Spring 5-12-2023

Imperfect Immunity and the Stability of a Modified Kermack-McKendrick Model

Kaylee Sims

IMPERFECT IMMUNITY AND THE STABILITY OF A MODIFIED KERMACK-MCKENDRICK MODEL

by

Kaylee Sims

A thesis submitted to the faculty of The University of Mississippi in partial fulfillment of the requirements of the Sally McDonnell Barksdale Honors College

Oxford

May 2023

Approved by:

_____

Advisor: Dr. Samuel Lisi

_____

Reader: Dr. Laura Sheppardson

_____

Reader: Dr. Ruaa Al Juboori

Dedicated to Kevin and Kathy Sims, with love.

ACKNOWLEDGEMENTS

I would like to start by thanking my advisor, Dr. Samuel Lisi, for his continual kindness, guidance, and encouragement. Dr. Lisi, you have never failed to show me the power of curiosity and the value of chipping away at difficult problems. I came into your office with only vague ideas of what higher mathematics and mathematical research constituted, and I am leaving with a new worldview, an enhanced education, and a renewed confidence in my career path. I cannot understate how thankful I am.

I would also like to thank the members of my thesis committee, Dr. Laura Sheppardson and Dr. Ruaa Al Juboori, for their invaluable time and profound insights, which graced this project with a new depth. Further, I am extremely grateful to Dr. John Samonds, Dr. Laura Sheppardson, Dr. Gerald Buskes, Professor Stephanie Blue, Ms. Deborah Stamper, and Mrs. Michelle Ogburn for their key roles in shaping me into the student that I am today. Thank you for inspiring and challenging me.

Finally, I would like to express my gratitude to the Sally McDonnell Barksdale Honors College itself. Its administrators, professors, benefactors, and students have repeatedly gifted me with sincere support and inspiration. Together, the Honors College and the Mathematics Department have been the cornerstone of my undergraduate experience and have truly changed me for the better.

ABSTRACT

The classic Kermack-McKendrick model of mathematical epidemiology suggests that a population is only in equilibrium when there is no disease present. In the modern era, we have several cyclic infectious diseases that show no signs of eradication, despite global health measures. In this thesis, we introduce a coefficient of waning immunity in order to produce a modified Kermack-McKendrick model and analyze whether the model yields system stability with a certain amount of infection present. Ultimately, the model is incongruent with real-world case data, with its most glaring failure being exponential dampening of the height of each disease case peak due to the system's complex eigenvalues.

Contents

List of Figures

List of Tables

1 An Analysis of the Standard "SIR" Model of Compartmental Epidemiology

"All models are wrong, but some are useful." - George Box

Compartmental models essentially seek to assign every object in a closed, dynamic system into one of two or more disjoint compartments and study the interactions between each compartment. These models are typically governed by one or more differential equations. Compartmental models are often used to understand population dynamics, by seeking to model future changes to that population and alternate outcomes. For example, the Lotka-Volterra model was created to study variations in animal populations, and the Bass model has been used to study and forecast product sales. Analyzing compartmental models using calculus and differential equations allows us to identify equilibrium points within the system and classify the stability of each point. Here, we will discuss the widely-used Kermack-McKendrick or "SIR" compartmental model of mathematical epidemiology and study a proposed modification.

1.1 Equations and Assumptions

The standard "susceptible, infected, recovered" compartmental model places every person in a given population into one of three disjoint compartments in the presence of an emerging infectious disease. The three compartments are susceptible persons, infected persons, and recovered persons. It is governed by three ordinary differential equations. The model requires several assumptions:

- All susceptible persons are assumed to be equally vulnerable to the disease, and all infected persons are equally likely to die from the disease.
- The rate of interaction between susceptible and infected persons is constant.
- Individuals that die of the disease move to the "recovered" compartment.
- Infected individuals move to the "recovered" compartment once they are no longer infectious, even if they are still symptomatic.
- An infected person will either die from the disease or gain permanent immunity from re-infection.
- Births and deaths unrelated to the disease are not considered. No individuals enter or leave the system, they may only change compartments.

Kermack and McKendrick first introduced this compartmental model in 1927 [9], thus the classic SIR model is also known as a Kermack-McKendrick model. For the sake of consistency, I have substituted their

original chosen variables with the variables I will use throughout this document. The basic SIR model relies upon the following system of differential equations:

$$\frac{ds}{dt} = -\epsilon s(t)i(t) \tag{1}$$
$$\frac{di}{dt} = \epsilon s(t)i(t) - \phi i(t) \tag{2}$$
$$\frac{dr}{dt} = \phi i(t) \tag{3}$$

where t represents time, $s(t)$ represents the susceptible population at a given point in time, $i(t)$ represents the infected population at a given point in time, and $r(t)$ represents the recovered population at a given point in time. Thus $\frac{ds}{dt}, \frac{di}{dt}, \frac{dr}{dt}$ represent the rate of change in each respective compartment over time. Let ϵ, which the Kermack-McKendrick model named k, denote the fixed rate of interactions between the susceptible and infected compartments per time period t that result in new infections. Let ϕ, which the Kermack-McKendrick model named l, denote the fixed fraction of the infected population that will recover and no longer be infectious during the time period t. ϕ is often called the "removal rate". ϕ can be thought of as a reciprocal of the average length of infectiousness. That is, if a person is infectious for five (5) days and the time period t is in days, $\phi = \frac{1}{5} = 0.2$.

Since the total population is assumed to be constant, the SIR model also uses the equation:

$$s(t) + i(t) + r(t) = N \tag{4}$$

where N denotes the unchanging total population. We will let $N = 1$, which allows us to think of the values of $s(t)$, $i(t)$, and $r(t)$ as the density of the population in each compartment. From here, we derive the following identity.

$$s(t) + i(t) + r(t) = 1 \tag{5}$$

The movement between each compartment and the variable governing this movement may be summarized via flowchart in Figure 1.

