THE EFFECT OF COVID-19 MITIGATION STRATEGIES ON THE SPREAD OF THE INFLUENZA VIRUS

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Abstract

The use of non-pharmaceutical interventions to control the spread of COVID-19, such as mask mandates and social distancing orders, will likely affect the spread of other infections. We develop a compartmental SVEIRS model with four stage progressions for the infectious states to study the effect of mask wearing on the spread of influenza viruses. Our model confirms the Centers for Disease Control and Prevention recommendations against mask wearing for individuals with symptoms of the flu as an effective preventative measure in lieu of vaccinations during normal circumstances. We also show that universal mask wearing, regardless of symptoms, could be sufficient to prevent an influenza epidemic during the COVID-19 pandemic, even in the absence of influenza vaccinations.

1. INTRODUCTION

During any pandemic, especially the COVID-19 pandemic, it is important not to forget that the seasonal flu will continue to circulate. Although it's difficult to predict the severity of the flu in any given year, experts warn that an active influenza season could put additional strain on hospital resources that are desperately needed to deal with COVID-19 infections [8]. In the southern hemisphere, which usually experiences it's peak influenza season July through October, the 2020 influenza season was not as severe as in past years; even in countries with weak COVID-19 mitigation policies, the 2020 influenza season was lighter than normal. In Australia, where there are high rates of social distancing and mask compliance, the 2020 influenza season was exceptionally light, with only 315 reported cases of the flu, down 99.8% from average levels [8]. This suggests that compliance with COVID-19 specific mitigation strategies, such as stay-at-home orders, social distancing, and mask mandates, is sufficient to prevent an influenza epidemic.

Typically, the flu vaccine is the primary intervention to control the spread of influenza viruses and in normal circumstances it is more cost effective than stay-at-home orders, such as those issued for the COVID-19 pandemic. However, vaccination is more costly than a mask mandate so we would like to study the effects of mask wearing on the spread of influenza viruses more closely. For example, would mandating wearing a mask outside of the household for 7 days after symptoms of the flu start, regardless of when symptoms disappear, be a viable recommendation by health experts against the spread of the flu, especially in absence of a flu vaccine. Unfortunately, our model shows that such a mask mandate may not be sufficient to prevent an influenza epidemic for especially virulent strains of the influenza virus which is consistent with current Centers for Disease Control and Prevention (CDC) guidelines [1]. We would also like to know if a universal mask mandate for the COVID-19 pandemic, or any pandemic, would be sufficient to prevent an influenza epidemic, possibly even in the absence of a flu vaccine. Our model shows that a universal mask mandate could be sufficient and may explain the unusually light 2020 influenza season in the southern hemisphere.

2. FORMULATION OF THE MODEL

In order to model the spread of the influenza viruses we will develop a compartmental based SVEIRS model with four stage progressions for the infected population. In our model, we introduce a vaccinated population which will be immune from influenza infection for a period of time, eventually returning to the susceptible population. This waning of vaccine immunity can be interpreted as the change in circulating influenza strains each year which are assumed not to be covered by the previous year's vaccine. We also use a latent state incubation period and stage progression for the infected states which will allow us to vary the contacts and infectivity over the course of illness. This flexibility will allow us to incorporate behavior change and understand more carefully how mask wearing, particularly in the later stages of the disease, when most people will have returned to work or school, will affect the spread of influenza causing viruses. Finally, we will allow recovered individuals to become susceptible again which is also a result of the change in viral strains from year to year.

2.1 Assumptions

We will have to make assumptions about our populations to build our model. In our model, we will assume that once an individual is exposed to the virus they will go through an incubation period before becoming infectious. We also will assume vaccination offers 100% immunity from the influenza virus, but only for a period of time after which a vaccinated individual will return to the susceptible population. When an individual progresses to the first infectious state they are not symptomatic but are now slightly infectious. The second stage of infection will be the peak of the illness when malaise has forced a behavior change to reduce the number of contacts and the person is at their peak infectivity. In the third stage of illness, malaise has begun to recede and the individual will start to increase their contacts as their infectivity decreases. In the last stage of infection, the individual may have little to no symptoms, will have returned to normal contact levels, and will still be slightly infectious.

We will also have a parameter in our model that will account for a reduction in infectivity if a mask is worn by an infectious individual. We will incorporate mask wearing into our model under two different circumstances. The first circumstance will be referred to as a pandemic situation. In a pandemic situation it will be assumed that mask wearing is universal due to the mask mandates for the pandemic of a different disease. In this case, individuals will wear masks around their contacts throughout the course of their illness, regardless of their symptoms. This will include mask wearing around individuals in their household when staying home for the second infectious stage when malaise is at a peak, since in a pandemic individuals will assume that their illness is the disease of the pandemic and they will take extra precautions to prevent the spread of that disease. The second circumstance will be referred to as the typical situation or pandemic free situation. In this case we will want to understand the effect of a mask mandate for those infected with the flu. Since an individual may not know they have the flu until symptoms

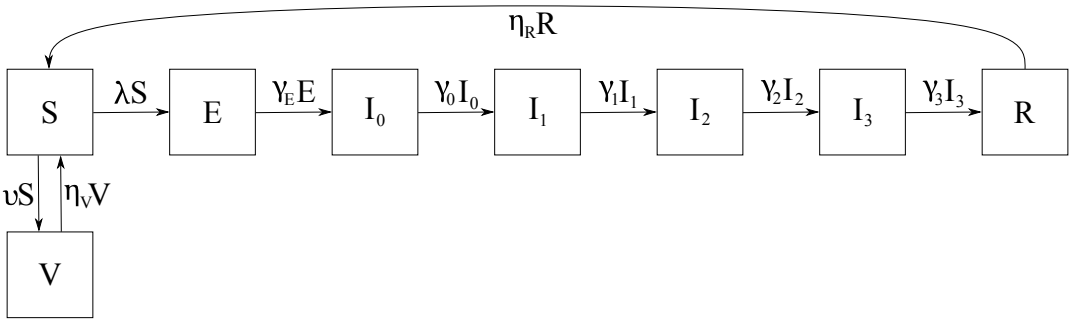


Figure 1: Block diagram for the SVEIRS model with four stage progressions for the infectious population. In this model, we see that vaccinated or recovered individuals are only temporarily removed from the susceptible population.

begin, they will not be wearing masks for the first infectious stage. And we will assume the mask mandate can only be enforced outside of the household, so individuals will not wear masks while in the second infectious stage due to them staying home for malaise.

In the recovered stage individuals will have immunity from infection, however this immunity will eventually end and they will reenter the susceptible population.

2.2 Parameters

Now we will quantify our assumptions by defining parameters to account for them in our equations. Table 1 summarizes each parameter. We start by dividing our population into eight groups. The first group is the susceptible population, denoted S. Then we have the latent population which we denote E and the vaccinated population denoted V, which is immune from infection. There are four stages of infected individuals denoted I_0, I_1, I_2, and I_3. Finally there is a recovered population which is also immune from infection, denoted R.

Susceptible people become exposed to the virus and leave the susceptible population to join the latent population at the rate

$$\sum \alpha_i I_i$$

where α_i describes the force from infection for the infectious stage I_i. This term can be broken down into three components,

$$\alpha_i = C_i \beta_i P_S,$$

where C_i is the number of contacts per person in I_i per day, β_i is the probability a person in I_i will transmit the virus to a contact i.e. the infectivity of a person in I_i, and P_S is the probability that a contact is susceptible. Together, we can interpret α_i as

$$\alpha_i = \begin{pmatrix}\text{Average number of}\\ \text{contacts per person}\\ \text{in } I_i \text{ per day}\end{pmatrix} \times \begin{pmatrix}\text{Probability that a}\\ \text{person in } I_i\\ \text{transmits the virus}\\ \text{to a contact}\end{pmatrix} \times \begin{pmatrix}\text{Probability that}\\ \text{a contact is}\\ \text{susceptible}\end{pmatrix}.$$

We further break down the parameters β_i and P_S to provide more clarity for our model. For our infectivity parameter we have

$$\beta_i = \begin{cases} b_i & \text{if } i = 0,1, \\ (1-\rho)b_i & \text{if } i = 2,3. \end{cases}$$

The probability of transmission of the influenza virus in stage I_i without intervention is given by b_i and ρ represents the mask coefficient. When there is no mask wearing we set $\rho = 0$, otherwise ρ represents the efficiency of a mask to prevent the transmission of the influenza virus. In a pandemic situation we will substitute the parameter β_{P_i} for β_i, and this will be the infectivity of a person in I_i during the pandemic situation. We define

$$\beta_{P_i} = (1-\rho)b_i$$

for all infectious stages to represent universal mask wearing.

