, infected and recovered individuals increase. This means that when the spreading disease is occurred, the number of susceptible individuals decrease since the susceptible individuals contact with infected individuals. Thus, susceptible individuals can require exposed individuals. After 2 - 10 days [30], the exposed individuals is progression to symptoms development, therefore, exposed individual is called infected individuals. After that infected individuals is hospitalized about 3-5 days [30] and then infected individuals is becomes recovered individuals. It can be concluded that SARS is highly infectious

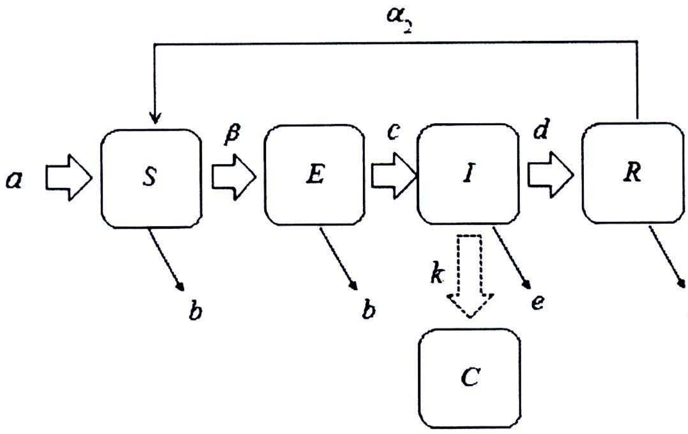

Figure 4.9 Schematic diagram of $SEIRS$ model with cumulative number of SARS cases.

base on the gradient of the susceptible curve. Figure 4.11 shows that the duration for the outbreak is about 60 days. The maximum number of infected individuals is 160 and the outbreak reached its peak about 18 days. The resulting curve for C fits very well with the total number of confirmed cases of SARS from 17 March 2003 to 26 April 2003 (totally 54 days) [27] as shown in Figure 4.12.

To study the dynamic of SARS in two cities, it is assumed that the all individuals can travel from one city to another city at the rate α_1. It is assumed that both cities are identical, i.e. the demographic are the same for each city. When the spread disease is occurred, the disease is transmitted with transition rate $\gamma\alpha_1$. Thus, the effect of transport–related infection, γ is monitored to forecast the total number of infected individuals and duration of its outbreak. In this case the model (3.5) is simulated by using parameter values $\alpha_1 = 0.9$ and various values of γ: $\gamma = 0$, $\gamma = 0.2$ and $\gamma = 1$, whilst retaining the same values of other parameters in the previous experiment. The initial conditions are used $S_1(0) = 1,100$, $E_1(0) = 95$, $I_1(0) = 95$, $R_1(0) = 0$, $C_1(0) = 95$, $S_2(0) = 1,100$, $E_2(0) = 5$, $I_2(0) = 5$, $R_2(0) = 0$, $C_2(0) = 0$. The purpose of this experiment is not to forecast the real size of the SARS epidemics in two cities but to study the effect of transport–related infection rate when the individuals in one city travel to another city. The cumulative number of cases and trajectory of infected individuals, in two cities, are shown in Figures 4.13 - 4.14, respectively. The results show that the total number of SARS in both cities increases with increase of γ (see Figure 4.13). It is also seen that the maximum number of infected individuals are 130, 150, 240 and the outbreak reached its peak about 22 days, 20 days, 10 days as γ increase, $\gamma = 0$, $\gamma = 0.2$, $\gamma = 1$, respectively (see Figure 4.14). This confirms that the size and duration of an outbreak can be influenced by transport-related infection. Thus, to reduction and to prevention the spread of SARS, it should have the control measure of the traveling of individual from one city to another city.

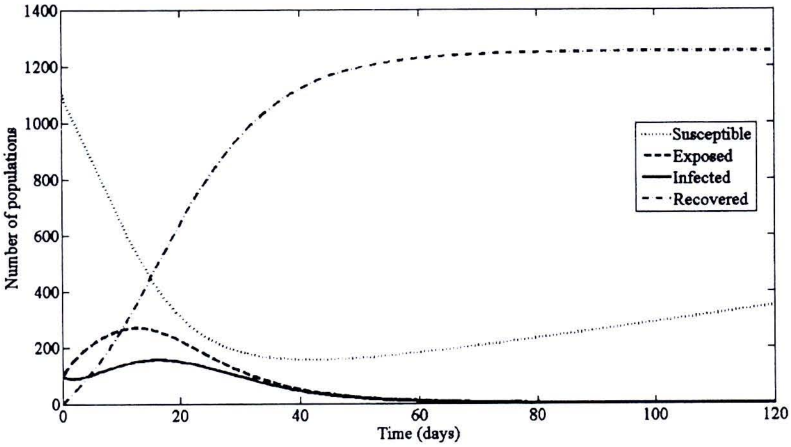

Figure 4.10 The dynamics of the model (3.6) with the parameter values: $a = 3$ day^{-1}, $b = 0.000034$ day^{-1}, $c = \frac{1}{6.4}$ day^{-1}, $d = \frac{1}{4}$ day^{-1}, $e = 0.007934$ day^{-1}, $\alpha_2 = 0.001$ day^{-1}, $k = \frac{1}{3}$ day^{-1} and $\beta = 0.679$ day^{-1}.

