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Modeling COVID-19 Transmission

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Abstract

In the search of better mathematical methods to predict the development of the current epidemic, we established a new model called SEQIR to simulate the transmission of COVID-19 based on the properties of COVID-19, after conducting a comparative study of several common models of epidemic transmission. Furthermore, associating with the real time data in Worcester, we quantified the effects of different control policies on prevention of epidemic spread.

In order to improve the simulation and prediction effect of the new model, we then explored the SEQIR model with time delay and latent period, and evaluated two steady states, which are disease-free equilibrium and endemic equilibrium. By using Routh–Hurwitz stability criterion and analyzing the dynamic stability of the equilibrium points, we indicated changing the value of time delay in the system does not have influence on disease spread. Under the endemic equilibrium condition, when R_0 is larger than 1, the system is locally stable for any value τ of time delay.

Executive Summary

In early December 2019, the first case of coronavirus disease was found in Wuhan City, Hubei Province in China. Its pathogen was named severe acute respiratory syndrome coronavirus 2SARS-CoV-2 by the International Committee on Taxonomy of Viruses. Coronavirus poses a continuous threat to human health with its high contagious efficiency, serious infection consequences and elusive epidemic time [23]. With the outbreak of the coronavirus in 2020, the number of confirmed diagnoses exceeds 140 million and more than 3 million deaths across more than 200 countries [14]. Due to the critical epidemic factors, numbers of countries have published a series of policies to lockdown the border. The United States even declared "state of emergency". People are paying more and more attention to models that predict the number of infections and deaths. In this research, mathematical models are built to simulate the transmission of the trend of COVID-19 in Worcester. The general mathematical model of epidemic disease is studied. However, the coronavirus is more contagious than the common disease, and the infections have a latent period, which is hard to distinguish between incubation and healthy people. Therefore, a new dynamical system is built based on the SEIR model, to visualize and pursue the influence of control policy on the epidemic. First, the real time data from The New York Times is fitted with the model to calculate the parameter of the model. The epidemic situation is analyzed through forward Euler's method, the impact of different prevention and control policies such as face mask and self-isolation is indicated by models. To understand the spread and envision the peak of the virus, we must be able to predict the steady states of the equations. Thus, we use linearization to format the systems of equations. Linearization can present the curve of the movement of the virus. Since the time delay system is nonlinear, we need to interpret the equations by making it to be linear. Firstly, we depict the process of linearization by the SEIQR model. Then, we transform the equation into the matrix form. We explore the differential equations with three dimensional cases, and finally find the estimated stable points. This study utilizes given data of the virus that occurs and mathematical models to investigate the behavior of it. Through our research, we found that the quarantine policy can effectively suppress the spread of the disease. We cannot avoid necessary daily contact, which would cause the virus spreading over the community. The significance of the model is to predict the number of infected people, and to explore the outbreak period.

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Contents

List of Figures

List of Tables

Chapter 1

Introduction

1.1 What is COVID-19?

COVID-19 (Corona-virus disease 2019) is a contagious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The first recognized case was confirmed in Wuhan, China, in December 2019 [18]. The rapid spread of the disease causes a worldwide pandemic.

Transmission of COVID-19 occurs mainly when infected people are in close contact with another person. Small droplets containing the virus can spread from infected people's nose and mouth as they breathe, cough, sneeze, sing, or speak. Other people will be infected if the virus gets into their mouth, nose, or eyes. The transmission through aerosols which is particulate matter suspended in the air is also possible [6]. COVID-19 will not only spread through infected people with identical symptoms, but some asymptotic infected people can also transmit the virus. Start with the day exposure to the virus to fourteen days after, symptoms of COVID-19 may be identical to the infected people [4]. People remain infectious for up to ten days after the onset of symptoms in moderate cases and up to twenty days in severe cases [7].

As the most serious global pandemic in recent decades, this Coronavirus appeared and spread suddenly without any warning. After it officially named as COVID-19 by WHO on February 12, 2020, many scientists all over the world began to focus on the studying this Coronavirus. They found that the Coronavirus disease is an infectious disease caused by Severe acute respiratory syndrome Coronavirus 2 [30]. The gene sequence of this kind of virus belongs to the same lineage as SARS and MERS viruses. It can enter the human body through the respiratory tract, causing the pulmonary infection, cardiac infection, and infecting other major organs [4]. This virus is transmitted through oral and nasal secretions, or through aerosols, particulate matter suspended in the air. Coronavirus will not only spread through overtly infected people, but some infected people will also be asymptomatic, which makes the diagnosis more difficult [9]. A certain level of mortality among the patients confirmed was ineluctable.

As the research continues, the spread of the virus led to an outbreak of epidemic all over the world. Except for China, which quickly contained the spread of the disease within two months with its most decisive method and restart its civil economic activity in October, 2020, most country in this world are still struggling in the midst of the epidemic, some were suffering from their political game, some were wavering under their own practical economic pressure. Up to now, the epidemic of COVID-19 seem still getting worse in this planet day by day, with more than 165 million confirmed infected person and more than 3 million loss of human life, developed into the first truly global epidemic [28].

1.2 Influence of COVID-19

As a massive and rapidly spreading epidemic, COVID-19 changed the world a lot, and its follow-up effects keep on going with the slow-rolling crisis. They had not only changed our daily life macroscopically and personally but also will create great uncertainty about our future.

The first negative and the most obvious influence the COVID-19 imposed on this world is the economic decline. Many countries issued travel bans and lockdown orders to prevent the rapid spread of the virus. The European Council announced that to curb the spread of COVID-19 on March 17, EU members agreed to impose a 30-day restriction on "non-essential travel" to Europe, the US government issued a 60 days temporary restriction on travel at US land border ports on April 23 [10]. Unfortunately, all these lockdown measures and restrictions on travel or social contacts, will slow global economic growth by reducing market volume at a civil level and international trade aspects,which was already very well represented in the past year of 2020.

As the panic spreads across countries, stricter quarantine measures are implemented worldwide, which not only has seriously affected the market supply of various products and services, but also limited the normal growth of demand in various markets.

According to statistics from the World Bank, the global economy shrank by 4.3% last year, the minimum growth rate since the financial crisis, which is 2.5 times more than the financial crisis ten years ago [15]. The violent fluctuations in the consumer market during the epidemic will not be dominated even by the government or large financial groups. To control the spread of the epidemic, population mobility must be minimized. Locking down cities and countries and closing borders are decisive measures. Large-scale personnel gathering will also be restricted which will affect the service industry, modern industrial production, transportation inevitably, because of our daily consumption decline. Countries with an incomplete industrial chain, or overly relied on partial industries, especially service industries, find it very difficult to maintain normal economic activities. The GDP of many countries expected to shrink by more than 5%, and the fiscal deficit as a percentage of GDP continues to increase [12].

Due to the locking down measure, travel ban, and infected population, the world labor force was shrunken first time from 1990, which further crippling the world economy. Since the organ damage among those severe symptoms infected person of COVID-19 are permanent and irreversible, so the lost of labor force will be a long term problem (until the younger generation join the labor market), which can reduce abnormally as its growing pattern for decades [11]. Furthermore, the continued medical care for infected persons will be a heavy burden for their family and society as well.

Compare to these economic loss, the spiritual damage caused by the epidemic will be a much more complicated problem, which can haunt many families and our society for a long time. Of course the severe infected people and their families will bear the torture from the side effects of disease and higher economic pressure [16]. While many people, enterprises and organizations who suffered heavy losses during the epidemic will blame it all on the incompetence of their governments, the pressure could inspire a corresponding hatred and distrust of authority, increase the difficulty of governance in various countries and regions, and force some poor governance ability politicians to pass the buck for their inadequate response to the epidemic to specific countries or communities. These irresponsible political manipulation will definitely disrupt normal international trade and international cooperation, significantly delayed the full recovery of the global economy after the epidemic, but also inspire some prejudice against some countries or some communities, just like what is going on in many states of US, including New York, California, and Texas, more than 50% people haves anti-China sentiment, and many East Asians have suffered physical and verbal abuse, including a stabbing case against Asian people [8]. Many Asians who have been violated are actually authentic American citizens who have

citizenship from birth, but they cannot be treated fairly because of their race.

Serious reflections after the epidemic are necessary and some changes have already started silently. Behind the bankruptcy of many physical chain like Art Van Furniture, which has 194 stores in 9 states across US, with annual sales of more than $1 billion, dozens of U.S. retailers filed for bankruptcy, resulting in the closure of more than 47,000 chain stores across the United States, while many E-commerce Companies maintained a steady development, benefited from the rigid demand during the quarantine period and their safer way of trade [21]. This change will be possible to cause the industrial restructuring after the epidemic, which should arouse the timely attention of relevant enterprises and economic -financial administrative authorities [21]. Further more we need face the reality that our global industrial chain is a delicate but fragile system as the global health care and epidemic prevention and control mechanism, the failure of an enterprise can trigger numerous chain reactions and may cause the paralysis of the entire industrial chain. The globalized industrial division of labor has improved production efficiency, but at the same time it has made the system more fragile and magnified when it encounters the impact of emergencies like COVID-19.

Those countries can instantaneously adapt to the change, and take effective measures to bring the epidemic under control rapidly, will get more opportunity to promote their economies during this process, while the countries have more complete industry chains will have the advantage of further rapid development. On the contrary, those countries cannot respond to the changes brought about by the epidemic timely in the right way, their economic development will stagnate or even decline. So we can foresee a major reshuffle of national power generated by the epidemic of COVID-19, leading to changes in the international economic pattern, even geopolitical layout.

1.3 The academic assignments of this study

By May 2, 2021, a total of 90,686 cases of Coronavirus in China have been diagnosed, and a total of 165 million cases have been diagnosed globally [28]. With the broadcast of the Coronavirus, the research has also expanded from the origin of the virus, the natural host, the intermediate host, and the patient zero to the study of the virus's transmission, mode of transmission, gene mutation, anti-epidemic prevention system design, and so on. Above all, it is particularly important to evaluate the effects of various epidemic prevention measures on disease control. Admittedly, the impact of the vaccine on the epidemic is crucial, but the development process of vaccines is also challenging. On July 20, 2020, the world's top medical journal "The Lancet" published online the Phase II randomized controlled trial results of the Corona-virus vaccine. As the world's first official release of Phase II clinical trial data of the new Corona-virus vaccine, it fully demonstrates the positive role of vaccination in controlling the epidemic [31]. Under the pressure of inadequate global vaccine production and supply capacity, the anti-epidemic effect of the vaccine will be much less effective while the emergence of newly mutated viruses. Conversely, the serious lockdown to cut off the source of infection and the transmission of the virus just like China seems much more effective. Therefore, it is especially important to find out the dynamic law of the development and evolution of COVID-19 under the conditions of various factors intervention.

In the early stage of the epidemic, epidemiologists and statisticians established a transmission dynamics model. Based on the data at the beginning of the outbreak, they predicted the peak of the spread of the epidemic, which provided important information for the government's decision on epidemic prevention. In the evaluation of prevention and control, the effective reproduction number is often used as the basis for evaluation. In classic epidemiological models, the R_0 value is often used to describe the transmission rate of an epidemic, which can reflect the potential and severity of an

infectious disease outbreak.

Among the common epidemic models such as SI, SIS, SIR, SIRS and SEIR models, SEIR model with the combination of these groups of people based on different infectious diseases can produce different models, which seems can simulate it better of the actual situation of the development and change of COVID-19 along the time axis. Since the COVID-19 has a longer time of incubation period (an average of 14 days, many cases more than 24 days were found all over the world), the process of infected statistics may be omitted and delayed due to the different management levels in different countries and regions. Researchers have had to design many more complex simulating equations to modify linear models based on statistical data to make it more closer to the reality of the development and spread of COVID-19 [17]. This simulation modification not only greatly increases the workload and difficulty of changing in dynamic simulation, but often encounters a serious challenge, which is that simulating equations are insoluble in many cases.

