Epidemic Modeling

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1 Introduction

Papers written in 1927 by epidemiologists William Kermack and Anderson McKendrick detailing one of the first compartmental differential equation epidemic models, fittingly called the "Kermack–McKendrick theory" and modernly shortened to the "SIR model," marked a huge breakthrough in mathematical analysis of a phenomenon previously untouched by numerical methods. By dividing the problem of epidemic modeling into three compartments ("susceptibles", "infecteds", and "recovereds") and representing the time-dependent relationship of these three variables as an intuitive system of differential equations, Kermack and McKendrick were able to build a workable epidemiological model from which to improve upon. In subsequent papers written by the duo, other confounding factors were incorporated into the model to improve its real-world accuracy including birth, death, migration, and even imperfect immunity.

Since Kermack and McKendrick, significant progress has been made by succeeding epidemiologists leading to increasingly accurate predictions, but as a result, models have gotten increasingly complex. Many of the more complex models fall outside the scope of what is possible for this project.

2 Goals

Some goals for this project include the following:

1. Go through list of basic models and discuss their inner workings.
2. Discuss accuracy of basic models.
3. Apply basic models to previous pandemics and discuss accuracy.
4. Apply more complex models to Covid-19 and discuss accuracy.
5. Provide suggestions as to what factors might improve current Covid-19 situation.

3 Models

Some of the models to be discussed in this section are:

1. SI
2. SIS
3. SIR
4. SIR with vital dynamics
5. SEIRD

3.1 SI Model

The most basic of the models is the SI model which is even simpler than the model put forth by Kermack and McKendrick in 1927. The idea behind this model is that initially, most of the population in question is in the "susceptible" group, with at least one person in the infected category. As the epidemic begins, those in the "infected" category will naturally come into contact with those in the "susceptible" category at a certain rate, moving them into the "infected" category. The beauty of this model is that this is where the model ends. The trouble with this model is, also, that this is where the model ends. Clearly, there is a way to transition only from the "susceptible" category to the "infected" category. No one would ever recover from the disease and eventually, everyone in the population would have contracted it and would be living with it for the rest of time. It is easy to see that this model is unrealistic, although it does give us a chance to introduce some of the basic differential equations and a key variable, β. The variable β represents the transmission rate, or, in other words, the average number of transmissions per person per unit time in the population. The system of differential equations for this model is:

$$S'(t) = -\beta * S(t) * I(t)$$
$$I'(t) = \beta * S(t) * I(t)$$

Change in the number of susceptibles in a population is thus dependent on interactions between susceptibles and infecteds. For every interaction between a susceptible and an infected there is a certain probability that that interaction will lead to the movement of a susceptible to the infected category which is represented by the variable β. If a susceptible becomes infected the susceptible category decreases and the infected category increases which explains the signs of the equations.

It is important to make a note of the limits here as time approaches infinity in this model. As long as the infection rate is not zero (why would we be modelling a noninfectious epidemic?), the infecteds will always approach N, where N is the size of the entire population.

From here on out, let s_0, i_0, and r_0 represent the initial values of the susceptibles, infecteds, an recovereds respectively.

When graphed the SI model resembles the following image for $\beta > 0$:

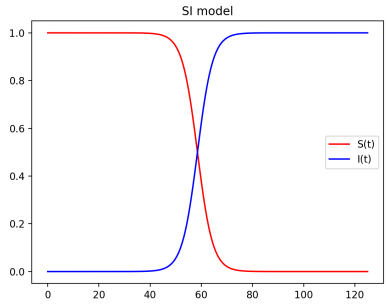

Figure 1: $\beta = 1/3$, $s_0 = 1$ and $i_0 = 3.04 \cdot 10^{-9}$

3.2 SIS model

The SIS model is the next step forward from the SI model. Now, instead of the infecteds being sick for all eternity, we allow the infecteds to recover and join the susceptible pool with the possibility of getting sick again immediately after they reenter. Because there are still only two categories (namely susceptible and infected), this situation can still be modelled by a system of two differential equations. However, two changes must be made to the previous system.

The first is that a new variable γ must be added into the equation. This variable γ represents the recovery rate of people in the population. In other words it tells us how long on average it takes for an average infected person to recover from the disease and join the susceptible pool again.