The parameter P_S represents the probability that a contact is susceptible and we define P_S in terms of the relative population sizes and contacts so that P_S will vary over time. We define

$$P_S = \frac{C_N S}{N_C}$$

where C_N is the number of contacts per person per day in the absence of illness. Thus C_N describes the number of contacts per day for a person in either S, V, E, or R, and

$$N_C = C_N S + C_N V + C_N E + C_0 I_0 + C_1 I_1 + C_2 I_2 + C_3 I_3 + C_N R$$

is the total number of contacts per day for the entire population.

The force from infection, $\alpha_i$contains the transmissibility parameter which is defined as $C_i \beta_i$ for each I_i. This parameter describes the overall impact on transmission each stage has by taking into consideration both the number of contacts and the infectivity. Since we want to vary C_i and β_i by infectious stage it is important to be have the notion of transmissibility since it will allow us to compare the rate of transmission of the virus by each stage taking both contacts and infectivity into account. We would expect to see lower transmissibility, for example, during the peak of the illness since although infectivity is highest during stage I_1, the number of contacts is very low. In contrast, during the first stage of infection we might expect to have a higher transmissibility. Despite the infectivity being low, there are no symptoms present in the first stage of infection so the individual has no behavior change and a normal amount of contacts per day.

We can also describe the rate at which individuals become exposed to the virus and leave the susceptible population from the force of infection perspective, λ. The force of infection can be equated to α_i by

$$\lambda S = \sum \alpha_i I_i.$$

Similarly, we can break down the force of infection, λ, as

$$\lambda = \sum C_N \beta_i P_i$$

where C_N is again the number of contacts per person per day for a non infected individual, β_i is the infectivity of a person in I_i, and P_i is now the probability that a contact of a susceptible person is in the infectious stage I_i. Heuristically we can think of the force of infection as

$$\lambda = \begin{pmatrix} \text{Average number of} \\ \text{contacts per person} \\ \text{in } S \text{ per day} \end{pmatrix} \times \begin{pmatrix} \text{Probability that a} \\ \text{person in } I_i \\ \text{transmits the virus} \\ \text{to a contact} \end{pmatrix} \times \begin{pmatrix} \text{Probability that} \\ \text{a contact is} \\ \text{infectious in} \\ \text{stage } I_i \end{pmatrix}.$$

Again, the appropriate substitution of β_{P_i} can be made for β_i in a pandemic situation.

Now we return to the movement of people through the susceptible population. Besides leaving the susceptible population after becoming exposed to the virus, an individual can leave the susceptible population through vaccination which will happen at rate ν relative to the size of the susceptible population. People can reenter the susceptible population in one of two ways. The first way is when a recovered individual's immunity wears off. We quantify the average number of days that a person is immune from infection after recovering from illness as the parameter ζ_R. Assuming a normal distribution on this average time ζ_R we can define

$$\eta_R = \frac{1}{\zeta_R}$$

as the average rate at which a person becomes susceptible again after recovering from illness.

The second way to enter the susceptible population is to lose immunity from the vaccine. This happens on average after ζ_V days. The rate at which vaccinated individuals return to the susceptible population is

$$\eta_V = \frac{1}{\zeta_V}.$$

We can completely describe the movement of individuals through both the susceptible and vaccinated states now. The change with respect to time of the susceptible population is given by the equation

$$\dot{S} = \eta_R R + \eta_V V - \alpha_0 I_0 - \alpha_1 I_1 - \alpha_2 I_2 - \alpha_3 I_3 - \nu S \tag{1}$$

or

$$\dot{S} = \eta_R R + \eta_V V - \lambda S - \nu S. \tag{2}$$

The change with respect to time for the vaccinated population is

$$\dot{V} = \nu S - \eta_V V. \tag{3}$$

Table 1: Table of parameters used to describe the system of differential equations. Each parameter is accompanied by a heuristic description and its dimensions.

Parameter	Description	Dimensions
	Variables	
$S(t)$	Susceptible population at time t.	people
$V(t)$	Vaccinated population at time t.	people
$E(t)$	Latent stage population at time t.	people
$I_0(t)$	Infectious stage 0 population at time t.	people
$I_1(t)$	Infectious stage 1 population at time t.	people
$I_2(t)$	Infectious stage 2 population at time t.	people
$I_3(t)$	Infectious stage 3 population at time t.	people
$R(t)$	Immune population at time t.	people
$T(t)$	Incidence at time t.	people
	Independent Parameters	
b_i	Infectivity of a person in I_i.	people contacts^{-1}
ρ	Mask coefficient.	dimensionless
C_i	Average number of contacts per day per person in I_i.	contacts people^{-1} day^{-1}
C_N	Average number of contacts per day per person in S, V, E or R.	contacts people^{-1} day^{-1}
τ_i	Average time spent in infectious stage I_i.	days
τ_E	Average time spent in latent state E.	days
ζ_R	Average time a recovered person is immune from infection.	days
ζ_V	Average time a vaccinated person is immune from infection.	days
ν	Rate at which the vaccine is administered.	day^{-1}
	Dependent parameters	
$I(t)$	Prevalence of infection at time t. $I(t) = E(t) + I_0(t) + I_1(t) + I_2(t) + I_3(t)$.	people
$N(t)$	Total population. $N(t) = S(t) + V(t) + E(t) + I_0(t) + I_1(t) + I_2(t) + I_3(t) + R(t)$.	people
N_C	Total contacts per day. $N_C = C_N S + C_N V + C_N E + C_0 I_0 + C_1 I_1 + C_2 I_2 + C_3 I_3 + C_N R$.	contacts day^{-1}
P_S	Probability that a contact is susceptible. $P_S = \frac{C_N S}{N_C}$.	dimensionless
P_i	Probability that a contact is in infectious stage I_i. $P_i = \frac{C_i I_i}{N_C}$.	dimensionless
β_i	Infectivity of a person in I_i. $\beta_i = b_i$ for $i = 0, 1$ and $\beta_i = (1-\rho) b_i$ for $i = 2, 3$.	people contacts^{-1}
β_{P_i}	Infectivity of a person in I_i during a pandemic situation. $\beta_{P_i} = (1-\rho) b_i$.	people contacts^{-1}
α_i	Force from infection for infectious stage I_i to the susceptible population. $\alpha_i = C_i \beta_i P_S$.	day^{-1}
λ	Force of infection for the susceptible population. $\lambda = \sum C_N \beta_i P_i$.	day^{-1}
γ_i	Average rate of leaving infectious stage I_i. $\gamma_i = \frac{1}{\tau_i}$.	day^{-1}
γ_E	Average rate of leaving latent stage E. $\gamma_E = \frac{1}{\tau_E}$.	day^{-1}
η_R	Average rate of becoming susceptible again after recovering from infection. $\eta_R = \frac{1}{\zeta_R}$.	day^{-1}
η_V	Average rate of becoming susceptible again after vaccination. $\eta_V = \frac{1}{\zeta_V}$.	day^{-1}

Next, let's look at the movement of individuals through the infectious stages. The average time spent in each stage I_i is given by τ_i and hence the average rate at which people leave stage I_i, again assuming normal distribution, is

$$\gamma_i = \frac{1}{\tau_i}.$$

Similarly, the average time spent in the latent stage E is τ_E and the average rate of leaving E is

$$\gamma_E = \frac{1}{\tau_E}.$$

So, we can write the equations describing the rate of change for the latent stage and each infectious stage as

$$\dot{E} = \alpha_0 I_0 + \alpha_1 I_1 + \alpha_2 I_2 + \alpha_3 I_3 - \gamma_E E \tag{4}$$
$$\dot{I}_0 = \gamma_E E - \gamma_0 I_0 \tag{5}$$
$$\dot{I}_1 = \gamma_0 I_0 - \gamma_1 I_1 \tag{6}$$
$$\dot{I}_2 = \gamma_1 I_1 - \gamma_2 I_2 \tag{7}$$
$$\dot{I}_3 = \gamma_2 I_2 - \gamma_3 I_3. \tag{8}$$

Finally, we need to discuss the change in the recovered population. The average rate at which people enter the recovered or immune state is $\gamma_3 I_3$. Recalling ζ_R and η_R from above, we can describe the change in the recovered population with the equation

$$\dot{R} = \gamma_3 I_3 - \eta_R R. \tag{9}$$

2.3 System of equations

Now we can write each of the equations representing the changes in each population with respect to time as a system of differential equations:

$$\dot{S} = \eta_R R + \eta_V V - \alpha_{S_0} I_0 - \alpha_{S_1} I_1 - \alpha_{S_2} I_2 - \alpha_{S_3} I_3 - \nu S = \eta_R R + \eta_V V - (\lambda + \nu) S \tag{10a}$$
$$\dot{V} = \nu S - \eta_V V \tag{10b}$$
$$\dot{E} = \alpha_0 I_0 + \alpha_1 I_1 + \alpha_2 I_2 + \alpha_3 I_3 - \gamma_E E = \lambda S - \gamma_E E \tag{10c}$$
$$\dot{I}_0 = \gamma_E E - \gamma_0 I_0 \tag{10d}$$
$$\dot{I}_1 = \gamma_0 I_0 - \gamma_1 I_1 \tag{10e}$$
$$\dot{I}_2 = \gamma_1 I_1 - \gamma_2 I_2 \tag{10f}$$
$$\dot{I}_3 = \gamma_2 I_2 - \gamma_3 I_3 \tag{10g}$$
$$\dot{R} = \gamma_3 I_3 - \eta_R R. \tag{10h}$$

Figure 1 summarizes this model.

