# NUMERICAL MODELS OF COVID-19

by

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### ABSTRACT

BENJAMIN CUNG. Numerical models of COVID-19. (Under the direction of DR. KEVIN MCGOFF)

COVID-19 is an ongoing infectious disease where individuals progress through the following four stages: susceptible (S), exposed (E), infected (I), and recovered (R). The standard mathematical model for the spread of such diseases through a large population is a system of ordinary differential equations, called the SEIR model. However, the qualitative features of the outbreak predicted from the SEIR model do not match with what the actual course of COVID-19 is doing in the U.S. population. In this thesis, we explore several different modifications of the standard SEIR model to determine and observe whether these modifications can recreate the qualitative features of the real world data.

## **DEDICATION**

Dedicated to Mr. John Osborne, my mathematics private tutor throughout elementary to high school, to even my first year of my undergraduate career when I was not at my best from the beginning. Thank you for the 10 years of non-stop teaching, guidance, and your continuous support and belief in me. My affinity for math continuously grew large throughout the years and I would not be pursuing this field of study if it were not for you.

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# LIST OF ABBREVIATIONS

CDC Centers for Disease Control and Prevention

ODE Ordinary differential equations

SEIR Susceptible Exposed Infected Recovered

#### CHAPTER 1: INTRODUCTION

#### 1.1 What is COVID-19?

COVID-19 is an ongoing disease that is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Some possible symptoms of this disease can include but are not limited to shortness of breath or difficulty breathing, chills, headaches, loss of senses, nausea/vomiting, etc. As of April 2021, the CDC has provided the statistics that there are 32.1 million reported cases, 572 thousand reported deaths, and the number of recovered individuals is unknown in the United States. COVID-19 can be passed on to susceptible individuals an infected person comes in contact. An individual can also obtain COVID-19 simply from just touching some contaminated object or surface before coming into contact with their eyes, noses, or mouth. It is important to understand that if an individual infected with this pathogen were to pass this into a population, then the individuals who are susceptible to the disease will become infected as time passes and eventually will be moved to the infected group increasing the rate of transmission [1]. In order to investigate this occurrence, a typical SEIR model is used to help model this disease.

### 1.2 The SEIR Model

Before using this model to help analyze the pathogen rate behavior, it is important to understand what an SEIR model is, and what a typical SEIR model is going to encapsulate. An SEIR model is a mathematical model highlighting the individuals of a population progressing through the four compartments of an infectious disease. This model is a system of four ordinary differential equations consisting of the following variables:

- S the number of susceptible individuals (capable of contracting the disease)
- E the number of exposed individuals (contracted the virus but not infectious)
- I the number of infected individuals (capable of transmitting disease)
- R the number of recovered individuals (removed from the population).

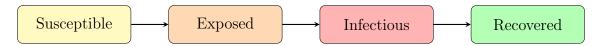


Figure 1.1: Diagram of SEIR compartment stages

Figure 1.1 details the sketch of the stages of progression of an infectious disease. With this diagram, we need the rates of transition of each stage. These rates are determined by the following system of ODEs:

$$\frac{dS}{dt} = -\frac{\beta SI}{N} \tag{1.1}$$

$$\frac{dE}{dt} = \frac{\beta SI}{N} - \mu E \tag{1.2}$$

$$\frac{dI}{dt} = \mu E - \gamma I \tag{1.3}$$

$$\frac{dR}{dt} = \gamma I. \tag{1.4}$$

The parameters are:

- $\bullet$  N the number of individuals in the population
- $\bullet$   $\mu$  the rate of progression of infection from exposed
- $\bullet$   $\gamma$  the rate at which individuals recover from infection
- $\beta$  captures the infection rate of the pathogen.

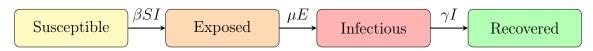


Figure 1.2: SEIR diagram with equations

Observe from the parameters listed above that N is fixed implying that we have no control over the number of people in the population. Also both  $\mu$  and  $\gamma$  are both parameters with no control as these two parameters are determined by biology. On the other hand, the parameter  $\beta$  may be significantly altered by human behavior, and we will focus our attention on this parameter. There are two factors that this parameter encapsulates. It may be viewed as a product of the rate of interaction in the population and the rate of transmission per interaction [2]. These factors are clearly determined by the average behavior of the population. The rate of interaction is generally determined on how often do people go out and interact with people and the rate of transmission per interaction is determined from individuals following rules such as social distancing or wearing their mask. Next we compare a typical SEIR model output with the real world statistics (see Figure 1.3).

## 1.3 A Comparison of Between Real World Statistics and an SEIR Model

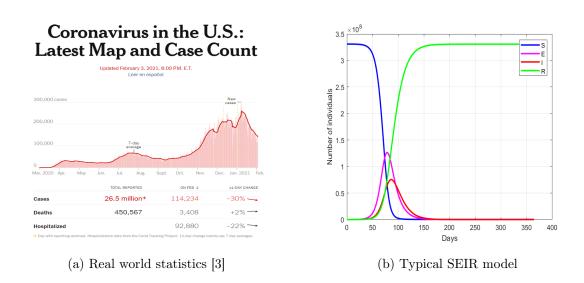


Figure 1.3: Side by side comparison

Figure 1.3a describes the number of reported cases spanning from March 2020 to February 2021. On the other hand, Figure 1.3b is a typical SEIR model using our choices of input for the parameters after one run of the ODEs. The parameters used for this figure are as follows:

- $N = 3.31 * 10^8$  (total population from U.S.)
- $\mu = 1/14$  (average incubation period 2 weeks)
- $\beta = 0.7$  (population choice)
- $E_0 = 1,000$  (initial number of exposed)
- $I_0 = 10,000$  (initial number of infected)
- T = 365 (span of 1 year)

So with our two graphs provided, here is an observation that we want to pay attention to. Notice that from the real world statistics graph that the curve produces multiple peaks. On the other hand, the SEIR model produces only one peak from both the exposed and the infected curves. It is also important to note that no matter what choice of input is used for the parameters, the model will always produce one peak despite the number of simulations tested. The takeaway from this observation is that the qualitative data from both figures do not match. So the following question, which helps motivate the analysis of this research, is what are some mechanisms that could possibly explain the qualitative behavior observed in the real world data?