Figure 1: Compartmental Flow of the Standard SIR Model

1.2 Analysis via Phase Portrait

In this section, we will analyze the classic Kermack-McKendrick compartmental model, following an outline proposed by Strogatz in Excercise 6.5.6 [11]. We will assume that $\epsilon, \phi > 0$ and will eliminate the equation for the recovered compartment, since it does not affect the interaction between the susceptible and infected populations. Following Strogatz's notation, we "let $x(t) \geq 0$ denote the size of the healthy population and $y(t) \geq 0$ denote the size of the sick population." That is, for this section, $s(t) = x(t)$ and $i(t) = y(t)$. His final prompt asks "An epidemic is said to occur if $y(t)$ increases initially. Under what circumstances does an epidemic occur?" To answer his question, we will consider three phase portraits, corresponding to $\frac{\epsilon}{\phi} = 1$, $\frac{\epsilon}{\phi} < 1$, and $\frac{\epsilon}{\phi} > 1$. Afterwards, we will discuss the significance of this ratio and the fixed points of each phase plane.

1.2.1 $\frac{\epsilon}{\phi} = 1$

For the condition $\frac{\epsilon}{\phi} = 1$ to be true, $\epsilon = \phi$ must be true. Since we are only interested in the value of the ratio, we may choose $\epsilon = \phi = 1$ without loss of generality.

With this condition, we derive the fixed points $(x, 0)$ and $(0, 0)$ by setting Equations 1 and 2 equal to zero and substituting $x = s$ and $y = i$. When viewing all phase plane, we must keep Equation 5 in mind, which tells us that $x + y \leq 1$ in all scenarios. Therefore, for example, the coordinate (0.5, 0.7) is not relevant to our system. The phase plane of the system $< -xy, xy - y >$ is shown in Figure 2. Since we are primarily interested in Quadrant I, where there are real values for the susceptible population and infected population, the phase portrait does not include the full coordinate plane. We may observe that $y(t)$, that is, the infected population, does not increase initially for any possible combinations of (x,y). Thus, under the condition that $\frac{\epsilon}{\phi} = 1$, an epidemic should not occur.

1.2.2 $\frac{\epsilon}{\phi} < 1$

To enable $\frac{\epsilon}{\phi}$ to be small, we will choose $\epsilon = 0.5$ and $\phi = 1$. This yields $\frac{\epsilon}{\phi} = 0.5$. With these conditions, we derive the same fixed points, $(x, 0)$ and $(0, 0)$. Quadrant I of the resultant phase portrait of the system $< -0.5xy, 0.5xy - y >$ is shown in Figure 3. Clearly, we can see that $y(t)$ sharply decreases not only initially, but at all values shown, which should not allow an epidemic to occur. This also follows intuition, since $\epsilon = 0.5$ indicates a small number of interactions resulting in new infections, and $\phi = 1$ indicates that a person is infectious for exactly one period of time t.

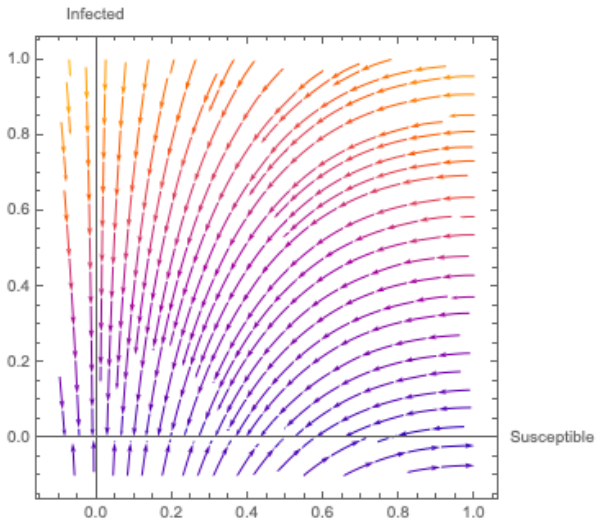

Figure 2: $\frac{\epsilon}{\phi} = 1$ Phase Portrait

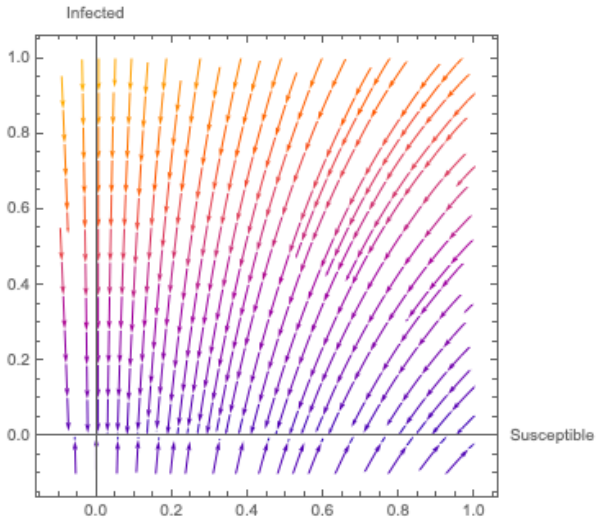

Figure 3: $\frac{\epsilon}{\phi} = 0.5$ Phase Portrait

1.2.3 $\frac{\epsilon}{\phi} > 1$

Here, we will first choose $\frac{\epsilon}{\phi} = 1.5$ by selecting $\epsilon = 0.3$ and $\phi = 0.2$. Once again, the fixed points remain $(x, 0)$ and $(0, 0)$. The phase portrait generated by $< -0.3xy, 0.3xy - 0.2y >$ is shown in Figure 4. This figure shows that there are several points where $y(t)$ does increase initially, such as the ordered pair $(0.8, 0.2)$, corresponding to a situation where 20% of the population is infected.

Next, we will choose $\frac{\epsilon}{\phi} = 15$ via $\epsilon = 1$ and $\phi = \frac{1}{15}$ to observe how the phase plane changes as this ratio grows. Figure 5 corresponds to $< -xy, xy - \frac{1}{15}y >$, and it clearly indicates a natural inclination for the infected proportion of the population to rise at all relevant coordinate pairs.