This brings us to the second change that needs to be made. The previous two equations that modelled $S'(t)$ and $I'(t)$ must include a term that factors in the transition of the recovered population from the infected pool to the susceptible pool. Therefore, the new equations for the SIS model are:

$$S'(t) = -\beta * S(t) * I(t) + \gamma * I(t)$$
$$I'(t) = \beta * S(t) * I(t) - \gamma * I(t)$$

In the susceptible group, every time an infected person recovers, this means an addition to the group of susceptibles, hence the $+ \gamma * I(t)$ in the $S'(t)$ equation. In the infected group, every time an infected person recovers, this means a loss from the group of infecteds, so a $-\gamma * I(t)$ term is thus appropriate in the $I'(t)$ equation.

Unlike the previous SI model, the limits as time approaches infinity in the SIS model are dependent on the cutoff value of β/γ, sometimes called the R_0 value or the basic reproduction number. Heuristically, the basic reproduction number is the average number of people a sick person will infect before they exit the infected group.

If the value of R_0 is greater than 1, then as $t \to \infty$, $I(t) \to 1 - \gamma/\beta$. This situation is modelled by the following graph:

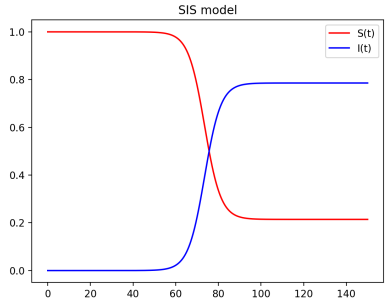

Figure 2: $\beta = 1/3$, $\gamma = 1/14$, $s_0 = 1$ and $i_0 = 3.04 \cdot 10^{-9}$

If the value of R_0 is less than 1, then as $t \to \infty$, $I(t) \to I_0 * e^{(\beta-\gamma)*t}$. This second situation is modelled by the following graph:

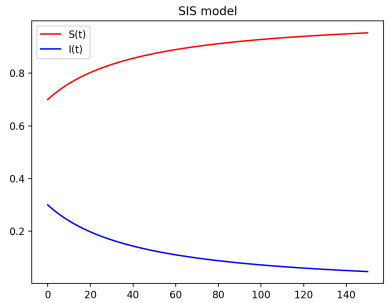

Figure 3: $\beta = 1/15$, $\gamma = 1/14$, $s_0 = 0.8$ and $i_0 = 0.2$

Note that because the above simulation depends on the epidemic effectively "dying out," it is most effective to model a situation in which there is already a significant portion of the population in the infected category. For example, the above graph has $s_0 = 0.8$ and $i_0 = 0.2$ as the starting susceptible and infected populations respectively.

3.3 SIR model without vital dynamics

This was the model first manufactured in 1927 by Kermack and McKendrick. Similar to the previous two models, there is one person initially infected and the majority of the initial population is labeled as susceptibles. As the epidemic begins, those in the "infected" category will naturally come into contact with those in the "susceptible" category at a certain rate, moving them into the "infected" category. At the same time, those in the "infected" category will stop showing symptoms and build immunity after a certain amount of time, removing them from the "infected" category and placing them in their final destination, the "recovered" category which differentiates this model from the SIS model discussed previously. This continues until nearly all the population has moved from the susceptible category to the recovered category.

Only one change needs to be made to convert the previous SIS model to this new model: the addition of a third differential equation to represent the "recovered" population. The system of equations we will use for this model is:

$$\begin{aligned} S'(t) &= -\beta * S(t) * I(t) \\ I'(t) &= \beta * S(t) * I(t) - \gamma * I(t) \\ R'(t) &= \gamma * I(t) \end{aligned}$$

In comparison with the set of equations for the SIS model, we have more or less moved the $+\gamma * I$ term from the susceptible equation to the recovered equation. A simple consideration of the models goal explains why. Now, instead of the infected population rejoining the susceptible pool, they instead join the recovered pool where they will stay for infinite time.

Similar to the SIS model, the SIR models behavior is dependent on the value of $\beta/\gamma = R_0$.

