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Honorable Mention

A Study of Infectious Diseases Through Mathematical Modeling

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A STUDY OF INFECTIOUS DISEASES BY MATHEMATICAL MODELS

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Abstract. Diseases are devastating. The SARS in 2003 and the swine influenza in 2009 sparked myriad of questions in our minds. Our major concern is the spread of germs. Throughout the entire project, we investigate disease-related issues and try to study the impacts of a disease by mathematical modeling.

We first start with the simplest model followed by more complicated ones. We focus on different factors that affect the spread of diseases. Diagrams are included in each chapter to see how the values of different groups vary. Then we come up with possible ways to prevent epidemics. Altering the models by adding more conditions, we find one that fits the real life situation - the SEIRS model. The situation in Hong Kong (*Swine Influenza* from April 2009 to April 2010 in Hong Kong) is simulated by putting the data into the model and our goal is fulfilled.

Introduction

Diseases had been bothering us for hundreds and thousands of years. Casualties, social destruction and havoc are the possible consequences. Pandemics were just as devastating as natural disasters, no matter when it occurred. Plague caused the death of 25 million people from 541 to 542 while Black Death was estimated to kill 75 million people. In the last century, about 50 million people were dead because of the Spanish flu. What a shocking number!

The spread of the above pandemics took place centuries ago. Is there any that happened in recent years? In 2003, there was an outbreak of SARS (Severe acute respiratory syndrome) all over the world, causing panic among the public. 8273 people were infected from November 2002 to July 2003. The mortality rate was

pretty high, reaching 9.6%. Apart from this, there was a widespread of swine influenza in 2009. Although the fatality rate was not as high as what we had first predicted, this aroused our awareness of public health.

If there is a remedy for all the diseases, then we will no longer be fearful of them. However, there may not be cure for some fatal diseases in reality. The occurrence of the above incidents sparked myriad of questions in our minds. We are deeply concerned about how a disease spreads. What are the factors influencing the outbreak of diseases? Are there any measures that are essential to prevent the widespread of disease? What is the most effective and advisable way to deal with the outbreak of diseases? Intrigued by this topic, we hope we can come up with answers to our queries in our project by mathematical means.

Throughout the project, we look into disease-related issues. We try to investigate the effects of a disease by mathematical modeling. In the meantime, we will find out the factors affecting the spread of diseases and possible ways to prevent epidemics. Additionally, data will be put in the model to simulate the situation in Hong Kong. Altering the models, we are longing for one that fits well in the real life situation. We hope we will be able to find out the rate of being infected and recovery and the number of people to be infected or should be vaccinated, etc. by substituting data. Our ultimate goal is to study how an disease spreads.

In the first chapter, we start with the simplest case – the SI model, with only susceptible group and infective model. We find that the results obtained do not comply with data of our daily lives. This prompts us to modify the model. So the SIS and SIR models are discussed in Chapter 3 and 4. As we know, a daily life situation can be very complicated with lots of variables. Apart from the susceptible, infective and removed group, other people may play a significant role too. That's why we introduce other elements in the model — SIVD, SIRS, SISR, TSIR, SEIR and SEIRS. In each section, we will first give a definition of all the variables we use and a detailed explanation of each model. A diagram will be shown to illustrate the idea lucidly. Then we will set the differential equations derived from the model. After that, we analyze the equations and see if we can obtain useful results. Substituting suitable values of the variables, we will plot graphs to observe the relationship between them. Finally, we will come up with a conclusion based on the observation.

Last but not least, we cannot finish the project without the help of our school's teachers, Mr. Wong and Mr. Cheung. They have guided and supervised us. We would like to acknowledge for their support.

1. Background

In this report, we would like to study how diseases are spread in a region. Through the investigation, we want to find out how the germs are spread, so as to find a way to reduce its damage to the society.

Through using differential equation and numerical simulation, we may figure out how the diseases are spread in the population. For instance, differential equations are used in Chapter 2 - The SI model.

Differential Equation:

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI \tag{2.1}$$

With the initial condition $I(0) = 1$, we can solve the differential equation (2.1).

$$I = \frac{N}{1 + (N-1)e^{-\beta SI}}, \qquad S = \frac{N(N-1)e^{-\beta SI}}{1 + (N-1)e^{-\beta SI}} \tag{2.2}$$

Then, we can plot the graph by using Excel with some t and defined β.

For example, $S = 6999999, I = 1, N = 7000000$ *at time* 0*, and* $\beta = 10^{-7}$.

Time	S	I	N
0	6999999	1	7000000
1	6999997.986	2.013752	7000000
2	6999991.834	4.055198	7000000
3	6999983.555	8.166162	7000000

$\vdots$

Numerical simulation:

We consider the same case,

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI \tag{2.1}$$

We consider the rate of change of S and I and take $\Delta t = 1$ day.

$$\begin{aligned} \text{So} \quad I(t+\Delta t) &= I(t) + \beta S(t) I(t) \Delta t \\ I(t+1) &= I(t) + \beta S(t) I(t) \\ \text{Also} \quad S(t+\Delta t) &= \beta(t) - \beta S(t) I(t) \Delta t \\ S(t+1) &= S(t) - \beta S(t) I(t) \end{aligned} \tag{2.3}$$

For example, $S = 6999999, I = 1, N = 7000000$ *at time* 0*, and* $\beta = 10^{-7}$.

$$
\begin{aligned}
& I(0) \\
&= 1 \quad \text{(defined)}
\end{aligned}
\qquad
\begin{aligned}
& S(0) \\
&= 6999999
\end{aligned}
$$

$$
\begin{aligned}
& I(1) \\
&= I(0+1) \\
&= I(0) + (10^{-7})S(0)I(0) \\
&= 1 + (10^{-7})(6999999)(1) \\
&= 1.70
\end{aligned}
\qquad
\begin{aligned}
& S(1) \\
&= S(0+1) \\
&= S(0) - (10^{-7})(6999999)(1) \\
&= 6999998.3
\end{aligned}
$$

$$
\begin{aligned}
& I(2) \\
&= I(1+1) \\
&= I(1) + (10^{-7})S(1)I(1) \\
&= 1.70 + (10^{-7})(6999998.3)(1.70) \\
&= 2.89
\end{aligned}
\qquad
\begin{aligned}
& S(2) \\
&= S(1+1) \\
&= S(1) - (10^{-7})S(1)I(1) \\
&= 6999998.3 - (10^{-7})(6999998.3)(1.7) \\
&= 6999997.11
\end{aligned}
$$

$$
\begin{aligned}
& I(3) \\
&= I(2+1) \\
&= I(2) + (10^{-7})S(2)I(2) \\
&= 2.89 + (10^{-7})(6999997.11)(2.89) \\
&= 4.913
\end{aligned}
\qquad
\begin{aligned}
& S(3) \\
&= S(2+1) \\
&= S(2) - (10^{-7})S(2)I(2) \\
&= 6999997.11 - (10^{-7})(6999997.11)(2.89) \\
&= 6999995.087
\end{aligned}
$$

Etc.

Then we can draw a table.

Time	S	I	N
0	6999999	1	7000000
1	6999998.3	1.7	7000000
2	6999997.11	2.89	7000000
3	6999995.087	4.913	7000000

$\vdots$

Then we can use these data to plot a graph with Excel.

These two methods have similar results and are used throughout our report frequently.

There are some similar projects done by other researchers. For example, David Smith and Lang Moore have done a project called "The SIR Model for Spread of Disease". [See reviewer's comment (3)] In this project, they are able to create a model of how Hong Kong flu is spread in New York City in 1968-1969. The model they used is SIR model, which will also be discussed in our project.

Symbols will not be mentioned in this section as they are independent in each model.

2. The SI Model

[See reviewer's comment (2)]

The SI model is the simplest model of an infectious disease which categorizes people as either susceptible (S) or infective (I). A susceptible person can become infective by contact with an infective. Let $\beta\Delta t$ be the probability of a susceptible person being infected by an infectious person in time interval Δt. So,

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI \tag{2.1}$$

Since there are $\beta SI\Delta t$ people moving out from S in Δt (S is decreasing), we put a "-" sign before βSI. On the other hand, there are $\beta SI\Delta t$ people being infected in Δt (I is increasing) so $\dfrac{dI}{dt}$ is positive.

$$S \xrightarrow{\beta SI} I$$

We defined the arrow ($\longrightarrow$) as flow of population from one side to another. Here it means that there are $\beta SI\Delta t$ people moving from S to I in Δt .

We assume that when $t = 0$, only 1 people is infective and other people is susceptible (i.e. $S = 6999999, I = 1$).

By solving (2.1) with initial condition $I(0) = 1$ [See reviewer's comment (4)]

$$I = \frac{N}{1 + (N-1)e^{-\beta SI}}, \qquad S = \frac{N(N-1)e^{-\beta SI}}{1 + (N-1)e^{-\beta SI}} \tag{2.2}$$

We assumed that the population size (N) are $7,000,000$. We also define that $S + I = N$, which is a constant, at any time t. These assumptions will also be used in the following chapters. Assuming that the people are well-mixed, meaning that they have an equal probability of meeting every person in the population.

Below are some cases of how S and I behave when time goes on.

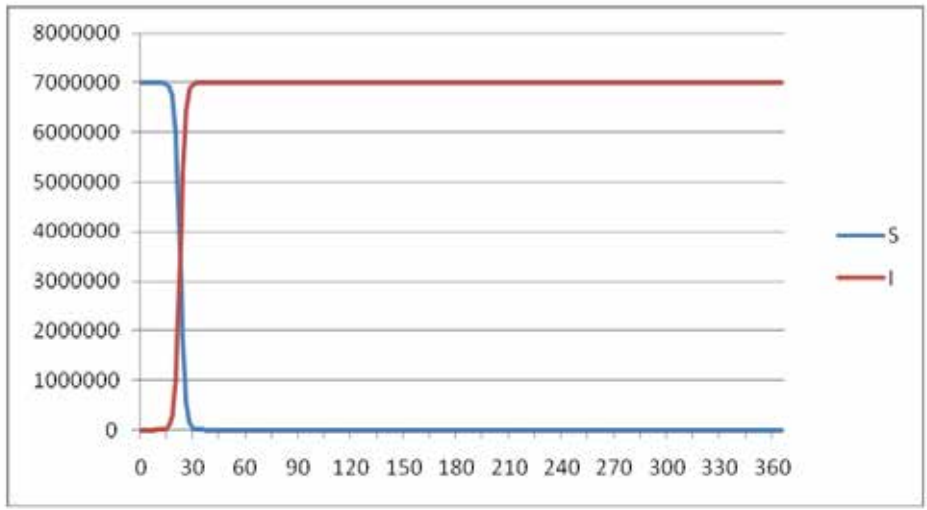


FIGURE 1. The number of people in class S, class I and in time t with $\beta = 10^{-7}$

From Figure 1, when beta equals 10^{-7}, S will decrease dramatically and so I increases sharply. In about 40 days, almost all people (i.e. 7 million people) will be infected.

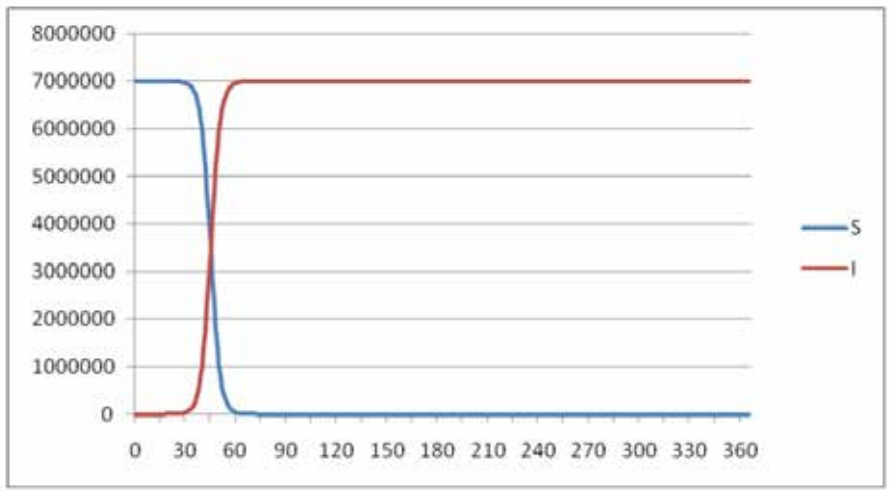


FIGURE 2. The number of people in class S, class I and in time t with $\beta = 5 \times 10^{-8}$

From Figure 2, when beta equals 5×10^{-8}, S will decrease slightly slower and so I increases in the same rate. In about 90 days, almost all people (i.e. 7 million people) will be infected.

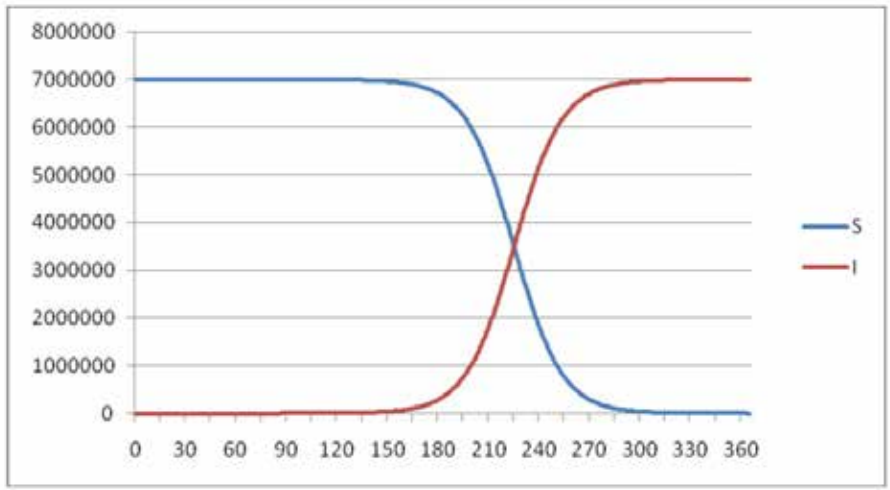


FIGURE 3. The number of people in class S, class I and in time t with $\beta = 10^{-8}$

From Figure 3, when beta equals 10^{-8}, S will decrease much slower and so I increases in the same rate. In about 400 days, almost all people (i.e. 7 million people) will be infected.

In all these three cases, as there is no way for the infective to recover, when $t \to \infty$, the entire population will turn to I. It is not applicable in real world situation usually, so it is only a start of this project, helping us to understand and then create a more applicable model.

3. The SIS Model

After recovering from some infectious diseases such as common cold and dysentery, a recovered person may have a low immunity. The infective person may become susceptible again. Therefore, we now introduce the SIS model by adding one more "S" into the SI model.

$$S \xrightarrow{\beta SI} I \xrightarrow{\gamma I} S$$

(The definitions of S, I and β are the same as that in the SI model. $\gamma \Delta t$ is defined as the probability that an infective person recovers during time Δt.)