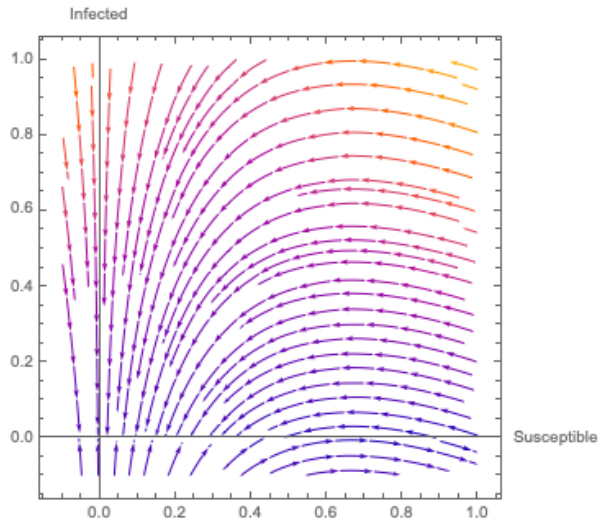

Figure 4: $\frac{\epsilon}{\phi} = 1.5$ Phase Portrait

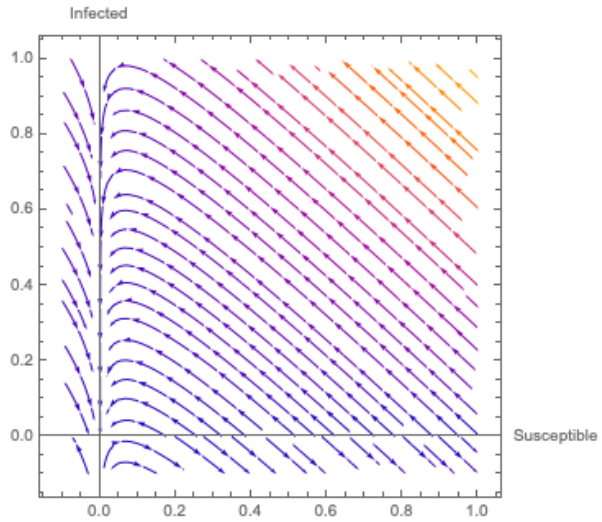

Figure 5: $\frac{\epsilon}{\phi} = 15$ Phase Portrait

1.3 The Significance of R_0

The Basic Reproduction Number quantifies the expected average number of new infections that will arise in a wholly susceptible population from one infected individual. This number is denoted by R_0, and essentially quantifies how quickly an infectious disease can spread in a given population. R_0 is dimensionless.

As Jones [8] explains, R_0 is the product of the rate of transmissibility ($\frac{infection}{contact}$), the rate of contact between susceptible and infected individuals ($\frac{contact}{time}$), and the length of time that a person is infectious

($\frac{time}{infection}$). That is:

$$R_0 \propto \frac{infection}{contact} \cdot \frac{contact}{time} \cdot \frac{time}{infection} \tag{6}$$

Since our variable ϵ assumes constant contact between the susceptible and infected compartments, ϵ can be thought of as the product of the rates of transmissibility and contact, and thus quantifies $\frac{infection}{time}$. Further, since ϕ is the reciprocal of the length of infectiousness, R_0 can be calculated using ϕ^{-1}, which is $\frac{time}{infection}$. Thus, using the variables in Equations 1, 2, and 3, we derive:

$$R_0 = \frac{\epsilon}{\phi} \tag{7}$$

This value is extremely significant, as it is widely accepted in the field of epidemiology [8] that when $R_0 \leq 1$, there is a low chance for an epidemic to occur, but when $R_0 > 1$, there is a much higher chance that an epidemic is imminent. The previous section illustrates this phenomenon via phase planes, and R_0's breakpoint also makes intuitive sense. $R_0 = 1$ corresponds to a situation in which one infection begets one new infection, essentially keeping the total number of cases steady. As R_0 increases, secondary infections increase at a higher rate, allowing for the population-wide exponential increase in infections seen in epidemics.

1.4 The Significance of the System's Fixed Points

Recall that for each value of $\frac{\epsilon}{\phi}$, which we now know as R_0, the fixed points of the phase plane were $(x, 0)$ and $(0, 0)$, where the x-coordinate corresponds to the susceptible population, $s(t)$ and the y-coordinate corresponds to the infected population, $i(t)$. Crucially, this indicates that the system is unstable whenever infection is present, and the system will return to a baseline equilibrium with no active infections. The fixed point (x,0) may be interpreted three different ways using Equation 5.

- For $x = s(t) = 1$, $i(t) = 0$ and $r(t) = 0$, indicating that the infection was never present in the population at all.
- For $0 < x < 1$, $i(t) = 0$ and $r(t) = 1 - x$, indicating that the infection has run its course and will not re-emerge due to the perfect immunity and/or death of the recovered population. There is some proportion of the population that remains susceptible but will never be infected.
- When $x = 0$, we have the second fixed point, $(0, 0)$, which indicates that $s(t) = 0$, $i(t) = 0$, and $r(t) = 1$. Here, stability is only achieved once the entire population has been infected and subsequently recovered or died.

2 The Need for Alterations to the Standard "SIR" Compartmental Model

The standard SIR model's biggest strength is also its biggest weakness: its simplicity. There are hundreds of real-world situations that defy the assumptions laid out in Section 1.1, yet the model and its variations are used repeatedly with reasonable accuracy to model epidemics, learn more about their theoretical nature, and estimate values for R_0. However, there are also obvious flaws and reasons why the standard SIR model cannot be relied upon for many complex epidemics. For example, the standard model does not account for vaccination, vulnerable and immunocompromised populations, nor global health measures such as masking or social distancing. In light of COVID-19, perhaps the most glaring weakness is that it assumes completely perfect disease immunity among the "recovered" compartment. With perfect immunity, as we saw previously, the system will always return to an equilibrium that has no new infections. Yet, in the real world, we see many instances of epidemics becoming a long-term, recurrent problem, as in the case of COVID-19 and H1N1. In the interest of preserving the beautiful simplicity of the SIR model, I sought to propose only one significant modification to the system of equations and analyze the resultant impacts on the stability of the system.

