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MATH 202

COVID-19 dynamics with

SEIR model

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Abstract:

A novel corona-virus has hit Chinese city, Wuhan, in December 2019 and subsequently hit major cities around the world resulting in increasing the number of mortalities. As a result, we want to present a model to predict the virus's growth rate worldwide. Such a model is very important to predict the approximate time when the peak of the epidemic is reached, and to study the way that the asymptomatic infections affect the spread of the virus. In addition, examining the relative effect of factors that govern the spread of the virus can help communities better prepare for an outbreak. By obtaining the number of positive tested cases for COVID-19 alongside with the time series from December 2019 in china until now, obtaining the cases in Italy from the moment that the virus widely spread till now, or using the USA statistics, and hence we can predict the spread of the virus around the world by analyzing this data to know exactly the behavior of this epidemic. We are trying to model the progression of the disease through a set of population using SEIR model which leads to a system of differential equations.

Introduction:

The first reported case in the COVID-19 outbreak was reported in the Chinese city, Wuhan, on 31 December 2019. Thailand was the second place that has the first case outside China on 13 January 2020. After that, COVID-19 spread to more than 50 other countries, so that WHO declares this epidemic as a Public Health Emergency of International Concern (PHEIC) on 30 January 2020. There are over 2,393,349 cases of confirmed COVID-19 worldwide as of 19 April. Typically, a rising infectious disease involves fast spreading, threaten the health of large numbers of people, and subsequently requires prompt action to prevent the disease at the community level. COVID-19 is caused by a new type of coronavirus which was previously named 2019-nCoV by the World Health Organization (WHO). This new virus is the seventh member of the coronavirus family, together with MERSnCoV and SARS-nCoV, that have the ability to spread among humans. The symptoms of the infection include fever, cough, shortness of breath, and diarrhea. In more severe cases, COVID-19 can cause pneumonia and even death. The incubation period of hosting COVID-19 can last for around 15 days. During the period of latent infection, the disease may still be infectious. The virus can spread from person to person through respiratory droplets and close contact. In our project, we are trying to model the progression of the disease through a set of population using a modified SEIR

model as SIR model will not be sufficient as the corona virus has a long incubation time which means that a person could be infected without showing any of the virus' symptoms and hence we will add more categories into the model's compartments (S: Susceptible, E: Exposed, I: Infected, R: Recovered). We will divide the susceptible category into two compartments; one for the normal population and other for the severe case which includes old people, and people who suffer from having weak immune systems or other diseases (C: Cumulative cases (both reported and not reported)) in order to add more accuracy to our model.

Problem definition:

Coronaviruses are a large family of viruses which are known to cause respiratory infections ranging in dangerous. The most discovered one (in December 2019 in China) and the one who concern us is COVID-19. According to World Health Organization (WHO), about 1 in every 5 people who catch it need hospital care. Because of the financial and the human loss, the world is suffering and since mathematics is the language the laws of the universe are written in, we will use mathematical modeling to describe how the virus grows and predict the spread of the virus.

Discussion:

Viruses are as old as humankind. They have been on our planet as old as we or close to it. Throughout the course of history, humans multiplied, increased in numbers, and dispersed throughout the world, new diseases emerged. With the beginning of the agricultural revolution around 10 000 years ago and the establishment of permanent settlements, people began living in closer proximity to one another as well as to animals. This facilitated the spread of bacteria and viruses between humans. An epidemic happens when a disease spreads between large numbers of people in a short period of time. When an epidemic goes global, it is called a pandemic. Around the 3rd century BCE in the Egyptian Empire, first emerged Smallpox. The earliest description that clearly resembles the disease appeared in China in 4th century CE. Smallpox is considered by many the most lethal and devastating viral infection in history and is also the only human disease which have been eradicated by vaccination. English doctor Edward Jenner eventually noticed that individuals who had been sick with the less dangerous disease of cowpox seemed to be immune from getting smallpox. This led to him developing the world's first systematic vaccination program in 1798, saving the lives of tens of thousands of people. Smallpox was found on the body of the ancient Egyptian mummy Ramses V. By

the 18th century. It had become responsible for about 20 per cent of deaths in Europe. Some estimated that the worldwide deaths from Smallpox in the 20th century were more than 300 million as 3 out of every 10 people with the disease died. Around mid-1300s, The Black Death in Europe killed third the total population which were more than 25 million people but it's hard to be sure as there was no systematic way of recording deaths and no censuses to record the initial size of the population. The disease was terrifyingly, wholesale contagious and terrifyingly efficient. People who were health could sleep at night but found dead in the morning. In 1918, The Spanish Flu pandemic infected around 500 million people which were third of the whole planet population at that time. It didn't stop there, it was also deadly as it took the lives of 50 million victims more than the first world war .at the time, there was no effective vaccine to treat the disease which made it worse. In just one year, the average life of expectation in United States fell by a dozen years. In Australia, The Black Death killed around 12,000 people and infected more than 2 million people. Smallpox, Spanish Flue and The Black Death are good examples of how old diseases are, how the suffering of humankind with viruses and the diseases caused by them is as old as humankind, how dangerous they are and how humankind over the course of history have always needed to find a cure for every new virus and

disease. The improvement of living conditions such as clean water, sewerage systems, better food, and personal hygiene methods has significantly reduced the incidence and impact of epidemics. Interventions such as antibiotics, anti-viral drugs and vaccination programs have also done much to prevent the spread of disease, with some diseases, like smallpox, being eradicated altogether. However, Today's highly mobile, interdependent, and interconnected world provides myriad opportunities for the rapid spread of infectious diseases, and radio nuclear and toxic threats. Infectious diseases are now spreading geographically much faster than at any time in history. It is estimated that 2.1 billion airline passengers travelled in 2006. An outbreak or epidemic in any one part of the world is only a few hours away from becoming an imminent threat somewhere else. Even though vaccines are available for many infectious diseases, these diseases still cause suffering and mortality in the world, especially in developing countries. The transmission mechanism from an infective to susceptible is understood for nearly all infectious diseases and the spread of diseases through a chain of infections is known. However, the transmission interactions in a population are very complex so that it is difficult to comprehend the large-scale dynamics of disease spread without the formal structure of a mathematical model. An epidemiological model uses a