In order to approach this question, our goal is to make four different modifications to the standard SEIR model and then analyze whether these modifications produce solutions that are qualitatively similar to the trends observed in the real world data. The four modifications that will be used in the following chapters are reactive  $\beta$ , reactive  $\beta$  with delay, fatigue, and policy intervention.

Reactive  $\beta$  involves making the transmission rate  $\beta$  dependent on people's reactions to the current level of infection of the population. In this modified model, if the current level of infection is high, then the population will take more precautions, resulting in a lower  $\beta$ . On the other hand, when the current level of infection is low, the population will relax some precautions, resulting in a higher  $\beta$ . The next modification we will discuss is reactive  $\beta$  with delay.

Reactive  $\beta$  with delay is the rate of change of pathogen transmission with the rate being dependent on people's reactions to level of infection from some number of days ago. People will get tested for COVID-19 to determine whether they are either infectious or not. However, the data to keep count for the number of infected needs to be recorded and the CDC will post the statistics to keep the tallies official for the public to view. The next modification we will discuss is fatigue.

Fatigue is defined as exhaustion that is caused from some physical or mental exertion. This is an important factor to implement to the SEIR model mainly due to individuals taking risks over time. Examples of fatigue may include not being able to see friends physically, or not being able to travel outside of homes/dorms (going to parties, friends' houses, vacation/traveling). When people do end up feeling fatigue, they get tired of being cautious. The last modification we will discuss is policy intervention.

Policy interventions are government actions/protocols or safety regulations designed specifically to help reduce or lower the rate of pathogen transmission. Some examples of policy interventions include a face mask/covering requirement, issuing a mandated curfew, the 6 feet social distancing policy, and opening small/large businesses. This is important to consider for our SEIR model as either lifting or issuing these policies affects the behavior of  $\beta$ . These factors will be added to the SEIR model separately rather than combined as we want to investigate the behavior of  $\beta$  using the specific modification being implemented to the model. For each modifica-

tion, we want to identify reasonable functions for  $\beta$ , run numerical simulations of the corresponding ODEs (over a range of parameters), and then analyze the solutions to determine whether they exhibit features witnessed by the real data.

### CHAPTER 2: REACTIVE $\beta$

### 2.1 Introduction to Reactive $\beta$ Model

The first modification that is going to be implemented and later analyzed into the SEIR model is reactive  $\beta$ . Recall that reactive  $\beta$  is determined by people's reactions towards the current infection level. The rate  $\beta$  is determined by people's behavior towards the level of infection, but also the biology factors into this parameter. Before including this modification into the model, we need to first choose a reasonable function of  $\beta$  to use that will help capture this factor.

## 2.2 Reactive $\beta$ Function

Going back to our definition of reactive  $\beta$ , the rate is dependent on people's behavior towards the level of infection. For example, if the outbreak has started without any awareness from individuals,  $\beta$  starts off as a high constant. Then when the individuals pay attention to the level of infection, they will take action by being cautious of the disease and follow various safety protocols to reduce the risk of the infection spreading. Hence, the rate of  $\beta$  will decrease. So our possible function to choose here is a monotonically decreasing function. Figure 2.1 listed on the next page displays a visual interpretation on what the modification is describing. As  $\beta$  is some large constant, individuals will react to that infection level which will steadily decrease as the number of infected individuals increases. Thus  $\beta$  will end up being a small constant but never 0 as this study is examined from a real-world perspective.

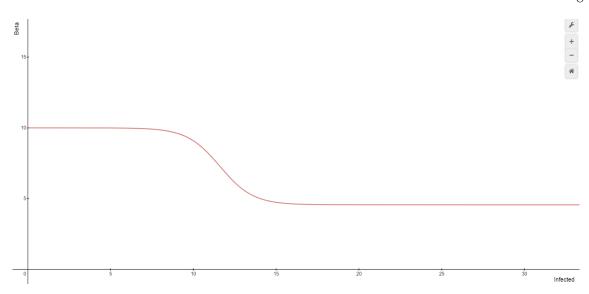


Figure 2.1: Graphical example of reactive  $\beta$ 

We choose the following parameterized function in our reactive  $\beta$  model:

$$\beta(I) = \beta_{high} \left( \frac{e^{-S(I-I_c)}}{1 + e^{-S(I-I_c)}} \right) + \beta_{low} \left( 1 - \frac{e^{-S(I-I_c)}}{1 + e^{-S(I-I_c)}} \right)$$
(2.1)

Here, the variable I is defined as the number of infected individuals and the parameters from Equation 2.1 are defined as follows:

- $\beta_{high}$  high value of  $\beta$  (capturing risky behavior whenever the number of infections is low)
- $\beta_{low}$  low value of  $\beta$  (capturing more cautious behavior whenever the number of infections is high)
- $\bullet$   $I_c$  critical number of infected
- $\bullet$  S the slope at the critical number.

With our  $\beta$  function and the parameters defined, we can now conduct the analysis. The next section of this chapter will be split into four subsections as we will conduct simulations modifying the four parameters listed above.

# 2.3 The Analysis of Reactive $\beta$

The following parameter values are defined as follow:

- $I_c = 2,000,000$
- $\beta_{high} = 1$
- $\beta_{low} = 0.002$
- S = 0.0001.