If the value of $R_0 > 1$, infecteds initially increase which make sense because people transmit at a higher rate than they recover. This situation is modelled by the following graph:

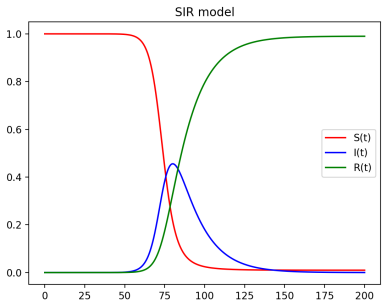

Figure 4: $\beta = 1/3$, $\gamma = 1/14$, $s_0 = 1$, $i_0 = 3.04 \cdot 10^{-9}$, $r_0 = 0$

If the value of $R_0 < 1$, infecteds will decrease initially which is intuitive because people are recovering from the disease faster than the transmission rate. This situation is modelled by the following graph:

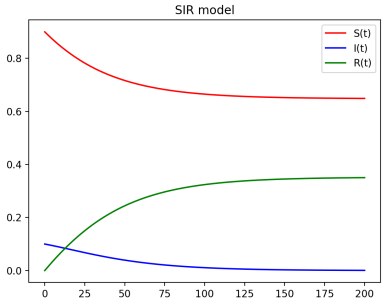

Figure 5: $\beta = 1/15$, $\gamma = 1/14$, $s_0 = 0.9$, $i_0 = 0.1$, $r_0 = 0$

Like the SIS model, the situation above with $R_0 < 1$ depends on their being a significant amount of the population infected at the start of the model, which is why the initial values for the susceptible and infected categories were changed to 0.9 and 0.1 respectively.

3.4 SIR model with vital dynamics

The next piece to the puzzle is adding in natural birth and death rates. It makes sense that not all people in each of the three SIR categories will end up surviving to change categories. Some of the individuals in each compartment will die of natural causes including those who are in the infected category. At the same time, there are people born into the world at a certain rate. For the sake of simplicity, most of the SIR with vital dynamics models keep the birth rate and the death rate the same so that the total population in the system remains stable. For our models, we will take the birth rate $\alpha = 0.005$ to be equal to the death rate $\mu = 0.005$.

There are two changes that need to be made to the previous model to convert it to the new one with vital dynamics incorporated. The first is that births need to be added into the susceptible population (here we assume no maternal immunity). The second is that deaths need to be subtracted away from each category. Thus our new set of differential equation is:

$$\begin{aligned} S'(t) &= -\beta * S(t) * I(t) + \alpha - \mu * S(t) \\ I'(t) &= \beta * S(t) * I(t) - \gamma * I(t) - \mu * I(t) \\ R'(t) &= \gamma * I(t) - \mu * R(t) \end{aligned}$$

Continuing the theme from previous models, the SIR model with vital dynamics's behavior is dependent on whether or not the R_0 value is greater than or less than 1.

If the value of $R_0 > 1$, all categories spike to a maximums before oscillating around their respective limits and coming to rest in the steady state. In the interest of finding the final values for each compartments as time goes to infinity, we will find the limits of each respective compartment by setting the three differential equations equal to zero:

$$\begin{aligned} 0 &= -\beta * S(t) * I(t) + \alpha - \mu * S(t) \\ 0 &= \beta * S(t) * I(t) - \gamma * I(t) - \mu * I(t) \\ 0 &= \gamma * I(t) - \mu * R(t) \end{aligned}$$

Solving these three equations, we obtain the limits for each of the three compartments:

$$\begin{aligned} s_\infty &= (\mu + \gamma)/\beta \\ i_\infty &= \alpha/(\mu + \gamma) - \mu/\beta \\ r_\infty &= 1 - s_\infty - i_\infty \end{aligned}$$

where r_∞ can be expressed in terms of the other two equations because of the fact that in an SIR model, $s + i + r = 1$ is always true.