Susceptible people are infected during time Δt is given by $\beta SI\Delta t$ and the infective people who recover during time Δt is given by $\gamma I\Delta t$, i.e.

$$\begin{aligned} I(t+\Delta t) &= I(t) + \beta S(t)I(t)\Delta t - \gamma I(t)\Delta t \\ S(t+\Delta t) &= S(t) - \beta S(t)I(t)\Delta t + \gamma I(t)\Delta t \\ \text{When } \Delta t \to 0, \qquad \frac{dI}{dt} &= \beta SI - \gamma I \\ \frac{dS}{dt} &= \gamma I - \beta SI \end{aligned} \tag{3.1}$$

Let N be the population size and $N = S + I$. Define the basic reproductive ratio as

$$\Re_0 = \frac{\beta N}{\gamma}.$$

We now need to find out the value of I by solving (3.1). Before that we make some assumptions first. We let I_0 and S_0 be the number of initial infective individuals and initial susceptible individuals respectively. Moreover, we assume $I_0 = 1$. Since S_0 should be much larger than I_0, we assume $S_0 \approx N$. We have the following two cases:

(i) For $\Re_0 = \dfrac{\beta N}{\gamma} \neq 1$,

$$I = \left\{ \frac{1}{N\left(1 - \dfrac{1}{\Re_0}\right)} + \left[1 - \frac{1}{N\left(1 - \dfrac{1}{\Re_0}\right)} \right] e^{-\beta N\left(1 - \frac{1}{\Re_0}\right)t} \right\}^{-1} \tag{3.2}$$

(ii) For $\Re_0 = 1$,

$$I = \frac{1}{\beta t + 1} \tag{3.3}$$

After solving for I, we have to think about whether the disease will spread out at the beginning and whether the disease will come to an equilibrium position. It is easy to see that the disease will spread out if I is increasing, i.e. $\dfrac{dI}{dt} > 0$. By equation (3.1) and our assumption $S_0 \approx N$, the disease will spread out $\iff \Re_0 > 1$. Likewise, the disease will not spread out $\iff \Re_0 \leq 1$. Therefore, we consider the following two cases:

(i) $\Re_0 > 1$, the disease will spread out at the beginning.
Taking limits on both sides of equation (3.2) as $t \to \infty$.

$$\lim_{t\to\infty} I = N\left(1 - \frac{1}{\Re_0}\right)$$

This indicates that when $\Re_0 > 1$, the disease will spread out and the final number of infected people will be closed to $N\left(1 - \frac{1}{\Re_0}\right)$.

(ii) $\Re_0 \leq 1$, the disease will not spread out at the beginning.
For $\Re_0 \neq 1$, take limits on both sides of equation (3.2) as $t \to \infty$.
For $\Re_0 = 1$, take limits on both sides of equation (3.3) as $t \to \infty$.
Both the above equations show that $\lim_{t\to\infty} I = 0$.
This indicates that when $\Re_0 \leq 1$, the disease will disappear.

In Hong Kong, the total number of people is around 7 millions. We assume $N = 7 \times 10^6$ and $\beta = 10^{-7}$. Here, we want to study how the change in γ affects I. From the above results, the disease will spread out when $\Re_0 > 1 \iff \gamma < 0.7$. On the other hand, the disease will disappear when $\Re_0 \leq 1 \iff \gamma \geq 0.7$. Now we use two graphs for illustration.

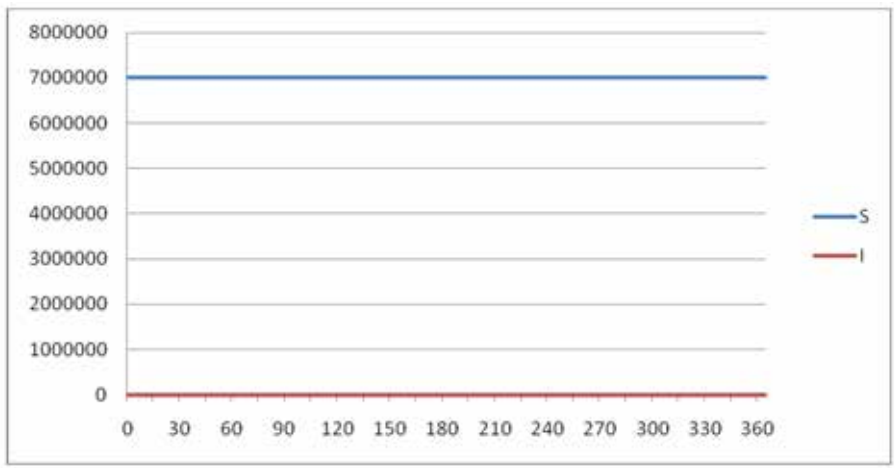


FIGURE 4. The number of people in class S, class I in time t with $\gamma = 0.8$

Since $\Re_0 = 0.875 < 1$, the disease will not spread out. The line of I levels off so the disease will disappear. This is due to the rate of recovery which is greater than the rate of being infected.

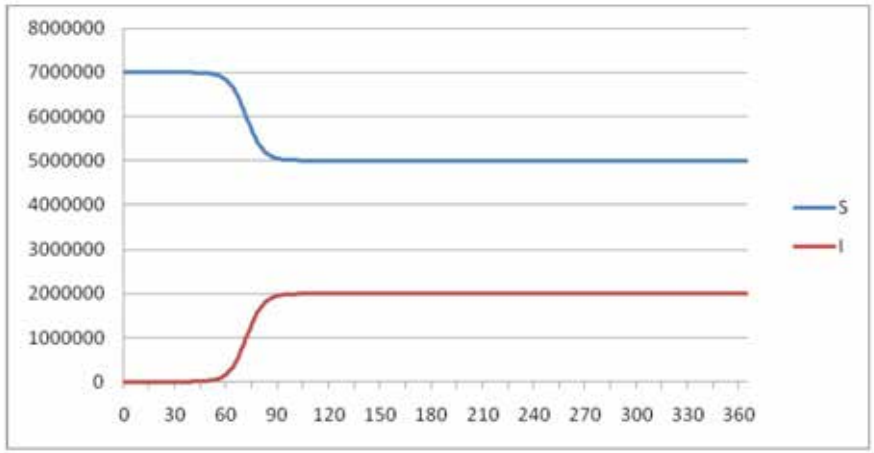


FIGURE 5. The number of people in class S, class I in time t with $\gamma = 0.5$

Since $\Re_0 = 1.4 > 1$, the disease will spread out. During 1^{st} to 60^{th} day, I increases slowly. During 60^{th} to 90^{th} day, I increases dramatically. After 90^{th} day, I becomes stable and the final number of infected people is around $7 \times 10^6 \left(1 - \frac{1}{1.4}\right) = 2$ millions. During this period, the rate of recovery equals the rate of being infected.

Conclusion:

If we want the disease to disappear, we have to make $\Re_0$ less than or equal to 1. Under the condition that β and N are fixed, γ should be maximized. The medicine used to treat the disease can increase the rate of recovery, i.e. increase γ. The SIS model still has some limitations because the number of infected people will keep constant (reach the equilibrium position) after some time. This certainly does not match with the real situation.

4. The SIR Model

In the SIR model, people are categorized into three groups: the susceptible group, the infective group and the removed group. The people in the susceptible group will become infective if they contact people who are infective. Sometimes, the infective people die or are vaccinated. They are not susceptible or infective anymore. In this case, we call these group 'removed'.

$$S \xrightarrow{\beta SI} I \xrightarrow{\gamma I} R$$

(The definitions of S, I and β are the same as mentioned in the previous chapters.)

In this chapter, R is the number of people in the removed group and γ is the probability that one random infective person becomes removed.

We can set three differential equations:

$$\frac{dS}{dt} = -\beta SI \qquad \frac{dI}{dt} = \beta SI - \gamma I \qquad \frac{dR}{dt} = \gamma I$$

Now, we define the basic reproductive ratio as

$$\Re_0 = \frac{\beta N}{\gamma} \tag{4.1}$$

When will an epidemic occur?

For convenience, let $\hat{S}, \hat{I}$ and $\hat{R}$ be the fraction of group S, I and R in the whole population respectively and we take γ^{-1} as time. [See reviewer's comment (5)]

$$\hat{S} = \frac{S}{N} \qquad \hat{I} = \frac{I}{N} \qquad \hat{R} = \frac{R}{N} \qquad \hat{t} = \gamma t \tag{4.2}$$

$$\frac{d\hat{S}}{d\hat{t}} = -\Re_0 \hat{S}\hat{I} \qquad \frac{d\hat{I}}{d\hat{t}} = \Re_0 \hat{S}\hat{I} - \hat{I} \qquad \frac{d\hat{R}}{d\hat{t}} = \hat{I}$$

An epidemic will occur if the percentage of infective group is keep on increasing,

$$\text{i.e.} \qquad \frac{d\hat{I}}{d\hat{t}} = \hat{I}(\Re_0 \hat{S} - 1), \qquad \Re_0 \hat{S} - 1 > 0 \qquad \text{with} \qquad \hat{S} \approx \hat{S}_0 \tag{4.3}$$

By (4.1) and (4.2),

$$\frac{\beta S_0}{\gamma} > 1 \tag{4.4}$$

We can know whether there will be an epidemic by substituting β, γ and S_0 into (4.4).

We have another question: What is the least number of people that have to be vaccinated if we want to prevent the epidemics?

Let p be the fraction of people that has to be vaccinated in order to prevent epidemics. The vaccinated people belong to the removed group.

The fraction of people in the susceptible group is therefore $1 - p$, i.e. $\hat{S} = 1 - p$.

By (4.3),

$$\Re_0(1 - p) > 1$$

By calculation,

$$p < 1 - \frac{1}{\Re_0} \tag{4.5}$$

An epidemic will occur if p satisfies (4.5).

Therefore, to prevent an epidemic, the least value of $p = 1 - \frac{1}{\Re_0}$.

In this chapter, we want to study the change in I and R for different values of γ. We take $N = 7 \times 10^6, \beta = 10^{-7}$. The disease becomes epidemic when

$$\Re_0 > 1 \iff \gamma < 0.7.$$

(i)

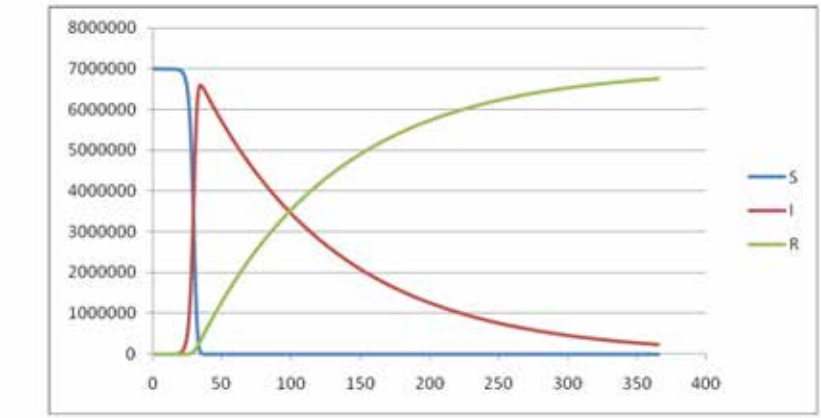


FIGURE 6. The number of people in class S, class I and class R in time t with $\beta = 10^{-7}$, $\gamma = 0.01$, $\Re_0 = 70$.

I increases drastically from the 16^{th} to the 38^{th} day. Then, I decreases from the 39^{th} day to the 365^{th} day. R increases sharply at first. As time goes on, the rate of increase of R decreases.

(ii)

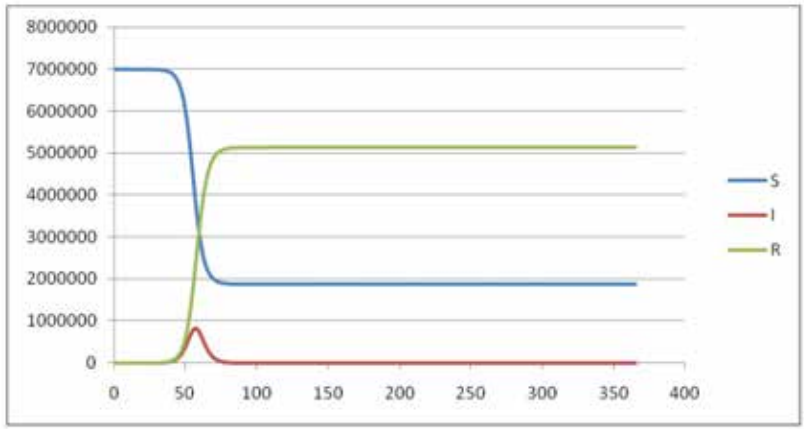


FIGURE 7. The number of people in class S, class I and class R in time t with $\beta = 10^{-7}$, $\gamma = 0.4$, $\Re_0 = 1.75$.

Compared to Figure 6, S drops at a lower rate for a longer period of time. Finally it remains constant at 1873106. On the contrary, there is a significant rise in R, which finally levels off at 5126894. I first goes up and then declines, but over a shorter period of time.

(iii)

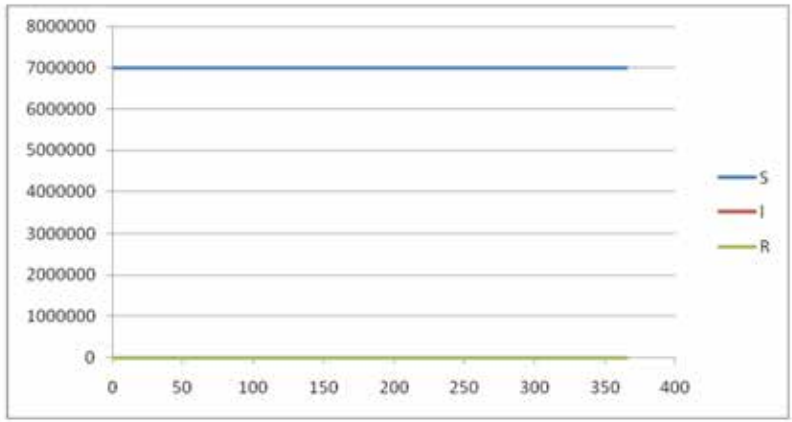


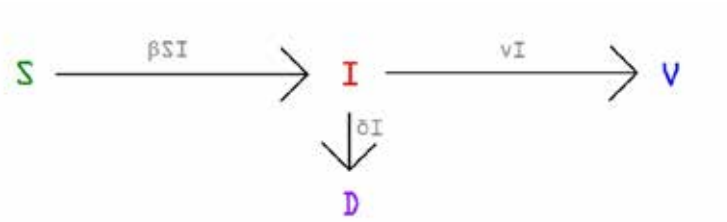
FIGURE 8. The number of people in class S, class I and class R in time t with $\beta = 10^{-7}$, $\gamma = 0.8$, $\Re_0 = 0.875$.

S remains unchanged and levels off and so is R.

From the above graphs, we can conclude that as the value of γ increases, more people will be still susceptible and less will be infected. For a constant value of β, γ determines how the disease will spread out. To reduce the number of class I, we ought to raise the value of γ. For instance, the government may offer vaccines at lower prices and separate the infected people from the susceptible ones.

5. The SIVD Model

In the SIVD model, people are characterized into four classes: susceptible S, infective I, vaccinated V and death D. Vaccinated and death individuals are no longer susceptible or infective. In the previous SIR model, the infectives move from the I class directly into the R class, but now they move into two classes, i.e. class V and D. The model can be diagramed as



We assume that the probability that an infective dies is given by δ, and ν will be the probability that an infective is vaccinated.

With a constant population size ($S + I + V + D = N$), we have the corresponding differential equations

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \nu I - \delta I, \qquad \frac{dV}{dt} = \nu I, \qquad \frac{dD}{dt} = \delta I \tag{5.1}$$

With reference to the SIR model, the infectives leave the I class with constant rate, so

$$\gamma = \delta + \nu$$

and the reproductive ratio becomes

$$\Re_0 = \frac{\beta N}{\delta + \nu} \tag{5.2}$$

In our project, we let β be the probability that a random infective person infects a random susceptible, with a value of 0.0000001 (i.e. 10^{-7}). Considering Hong Kong, we assume there is only 1 infective among the whole population ($N = 7000000$) initially, when $t = 0$.