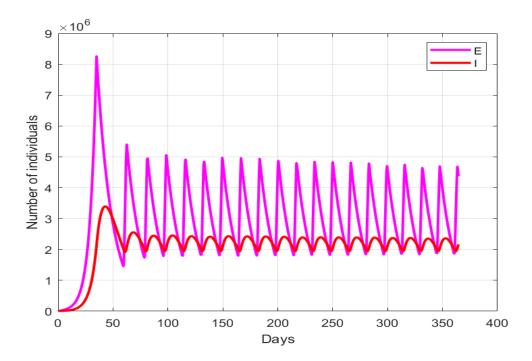


Figure 2.2: Model with selected parameters

Figure 2.2 displays the model with the parameters listed above. With these values, we see that the model produces oscillations. We see that when the number of infected individuals is high enough, then people will react, change their behavior, and then the number will decrease. When the number of infected individuals are low enough, then people will react and take risk which will increase the number. Now that we know what the model looks like with our selected values, in the following subsections, we perform an analysis of the role of each parameter in this model.

## 2.3.1 Critical Number of Infected Analysis

We will look at different models with different choices of the critical number. We performed seven simulations, each with a different choice of parameter  $I_c$ . Our choices were  $I_c = k \cdot 10^6$  where k = 1, ..., 7. We will also refer to our simulations as trials throughout this thesis.

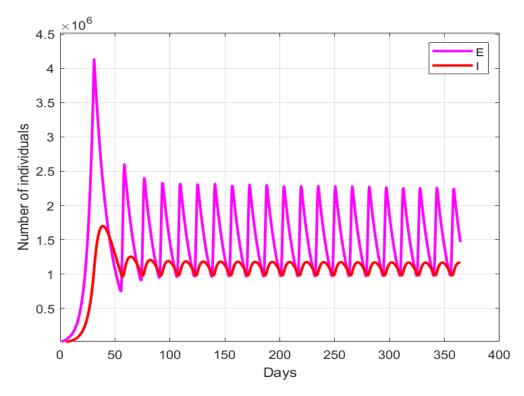


Figure 2.3: Trial #1:  $I_c = 1,000,000$ 

We observe that there are multiple peaks implying an oscillation appears. This is simply because individuals are reacting fast to infection level early on since the  $\beta_{high}$  value is large. The crucial point to understand here is that our  $I_c$  acts as a baseline, where as time passes, when the number of infected is less than this critical number, the majority of the population is behaving according to the  $\beta_{high}$  value and when the number of infected is greater than this critical number, the majority of the population is behaving according to the  $\beta_{low}$  value. We see that once the infected curve reaches the critical number, then these oscillations appear due to the  $\beta_{low}$  value as people are

reacting to the infection level. Also, the oscillation we see serves as an average since our function from Equation 2.1 contains a "weight" function such that:

$$0 \le \frac{e^{-S(I-I_c)}}{1+e^{-S(I-I_c)}} \le 1. \tag{2.2}$$

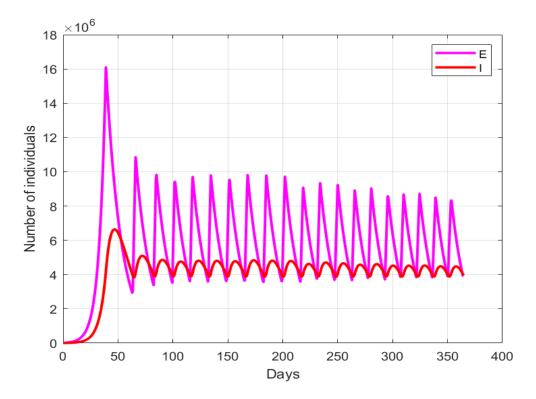


Figure 2.4: Trial #4:  $I_c = 4,000,000$ 

In Figure 2.4, the  $I_c$  value is 4,000,000 and we can see that the critical number input here is the baseline for this model. Of course, with a higher input from our previous simulation, the number of both infected and exposed rises and the behaviors of the oscillations are due to the  $\beta_{low}$  value. Similarly, before the infected curve reaches the baseline, the rise of infected is dependent on the  $\beta_{high}$  value.

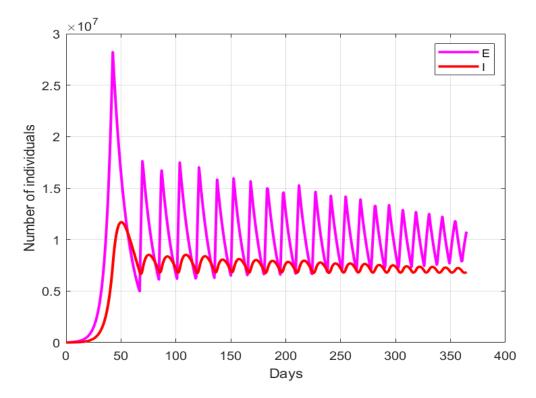


Figure 2.5: Trial #7:  $I_c = 7,000,000$ 

Finally in Figure 2.5, our model is displayed with the  $I_c$  value being 7,000,000 performing the same behaviors with the baseline of the critical number and our  $\beta_{low}$  and  $\beta_{high}$  just as the previous figures. However, notice that for this figure, the magnitude of these oscillations is decreasing in size. In other words, the oscillation is dampening. This is caused by the number of individuals who were part of the susceptible group transitioning into the exposed state and eventually moving to the infected state. The takeaway from this investigation is that the higher number of susceptible individuals, the larger the peak, and the smaller the number of susceptible, the smaller the peak.

2.3.2 
$$\beta_{high}$$
 Analysis

Next, all the parameters except for  $\beta_{high}$  will be fixed as this section will focus on our control for the  $\beta_{high}$  value. We performed ten simulations, each with a different choice of parameter  $\beta_{high}$ . Our choices were  $\beta_{high} = 0.2k$  where k = 1, ..., 10.