To help us understand the behavior of our system better we also want to define a few diagnostic equations. First we have the total population, given by N,

$$N = S + V + E + I_0 + I_1 + I_2 + I_3 + R. \tag{11}$$

Since we are not assuming any migration in or out of the population system, $\dot{N} = 0$ always. The total population can also be useful for making sure that our initial conditions are balanced. For our initial conditions we have the values

$$S(0), V(0), E(0), I_0(0), I_1(0), I_2(0), I_3(0), R(0)$$

which means that when our population is normalized we need to have

$$1 = N(0) = S(0) + V(0) + E(0) + I_0(0) + I_1(0) + I_2(0) + I_3(0) + R(0).$$

We will denote $N(0)$ as N^0 sometimes. Next, we have the equation that describes the change in incidence over time

$$\dot{T} = \alpha_0 I_0 + \alpha_1 I_1 + \alpha_2 I_2 + \alpha_3 I_3. \tag{12}$$

The incidence can be thought of as the cumulative number of infections at any given time. Since we have a closed population system and we assume that individuals can be reinfected, our incidence can grow larger than the total population as time increases. Finally, we have an equation describing the prevalence of the infection, or the current total of infected individuals, at any given time:

$$I = E + I_0 + I_1 + I_2 + I_3. \tag{13}$$

We can also write our initial conditions in terms of the prevalence, giving us

$$S(0) = N(0) - V(0) - I(0) - R(0).$$

3. Interpretation of the model

Now that we have established our model we wish to analyze the characteristics of its behavior. We will do this using two important concepts for describing a system of equations that models the spread of a disease: the reproductive number and the steady states.

3.1 Reproductive Number

The reproductive number is a very useful indicator of the spread of a disease. The reproductive number is defined as the average number of secondary infections. We typically are interested in two versions of the reproductive number. The *basic* reproductive number, $\mathscr{R}_0$, indicates the growth of a disease at the very beginning of an epidemic when the disease is introduced into a fully, or nearly fully, susceptible population. In other words, in the basic reproductive number we set the parameter $P_S = 1$. The second version of the reproductive number is the *effective* reproductive number, $\mathscr{R}_e$.The effective reproductive number indicates that spread of a disease in the middle or end stages of an epidemic when $P_S < 1$.

We can use the reproductive number to analyze the potential for growth in an epidemic by considering what the value of the reproductive number means. Since the reproductive number tells us the average number of secondary infections we can see that if the reproductive number is less than 1, then on average every infected person is infecting less than one other person so the disease will eventually die out. On the other hand, if the reproductive number is larger than 1, then every infected individual is infecting on average more than one other individual so the disease will grow into an epidemic. If the reproductive number is equal to 1 we consider the disease to be in a steady state or equilibrium where the number of current infections is neither growing nor decreasing.

The basic reproductive number is always larger than the effective reproductive number, making it a useful tool to analyze the effectiveness of mitigation and treatment strategies when the susceptibility of the population is unknown. In other words if an intervention reduces the basic reproductive number to less than 1, then regardless of the susceptibility of the population, the effective reproductive number will also be less than one.

We will first derive the reproductive number for our model heuristically. We see that α_i, which has dimensions day^{-1}, describes the number of new infections per day from a person in I_i. So, to get the total number of new infections from a person in I_i over the time spent in I_i we multiply α_i by τ_i. To account for the total new infections caused by a person as they move through each of the infectious stages we have the count

$$\mathscr{R}_e = \sum \alpha_i \tau_i \tag{14}$$

as the average number of new infections caused by a single infected person.

We can take the effective reproductive number with α_i broken into each of its terms to get

$$\mathscr{R}_e = \sum C_i \beta_i P_S \tau_i. \tag{15}$$

Then setting $P_S = 1$ in (15) gives us the basic reproductive number for our model,

$$\mathscr{R}_0 = \sum C_i \beta_i \tau_i \tag{16}$$

To be sure that we have derived the correct reproductive number we can use the Next Generation Method to construct the effective reproductive number. To do this we start be constructing matrices $\dot{X}$, $F(X)$, and $V(X)$, where $\dot{X}$ represents the change in the infectious populations, $F(X)$ is the new infections entering the system, and $V(X)$ is the progressions between infectious populations, such that $\dot{X} = F(X) - V(X)$. Let

$$\begin{pmatrix} \dot{E} \\ \dot{I_0} \\ \dot{I_1} \\ \dot{I_2} \\ \dot{I_3} \end{pmatrix} = \begin{pmatrix} \alpha_0 I_0 + \alpha_1 I_1 + \alpha_2 I_2 + \alpha_3 I_3 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} \gamma_E E \\ -\gamma_E E + \gamma_0 I_0 \\ -\gamma_0 I_0 + \gamma_1 I_1 \\ -\gamma_1 I_1 + \gamma_2 I_2 \\ -\gamma_2 I_2 + \gamma_3 I_3 \end{pmatrix}$$

be these matrices. Next we take the Jacobian of F and V to get

$$J_F = \begin{pmatrix} 0 & \alpha_0 & \alpha_1 & \alpha_2 & \alpha_3 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$J_V = \begin{pmatrix} \gamma_E & 0 & 0 & 0 & 0 \\ -\gamma_E & \gamma_0 & 0 & 0 & 0 \\ 0 & -\gamma_0 & \gamma_1 & 0 & 0 \\ 0 & 0 & -\gamma_1 & \gamma_2 & 0 \\ 0 & 0 & 0 & -\gamma_2 & \gamma_3 \end{pmatrix}.$$

Our Next Generation Matrix, NGM, is defined as $NGM = J_F J_V^{-1}$, so we need the inverse of J_V:

$$J_V^{-1} = \begin{pmatrix} \frac{1}{\gamma_E} & 0 & 0 & 0 & 0 \\ \frac{1}{\gamma_0} & \frac{1}{\gamma_0} & 0 & 0 & 0 \\ \frac{1}{\gamma_1} & \frac{1}{\gamma_1} & \frac{1}{\gamma_1} & 0 & 0 \\ \frac{1}{\gamma_2} & \frac{1}{\gamma_2} & \frac{1}{\gamma_2} & \frac{1}{\gamma_2} & 0 \\ \frac{1}{\gamma_3} & \frac{1}{\gamma_3} & \frac{1}{\gamma_3} & \frac{1}{\gamma_3} & \frac{1}{\gamma_3} \end{pmatrix}.$$

Finally we have

$$NGM = \begin{pmatrix} \frac{\alpha_0}{\gamma_0} + \frac{\alpha_1}{\gamma_1} + \frac{\alpha_2}{\gamma_2} + \frac{\alpha_3}{\gamma_3} & \frac{\alpha_0}{\gamma_0} + \frac{\alpha_1}{\gamma_1} + \frac{\alpha_2}{\gamma_2} + \frac{\alpha_3}{\gamma_3} & \frac{\alpha_1}{\gamma_1} + \frac{\alpha_2}{\gamma_2} + \frac{\alpha_3}{\gamma_3} & \frac{\alpha_2}{\gamma_2} + \frac{\alpha_3}{\gamma_3} & \frac{\alpha_3}{\gamma_3} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

The reproductive number is the dominant eigenvalue of the Next Generation Matrix, which is clearly

$$\mathscr{R}_e = \frac{\alpha_0}{\gamma_0} + \frac{\alpha_1}{\gamma_1} + \frac{\alpha_2}{\gamma_2} + \frac{\alpha_3}{\gamma_3}.$$

Given that $\gamma_i = \frac{1}{\tau_i}$, we get

$$\mathscr{R}_e = \alpha_0\tau_0 + \alpha_1\tau_1 + \alpha_2\tau_2 + \alpha_3\tau_3,$$

which confirms our heuristic interpretation of the reproductive number.