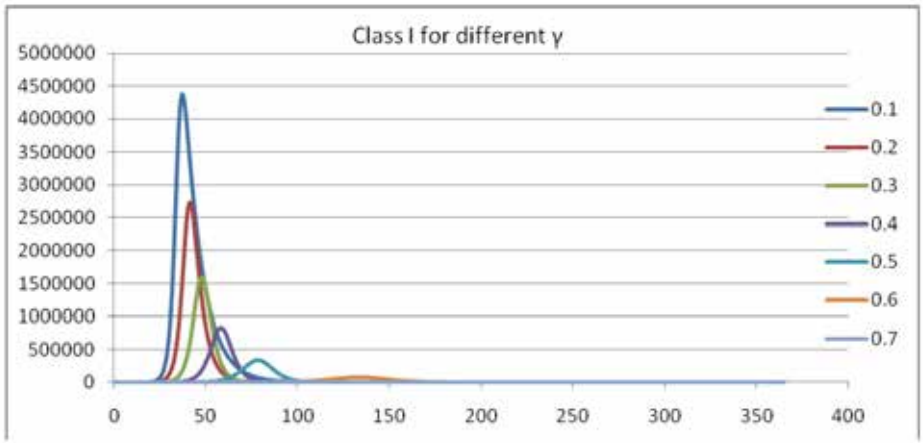


FIGURE 9. The number of people in class I for different values of γ in time t.

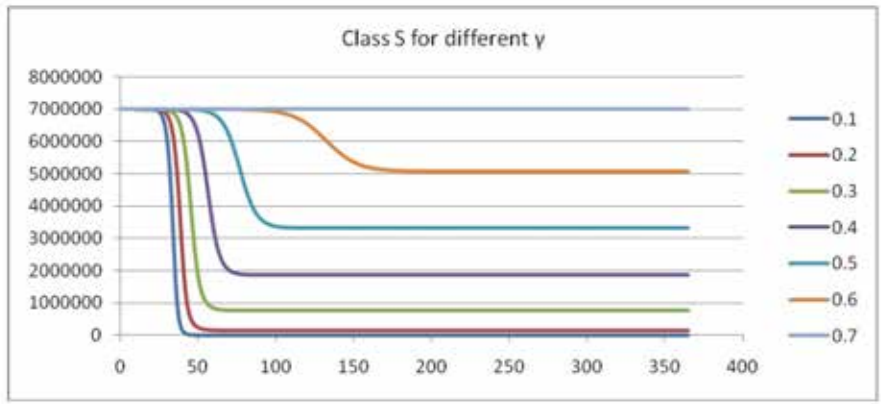


FIGURE 10. The number of people in class S for different values of γ in time t.

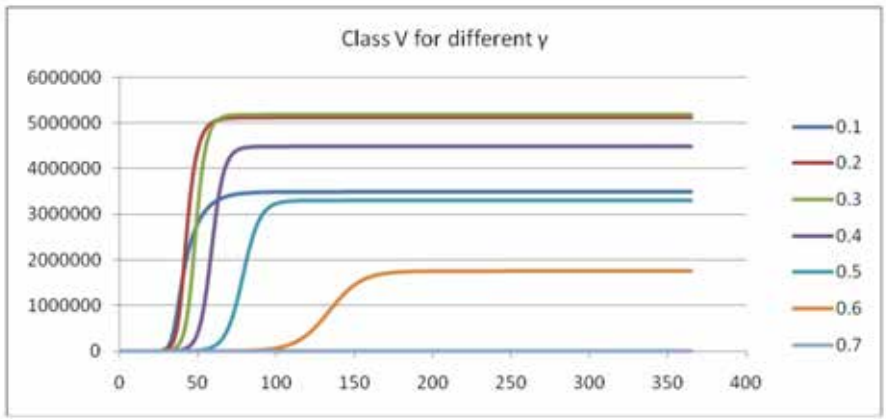


FIGURE 11. The number of people in class V for different values of γ in time t.

From Figures 9, 10 and 11, altering the value of γ will affect the number and the peak value of infectives, as well as the time of outbreak of disease. As γ increases, the number of infectives rises to the peak value in a shorter time and with a larger value. Moveover, the number of people who are susceptible will be bounded below by a larger value. However, the number of people who are vaccinated has a special pattern as γ varies.

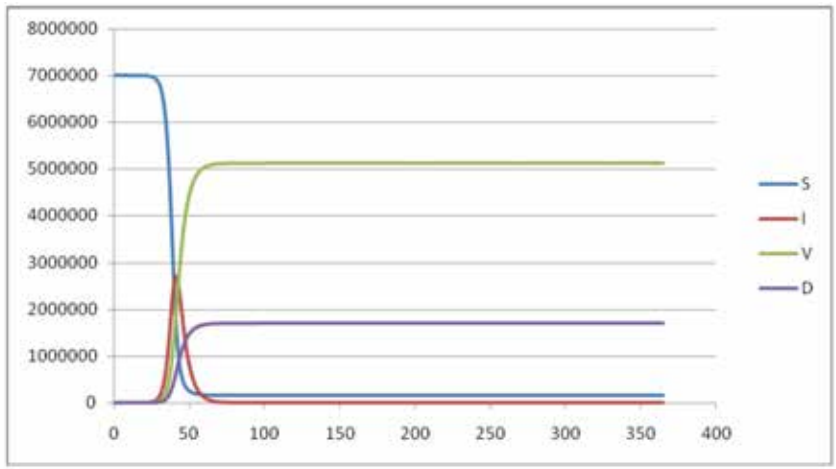


FIGURE 12. The number of people in class S, class I, class R and class D in time t with $\beta = 0.0000001, \delta = 0.05, \nu = 0.15, \gamma = 0.20$.

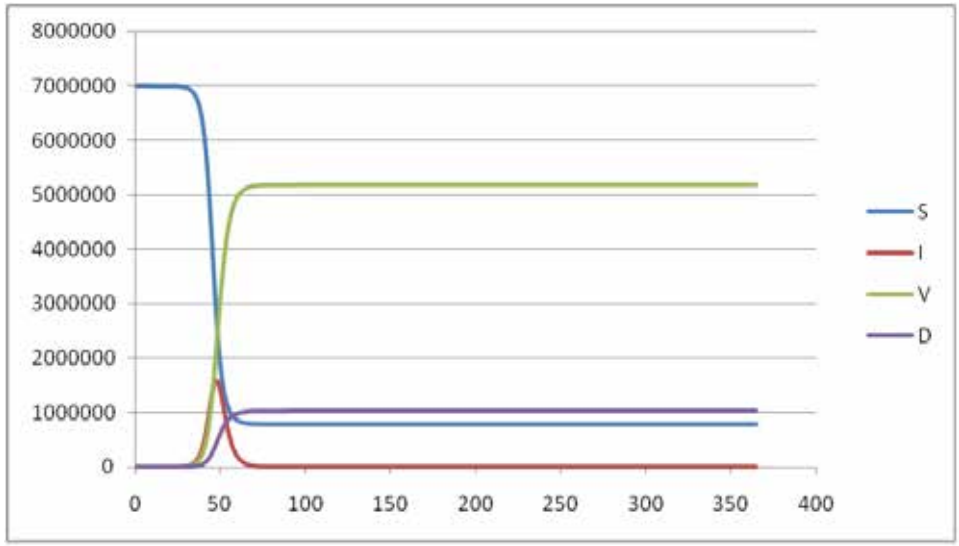


FIGURE 13. The number of people in class S, class I, class R and class D in time t with $\beta = 0.0000001, \delta = 0.05, \nu = 0.25, \gamma = 0.30$.

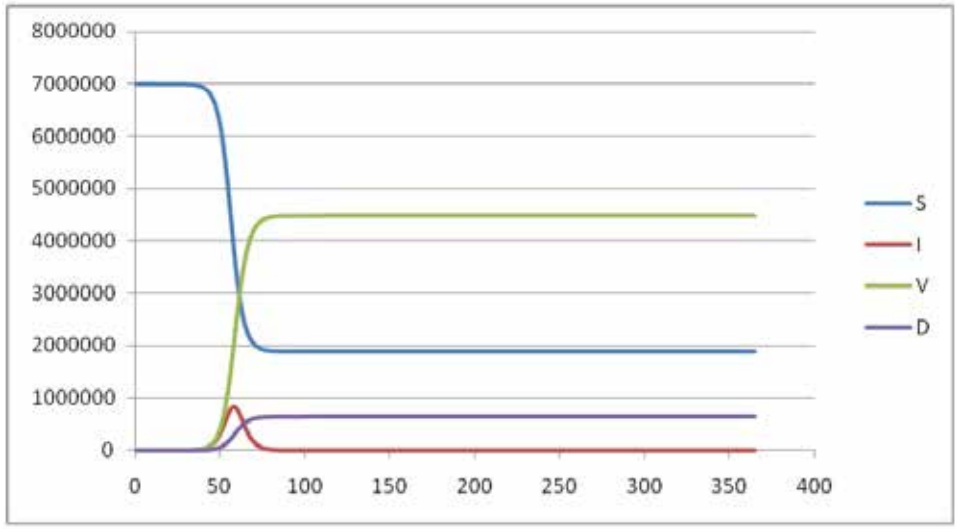


FIGURE 14. The number of people in class S, class I, class R and class D in time t with $\beta = 0.0000001, \delta = 0.05, \nu = 0.35, \gamma = 0.40$.

6. The SISR Model

We develop the SIR Model in Chapter 3 to a slightly more complicated model for infectious diseases, SISR Model, where we assume that the infective people may become susceptible again. People are also characterized into three classes: susceptible S, infective I and removed R. We assume that the return of class I to class S occurs at a rate proportional to the population of infective people. The model may be diagrammed as

$$S \underset{\mu \mathrm{I}}{\overset{\beta \mathrm{SI}}{\rightleftarrows}} I \xrightarrow{\gamma I} R$$

and the corresponding coupled differential equations are

$$\frac{dS}{dt} = -\beta SI + \mu I, \qquad \frac{dI}{dt} = \beta SI - \mu I - \gamma I, \qquad \frac{dR}{dt} = \gamma I, \tag{6.1}$$

with the constant population constraint $S + I + R = N$. For convenience, we nondimensionalize (2.1) using N for population size and γ^{-1} for time; that is, let

$$\hat{S} = \frac{S}{N}, \qquad \hat{I} = \frac{I}{N}, \qquad \hat{R} = \frac{R}{N}, \qquad \hat{t} = \gamma t \tag{6.2}$$

and define the basic reproductive ratio

$$\Re_0 = \frac{\beta N}{\gamma} \tag{6.3}$$

The nondimensioal SISR equations are

$$\frac{d\hat{S}}{d\hat{t}} = -\Re_0 \hat{S}\hat{I} + \frac{\mu}{\gamma}\hat{I}, \qquad \frac{d\hat{I}}{d\hat{t}} = \Re_0 \hat{S}\hat{I} - \frac{\mu}{\gamma}\hat{I} - \hat{I}, \qquad \frac{d\hat{R}}{d\hat{t}} = \hat{I}, \tag{6.4}$$

with nondimensional constraint $\hat{S} + \hat{I} + \hat{R} = 1$.

An epidemic occurs when a small number of infective is introduced into a susceptible population results in an increasing number of infective. The linear stability problem may be solved by considering only the equation for $d\hat{I}/d\hat{t}$ in (6.4). At $t = 0$, $\hat{I} > 0$ and $\hat{S} \approx \hat{S}_0$, we have

$$\frac{d\hat{I}}{d\hat{t}} = (\Re_0 \hat{S}_0 - \frac{\mu}{\gamma} - 1)\hat{I} > 0,$$

so that an epidemic occurs if $\Re_0, \hat{S}_0 - \dfrac{\mu}{\gamma} - 1 > 0$. With the basic reproductive ratio given by (6.3), and $\hat{S}_0 = S_0/N$, where S_0 is the number of initial susceptible individuals, an epidemic occurs if

$$\frac{\beta S_0}{\gamma + \mu} > 1 \tag{6.5}$$

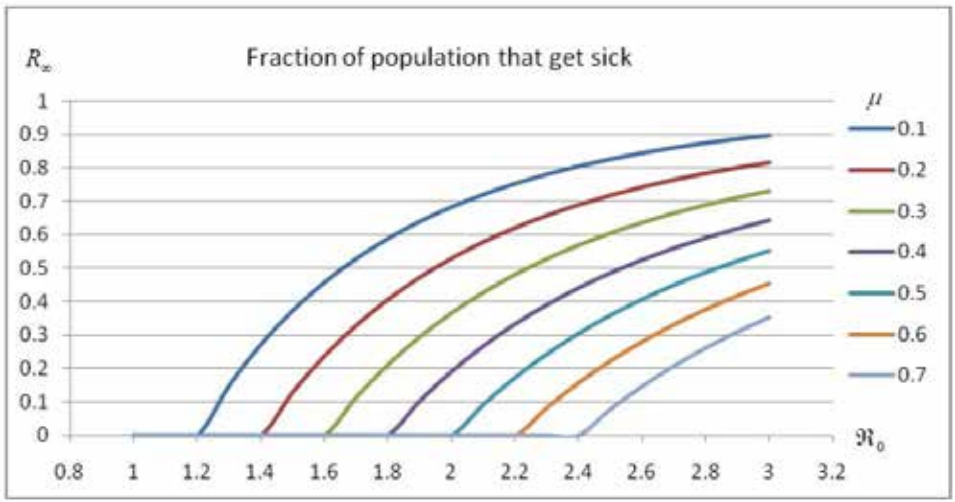


FIGURE 15. The fractions of the population that get sick in the SISR model as a function of the basic reproduction ratio $\Re_0$ with different values of μ.

From Figure 15, we can see that with the increase of the reproduction ratio $\Re_0$, the fractions of population that get sick increase and with the increase of the rate that class I return to class S, μ, the fractions of population that get sick decrease.

As we do not want an epidemic to occur, the value of $\Re_0$ should decrease or the value of μ should increase. As $\Re_0 = \beta N/\gamma$ by (6.3), $\Re_0$ is proportional to β and inversely proportional to γ, we can also decrease the value of β or increase the value of γ to decrease the value of $\Re_0$.

If an epidemic occurs, we want to find the fraction of the population gets sick in order to know how serious the epidemic is. We can use the equations in (6.4) to calculate the fraction of the population gets sick, $\hat{R}_\infty$. To compute $\hat{R}_\infty$, it is simpler to work with a transformed version of (6.4). By the chain rule,

$$d\hat{S}/d\hat{t} = (d\hat{S}/d\hat{R})/(d\hat{R}/d\hat{t}),$$

so that

$$\frac{d\hat{S}}{d\hat{R}} = \frac{d\hat{S}d\hat{t}}{d\hat{R}/d\hat{t}} = -\Re_0\hat{S} + \frac{\mu}{\gamma}.$$

Using the integrating factor, the equation becomes

$$\frac{d}{d\hat{R}}e^{\Re_0\hat{R}}\hat{S} = \frac{\mu}{\gamma}e^{\Re_0\hat{R}}.$$

By integrating the equation,

$$\hat{S} = \frac{\mu}{\gamma \Re_0} + Ce^{-\Re_0 \hat{R}}.$$

Putting the initial condition, $\hat{R} = 0$ and $\hat{S} = 1$ into the above equation, we can find

$$\hat{S} = \frac{\mu}{\gamma \Re_0} + (1 - \frac{\mu}{\gamma \Re_0})e^{-\Re_0 \hat{R}}.$$

We define that when $\hat{R} = \hat{R}_\infty$, $\hat{S} = 1 - \hat{R}_\infty$ and $\hat{I} = 0$,

$$1 - \hat{R}_\infty = \frac{\mu}{\gamma \Re_0} + (1 - \frac{\mu}{\gamma \Re_0})e^{-\Re_0 \hat{R}}.$$

Rearrange the equation

$$1 - \hat{R}_\infty - \frac{\mu}{\gamma \Re_0} - (1 - \frac{\mu}{\gamma \Re_0})e^{-\Re_0 \hat{R}} = 0.$$

We can compute the value of $\hat{R}_\infty$ by putting the values of $\mu, \gamma, \Re_0$ into (6.6), so we can know how serious the epidemic is and take immediate action to prevent the disease from spreading out.