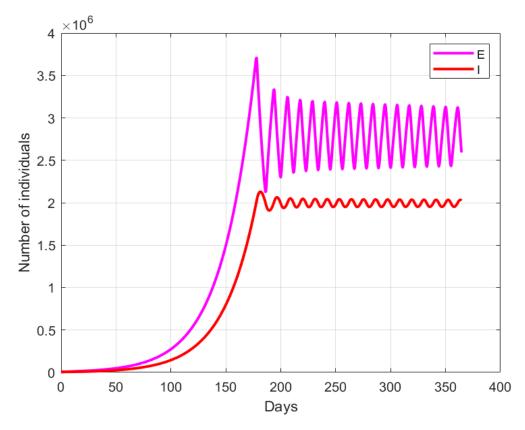


Figure 2.6: Trial #1:  $\beta_{high} = 0.2$ 

In Figure 2.6, our parameter input for  $\beta_{high}$  is 0.2. This means that individuals are not taking as much risks and remaining highly cautious. As we see from the figure, it takes approximately almost 180 days to reach the critical number (fixed  $I_c = 2,000,000$ ) and then eventually the oscillations appear which is dependent upon the  $\beta_{low}$  value (fixed  $\beta_{low} = 0.002$ ).

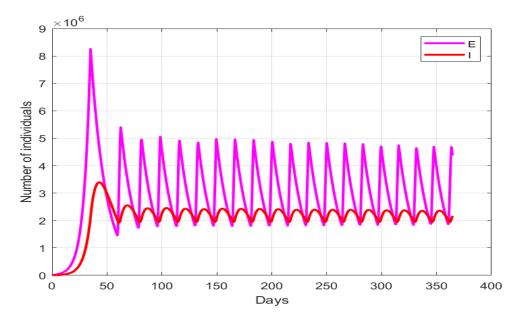


Figure 2.7: Trial #5:  $\beta_{high} = 1$ 

Next in Figure 2.7, the  $\beta_{high}$  value is 1 and we see that the infected curve has reached the critical number of infected faster than from the previously displayed simulation. Additionally, the size of these oscillations increases which is directly related to the  $\beta_{high}$  value.

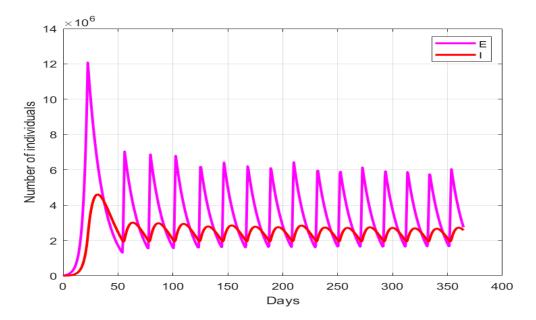


Figure 2.8: Trial #10:  $\beta_{high} = 2$ 

On the final trial, Figure 2.8 produces this model when the  $\beta_{high}$  is 2. The infected curve has reached the critical number even faster and of course, the size of the peaks has increased. In general, if the  $\beta_{high}$  value is high, then the curve reaches the  $I_c$  value fast and if the  $\beta_{high}$  value is low, then the curve reaches the  $I_c$  value slowly. Additionally, throughout these 3 figures, we see that throughout all these oscillations, there is a small window frame where the infected is just below the critical number for a short amount of time. As mentioned from before, the weight function from Equation 2.2 serves as an average function. So both our  $\beta_{high}$  and  $\beta_{low}$  values create this weighted average so that the oscillations stay just in between our input of the critical number of infected.

## 2.3.3 $\beta_{low}$ Analysis

In this subsection, we will now analyze the  $\beta_{low}$  parameter being manipulated to the SEIR model with the other parameters being fixed. We performed twenty simulations, each with a different choice of parameter  $\beta_{low}$ . Our choices were  $\beta_{low} = 0.01k$  where  $k = 1, \ldots, 20$ .

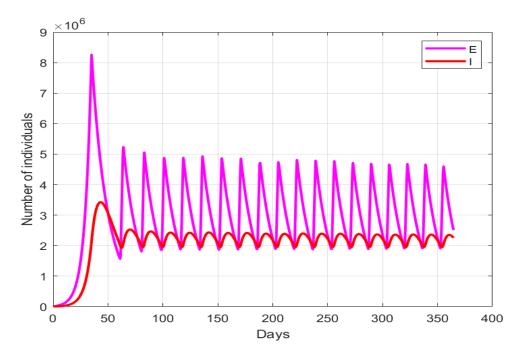


Figure 2.9: Trial #1:  $\beta_{low} = 0.01$ 

In Figure 2.9, our parameter choice for  $\beta_{low}$  is 0.01 with the  $\beta_{high}$  value being 1 which is fixed for all simulations below. As seen here, the outbreak occurs quickly once reaching the critical number of infected (fixed  $I_c = 2,000,000$ ) and oscillations appear due to  $\beta_{low}$  and  $\beta_{high}$  acting as an average towards the baseline.

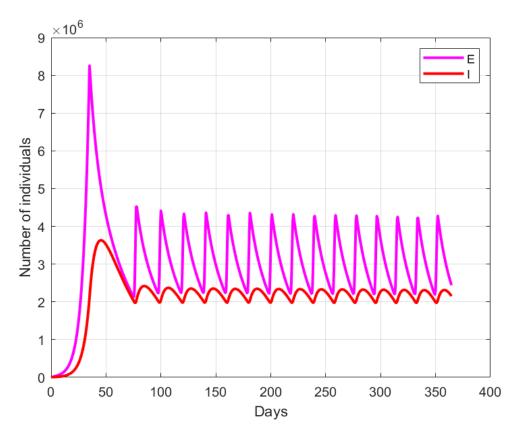


Figure 2.10: Trial #5:  $\beta_{low} = 0.05$ 

Next in Figure 2.10, the input used here for  $\beta_{low}$  is 0.05. We see here that the behavior is a little similar to that in the model from Figure 2.8. Notice that the magnitude of the oscillation has decreased. Additionally, we see that both the exposed and the infected curve never intersect each other aside from reaching the second peak of infected mainly due to both  $\beta_{low}$  and  $\beta_{high}$  affecting this shape.