We can use the reproductive number to analyze the variable parameters in our model. For example, P_S is a factor in the force from infection α_i and we can write

$$\mathscr{R}_e = P_S\left(\sum C_i\beta_i\tau_i\right) = S \cdot \frac{C_N}{N_C}\left(\sum C_i\beta_i\tau_i\right)$$

which shows us that a decrease in the number of susceptible people either through infection or vaccination will proportionality decrease the effective reproductive number. If we breakdown our reproductive number in terms of β_i we get

$$\mathscr{R}_e = C_0(b_0)P_S\tau_0 + C_1(b_1)P_S\tau_1 + C_2(b_2(1-\rho))P_S\tau_2 + C_3(b_3(1-\rho))P_S\tau_3$$

for the pandemic free situation and

$$\mathscr{R}_e = C_0(b_0(1-\rho))P_S\tau_0 + C_1(b_1(1-\rho))P_S\tau_1 + C_2(b_2(1-\rho))P_S\tau_2 + C_3(b_3(1-\rho))P_S\tau_3$$

for the pandemic situation. We see that an increase in wearing masks and increasing the efficiency of masks, ρ, will correspond to a decrease in the reproductive number in both scenarios, but its effect will be larger in the pandemic scenario.

3.2 Steady state

Now that we have developed the reproductive number to help us understand the beginning and middle stages of an epidemic, we need to examine the steady state behavior of our system to understand the end stages of our epidemic. In particular, our system has two equilibrium states, the disease free equilibrium and the endemic equilibrium.

The disease free equilibrium is straightforward. Without infections, the only movement we will see is in the vaccination of susceptible individuals and in the loss of vaccination immunity. Setting each of the time derivatives equal to 0 and latent, infectious, and recovered populations to zero, we are left with

$$0 = \dot{S} = \eta_V V - \nu S$$
$$0 = \dot{V} = \nu S - \eta_V V.$$

This means our disease free equilibrium exists when

$$S = \frac{\eta_V V}{\nu} \tag{17a}$$
$$V = \frac{\nu S}{\eta_V} \tag{17b}$$
$$E = 0 \tag{17c}$$
$$I_0 = 0 \tag{17d}$$
$$I_1 = 0 \tag{17e}$$
$$I_2 = 0 \tag{17f}$$
$$I_3 = 0 \tag{17g}$$
$$R = 0. \tag{17h}$$

The endemic equilibrium is more complicated. When a disease is in an endemic state it means that while people are continuing to get vaccinated, infected, and recovered, there is no change in the size of the populations. At the endemic equilibrium $\mathscr{R}_e = 1$. To derive the necessary conditions for an endemic equilibrium we need to establish that $\dot{T}^* > 0$, $I^* > 0$, and $\dot{N} = 0$, where $*$ represents the size of the populations at the endemic equilibrium. If $\mathscr{R}_0 < 1$ then there will be no endemic state and the disease will die out. If $\mathscr{R}_0 > 1$ then we will solve for the endemic steady state by setting all derivatives equal to 0:

$$0 = \dot{S}^* = \eta_R R^* + \eta_V V^* - \alpha_0^* I_0^* - \alpha_1^* I_1^* - \alpha_2^* I_2^* - \alpha_3^* I_3^* - \nu S^* \tag{18a}$$
$$0 = \dot{V}^* = \nu S^* - \eta_V V^* \tag{18b}$$
$$0 = \dot{E}^* = \alpha_0^* I_0^* + \alpha_1^* I_1^* + \alpha_2^* I_2^* + \alpha_3^* I_3^* - \gamma_E E^* \tag{18c}$$
$$0 = \dot{I_0}^* = \gamma_E E^* - \gamma_0 I_0^* \tag{18d}$$
$$0 = \dot{I_1}^* = \gamma_0 I_0^* - \gamma_1 I_1^* \tag{18e}$$
$$0 = \dot{I_2}^* = \gamma_1 I_1^* - \gamma_2 I_2^* \tag{18f}$$
$$0 = \dot{I_3}^* = \gamma_2 I_2^* - \gamma_3 I_3^* \tag{18g}$$
$$0 = \dot{R}^* = \gamma_3 I_3^* - \eta R^*. \tag{18h}$$

We will use these equations to solve for our endemic equilibrium conditions. First, we add $\dot{S}^*$, $\dot{V}^*$ and $\dot{E}^*$ which gives us

$$\gamma_E E^* = \eta_R R^*$$
$$E^* = \tau_E \eta_R R^*. \tag{19}$$

Then, since $\dot{I_0}^* = 0$ we have,

$$\gamma_0 I_0^* = \gamma_E I_E^*$$
$$I_0^* = \frac{\tau_0}{\tau_E} I_E^*$$
$$I_0^* = \tau_0 \eta_R R^* \tag{20}$$

by substituting (19). Similarly, we have

$$\gamma_1 I_1^* = \gamma_0 I_0^*$$
$$I_1^* = \frac{\tau_1}{\tau_0} I_0^*$$
$$I_1^* = \tau_1 \eta_R R^*, \tag{21}$$

$$\begin{aligned}\gamma_2 I_2^* &= \gamma_1 I_1^* \\ I_2^* &= \frac{\tau_2}{\tau_1} I_1^* \\ I_2^* &= \tau_2 \eta_R R^*, \end{aligned} \tag{22}$$

and

$$\begin{aligned}\gamma_3 I_3^* &= \gamma_2 I_2^* \\ I_3^* &= \frac{\tau_3}{\tau_2} I_2^* \\ I_3^* &= \tau_3 \eta R^* \end{aligned} \tag{23}$$

by substituting (20), (21), and (22) respectively.

Next, we take $\dot{S} + \dot{V}$ which gives us

$$\begin{aligned}\eta_R R^* &= \sum \alpha_i^* I_i^* \\ \eta_R R^* &= \sum C_i \beta_i P_S^* I_i^*. \end{aligned}$$

We can substitute (20), (21), (22), (23) and (19), giving us

$$\begin{aligned}\eta_R R^* &= \sum C_i \beta_i P_S^* \tau_i \eta_R R^* \\ 1 &= \sum C_i \beta_i P_S^* \tau_i \\ 1 &= P_S^* \mathscr{R}_0 \\ 1 &= S^* \cdot \frac{C_N}{N_C^*} \mathscr{R}_0 \\ S^* &= \frac{N_C^*}{C_N \mathscr{R}_0} \\ S^* &= \frac{S^*}{\mathscr{R}_0} + \frac{V^*}{\mathscr{R}_0} + \frac{E^*}{\mathscr{R}_0} + \frac{\sum C_i I_i^*}{C_N \mathscr{R}_0} + \frac{R^*}{\mathscr{R}_0} \end{aligned} \tag{24}$$

$$S^* \left(1 - \frac{1}{\mathscr{R}_0}\right) = \frac{V^*}{\mathscr{R}_0} + R^* \left(\frac{\eta_R \tau_E}{\mathscr{R}_0} + \frac{\sum C_i \tau_i \eta_R}{C_N \mathscr{R}_0} + \frac{1}{\mathscr{R}_0}\right). \tag{25}$$

Finally, $\dot{V}^* = 0$ gives us

$$\begin{aligned}\nu S^* &= \eta_V V^* \\ S^* &= \frac{\eta_V V^*}{\nu}. \end{aligned} \tag{26}$$

From (24) we see that at the endemic equilibrium, $P_S^* \mathscr{R}_0 = \mathscr{R}_e = 1$, which we expected to be true.

