MONITORING PANDEMICS USING THE SIR MODEL

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July 30, 2021

EQUATIONS

$$S = S(t)$$

 $l = I(t)$
 $s(t) + i(t) + r(t) = 1$
 $dS/dt = -bSI/N$
 $dI/dt = bSI/N - kI$
 $dR/dt = kI$
 $dI = (b * S * I/N - K * I) * (1 - alpha)$

Preface

In all that is based on the principles of mathematics and physics(Everything), there exists a fundamental truth. Every model created by humans for the purpose of understanding is simply an approximation. Even with the promise of morals law, and our expectation of exponential growth in computational power; We cannot expect to understand a system as is. However, we don't have to. Humanity has arguably made it very far by sheer approximation alone. In this essay, we are interested in the extremely complicated nature of infectious diseases, and the deceivingly simple mathematics behind them.

The SIR model is a mathematical tool that uses Ordinary Differential Equations (ODEs) to visualize and monitor the progression of infectious diseases in a society. The model describes the relationship between the susceptible, infected, and recovered populations, specifically how the number of people in each population change over time as they move from one to another. Susceptible individuals can become infected when coming in contact with an infected person while infected individuals recover, gain immunity, and move to the recovered population. The recovered population is also referred to as the "removed class" because it contains all those who can no longer transmit the virus, therefore the individuals in this population could be alive or dead. The changes that occur with the susceptible and the infected populations are the only ones that we take into consideration when developing our model.

The layman explanation of the SIR model

The subject in question is the SIR model. This is a compartmental model meaning we separate the subject into categories where each individual can transition between the categories. The three categories in the SIR model consist of susceptible, infected, and recovered. The layman's explanation there is an infinite amount of variables at play for any pandemic or endemic. You just need to know some initial conditions such as how many people are infected, how easily can the disease be spread, how long do people tend to spread it, and how many people we talking about. This is all the information you need to make a solid prediction of how a pandemic will develop. It is important to mention that if you "tend"

Mathematical Analysis

There are 2 main parameters that control changes in the populations: the infection rate (b) and the recovery rate (k). The infection rate determines how fast susceptible individuals become infected per day, and the recovery rate determines how fast infected individuals become non-contagious. We take these individual populations and divide each of them by the total population to calculate the average infection number (bS/N) and the average number of recovered patients per day (kI). Now we will break down each equation. The model is a system of 2-D, autonomous differential equations since the equations have 2 state variables S and I.

$$dR/dt = kI$$

➤ Equation 1 calculates the rate that the susceptible population is decreasing as people become infected. The negative since represents the constant negative slope for this population.

$$dS/dt = -bSI/N \tag{2}$$

➤ Equation 2 describes the changes of the infected population. The first term reflects how susceptible individuals are who got sick now join the infected. The second term represents the rate infected individuals become non-contagious and move to the removed/recovered population.

$$dI/dt = bSI/N - kI$$

In continuation, the SIR model will be shown in terms of real numbers and having been solved. Due to the model being two-dimensional we will need two initial conditions to compensate for the total two state variables being represented in equations (1) and (2). In this case, the initial susceptible count will be represented by 0.9, (90%). This means that about 90% of the population

is at risk of contracting the disease. The second initial condition which is the infected will be 0.1. (10%). One may be wondering if we would include another variable as well to represent the population who has either died or contracted the disease. The answer to that is no, the reason being is that this portion of the population will have no impact unless they contract the disease once more or infect others, therefore we null them from consideration. There will be parameter values included as well which can be any number chosen to represent the rate changes. For equation (1), the rate b/N will be represented by the number 2. In equation (2) the number chosen is 1.5. In order to show the relationship and go more in-depth the two-state variables S and I will need a phase line from the values zero to one as shown in figure 1. It is also necessary to plot the initial conditions 0.9 and 0.1 onto their designated phase line. The overall solution to this model will be two functions S(t) and I(t) which give the evolutional fractions overtime of the susceptible and infected individuals. Figure (1b) begins by demonstrating the initial conditions of the Susceptible population. The reason why the slope is downward, in the beginning, is since as more individuals become infected the number of individuals left to contract the disease is less. This can be demonstrated in terms of values for instances after having solved S'(0) and S(0)I(0) we get a resultant of -0.18 which was expected to occur. We then follow up by representing this shift onto our phase line for the susceptible with an arrow pointing left, away from the initial 0.9. In contrast, the rate of change for the Infected, (figure 1c), demonstrates an upward slope commencing from the initial 0.1. The cause for this positive slope is due to the rate of infection increasing, meaning many more individuals who were initially not sick contracted the disease. Mathematically, the I'(0) and the S(0)I(0) result in a positive integer 0.13 which backs up the original statement. When shown on the phase line it is represented by an arrow starting at 0.1 and moving away, shown in figure (1d). We then take into consideration what would occur as time