So we need to find a more convenient and efficient way to simulate the trend of COVID-19 transmission, and avoid massive computation processes.

After contrastive study on the mathematical simulation models of many epidemic diseases and combination with the transmission characteristics of COVID-19, we developed a new model called SEQIR for the data fitting of COVID-19. The goal is trying to reveal the objective law of the spread of the epidemic and find out which kind of epidemic prevention methods would be more effective. According to the study in Chapter 3, we continuously explore the SEQIR model and subjoin time delay into the system. When we try to modify the solution of system at steady-state, we do not need to solve all equations of SEQIR model, while some equations are unsolvable. The study will start from analysis of this new model and find out whether or not time delay (incubation period) will affect the stability of solutions, get the steady-states and characteristic equations, developing a reliable, efficient epidemic transmission evolution model.

Chapter 2

Mathematical Modeling about COVID-19

2.1 Introduction to epidemic model

Infectious disease dynamics is a crucial method for theoretical and quantitative research on infectious diseases. The mathematical model reflecting on dynamic characteristics of infectious diseases is established based on the spread and evolution of the disease within a community and other social factors related to it. Through the variable and quantitative analysis and numerical simulation of the model, it indicates the development process of the disease, predicts the development and change, and analyzes the key factors of the disease epidemic, so as to seek the best strategy for its prevention and control. Compared with the traditional statistical method, the dynamic method can better reflect the epidemic law from the aspect of disease transmission.

The earliest research on infectious disease models began in the 20th century. In 1906, W.H. Hamer constructed a discrete event model to understand the repeated epidemics of measles [3]. In 1911, Sir Ronald Ross used a differential equation model to study the spread of malaria in mosquitoes and among people. The results showed that if the number of mosquitoes is reduced below a critical value, the malaria epidemic will be controlled. In 1926, Kermack and McKendrick studied the Black Death and the Bombay plague and constructed the SIR compartment model. In 1932, the SIS compartment model was proposed. Modeling and research on the dynamics of infectious diseases began to flourish in the middle of the 20th century [3].

2.2 Mathematical Epidemic Model

There are many types of mathematical models of infectious diseases according to various factors such as the different speeds of infectious diseases, different spatial scopes, diverse transmission routes, and dynamic mechanisms. If the model is divided according to continuous time, it can be divided into ordinary differential equations, partial differential equations, and other equation models. If it is divided based on discrete time, then it is the difference equation.

The common epidemic models can be divided into $SI, SIS, SIR, SIRS$ and $SEIR$ models according to the specific characteristics of epidemic diseases.The S, E, I, R represent different categories of people:

- S (Susceptible) refers to the group of healthy people lacking immune capacity, who are easy to be infected after contact with infected people.
- E (Exposed) refers to a person who has encountered an infected person but is not yet contagious, may be used for an infectious disease with an incubation period.

- I (Infected) refers to people who have disease that can be transmitted to S, turning them into E or I.

- R (Recovered) refers to a person who is immune after recovery from a disease. A life-long immune infectious disease cannot be re-converted to S, E, or I. If the immune period is limited, it can be re-converted to S, and thus infected.

The combination of these four groups of people based on different infectious diseases can produce different models. In addition, models that will be discussed in this chapter is based on these basic assumptions:

1. The total population of the region is $N(t)$. Neglect all population dynamics such as birth, death, migration, etc. Assume that all the population is in a closed environment and the population changes over time based on disease is more significantly than the population changes over time based on nature birth and death rate. Therefore, the total population always remains a constant, such that:
$$N(t) \equiv K$$

2. Once a patient comes into contact with a susceptible person, there will be a chance of infection. Assume at time t, the number of infectious people $I(t)$ will contact with r susceptible people on average per person in the unit time, and there is β probability in percentage to contagious health people. The proportion of healthy people is $\frac{S}{N}$.

3. Assume at time t, the percentage of infected people among total population N is $\frac{I(t)}{N}$. The number of susceptible people $I(t)$ will contact r people on average in the unit time. $\frac{rI(t)}{N}$ is the infected people that each susceptible person will contact per day, and there is β probability per infected person will transmit the virus to health people. Therefore, the number of susceptible people transmit to infected people is:
$$\frac{r\beta S(t)I(t)}{N}$$

4. Assume at time t, the number of people recovered from the infected per unit time is proportional to the number of patients, and the proportional coefficient, γ, is the probability of people will recover per unit time.

5. Assume at time t, some of exposed people would be recovered due to self-immunity, which means not everyone in exposed group would develop the disease after incubation period. Therefore, α represents the incidence rate.

The table of parameters for standard epidemic models are shown below:

Table 2.1: Standard Parameters for Epidemic Models.

Notation	Unit	Parameter Name
β	% $people^{-1}$	Probability per infected person will transmit the virus
r	$people\ day^{-1}$	Average numbers of people contact with infected people per day
γ	% day^{-1}	Probability of people will recover per day
α	% day^{-1}	Incidence rate of exposed people per day

2.3 SI Model

This model only involves two groups of people, S and I. The susceptible person who contacts the infected person becomes the infected person. There is no incubation period, cured condition, and immunity. Take a day as the smallest unit of time in the model. The total number of people is N, regardless of the birth and death of the population, immigration, and emigration, so the total number remains the same. At time t, the number of two groups of people is $S(t)$, $I(t)$. When the initial time is $t = 0$, the initial number of people is S_0. Once an individual in the SI model is infected, he or she is permanently infected. At a given time t, the number of individuals whose $S(t)$ and $I(t)$ represent the state of susceptibility and infection at that time, respectively, obviously has:

$$S(t) + I(t) = N$$

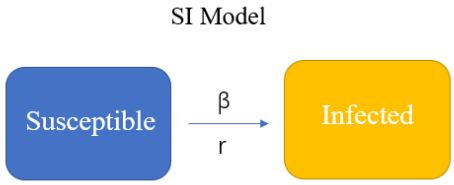


Figure 2.1: The schematic diagram of SI Model.

When susceptible people become infected, the number of susceptible will be decreased, $\frac{-r\beta S(t)I(t)}{N}$, on the other hand, the infected people will be increased, $\frac{r\beta S(t)I(t)}{N}$.

$$\begin{cases} \dfrac{dS}{dt} = -\dfrac{r\beta S(t)I(t)}{N} \\ \dfrac{dI}{dt} = \dfrac{r\beta S(t)I(t)}{N} \end{cases} \tag{2.1}$$

With MatLab, we can solve this system of equations:

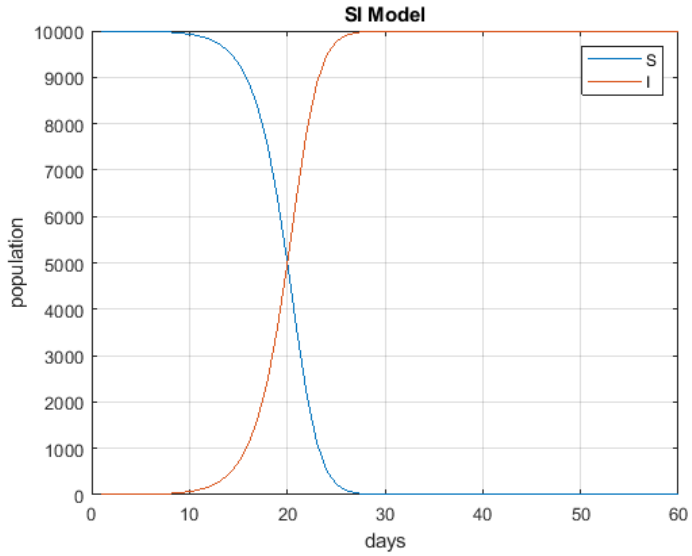


Figure 2.2: Simulate SI Model with parameters: N=10000, $\beta = 0.03, r = 20$.

From Fig. 2.2, it is clear that the total population will be constant which corresponds in the previous assumption. Between day 10 to day 25, and the infection rate keeps growing rapidly. In unit time t, the number of infections per infected person is proportional to the number of susceptible people. Without recovery rate, people will be infected eventually.

2.4 SIR Model

The Kermack-McKendrick SIR compartment model divides the regional population into the following three categories: susceptible, infected, recovered. The process of susceptible persons from illness to removal can be described by the following block diagram.

In this model, there are three groups of people, S (Susceptible), I (Infected), and R (recovered). Recovered people will have immunity and once cured will not be infected again, which means the cured people are not involved in this model anymore. The total population N is constant, and at time t, the susceptible, infected, and recovered are $S(t)$, $I(t)$ and $R(t)$. It follows that:

$$S(t) + I(t) + R(t) = N$$

Set the infectious rate as β and recovery rate as γ, which is the ratio of infected people becoming recovered people at time t.

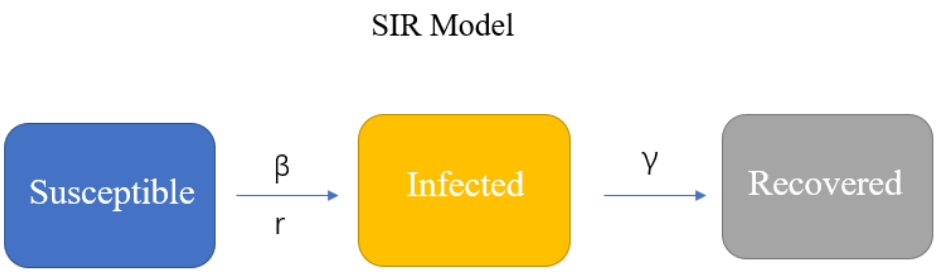


Figure 2.3: The schematic diagram of SIR Model.

From the model, the equation can be written as:

$$\begin{cases} \dfrac{dS}{dt} = -\dfrac{r\beta S(t)I(t)}{N} \\ \dfrac{dI}{dt} = \dfrac{r\beta S(t)I(t)}{N} - \gamma I(t) \\ \dfrac{dR}{dt} = \gamma I(t) \end{cases} \tag{2.2}$$

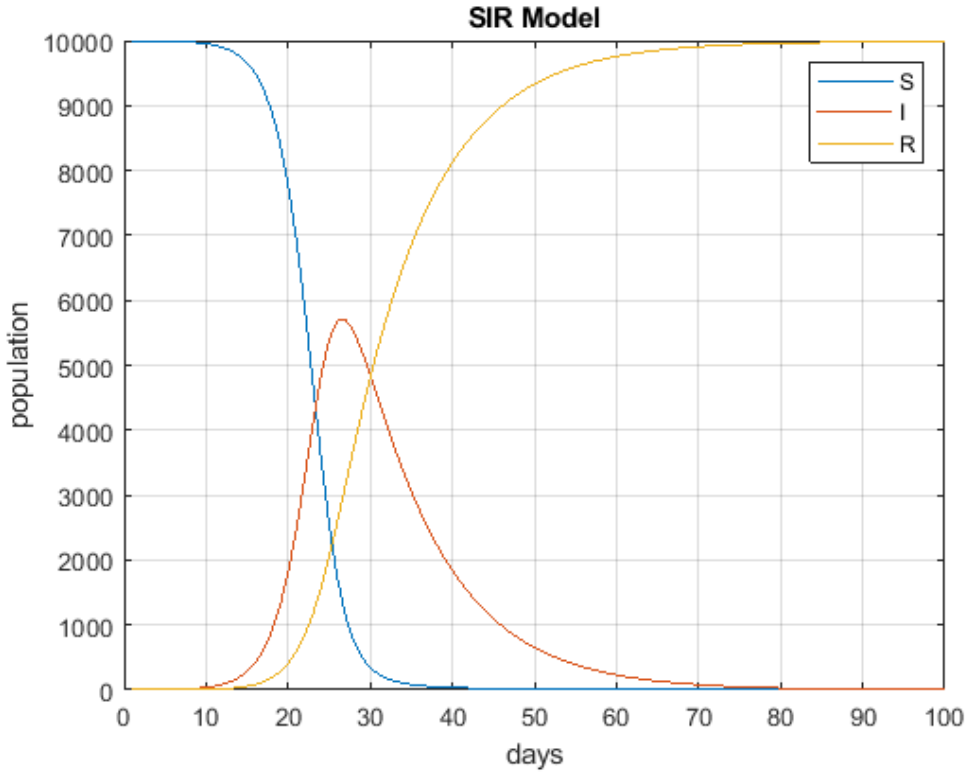


Figure 2.4: Simulate SIR Model with N=10000, $\beta = 0.03, \gamma = 0.1, r = 20$.

