CHAPTER 5 DISCUSSIONS AND CONCLUSIONS

In this thesis has formulated an $SEIRS$ model with transport–related infection for studying the spreading disease in two cities, and is given by

in city 1:

$$\begin{aligned}
\frac{dS_1}{dt} &= a - bS_1 - \frac{\beta S_1 I_1}{N_1} + \alpha_2 R_1 - \alpha_1 S_1 + \alpha_1 S_2 - \frac{\gamma \alpha_1 S_2 I_2}{N_2}, \\
\frac{dE_1}{dt} &= \frac{\beta S_1 I_1}{N_1} - (b + c + \alpha_1) E_1 + \alpha_1 E_2 + \frac{\gamma \alpha_1 S_2 I_2}{N_2}, \\
\frac{dI_1}{dt} &= cE_1 - (e + d + \alpha_1) I_1 + \alpha_1 I_2, \\
\frac{dR_1}{dt} &= dI_1 - (b + \alpha_1 + \alpha_2) R_1 + \alpha_1 R_2,
\end{aligned} \tag{5.1}$$

in city 2:

$$\begin{aligned}
\frac{dS_2}{dt} &= a - bS_2 - \frac{\beta S_2 I_2}{N_2} + \alpha_2 R_2 - \alpha_1 S_2 + \alpha_1 S_1 - \frac{\gamma \alpha_1 S_1 I_1}{N_1}, \\
\frac{dE_2}{dt} &= \frac{\beta S_2 I_2}{N_2} - (b + c + \alpha_1) E_2 + \alpha_1 E_1 + \frac{\gamma \alpha_1 S_1 I_1}{N_1}, \\
\frac{dI_2}{dt} &= cE_2 - (e + d + \alpha_1) I_2 + \alpha_1 I_1, \\
\frac{dR_2}{dt} &= dI_2 - (b + \alpha_1 + \alpha_2) R_2 + \alpha_1 R_1.
\end{aligned} \tag{5.2}$$

The basic reproduction number of the model is derived by using the next generation method. This number is the threshold condition for the existence of the endemic state. Stability analysis of the model shows that the disease–free equilibrium is locally asymptotically stable, that is no endemic equilibrium, if the basic reproduction number is less than or equal unity. On the other hand, if the basic reproduction number is greater than unity, the endemic equilibrium is locally asymptotically stable. The models (5.1) and (5.2) are analyzed by splitting into three cases: no individual travel, only susceptible and exposed individuals travel, and all individuals travel between two cities, respectively.

If there is no the movement of individuals among two cities, the models (5.1) and (5.2) are reduced to well known $SEIRS$ model:

$$\begin{aligned}
\frac{dS}{dt} &= a - \frac{\beta SI}{N} - bS + \alpha_2 R, \\
\frac{dE}{dt} &= \frac{\beta SI}{N} - (b+c)\, E, \\
\frac{dI}{dt} &= cE - (e+d)\, I, \\
\frac{dR}{dt} &= dI - (b+\alpha_2)\, R.
\end{aligned} \tag{5.3}$$

The basic reproduction number of (5.3) is given by

$$\mathcal{R}_0 = \frac{\beta c}{(b+c)(e+d)}.$$

It is found that the model (5.3) has two equilibrium, namely, disease–free equilibrium, (S^0, 0, 0, 0), and endemic equilibrium, (S^*, E^*, I^*, R^*), respectively. Theorems 3.1 – 3.2 given in Chapter 3 verify that the disease–free equilibrium of model (5.3) is stable provided $\mathcal{R}_0 < 1$, and the endemic equilibrium is proved to be stable for $\mathcal{R}_0 > 1$. The numerical results in Figures 4.1 – 4.2 confirm these behaviors, in the sense that, the numerical results of this model converge to the correct steady–state (for $\mathcal{R}_0 < 1$, this is disease–free equilibrium point while for $\mathcal{R}_0 > 1$, it is an endemic equilibrium point).

When, only susceptible and exposed individuals can travel to another city while the infected and recovered individuals are inhibited from travelling to another city, the models (5.1) and (5.2) become

in city 1:

$$\begin{aligned}
\frac{dS_1}{dt} &= a - bS_1 - \frac{\beta S_1 I_1}{N_1} + \alpha_2 R_1 - \alpha_1 S_1 + \alpha_1 S_2, \\
\frac{dE_1}{dt} &= \frac{\beta S_1 I_1}{N_1} - (b+c+\alpha_1)\, E_1 + \alpha_1 E_2, \\
\frac{dI_1}{dt} &= cE_1 - (e+d)\, I_1, \\
\frac{dR_1}{dt} &= dI_1 - (b+\alpha_2)\, R_1,
\end{aligned} \tag{5.4}$$

in city 2:

$$\begin{aligned}
\frac{dS_2}{dt} &= a - bS_2 - \frac{\beta S_2 I_2}{N_2} + \alpha_2 R_2 - \alpha_1 S_2 + \alpha_1 S_1, \\
\frac{dE_2}{dt} &= \frac{\beta S_2 I_2}{N_2} - (b + c + \alpha_1) E_2 + \alpha_1 E_1, \\
\frac{dI_2}{dt} &= cE_2 - (e + d) I_2, \\
\frac{dR_2}{dt} &= dI_2 - (b + \alpha_2) R_2.
\end{aligned} \tag{5.5}$$