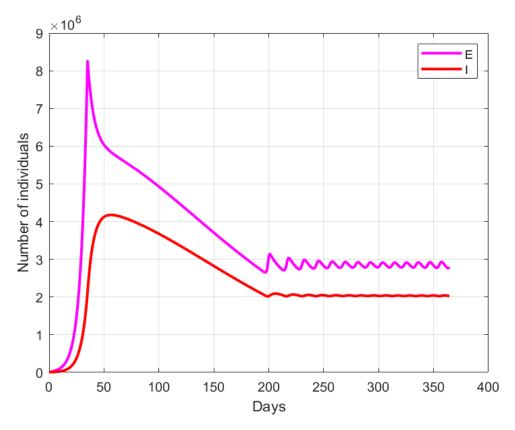


Figure 2.11: Trial #10:  $\beta_{low} = 0.1$ 

In Figure 2.11, the  $\beta_{low}$  value is 0.1. We see that there is an exponential rise on the exposed curve as the infected curve reaches the critical number. After the first peak of the infected curve, there is a slow decay which does not reach to the critical baseline until the 200<sup>th</sup> day. This, of course, is due to the  $\beta_{low}$  value as k increases. Additionally, the size of the oscillation is minuscule as it averages itself on the baseline.

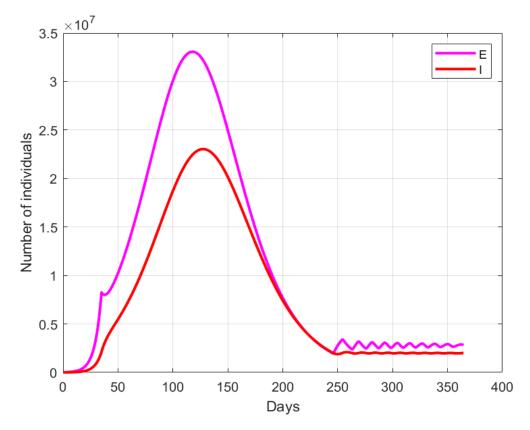


Figure 2.12: Trial #20:  $\beta_{low} = 0.2$ 

Lastly, in Figure 2.12, the  $\beta_{low}$  value for this simulation is 0.2. What is interesting to observe here is that in the early stages of the number of exposed, there is a cusp that exists around the 35<sup>th</sup> day. Through the small window of time, the exposed curve decreases and then increases fast. As we see for the infected curve, the number of infected cases continues to rise. This value of  $\beta_{low}$  is high enough to produce an outbreak on its own, which is why we see the level of infections continues to rise even after the system passes  $I_c$ . The cusp is likely due to the least number of susceptible transitioning to the exposed as there was already a substantially large number of susceptible already moving to exposed during an earlier stage throughout the year. We see that if  $\beta_{low}$  is behaving above the critical number baseline, there will exist a rise in the system and then a decay if  $\beta_{low}$  is under the critical number. Additionally, if  $\beta_{low} = \beta_{high}$ , then the outbreak continues.

# 2.3.4 Slope Analysis

For the final parameter analysis of reactive  $\beta$ , the slope will be in our control while the other three parameters are fixed. We performed 21 simulations, each with a different choice of parameter S. Our choices were  $S = 10^{-6}(9+k)$  where k = 1, ..., 21.

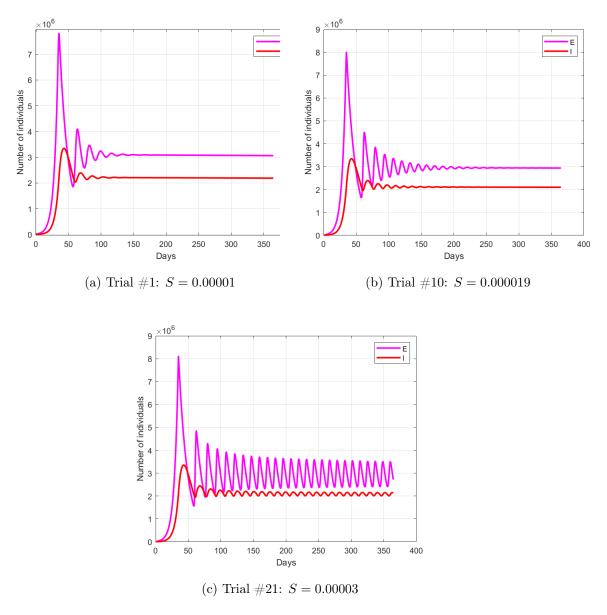


Figure 2.13: 3 different simulations with different slope values

In Figure 2.13, we have three different trials with different slope values listed from the previous page. If we inspect each sub-figures carefully, we observe that a small slope value (Figure 2.13(a)) will produce dampening oscillations. It is also worth mentioning that because that the size of these oscillations is minuscule, individuals are not as reactive towards the level of infection as if there is some fast switch between our fixed values of  $\beta_{low}$  and  $\beta_{high}$ . Increasing in each step size, Figure 2.13(b) and Figure 2.13(c) shows that individuals are reacting rapidly towards the infection level and trying to average itself on the baseline of the fixed critical number.

#### CHAPTER 3: REACTIVE $\beta$ WITH DELAY

### 3.1 Introduction to Reactive $\beta$ with Delay Model

The next modification to be implemented is reactive  $\beta$  with delay. As the name implies, it is referring to the same definition of reactive  $\beta$  but adding delay to this factor. Recall that adding delay to reactive  $\beta$  means we are looking at the number of infections from a number of days ago. This is an important qualitative feature to model into the SEIR model if we can observe the trends occurring in the real-world data in Figure 1.3(a).