The graph of the SIR model is displayed below and confirms the derived limits for the given parameters:

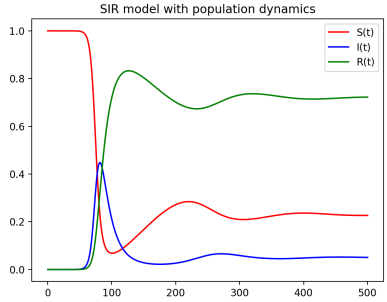

Figure 6: $\beta = 1/3$, $\gamma = 1/14$, $\alpha = 5/1000$, $\mu = 5/1000$, $s_0 = 1$, $i_0 = 3.04 \cdot 10^{-9}$, $r_0 = 0$

An explanation for the difference between this graph and the SIR graph without population dynamics and $R_0 > 0$ is that the latter had only a negative term in the $S'(t)$ equation and only a positive term in the $R'(t)$ equation. Therefore, susceptibles always had to decrease and infected always had to increase. With the addition of births in the $S'(t)$ equation and deaths in the $R'(t)$ equation of the new system, there is a balance between the positive terms and the negative terms that leads to the observed oscillation.

If the value of $R_0 < 1$, akin to previous models, infecteds drop away to zero. Different from the previous SIR model without vital dynamics however, the population returns to all susceptible and no recovered as time $\to \infty$.

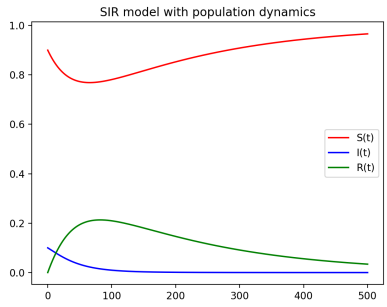

Figure 7: $\beta = 1/15$, $\gamma = 1/14$, $\alpha = 5/1000$, $\mu = 5/1000$, $s_0 = 0.9$, $i_0 = 0.1$, $r_0 = 0$

Intuitively, it makes sense that in a population with the infection rate lower than the recovery rate that eventually the infecteds will drop to zero. It also makes sense that those who had initially recovered from the disease and had immunity would eventually pass away and be replaced by children born into the susceptible compartment.

3.5 SEIRD model

For this next model, we will skip a step or two and answer multiple questions at the same time. Usually, when one contracts an illness, one is not immediately contagious. There is a period between when a person is infected and when that person becomes symptomatic and has the ability to transmit the illness. We will say that the individuals in this in-between period in our new model are in the "Exposed" compartment. Therefore, our new model will incorporate a fourth differential equation represented by $E'(t)$.

Furthermore, in our previous models, people contracted the disease and either remained infected for all eternity or they entered the "recovered" compartment after a certain period of time. This seems to miss the point of why an epidemic is worth modelling in the first place: An epidemic is most relevant when people are killed. To improve our model, we need a way to remove those people who have died from the population, so in our model, we will add a fifth differential equation $D'(t)$ to represent the deceased due to the epidemic.

To make this change, we also need to introduce a new variable which we'll call ϕ. ϕ in our equation will represent the fatality rate. Note that this is different than the previously defined mortality rate μ as μ was the death rate unrelated to the epidemic whereas ϕ is the chance that any given person will pass away after being infected.

The SEIRD model system of equations is:

$$\begin{aligned} S'(t) &= -\beta * S(t) * I(t) \\ E'(t) &= \beta * S(t) * I(t) - \sigma * E(t) \\ I'(t) &= \sigma * E(t) - \gamma * I(t) \\ R'(t) &= \gamma * (1 - \phi) * I(t) \\ D'(t) &= \gamma * \phi * I(t) \end{aligned}$$

Once again, the behavior of this model is heavily dependent on the value of R_0. If the value of $R_0 > 0$, then the graph for the model looks like this:

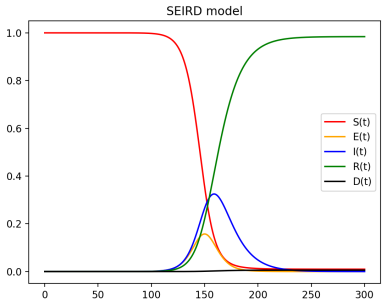

Figure 8: $\beta = 1/3$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $s_0 = 1$, $i_0 = 3.04 \cdot 10^{-9}$, $r_0 = 0$

and if the value of $R_0 < 0$, then the graph for the model looks like this:

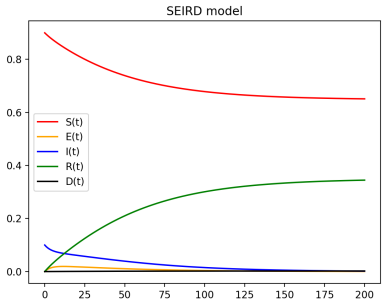

Figure 9: $\beta = 1/15$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $s_0 = 0.9$, $i_0 = 0.1$, $r_0 = 0$

Both of these graphs closely resemble their counterparts for the SIR model without vital dynamics. The only differences are the addition of the "exposed" category and that some of the recovered population is placed in the "deceased" category instead for each graph.