2.1 Introduction of a Coefficient of Waning Immunity

Let θ be known as the "coefficient of waning immunity". This coefficient represents the average amount of time before a surviving individual in the recovered compartment loses natural immunity and returns to the susceptible compartment. As we collectively observed with the COVID-19 outbreak, the ability to be re-infected absolutely changes the dynamics of disease spread. Since compartmental models of epidemiology are primarily used to model future outcomes, this modification was proposed to see how condensing waning immunity to one variable would affect the resultant system. Since θ involves interactions purely between the susceptible and recovered compartments, the modified system of equations becomes:

$$\frac{ds}{dt} = -\epsilon s(t)i(t) + \theta r(t) \tag{8}$$

$$\frac{di}{dt} = \epsilon s(t)i(t) - \phi i(t) \tag{9}$$

$$\frac{dr}{dt} = \phi i(t) - \theta r(t) \tag{10}$$

Please note that when $\theta = 0$, the system is identical to the original system of equations in Section 1.1, thus $\theta = 0$ represents perfect immunity, and the larger θ is, the shorter the average period of immunity is for the

given disease. The new flow between each compartment and its associated variable is shown in Figure 6.

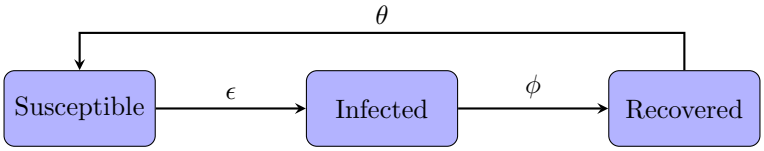

Figure 6: Compartmental Flow of the Modified SIR Model

2.2 Introduction of a New Non-dimensional Parameter, μ

We must reduce the number of current variables so that we may later analyze our system via a two-dimensional plot. We will also define a new dimensionless parameter denoted by μ. Since ϕ is in units of $\frac{infection}{time}$ and θ is in units of $\frac{immunity}{time}$, we may quantify and non-dimensionalize the relationship between ϕ and θ by deriving μ such that:

$$\mu = \frac{\theta}{\phi} \tag{11}$$

Recall that if a person is infectious for five days, $\phi = \frac{1}{5}$, thus ϕ is generally small. In many recent epidemics, the period of natural immunity represented by θ has lasted far longer than the associated period of infectiousness, thus we may assume that θ is generally much smaller than ϕ. Then, for most real-world scenarios of imperfect disease immunity, μ is very small.

2.3 Time Scale Change

At present, we have condensed our system into three parameters, R_0, μ, and time (t). We will change our time scale and non-dimensionalize it by choosing:

$$t = \frac{\tau}{\phi} \tag{12}$$

and allowing the new time period, τ to be represented by one cycle of the infectious period by choosing $\phi = 1$ when scaled.

By applying the chain rule to Equations 8, 9, and 10, we have:

$$\frac{ds}{d\tau} = -\frac{\epsilon}{\phi} si + \frac{\theta}{\phi} r \tag{13}$$

$$\frac{di}{d\tau} = \frac{\epsilon}{\phi} si - i \tag{14}$$

$$\frac{dr}{d\tau} = i - \frac{\theta}{\phi} r \tag{15}$$

2.4 Final Non-Dimensionalization of the System

Finally, using the identities put forth in Equations 7 and 11, we derive:

$$s' = -R_0 si + \mu r \tag{16}$$

$$i' = R_0 si - i \tag{17}$$

$$r' = i - \mu r \tag{18}$$

which we may now use to perform a stability analysis of the modified system.

3 Stability Analysis of the New System

We have previous noted that μ is assumed to be very small, therefore we may safely approximate $\mu^n \approx 0$ for all $n > 1$. This approximation will be used throughout this chapter.

3.1 Deriving Fixed Points of the Modified System

Here, we will eliminate r by applying Equation 5 to the system of equations found in Section 2.4 via substitution. This system becomes:

$$s' = -R_0 si + \mu(1 - s - i) \tag{19}$$

$$i' = R_0 si - i \tag{20}$$

$$r' = i - \mu(1 - s - i) \tag{21}$$

Since this substitution makes Equation 21 redundant, we will proceed using Equations 19 and 20 to represent the entire system. As we did within Chapter 1, for fixed points, we will let the first coordinate in the ordered plane denote the susceptible population, s, and the second coordinate will correspond to the infected population, i. The fixed point $(1, 0)$ exists, which may be considered the trivial point, since this is the system with no disease present. We also have the critical point $\left(\frac{1}{R_0}, \frac{\mu}{1+\mu} \cdot \frac{-1}{R_0}\right)$. Please note that because μ will be small for real-world epidemics, $\mu \approx \frac{\mu}{1+\mu}$, and $\left(\frac{1}{R_0}, \frac{\mu}{1+\mu} \cdot \frac{-1}{R_0}\right) \approx (\frac{1}{R_0}, \mu(1 - \frac{1}{R_0}))$.

Our challenge becomes interpreting this critical point. If we choose $R_0 = 2.5$ and $\mu = 0.05$, which corresponds, for instance, to a generic disease with $\epsilon = 0.5$ capable of an epidemic with a 5-day period of acute infectiousness and a 90-day period of immunity, the fixed point gains a real value of (0.4, 0.03), which means the recovered proportion is 0.57 and 3 percent of the population is actively infected. If we choose $R_0 = 10$ and $\mu = 0.05$, we have a disease with a much higher ϵ value but, potentially, the same values for ϕ and θ as the previous example. The fixed point becomes (0.1,0.045), which corresponds to a system where only 10 percent of the population remains susceptible and 4.5 percent are actively infected.