microscopic description (the role of an infectious individual) to predict the macroscopic behavior of disease spread through a population. Since repeatable experiments and accurate data are usually not available in epidemiology, mathematical models and computer simulations can be used to perform needed theoretical experiments. Calculations can easily be done for variety of parameter values and data sets. Mathematical models have both limitations and capabilities that must recognized. Sometimes questions cannot be answered by using epidemiological models, but sometimes the modeler can find the right combination of available data, an interesting question, and a mathematical model which can lead to the answer. Comparisons can lead to a better understanding of the processes of disease spread. Modeling can often be used to compare different diseases in the same population, the same disease in different populations, or the same disease at different times. Epidemiological models are useful in comparing the effects of prevention or control procedures. Hethcote and Yorke (1984) use models to compare gonorrhea control procedures such as screening, rescreening, tracing infectors, tracing infectees, post-treatment vaccination and general vaccination. Communicable disease models are often the only practical approach to answering questions about which prevention or control procedure is most effective. Quantitative

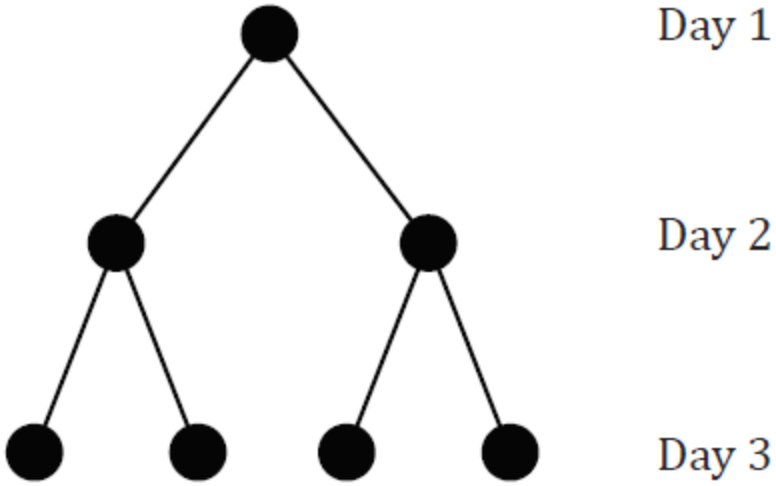
predictions of epidemiological models are always subject to some uncertainty since the models are idealized and the parameter values can only be estimated. However, predictions of the relative merits of several control methods are often robust in the sense that the same conclusions hold over a broad range of parameter values and a variety

of models. Strategies for rubella vaccination are compared using a cost benefit analyses in the article on rubella by Hethcote (1989) in this volume. Optimal strategies for vaccination can be found theoretically by using modeling. Longini, Ackerman and Elveback (1978) use an epidemic model to decide which age groups should be vaccinated first to minimize cost or deaths in an influenza epidemic. Hethcote (1988) uses a modeling approach to estimate the optimal age of vaccination for measles. A primary conclusion of this paper is that better data is needed on vaccine efficacy as a function of age to better estimate the optimal age of vaccination. Thus, epidemiological modeling can be used to identify crucial data that needs to be collected. An underrecognized value of epidemiological modeling is that it leads to a clear statement of the assumptions about the biological and sociological mechanisms which influence disease spread. The parameters used in an epidemiological model must have a clear interpretation such as a contact rate or a duration of infection. Models can

be used to assess many quantitative conjectures. The hope is that this powerful combination of biological discoveries, mathematical modelling and public health initiatives can help the world eradicate other diseases.

1-The Basic Exponential Model:

The spread of a contagious disease depends on both the amount of contact between individuals and the chance that an infected person will transmit the disease to someone they meet. If the transmission risk of the disease is 100 per cent and each infectious person meets two other people before they recover, the disease will soon begin to spread very quickly. If recovery takes one day, this situation will result in the number of sick people doubling each day. We can model this situation by the following equation: ($y = 2^{t-1}$) where (y) is the total number of people who are infected, and (t) is the time in days which has elapsed since the initial outbreak. We can too also describe the situation graphically:



Such a scenario, where the number of infections multiplies by a constant factor each day,

is called exponential growth. It can be used as a simplistic model of how an infection could potentially spread through a population.

2-The Reproduction Number R_0:

The basic reproduction number measures how many people an infected individual will transmit the disease to before they recover, and it is given the symbol R_0. More precisely, it is the number of secondary infections produced by an infected individual in a population that is totally susceptible. This number is important in determining how quickly an infection will spread through a population. For example, if the value of R_0 for measles is 14, then each case of measles would produce 14 new secondary cases. This would spread through the

population much faster than in our previous example, where the value of R_0 was only equal to 2. The basic reproduction number is affected by several factors:

- **The rate of contact between individuals in the host population.**
- **The probability of the infection being transmitted during contact.**
- **The duration of infectiousness.**

The magnitude of R_0 not only indicates the speed of how a disease will spread, but whether it not it will spread at all. If it is the case that

- $R_0 > 1$

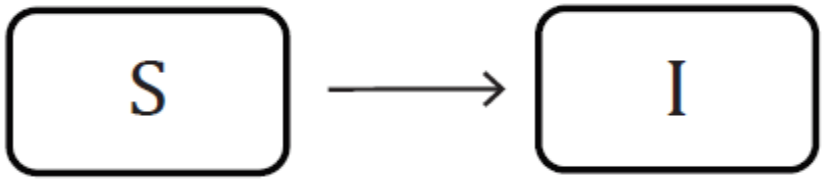
then the infection will spread throughout the population. But if

- $R_0 < 1$

then the infection will not be able to take hold and will eventually die out. Generally, the greater the value of R_0, the harder it is to control an epidemic.