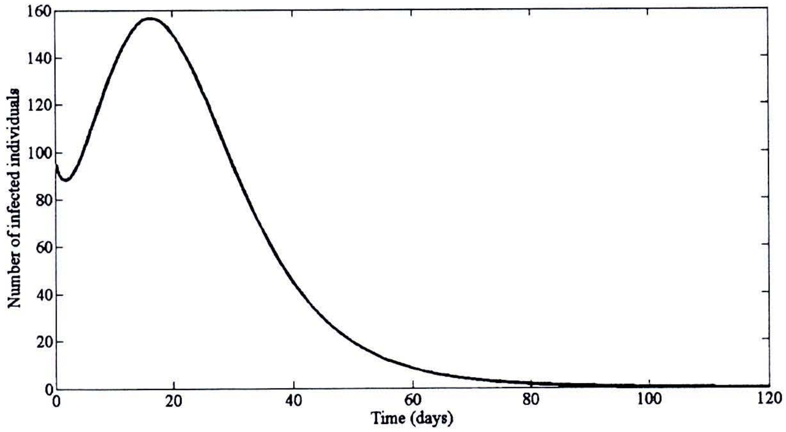

Figure 4.11 The trajectory of infected individuals of model (3.6) with parameters values: $a = 3$ day^{-1}, $b = 0.000034$ day^{-1}, $c = \frac{1}{6.4}$ day^{-1}, $d = \frac{1}{4}$ day^{-1}, $e = 0.007934$ day^{-1}, $\alpha_2 = 0.001$ day^{-1}, $k = \frac{1}{3}$ day^{-1} and $\beta = 0.679$ day^{-1}.

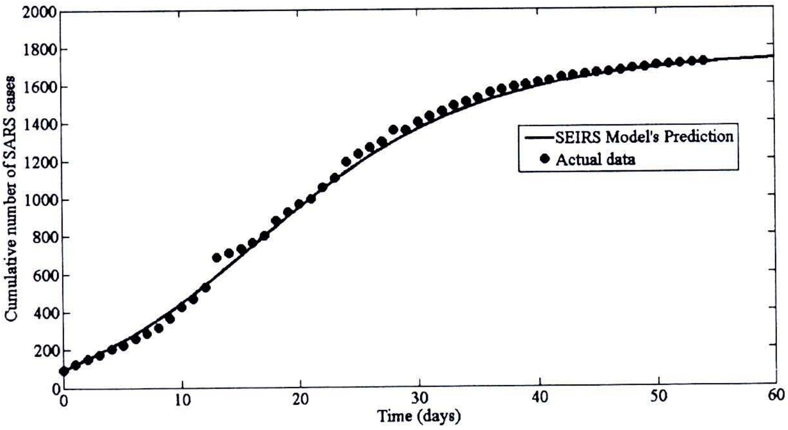

Figure 4.12 Comparison the cumulative numbers of SARS between actual data by WHO [27] (dotted lines) and *SEIRS* model's prediction (solid lines).