We want to study how S, I, R change with time t by substituting different values of β, γ, μ. Here, we assume that the values of γ and μ will not be too small. The values of γ and μ cannot be too small, this SISR model will be similar to the SIS model. On the other hand, if the value of μ is too small, this model will be similar to the SIR model. From (6.5), we know that an epidemic occurs if $\beta S_0/(\gamma + \mu) > 1$. By putting $\beta = 10^{-7}$ and $S_0 = 7 \times 10^6$, we know an epidemic occurs when $\gamma + \mu < 0.7$. So we substitute different values of γ and μ where $\gamma + \mu < 0.7$ to see the changes of the number of people in different groups.

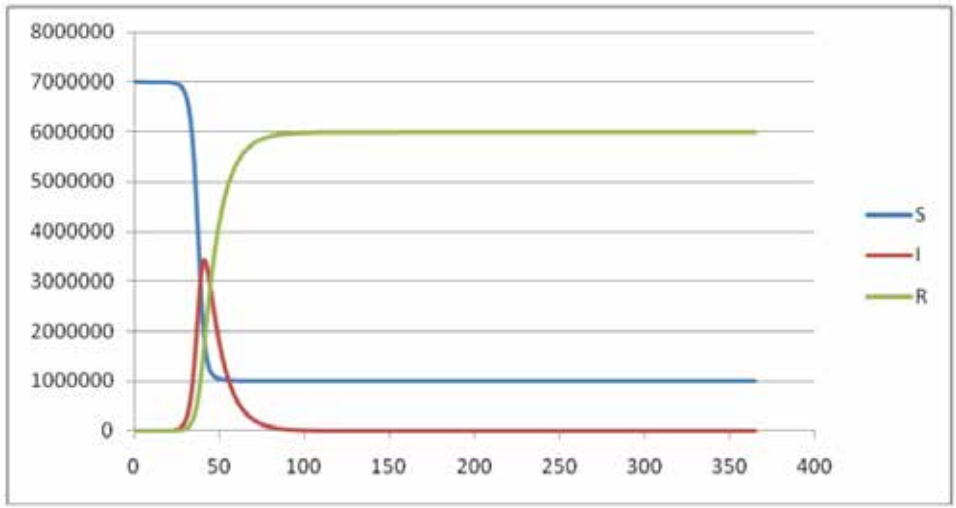


FIGURE 16. The number of people in class S, class I and class R in time t with $\gamma = 0.1$ and $\mu = 0.1$.

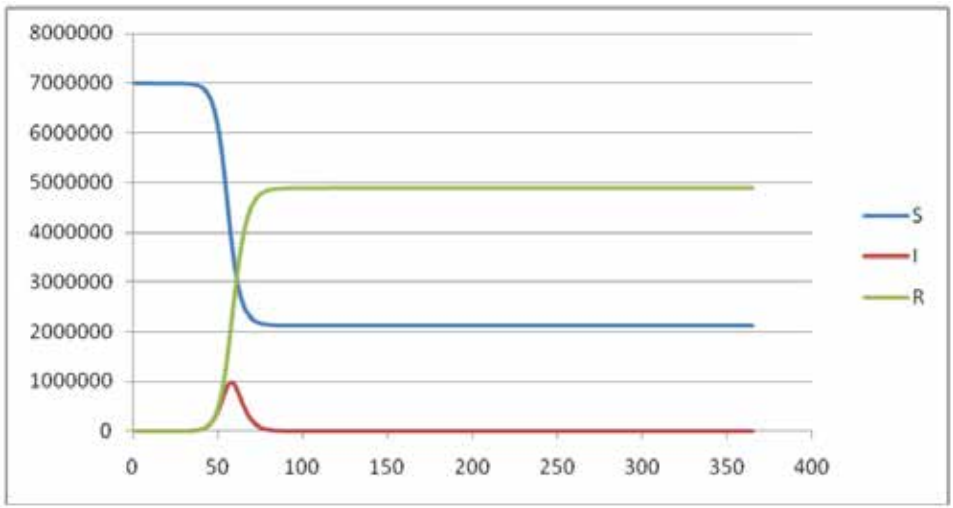


FIGURE 17. The number of people in class S, class I and class R in time t with $\gamma = 0.3$ and $\mu = 0.1$.

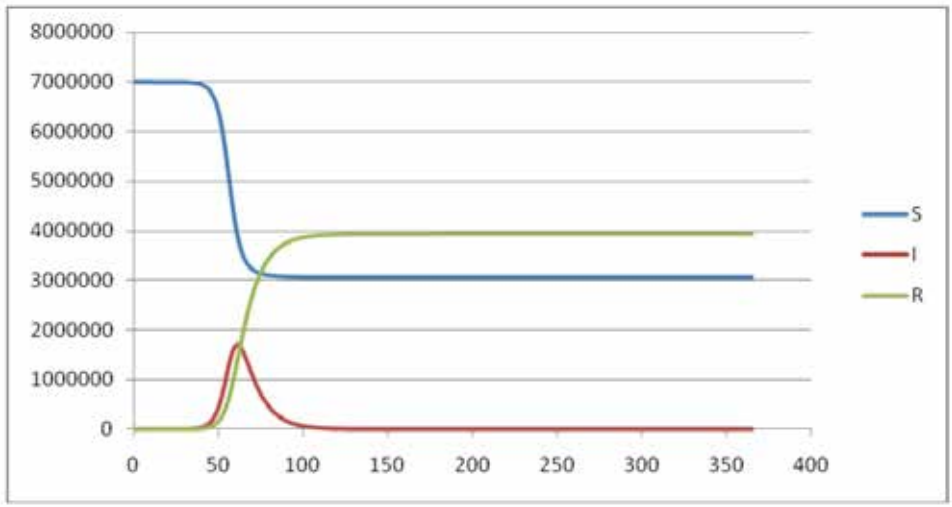


FIGURE 18. The number of people in class S, class I and class R in time t with $\gamma = 0.1$ and $\mu = 0.3$.

From Figure 16, 17,18, we can see that when the value of γ or μ increases, the rate of change of I will decrease and so there are less people become infective and more people remain susceptible.

To prevent infectious diseases from spreading out, the government should provide medicine to the patients, so the recover rate will be higher. The values of γ or μ will then increase, the rate of change of infective people will decrease. Therefore, the diseases will not spread out.

7. The SIRS Model

In this chapter, the SIRS Model we introduced is similar to the SIR Model and the SISR Model. Here, we assume that the recovered people may lose their immunity and become susceptible again. Tuberculosis is one of the diseases of the SIRS model. We define that the return of class R to class S occurs at a rate proportional to the population of recovered people. The model may be diagrammed as

$$S \xrightarrow{\beta SI} I \xrightarrow{\gamma I} R \xrightarrow{\mu\gamma R} S,$$

and the corresponding coupled differential equations are

$$\frac{dS}{dt} = -\beta SI + \mu R, \qquad \frac{dI}{dt} = \beta SI - \gamma I, \qquad \frac{dR}{dt} = \gamma t - \mu R \tag{7.1}$$

with the constant population constraint $S + I + R = N$. We define the basic reproductive ratio

$$\Re_0 = \frac{\beta N}{\gamma} \tag{7.2}$$

The nondimensioal SISR equations are

$$\frac{d\hat{S}}{d\hat{t}} = -\Re_0 \hat{S}\hat{I} + \frac{\mu}{\gamma}\hat{R}, \quad \frac{d\hat{I}}{d\hat{t}} = \Re_0 \hat{S}\hat{I} - \hat{I}, \quad \frac{d\hat{R}}{d\hat{t}} = \hat{I} + \frac{\mu}{\gamma}\hat{R} \tag{7.3}$$

with nondimensional constraint $\hat{S} + \hat{I} + \hat{R} = 1$.

Similarly, an epidemic occurs when a small number of infective introduced into a susceptible population results in an increasing number of infective. This may be solved by considering only the equation for $d\hat{I}/d\hat{t}$ in (7.3). At $t = 0, \hat{I} > 0$, we have

$$\frac{d\hat{I}}{d\hat{t}} = (\Re_0 \hat{S}_0 - 1)\hat{I}$$

so that an epidemic occurs if $\Re_0 \hat{S}_0 - 1 > 0$. With the basic reproductive ratio given by (7.2), and $\hat{S}_0 = S_0/N$, where S_0 is the number of initial susceptible individuals, an epidemic occurs if

$$\frac{\beta S_0}{\gamma} > 1 \tag{7.4}$$

This result is the same as the SIR Model because the rate of change of the infective group is the same. The difference between the two models is the rate of change of the susceptible group and recovered group.

Let (S_*, I_*, R_*) be the fixed point of (7.1). The first equilibrium point is at the beginning, i.e. $(N - 1, 1, 0)$. We found that another equilibrium point when

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \tag{7.5}$$

From the equations in (7.1), we can deduce that this occurs when

$$\beta SI = \gamma I = \mu R \tag{7.6}$$

By solving (7.6) and $S + I + R = N$, we can have

$$(S_*, I_*, R_*) = \left(\frac{\gamma}{\beta}, \frac{\mu\left(N - \frac{\gamma}{\beta}\right)}{\gamma + \mu}, \frac{\gamma\left(N - \frac{\gamma}{\beta}\right)}{\gamma + \mu} \right) \tag{7.7}$$

We can see that if $N < \frac{\gamma}{\beta}$, the number of people in class I and R will be negative and this will not be the real case in our life, so we say that this equilibrium point only exists when $N \geq \frac{\gamma}{\beta}$. This quantity $\frac{\gamma}{\beta}$ is the threshold level for the disease.

From (7.4), an epidemic occurs if $\beta S_0/\gamma > 1$. After substituting $S_0 = N$ and rearranging the term, we can have an epidemic occurs if

$$N \geq \frac{\gamma}{\beta} \tag{7.8}$$

Therefore, when $N < \frac{\gamma}{\beta}$, an epidemic will not occur and $(S_*, I_*, R_*) = (N, 0, 0)$ when $t \to \infty$.

We found that when it reaches the equilibrium point (7.7) with the constant values of β, γ, μ. The number of people will be unchanged as the equilibrium point (7.7) after any time t. It is because the number of people entering and leaving class S, I and R at a time interval Δt is the same. When $t \to \infty$, this equilibrium point (7.7) will also occur.

Here, we substitute the values of N, β, γ, μ to see the changes among class S, I and R with time. In this model, we also assume $N = 7000000$ and $\beta = 10^{-7}$.

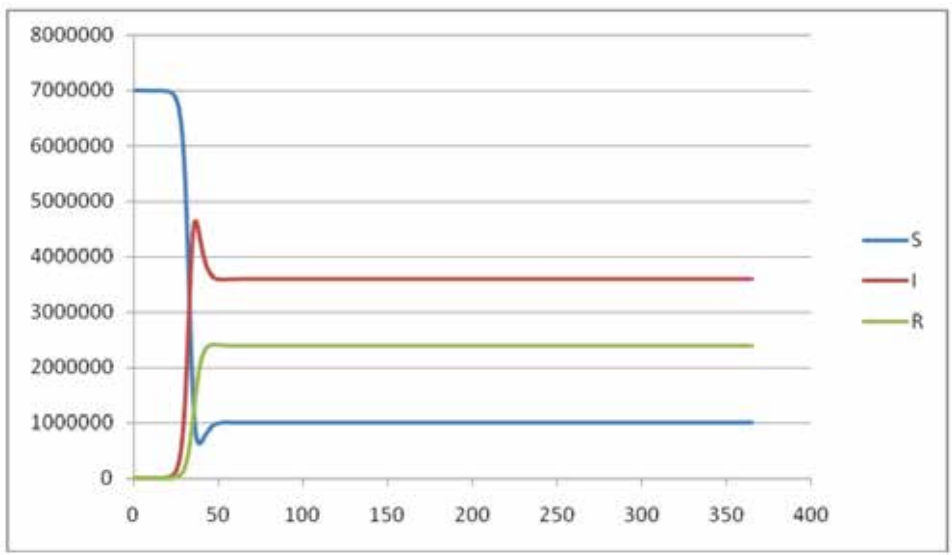


FIGURE 19. The number of people in class S, class I and class R in time t with $\gamma = 0.1$ and $\mu = 0.15$.

After putting the values of N, β, γ, μ into the equilibrium point in (7.7), we have

$$(S_*, I_*, R_*) = (1000000, 3600000, 2400000)$$

It has the same result as Figure 19.

We found that it reaches this equilibrium point after 90 days. [See reviewer's comment (6)] After 90 days, there are same number of people from class S, I, R go to class I, R, S respectively in each period.

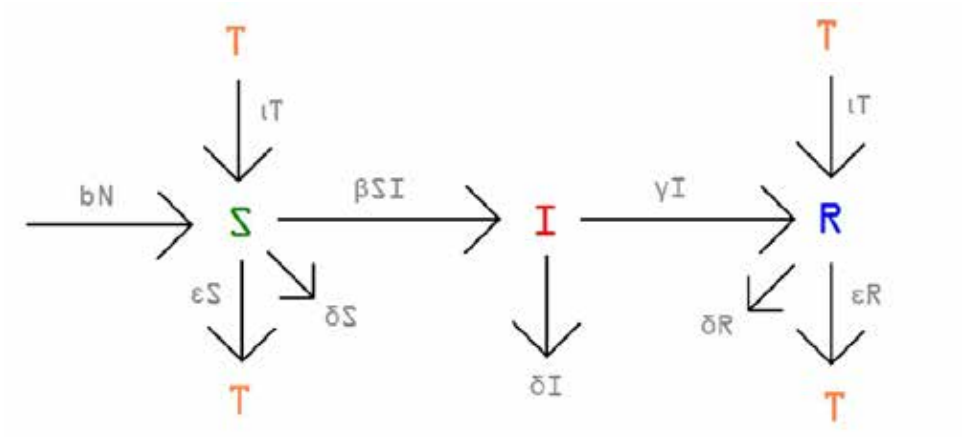
Does it mean the number of susceptible people, infective people, recovered people remain steady after a period of time in our daily life? If not, then what is the use of above theory?

Actually, it is impossible to happen in our daily life. As the development of the medical treatments, the value of γ will increase in a short period of time and there is less people become infective. Also, everyone has different measures to prevent being infected, so the value of β is not the same in every estate. Therefore the real case of the infectious disease will absolutely be different from the above model.

However, we can use the number of infective people in the first few days to estimate the values of β, γ, μ and use the above model to estimate how many people will be affected by this infectious disease if we do nothing to the disease where the values of β, γ, μ remains the same as the beginning. So we can know how serious is the disease and the authority can take action to prevent the disease from spreading out.