From Fig 2.4, the maximum of infection is at 27 days after epidemic started to spread. First, we can see that the stability point of the system is $I = 0$, $S = 0$, which means the virus will eventually pass. Then, we can predict an exponential upward and then a downward trend. The faster it spreads, the faster it ends. The entire population is filtered by the virus and goes through the process from S to I to R.

2.5 SEIR Model

The model demonstrates that COVID-19 will pass, and if the infection rate of the virus is high, it will pass quickly. The core parameter is the transmission speed, which determines how long the entire population contracted the disease, then the entire population will be filtered, and the epidemic will be over. It depends on the susceptibility of the virus and the contact density of the population. It is also the most primitive basis for us to reduce the contact density to control transmission. However, many core elements are obviously neglected, like whether the recovering person after the first infection will be infected again, and whether when the contact occurs, at the same time susceptible becomes infectious, which means that the contact and the onset of the infection are almost simultaneous. In the models above, the most important parameter, incubation period, for COVID-19 is neglected. Therefore, the model can be modified as:

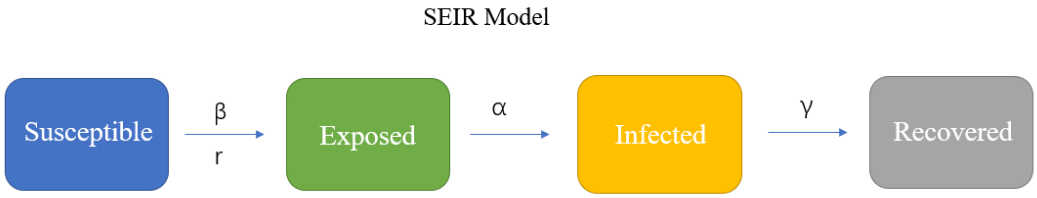


Figure 2.5: The schematic diagram of SEIR Model.

Set the infected rate as β, recovery rate as γ, the rate of exposed people in incubation period to infected people as α.

$$\begin{cases} \dfrac{dS}{dt} = -\dfrac{r\beta S(t)I(t)}{N} \\ \dfrac{dE}{dt} = \dfrac{r\beta S(t)I(t)}{N} - \alpha E(t) \\ \dfrac{dI}{dt} = \alpha E(t) - \gamma I(t) \\ \dfrac{dR}{dt} = \gamma I(t) \end{cases} \tag{2.3}$$

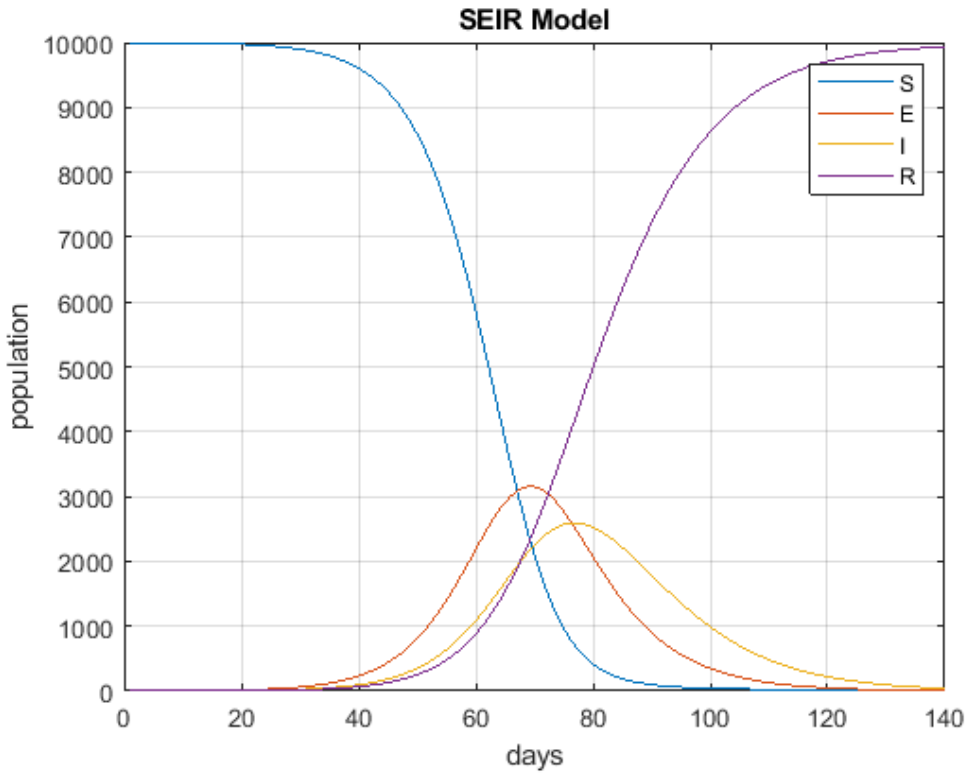


Figure 2.6: Simulate SEIR Model with N=10000, r=20, $\beta = 0.03, \gamma = 0.1, \alpha = 0.1$.

From Fig 2.5, there is maximum infection date at day 77. Part of susceptible people will contact healthy people, they will be exposed under the threaten of the virus, but not be infected. After

contacting other people, infected people become exposed, and there is still possibility that exposed people will not be infected. We can see that compared with other models the infected people are least on SEIR model.

2.6 Basic reproduction number

R_0, known as the basic reproductive number, is defined as the average number of people could be infected by an infected person in an epidemic, when there is no immunity in the community if no intervention gets involved from outside. If $R_0 < 1$, that is, the maximum number of people a patient can infect on average is less than 1, then the infection will gradually disappear. If $R_0 \geq 1$, the disease will continue to develop and become a pandemic [27].

So we can take the SIR system as an example to examine the epidemiological implications of the basic reproductive number here,

$$\begin{cases} \dfrac{dS}{dt} = -\dfrac{r\beta S(t)I(t)}{N} \\ \dfrac{dI}{dt} = \dfrac{r\beta S(t)I(t)}{N} - \gamma I(t) \\ \dfrac{dR}{dt} = \gamma I(t) \end{cases} \tag{2.4}$$

Add both sides of this equation to get

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$$

So

$$S(t) + I(t) + R(t) = k$$

where k is a constant number.

Since the first two equations do not include the $R(t)$, and $\frac{dR}{dt}$ can be calculated from $I(t)$; therefore we can get some conclusion from them,

$$\begin{cases} \dfrac{dS}{dt} = \dfrac{-r\beta I(t)S(t)}{N} \\ \dfrac{dI}{dt} = \dfrac{r\beta I(t)S(t)}{N} - \gamma I(t) \end{cases} \tag{2.5}$$

Let $\kappa = \frac{r\beta}{N}$, then we can write $Eq(2.5)$ to:

$$\begin{cases} \dfrac{dS}{dt} = -\kappa I(t)S(t) \\ \dfrac{dI}{dt} = I(t)(\kappa S(t) - \gamma) \end{cases} \tag{2.6}$$

Since $\frac{dS}{dt} < 0$, $S(t)$ presents a monotonic decline trend, with a lower limit of 0.

$$\lim_{t\to\infty} S(t) = S_\infty = 0$$

According to the $Eq(2.6)$,

$$\begin{cases} \dfrac{dI}{dS} = -1 + \dfrac{\rho}{S} \\ \rho = \dfrac{\gamma}{\kappa} \end{cases} \tag{2.7}$$

When $S = \rho$, I reaches its maximum value, then we can plot the trajectory diagram on the plane (S, I) in the *Figure* 2.7:

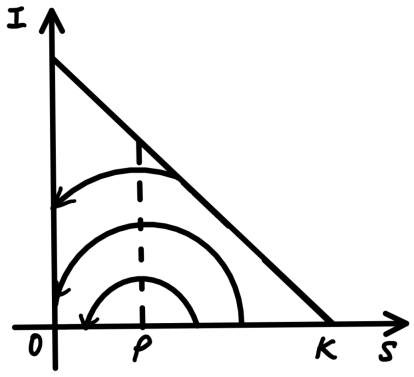


Figure 2.7: The trajectory map of System of Equations (2.7).

As the plot shown, all the equilibrium points of $Eq(2.6)$ are on the S-axis, and when $I = 0$, the $Eq(2.6)$ is in the steady state. When the initial value $S(0) = S_0 > \rho$, with the growth of time, the number of infected persons $I(t)$ will first increase, until $I(t)$ meets to the maximum value of $I(\rho)$, and then, it will gradually decrease and eventually die out.

This phenomenon indicates that if $S_0 > \rho$, or $S_0\kappa\frac{1}{\gamma} > 1$ the disease will be prevalent. Then,

$$R_0 = S_0\kappa\frac{1}{\gamma} = \frac{S_0}{\rho}$$

When $R_0 > 1$ the disease will become epidemic, the condition will be totally different when $R_0 < 1$, which means the disease will not cause an epidemic.

Since the γ is recovery rate, $\frac{1}{\gamma}$ is the average recovery time, which represents how long it takes for a person to become healthy again. Since the parameter γ is known from the research of COVID-19, if we have x numbers of patient, in the unit time, there are $x\gamma$ infected people would be recovered. Therefore, after $\frac{1}{\gamma}$ days, all patient will be recovered.

Chapter 3

SEQIR Model

3.1 Building a Model

Based on the basic model, a developed model is established. This new model includes time delay to simulate COVID-19. Since COVID-19 has the strong infectivity, therefore there is the home quarantine policy. Beside basic four groups of people, we add the quarantined population, Q, into the model and apply new coefficients representing parameters for latent period. Due to the home-quarantine-policy, many people decide to work and study at home, which decreased the average contact people per day, but there is barely a chance to not contacting other people at all, so there is still possibility that health quarantined people will be infected. Since the population is constant, so $N(t) = S(t) + E(t) + I(t) + Q(t) + R(t)$, where Q is the quarantined people at time t.

Along with the assumptions mentioned in Chapter 2, this model is established based on additional assumptions:

1. Recovered people will not be reinfected.
2. People who are quarantining at home will also have the possibility to be infected.