passes, for single autonomous differentials the state variables would move continuously in the original direction but in terms of our model presented we will have two resulting levels. One which levels off at about S=0 or some other integer S. In the phase line it will be represented by multiple arrows pointing away within the boundary of 0-1. The reason for this is because the rate of change susceptibles can never be positive, only negative or zero. Which is shown in Figure (2a). In terms of I(t), it can never be larger than one since it only represents a fraction of individuals who are infected therefore at some point the growth must terminate. When factoring in the rate of susceptibles balances the rate of recovery resulting in the number of infected individuals remaining constant. Therefore in the following equations, it results in I=0 and S=0.25 being the stopping points to where the number of infected individuals runs out or the fraction of the susceptibles drops to a quarter. That is why the true cause for the growth stop of I is in due part of the value S, since the number of infected will stop as there are no more individuals left to be infected. Therefore we commence to include the arrows following the original line away from 0.1 but without a specific model representing the method in how S(t) evolves we are unable to see where the critical points for I are, figure (2b). In the below figure (2c) we assume that the S rate of change graph aligned with the I rate of change graph will change at where 0.25 of S is. If this were to be the case then we could safely assume that I(t) would stop increasing and begin to drop. We would also assume that the local MAX of I(t) would be at the point in changes from increase to decrease. With this change in mind, we then must adjust our original phase line to demonstrate the decrease. This results in arrows pointing the opposite direction above our previous lines to demonstrate this change, as demonstrated in figure (2d). After having gone over the relationships between the values and the model we will now discuss the impact of our values in terms of the epidemic. Unfortunately, since we did not set a specific point to where S becomes

constant we are unable to determine whether or not all or some susceptible individuals got infected. Based on our representation we have two case scenarios, the first being if S(t) approached zero then the epidemic in those terms was as bad as it gets. The other case scenario is if S(t) had remained fairly large and constant then that would have meant that many humans escaped the disease. This can be seen in Figures (2b).

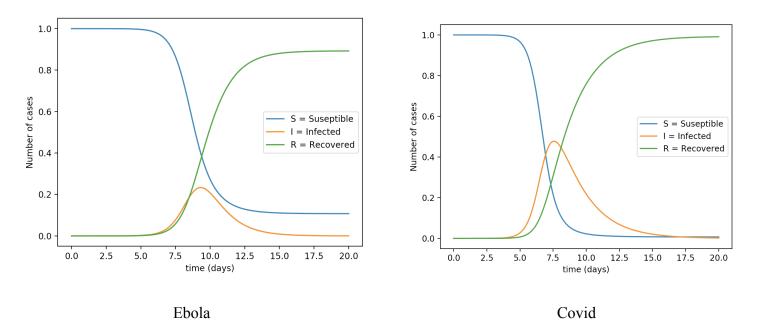
Covid-19 vs the Ebola Virus

It was interesting to consider two of the most recently famous deadly diseases in the public lexicon. There is ebola, and there is covid-19. The nature of the two diseases can be described as polar opposites. The Ebola virus was first reported in 2014; the pandemic caused by the virus ended in 2016, and there were a total of 28,000 cases over this approximately 2 year period. On the other hand "the first case of the novel COVID-19 appeared in December 2019 and as of April 28, 2020, there have been nearly 3 million cases and close to 212,000 deaths" [6]. Each virus has various feature differences: Most Ebola cases were severe cases and the infected suffered from diarrhea, vomiting, and bleeding. Conversely, 80% of Covid cases have been mild or less where the infected suffer from coughing, strained breathing, and pneumonia in the worst cases. A major difference between the two is that Ebola could only be transmitted in the latest stages when infected individuals would experience symptoms that would leave them bedridden, but Covid-19 can be transmitted days before an infected individual shows symptoms, meaning that transmission is much easier for Covid.