8. The TSIR Model

In this chapter, the TSIR Model that we are going to introduce is similar to the SIR Model. People are characterized into four classes in this model: tourist T, susceptible S, infective I and removed R. We assume that there are a fixed number of tourists coming to Hong Kong per day, which are about 94000 after knowing that there are about 8.6 million tourists per season. We define that every tourist coming to Hong Kong will either belong to class S or class R since an infective can hardly pass the health test before they travel to Hong Kong. We also assume that the immigration rate ι is inversely proportional to the population of the entire world and the emigration rate ϵ is proportional to the population of class S and class R. And this time, birth rate b and death rate δ are also introduced to the model. The model can be diagrammed as



We have the corresponding differential equations

$$\frac{dT}{dt} = \epsilon S + \epsilon R - 2\iota T, \frac{dS}{dt} = bN + \iota T - \beta SI - \delta S - \epsilon S,$$
$$\frac{dI}{dt} = \beta SI - \gamma I - \delta I, \frac{dR}{dt} = \gamma I + \iota T - \delta R - \epsilon R \tag{8.1}$$

[See reviewer's comment (7)]

To make things easy, we assume the population size is constant ($S + I + R = N$), and birth rate equals to death rate, i.e. $b = \delta$. Therefore T is a constant,

$$\frac{dT}{dt} = \epsilon S + \epsilon R - 2\iota T = 0,$$

and the emigration rate ϵ is given by

$$\epsilon = 2\iota T/(S + R),$$

and define the basic reproductive ratio

$$\Re_0 = \frac{\beta N}{\gamma} \tag{8.2}$$

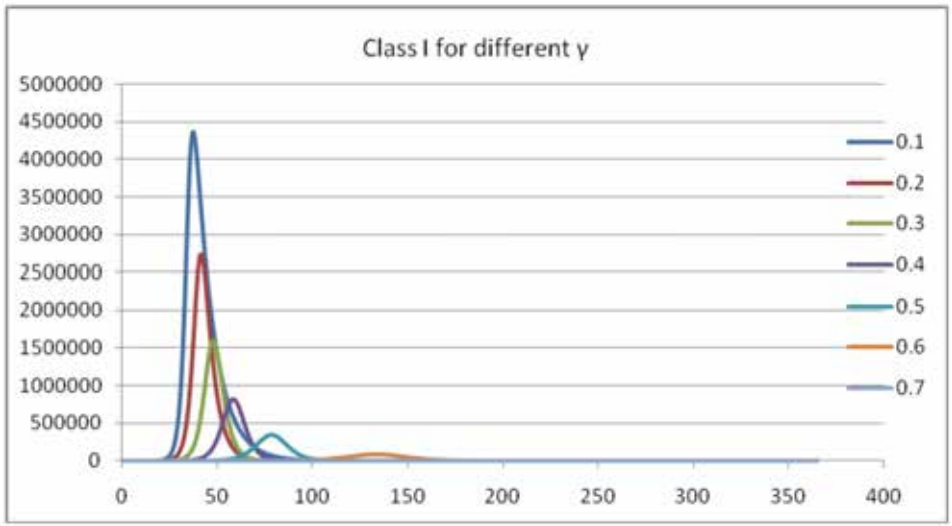


FIGURE 20. The number of people in class I for different values of γ in time t.

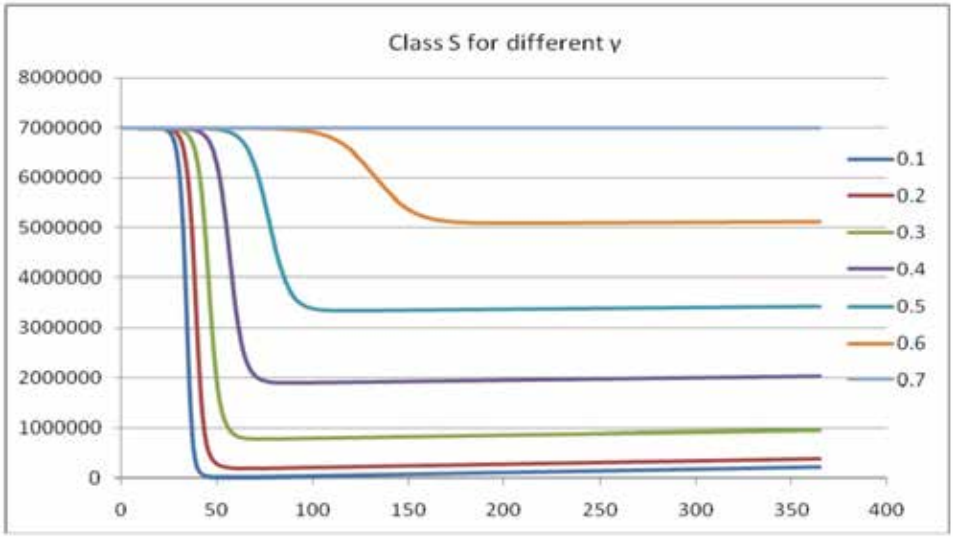


FIGURE 21. The number of people in class S for different values of γ in time t.

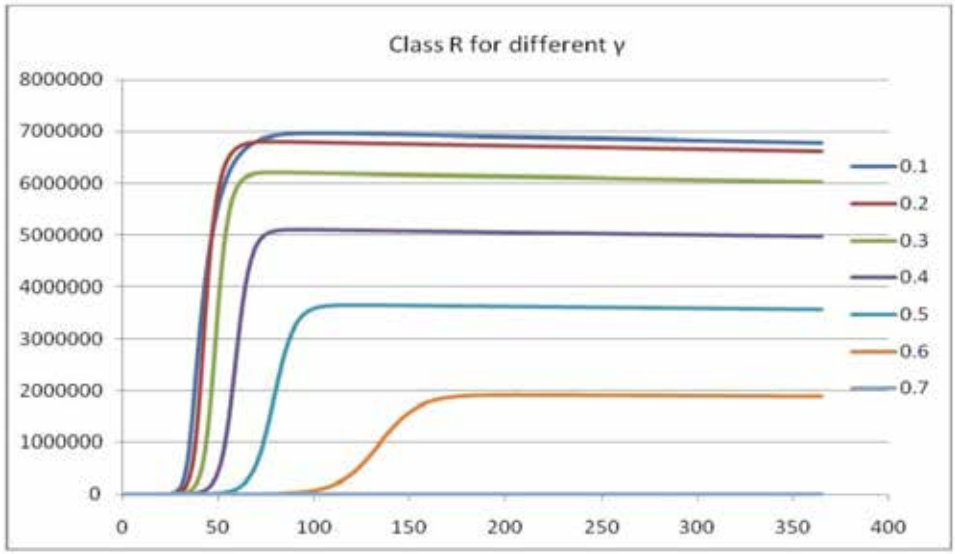


FIGURE 22. The number of people in class R for different values of γ in time t.

The immigration rate ι is equal to 94000 (number of tourists coming to Hong Kong per day) divided by 1.2×10^{10} (the population of the entire world). As it is rather complicated for our group to solve the differential equations, MS Excel is employed to find the approximate value of each class.

From the graphs above, as γ increases, the number of infectives rises to the peak value in a shorter time and with a larger value. Moveover, the number of people who are susceptible will reach its minimum value in a later time and by a larger value. The class R will reach its maximum in a later time and by a smaller value.

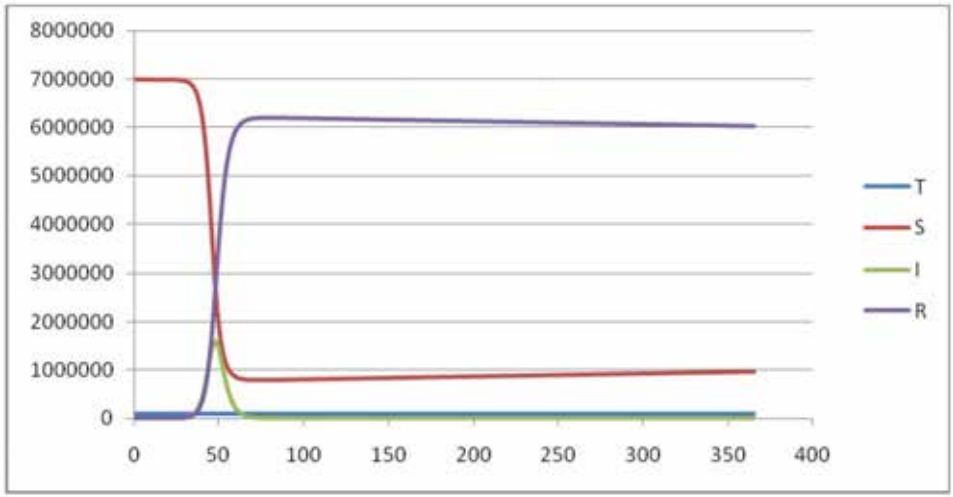


FIGURE 23. The number of people in class T, class S, class I and class R in time t with $\beta = 0.0000001, b = \delta = 0.0001, \gamma = 0.30$.

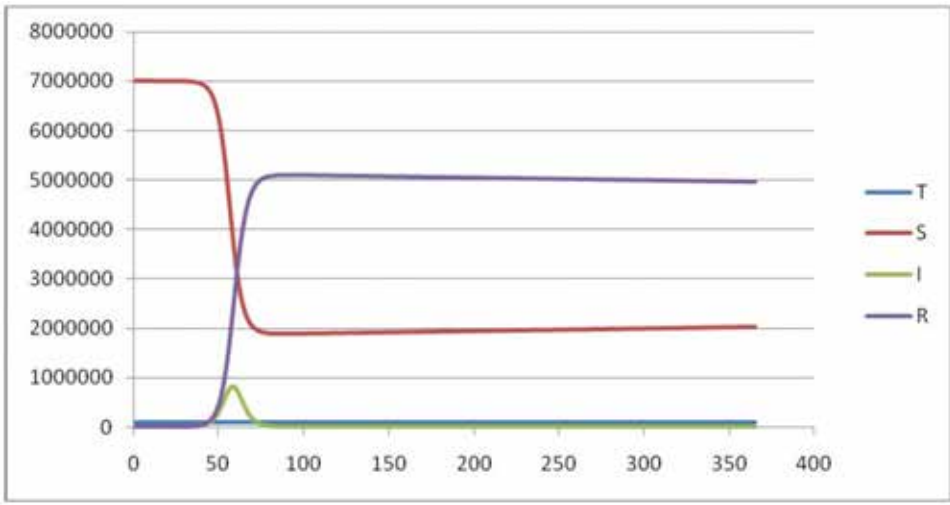


FIGURE 24. The number of people in class T, class S, class I and class R in time t with $\beta = 0.0000001, b = \delta = 0.0001, \gamma = 0.40$.

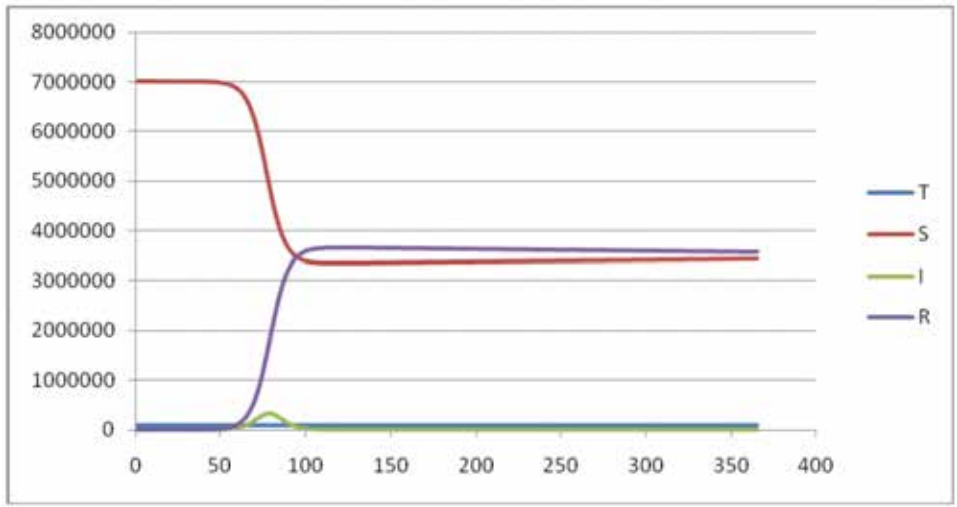


FIGURE 25. The number of people in class T, class S, class I and class R in time t with $\beta = 0.0000001, b = \delta = 0.0001, \gamma = 0.50$.

However, there are still rounding-off errors in calculations; making the total population N not constant but increasing. Hence, class S will increase in long term and class R will decrease in long term.

If we change the birth rate and death rate, the curves of class S, I and R create special patterns.

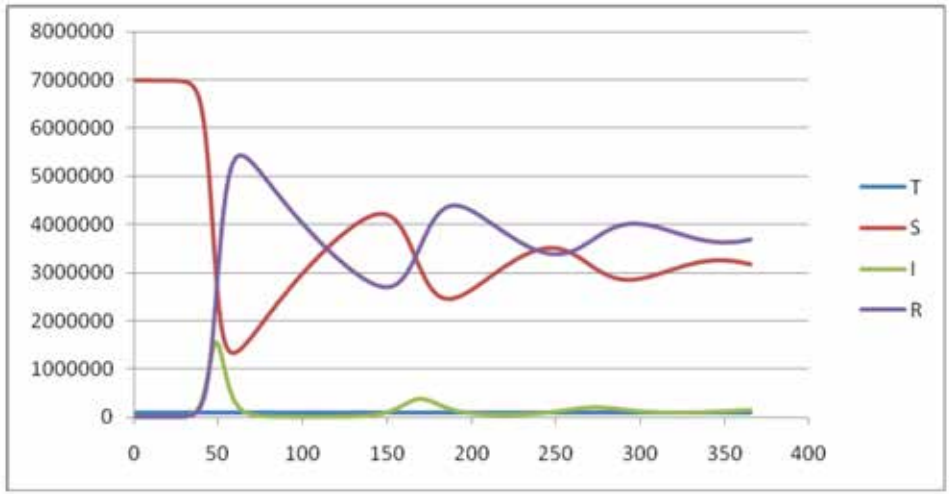


FIGURE 26. The number of people in class T, class S, class I and class R in time t with $\beta = 0.0000001, b = \delta = 0.01, \gamma = 0.30$.

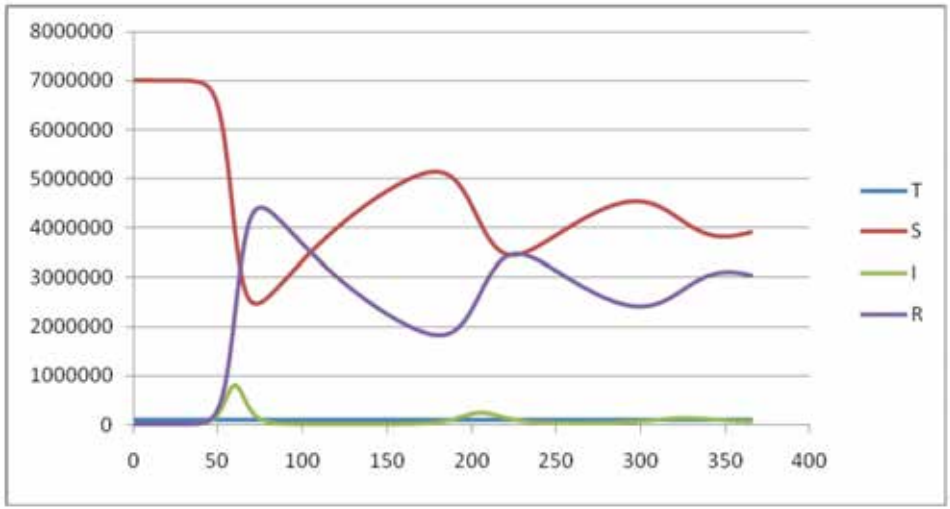


FIGURE 27. The number of people in class T, class S, class I and class R in time t with $\beta = 0.0000001, b = \delta = 0.01, \gamma = 0.40$.