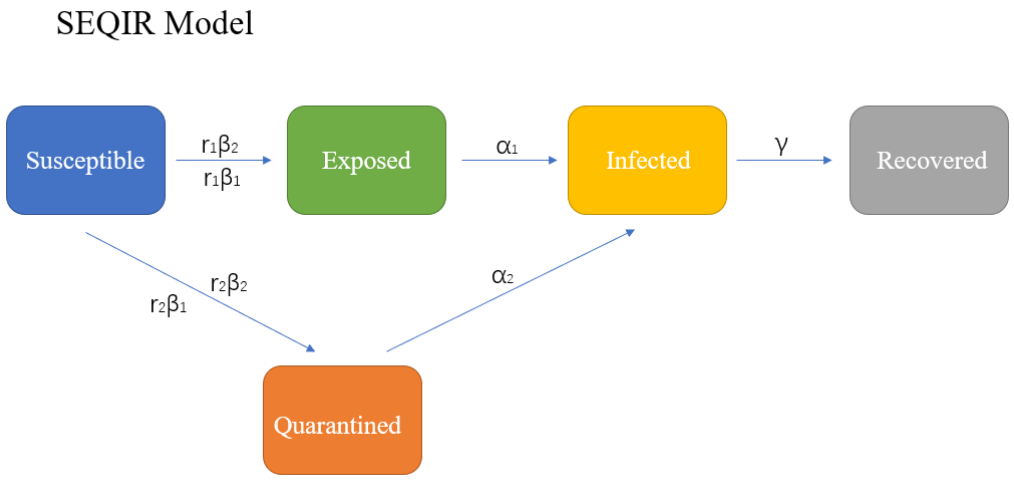


Figure 3.1: Schematic diagram for the SEQIR Model

From the figure above, we ignore the natural death and birth rate, and write the system as:

$$\begin{cases} \dfrac{dS}{dt} = -\dfrac{r_1\beta_1 S(t)I(t)}{N} - \dfrac{r_1\beta_2 S(t)Q(t)}{N} - \dfrac{r_2\beta_1 S(t)I(t)}{N} - \dfrac{r_2\beta_2 S(t)Q(t)}{N} \\ \dfrac{dE}{dt} = \dfrac{r_1\beta_1 S(t)I(t)}{N} + \dfrac{r_1\beta_2 S(t)Q(t)}{N} - \alpha_1 E(t) \\ \dfrac{dQ}{dt} = \dfrac{r_2\beta_1 S(t)I(t)}{N} + \dfrac{r_2\beta_2 S(t)Q(t)}{N} - \alpha_2 Q(t) \\ \dfrac{dI}{dt} = \alpha_1 E(t) + \alpha_2 Q(t) - \gamma I(t) \\ \dfrac{dR}{dt} = \gamma I(t) \end{cases} \tag{3.1}$$

Table 3.1: Standard parameter set for SEQIR Model

Notation	Unit	Parameter Name
r_1	people day^{-1}	average contact number of susceptible people
r_2	people day^{-1}	average contact number of quarantined people
β_1	people^{-1}	infection rate for each infected people
β_2	people^{-1}	infection rate for each quarantined people
α_1	% day^{-1}	incidence rate of exposed people per day
α_2	% day^{-1}	incidence rate of quarantined people per day
γ	% day^{-1}	recovery rate per day

The quarantined group and normal susceptible group had different numbers of contacting per day, so there are two values of r in the model. Due to the policies of preventing COVID-19 in public areas, people usually wore masks and kept social distance from each other, but people who quarantined at home either not contacting people at all or acting closely to family members. So, there were distinct values of infection rate β. With different contacting access for susceptible people and quarantined people, there were also two values of α incidence rates of two groups people.

3.2 Initial Value Problem and Numerical Solution

Most physical objects have overly complex relationships. Their state varies with time, place, and condition. Differential equations are about finding connections and patterns between their states and their transitions. In other words, the differential equation is the relationship between a function or functions and their derivatives. The initial value problem is the function (and possibly the derivative) of a given function at an initial point to be solved. In practical terms, we are concerned with the approximation of certain independent variables at a series of discrete points within a defined range. In real life, modeling the differential equation of problem modeling can be complex to solve. Therefore, there are common-used approaches to solve the equations. The first approach is to solve the problem approximately and reduce the problem to a differential equation that can be solved precisely, then approximate the solution of the original problem with the solution of the simplified equation [26].

We are approaching the Model by Euler's Method which is usually used to find the numerical solution of the initial value problem in ordinary differential equations. Our model is using forward Euler's method. This method uses the first two terms of the Taylor series expansion:

$$f(a+h) = f(a) + hf'(a)$$

Let

$$\frac{dy}{dx} = f(x,y), a \leq x \leq b, y(x_0) = y_0$$

Step one is to use a discrete method. Split the region from a to b into n small regions, $a \leq x \leq b$ is discretized into increments with h. The step length would be $h_i = (x_{i+1} - x_i)$. We want to approach a point x which is the solution of the initial problem. Let h be a small value, at point x, it can also be approximated as $x = (x_0) + h$. At the point $x_i = a + ih_i$, the equal length would be $h_i = h = \frac{b-a}{n}$.

To calculate the series of approximate value, $y_i = yx_i$, of function $y(x)$ at $a = x_0 < x_1 < \ldots < x_n = b$, it is necessary to take integrals from x_n to x_{n+1} at function $\frac{dy}{dx} = f(x,y), a \leq x \leq b$. Therefore,

$$\int_{x_n}^{x_{n+1}} y'dx = \int_{x_n}^{x_{n+1}} f(x, y(x))dx$$

$$y(x_{n+1}) - y(x_n) = \int_{x_n}^{x_{n+1}} f(x, y(x))dx$$

At Point x_n, replace the left side of the differential equation with the forward difference formula.

$$y'(x_n) \approx \frac{y(x_{n+1}) - y(x_n)}{h}$$

$$y_{x+1} \approx y(x_n) + hy'(x+n) = yn + hf(x_n, y_n)$$

From the equation above, there exist equations from y_1 to y_{n+1}

$$y_{n+1} = y_n + hf(x_n, y(x_n))$$

For each step of calculation y_n, it is only related to y_{n-1}. This method is called the forward Euler method. The advantage of it is it gives an explicit equation which is easier to implement and requires smaller operation per time-step. Adjoint to forward Euler method, backward Euler method is an implicit equation, which can be approached by:

$$y_{n+1} = y_n + hf(x_{n+1}, y_{n+1})$$

Since the time-step, h, is small, the error does not influence the model rapidly. Therefore, in our MatLab, we use the forward Euler method to approximate the equation.

3.3 Analyze the Model

With MatLab, we are capable of simulating the rise and fall of epidemics in Worcester. The purpose of fitting the data is to obtain a continuous function or a more dense discrete equation that is consistent with the realistic data. The data of death rate and recovery rate are based on WHO Coronavirus Dashboard [28]. The population of Worcester is about 185100 [20].

The trend of the actual data and estimated model is similar but the variables of our model are larger than the real data. The cause of such a huge difference is because we do not include the death rates and based on our assumption, all people in Worcester are eventually going to be infected, while in real life it is not possible. As we can see that after 120 days, the trend of epidemic is going smoother. The estimated graph with the variables:

Table 3.2: Values of parameter set for SEQIR Model.

Notation	r_1	r_2	β_1	β_2	α_1	α_2	γ
Value	2	0.5	0.2	0.2	0.05	0.1	0.97

Due to the strong intervention by the government during the epidemic period, the influence mechanism is more complicated, and it is difficult to directly adjust the parameters and fit the model to reflect the real situation.

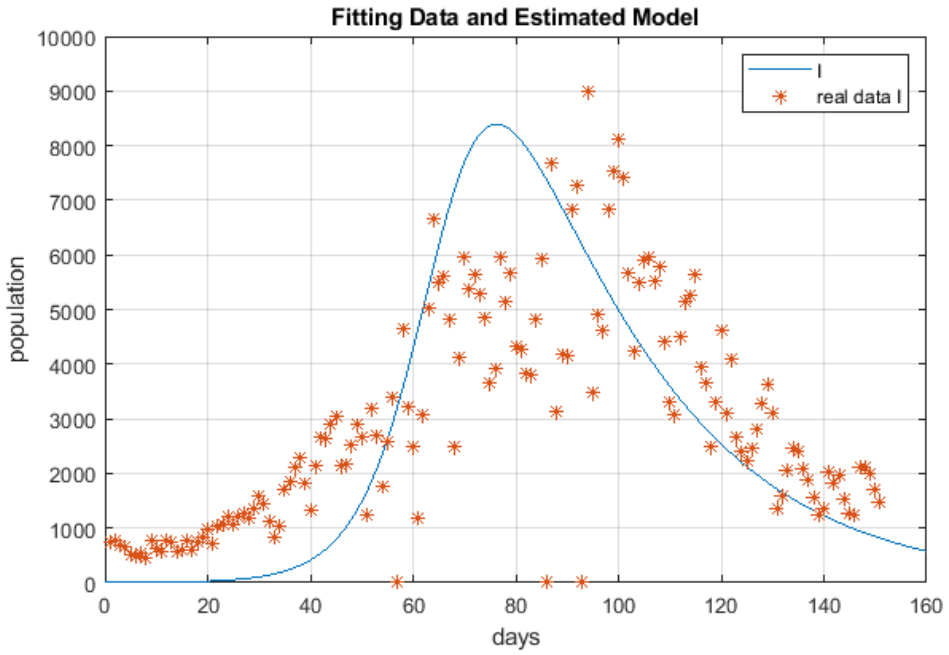


Figure 3.2: Fitting Worcester Infected Population Data using Estimated Model

The real life data in Fig3.2 is grouped and obtained from The New York Times [25]. Since the data was missing due to public holidays, the patients shown on the graph for real data is 0 for these days. The data is from October, 2020 to February, 2021.

As we change the number of quarantines, the number of infected people decreased; the infected people have arrived the peak early, and the curve of infection has also become smoother. Even if some people decide to quarantine at home, they still cannot avoid contact with exposed persons. Therefore, the number of quarantined people on our graph is decreasing, and the number of exposed people is increasing. Fig 3.3 indicates that.

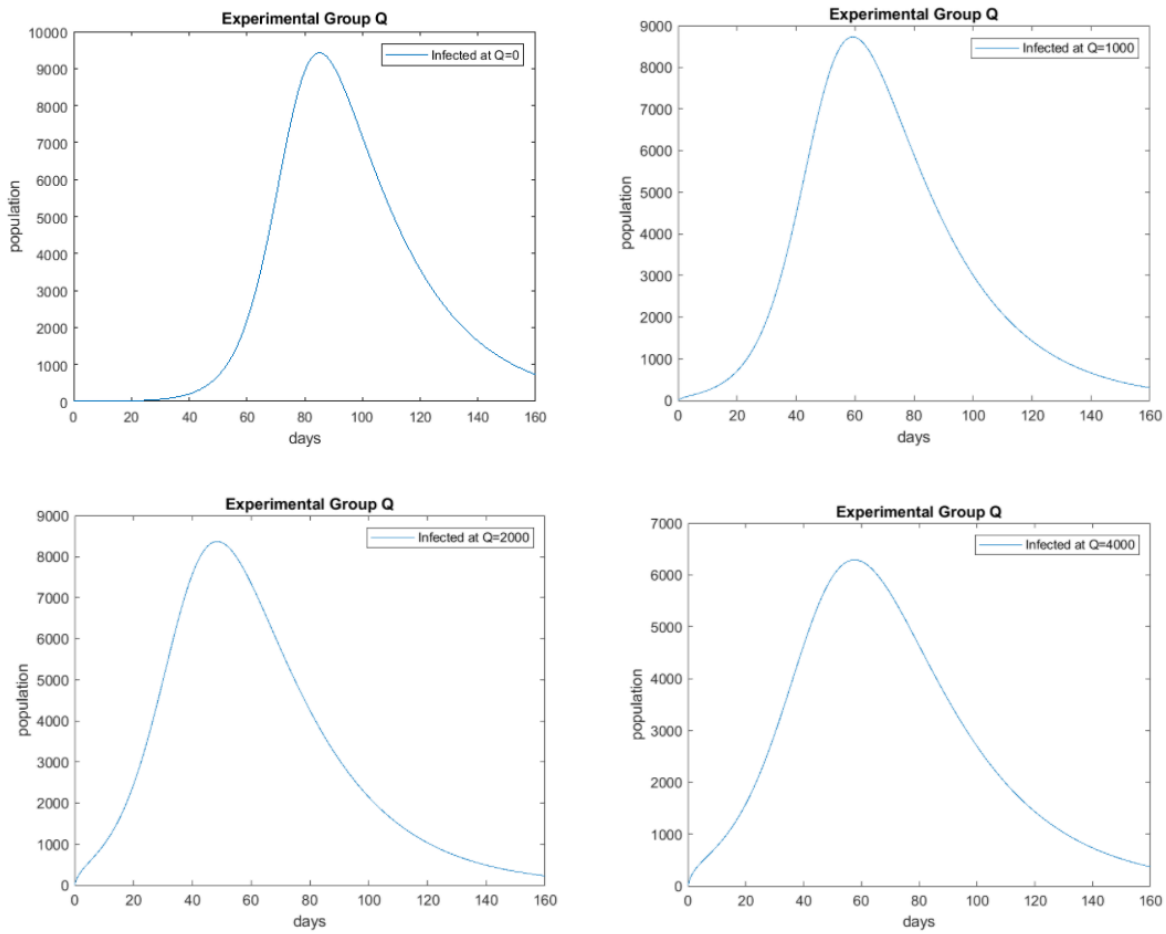


Figure 3.3: Model Simulation 1 - The Different values of quarantined population; N=185100, r=10, Q=0, Q=1000,Q=2000,Q=4000

As we keep social distance, the less people contact with other, the less people will be infected. One of the most special features of the COVID-19 is that people who are infected during the incubation period are also contagious. This has been confirmed by medical workers. For an infected person in the incubation period, he does not know that he has been infected. If the person does not wear mask or self-quarantine, everyone else contacted with him would have risk of infection. Therefore, an infected person in the incubation period may also become an important spreader of the virus.