By analyzing (5.4) and (5.5), it is found that the models (5.3), (5.4) and (5.5) have identical the basic reproduction number. This specifies that the dynamics in both cities of the models (5.4) and (5.5) are coincided with the model (5.3) as guaranteed by Theorem by 3.3 – 3.4 and Figures 4.3 – 4.4. It can be concluded that, from the obtained results, 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. In addition, if a disease has appeared only in city 1 and $\mathcal{R}_0 > 1$, the travelling of exposed individuals will bring the disease to city 2 and the disease will break out later in city 2 (see Figure 4.4).

When all individuals travel between two cities, the models (5.1) and (5.2) are analyzed. It is found that the basic reproduction number of this model is

$$\mathcal{R}_{0\gamma} = \mathcal{R}_0 + \frac{\gamma \alpha_1 c}{(b + c)(e + d)}.$$

Theoretical results in Lemma 3.2 and Theorem 3.5 show that the disease–free equilibrium is locally asymptotically stable when $\mathcal{R}_{0\gamma} < 1$ and if $\mathcal{R}_{0\gamma} > 1$, the endemic equilibrium is locally asymptotically stable, respectively. Figures 4.5 – 4.8 confirm that the all numerical results are consistent with the theoretical results.

Further, comparing $\mathcal{R}_0$ and $\mathcal{R}_{0\gamma}$, it is seen that $\mathcal{R}_{0\gamma} > \mathcal{R}_0$ for $\gamma > 0$. This suggests that even if the disease dies out separately in two cities in the absence of transport-related infection, it is possible that the disease will cause endemic disease due to transport-related infection.

Now, consider the coexistence steady state $P^*_\gamma(S^*_\gamma, E^*_\gamma, I^*_\gamma, R^*_\gamma, S^*_\gamma, E^*_\gamma, I^*_\gamma, R^*_\gamma)$ when the disease is endemic in both cities, it is seen that

$$\frac{\partial S^*_\gamma}{\partial \gamma} < 0, \quad \frac{\partial E^*_\gamma}{\partial \gamma} > 0, \quad \frac{\partial I^*_\gamma}{\partial \gamma} > 0, \quad \frac{\partial R^*_\gamma}{\partial \gamma} > 0, \tag{5.6}$$

which implies that the total number of susceptible individuals decreases with the increase of γ. On the other hand, the total number of exposed, infected and recovered individuals increases with the increase of γ. It is also found that, as γ is increased, the proportion of total number of exposed, infected and recovered individuals increases while the proportion of total number of susceptible individuals

decrease since $\frac{\partial}{\partial\gamma}\left(\frac{E^*_\gamma + I^*_\gamma + R^*_\gamma}{N^*_\gamma}\right) > 0$, and $\frac{\partial}{\partial\gamma}\left(\frac{S^*_\gamma}{N^*_\gamma}\right) < 0$, respectively. Moreover, the final size of populations decreases sine $\frac{\partial N^*_\gamma}{\partial\gamma} < 0$ for $\gamma > 0$. Therefore, epidemic model with transport–related infection will cause an endemic disease more seriously on spreading disease. According to Theorem 3.5 and the discussion behind this theorem, if there is no restriction on infective traveling, then transport–related infection intensifies the disease spread, in the sense of that, size of patients increase when $\mathcal{R}_{0\gamma} > 1$.

The model (5.3) is used to study the outbreak of SARS in Hongkong from 17 March 2003 to 26 April 2003 [27]. The simulation results of the model (5.3) fit well the total number of recorded SARS cases and the time of SARS infection, see Figure 4.12. Finally, the models (5.1) and (5.2) are simulated with SARS parameters to predict the SARS transmission between two cities. The results obtained show that the transport–related infection has effected to increase the number of infected individuals and to faster spread disease in both cities (see Figures 4.14).

Suggestions

The objective of thesis is just to develop the epidemic model with transport–related infection but not to forecast the real size of the SARS epidemics in two cities because of the lack of data completeness report. Moreover, the $SEIRS$ model can be predicted well for SARS outbreak within city since there is the completeness of the reported data. This suggests that the reliability of developed model rest on the accuracy and completeness of reported cases. Therefore, the developed in models (5.1) and (5.2) will be useful if it can be applied the real data of SARS or other diseases. It hope that this study can be helpful in providing the information to public health authorities and policy maker to reduce spreading disease when its occurs.

In this thesis, it is assumed that both cities are identical, and the individuals have no infectious force in the latent period. If there are no such an assumptions, the dynamics of the model may be different. This is left for future work.