To solve explicitly for the endemic equilibrium conditions we can take the linear relationships (19), (20), (21), (22), (23), (25), (26), and N^0 to form the matrix system $Ax = B$:

$$\begin{pmatrix} 0 & 0 & 1 & 0 & 0 & 0 & 0 & -\tau_E \eta_R \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & -\tau_0 \eta_R \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 & -\tau_1 \eta_R \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 & -\tau_2 \eta_R \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & -\tau_3 \eta_R \\ 1 - \frac{1}{\mathscr{R}_0} & -\frac{1}{\mathscr{R}_0} & 0 & 0 & 0 & 0 & 0 & -\left(\frac{\eta_R \tau_E}{\mathscr{R}_0} + \frac{\sum C_i \tau_i \eta_R}{C_N \mathscr{R}_0} + \frac{1}{\mathscr{R}_0}\right) \\ 1 & -\frac{\eta_V}{\nu} & 0 & 0 & 0 & 0 & 0 & 0 \\ 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \end{pmatrix} \begin{pmatrix} S^* \\ V^* \\ E^* \\ I_0^* \\ I_1^* \\ I_2^* \\ I_3^* \\ R^* \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ N^0 \end{pmatrix}.$$

Solving for x in MATLAB, we can state explicitly the endemic equilibrium conditions:

$$S^* = \eta_V \frac{G}{M} \tag{27a}$$
$$V^* = \nu \frac{G}{M} \tag{27b}$$
$$E^* = -\tau_E \frac{K}{M} \tag{27c}$$
$$I_0^* = -\tau_0 \frac{K}{M} \tag{27d}$$
$$I_1^* = -\tau_1 \frac{K}{M} \tag{27e}$$
$$I_2^* = -\tau_2 \frac{K}{M} \tag{27f}$$
$$I_3^* = -\tau_3 \frac{K}{M} \tag{27g}$$
$$R^* = -\zeta_R \frac{K}{M} \tag{27h}$$

where

$$M = \mathscr{R}_0 C_N \eta_V \left(1 + \eta_R \left(\sum \tau_i + \tau_E\right)\right) - C_N \eta_R \left(\sum \tau_i\right)\left(\eta_V + \nu\right) + \eta_R \left(\sum C_i \tau_i\right)\left(\eta_V + \nu\right)$$
$$G = N^0 \left(C_N + \eta_R \left(\sum C_i \tau_i + C_N \tau_E\right)\right)$$
$$K = N^0 C_N \eta_R \left(\eta_V + \nu - \mathscr{R}_0 \eta_V\right).$$

4. Computational analysis

Now that we have formulated our model and analyzed some of its properties we will run 3 year simulations of our model in MATLAB. The values for our parameters and initial conditions for our populations are summarized in Table 2.

In all our simulations we will consider two different estimated values for b_0 and b_3 which will determine our base line, or intervention free, basic reproductive number to account for the variation in the influenza reproductive number. According to [4], the influenza reproductive number can range from 0.9 to 2.1. To model an influenza epidemic that falls within this standard range we set $b_0 = b_3 = 0.01$. This will give a basic reproductive number of $\mathscr{R}_0 = 1.68$ for our model which sits in the middle of this range. We will consider this the low basic reproductive number scenario. However, some influenza strains, such as the 1918 pandemic strain, can have reproductive numbers up to 2.8 [4]. To model this scenario we take $b_0 = b_3 = 0.02$ which gives us a basic reproductive number of $\mathscr{R}_0 = 2.12$ for our model. We will consider this the high basic reproductive number scenario.

To start, we have the intervention free simulation. In this simulation we have $\nu = 0$ to represent the absence of vaccination intervention and we will be setting $\rho = 0$ to represent no mask wearing. Figure 2 shows the simulation. We see that having waning immunity from infection after recovering simulates a cyclic prevalence pattern which has periods of higher prevalence followed by periods of lower prevalence. As expected, the incidence eventually surpasses the total population as people can be infected more than once. Compared to the low basic reproductive number scenario, the high reproductive number scenario had higher incidence and the period of the prevalence waves is slightly shorter. Although 3 years is not sufficient to attain endemic status, we see that the model is headed in the direction of becoming endemic with positive change in incidence.

The first intervention we will analyze is vaccination. Figure 3 shows the simulation with only the vaccination intervention. Although vaccination does not influence the basic reproductive number we see that decreasing the susceptible population through vaccination reduces the effective reproductive number to less than 1 and the disease dies out. The main difference between the two basic reproductive number scenarios is that we have higher incidence in the high basic reproductive number scenario. As is consistent with CDC guidelines on influenza intervention strategies, vaccination is useful at containing an influenza epidemic [1].

The next intervention we want to analyze is the mask mandate in a pandemic free situation. In this simulation, which is shown in Figure 4, the population is mandated to wear masks outside of the household for 7 days after symptoms begin. Should the mask intervention be enough to reduce the basic reproductive number to below 1, as in the low basic reproductive number scenario, the disease will die out. In the high basic reproductive number scenario, where the mask intervention reduces the

Table 2: Table of initial conditions and parameter values used in MATLAB simulations. In our simulations we will consider a normalized population.

Parameter	Value	Reference
Initial Conditions		
$N^0 = N(0)$	1	normalized
$V(0), R(0)$	0	normalized
$E(0), I_0(0), I_1(0), I_2(0), I_3(0)$	0.002	normalized
$S(0) = N^0 - I(0) - V(0) - R(0)$	0.99	normalized
Parameters		
b_0	0.01, 0.02	estimated
b_1	0.09	[6]
b_2	0.05	estimated
b_3	0.01, 0.02	estimated
ρ	0.70	[3]
C_0	11	[7]
C_1	3	[5]
C_2	7	estimated
C_3	11	[7]
C_N	11	[7]
τ_E	2	[2]
τ_0	1	[2]
τ_1	2	[2]
τ_2	2	[2]
τ_3	3	[2]
ζ_R	240	[2]
ζ_V	240	[2]
ν	0.01	[9]

basic reproductive number but not to below 1, the disease will become endemic. However, compared to the intervention free scenario, the mask mandate in the high basic reproductive number scenario has a significantly lower incidence rate.

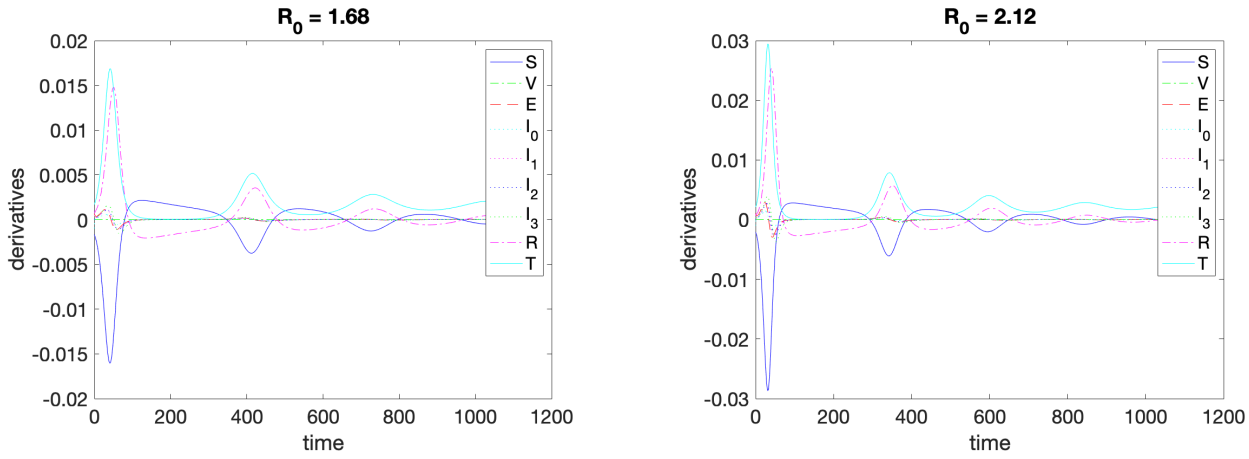
The final intervention we are interested in is a universal mask mandate in a pandemic situation. Figure 5 shows the effect of universal mask wearing, regardless of symptoms or illness. In both scenarios, universal mask wearing correlates to significant reductions of the basic reproductive number to less than 1. The disease dies out and the incidence remains low in these scenarios.

Now we want to look at the effect of combined interventions. The first combined intervention will be for a pandemic free situation. Figure 6 shows the effect of vaccination and a mask mandate. In both reproductive number scenarios, the disease dies out. However, we see that the use of vaccinations and masks when the basic reproductive number is high is sufficient to reduce the effective reproductive number to less than 1. This can be compared to the scenario with the mask only intervention, which was not sufficient to cause the disease to die out, and the vaccination only scenario, which was sufficient to prevent an epidemic. This is expected as the CDC does not recommend mask wearing as an intervention for the influenza virus in non-healthcare settings when there is a vaccination available [1].

The second combined intervention scenario we analyze is the use of vaccination and universal mask wearing in a pandemic situation. Figure 7 shows the combined effect of universal mask wearing and vaccination on the model. In either reproductive number scenario, the disease does not become epidemic because the basic reproductive number is less than 1. Compared to the simulation with only universal mask wearing, the effect on the model is not much different with the addition of vaccinations.