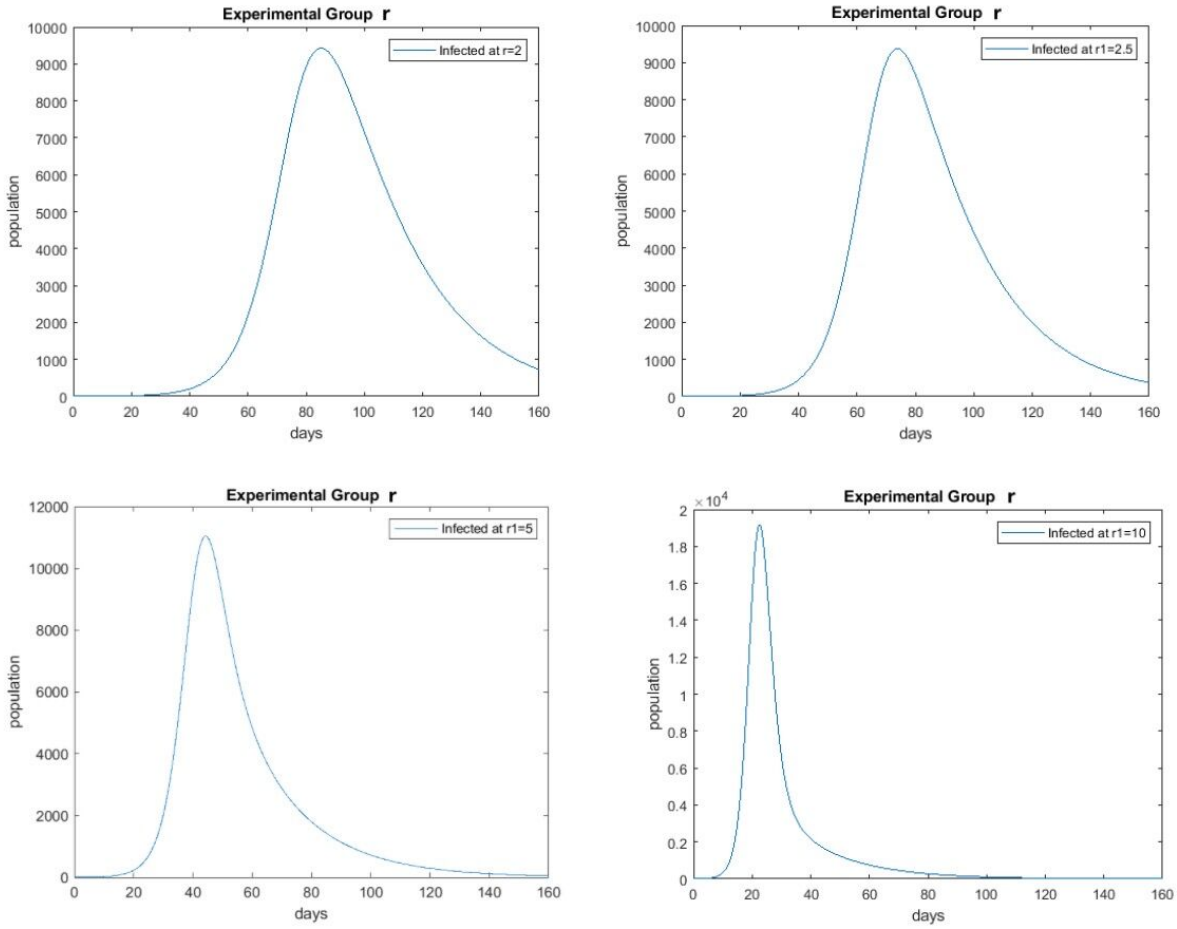


Figure 3.4: Simulation 2 - The different values of average contact number; N=185100, Q =0,r1=2, r1=2.5, r1=5, r1=10

3.4 Conclusion

Through the comparison of the above two situations, we can conclude that home quarantine and social distance can reduce the infectivity of infected persons during the incubation period. It is obviously beneficial for epidemic prevention and control. The effect of maintaining social distancing is even better than mask and quarantine. When an epidemic is approaching, reducing the route of transmission is an important way to reduce the number of infected people.

Although the improved model can make a more ideal trend analysis of the epidemic situation, the treatment of the later stage of the epidemic situation is still a deviation from the real situation. It is mainly caused by three reasons. Firstly, the SEIR model is too simple, and the situation is too idealized. The infection of COVID-19 is not only transmitted by infected people, but also by some susceptible people, while this part of the population is not reflected in the establishment of the model. Second, the COVID-19 is transmitted overseas, and the data gap between countries is huge. In this case, we do not make a good fit for the data. Thirdly, it is assumed that the population base of the data is large, which is a great challenge for the establishment of the model, because the general model requires a fixed population and no population communication. The most important point is to understand the implications of network dynamics for Disease Control. If we know the dynamics of transmission, we can effectively cut down the paths of disease spreading within a complex network and

make the most effective blocking strategy according to the characteristics of the virus. In the long run, it requires us to consider disease control as part of the design of social networks. Many problems have been exposed during the outbreak, such as the control of high-risk transmission routes, how to record and lock transmission routes to cut off in the first place, and how to build a most effective medical network, etc. Such a long-term approach would minimize the social risk of future transmission of a virus that is more dangerous than the COVID-19.

Chapter 4

Stability analysis on time delay of SEQIR Model

4.1 Time delay of SEIQR Model

Based on the model in Chapter 3, we studied other models for the topic of dynamic model. With reference to the model of article [29], we conceived of this model of COVID-19. We developed the model by adding natural mortality without disease in this model and changing the method of the quarantined people were calculated. Except the people who are quarantined at home, there are also infected people quarantined at hospital. Therefore, we included quarantined people from infected period. The time delay is applicable for simulating COVID-19, so we also learnt the time delay of the model from articles [22] [19].

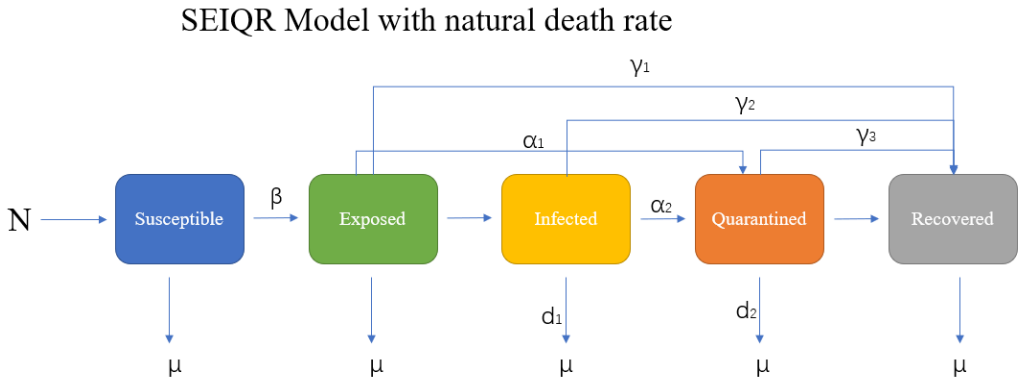


Figure 4.1: The schematic diagram of SEIQR Model with natural death rate.

Moreover, we introduced time delay to our system of equations. The mathematical model with time delay reflects that the law of motion change at time t not only depends on time t, but also could be affected by some conditions before time t. The mathematical model with time delay reflects that the law of motion change at time t not only depends on time t, but also could be affected by some conditions before time t.

In this model, we combined r and β from previous model. According to Chapter 3, r was the average number of contacting people per day, β was the probability of infection of the disease per person. This new β meant the effective contact rate, where $\beta_{new} = r\beta$.

In addition, the death rate of infected group and quarantined group included natural death rate, μ, and mortality of COVID-19, d_1 and d_2. Therefore, the death rate of these two groups were higher than other groups.

Table 4.1: Basic parameter set for SEIQR Model

Notation	Unit	Parameter Name
β	day^{-1}	Effective contact rate per day
N	people	Total population of the region
μ	day^{-1}	natural death rate per day
α_1	day^{-1}	quarantine rate for exposed people
α_2	day^{-1}	quarantine rate for infected people
d_1	day^{-1}	mortality of COVID-19 in infected group
d_2	day^{-1}	mortality of COVID-19 in quarantined group
γ_1	day^{-1}	recovery rate of exposed people
γ_2	day^{-1}	recovery rate of infected people
γ_3	day^{-1}	recovery rate of quarantined people

$$\begin{cases} \dfrac{dS}{dt} = N - \dfrac{\beta S(t)I(t)}{N} - \mu S(t) \\ \dfrac{dE}{dt} = \dfrac{\beta S(t)I(t)}{N} - \dfrac{\beta e^{-\mu\tau} S(t-\tau)I(t-\tau)}{N} - \alpha_1 E(t) - \gamma_1 E(t) - \mu E(t) \\ \dfrac{dI}{dt} = \dfrac{\beta e^{-\mu\tau} S(t-\tau)I(t-\tau)}{N} - \alpha_2 I(t) - \gamma_2 I(t) - d_1 I(t) - \mu I(t) \\ \dfrac{dQ}{dt} = \alpha_1 E(t) + \alpha_2 I(t) - d_2 Q(t) - \gamma_3 Q(t) - \mu Q(t) \\ \dfrac{dR}{dt} = \gamma_1 E(t) + \gamma_2 I(t) + \gamma_3 Q(t) - \mu R(t) \end{cases} \tag{4.1}$$

$S(t-\tau)$ and $I(t-\tau)$ represent the susceptible and infected population at $t-\tau$ moment respectively, and τ is the incubation period of the virus.

If we assume that the incubation period of the virus is τ, the number of infected cases at time t is $x(t)$. $x(-\tau) = x_0$, $e^{\mu\tau}$ is the probability distribution of the change in the number of people in the incubation period.

We firstly need to understand the meaning of $e^{-\mu\tau}$. Let N(t) be the total population at time t, and $N(0) = x_0$. The death rate is μ, and the pattern of the changing of population can be written as:

$$\begin{cases} \dfrac{dN}{dt} = -\mu N \\ N(0) = x_0 \end{cases} \tag{4.2}$$

The solution is:

$$N(t) = x_0 e^{-\mu t}$$

$$\frac{N(t)}{N_0} = e^{-\mu t}$$

It is shown that ratio of population $\frac{N(t)}{N_0}$ during the illness period has an exponential distribution $e^{-\mu t}$, which means the probability of people surviving form time $t = 0$ to time $t = t$ is $e^{-\mu t}$.

If there is incubation period τ at time $t-\tau$, the exposed people at time t will become infected people. Since there is natural death rate, the probability that exposed people will survive at time t is:

$$\beta I(t-\tau)S(t-\tau)e^{-\mu\tau}$$

In this chapter, we determined the basic reproduction number, which is the threshold expression to distinguish whether a disease is prevalent or not. While we use Routh-Hurwitz stability criterion to evaluate the stability of equilibrium points with different value of time delay, we can determine whether a linear system has a positive root in the right half of the complex plane in a polynomial equation without having to solve the equation. Characteristic equation has to be on the left-half plane just like the poles of the transfer function to be a stable system [2].