Due to these differences, the Covid-19 virus and the Ebola virus have different reproduction, infection, and recovery/mortality rates and have had very different impacts on the world. "The reproductive number R describes the average number of individuals that a person infected with a particular pathogen infects" [5]. The estimated reproduction rate of Covid-19 is

between 1.4 and 5 as there are multiple variants of the virus worldwide, while Ebola's reproduction rate is estimated to be between 1.5 and 2.5 [5]. We see that there is a stark difference in the range of Covid's reproduction rate over Ebola's; this is why the Covid-19 virus seems to spread so quickly worldwide when compared to Ebola. We calculate the reproduction rate using this equation: Ro = bS/k (4)

If we compare plots of the two viruses we get:

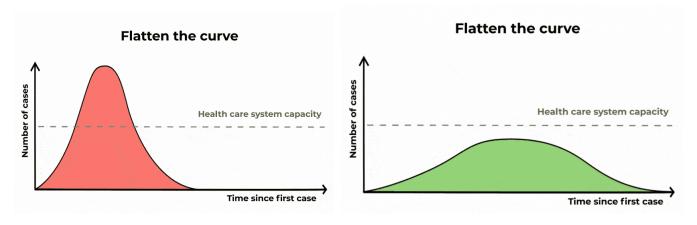


On the Ebola plot, we can see that not all of the susceptible population actually catches the virus; the virus begins to naturally die down on its own between 10 and 12.5 days because the reproduction rate drops below 1, which is the minimum number to sustain a pandemic. On the covid plot, the infection curve reaches its peak about 30% faster than Ebola (around day 7) and infects the whole susceptible population by day 10. From the plots above we can also see

that the infection curve for covid is steeper and its peak is double the peak of Ebola's infection curve. These comparisons help us to conclude that Covid is about 2x more infective than Ebola.

Additional Parameters

Several methods have been implemented to help slow down the Covid-19 virus' infection rate and "flatten the curve" as we so frequently heard Dr. Faucii, the director of the U.S. National Institute of Allergy and Infectious Diseases (NIAID), and other medical professionals echo during this pandemic. The two biggest reasons for the push have been to limit the number of fatalities and to prevent the health care system from collapsing due to an influx of more infected patients than what the hospitals can handle.



From source [1]

Social distancing measures are the most effective ways we have implemented to flatten the curve. These measures include: limiting the capacity of places where large gatherings can occur such as restaurants, bars, theatres, etc, making it mandatory to wear masks, and enforcing space restrictions of 6ft apart in public spaces. The result of these measures can be shown by adding an additional parameter to our SIR model, specifically to the dI/dt equation as this controls the infection rate; the parameter is named alpha in our model.

The range is $0 \le \text{alpha} \le 1$, and this represents the different intensities of the social distancing measures. For example, alpha $\cong 1$ is a complete lockdown (being equal to 1 is in theory since people need to go to grocery stores to get supplies), alpha = 0.5 would be social distancing measures running at 50%, and alpha = 0 would be no social distancing. The resulting dI/dt equation is below.