3.2 Evaluating the Eigenvalues of the New System

3.2.1 Eigenvalues of the "Trivial Point"

Using the fixed point (1,0), we can calculate the Jacobian Matrix for our modified system of equations via:

$$\begin{bmatrix} \nabla s'(1,0) \\ \nabla i'(1,0) \end{bmatrix} = \begin{bmatrix} -\mu & -R_0 - \mu \\ 0 & R_0 - 1 \end{bmatrix}.$$

Since this is an upper triangular matrix, we may easily find that the eigenvalues for the system at this point are $\lambda_1 = -\mu$ and $\lambda_2 = R_0 - 1$, its trace is $R_0 - 1 - \mu$, and the determinant is $\mu(1 - R_0)$. In the presence of an epidemic, $R_0 > 1$, so we would expect the point to have one negative real eigenvalue and one positive real eigenvalue, corresponding to a saddle point.

3.2.2 Eigenvalues of the Second Fixed Point

Using the critical point $\left(\frac{1}{R_0}, \frac{\mu}{1+\mu} \cdot \frac{-1}{R_0}\right) \approx \left(\frac{1}{R_0}, \mu(1-\frac{1}{R_0})\right)$, we compute the Jacobian Matrix $\begin{bmatrix} -R_0\mu & -1-\mu \\ R_0\mu - \mu & 0 \end{bmatrix}$ which has a trace of $-R_0\mu$ and a determinant of $\mu(R_0 - 1)$.

We may find the system's eigenvalues via the characteristic function:

$$0 = \begin{vmatrix} -R_0\mu - \lambda & -1-\mu \\ R_0\mu - \mu & 0 - \lambda \end{vmatrix} = (-R_0\mu - \lambda)(-\lambda) - (-1-\mu)(R_0\mu - \mu) \tag{22}$$

which we may solve using the quadratic formula and the approximation introduced at the start of Chapter 3. This yields:

$$\lambda \approx \frac{-R_0\mu \pm \sqrt{-4\mu(R_0 - 1)}}{2} \tag{23}$$

which simplifies to:

$$\lambda \approx \frac{-R_0\mu}{2} \pm \sqrt{\mu(1 - R_0)} \tag{24}$$

which is rather difficult to make sense of without substituting in numerical values for μ and R_0. However, we may make some general observations. In the presence of an epidemic, $R_0 > 1$, so $\mu(1 - R_0) < 0$, which guarantees a complex eigenvalue. Recall that $\mu, R_0 \geq 0$ by definition, and it becomes obvious that the real part of the eigenvalues will always be strictly negative if an epidemic with imperfect immunity is present. The presence of complex eigenvalues with a negative real part indicates that the phase portrait will show a stable spiral near the critical point. Here, we see the first notion of stability in the system when there is an active infection in the population.

3.3 Generating Phase Portraits by Inserting Numerical Values for μ and R_0

In this section, we will use approximate values for μ and R_0 that correspond to real-world diseases capable of producing an epidemic, so that we may make sense of their phase portraits and fixed points and eventually compare the results to real-world data. Since μ is not clearly defined for any one disease and seasonal

immunity is not fully understood, estimations will be made based upon current literature for each disease.

3.3.1 Disease 1: Seasonal Influenza

Disease 1 is based upon seasonal influenza, without differentiating between sub-types. We will assign an average R_0 of 1.28 [3] and an average ϕ of $\frac{1}{4}$ [1] based upon available literature. An average θ of $\frac{1}{240}$ corresponds to waning immunity around the seasonal four-month period of acute flu infections observed in the Centers for Disease Control and Prevention's published yearly flu data. Using these values, we attain $\mu = 0.017$. The fixed point $(\frac{1}{R_0}, \mu(1-\frac{1}{R_0}))$ from Section 3.1 becomes (0.78, 0.00374), indicating convergence to a very small proportion of actively infected individuals with a significant number of recovered individuals. The phase portrait is shown in Figure 7. Here, we see a barely perceptible stable spiral around the critical point, as expected, and a saddle point across the horizontal axis at (1,0).

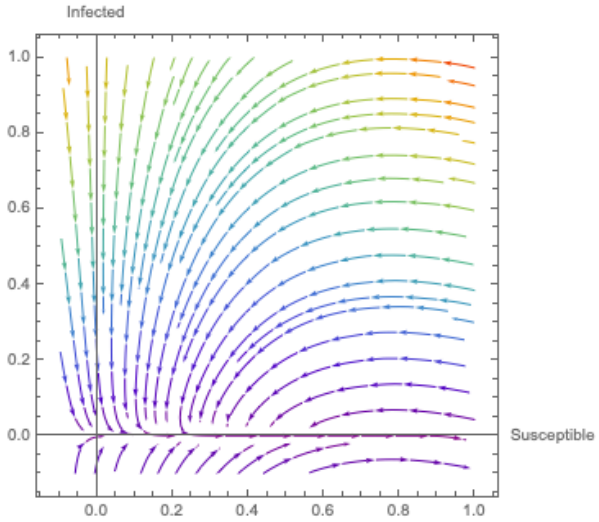

Figure 7: Phase Portrait Corresponding to an Approximation of Seasonal Influenza

3.3.2 Disease 2: COVID-19

The parameters for disease 2 shall be based upon initial values of COVID-19, also known as SARS-CoV-2, found in current literature. We will average the mean and median R_0 determined across multiple studies [10] and assign $R_0 = 3$ to disease 2. Another study found that the average length of infectiousness and viral shedding was eight days [12], yielding $\phi = \frac{1}{8}$. The length of natural immunity varies wildly and is dependent upon the disease strain and an individual's vaccination status, but the Centers for Disease Control states that previously infected individuals "have a low risk of subsequent infection for at least 6 months" [4]. We will assign $\theta = \frac{1}{180}$, corresponding to an average of six months of immunity. These values give an approximate μ

of 0.044. The fixed point gains a real value of (0.33, 0.0296), indicating convergence when there is a sizeable amount of active infections and a significant majority of the population has already been infected. The corresponding phase portrait is shown in Figure 8, which also displays the expected stable spiral at (0.33, 0.0296) and a saddle point at (1, 0). This phase portrait shows a much clearer spiral, indicating that the system would prefer to hover around an approximate 3 percent case rate at all times.