4.2 Steady States

This section introduces the steady states. In real life, we can only simulate many problems with nonlinear system equations, but we wish to approach the solution of equations with linear system. The equilibrium point is the point where the derivative of each variable in the system equation is 0. The nonlinear system can be approximated to form an independent new linear system at any point. This means that it is possible to meet the balance requirement at any point. An equilibrium point can be stable or unstable [1].

Since we have a constant population $N(t) = N = S(t) + E(t) + I(t) + Q(t) + R(t)$, we are able to simplify the equations. According to Eq.(4.1), there is no Q(t) and R(t) involved in the first three equations, which means once we know S(t), E(t) and I(t), we can express Q(t) and I(t) respect to first three equations. Therefore, we can omit $\frac{dQ}{dt}$ and $\frac{dR}{dt}$ during the calculation of steady states. The following three equations will be considered are:

$$\begin{cases} \dfrac{dS}{dt} = N - \dfrac{\beta S(t)I(t)}{N} - \mu S(t) \\ \dfrac{dE}{dt} = \dfrac{\beta S(t)I(t)}{N} - \dfrac{\beta e^{-\mu\tau}S(t-\tau)I(t-\tau)}{N} - \alpha_1 E(t) - \gamma_1 E(t) - \mu E(t) \\ \dfrac{dI}{dt} = \dfrac{\beta e^{-\mu\tau}S(t-\tau)I(t-\tau)}{N} - \alpha_2 I(t) - \gamma_2 I(t) - d_1 I(t) - \mu I(t) \end{cases} \tag{4.3}$$

Now let S^*, E^*, I^* be equilibrium points,

$$\lim_{t\to\infty} S(t) = \lim_{t\to\infty} S(t-\tau) = S^*$$

$$\lim_{t\to\infty} E(t) = \lim_{t\to\infty} E(t-\tau) = E^*$$

$$\lim_{t\to\infty} I(t) = \lim_{t\to\infty} I(t-\tau) = I^*$$

By setting $\frac{dS}{dt}$, $\frac{dE}{dt}$, and $\frac{dI}{dt}$ to be zero, we can calculate the steady-states.

$$0 = N - \frac{\beta S^* I^*}{N} - \mu S^* \tag{4.4a}$$

$$0 = \frac{\beta S^* I^*}{N} - \frac{\beta e^{-\mu\tau} S^* I^*}{N} - \alpha_1 E^* - \gamma_1 E^* - \mu E^* \tag{4.4b}$$

$$0 = \frac{\beta e^{-\mu\tau} S^* I^*}{N} - \alpha_2 I^* - \gamma_2 I^* - d_1 I^* - \mu I^* \tag{4.4c}$$

From $Eq(4.4c)$,

$$0 = I^*(\frac{\beta e^{-\mu\tau} S^*}{N} - (\alpha_2 + \gamma_2 + d_1 + \mu)) \tag{4.5}$$

In order to satisfy $Eq(4.5)$, there are two conditions should be considered:

$$I^* = 0$$

or,

$$S^* = \frac{N(\alpha_2 + \gamma_2 + d_1 + \mu)}{\beta e^{-\mu\tau}}$$

Substitute $I^* = 0$ into $Eq(4.4)$.

$$\begin{cases} 0 = N - \dfrac{\beta S^* 0}{N} - \mu S^* \\ 0 = \dfrac{\beta S^* 0}{N} - \dfrac{\beta e^{-\mu\tau} S^* 0}{N} - \alpha_1 E^* - \gamma_1 E^* - \mu E^* \end{cases} \tag{4.6}$$

$$\begin{cases} S^* = \dfrac{N}{\mu} \\ 0 = 0 \end{cases} \tag{4.7}$$

When $I^* = 0$, steady state E_1 is:

$$E_1 = (\frac{N}{\mu}, 0, 0)$$

.

To simplify the calculation, let $(\alpha_2 + \gamma_2 + d_1 + \mu) = a$. Based on the basic reproduction number we introduced in Chapter 2, the R_0 of this system is:

$$R_0 = \frac{\beta e^{-\mu\tau}}{a}$$

Substitute $S^* = \frac{N}{R_0}$ into $Eq(4.4)$.

$$\begin{aligned} 0 &= N - \frac{\beta S^* I^*}{N} - \mu S^* \\ 0 &= N - \beta \frac{1}{R_0} I^* - \mu \frac{N}{R_0} \\ I^* &= \frac{N(\mu - R_0)}{\beta} \end{aligned}$$

Substitute I^* and S^* into $Eq(4.4b)$, we have:

$$\begin{aligned} 0 &= \frac{\beta S^* I^*}{N} - \frac{\beta e^{-\mu\tau} S^* I^*}{N} - \alpha_1 E^* - \gamma_1 E^* - \mu E^* \\ (\alpha_1 - \gamma_1 - \mu) E^* &= \frac{aN(-\mu a + \beta e^{-\mu\tau})}{e^{-\mu\tau}} - \frac{aN(-\mu a + \beta e^{-\mu\tau}) e^{-\mu\tau}}{e^{-\mu\tau}} \\ E^* &= \frac{N(R_0 - \mu)(1 - e^{-\mu\tau})}{R_0(\alpha_1 + \gamma_1 + \mu)} \end{aligned}$$

When $S^* = \frac{aN}{\beta e^{-\mu\tau}} = \frac{N}{R_0}$, steady state E_2 is:

$$E_2 = (\frac{N}{R_0}, \frac{N(R_0-\mu)(1-e^{-\mu\tau})}{R_0(\alpha_1+\gamma_1+\mu)}, \frac{N(R_0-\mu)}{\beta})$$

4.3 Characteristic Equation

There are mainly two kinds of methods to study the stability of time-delay dynamic systems. One is based on Lyapunov function/functional series, and the other is based on characteristic root analysis of system equations. Since the results obtained by these methods are often too conservative, there is no rule to follow in constructing Lyapunov function. The estimation of total derivatives along the system trajectory depends on the inequality estimation skill, so characteristic root analysis is always used to study the stability of time-delay dynamic systems in practical applications.

If the linearized delay differential equation at at the equilibrium point of a nonlinear delay differential equation has no characteristic root with the real part of zero, then the local stability of the zero solution of the nonlinear delay differential equation is consistent with the zero solution of its linearized equation. In particular, if all the characteristic roots of a delayed linearized differential equation have negative real parts, then the zero solution of the equation is asymptotically stable, and thus the zero solution of the original equation is also asymptotically stable. Therefore, the focal point of local stability analysis is to analyze the distribution of characteristic roots on the complex plane[13].

Characteristic equations with time delay differential system have the polynomial form:

$$P(\lambda,\tau) = P_1(\lambda) + P_2(\lambda)e^{=\lambda\tau} = 0$$

In SEIQR we used the Hurwitz criterion to find the root of characteristic equation for each steady state in different time delay value.

$$A_1 = \begin{bmatrix} \frac{\partial\frac{dS}{dt}}{\partial S(t)} & \frac{\partial\frac{dS}{dt}}{\partial E(t)} & \frac{\partial\frac{dS}{dt}}{\partial I(t)} \\ \frac{\partial\frac{dE}{dt}}{\partial S(t)} & \frac{\partial\frac{dE}{dt}}{\partial E(t)} & \frac{\partial\frac{dE}{dt}}{\partial I(t)} \\ \frac{\partial\frac{dI}{dt}}{\partial S(t)} & \frac{\partial\frac{dI}{dt}}{\partial E(t)} & \frac{\partial\frac{dI}{dt}}{\partial I(t)} \end{bmatrix} \tag{4.8}$$

$$= \begin{bmatrix} -\frac{\beta I(t)}{N} - \mu & 0 & -\frac{\beta S(t)}{N} \\ \frac{\beta I(t)}{N} & -\alpha_1 - \gamma_1 - \mu & \frac{\beta S(t)}{N} \\ 0 & 0 & a \end{bmatrix} \tag{4.9}$$

$$= \begin{bmatrix} -\frac{\beta I^*}{N} - \mu & 0 & -\frac{\beta S^*}{N} \\ \frac{\beta I^*}{N} & -\alpha_1 - \gamma_1 - \mu & \frac{\beta S^*}{N} \\ 0 & 0 & -a \end{bmatrix} \tag{4.10}$$

$$A_2 = \begin{bmatrix} \frac{\partial \frac{dS}{dt}}{\partial S(t-\tau)} & \frac{\partial \frac{dS}{dt}}{\partial E(t-\tau)} & \frac{\partial \frac{dS}{dt}}{\partial I(t-\tau)} \\ \frac{\partial \frac{dE}{dt}}{\partial S(t-\tau)} & \frac{\partial \frac{dE}{dt}}{\partial E(t-\tau)} & \frac{\partial \frac{dE}{dt}}{\partial I(t-\tau)} \\ \frac{\partial \frac{dI}{dt}}{\partial S(t-\tau)} & \frac{\partial \frac{dI}{dt}}{\partial E(t-\tau)} & \frac{\partial \frac{dI}{dt}}{\partial I(t-\tau)} \end{bmatrix} \tag{4.11}$$

$$= \begin{bmatrix} 0 & 0 & 0 \\ -\frac{\beta e^{-\mu\tau} I(t-\tau)}{N} & 0 & -\frac{\beta e^{-\mu\tau} S(t-\tau)}{N} \\ \frac{\beta e^{-\mu\tau} I(t-\tau)}{N} & 0 & \frac{\beta e^{-\mu\tau} S(t-\tau)}{N} \end{bmatrix} \tag{4.12}$$

$$= \begin{bmatrix} 0 & 0 & 0 \\ -\frac{\beta e^{-\mu\tau} I^*}{N} & 0 & -\frac{\beta e^{-\mu\tau} S^*}{N} \\ \frac{\beta e^{-\mu\tau} I^*}{N} & 0 & \frac{\beta e^{-\mu\tau} S^*}{N} \end{bmatrix} \tag{4.13}$$

The Jacobian matrix of the differential equations is $J = A_1 + A_2 e^{-\mu\tau}$

$$J = \begin{bmatrix} \frac{-\beta I^*}{N} - \mu & 0 & -\frac{-\beta S^*}{N} \\ \frac{\beta I^*}{N} - \frac{e^{-\mu\tau} e^{-\lambda\tau}}{N} & -\alpha_1 - \gamma_1 - \mu & \frac{\beta S^*}{N} - \frac{-\beta S^* e^{-\mu\tau} e^{-\lambda\tau}}{N} \\ \frac{\beta I^* e^{-\mu\tau} e^{\lambda\tau}}{N} & 0 & -a + \frac{\beta S^* e^{-\lambda\tau} e^{-\mu\tau}}{N} \end{bmatrix} \tag{4.14}$$

Therefore, we have Jocobian matrix for steady states at E_1.

$$J_{E_1} = \begin{bmatrix} -\mu & 0 & \frac{\beta}{\mu} \\ -\frac{e^{-\mu\tau} e^{-\lambda\tau}}{N} & -\alpha_1 - \gamma_1 - \mu & \frac{\beta - \beta(e^{-\mu\tau-\lambda\tau})}{\mu} \\ 0 & 0 & -a + \frac{\beta e^{-\mu\tau-\lambda\tau}}{\mu} \end{bmatrix} \tag{4.15}$$

The Jocobian matrix for steady states at E_2 is :

$$J_{E_2} = \begin{bmatrix} -R_0 & 0 & -\frac{\beta}{R_0} \\ R_0(1 - e^{-\mu\tau-\lambda\tau}) & -\alpha_1 - \gamma_1 - \mu & \frac{\beta}{R_0} \\ R_0 e^{-\mu\tau-\lambda\tau} & 0 & -a + \frac{\beta}{R_0} e^{-\mu\tau-\lambda\tau} \end{bmatrix} \tag{4.16}$$