### 3.2 Reactive $\beta$ with Delay Function

With our definition established, next comes the function construction for this modification. Since this factor is still using reactive  $\beta$ , we can still use the same function from Equation 2.1. However, we are now adding the delay factor to the reactive  $\beta$  function, so we need to be careful when manipulating our equation from Section 2.2. We are still modeling population level infections to infected levels but in this section, the reactions are delayed. Let us consider adding a parameter specifically for delay and define it as  $\delta$  such that  $\delta > 0$ . We can define a new reactive  $\beta$  function as follow:

$$\beta(I(t-\delta)) = \beta_{high} \left( \frac{e^{-S(I(t-\delta)-I_c)}}{1 + e^{-S(I(t-\delta)-I_c)}} \right) + \beta_{low} \left( 1 - \frac{e^{-S(I(t-\delta)-I_c)}}{1 + e^{-S(I(t-\delta)-I_c)}} \right), \quad (3.1)$$

with t being the current time. With our function defined, we will now analyze several simulations where the parameter in our control will be  $\delta$ .

## 3.3 The Analysis of Reactive $\beta$ with Delay

In this section, we will analyse the delay parameter that is added to our reactive  $\beta$  function. We performed 15 simulations, each with a different choice of parameter  $\delta$ . Our choices were  $\delta = 0, \dots, 14$ . For the following parameters, they will all be fixed except for  $\delta$  as follows:

- $I_c = 2,000,000$
- $\beta_{high} = 2$
- $\beta_{low} = 0.02$
- S = 0.0001.

We will be analyze Trials #1, 5, and 15 to see what sort of observations can be made from these simulations when delay is added to reactive  $\beta$ .

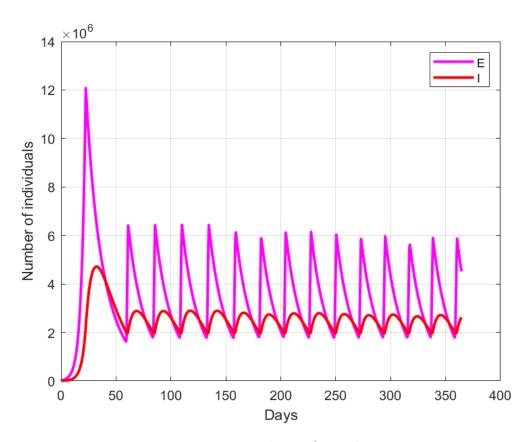


Figure 3.1: Trial #1:  $\delta = 0$  days

Here in Figure 3.1, the delay value is 0. Just from the figure alone, if 0 days of delay was added, then the model just acts as reactive  $\beta$  on its own. Obviously, the  $\beta_{low}$  and  $\beta_{high}$  act on their own the same way from the previous chapter.

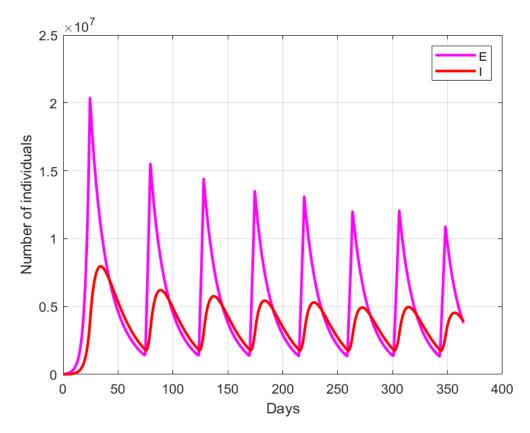


Figure 3.2: Trial #5:  $\delta = 4$  days

Next in Figure 3.2, our fifth simulation produces the model when the  $\delta$  value is 4 days. The number of peaks has decreased from the previous figure including the magnitude of each peak.

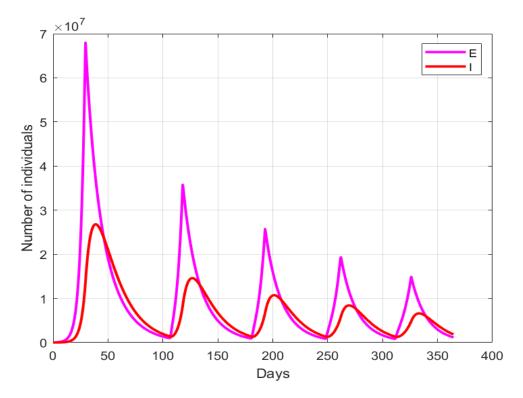


Figure 3.3: Trial #15:  $\delta = 14$  days

Finally in Figure 3.3, the  $\delta$  value is 14 days. There exist less peaks and the size of each peak of the oscillation decreases even more. We can conclude that the longer the delay, the fewer number of peaks and the oscillations last longer. When we are looking at the number of infections from a number of days ago, then the system takes a long time to turn itself around from rise to decay until it forms a new peak.

#### CHAPTER 4: FATIGUE

### 4.1 Introduction to Fatigue Model

The next modification that we are going to analyze is fatigue. Recall that fatigue is feeling overtired or lack of energy. People start to feel tired from staying at home or not traveling outside in order to follow safety protocols. As a result, individuals will then violate safety protocols such as not wearing a mask or violating the 6-feet social distancing policy. Due to fatigue, the transmission rate  $\beta$  will increase overtime.

## 4.2 Fatigue Function

In order to carefully choose our fatigue function to be implemented to the SEIR model, we must think about how fatigue works. As mentioned, people start feeling fatigue and will later violate safety rules. The way we can think of this is that the transmission rate  $\beta$  from the beginning of time starts really low and remains constant for a certain duration. As time passes, people will get tired of being cautious and will take risks. Taking these risks will increase the level of infection and hence  $\beta$  increases. So with this description, the function must be monotonically increasing. Keep in mind that the function we are constructing must be a function of time with the output of the rate of  $\beta$ .