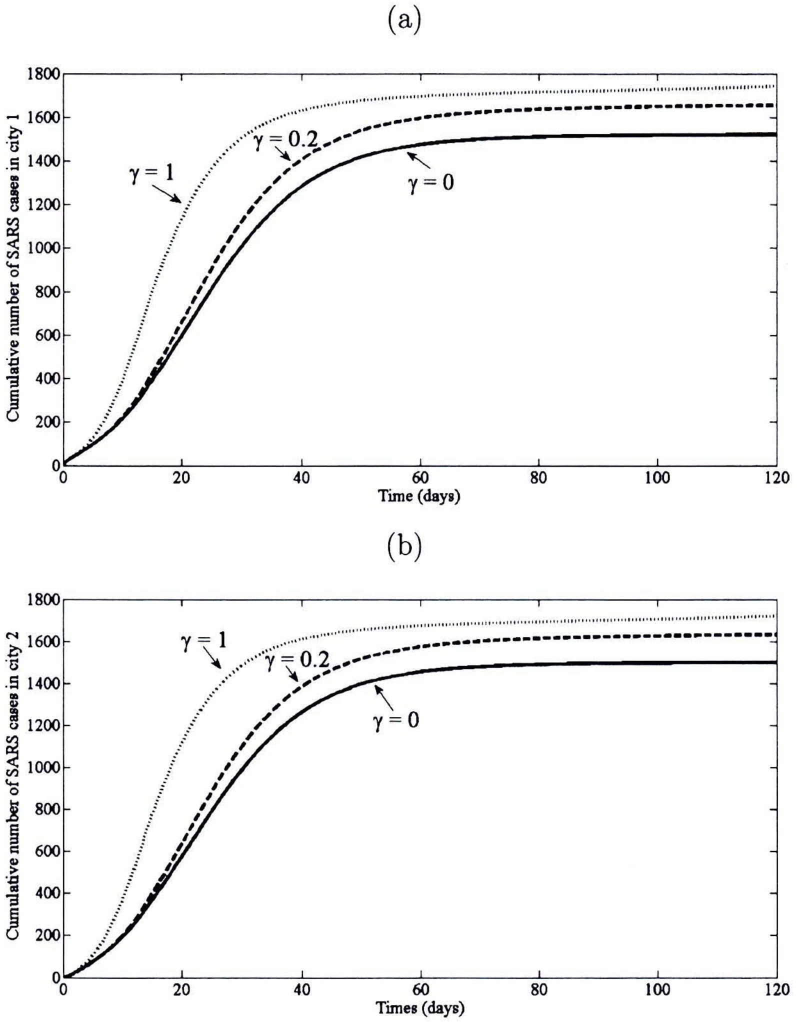

Figure 4.13 The cumulative number of SARS cases of the model (3.5) with various of γ: $\gamma = 0$, $\gamma = 0.2$, $\gamma = 1$.

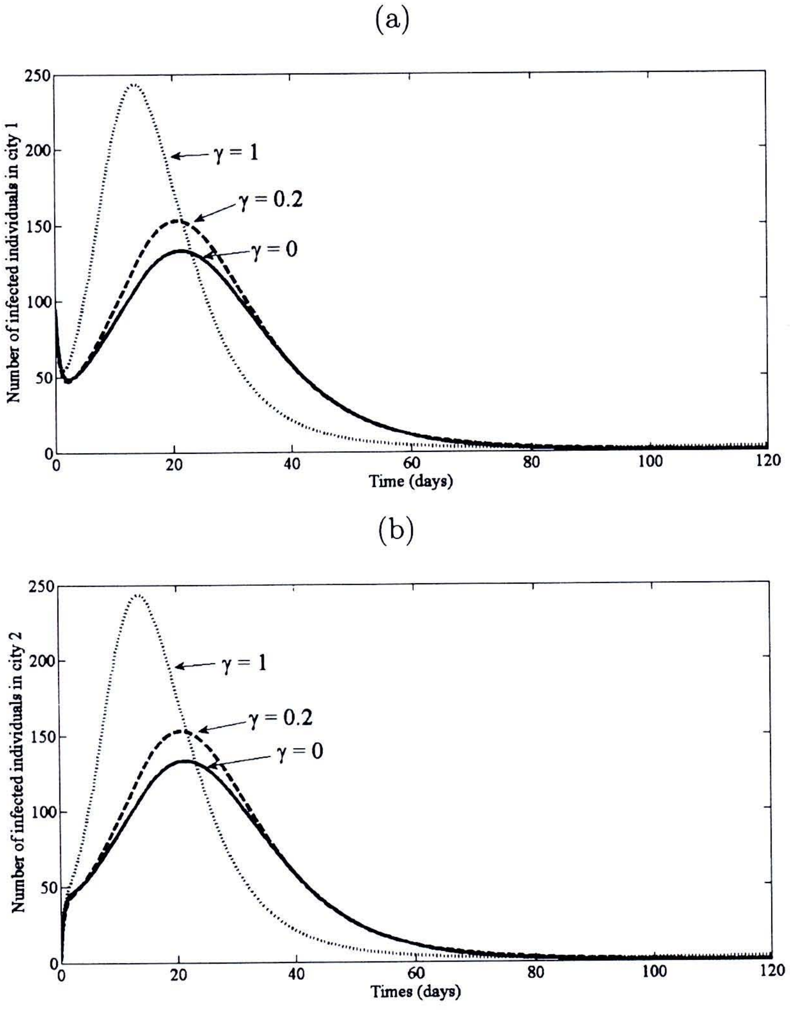

Figure 4.14 The trajectory of infected individuals of the model (3.5) with various of γ and other parameter values : $a = 3 \ \text{day}^{-1}$, $b = 0.000034 \ \text{day}^{-1}$, $c = \dfrac{1}{6.4} \ \text{day}^{-1}$, $d = \dfrac{1}{4} \ \text{day}^{-1}$, $e = 0.007934 \ \text{day}^{-1}$, $\alpha_2 = 0.001 \ \text{day}^{-1}$, $k = \dfrac{1}{3} \ \text{day}^{-1}$, $\beta = 0.679 \ \text{day}^{-1}$, and $\alpha_1 = 0.9 \ \text{day}^{-1}$.