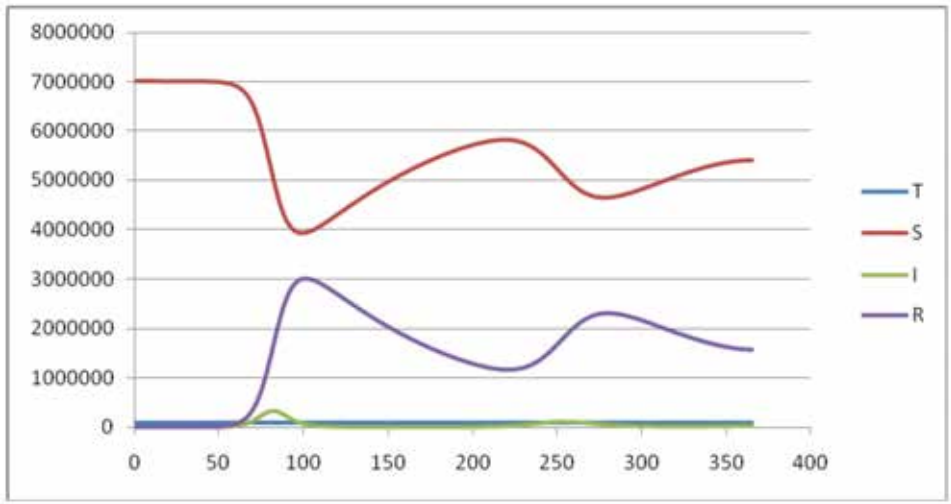


FIGURE 28. The number of people in class T, class S, class I and class R in time t with $\beta = 0.0000001, b = \delta = 0.01, \gamma = 0.50$.

From Figures 8.7, 8.8 and 8.9, the shapes of the lines representing class S, class I and class R become more complicated when the birth rate and death rate are increased to 0.01. The lines seem to be showing a particle undergoing damped simple harmonic motion. The frequency of oscillations is higher when γ is smaller.

9. The SEIR Model

The SEIR Model is developed from the SIR model and is different from the model in the above chapter. We add one more type of people in the model, the exposed people (E). These people have been infected by the disease but they are not yet infectious. They may not know that they are affected as they do not have any characteristics of being sick. We assume that the rate of the exposed people become the infectious people is directly proportional to the number of exposed people. Here, we also do not consider the birth rate and the death rate first. The model may be diagrammed as

$$S \xrightarrow{\beta SI} E \xrightarrow{\alpha E} I \xrightarrow{\gamma I} R,$$

and the corresponding coupled differential equations are

$$\frac{dS}{dt} = -\beta SI, \frac{dE}{dt} = \beta SI + \alpha E, \frac{dI}{dt} = \alpha E - \gamma I, \frac{dR}{dt} = \gamma I \tag{9.1}$$

[See reviewer's comment (8)]

with the constant population constraint $S + I + R = N$ and $0 < \alpha \leq 1$.

We define the basic reproductive ratio

$$\Re_0 = \frac{\beta N}{\gamma}. \tag{9.2}$$

Now, we consider two cases of this model. The difference between them is the initial conditions.

Case 1: $S(0) = 6999999, E(0) = 1, I(0) = 0$ and $R(0) = 0$

This case assumes that there is an exposed person at the beginning as one should be exposed first before being infectious. We consider the initial time at the first person who is affected and the one is exposed. This case is more theoretical.

Case 2: $S(0) = 6999999, E(0) = 0, I(0) = 1$ and $R(0) = 0$

This case assumes that there is an infectious person at the beginning as we only know one got the disease after getting the characteristics of the disease, otherwise we do not know one is affected. Here, we consider the initial time at the first person who is affected and the one is infectious. This case is more like the real case in our daily life.

To calculate when an epidemic occurs, we have to use $dE/dt + dI/dt > 0$ instead of using $dI/dt > 0$ because the exposed people are already affected by the disease and will become infectious after a short period of time, so they will spread out the disease. We use the initial conditions of the above cases, we have

$$E(0) + I(0) = 1. \tag{9.3}$$

If $\dfrac{dE}{dt} + \dfrac{dI}{dt} \leq 0$, the number of people in these two group will not increase and we have

$$E(t) + I(t) \leq 1. \tag{9.4}$$

so the disease will not spread out. The epidemic occurs when

$$E(t) + I(t) > 0. \tag{9.5}$$

To solve this inequality, we have to consider the equations in (9.1). We have

$$\frac{dE}{dt} + \frac{dI}{dt} = (\beta S - \gamma)I > 0. \tag{9.6}$$

so that an epidemic occurs if $\beta S - \gamma > 0$. After rearranging the term,

$$\frac{\beta S}{\gamma} > 1. \tag{9.7}$$

From (9.2), $\Re_0 = \beta N/\gamma$ and $S_0 \approx N$, so we can say an epidemic occurs when

$$\Re_0 > 1$$

If $\Re_0 \leq 1, I(t) \leq 1$ in any time t. As the number of people is not zero at the beginning, it will still affect some susceptible people theoretically and they will become exposed. Therefore $\lim_{t\to\infty}(S(t), E(t), I(t), R(t)) \neq (N-1, 0, 0, 1)$. However, one will become recovered in a very short period of time in our daily life, so this will not affect a lot of the susceptible people. We have $\lim_{t\to\infty}(S(t), E(t), I(t), R(t))$ when $\Re_0 \leq 1$ is $(N-n, 0, 0, n)$ where n is a very small number.

Now we want to see if there are any more differences between the two cases. We plot the graphs by substitute the same value of β, α, γ where $\Re_0 > 1$ in both cases to see the changes among class S, I and R with time.

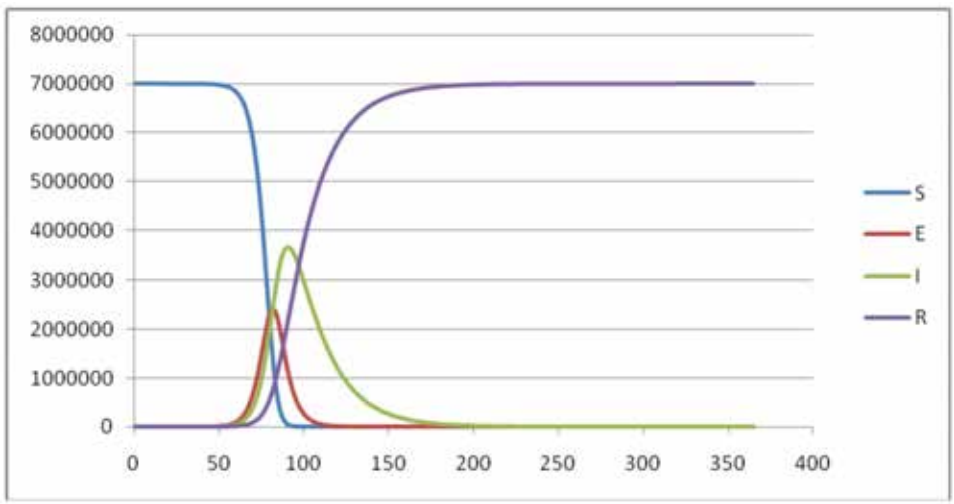


FIGURE 29. The number of people in class S, class E, class I and class R in time t with $\beta = 10^{-7}, \alpha = 0.15$ and $\gamma = 0.05$ where $S(0) = 6999999, E(0) = 1, I(0) = 0$ and $R(0) = 0$.

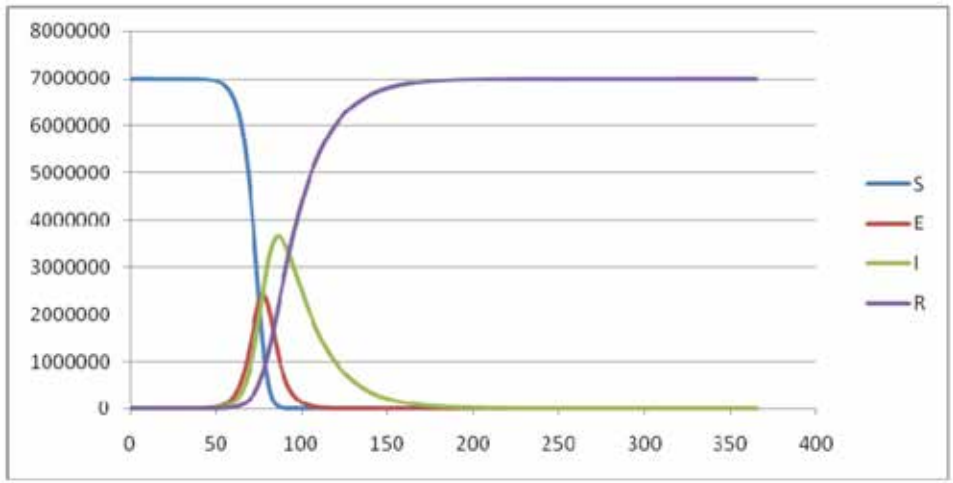


FIGURE 30. The number of people in class S, class E, class I and class R in time t with $\beta = 10^{-7}, \alpha = 0.15$ and $\gamma = 0.05$ where $S(0) = 6999999, E(0) = 0, I(0) = 0$ and $R(0) = 0$.

The shapes of the curves in Figure 29 are very similar to that in Figure 30. The difference is that the curve of class E and class I in case 2 (Figure 30) reaches the peak value a few days earlier than that in case 1 (Figure 29). Both cases go to the same equilibrium position at the end. So we can see that both cases will give similar results while case 2 will go to the peak values a bit faster than case 1.

For convenience, we will use case 2 to study SEIR in our following study and discussion, so we do not need to consider two cases in our further study.

Now, we want to study if we increase the value of α, γ where $\Re_0 > 1$ to see the differences between them.

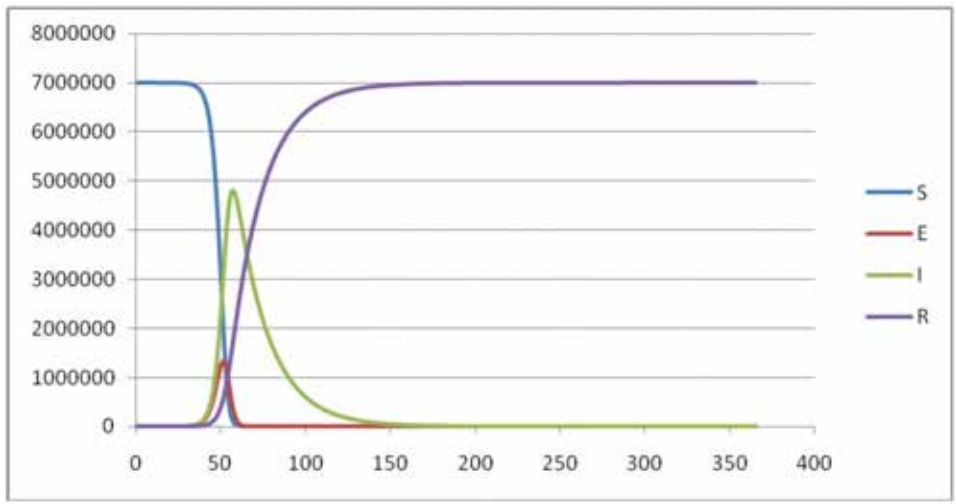


FIGURE 31. The number of people in class S, class E, class I and class R in time t with $\beta = 10^{-7}, \alpha = 0.5$ and $\gamma = 0.05$ where $S(0) = 6999999, E(0) = 0, I(0) = 1$ and $R(0) = 0$.

By comparing Figure 31 with the Figure 30, we can see that the number of infective people reaches its peak value earlier and has a higher peak value when the value of α increases. The number of recovered people at the end between these two figures is the same. This shows that the changes of α will only affect the time and the people of peak value of class I while the number of people in each class after a long period of time is the same.

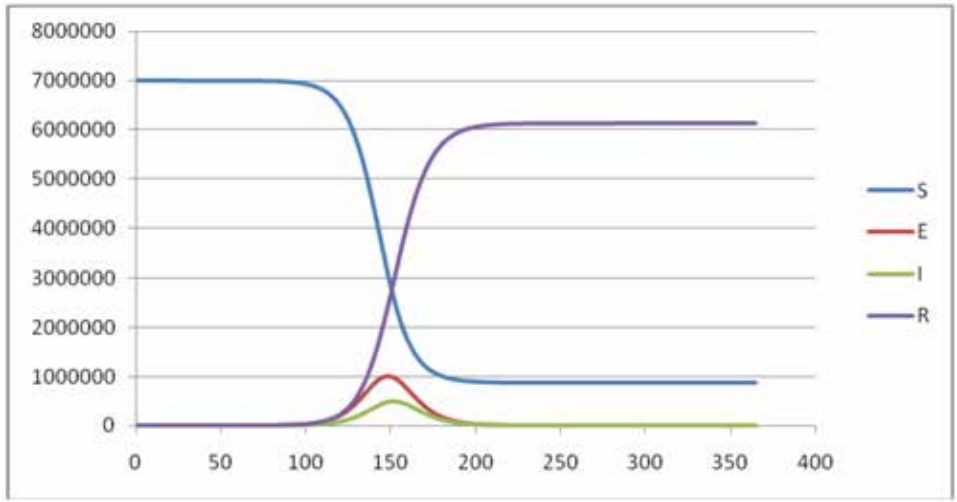
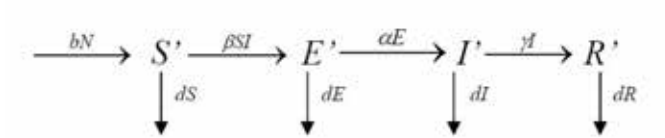


FIGURE 32. The number of people in class S, class E, class I and class R in time t with $\beta = 10^{-7}, \alpha = 0.15$ and $\gamma = 0.3$ where $S(0) = 6999999, E(0) = 0, I(0) = 1$ and $R(0) = 0$.

By comparing Figure 32 with Figure 30, we can see that the number of infective people reaches its peak value later and has a lower peak value when the value of γ increases. The number of recovered people at the end is less than that in Figure 30. This shows that the change of γ will affect the time and the peak value of class I. Also the number of people in class S and class R are also affected.

Now, we add the birth rate and the death rate into the model. We let b be the birth rate and d be the death rate. The model may be diagrammed as



and the corresponding coupled differential equations are

$$\frac{dS'}{dt} = bN - \beta S'I' - dS', \frac{dE'}{dt} = \beta S'I' + \alpha E' - dE',$$
$$\frac{dI'}{dt} = \alpha E' - \gamma I' - dI', \frac{dR'}{dt} = \gamma I' - dR', \tag{9.8}$$

[See reviewer's comment (9)]

with the constant population constraint $S' + E' + I' + R' = N$ and $0 < \alpha \leq 1$. As the population is constant, we have

$$\frac{dS'}{dt} + \frac{dE'}{dt} + \frac{dI'}{dt} + \frac{dR'}{dt} = 0. \tag{9.9}$$

To solve this equation, we have to consider the equations in (9.8). We will then have

$$b = d. \tag{9.10}$$

We define the basic reproductive ratio

$$\Re_0 = \frac{\alpha\beta N}{(b+\alpha)(b+\gamma)}. \tag{9.11}$$

An epidemic occurs when $\Re_0 > 1$.

From (9.11), if the birth rate or the recovery rate of the infectious people increases, the reproductive ratio will decrease. Therefore the epidemic may not occur or occur in a slower rate. This means less people will be affected.

What is the difference between adding and without adding the birth rate and the death into the model?

We consider both cases at a certain time and the values of α, β, γ, N are the same. We can observe that the number of the exposed people, infectious people and the recovered people in the model with the birth rate and the death rate will be lower than that without the birth rate and the death rate. It is because some exposed people, infectious people and the recovered people died and so the number will be lower.