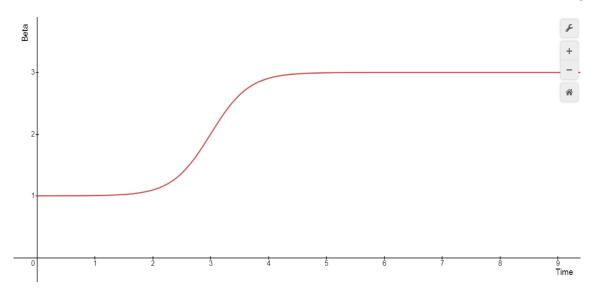


Figure 4.1: Graphical example of fatigue

Figure 4.1 gives the visual example of how our fatigue function should be fabricated.

The function for fatigue is defined including new parameters:

$$\beta(t) = \beta_{high} \left( \frac{e^{S(t-t_c)}}{1 + e^{S(t-t_c)}} \right) + \beta_{low} \left( 1 - \frac{e^{S(t-t_c)}}{1 + e^{S(t-t_c)}} \right), \tag{4.1}$$

where:

- t = time in days
- $t_c$  = critical point in time.

Additionally, we see that there is a "weight" function that acts as an average such that:

$$0 \le \frac{e^{S(t-t_c)}}{1 + e^{S(t-t_c)}} \le 1$$

We will now analyze a couple of models with different values of the critical time to determine what this qualitative feature is behaving like. Keep in mind that the other parameters will be kept fixed continuing on in this chapter.

# 4.3 Fatigue Analysis

In this section, we will now analyze two different models with the fatigue feature implemented with two different  $t_c$  values. Listed below are the parameters that will be kept fixed:

- $\beta_{high} = 0.5$
- $\beta_{low} = 0.01$
- S = 0.001

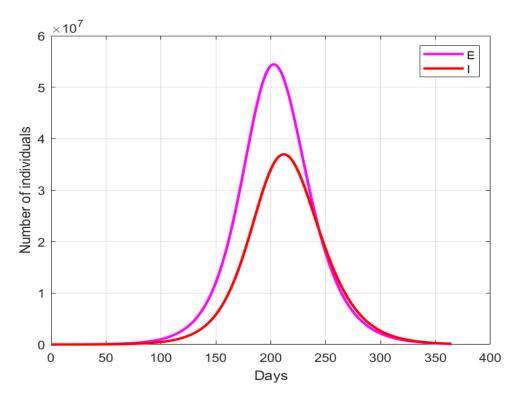


Figure 4.2:  $t_c = 91 \text{ days}$ 

In Figure 4.2, the  $t_c$  value is at the 91<sup>st</sup> day. We see that the value of  $\beta_{low}$  is acting upon before  $t_c$  and  $\beta_{high}$  is acting upon after  $t_c$ . This is why we see this slow increase before  $t_c$  and the outbreak rises fast after the  $t_c$  value.

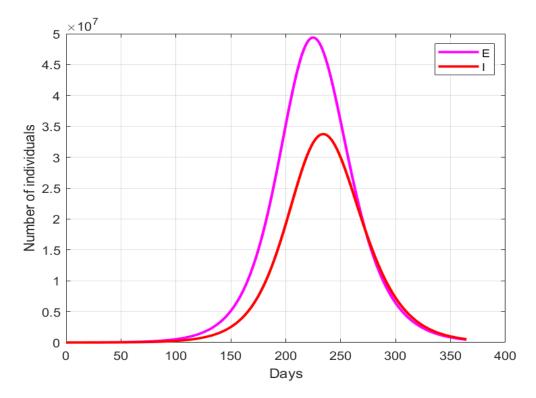


Figure 4.3:  $t_c = 182 \text{ days}$ 

In Figure 4.3, the  $t_c$  value is at the  $182^{\rm nd}$  day. The shape of the model is relatively the same as from Figure 4.2 which creates this shift. In other words, if the  $t_c$  value is low, then the outbreak occurs earlier whereas if the  $t_c$  value is high, the outbreak occurs later. Now we can infer from these two graphs that whenever fatigue occurs, the outbreak rises at the critical time and it is burning through all the susceptible transitioning to exposed and exposed moving to infected.

#### CHAPTER 5: POLICY INTERVENTION

### 5.1 Introduction to Policy Intervention Model

The last modification that is going to be useful to analyze is policy intervention. To put it simply, if a policy were to be issued for all individuals to follow and obey,  $\beta$  will decrease and if a policy were to be lifted, then  $\beta$  will increase, since people are more likely to take risk once the policy is no longer in effect. Now that we have a clear understanding of what policy interventions can do to affect the rate of  $\beta$ , we will now choose a realistic  $\beta$  function that captures this modification.

### 5.2 Policy Intervention Function

From the previous section, we established what a policy intervention is and why the purpose of implementing this factor is important to the SEIR model. In order to find a reasonable choice of a  $\beta$  function representing policy interventions, we restate what policy interventions will do to the rate of transmission. We mention that  $\beta$  will increase when a policy has been lifted and  $\beta$  will decrease when a policy has been issued or mandated. Hence, we model these intervention by making  $\beta$  a piecewise constant function of time. It also makes sense to say that  $\beta$  is constant with changes occurring when new policies are put in place. Figure 5.1 provides a visual example of how our policy intervention function should look.

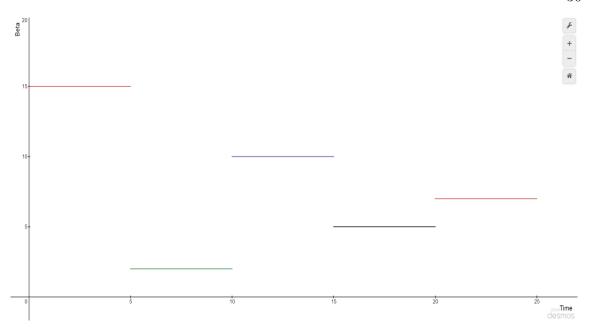


Figure 5.1: Graphical example of policy intervention

Thus, the best choice for the  $\beta$  function used for policy interventions is a piece-wise constant function. Then for all  $\beta_k > 0$ , and  $t_k > 0$  where k = 1, ..., n, our piece-wise constant function is defined as follow:

$$\beta(t) = \begin{cases} \beta_1, & 0 < t \le t_1 \\ \beta_2, & t_1 < t \le t_2 \\ & \vdots \\ \beta_n, & t_{n-1} < t \le t_n. \end{cases}$$
(5.1)

We will now conduct an analysis of policy interventions and investigate what their impact on the model outputs.