4 Applying models to Covid-19

The challenging part about modelling a complex, real-world pandemic like Covid-19 is that there is a gargantuan amount of factors that need to be considered in order to come up with a suitable model that will be able to effectively predict any long term outcomes. In this section, an attempt will be made to concisely group many of the necessary factors together and to make a conjecture as to what a possible model could look like. The list of these factors includes:

- determination of what model would mimic the behavior of the virus the best
  - Many current models use an SEIR/SEIRD model for the basis of their predictions
- determination of important rates, constants, and other variables
  1. birth rate
  2. death rate
  3. transmission rate
  4. recovery rate
  5. incubation period
  6. fatality rate
  7. the possibility of maternally-derived immunity and the rate at which this occurs
  8. asymptomatic carrier state and the rate at which this occurs
  9. the effect of age on the aforementioned rates for different individuals
  - Difficulty arises with many of these variables as they are not always constant. Real models have to be updated daily with new values for these constants.
- Control measures and their effects must be incorporated into the models.
  - Current control measures for Covid-19 include social distancing, mobility, mask usage, lockdown, and vaccines.
  - Many of these control measures are dependent upon political ideology in any given area and all of them have seen shifts in their usage over time in the United States.

4.1 SEIRD model with vital dynamics

In programming, we don't start with a blank source file, code the entire program, and then compile and run it to see if our code works properly. Instead we build and test the program incrementally to help us narrow down possible sources of bugs. We will do the same here adding in individual factors and testing them graphically.

Our base model will be the SEIRD model already discussed at the end of the previous section. The first factor we will add in to the model is vital dynamics (constant population). The system of equation used for this model is:

$$\begin{aligned} S'(t) &= -\beta * S(t) * I(t) + \alpha - \mu * S(t) \\ E'(t) &= \beta * S(t) * I(t) - \sigma * E(t) - \mu * E(t) \\ I'(t) &= \sigma * E(t) - \gamma * I(t) - \mu * I(t) \\ R'(t) &= \gamma * (1 - \phi) * I(t) - \mu * R(t) \\ D'(t) &= \gamma * \phi * I(t) - \mu * D(t) \end{aligned}$$

The system of equations is rapidly getting more complex and the model does not yet have close to all of the factors it need. The graph of the model after adding in vital dynamics looks like this:

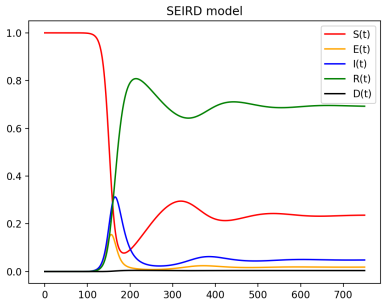

Figure 10: $\beta = 1/3$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $\alpha = 5/1000$, $\mu = 5/1000$, $s_0 = 1$, $i_0 = 3.04 \cdot 10^{-9}$, $r_0 = 0$

This seems to make sense. Before, when we added in vital dynamics to our SIR model, it produced a graph with initial spikes to maximum values in the susceptible, infected, and recovered compartments that then oscillated as they approached their steady state solutions. This model is consistent with that as well.

4.2 SEIRD model with vital dynamics and vaccination of newborns

One of, if not the best, prevention measures for stopping the spread of a virus is the creation of vaccines that provide immunity for a certain time period for individuals in a given population. For the sake of simplifying the model, we will attempt to add a vaccination factor into our model represented by a new compartment $V'(t)$, but we will assume that vaccination gives lifelong immunity to its recipients. In this model, we will consider the scenario when a portion of newborns are vaccinated at a rate given by ν.