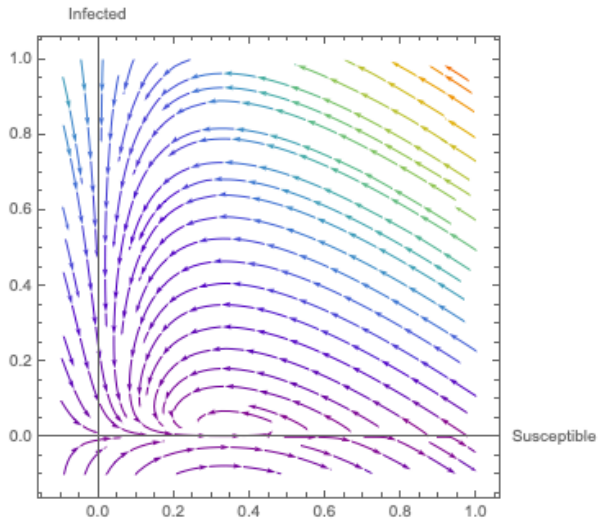

Figure 8: Phase Portrait Corresponding to an Approximation of COVID-19

3.3.3 Disease 3: Measles

Disease 3 is based upon Rubeola, commonly known as measles. Surviving individuals previously infected with the disease, but not necessarily those that were vaccinated, are expected to have lifelong, perfect immunity [2]. We will examine this disease as a control of sorts, since the modified system of equations herein is designed to apply to epidemics where there are signs of waning immunity. For measles, we will choose $R_0 = 15$ [7]. Recall that for perfect immunity, $\theta = 0$, so regardless of the value of ϕ, we arrive at $\mu = 0$. The phase portrait is shown in Figure 9, which is nearly identical to Figure 5, which had an identical R_0 assigned to it. This indicates that the addition of the parameters θ and μ still allows for some consistency with the original Kermack-McKendrick model. The critical points are (0.0667, 0) and (1,0). Notably, we see a directional change in the flow of the diagram for all values of (0.0667, i) for all possible values of $0.0667 < i < 1 - 0.0667$.

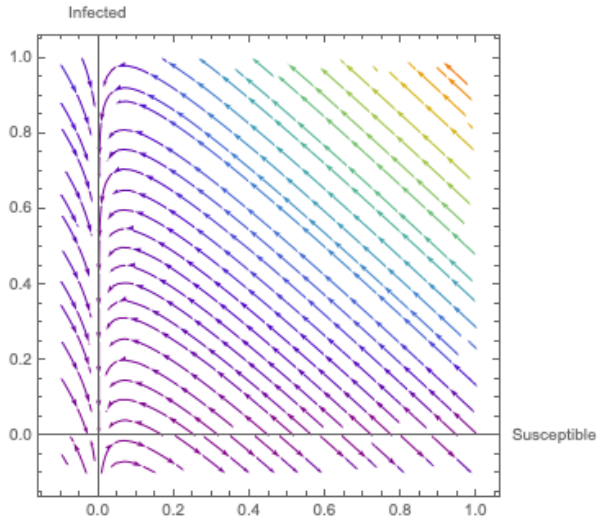

Figure 9: Phase Portrait Corresponding to an Approximation of Measles

4 Drawing Conclusions

To explore the viability of the proposed modified Kermack-McKendrick model detailed herein, in this section we will use our chosen parameters to project case data and compare it to actual epidemic case data. Since the chosen parameters by themselves cannot convey the full picture of widespread infectious diseases, although they are all based on a thorough review of scientific literature, please be aware that the primary goal is to analyze patterns and see what conclusions can be drawn.

4.1 Comparison to Actual Case Data

4.1.1 Influenza Comparison

Using our modified equations and chosen parameters, we can attempt to project cases of disease 1 over 200 cycles of τ. The results are shown in Figure 10. Please note that τ is non-dimensional and $\phi = 1$ in our modified system, so the x-axis may be read as "cycles of infectiousness." That is, for disease 1 with our chosen value of ϕ, each integer on the horizontal axis represents 4 days.

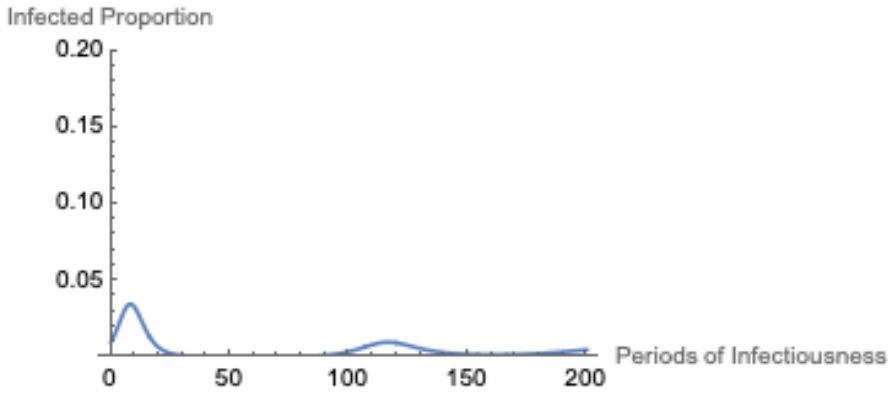

Figure 10: Projected Cases Using Approximate Influenza Parameters

Figure 10 shows two discernible peaks and the beginnings of a third, with each peak becoming less and less extreme. Here we see some notion of seasonality, which is promising, but when we compare Figure 10 to Table 1, which contains estimated total cases of Influenza as published by the Centers for Disease Control and Prevention [6], we can clearly see that Influenza does not get less severe as time goes on, as the model forecasted. In reality, there is significant fluctuation in the size of each peak of cases from year to year.