3-The SI model:

A more realistic way of modelling the spread of disease is using the SI model. This is the simplest one among epidemic models. At each time *t* (measured in days), we divide the population into the number who are susceptible, S(t), and the number who are infectious, *I* (*t*). We let *N* be the total population size, which we take to be a constant.



In our model, we assume that every member of the population is either susceptible or infectious, giving us the equation

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

Differentiating both sides with respect to *t*, and remembering that *N* is a constant, we get a relationship between how the numbers of susceptible and infected people are changing over time:

- $\frac{dI}{dt} = -\frac{dS}{dt}$

It often makes sense to study the proportions of infected and susceptible people rather than the raw numbers. So, we define new variables as follows:

- $i(t) = \frac{I}{N}$**, the proportion of the population who are infected**
- $s(t) = \frac{S}{N}$**, the proportion of the population who are susceptible.**

Then we know that *i* + s = 1 or, rearranging, that *s=1-i.* In the SI model we assume that any infected people will continue to spread the disease until the end of the epidemic, that is, they never recover. The rate at which the disease spreads will be proportional to *s* (the more susceptible people there are, the

faster the disease can spread) and also to *i* (the more infected people there are, the faster the disease will spread). This observation leads us to write down our first differential equation:

- **Rate of infection =*β* (proportion of susceptible) x (proportion of infectees)**

  = *β S*(*t*) *i*(*t*), or *β s i*

Here *β* is a constant called the transmission rate and is the average number of people each infectious person spreads the disease to each day. It can be calculated by multiplying the transmission risk with the average number of contacts per day.

We can rewrite this equation solely in terms of *i* :

- $\frac{d\,i}{d\,t} = \beta\,(1 - i)i$

Every person who becomes infected is one less person who is susceptible. In other words, the fraction of susceptible people will go down at the same rate as the fraction of infected people go up. This gives us a second equation:

- $-\frac{d\,i}{d\,t} = \frac{d\,s}{d\,t} = -\beta\,si$

A way of representing this model graphically.

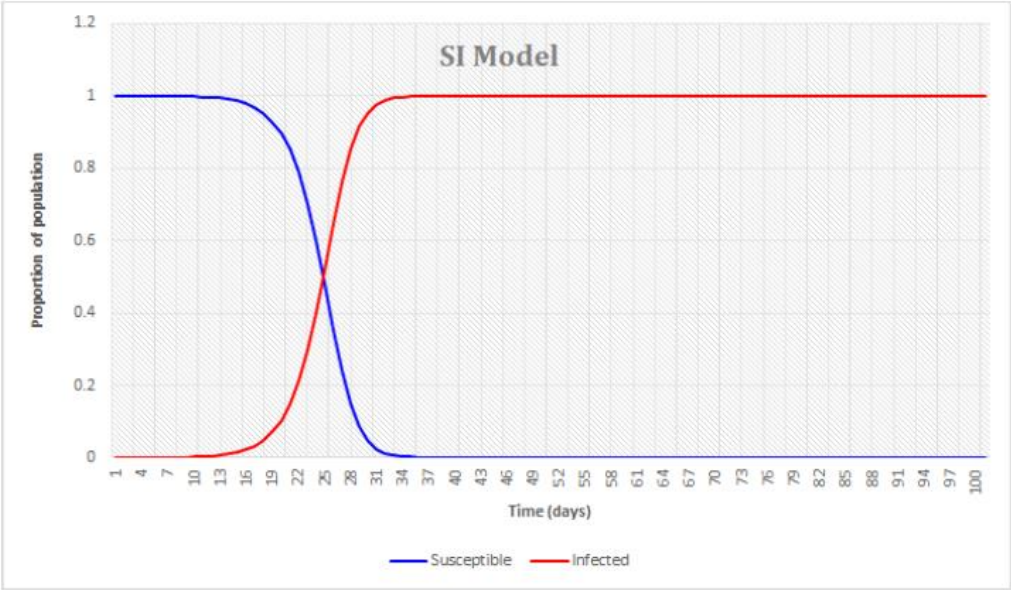


Figure 3: The SI model of the spread of disease, showing the proportion of susceptible (blue) and infected (red) people over 100 days.

4-The SIR model:

The SIR model more accurately represents how an infection would spread through a population because it takes into consideration that some people will recover from the disease and no longer be susceptible. This model assumes that people who recover from the infection become immune and cannot become infected a second time. First, we can investigate how the graphs of susceptible and infectious people might be different if we assume that infectious people recover after a fixed amount of time. In Figure 4 below, we look at the evolution of infection *A*, which has a reproduction number R_0 of 3, an infectious duration of 5 days, and an initially infected proportion of 1 per cent of the population.

The blue line is the proportion of the population who are susceptible, *s*(*t*). The red line is the proportion of the population who are actively infected, *i* (*t*).