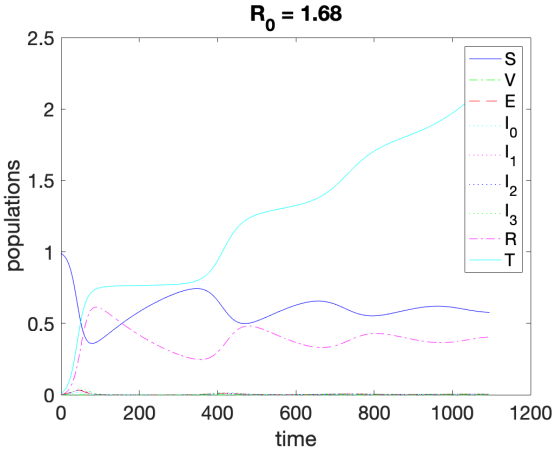
5. Conclusions

By developing an SVEIRS compartmental model for the influenza disease with multiple stage progressions for the infected populations we were able to analyze the effect of mask wearing as a possible intervention for preventing a flu epidemic in both a pandemic and pandemic free situation. First, we were able to confirm the basis of existing guidelines from the CDC on interventions for influenza. In particular, we showed in our model that vaccination is a very useful strategy to prevent an influenza epidemic, even for strains with high reproductive numbers. We were also able to show that in a pandemic free scenario, a mandate for wearing masks outside of the household for 7 days after the onset of symptoms may not be sufficient to

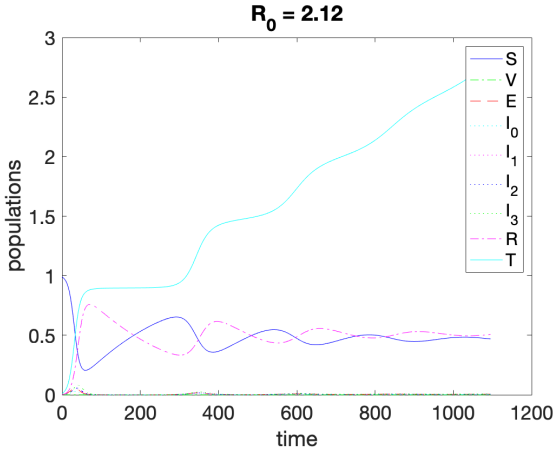


(a) Time derivatives for $b_0 = b_3 = 0.01$.

(b) Time derivatives for $b_0 = b_3 = 0.02$.



(c) Populations over time for $b_0 = b_3 = 0.01$.

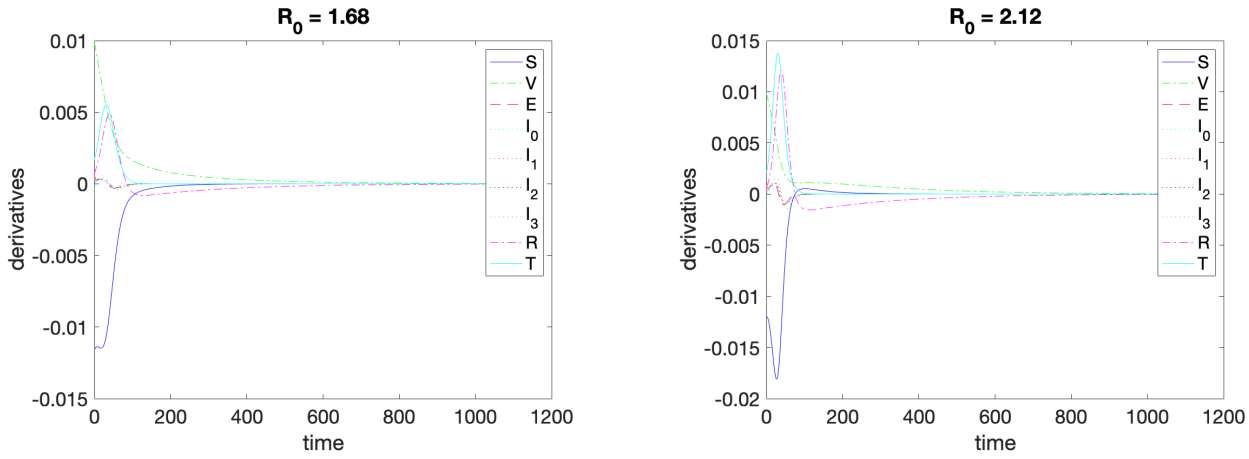


(d) Populations over time for $b_0 = b_3 = 0.02$.

Figure 2: Simulations of the model without vaccination or mask wearing interventions over 3 years using the values in Table 2. The loss of immunity after recovering from infection creates a cyclic pattern of prevalence. The disease has not yet reached the endemic state after 3 years for both basic reproductive number scenarios.

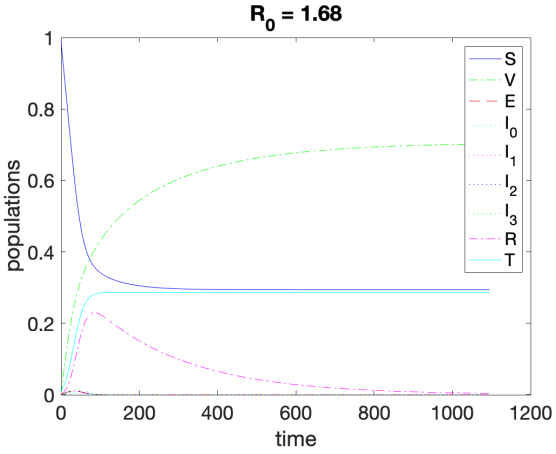
prevent an influenza epidemic in the absence of vaccinations. This is consistent with CDC guidelines on interventions to prevent the spread of influenza which do not recommend mask wearing and maintain vaccination as the primary intervention. However, in a pandemic situation, much like the COVID-19 pandemic, where there is universal mask wearing to prevent the spread of the pandemic disease, our model showed that universal mask wearing is sufficient to prevent an influenza epidemic in the absence of vaccination. Furthermore, combining vaccination intervention with universal mask wearing does not significantly change the outcome of the influenza model meaning that in such a pandemic situation, vaccination may not be necessary or cost efficient for preventing the spread of influenza.

Our model suggests our expected results and suggests that the interventions placed on Australia's population due to the COVID-19 pandemic may be responsible for the unusually low incidence of influenza during the 2020 season. However, the model cannot provide concrete evidence. For one, our model assumes that vaccination is 100% effective which is certainly not the case for the flu vaccine. There are lots of factors including time since vaccination and match of the vaccine flu strains to circulating strains that effect the overall efficacy of the flu vaccine at preventing influenza infection in the population. Estimates are that the influenza vaccine is typically 40-60% effective for the population in an average year [1]. Furthermore there are certain aspects of the influenza infection that were not incorporated into this model including the completely asymptomatic cases of the flu and the death rate for the disease. We also see that some assumptions about our population are idealistic and not necessarily representative of what actually happens. For one, the model includes no migration which occurs naturally in the population due to travel, moving, birth, death, etc.. Moreover, our model for vaccination may not accurately model the actual

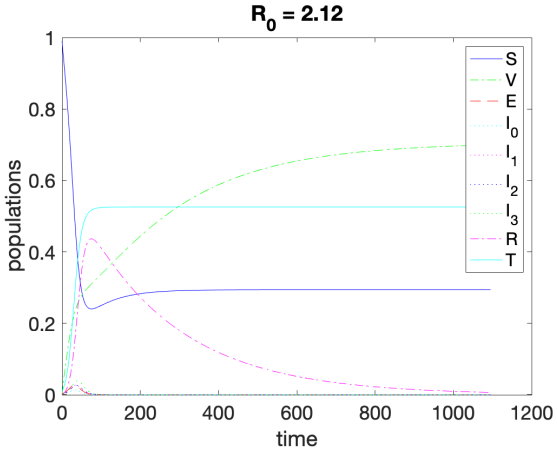


(a) Time derivatives for $b_0 = b_3 = 0.01$.

(b) Time derivatives for $b_0 = b_3 = 0.02$.



(c) Populations over time for $b_0 = b_3 = 0.01$.



(d) Populations over time for $b_0 = b_3 = 0.02$.