Table 1: CDC Yearly Influenza Data [6]

Influenza Season	Estimated Total Cases
2010-2011	21,000,000
2011-2012	9,300,000
2012-2013	34,000,000
2013-2014	30,000,000
2014-2015	30,000,000
2015-2016	24,000,000
2016-2017	29,000,000
2017-2018	41,000,000
2018-2019	29,000,000
2019-2020	36,000,000

4.1.2 COVID-19 Comparison

As before, we will use our chosen parameters to attempt to project cases for disease 2, as shown in Figure 11. We defined disease 2 with $\phi = \frac{1}{8}$, so each integer on the horizontal axis corresponds to 8 days. This projection shows a very large peak with rougly 30 percent of the population infected, and then a sharp decline in cases before another small peak and decline. From there, the model suggests that roughly 2.96% of the population will be infected at any given time, corresponding to the fixed point (0.33, 0.0296) that served as the center of the stable spiral in Figure 8.

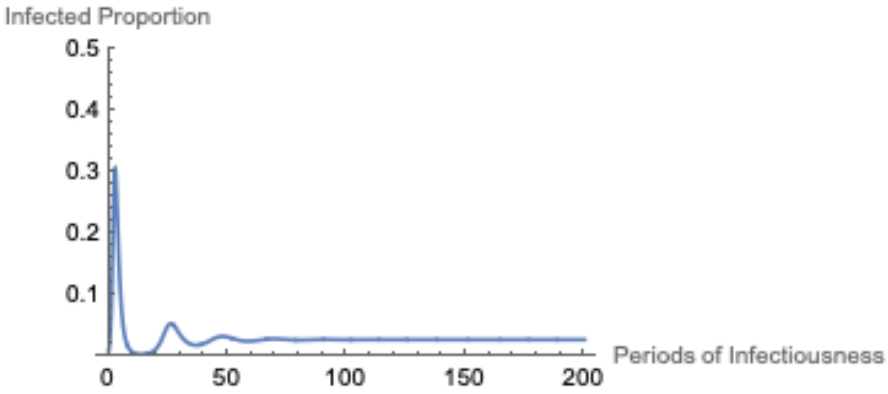

Figure 11: Projected Cases Using Approximate COVID-19 Parameters

When we compare Figure 11 to actual COVID-19 case data as reported to the Centers for Disease Control and Prevention [5] found in Figure 12, several glaring errors become evident. The actual data contains several peaks rather than the two that the model implied, and there is nowhere near a constant 3 percent case rate. In 2021, the total population of the United States was approximately 332 million people, meaning a 2.96% case rate would correspond to roughly 9.8 million cases in an 8 day period. This case projection is unreasonable when compared to CDC data, which shows that reported cases never surpassed 6 million cases

in 7 days.

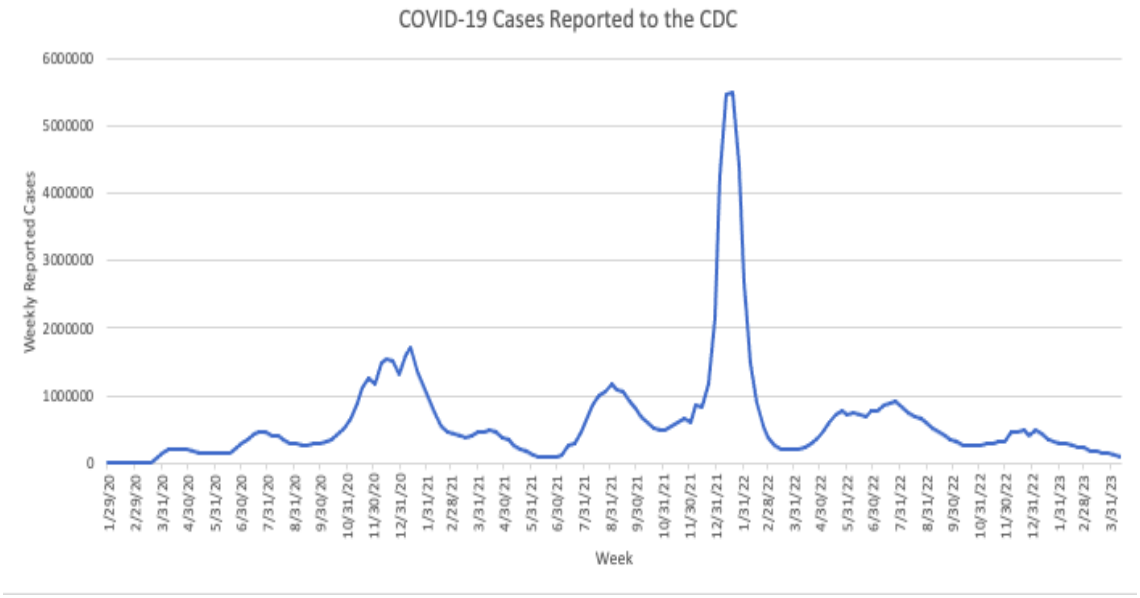

Figure 12: Weekly COVID-19 Cases Reported to the CDC [5]

4.1.3 Measles Comparison

Figure 13 shows the projected cases for disease 3. Since there has not been recent widespread susceptibility to the measles, there is no current data to compare this projection to. Since the measles, without widespread vaccination, is incredibly devastating and confers life-long immunity [2] upon survival, the projection is entirely reasonable. However, this projection is only relevant as a control, as the aim of the modified system was to document how imperfect immunity affects case projections.