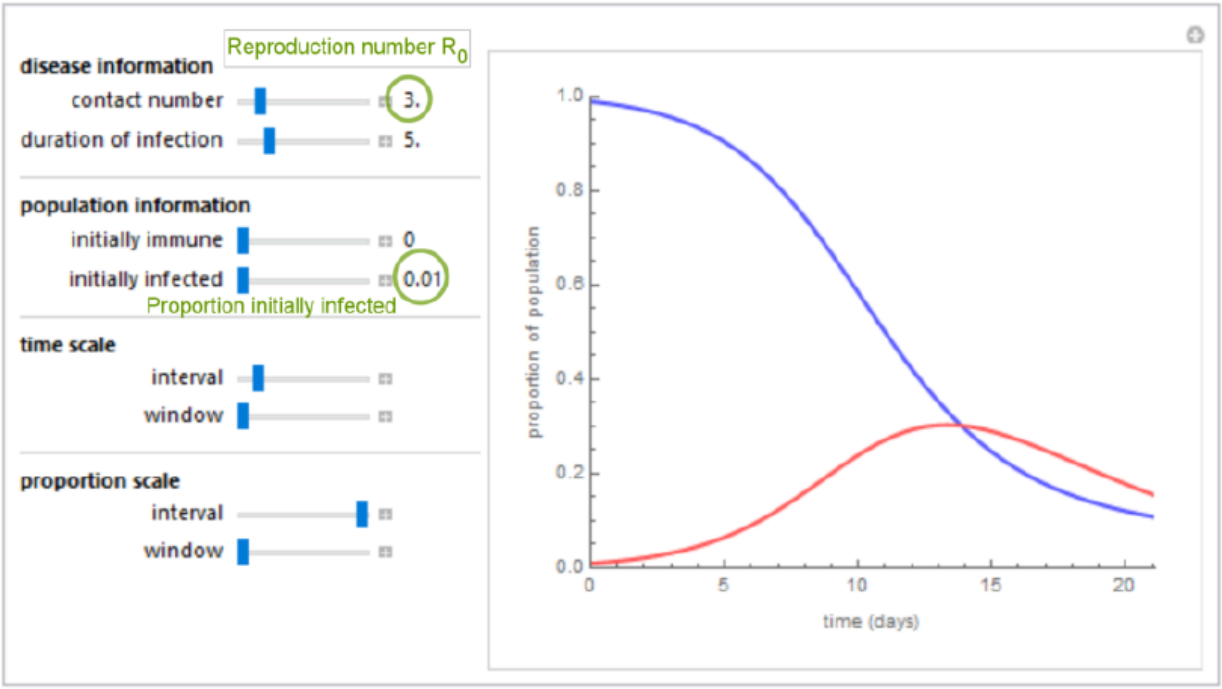


Figure 4: SIR model for infection *A*

In an SIR model, the population is divided into three types:

- **Susceptible (*S*) (not infected),**
- **Infectious (*I*),**
- **Recovered (*R*) (that is, vaccinated or recovered with immunity).**

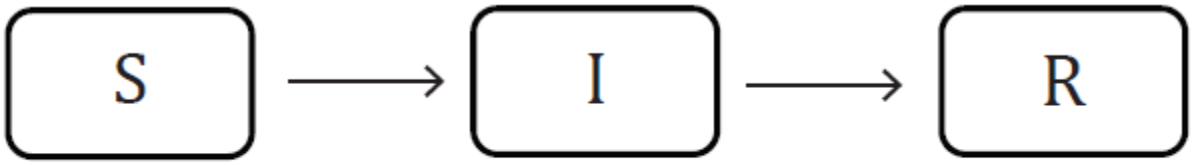


Figure 7: The SIR model of the spread of disease.

We will make three assumptions of how these categories of the population relate to each other:

1- **The number of infected people increases at a rate proportional to both the number of infected and the number of susceptible people. The number of susceptible people decreases at this same rate. The ratio involved is the transmission rate β ,the same as in the SI model.**

2- **The number of recovered people increases at a rate proportional to the number of infected people. The ratio involved is called the recovery rate γ**

3- **A susceptible person who catches the disease becomes infectious immediately. We model each of *S*, *I* and *R* as functions of a time variable *t,* which is measured in days Our variables then become**

- ***S* = *S*(*t*) = number of susceptible individuals at time *t.***
- ***I* = *I* (*t*) = number of infected individuals at time *t.***
- ***R* = *R*(*t*) = number of recovered individuals at time *t*.**

Just as in the SI model, it makes more sense to consider the proportions of each type of individual rather than their actual number. If the total population again has size *N*, the new variables become:

- $s(t)=\frac{S(t)}{N}$ **= the proportion of susceptible individuals at time *t.***

- $i(t) = \frac{I(t)}{N}$ = the proportion of infected individuals at time *t.*
- $-r(t) = \frac{R(t)}{N} =$ the proportion of susceptible individuals at time *t*.

Then we can write the equation $s(t) + i(t) + r(t) = 1$.

Graphing the SIR Model:

The evolution of the equations in the SIR model can be represented graphically. The following graph shows the development of an infection where the average infectious period is ten days and each infected person spreads the infection to one person every two days. At the beginning of the infection, when $t = 0$, the number of susceptible individuals is S = 5000000, while the number of infected individuals is $I = 10$. We can assume that no one has recovered from the infection in its initial stage of outbreak, so to start with we have $R = 0$. The transmission rate β is 0.5 because this is the average number of people an infectious person passes the disease to each day. The recovery rate γ is 0.1 because if each person is infectious for 10 days then we would expect a tenth of the people to recover each day. The recovery rate is always the inverse of the infectious period. The blue line represents the number of susceptible

individuals. The red line represents the number of infected individuals. The yellow line represents the number of recovered individuals.