The characteristic equations can be calculated by the determinant of difference between identity matrix with same size and eigenvalues of matrix itself as:

$$P_{E_1}(\lambda, \tau) = det(J_{E_1} - \lambda)I$$

$$P_{E_1} = \begin{bmatrix} -\mu - \lambda & 0 & \frac{\beta}{\mu} \\ -\frac{e^{-\mu\tau} e^{-\lambda\tau}}{N} & -\alpha_1 - \gamma_1 - \mu - \lambda & \frac{\beta - \beta(e^{-\mu\tau-\lambda\tau})}{\mu} \\ 0 & 0 & -a + \frac{\beta e^{-\mu\tau-\lambda\tau}}{\mu} - \lambda \end{bmatrix} \tag{4.17}$$

The first characteristic equation with E_1 is

$$(-\mu - \lambda)(-\alpha_1 - \gamma_1 - \mu - \lambda)(-a + \frac{\beta e^{-\mu\tau-\lambda\tau}}{\mu} - \lambda) = 0$$

We have $\lambda_1 = -\mu$, $\lambda_2 = -(\mu + \alpha_1 + \gamma_1)$, so other roots are based on:

$$\begin{aligned} 0 &= \lambda + a - \frac{\beta e^{-\mu\tau-\lambda\tau}}{\mu} \\ \lambda &= \frac{\beta e^{-\mu\tau-\lambda\tau}}{\mu} - a \\ \lambda &= -a(1 - \frac{\beta e^{-\mu\tau-\lambda\tau}}{a}) \\ &= -a(1 - R_0 e^{-\lambda\tau}) \end{aligned}$$

Meanwhile, $e^{ib\tau} = acos(b\tau) - isin(b\tau)$, the magnitude of $e^{ib\tau}$, $|e^{ib\tau}| = 1$.

$$\begin{aligned} e^{-\lambda\tau} &= e^{-(a+ib)\tau} \\ &= e^{-a\tau} e^{ib\tau} \end{aligned}$$

Proved by contradiction, we assume that λ has a positive real root. The real part of λ should be positive, and it should be a positive real number.

$$\begin{aligned} &R_e(\lambda) \geq 0 \\ &R_e(\lambda) \leq -a(1 - R_0 e^{-\lambda\tau}) \\ &R_e(\lambda) \leq -a(1 - R_0) \end{aligned}$$

When $R_0 < 1$, we have $R_e(\lambda) < 0$. By contradiction, we proved that λ does not have a positive real root, when $R_0 < 1$.

On the other hand, when $R_0 > 1$, λ has positive real roots.

If $R_0 < 1$, equilibrium point E_1 is locally stable; if $R_0 > 1$, equilibrium point E_1 is unstable, while there exists an endemic equilibrium point, E_2. Since the eigenvalues of this characteristic equation is always negative when $R_0 < 1$, the eigenvalues do not cross from left-half plane to right-half plane. There is not a Hopf bifurcation.

We now were trying to prove for any τ, when $R_0 > 1$, E_2 is locally stable.

$$P_{E_2} = \begin{bmatrix} -R_0 - \lambda & 0 & -\frac{\beta}{R_0} \\ R_0(1 - e^{-\mu\tau-\lambda\tau}) & -\alpha_1 - \gamma_1 - \mu - \lambda & \frac{\beta}{R_0} \\ R_0 e^{-\mu\tau-\lambda\tau} & 0 & -a + \frac{\beta}{R_0} e^{-\mu\tau-\lambda\tau} - \lambda \end{bmatrix} \tag{4.18}$$

The characteristic equation for E_2 is:

$$P_{E_2}(\lambda, \tau) = (-\alpha_1 - \gamma_1 - \mu - \lambda)((-R_0 - \lambda)(-a + \frac{\beta}{R_0} e^{-\mu\tau-\lambda\tau} - \lambda) + \frac{\beta}{R_0} R_0 e^{-\mu\tau-\lambda\tau}) \tag{4.19}$$

Let $\alpha_1 + \gamma_1 + \mu = b$

$$Equ(4.19) = (-b - \lambda)((-R_0 - \lambda)(-a + \frac{\beta}{R_0} e^{-\mu\tau-\lambda\tau} - \lambda) + \beta e^{-\mu\tau-\lambda\tau})$$

Therefore, the first root of the characteristic equation is obvious

$$\lambda_1 = -b$$

Now, we are going to calculate other possible roots.

$$0 = (-R_0 - \lambda)(-a + \frac{\beta}{R_0}e^{-\mu\tau-\lambda\tau} - \lambda) + \beta e^{-\mu\tau-\lambda\tau} \tag{4.20a}$$

$$= (R_0 + \lambda)(\lambda + a - \frac{\beta}{R_0}e^{-\mu\tau-\lambda\tau}) - (\beta e^{-\mu\tau-\lambda\tau}) \tag{4.20b}$$

We first consider the condition when $\tau = 0$, $R_0 = \frac{\beta}{a}$, $\frac{\beta}{R_0}e^{-\mu\tau-\lambda\tau} = a$.

$$\begin{cases} -a + a + \dfrac{\beta}{a} > 0 \\ \beta + \dfrac{\beta}{a}(a - a) > 0 \end{cases} \tag{4.21}$$

According to Hurtwiz criterion, when $\tau = 0$, there exists an locally stable point for E_2.

Under the condition when $\tau \neq 0$, if there exists a pure imagery root $\lambda = i\omega$. We substitute it into *Eq.*(4.20), and separate the real root and imagery parts.

$$\begin{aligned} 0 &= (R_0 + \lambda)(\lambda + a - \frac{\beta}{R_0}e^{-\mu\tau-\lambda\tau}) - (\beta e^{-\mu\tau-\lambda\tau}) \\ &= \lambda^2 + (R_0 + a)\lambda - (\frac{\beta\lambda}{R_0} + 2\beta)e^{-\mu\tau-\lambda\tau} - aR_0 \\ &= -\omega^2 + (R_0 + a)i\omega - (\frac{i\beta\omega}{R_0} + 2\beta)e^{-\mu\tau}(cos(\omega\tau) - isin(\omega\tau)) - aR_0 \end{aligned}$$

$$\begin{cases} -\omega^2 - 2\beta e^{-\mu\tau}cos(\omega\tau) - \omega\dfrac{\beta}{R_0}e^{-\mu\tau}sin(\omega\tau) - aR_0 = 0 \\ i(\omega(R_0 + a) + 2\beta e^{-\mu\tau}sin(\omega\tau) - \omega\dfrac{\beta}{R_0}e^{-\mu\tau}cos(\omega\tau)) = 0 \end{cases} \tag{4.22}$$

Eq.(4.22) can be written as:

$$\begin{cases} sin(\omega\tau) = \dfrac{-\omega^3 + (-2R_0^2 - 3aR_0)\omega}{\frac{\beta}{R_0}e^{-\mu\tau}(\omega^2 + 4R_0^2)} \\ cos(\omega\tau) = \dfrac{(-R_0 + a)\omega^2 - 2aR_0^2}{\frac{\beta}{R_0}e^{-\mu\tau}(\omega^2 + 4R_0^2)} \end{cases} \tag{4.23}$$

We can expand the *Eq.*(4.23) by $R_0 = \frac{\beta e^{-\mu\tau}}{a}$

$$\begin{cases} sin(\omega\tau) = \dfrac{-\omega^3 + (-2\frac{\beta e^{-2\mu\tau}}{a^2} - 3a\frac{\beta e^{-\mu\tau}}{a})\omega}{\frac{a}{e^{-\mu\tau}}(\omega^2 + 4\frac{\beta e^{-2\mu\tau}}{a^2})} \\ cos(\omega\tau) = \dfrac{(-\frac{\beta e^{-\mu\tau}}{a} + a)\omega^2 - 2a\frac{\beta e^{-2\mu\tau}}{a^2}}{\frac{a}{e^{-\mu\tau}}(\omega^2 + 4\frac{\beta e^{-2\mu\tau}}{a^2})} \end{cases} \tag{4.24}$$

By squaring and adding the $Eq(4.23)$, we get:

$$\omega^6 + (5R_0^2 + 4aR_0)\omega^4 + (16R_0^4 + 16R_0^3 + 26R_0^2a^2)\omega^2 + 4a^2R_0^4 + \beta^2e^{-2\mu\tau}16R_0^2 = 0 \tag{4.25}$$

$$\begin{cases} p_1 = 5R_0^2 + 4aR_0 \\ p_2 = 16R_0^4 + 16R_0^3 + 26R_0^2a^2 \\ p_3 = 4a^2R_0^4 + \beta^2e^{-2\mu\tau}16R_0^2 \end{cases} \tag{4.26}$$

Let $\omega^2 = \psi$ we can know that:

$$h(\psi) = \psi^3 + p_1\psi^2 + p_2\psi + p_3 = 0.$$

Since $R_0 > 1$,$a > 0$, we have

$$p_1 > 0, p_2 > 0, p_3 > 0$$

In conclusion, there exists a locally asymptotically stable point for E_2.

4.4 Numerical Simulation of SEIQR Model

Epidemic trend of infectious diseases is related to R_0, so it is necessary to study and analyze the properties of R_0. This section described the factors that affect R_0 through graphically through MATLAB to provide evidence for disease prevention and control. The study analyzed the dynamic stability of the equilibrium point of the model, and defined the spread and extinction of the threshold value, R_0.

We estimated two sets value of R_0 form $Eq(4.1)$:

The first set of values:

$$\beta = 0.2, \alpha_1 = 0.3, \alpha_2 = 0.2, \gamma_1 = 0.1, \gamma_2 = 0.1, \mu = 0.05, \tau = 14, N = 1, d_1 = 0.02$$

It is easy to conclude that the $R_0 = 0.25465 < 1$, therefore, there is one disease-free equilibrium point at $E_0 = (0.94297, 0, 0)$. When approaching E_0, the system is locally asymptotically stable.

The second set of values:

$$\beta = 0.9, \alpha_1 = 0.3, \alpha_2 = 0.2, \gamma_1 = 0.1, \gamma_2 = 0.1, \mu = 0.05, \tau = 14, N = 1, d_1 = 0.02$$

It is easy to conclude that the $R_0 = 1.14596 > 1$, therefore, there are one disease-free equilibrium point at $E_0 = (1.07965, 0, 0)$, and one endemic equilibrium point $E_1 = (0.0.87263, 0.92514, 1.21773)$. When approaching E_1, the system is locally asymptotically stable.

The following numerical simulation examples show that when $R_0 < 1$, the disease-free equilibrium point of the model is locally asymptotically stable, and when $R_0 > 1$, the endemic equilibrium point of the model becomes locally asymptotically stable.

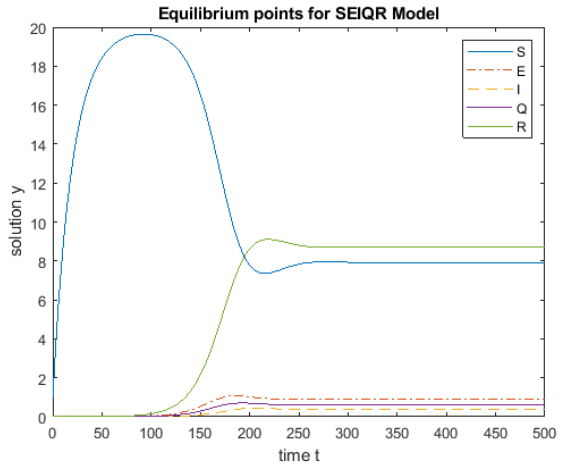


Figure 4.2: Equilibrium point for $R_0 < 1$, when $\beta = 0.2, \alpha_1 = 0.3, \alpha_2 = 0.2, \gamma_1 = 0.1, \gamma_2 = 0.1, \mu = 0.05, \tau = 14, N = 1, d_1 = 0.02$.