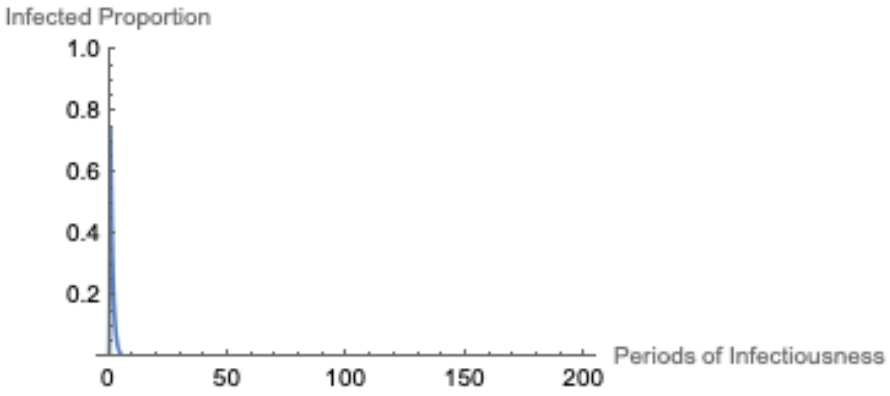

Figure 13: Projected Cases Using Approximate Measles Parameters

4.2 Key Insights, Failures, and Final Thoughts

Through only two direct comparisons between projected cases and actual disease cases, glaring failures of the model become evident. As Figures 10 and 11 show, the model predicts that each peak in cases will be smaller than the last. This is directly linked to the eigenvalues of the system, which, as we identified in Section 3.2.2, will always be complex eigenvalues with a negative real part for any system with imperfect immunity and $R_0 > 1$. The general solution of a system with $\lambda = p \pm qi$, with $\bar{v}$ as its associated complex eigenvector, is $\bar{x} = c_1\bar{x_1}(t) + c_2\bar{x_2}(t)$ where:

$$\bar{x_1}(t) = Re[\bar{v}(cos(qt) + isin(qt))e^{pt}] \tag{25}$$

$$\bar{x_2}(t) = Im[\bar{v}(cos(qt) + isin(qt))e^{pt}] \tag{26}$$

since p represents the real part of the eigenvalue, p is negative for our system, therefore e^{pt} produces an exponential dampening effect where each peak will get unavoidably smaller. While the *sin* and *cos* portions give rise to the oscillation that we see in diseases exhibiting seasonality, the negative dampening simply does not match actual case data, and this is the largest failure of the model.

There are several reasons for the failure of the model. While "SIR" models are traditionally used in the presence of a new disease to model future outcomes, the model fails to account for pathogen evolution that outpaces the rate at which immunity wanes. Further, evolution is generally unpredictable, so making this adjustment would be difficult to do with traditional parameters. The model also treats the parameter θ as having equal weight as the tried and true crucial parameters ϵ and ϕ, which may result in an overestimation of the impact that waning immunity has on disease spread and case projections. The chosen diseases used for data comparison are also incredibly complex, with the presence of numerous pathogen variants and the introduction of vaccines and global health measures interrupting the supposedly closed system. Further, the model ignores births, which would naturally increase the susceptible population over time in situations such as Figure 13, allowing for potential disease re-emergence even in the case of perfect immunity.

In all, we can conclude that the coefficient of waning immunity significantly altered the stability of the system and did give some indication of system stability with disease present, but ultimately failed to be applicable to any real-world scenario or congruent with real-world data, thus the modified Kermack-McKendrick model proposed herein would need many more modifications to be useful. Waning immunity on its own is not enough to model seasonal oscillations via complex eigenvalues, and waning immunity does not account for the rapid pace of pathogen evolution seen in modern epidemics.

References

[1] Key facts about influenza (flu). *Centers for Disease Control and Prevention*, Oct 2022.

[2] Francesco Paolo Bianchi, Simona Mascipinto, Pasquale Stefanizzi, Sara De Nitto, Cinzia Germinario, and Silvio Tafuri. Long-term immunogenicity after measles vaccine vs. wild infection: An italian retrospective cohort study. *Human Vaccines amp; Immunotherapeutics*, 17(7):2078–2084, 2021.

[3] Matthew Biggerstaff, Simon Cauchemez, Carrie Reed, Manoj Gambhir, and Lyn Finelli. Estimates of the reproduction number for seasonal, pandemic, and zoonotic influenza: A systematic review of the literature. *BMC Infectious Diseases*, 14(1), 2014.

[4] Centers for Disease Control and Prevention. Science brief: Sars-cov-2 infection-induced and vaccine-induced immunity, Oct 2021.

[5] Centers for Disease Control and Prevention. Cdc covid data tracker, Apr 2023.

[6] Centers for Disease Control, Prevention, National Center for Immunization, and Respiratory Diseases. Past seasons estimated influenza disease burden, Oct 2022.

[7] Fiona M Guerra, Shelly Bolotin, Gillian Lim, Jane Heffernan, Shelley L Deeks, Ye Li, and Natasha S Crowcroft. The basic reproduction number (r 0) of measles: A systematic review. *The Lancet Infectious Diseases*, 17(12), Dec 2017.

[8] James Holland Jones. Notes on r0. *Stanford University*, May 2007.

[9] William Ogilvy Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772):700–721, 1927.

[10] Ying Liu, Albert A Gayle, Annelies Wilder-Smith, and Joacim Rocklöv. The reproductive number of covid-19 is higher compared to sars coronavirus. *Journal of Travel Medicine*, 27(2), Mar 2020.

[11] Steven H. Strogatz. *Nonlinear dynamics and chaos*. Westview Press, Boulder, CO, second edition, 2015. With applications to physics, biology, chemistry, and engineering.

[12] Jeroen J. van Kampen, David A. van de Vijver, Pieter L. Fraaij, Bart L. Haagmans, Mart M. Lamers, Nisreen Okba, Johannes P. van den Akker, Henrik Endeman, Diederik A. Gommers, Jan J. Cornelissen, and et al. Duration and key determinants of infectious virus shedding in hospitalized patients with coronavirus disease-2019 (covid-19). *Nature Communications*, 12(1), 2021.