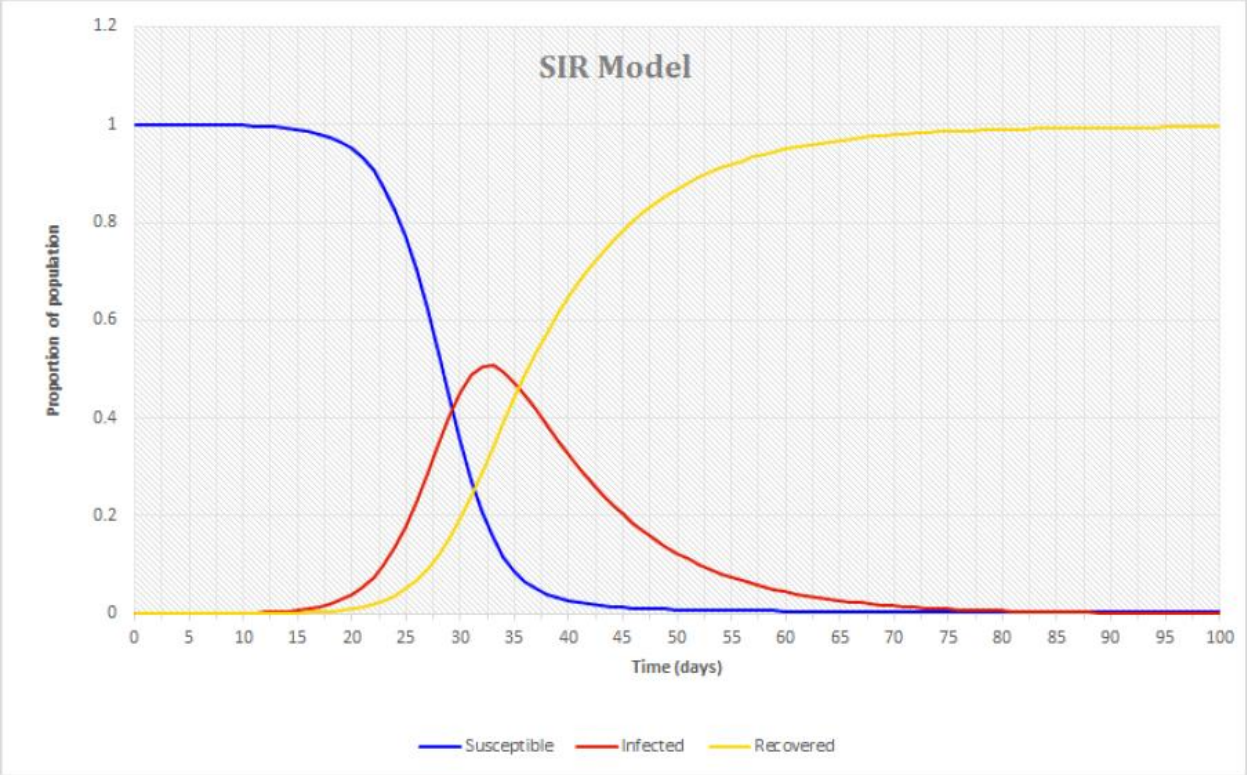


Figure 8: SIR model graph over 100 days with an initial susceptible population of 5 000 000, an initial infected population of 10, an initial recovered population of 0, a transmission rate β of 0.5, and a recovery rate γ of 0.1.

As the proportion of recovered people increases, the proportion of susceptible people decreases, as shown respectively by the yellow and blue lines. The red line represents the trend of infected individuals, and how their numbers change over time. In this example, both the blue and red lines decrease until they hit zero. This means that the entire population has become infected with the disease and moved into the recovered phase. When the blue line does NOT

reach zero, this means the disease has died out before everyone in the population has contracted it. In the SIR model, the basic reproduction number *R*0 can be calculated using the ratio of the transmission rate to the recovery rate:

- $R_0 = \frac{\beta}{\gamma}$

5. The SIS Model:

The model is for diseases for which infection does not confer immunity. It is called an SIS model since individuals return to the susceptible class when they recover from the infection. The compartmental diagram for an SIS model. Naturally occurring births and deaths (vital dynamics) are included, but the behavior of solutions is similar when vital dynamics are not included. The initial value problem (IVP) for this SIS model formulated in terms of class sizes is

$$\begin{aligned} (NS(t))' &= -\lambda SNI + \gamma NI + \mu N - \mu NS \\ (NI(t))' &= \lambda SNI - \gamma NI - \mu NI \\ NS(0) &= NS_0 > 0, \quad NI(0) = NI_0 > 0, \quad NS(t) + NI(t) = N \end{aligned} \tag{4.1}$$

where λ is a positive constant and primes denote derivatives with respect to time *t.* If each equation above is divided by the constant population size *N,* then the IVP in terms of the fractions in the classes is

$$S'(t) = -\lambda IS + \gamma I + \mu - \mu S$$
$$I'(t) = \lambda IS - \gamma I - \mu I$$
$$S(0) = S_0 > 0, \quad I(0) = I_0 > 0, \quad S(t) + I(t) = 1.$$

Since S(t) can be found from I(t) by using S(t) = 1 - I(t), it is sufficient to consider

$$I'(t) = [\lambda - (\gamma + \mu)]I - \lambda I^2$$
$$I(0) = I_0 > 0.$$

Since this is a Bernoulli differential equation, the substitution $y = I^{-1}$ converts into a linear differential equation from which the unique solution is found to be

$$I(t) = \begin{cases} \dfrac{e^{(\gamma+\mu)(\sigma-1)t}}{\sigma[e^{(\gamma+\mu)(\sigma-1)t} - 1]/(\sigma - 1) + 1/I_0} & \text{for} \quad \sigma \neq 1 \\ \dfrac{1}{\lambda t + 1/I_0} & \text{for} \quad \sigma = 1 \end{cases}$$

where *a* is the contact number *λ(γ+μ)*. The theorem below follows from the explicit solution. Theorem 4.1. *The solution* I(t) *of* approaches 1 - *I/a as t* ->∞ *if a*> I *and approaches* 0 *as t* -> ∞ *if a* =< I. This theorem means that for a disease without immunity with any positive initial infective fraction, the infective fraction approaches a constant endemic value if the contact number exceeds I; otherwise, the disease dies out. A threshold result for an SI model is obtained

from Theorem 4.1 by taking the removal rate *y* to be zero in the model. If both the removal rate y and the birth and death rate *μ* are zero, then *σ* = ∞ so that there is no threshold and eventually everyone is infected. This model with γ= *μ* = 0 is the "simple epidemic model". Some authors such as May (1986) use other terminology; he uses the basic reproductive rate R_0 is place of the contact number *σ* and effective reproductive rate $R_0 S$ instead of the replacement number *σ S.* The prevalence is defined as the number or cases of a disease at a given time so that it corresponds to *NI.* Since the incidence is defined to be the number of new cases per unit time, it corresponds to the *λSNI* term in model. At an endemic equilibrium the prevalence is equal to the incidence times the average duration of infection 1/(γ +μ)since the right side of the second equation is zero at an equilibrium. The incidence and the prevalence of some diseases oscillate seasonally in a population. This oscillation seems to be caused by seasonal oscillation in the contact rate λ. For example, the incidence of childhood diseases such as measles and rubella increase each year in the winter when children aggregate in schools. If the contact rate λ changes with time *t,* then the *λ(t).* If λ.(t) is periodic with period *p,* then Hethcote has found the asymptotic behavior of solutions I(t). If the average contact number $\sigma^- = \lambda^-/(\gamma + \mu)$ satisfies *u* =< 1, then I(t) damps in an oscillatory manner to 0 for large *t.*

However, if $\sigma^{-} > 1$, then I(t) approaches an explicit periodic solution for large *t.* Gonorrhea is an example of a disease for which infection docs do not confer immunity.