Besides, the rate of the epidemic will be slower than that without the birth rate and the death rate. From (9.1) and (9.8),

$$\frac{dE}{dt} + \frac{dI}{dt} > \frac{dE'}{dt} + \frac{dI'}{dt} \tag{9.12}$$

So the time for the epidemic in the case with the birth rate and the death rate will last longer. However, the number of susceptible, exposed, infectious, recovered after a long period are nearly the same in both cases.

We can calculate the values of the birth rate and the death rate by considering the number of births and the number of deaths in Hong Kong. In Hong Kong, the number of births and the number of deaths in 2009 is 82100 and 40200 respectively. For simply calculation, we assume the number of births is the same at every day, i.e. approximately 225 births in a day.

As we define the time interval Δt is one day and the value of N is a constant at every time t, we have $bN = 225$

$$b = \frac{225}{7 \times 10^6} \implies b \approx 3.21 \times 10^{-5}.$$

Now, we assume $b \neq d$, the value of N will not be a constant. In this case, we will also have $bN = 225$ and $dN = 110$, but the difference is that the values of b and d will vary with time t. The values of b and d will be inversely proportional with the number of people N.

At $t = 0, N = 7 \times 10^6$, we will have

$$b(0) \approx 3.21 \times 10^{-5} \text{ and } d(0) \approx 1.57 \times 10^{-5}.$$

If the values of b and d remain unchanged while the value of N increases, the number of N after a year will be more than expected. This will become an error in calculating the number of people in each group, so the results will be less accurate.

10. The SEIRS Model

After studying chapter 7, the SIRS model, and chapter 9, the SEIR model, we mix these two models and develop a new model, the SEIRS model. Here, we assume that the recovered people may lose their immunity and become susceptible again. We also assume the return of class R to class S occurs at a rate proportional to the population of recovered people and the rate of the exposed people become the infectious people is directly proportional to the number of exposed people. The model may be diagrammed as

$$S \xrightarrow{\beta SI} E \xrightarrow{\alpha E} I \xrightarrow{\gamma I} R \xrightarrow{\mu I} S,$$

and the corresponding coupled differential equations are

$$\frac{dS}{dt} = -\beta SI + \mu I, \frac{dE}{dt} = \beta SI - \alpha E, \frac{dI}{dt} = \alpha E - \gamma I, \frac{dR}{dt} = \gamma I - \mu I \qquad (10.1)$$

with the constant population constraint $S + I + R = N$ and $0 < \alpha \leq 1$.

[See reviewer's comment (10)]

We define the basic reproductive ratio

$$\Re_0 = \frac{\beta N}{\gamma}. \tag{10.2}$$

The epidemic occurs when

$$\frac{dE}{dt} + \frac{dI}{dt} > 0. \tag{10.3}$$

To solve this equation, we have to consider the equations in (10.1). We have

$$\frac{dE}{dt} + \frac{dI}{dt} = (\beta S - \gamma)I > 0. \tag{10.4}$$

so that an epidemic occurs if $\beta S - \gamma > 0$. After rearranging the term,

$$\frac{\beta S}{\gamma} > 1. \tag{10.5}$$

From (10.2), $\Re_0 = \beta N/\gamma$ and $S_0 \approx N$, so we can say an epidemic occurs when

$$\Re_0 > 1.$$

This result is the same as the SEIR model because the sum of the rate of change of the exposed group and infective group is the same. An epidemic will occur with the same condition as the SEIR model.

Let (S_*, E_*, I_*, R_*) be the fixed point of (10.1). The first equilibrium point is at the beginning, i.e. $(N-1, 0, 1, 0)$. We found that another equilibrium point when

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0. \tag{10.6}$$

From the equations in (6.1), we can deduce that this occurs when

$$\beta SI = \alpha E = \gamma I = \mu R. \tag{10.7}$$

By solving (6.6) and $S + E + I + R = N$, we can have

$$(S_*, E_*, I_*, R_*) = \left(\frac{\gamma}{\beta}, \frac{\alpha(N - \frac{\gamma}{\beta})}{\frac{1}{\alpha} + \frac{1}{\gamma} + \frac{1}{\mu}}, \frac{\gamma(N - \frac{\gamma}{\beta})}{\frac{1}{\alpha} + \frac{1}{\gamma} + \frac{1}{\mu}}, \frac{\mu(N - \frac{\gamma}{\beta})}{\frac{1}{\alpha} + \frac{1}{\gamma} + \frac{1}{\mu}} \right). \tag{10.8}$$

After simplifying, we have

$$(S_*, E_*, I_*, R_*) = \left(\frac{\gamma}{\beta}, \frac{\gamma\mu\left(N - \frac{\gamma}{\beta}\right)}{\alpha\gamma + \gamma\mu + \mu\alpha}, \frac{\mu\alpha\left(N - \frac{\gamma}{\beta}\right)}{\alpha\gamma + \gamma\mu + \mu\alpha}, \frac{\alpha\gamma\left(N - \frac{\gamma}{\beta}\right)}{\alpha\gamma + \gamma\mu + \mu\alpha} \right). \tag{10.9}$$

We can see that if $dsN < \frac{\gamma}{\beta}$, the number of people in class E, I and R will be negative and this will not be the real case in our life, so we say that this equilibrium point only exists when $dsN \geq \frac{\gamma}{\beta}$.

When $N < \frac{\gamma}{\beta}$, an epidemic will not occur and $(S_*, E_*, I_*, R_*) = (N, 0, 0, 0)$ when $t \to \infty$.

When $t \to \infty$., this equilibrium point (10.9) will also occur.

By comparing the point (6.7) with the point (10.9), the value of S is the same in both cases. It means the number in group S will be $\frac{\gamma}{\beta}$ after a long period of time for both SIRS model and SEIRS model.

Here, we substitute the values of $N, \beta, \alpha, \gamma, \mu$ to see the changes among class S, E, I and R with time and see if there are any difference with the SIRS model and the SEIR model. In this model, we also assume $N = 7000000$ and $\beta = 10^{-7}$.

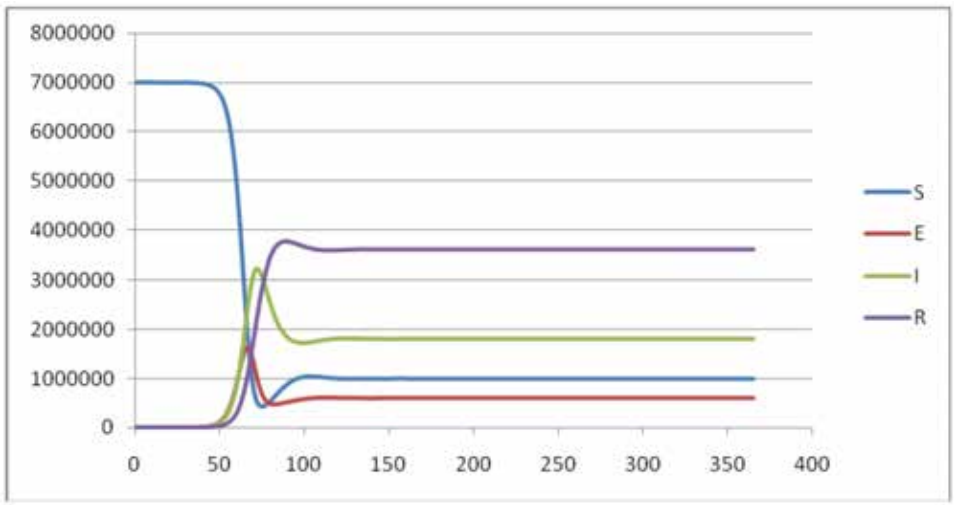


FIGURE 33. The number of people in class S, class E, class I and class R in time t with $\alpha = 0.3$, $\gamma = 0.1$ and $\mu = 0.05$.

We put the values of $N, \beta, \alpha, \gamma, \mu$ to the equilibrium point in (10.9), we have

$$(S_*, E_*, I_*, R_*) = (1000000, 600000, 1800000, 3600000).$$

It also shows the same result as Figure 33, the method of plotting graphs by using discrete data. We can see this is similar as the result in chapter 6.

From the Figure 33, we found that it reaches this equilibrium point after 4 months, i.e. 120 days. There are same number of perople from class S, E, I, R go to class E, I, R, S respectively in each period after it reaches the equilibrium point. So the number in each group will be unchanged and remain constant afterward.

11. Further study in The SEIR Model

After studying the models in the previous chapters, we still cannot find a suitable model which matches with the real situation in Hong Kong. The number of infectious is either too small or too large that means the epidemic either does not occur or occurs seriously that affect more than hundred thousand people. Here is an example of the real case of epidemic in Hong Kong:

Month	Apr 2009	May 2009	Jun 2009	Jul 2009	Aug 2009
Number of people affected	0	23	762	2887	8135
Month	Sep 2009	Oct 2009	Nov 2009	Dec 2009	Jan 2010
Number of people affected	16090	3780	901	1596	1092
Month	Feb 2010	Mar 2010	April 2010		
Number of people affected	469	573	148		

FIGURE 34. The number of people affected by *Swine Influenza* in each month

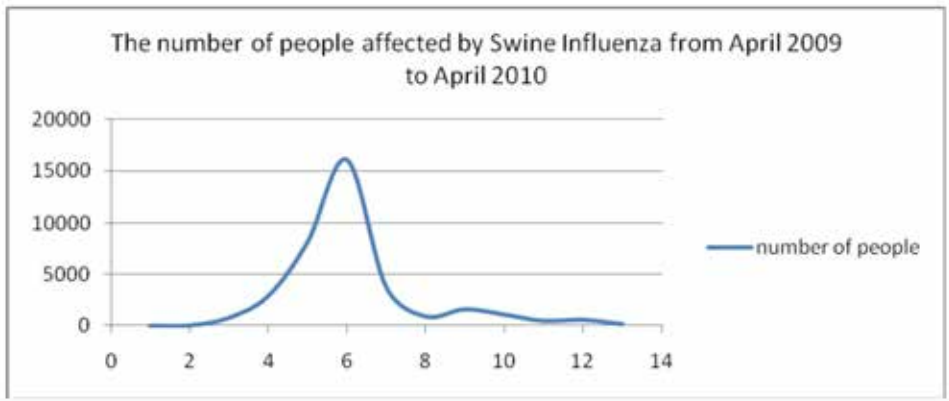


FIGURE 35. The number of people affected by *Swine Influenza* from April 2009 to April 2010

From Figure 34, 35, we can know that there are less than 40,000 people affected. This means that the above models in the previous chapter do not match with the real case.

Does it mean that the above theory is wrong? If not, what is the problem?

Actually, the above theory is correct with the above assumptions. The problem is that we assume the values of α, β, γ are constants at any time t, but the values of α, β, γ may vary in our daily life as there are many factors that will affect them.

Now, we want the recovery rate of infectious people to increase with time, so the number of infectious people will decrease and hence the epidemic will still occur with less people being affected. We assume $\gamma(t) = Ct^k$, where C is a constant. As we want the value of γ to increase with time but with a decreasing slope, we have

$$\gamma(t) > 0, \gamma'(t) > 0 \text{ and } \gamma''(t) < 0, \tag{11.1}$$

$$\gamma(t) = Ct^k > 0, \gamma'(t) = Ckt^{k-1} > 0 \text{ and } \gamma''(t) = Ck(k-1)t^{k-2} < 0. \tag{11.2}$$

After solving (11.2), we have

$$C > 0 \text{ and } 0 < k < 1. \tag{11.3}$$

The model may be diagrammed as

$$S \xrightarrow{\beta SI} E \xrightarrow{\alpha E} I \xrightarrow{\gamma I} R,$$

and the corresponding coupled differential equations are

$$\frac{dS}{dt} = -\beta SI, \frac{dE}{dt} = \beta SI - \alpha E, \frac{dI}{dt} = \alpha E - \gamma I, \frac{dR}{dt} = \gamma I, \tag{11.4}$$

with the constant population constraint $S + E + I + R = N$ and $0 < \leq 1$.

The epidemic occurs when

$$\frac{dE}{dt} + \frac{dI}{dt} > 0. \tag{11.5}$$

To solve this equation, we have to consider the equations in (11.4). We have

$$\frac{dE}{dt} + \frac{dI}{dt} = (\beta S - Ct^k)I > 0 \tag{11.6}$$

so that an epidemic occurs if $\beta S - Ct^k > 0$. After rearranging the term,

$$t < \left(\frac{\beta S}{C}\right)^{\frac{1}{k}}. \tag{11.7}$$

This shows the disease will not spread out after $t = \left(\frac{\beta S}{C}\right)^{\frac{1}{k}}$. To shorten the time of the disease spreading out, C and k should be larger. This means if there is any medicine which can control the disease as soon as possible, the recovery rate will increase and so the epidemic will no longer occur.

Now, we plot the graph by using the real data and numerical simulation of our model to compare if there is any difference between them. We use $\gamma(t) = Ct^{\frac{1}{2}}$ in the following examples as we find that when $k = 0.5$, the curve will be more similar to the real case. For further study, we can try different values of k to find a more accurate result.

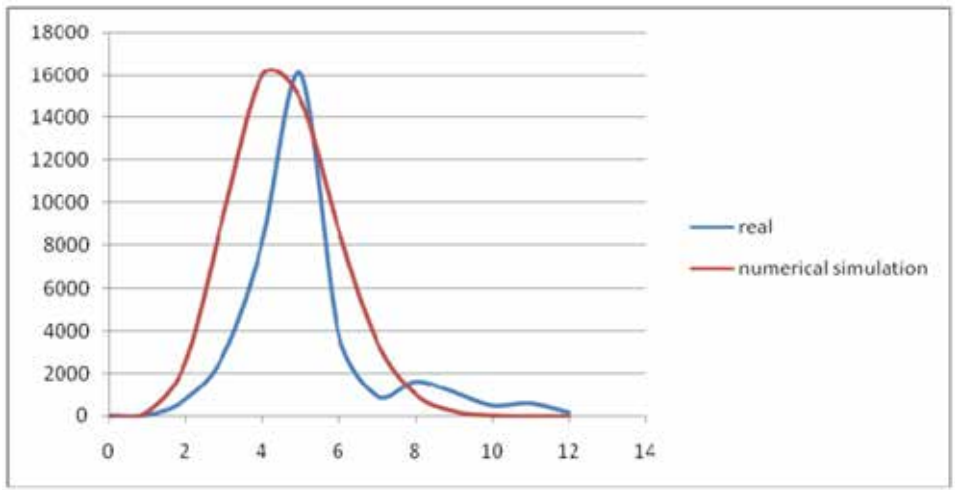


FIGURE 36. The number of people affected by *Swine Influenza* from April 2009 to April 2010 with $\beta = 10^{-7}$, $\alpha = 0.18$, $C = 0.064$ and $k = 0.5$ where $S(0) = 6999999$, $E(0) = 0$, $I(0) = 1$ and $R(0) = 0$.

From Figure 36, we can see that these two curves are similar. The curve by numerical simulation does not give too large or too small in the number of infectious people. The curves go to the peak value at a similar time and their peak values are nearly the same.

As the Centre for Health Protection only provides how many people got the disease in a month, we calculate how many people in each month to plot the curve. We denote $P(m)$ be the number of people who got *Swine Influenza* in the m^{th} month. In general,

$$P(m) = S(30m - 30) + E(30m - 30) - S(30m) - E(30m) \tag{11.8}$$

To derive this equation, we use the sum of the differences of the susceptible people and the exposed people between the beginning and the end of the month. For example, there are $S(0) + E(0) - S(30) - E(30)$ people got *Swine Influenza* in the first month. By putting $m = 1$ to $m = 12$, Figure 36 was plotted.