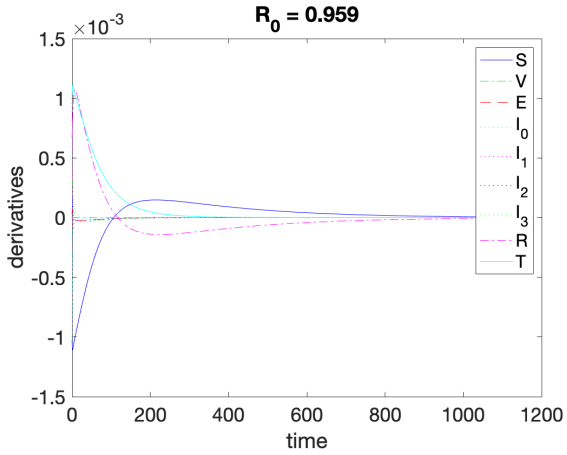
Figure 3: Simulations of the model with vaccination but no mask wearing interventions over 3 years using the values in Table 2. Reducing the susceptible population through vaccination is enough to cause the disease to die out eventually for both basic reproductive number scenarios.

vaccination of the population. Typically, most flu vaccines are administered in the fall within a span of a few months, not evenly distributed over the population over the entire year as in our model. While the waning of immunity in the model created a sense of seasonality, there are other more significant factors that affect the seasonality of the influenza virus so we cannot assume that seasonality is a component of our model.

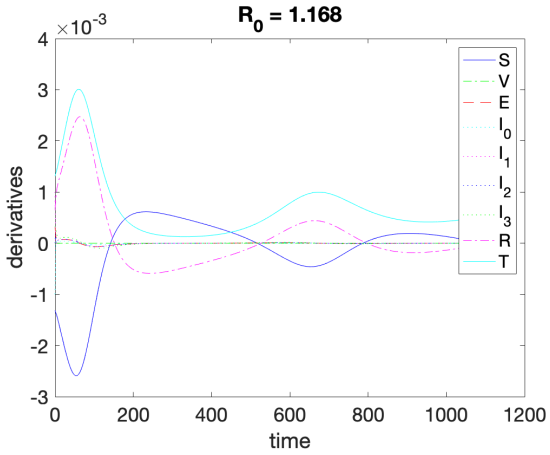
Since vaccinations are costly, it may be of interest for further study into the effectiveness of universal mask wearing on the influenza virus in the absence of vaccination as a potential cost saving measure during a pandemic situation such as the COVID-19 pandemic. Something to consider for such a scenario in the United States would be mask compliance rates. In the United States, mask compliance is a big issue for the COVID-19 pandemic and the rates of mask compliance may not be high enough to see large scale effects on the incidence of influenza in the United States. It might also be worth considering the effect of social distancing or reduction in contacts due to COVID-19 and how that would influence an influenza epidemic. Overall, based on the simulation of our model we might expect that mask mandate COVID-19 mitigation strategies will contribute to a decrease in influenza incidence during the northern hemisphere's 2020-2021 flu season.

6. Acknowledgments

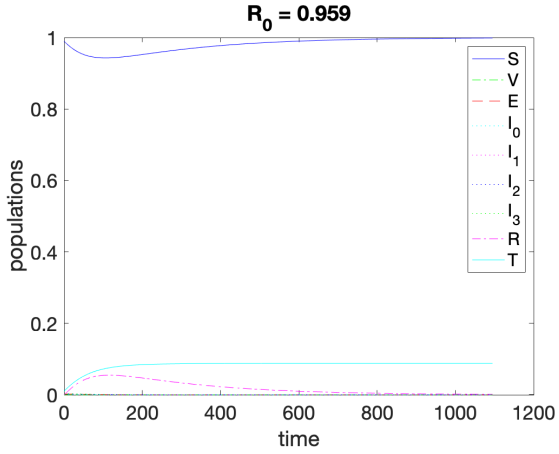
The first author would like to acknowledge the help of the second author on this project, particularly in helping to debug MATLAB code. They would like to thank the second author and Abigail Zion for all the correspondence, support, and advice over



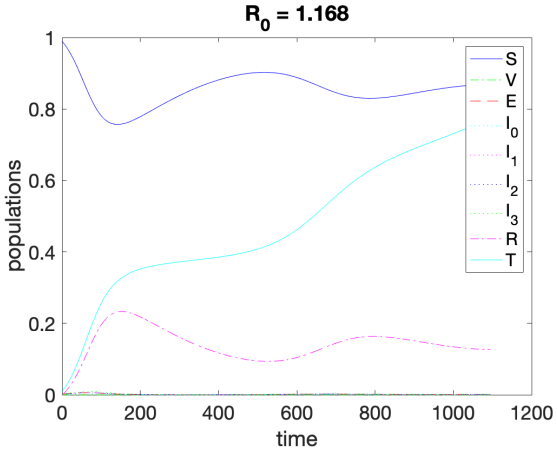
(a) Time derivatives for $b_0 = b_3 = 0.01$.



(b) Time derivatives for $b_0 = b_3 = 0.02$.



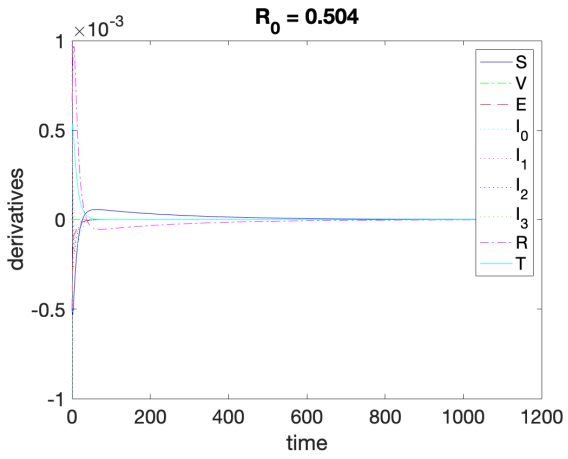
(c) Populations over time for $b_0 = b_3 = 0.01$.



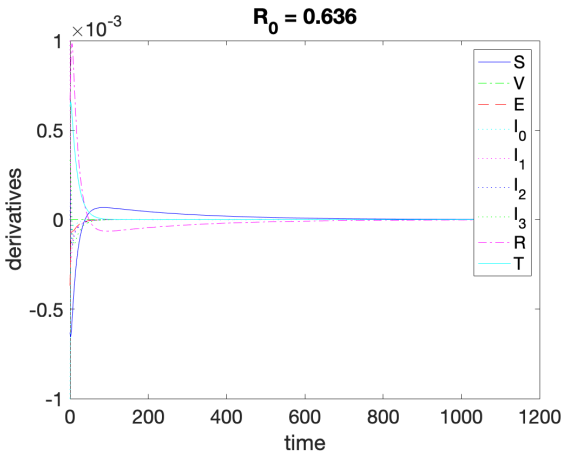
(d) Populations over time for $b_0 = b_3 = 0.02$.

Figure 4: Simulations of the model with a mask mandate in the pandemic free situation and without vaccination over 3 years using the values in Table 2. If the mask mandate reduces the basic reproductive number to less than 1, then the disease will die out. If the mask mandate reduces the reproductive number, but not to below 1, then the disease will eventually become endemic, although it will take longer than 3 years.

the course of the semester. They would also like to acknowledge the help of their classmates throughout the course on epidemiological understanding as well as working with systems of differential equations. Finally, they would like to acknowledge the support and advice of the professor, Dr. Hyman, throughout the class and on this project.



(a) Time derivatives for $b_0 = b_3 = 0.01$.



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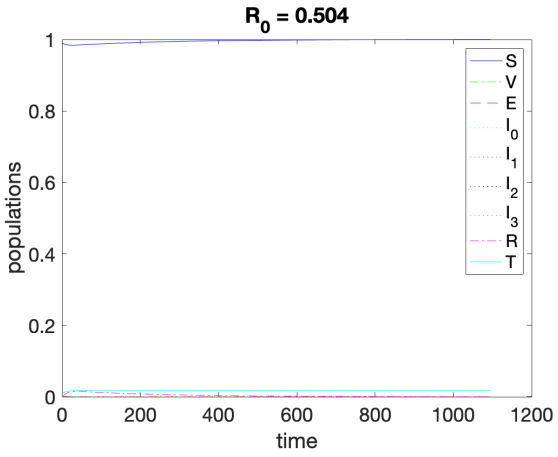
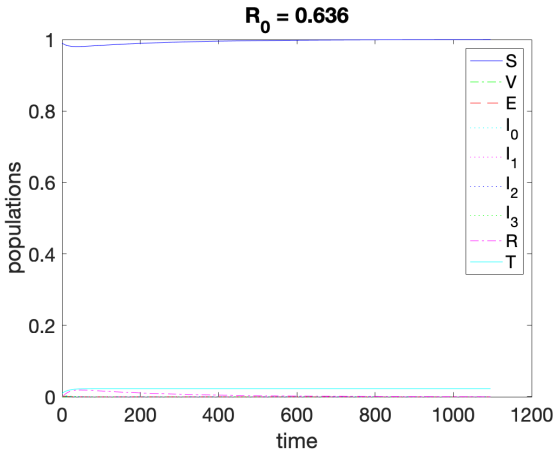
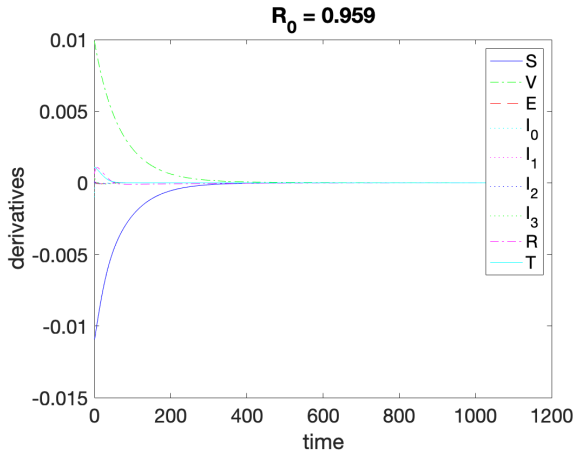


(c) Populations over time for $b_0 = b_3 = 0.01$.