Methodology:

A mathematical model is formulated to study the change in our state variables. The model can either be generated by tracing all the individuals in our system or by either classifying the community into groups. Despite the fact that tracing all the individuals in society would result in a very accurate model, it would be hard to trace the individuals in addition it would be very hard to compute it. As a result, compartmentalizing our state variables into groups would make tracing them easier and would result in a good approximation to the change occurring in the real world.

A general way in grouping the community would be SIR model, which classify the people into three groups:

1) S: stands for susceptible.
2) I: stands for the Infected.
3) R: stands for either the recovered or death cases.

This model is predictive for infectious diseases that are transmitted from human to human, and where recovery confers lasting resistance. However, in our case, this model would not be sufficient as the corona virus has a long incubation time which means that a person could be infected, however, the virus' symptoms may have not appeared immediately. Moreover, we will divide the susceptible to two compartments; one for the normal population and other for the severe case which includes old people, and people who suffer from having weak immune systems or other diseases.

As a result, our total population will consist of those 5 groups:

1) **Susceptible group (S).**
2) **Cumulative cases (both reported and not reported) (C).**
3) **Exposed group (E).**
4) **Infected group (I).**
5) **Recovered group (who are recovered or dead) (R).**

As a result, we would follow the SEIR model rather than SIR as adopting this model will help to represent the total population into the four mentioned groups above.

- $\frac{d\,s}{d\,t} = -$ B(t)*S*$\frac{I}{N}$ - b*S*$\frac{E}{N}$

- $\frac{d\,e}{d\,t} = b * \frac{S}{N} - B * S * \frac{I}{N} - qE$
- $\frac{dI}{dt} = qE - yI$
- $\frac{dR}{dt} = yI$
- $\frac{dN}{dt} = -bN$
- $\frac{dD}{dt} = dyI - hD$
- $\frac{dC}{dt} = qE$

Where

$B(t)=b(1-a)x(1-D/N)^K$

The transmission rate function, B(t), models the spread of the coronavirus by incorporating the influence of governmental actions, and the decreasing contacts among individuals due to the number of deaths.

The following values for constants are retrieved from a currently research paper that addresses modeling of COVID-19 as we are currently working on the method of getting these constants.

Constants:

1- **b is the transmission rate and equal to {0.5944, 1.68} (day^{-1}).**

2- **Q^{-1} is the mean latent period and equal to 3 days.**

3- **Y^{-1} is the mean infectious period and equal to 5 days.**

4- **d is the proportion of severe cases and equal to 0.2.**

5- **h^{-1} is the mean duration of public reaction and equal to 11.2 days.**

6- **a is Governmental action strength and equal to {0,0.4239,0.8478}.**

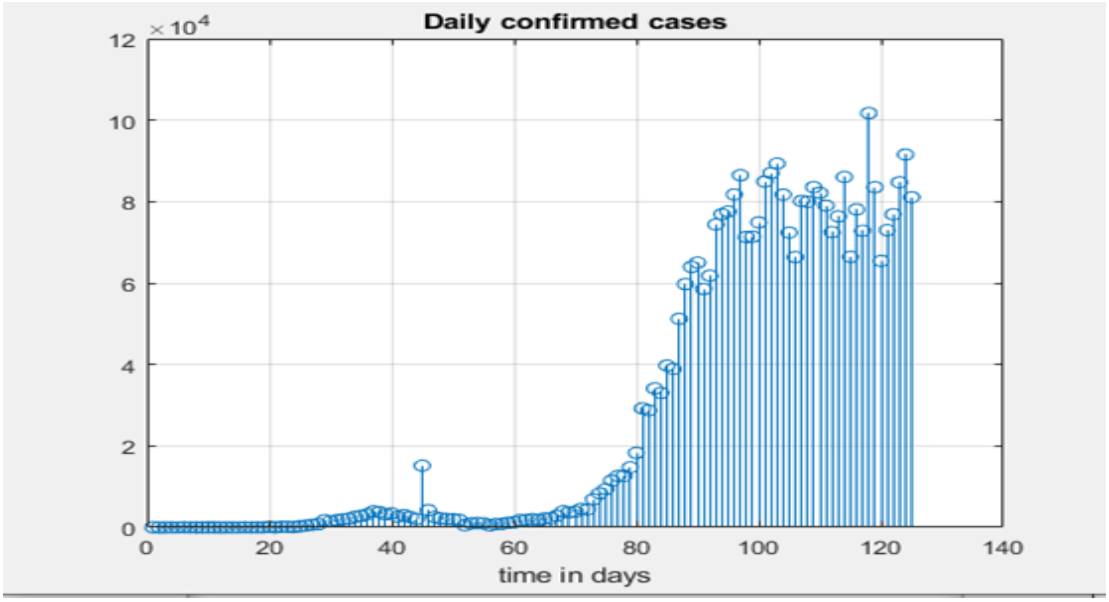
7- **k is intensity of responds and equal to 1117.3.**