We will first create a model with vaccination of newborns and a value for $\nu = 0.9$ which is not unreasonable as polio, MMR, and chickenpox all had vaccination rates for infants above 90%. The equations governing the new model look like this:

$$\begin{aligned}
S'(t) &= -\beta * S(t) * I(t) + \alpha * (1 - \nu) - \mu * S(t) \\
E'(t) &= \beta * S(t) * I(t) - \sigma * E(t) - \mu * E(t) \\
I'(t) &= \sigma * E(t) - \gamma * I(t) - \mu * I(t) \\
R'(t) &= \gamma * (1 - \phi) * I(t) - \mu * R(t) \\
D'(t) &= \gamma * \phi * I(t) - \mu * D(t) \\
V'(t) &= \alpha * \nu - \mu * V(t)
\end{aligned}$$

The graph showing this model with newborn vaccination is:

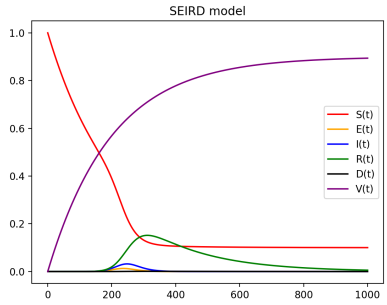

Figure 11: $\beta = 1/3$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $\alpha = 5/1000$, $\mu = 5/1000$, $\nu = 0.9$, $s_0 = 1$, $i_0 = 3.04 \cdot 10^{-9}$, $r_0 = 0$

People in every category naturally pass away and are replaced with newborns entering the model through the "susceptible" category with a chance to be immunized immediately. Those babies are slowly added into the "vaccinated" category. As time $\to \infty$, the vaccinated compartment approaches the rate of newborn vaccination, which in this model is 0.9%. Clearly, this strategy is hugely effective in the long term, but is less effective in the short term as the steady state of the above graph is around 1000 days.

4.3 SEIRD model with vital dynamics and vaccination of both newborns and non-newborns

We have previously discussed the model where newborns are vaccinated and the difference the factor made in our model was stark. It would be interesting to see how the model is affected if not only newborns but also non-newborns are vaccinated. In this last model, we will consider the scenario when non-newborns are vaccinated at a rate given by ϵ.

Changes to the system of differential equations are small compared to the previous system. A term is added to the "susceptible" and "vaccinated" compartments to signify the non-newborn population that is being vaccinated and removed from the susceptible group. The equations are thus:

$$
\begin{aligned}
S'(t) &= -\beta * S(t) * I(t) + \alpha * (1 - \nu) - \epsilon * S(t) - \mu * S(t) \\
E'(t) &= \beta * S(t) * I(t) - \sigma * E(t) - \mu * E(t) \\
I'(t) &= \sigma * E(t) - \gamma * I(t) - \mu * I \\
R'(t) &= \gamma * (1 - \phi) * I(t) - \mu * R(t) \\
D'(t) &= \gamma * \phi * I - \mu * D(t) \\
V'(t) &= \alpha * \nu + \epsilon * S(t) - \mu * V(t)
\end{aligned}
$$

This model changes with different rates of ϵ. For low values of epsilon, the graph looks like this:

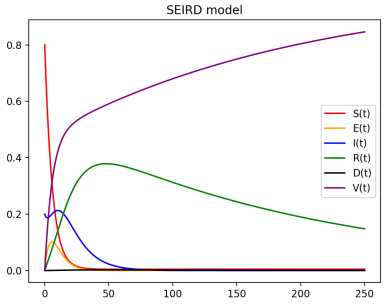

Figure 12: $\beta = 1/3$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $\alpha = 5/1000$, $\mu = 5/1000$, $\nu = 0.9$, $\epsilon = 0.1$, $s_0 = 0.8$, $i_0 = 0.2$, $r_0 = 0$

Note that, one final time, the slope for the infected compartment is always negative if we start with one infected person. For this reason we start the model with $s_0 = 0.8$ and $i_0 = 0.2$ for initial values of susceptibles and infected respectively. This is more applicable in a real-world context anyway as vaccines are not developed immediately but instead are developed and implemented with a portion of the population already having been infected.