$$dI = (b * S * I/N - K * I) * (1 - alpha)_{(5)}$$

Resulting plots after adding social distancing parameter:

$$alpha = 0$$

$$0.8 - \frac{1.0}{0.8} = 0.5$$

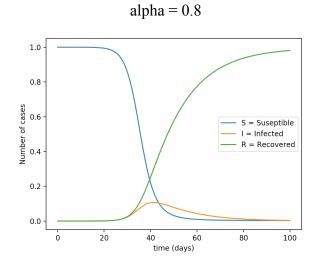
$$0.8 - \frac{1.0}{0.8} = 0.6$$

$$0.8 - \frac{1.0}{0.8} = 0.5$$

$$0.9 - \frac{1.0}{0.0} = 0.5$$

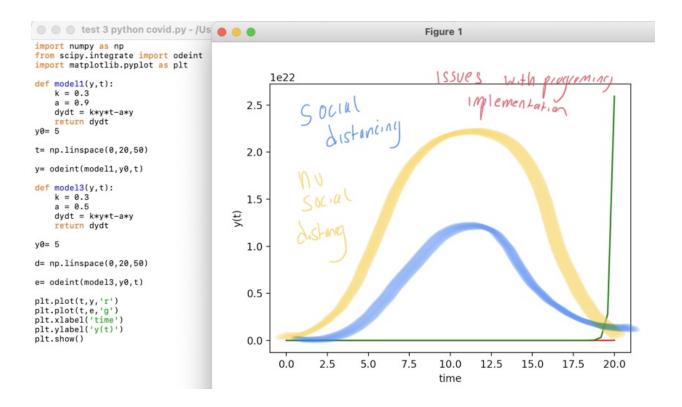
$$0.9 - \frac{1.0}{0.0} = 0.5$$

$$0.0 - \frac{1.0}{0.0} = 0.5$$



The three plots show the effects of three different levels of social distancing; 0%, 50%, and 80% intensity. The infection curve surges to 0.6 (60%) of the population when there is no social distancing in place (aka alpha = 0); this rate is too unsustainable. When 50% of social distancing (alpha = 0.5) is put in place the infection rate is halved to 0.3 (30%) of the population, and when 80% of social distancing (alpha = 0.8) is implemented, the infection rate is reduced significantly, peaking at 0.1 (10%) of the population. Social distancing also elongates the total time span of the pandemic, which is why the end date of the pandemic is 100 days instead of 60.

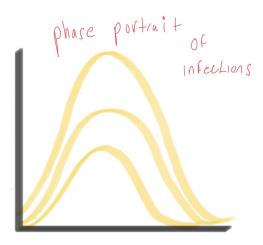
Experimental Python Graph. The plan was to have the infection rate function reduced by a certain function. This function was supposed to represent exist another version of equation #3, which represents the lower rate of infection. This can be simply explained by the lower rates of infection as distance between individuals increases. There were issues with programming implementation but the idea was to illustrate the benefits of social distancing in an intuitive manner. There is an abundance of research supporting this proposed model.



$$dS/dt = -bSI/N$$

$$dI/dt = bSI/N - kI$$
 The objective was to introduce

another variable that could represent the influence of social distancing on the population. For the rate of change of infected, it would have been another variable subtract from the rate, those the susceptible population would have decreased at a lower rate. However, the infection will continue as long as the susceptible population does not become part of the recovered or removed group. The only other way to join the removed population besides getting infected would require vaccination. We do want to point out that vaccination is technically a controlled infection, but this is more of an issue with terminology rather than mathematics. Ultimately, the benefit of social distance can reduce the peak of infections.



The phase graph of the SIR model can be described as various-sized bells. The purpose is to describe different models with different initial conditions. This leads to one of the biggest issues with the SIR model and the covid 19 pandemic. According to various research, most models during the beginning of the pandemic varied wildly. Many of them failed to predict the spike of summer 2020. The issue is that in order to have reliable predications there needs to be information of what is the initial number of cases. That number is fundamental since any slight adjustments to the initial conditions can cause predictions to vary wildly. That information scarcity was one of the major reasons why the SIR model failed. The other issue is the model accounts for the number of individuals infected however it does not account for social factors. Today, you can see the trends that pushed us toward our winter spike. The holidays were a major reason why covid spiked because people wanted to congregate in order to socialize and avoid the cold. There is no social parameter in the parameter for the SIR model, or a weather parameter. Thus the model is unprepared to take such factors into consideration. This does not mean the model is worthless. It just means that it's a model, it is very accurate under the right conditions of knowing initial parameters.