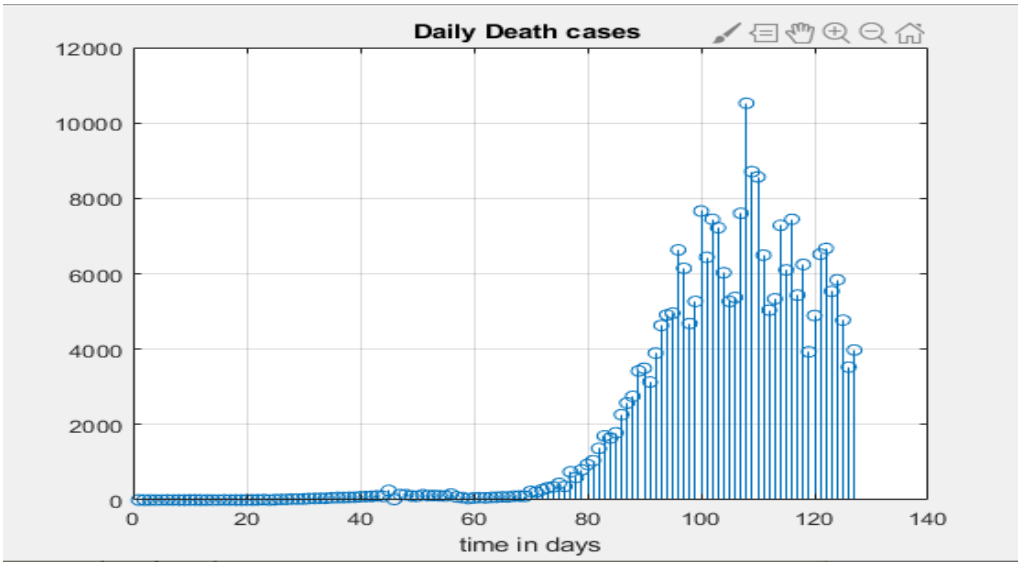
Analysis and Results:

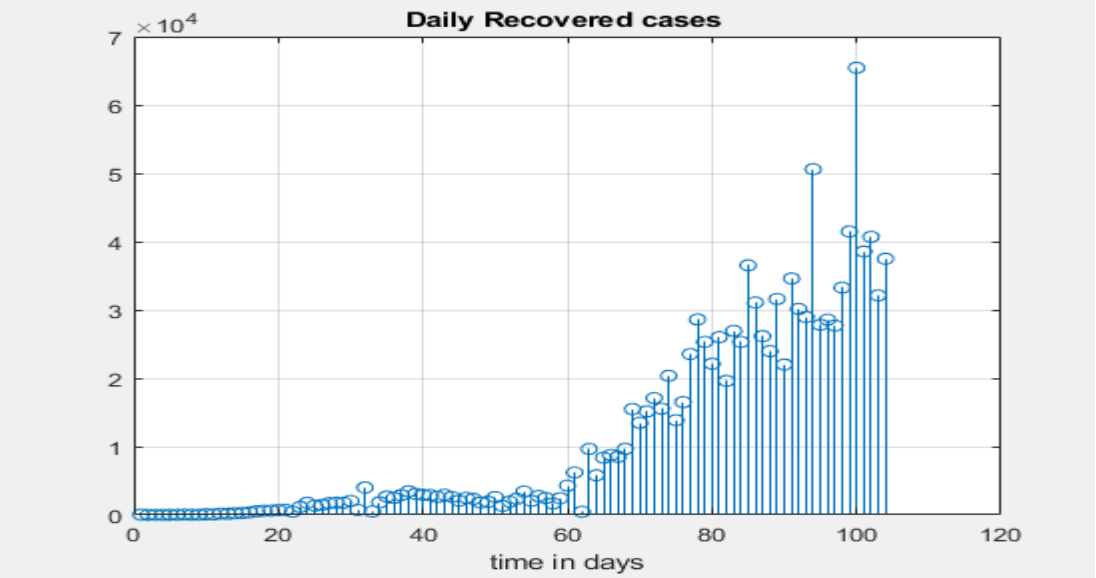
The reviewed literature we have looked for was using Poisson method to get the coefficients as transmission coefficient, mean latent and recovery period by trying to get the number of occurrence of an event and then use the suitable regression model to fit them, However, these models are only time dependent and this lead to ignore the effect of the proportional infected numbers, However, there are some parameters which cannot deal with such ignorance as the transmission ratio which tries to calculate how the virus spread according to the contact ratio, as a result using a Poisson regression to estimate it as time passes may give us an inaccurate result. Concluding that those models may give us deceiving results that need to be changed every once and then to be accurate. As a result, we tried to develop a new model to determine the coefficients used in the differential equations.

The developed model deals with the coefficients as a linear function whose independent variable is the number of the infected people and the dependent variable represents the ratio of the occurrence of an event which may be recovery, death or even transmission.

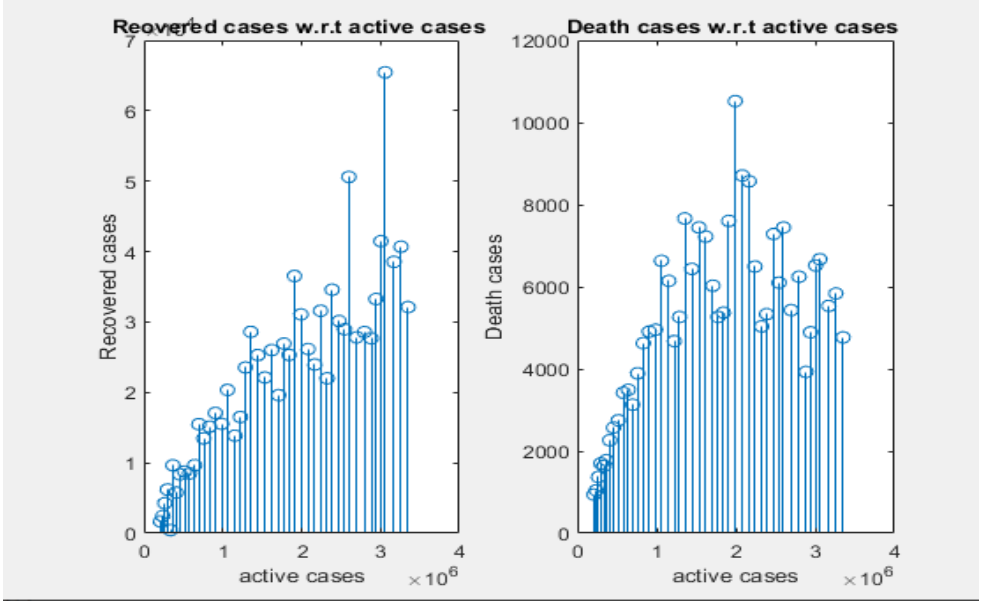
First, having the data of cumulative death cases, we discretize this data by subtracting death cases per day from the day after, so we have a discrete graph representing the discrete data of death cases per day. We have done the same for recovered cases graph and active infected cases graph.