To illustrate the difference between vaccination rates, two more graphs are included below with parameters outlined in the caption:

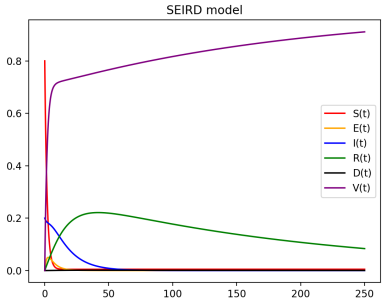

Figure 13: $\beta = 1/3$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $\alpha = 5/1000$, $\mu = 5/1000$, $\nu = 0.9$, $\epsilon = 0.5$, $s_0 = 0.8$, $i_0 = 0.2$, $r_0 = 0$

Note the lack of a peak of the infected compartment compared to the previous model. Slope for the infecteds is zero for all time t in this model. An interesting note here is that 50% vaccination is a pretty common immunization rate for yearly influenza. Vaccination rates for Covid-19 will likely be higher than 50%. Let's take a look at a model with a significantly higher rate of vaccination than 50%:

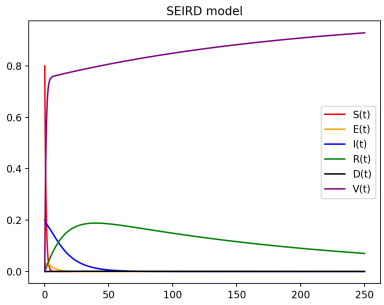

Figure 14: $\beta = 1/3$, $\gamma = 1/14$, $\sigma = 1/5$, $\phi = 6/1000$, $\alpha = 5/1000$, $\mu = 5/1000$, $\nu = 0.9$, $\epsilon = 0.99$, $s_0 = 0.8$, $i_0 = 0.2$, $r_0 = 0$

Although there is slight improvement in all categories of this model, it becomes apparent that after a certain vaccination rate threshold, eradicating a pandemic becomes almost entirely time dependent. Benefits of a vaccination rate as high as 99% are likely not great enough to offset the cost of the vaccines.

5 Conclusion

This was one of the more interesting projects I've undertaken. On the outset, I didn't know what I wanted to accomplish in the time frame I had. I had originally wanted to go through a progression of models (more to solidify my own understanding of them than anything else) from most basic SI to the SEIRD model which was the most complex model discussed in class. I then wanted to apply models to previous pandemics and originally planned to analyze the Spanish flu and the Swine flu, but quickly shifted gears as I realized retroactively fitting models to previous data, while perhaps an interesting and worthwhile application of epidemic modelling, would be terribly time consuming. So I instead focused my attention on extending the SEIRD model in the final section.

Reflecting on my results, many of the rudimentary models had been discussed to an extent in class, and thus were unsurprising. It was interesting to see the progression of the models as they became more complex with perhaps the most unexpected result coming from the addition of vital dynamics to the system in model 3.4. The mere addition of natural births and deaths into the system produced spikes and oscillations in the modelled epidemics representing changes in dominant terms in the differential equations. Of course, all vital dynamics models in this project came with the caveat that the birth rate and death rate was kept the same in an effort to keep the population constant which, in reality, is not entirely accurate.

On that note, perhaps the biggest takeaway I gleaned from this project was the sheer complexity of epidemic modelling. I started at the SI model and went through 7 progressions to reach my final model. However, I was nowhere near close to an accurate model with all necessary factors. Of the factors mentioned at the beginning of section 4, I still had yet to implement the carrier state, the effect of age, social distancing, mobility, mask usage, lockdown measures, and legal differences between regions of the United States. Even a model that did accurately include all of these factors would not be complete.

I think at the beginning of this project I had had this idea that epidemic modelling was a way to take something seemingly abstract and create an exact science out of it. Now, I realize that no matter how many factors you add into a model, there are almost certainly other unconsidered nuances that will throw off predictions. Therefore, the real goal of epidemic modelling should not be to create a perfect model for any given epidemic, but instead to create a sufficient model and learn how to adjust that model as factors, rates, and constants vary with time which is clearly easier said than done.

If I were to extend this project in the future, I would likely want to revisit the retrofitting aspect of epidemic modelling not only because I think it would be interesting to see what models fit best with various epidemics, but also because I realize now that retrofitting is actually a part of the process of updating current models to more accurately reflect events in the recent past.

TLDR: Interesting project that produced some unexpected results. Would have liked to retrofit some models in an extended project.

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