To estimate the values of β, α, C, we can use the time of the occurrence of the second case of infection, the total number of people who are infected in the first month and the time for an infectious person to recover. In the case of *Swine Influenza*, it takes about a week for an infectious person to recover. Also, the number of infected people in each month is known. However, we do not know the number of infected people on each day and so the time of the occurrence of the second case of infection has to be assumed. The government departments can have a set of more accurate data to adjust the values of β, α, C. Therefore, they can predict the number of people that will be affected and when the disease is the most serious. This helps the government take action for a new disease.

Now, we use the case of *Severe Acute Respiratory Syndrome (SARS)* to see if this model is also similar to the case of other epidemic.

Month	03 Feb	03 Mar	03 Apr	03 May	03 Jun	03 Jul
Number of people affected	0	610	979	150	16	0

FIGURE 37. The number of people affected by *SARS* in each month.

We plot the graph by using the above data and the numerical simulation to compare if there are any large differences between them and determine whether the model suits the real cases or not.

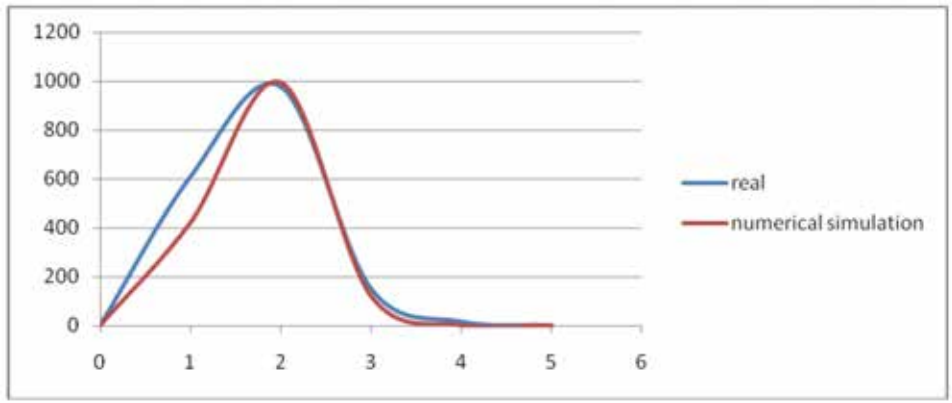


FIGURE 38. The number of people affected by *SARS* from February 2003 to July 2003 with $\beta = 10^{-7}, \alpha = 0.18, C = 0.064$ and $k = 0.5$ where $S(0) = 6999999, E(0) = 0, I(0) = 1$ and $R(0) = 0$.

From Figure 38, we can see that these two curves are also similar. Their peak values are similar and these curves go to the peak value at a similar time. From Figure 36 and 38, these graphs show this model will give similar result to the real case.

Now, we want to have some changes on $\gamma(t)$ to see if there is any difference. We use the case of *Swine Influenza* again. We assume $\gamma(t) = C \ln t$, where C is a positive constant as the value of γ increases with time with the decreasing slope.

The epidemic occurs when

$$\frac{dE}{dt} + \frac{dI}{dt} > 0. \tag{11.9}$$

To solve this equation, we have to consider the equations in (11.4). We have

$$\frac{dE}{dt} + \frac{dI}{dt} = (\beta S - C \ln t)I > 0 \tag{11.10}$$

so that an epidemic occurs if $\beta S - C \ln t > 0$. After rearranging the term,

$$t < e^{\frac{\beta S}{C}}. \tag{11.11}$$

This shows the disease will not spread out after $t = e^{\frac{\beta S}{C}}$.

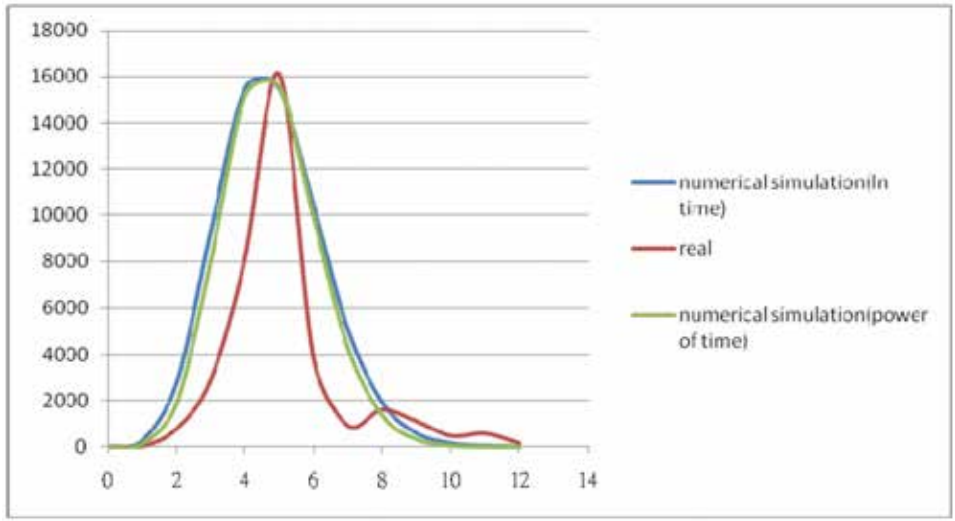


FIGURE 39. The number of people affected by *Swine Influenza* from April 2009 to April 2010 with $\beta = 10^{-7}, \alpha = 0.18, C = 0.064$ and $k = 0.5$ for the numerical simulation $(\gamma(t) = Ct^k); \beta = 10^{-7}, \alpha = 0.465$ and $C = 0.146$ for the method $(\gamma(t) = C \ln t)$ where $S(0) = 6999999, E(0) = 0, I(0) = 1$ and $R(0) = 0$.

Form Figure 39, we can see the curves plotted by two methods are similar to each other. These two methods can give similar result to the real case. However, the method by using the power of time will be better as there is one more constant k that will affect the result and it will be easier to control the values of β, α, C. We think the first method, assuming $\gamma(t) = Ct^k$, will be more applicable.

12. Summary

- Give an abstract, introduction of why we are studying this topic.
- Have some background information about the models. Explain briefly the modeling steps that lead to these models.
- Create the simplest model - SI model, with only two basic components. Simply describe how models work.
- Having the SIS SIR models as the beginning, introducing more concepts of how we develop the models.
- Developing SIVD SISR models by splitting or rearranging the existing components.
- Having the SIRS TSIR SEIR SEIRS models to show different results under different conditions. More and more conditions are added in order to be close to reality. Adding different conditions will change the result sharply.

- **A further study in the SEIR model** is made with comparison with real data of Swine Influenza from April 2009 to April 2010 in Hong Kong, resulting in a surprising result. Find out that time is an important factor in the model.
- Calculations abridged in earlier chapters are shown in Appendices.

13. Conclusion

First, we create the simplest model, the SI model. Then, by adding different new groups, we are able to make other different and more complicated models. After a series of investigation and research on these models, we are able to create a model which gives a similar result as the real data.

Actually, in this project, we are trying to create a model which is applicable in real life situation, aiming at predicting how the germs are spread. We try to investigate the spreading of a disease by mathematical modeling. First, we find out the factors affecting the spread of diseases and possible ways to prevent epidemics. Moreover, data are put into the model to simulate the situation in Hong Kong. We are able to find out the rate of being infected and recovery, and so as the number of people to be infected.

After making a number of models, and modifying them, we are able to create a model which fits the case in Hong Kong in 2009 (*Swine Influenza*). We believe that more work can be done on these topics, aiming at the same goal, creating a model in other situations which helps predict how germs are spread, so as to prevent them. We hope that this project can help to achieve this ultimate goal.

14. Appendices

1. Steps for solving equation (2.1)

$$\begin{aligned}
\frac{dI}{dt} &= \beta SI \\
\frac{dI}{dt} &= \beta(N-I)I \\
\frac{dI}{(N-I)I} &= \beta dt \\
\int \left(\frac{1}{N-I} + \frac{1}{I}\right) dI &= \int \beta dt \\
\ln\left|\frac{I}{N-I}\right| &= N\beta t + C_1 \\
\frac{I}{N-I} &= C_2 e^{N\beta t} \\
I &= \frac{NC_2}{C_2 + e^{-N\beta t}}
\end{aligned}$$

Put $I = 1, t = 0$, we have $C_2 = \dfrac{1}{N-1}$. Therefore,

$$I = \frac{N}{1 + (N-1)e^{-N\beta t}} \text{ and } S = \frac{N(N-1)e^{-N\beta t}}{1 + (N-1)e^{-N\beta t}}.$$

2. Steps for solving equation (3.1)

$$\begin{aligned} \frac{dI}{dt} &= \beta SI - \gamma I \qquad (3.1)\\ \frac{dI}{dt} &= \beta(N-I)I - \gamma I \\ \frac{dI}{dt} + (\gamma - \beta N)I &= -\beta I^2 \end{aligned}$$

(i) For $\Re_0 = \dfrac{\beta N}{\gamma} \neq 1$, this is in fact a Bernoulli differential equation. The general solution is

$$I = \left[\frac{(1-2)\int e^{(1-2)\int(\gamma-\beta N)dt}(-\beta)dt + C}{e^{(1-2)\int(\gamma-\beta N)dt}}\right]^{\frac{1}{1-2}}$$

$$I = \left[\frac{\beta}{\beta N - \gamma} + Ce^{(\gamma-\beta N)t}\right]^{-1}$$

Since $I = 1$ when $t = 0$ and $\Re_0 = \dfrac{\beta N}{\gamma}$, we have

$$I = \left\{\frac{1}{N\left(1 - \frac{1}{\Re_0}\right)} + \left[\frac{1}{N\left(1 - \frac{1}{\Re_0}\right)}\right] e^{-\beta N\left(1 - \frac{1}{\Re_0}\right)t}\right\}^{-1} \qquad (3.2)$$

(ii) For $\Re_0 = \dfrac{\beta N}{\gamma} = 1$, $\dfrac{1}{I^2}dI = -\beta dt$,

$$\begin{aligned} \frac{1}{I^2}dI &= -\beta dt \\ \int \frac{-1}{I^2}dI &= \int \beta dt \\ \frac{1}{I} &= \beta t + C \\ I &= \frac{1}{\beta t + C} \end{aligned}$$

Since $I = 1$ when $t = 0$, $C = 1$. Hence

$$I = \frac{1}{\beta t + 1} \qquad (3.3)$$

3. Steps for solving equations (4.2)

$$\begin{aligned}
&\frac{d\hat{S}}{d\hat{t}} && \frac{d\hat{I}}{d\hat{t}} && \frac{d\hat{R}}{d\hat{t}} \\
&= \frac{d\hat{S}}{dS} \times \frac{dS}{dt} \times \frac{dt}{d\hat{t}} && = \frac{d\hat{I}}{dI} \times \frac{dI}{dt} \times \frac{dt}{d\hat{t}} && = \frac{d\hat{R}}{dR} \times \frac{dR}{dt} \times \frac{dt}{d\hat{t}} \\
&= \frac{1}{N} \times (\beta SI) \times \frac{1}{\gamma} && = \frac{1}{N} \times (\beta SI - \gamma I) \times \frac{1}{\gamma} && = \frac{1}{N} \times (\gamma I) \times \frac{1}{\gamma} \\
&= \frac{-\beta N}{\gamma} \times \frac{S}{N} \times \frac{I}{N} && = \frac{\beta N}{\gamma} \times \frac{1}{N^2} \times SI - \frac{I}{N} && = \hat{I} \\
&= -\Re_0 \hat{S}\hat{I} && = \Re_0 \hat{S}\hat{I} - \hat{I}
\end{aligned}$$

4. Steps for solving inequality (4.3)

$$\begin{aligned}
\Re_0 \hat{S}_0 - 1 &> 0 \\
\Re_0 \hat{S}_0 &> 1 \\
\frac{\beta N S_0}{\gamma N} &> 1 \\
\frac{\beta S_0}{\gamma} &> 1.
\end{aligned}$$

5. Steps for getting (4.5)

$$\begin{aligned}
\Re_0(1-p) &> 1 \\
\Re_0 - p\Re_0 &> 1 \\
p\Re_0 &< \Re_0 - 1 \\
p &< 1 - \frac{1}{\Re_0}.
\end{aligned}$$

6. In chapter 11, the first case we used, i.e. assume $\gamma(t) = Ct^k$, we have

$$\begin{aligned}
S(t+\Delta t) &= S(t) - \beta S(t) I(t) \\
E(t+\Delta t) &= E(t) + \beta S(t) I(t) - \alpha E(t) \\
I(t+\Delta t) &= I(t) + \alpha E(t) - \gamma(t) I(t) \\
R(t+\Delta t) &= R(t) + \gamma(t) I(t)
\end{aligned} \tag{13.1}$$

The second method we used, i.e. assume $\gamma(t) = C \ln t$, we have

$$\begin{aligned}
S(t+\Delta t) &= S(t) - \beta S(t) I(t) \\
E(t+\Delta t) &= E(t) + \beta S(t) I(t) - \alpha E(t) \\
I(t+\Delta t) &= I(t) + \alpha E(t) - \gamma(t+\Delta t) I(t) \\
R(t+\Delta t) &= R(t) + \gamma(t+\Delta t) I(t)
\end{aligned} \tag{13.2}$$

There is a little bit difference between (13.1) and (13.2). It is because if we consider $\gamma(0)$ for $\gamma(t) = C \ln t$, the value of γ is undefined. So we will use $\gamma(t + \Delta t)$ instead of $\gamma(t)$.

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Reviewer's Comments

The reviewer has some comments about the presentation of this paper and the typos.

1. The reviewer has comments on the wordings, which have been amended in this paper.
2. The reviewer suggests merging Chapter 1 into Chapter 2, because there is no additional background introduction except the formula for SI model and there is even no definition for each variable.
3. A reference for this project may be needed.
4. How about rewriting the solution of SI model as follows:
   firstly, adding the two equations implies that $S + I$ must be a constant N, which is defined to be the population;
   secondly, with initial condition $I(0) = 1$ we can solve I and S using the method for separable ODEs.
5. Does "we take γ^{-1} as time" mean "we rescale time by γ^{-1}"?
6. It may be more mathematically strict to replace "reach" with "almost reach" or "is sufficient close to", since the solution can only approach but never touch the equilibrium due to uniqueness for ODE.
7. It is difficult to understand the TSIR model (8.1), especially the definition of tourist T. There always exist self-contradictions for the following possible cases:
   1) T means the total number of tourists inside Hong Kong at time t, but this obviously contradicts with the first equation in (8.1): $\dfrac{dT}{dt} = \varepsilon S + \varepsilon R - 2\iota T$;
   2) T means the total number of people who are outside Hong Kong at time t and may enter Hong Kong as tourists at an immigration rate ι, then T should be a very large number, contradicting with Figure **??**-**??**; and also it makes no sense to require $S + I + R$ and T to be constant from the ODE system.

   The reviewer suggests that:
   for the above case 2), set up the correct autonomous ODE system for T, S, I, R, which automatically implies $T + S + I + R =$ constant; or else,
   only consider S, I, R restricted in Hong Kong but affected by the incoming tourists (at a rate 94000 per day) and outgoing ones (proportional to $S+R$ as in the paper). This seems to better match the author's motivation for TSIR model.
8. The ODE for E should be
$$\frac{dE}{dt} = \beta SI - \alpha E,$$
   and the following population constraint should be $S + E + I + R = N$.
9. The ODE for E' should be
$$\frac{dE'}{dt} = \beta S'I' - \alpha E' - dE',$$
   and in the previous diagram, dS, dE, dI, dR should be dS', dE', dI', dR'.

10. The ODE for S, R should be

$$\frac{dS}{dt} = -\beta SI + \mu R, \frac{dR}{dt} = \gamma I - \mu R;$$

and in the previous diagram, μI from R to S should be μR;
and the following population constraint should be $S + E + I + R = N$.