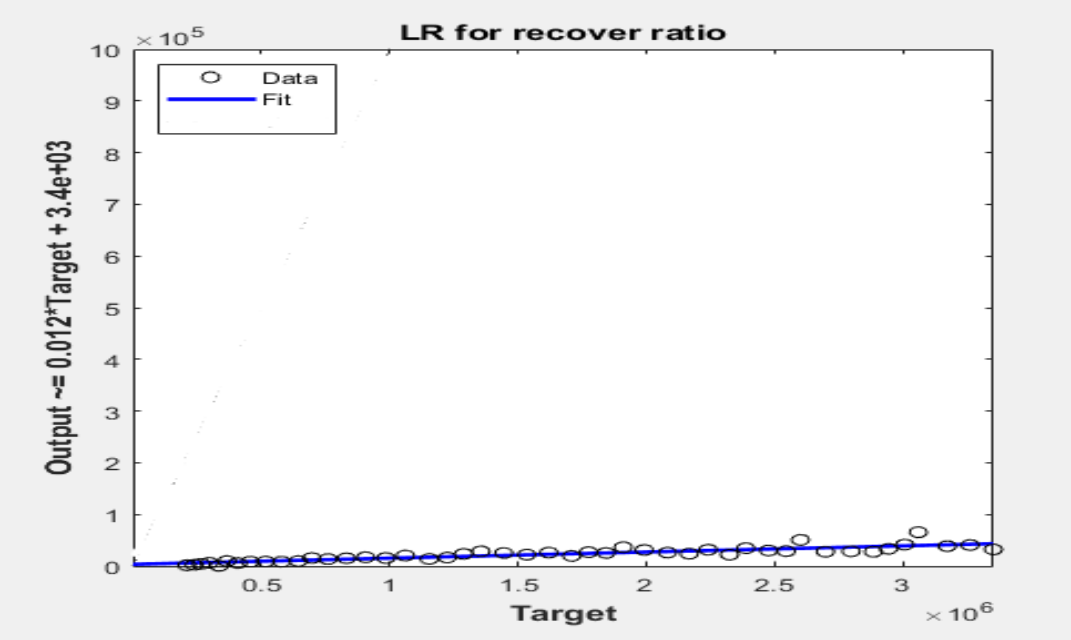


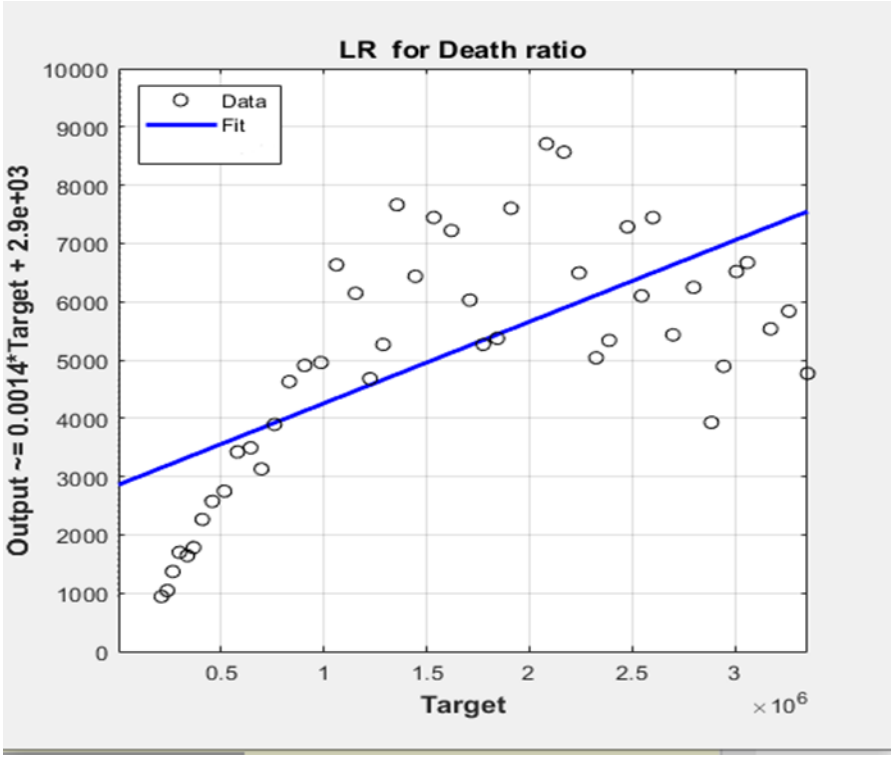
Second, we normalize the date of the two graphs by changing x-axis to make it represent the normalized active infected cases and then plot it with both



normalized Death cases and normalized recovered cases

Third, (1) To calculate the death rate coefficient, we made a regression model having the vector of death cases and the (vector of active infected cases-recovered cases).
The result will be the coefficient of death rate as a function of infected cases.
(2) To calculate the recovery rate coefficient, we made a regression model having the vector of recovered cases and the (vector of active infected cases-death cases).
The result will be the coefficient of recovery rate as a function of infected cases.





Conclusion:

The values determined for the reproduction rate, period and the infectious period are outside of the ranges that normally expected. This is likely due to the way in which the SEIR model is used, that is by collecting data about the number of infected patients and the number of recorded closed cases and opposed to addressing the full population. The two graphs illustrate how effective the policy and the social distancing is important as it affects the initial

conditions for each rate (SEIR). The model shows that the safety guidelines such as wearing of masks, social distancing, frequent wash of hands and cutting down on travel has effect on the susceptible people. This will poses a challenge to the long-term mathematical modeling of the disease.

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