### 5.3 Policy Intervention Analysis

In this section, we will analyze the model with the policy intervention factor added. This factor is meant to capture the typical statewide policy interventions. In particular:

- 3 outbreak starts with no awareness for the first 15 days
- 0.05 issued lockdown from Day 16 to Day 60
- 0.5 businesses open up from Day 61 to 80
- 0.1 mask mandate from Day 80 and onwards.

Thus, we have the following  $\beta$  function:

$$\beta(t) = \begin{cases} 3, & 0 < t \le 15 \\ 0.05, & 15 < t \le 60 \\ 0.5, & 60 < t \le 80 \\ 0.1, & 80 < t. \end{cases}$$
 (5.2)

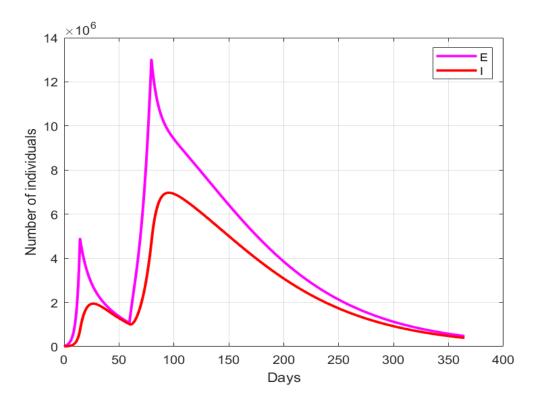


Figure 5.2: Policy Intervention Model with  $\beta(t)$ 

In Figure 5.2, the model is produced with the piecewise constant function. When  $\beta=3$  for  $0 < t \le 15$ , the system starts with a huge rise as no individuals are aware that the outbreak has begun. When  $\beta=0.05$  for  $15 < t \le 60$ , a lockdown has been set and we see the number of infected individuals decreasing. Next, when  $\beta=0.5$  for  $60 < t \le 80$ , some businesses have reopened and people are performing risky behavior which is why we see a rise in the model. Lastly, when  $\beta=0.1$  for 80 < t, a mask mandate has been issued and the infected curve decreases as this intervention reduces the rate of transmission. Thus, the disease dynamics can be seen to track the policy interventions.

## CHAPTER 6: CONCLUSIONS AND DISCUSSION

In this work we have evaluated four modifications of the standard SEIR model. We analyzed the behavior of solutions under these four types of modifications, and we have attempted to understand how the model outputs depend on the parameter choices. Let us now evaluate whether these modifications match the qualitative features of the real world data.

Reactive  $\beta$  gives us a strong understanding of why the number of infected rises and decays depending on the reaction to the level of infection. Both the real world data and this modified model yield oscillations in the number of infected individuals. However, the oscillations that appear in our model do not appear in the real world data, as the model oscillations decrease in magnitude, whereas the real oscillations increase in magnitude.

In our reactive  $\beta$  with delay model, the delay parameter helps us observe what would happen if people were to look back on some number of days ago, read the data, and react to the infection level with that information given. As before, the oscillations that appear in this model do not appear to match those observed in the real world data.

Fatigue is an important factor used here, as we observe anecdotally that people tend to take greater risks as time goes on. We see that depending on some time, people will eventually grow tired of following protocols and will carry out risky behavior. In our fatigue model, we see that there is only one peak, followed by decay towards zero. As there are no oscillations, we conclude that fatigue alone cannot explain the oscillations observed in the real world data.

Lastly, policy interventions form another important factor in understanding the

effects of pathogen transmission. Lifting off and mandating safety protocols and practices clearly affects the number of exposed and infected throughout time, and the level of infection determines whether the rules/regulations will be set in motion or removed.

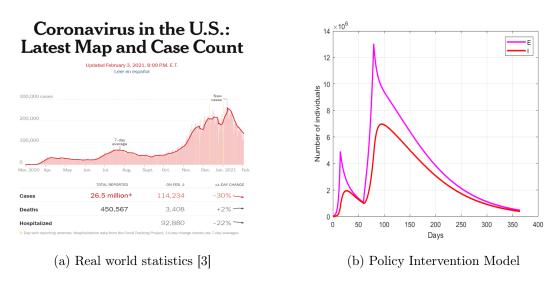


Figure 6.1: Side by side comparison

Looking at Figure 6.1(a), the first peak appears around April 2020. It is likely that there is some intervention taking place between that time window. Then the second peak appears around the summer, approximately July to August. Finally, there is a huge rise of infected taking place around early fall and lasting all the way to the winter season and peaks are appearing everywhere throughout those two seasons. Now if we pay attention to the model from Figure 6.1(b), we see that when using the  $\beta$  values and time values from Equation 5.2, the two models, if we compare them together, are similar qualitatively. But, our piecewise constant function (Equation 5.2) contains four  $\beta$  values which is enough to produce two peaks corresponding with the spring and summer seasons. Given the policy interventions that we have modeled, our model does not produce a third peak. On the other hand, there is clearly a large third peak over the winter in the real data. In this sense, the policy intervention model fails to capture the qualitative behavior of the real world data.

Of course, there are other possible modifications that can strongly affect the rise of infected of the disease that we not have not analyzed at all. The possibility of combining all four modifications in the model could produce results that match the main features observed in the real world data. Adding vaccinations to the model could provide us with the relationship between the recovery and the infected curves. There are also the superspreader events, where the rate of pathogen transmission rises drastically for a short period of time, and modeling this factor could also improve our models. We believe that further modifications along these lines present potentially interesting avenues for future research.

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