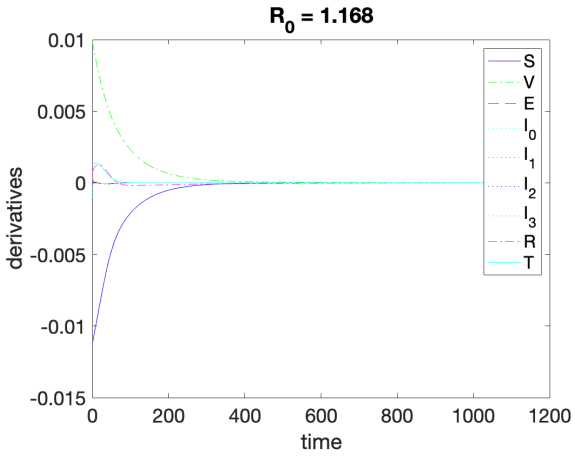


(d) Populations over time for $b_0 = b_3 = 0.02$.

Figure 5: Simulations of the model with mask wearing in a pandemic situation, when masks are worn regardless of symptoms of illness, and without vaccination over 3 years using the values in Table 2. In either basic reproductive number scenario, universal mask wearing in a pandemic situation is sufficient to reduce the basic reproductive number significantly. In both cases the disease will die out with a relatively small incidence.



(a) Time derivatives for $b_0 = b_3 = 0.01$.



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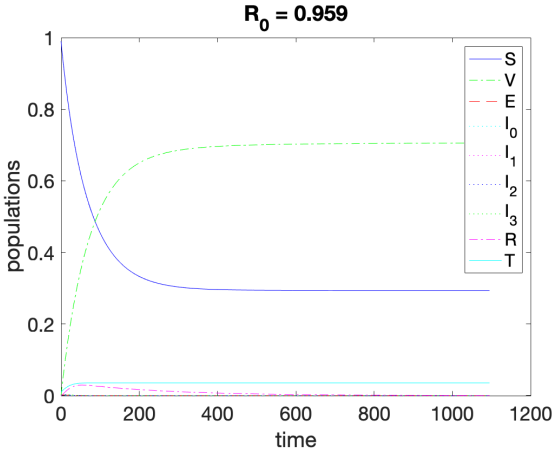
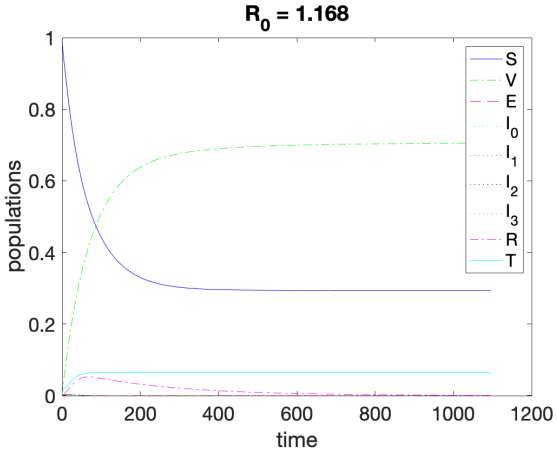
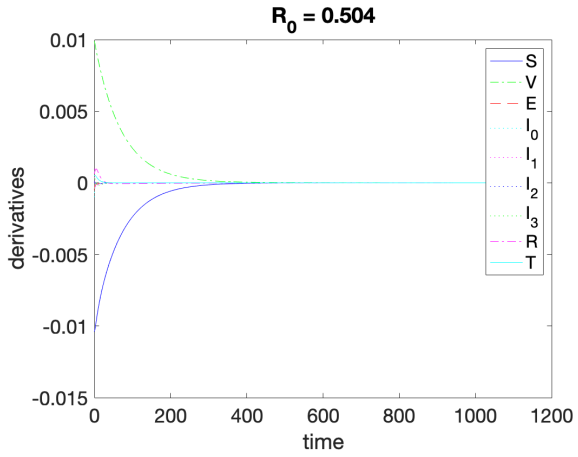


(c) Populations over time for $b_0 = b_3 = 0.01$.

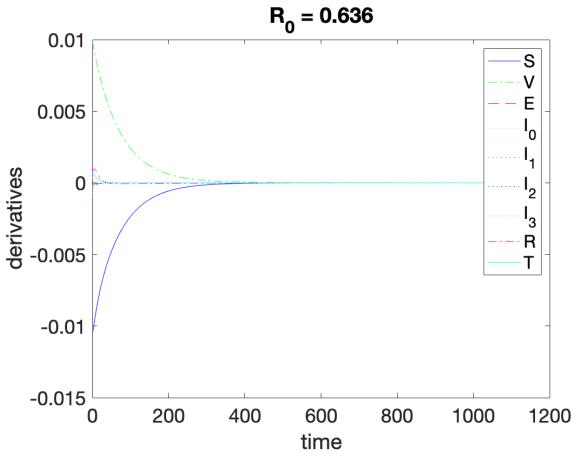


(d) Populations over time for $b_0 = b_3 = 0.02$.

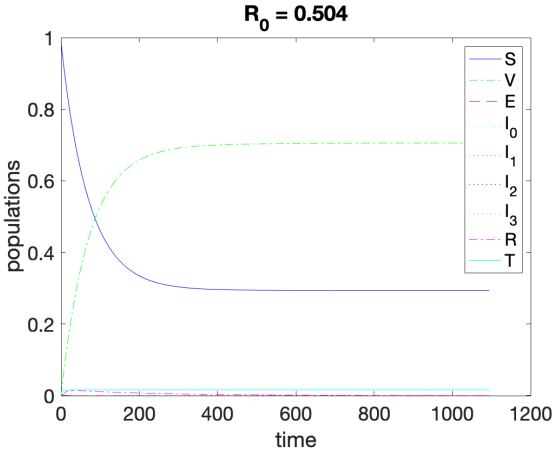
Figure 6: Simulations of the model with vaccination and the mask mandate interventions in a pandemic free situation over 3 years using the values in Table 2. In both basic reproductive scenarios, the combined intervention causes the disease to die out, although the incidence is lower in the low basic reproductive scenario.



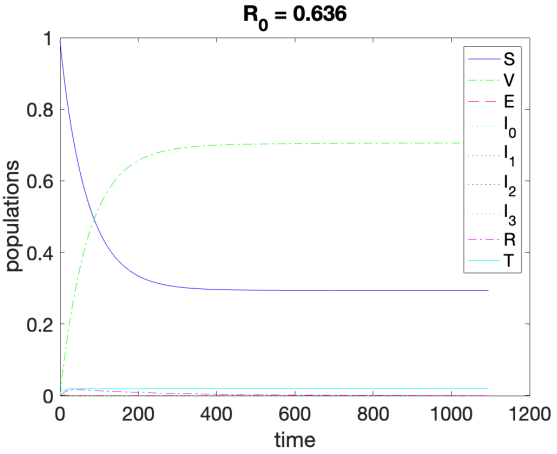
(a) Time derivatives for $b_0 = b_3 = 0.01$.



(b) Time derivatives for $b_0 = b_3 = 0.02$.



(c) Populations over time for $b_0 = b_3 = 0.01$.



(d) Populations over time for $b_0 = b_3 = 0.02$.

Figure 7: Simulations of the model with vaccination and universal mask wearing in a pandemic situation over 3 years using the values in Table 2. In both basic reproductive number scenarios, the disease dies out with low incidence. Compared to the same mask scenario without vaccination, the use of vaccination does not significantly change the outcome or incidence.

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