WORKS CITED

- Black, Andrew, et al. "How to Flatten the Curve of Coronavirus, a Mathematician
 Explains." *The Conversation*, 13 July 2021,
 theconversation.com/how-to-flatten-the-curve-of-coronavirus-a-mathematician-explains133514#:~:text=This%20means%20that%20if%20you,of%20saying%20slowing%20the
 %20spread.
- 2. Nykamp, Duane, director. *YouTube*, YouTube, 17 Nov. 2013, www.youtube.com/watch?v=06wnwSEHZPY.
- 3. Sadun, Lorenzo, director. *YouTube*, YouTube, 29 July 2014, www.youtube.com/watch?v=-e igCrev10.
- 4. Smith, David, and Lang Moore. "The Sir Model for Spread of Disease the Differential Equation Model." *The SIR Model for Spread of Disease The Differential Equation Model* | *Mathematical Association of America*, www.maa.org/press/periodicals/loci/joma/the-sir-model-for-spread-of-disease-the-differential-equation-model.
- Zimmer, Katarina. "Why r0 Is Problematic for Predicting Covid-19 Spread." *The Scientist Magazine*®, 2020,
 www.the-scientist.com/features/why-r0-is-problematic-for-predicting-covid-19-spread-67 690.
- 6. "How Does Covid-19 Compare to Other Pandemics (H1N1, EBOLA)." *UT Health East Texas*,

uthealtheasttexas.com/virtualcare/articles/how-does-covid-19-compare-other-pandemics-h1n1-ebola.

7. Moein, Shiva, et al. "Inefficiency of SIR Models in Forecasting COVID-19 Epidemic: A Case Study of Isfahan." *Nature News*, Nature Publishing Group, 25 Feb. 2021, www.nature.com/articles/s41598-021-84055-6.

Equations and Figures:

$$S(0) = 0.9$$

$$I(0) = 0.1$$

$$b/N = 2$$

$$K = 1.5$$

$$dS/dt = -2SI$$

$$dl/dt = 2SI - 0.5I$$

Figure 1)



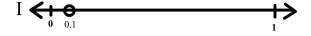
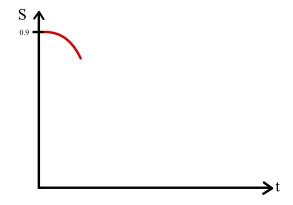


Figure 1b)





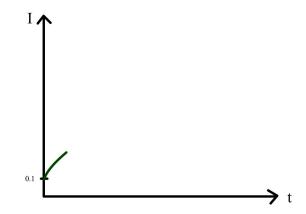
Equations)

$$S'(0) = -2S(0)I(0)$$

$$S'(0) = -2(0.9)(0.1)$$

$$S'(0) = -0.18$$

Figure 1c)



Equation)

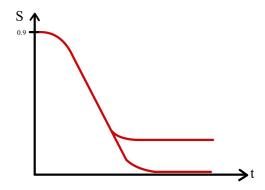
$$I'(0) = 2S(0)I(0) - 0.5I(0)$$

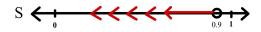
$$I'(0) = 0.13$$

Figure 1d)



Figure 2a)





Equation)

$$dS/dt = -2SI \le 0$$

$$dS/dt = -2SI = 0$$

$$IfS = 0 or I = 0$$

Equation)

$$dI/dt = 0 - 2SI - 0.5I$$

$$I(2S - 0.5) = 0$$

$$I = 0 or 2S - 0.5 = 0$$

$$I = 0 or S = 0.25$$

Figure 2b)



Figure 2c)

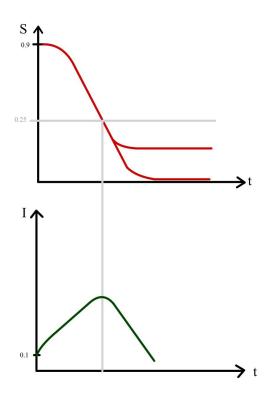


Figure 2d)