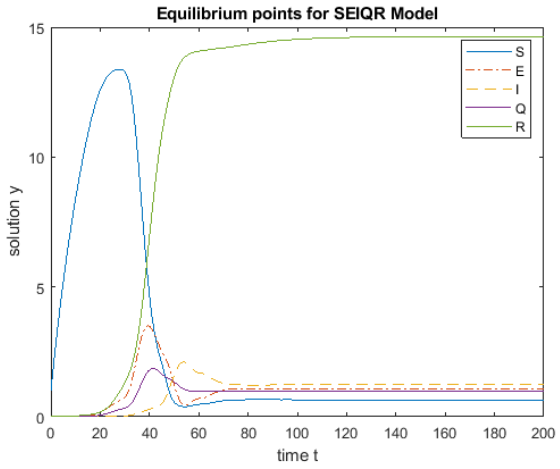
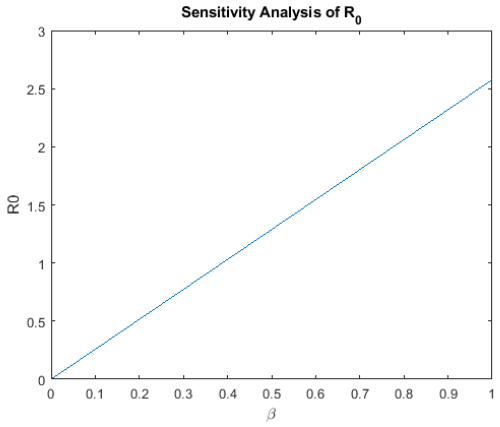
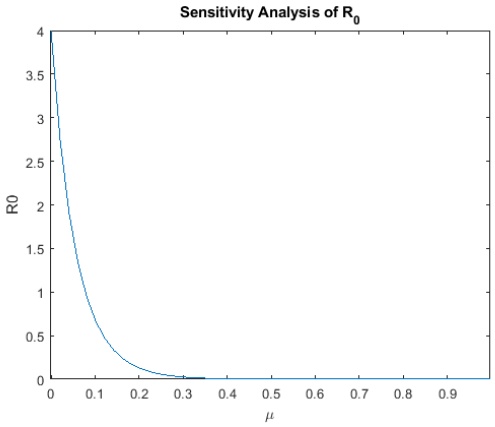


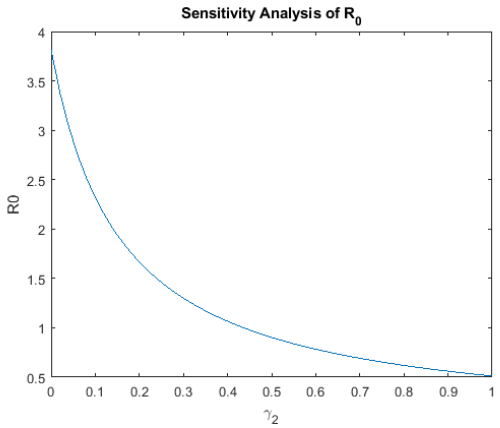
Figure 4.3: Equilibrium point for $R_0 > 1$, when $\beta = 0.9, \alpha_1 = 0.3, \alpha_2 = 0.2, \gamma_1 = 0.1, \gamma_2 = 0.1, \mu = 0.05, \tau = 14, N = 1, d_1 = 0.02$.

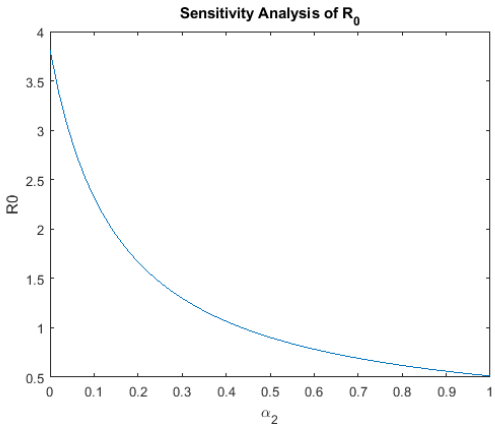
4.5 Sensitivity Analysis of R_0

Sensitivity analysis has been used to detect the influence of different parameters on model output. In this section, we study the influence of different parameters on the basic regeneration number R_0, so as to determine the important parameters that affect the model.









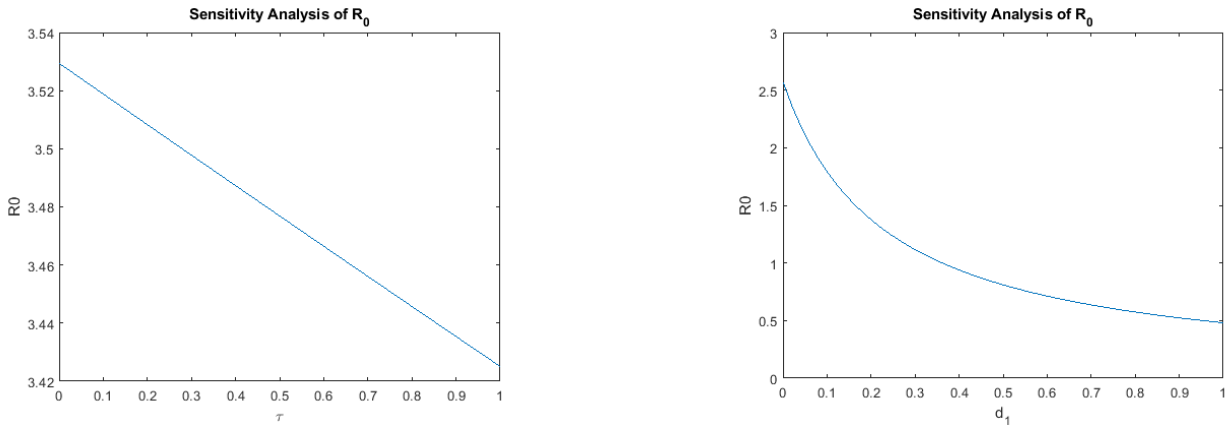


Figure 4.4: Plots of relationship between the basic reproduction number R_0 and the following parameters: (a) β (infected rate); (b) μ (natural death rate); (c) γ_2 (recovery rate of infected people); (d) α_2 (quarantine rate of infected people); (e) τ (time delay); (f) d_1 (death rate of infected people due to COVID-19).

It can be seen from the figure above that when β and R_0 are positively correlated, and other parameters are negatively correlated. Among all the negatively correlated parameters, d_1 has a smaller impact on R_0, and μ has a greater impact on R_0. In other words, when the parameter value is larger, controlling β is more important than controlling other parameters.

4.6 Conclusion

We studied the SEIQR model with time delay, and through mathematical analysis, gave the critical parameter conditions which proved there did not exist the Hopf bifurcation. It determined whether the disease is prevalent depends on the threshold basic reproduction number R_0. When $R_0 < 1$, the disease-free equilibrium point is locally asymptotically stable; when $R_0 > 1$, the endemic equilibrium point is locally asymptotically stable. Therefore, mathematically, the values of time delay, τ, would not affect the equilibrium points for the model. In this model, we had parts of exposed people who were infected would be recovered by natural immunity. These naturally recovered people had certain influence on the system.

Chapter 5

Conclusion

The development and evolution of most epidemics have two notable characteristics: the first one is time delay. The evolution of the system not only depends on the current state, but also is related to the past state. The second one is contagion. Living creatures and virus exists in the same planet sharing same natural environment. Virus can be cross transmitted among most creatures. Most epidemics can be co-transmitted among humans and animals. Therefore, the utilizes of dynamic model with time delay describes accurately the phenomenon of the development of epidemics.

In this MQP, there are three major parts. The first and second chapter introduces the background of COVID-19 and significance of several types of typical epidemiological system. These two chapters provide a solid knowledge for us on building the model for COVID-19 by using epidemiological systems. We consider the advantage and disadvantage of SI model, SIR model and SEIR model before constructing our model.

The third chapter establishes a COVID-19 model with the number of people in quarantine. After discussing the difficulties of building a model, we attempt to establish a model to simulate COVID-19. Through MATLAB simulations, we obtain important parameters that determine the number of infected people. Although the classic disease transmission dynamic model has made great achievements in predicting certain specific diseases, they are often too idealistic and ignore some important aspects, such as the number of contacts, the incubation period, and the evolution of disease. In this chapter, we consider the COVID-19 transmission model with an incubation period and a quarantine period. The numerical solution of the system is solved by forward Euler method.

The fourth part analyzes equilibrium points for nonlinear system of equations based on time delay. Utilizing the Routh–Hurwitz stability criterion, we prove the steady states of the equilibrium point of endemic diseases. It can be concluded that when the basic reproduction number is less than 1, the disease-free equilibrium point is locally asymptotically stable. When the basic reproduction number is greater than 1, the endemic equilibrium point is locally asymptotically stable.

Appendix A

MATLAB Code

This is MatLab code for simulating SEIQR Model.

```
%-------------------------------------------------------------------------
%-----------------------------------------Chapter 3 coding----------------
clear;clc;

%-------------------------------------------------------------------------
%-------------------------------------------------------------------------
N = 185100;
E = 0;
Q = 0;
I = 10;
S = N - I;
R = 0;

r1 = 2.5;
r2 = 0.5;
B1 = 0.25;
B2 = 0.105;
a1 = 0.5;
a2 = 0.05;
y = 0.97;
h = 0.01;

T = h:h:160;
for idx = 1:length(T)-1
    S(idx+1) = S(idx) + h*(- r1*B1*S(idx)*I(idx)/N - r1*B2*S(idx)*Q(idx)/N - r2*B1*S(
        idx)*I(idx)/N - r2*B2*S(idx)*Q(idx)/N);
    E(idx+1) = E(idx) + h*(r1*B1*S(idx)*I(idx)/N-a1*E(idx)+r1*B2*S(idx)*Q(idx)/N);
    Q(idx+1) = Q(idx) + h*(r2*B1*S(idx)*I(idx)/N + r2*B2*S(idx)*Q(idx)/N -a2*Q(idx));
    I(idx+1) = I(idx) + h*(a1*E(idx) + a1*Q(idx) - y*I(idx));
    R(idx+1) = R(idx) + h*(y*I(idx));
end
%-------------------------------------------------------------------------
%-----------------------------------------Chapter 4 coding----------------
clear;clc;

%-------------------------------------------------------------------------
%-------------------------------------------------------------------------
beta=1.9;
a1=0.3;
a2=0.2;
```

```
gamma1=0.01;
gamma2=0.01;
gamma3=0.05;
mu=0.05;
tau=14;
N=1;
d1=0.04;
d2=0.02;

ddeSEIQR = @(t,y,Z) [ N-beta*y(1)*y(3)/N-mu*y(1);
    (beta*y(1)*y(3)/N)-beta*exp(-mu*tau)*Z(1,1)*Z(3,1)/N-a1*y(2)-gamma1*y(2)-mu*y(2);
    beta*exp(-mu*tau)*Z(1,1)*Z(3,1)/N-a2*y(3)-gamma2*y(3)-d1*y(3)-mu*y(3);
    a1*y(2)+a2*y(3)-d2*y(4)-gamma3*y(4)-mu*y(4);
    gamma1*y(2)+gamma2*y(3)+gamma3*y(4)-mu*y(5)];



sol = dde23(ddeSEIQR,[14, 1],[0.999 0 0.001 0 0],[0, 200]);%dde23(@...,tau,history,
    tspan);

figure;
plot(sol.x,sol.y(1,:) )
hold on
plot(sol.x,sol.y(2,:),'-.' )
hold on
plot(sol.x,sol.y(3,:),'--')
hold on
plot(sol.x,sol.y(4,:))
hold on
plot(sol.x,sol.y(5,:))
hold off

title('Equilibrium points for SEIQR Model');
xlabel('time t');
ylabel('solution y');
legend('S','E','I','Q','R');
```

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