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Covid-19: Analysis with SIR-Model

We are interested in examining the spread of the current Covid-19 virus, as affected by quarantine measures and the asymptomatic/symptomatic condition of infected individuals. Our analysis is done with hopes of evaluating the efficiency of self-isolation through quarantine and the consideration of asymptomatic individuals who also pose a threat of spreading the virus. Our data is limited to a few regions around the world, including the United States, therefore posing possible incorrect conclusions and assumptions regarding the virus throughout the entire world. This in turn is due to the varying laws and rules imposed (or not) by leading rulers and government organizations and the varying number of contacts made each day in every region. Acknowledging this, we have hopes of constructing a model that may be used to represent the spread of Covid-19 throughout most regions of the world, through an adaptation of parameters based on the desired location.

Our model was initially based off of that of a standard SIR (Susceptible, Infected, Recovered) model, which also took into account the number of individuals who were in the process of recovering, and those who had died due to the virus [1]. With some additional research, we found an additional model in the form of SEIR (Susceptible, Exposed, Infected, Recovered). This model took into account the effectiveness of quarantines and the use of constant testing based on asymptomatic and symptomatic carriers [2]. Our aim was to incorporate the elements and conditions of how quarantine affected the transmission of the virus upon prolonged contact and the transmission from asymptomatic carriers in addition to those who are symptomatic. We did not incorporate the paper's analysis of the effects of the effectiveness of Covid testing on the containment of the virus itself, as data may be insufficient from not having consistent, universal testing for all affected. In addition, we included that assumption that recovered individuals may become susceptible to receiving the virus again, as seen in common SIRS models. For this paper we fix an *effectiveness* of quarantine in that of reducing the rates of contacts of individuals. We assume that complete isolation is nearly impossible, however we acknowledge that an improved quarantine will have a positive effect on mitigating the spread of the virus. We also acknowledge that it is difficult to conclude that the transmission of the disease from infected asymptomatic individuals and infected symptomatic individuals to be exactly the same. We also assume the duration of quarantine to be universal throughout all conditions when mandated by the region. Though errors may exist in the

representations of the model, our goal is to develop a model to better represent the Covid-19 pandemic taking into account the use of effective quarantine.

1. Model Explanation:

According to the analyzed SIR and SEIR models, we may categorize the population into the following groups and parameters:

S = Susceptible,

I = Infected,

R = Resolving,

D = Number of dead from pandemic,

C = Number of recovered people,

N = Total number of population.

Where $N = S + I + R + D + C$.

In assembling our model, we have made a handful of observations and assumptions that affect the output and accuracy of our equations. To start, we set the initial values of our parameters to represent the introduction of the virus to a society in which no individual has previously come in contact with the virus. Essentially, we are at the outbreak of the virus such that the amount of people affected are negligible in comparison to the total population of which we wish to examine. Therefore, the entirety of the population (N) is classified as susceptible (S) to start. The numbers of infected individuals (I), resolving individuals (R), those who died from infection (D), and those who have recovered from infection (C) are all initially zero. As more contacts are made throughout society and more individuals are infected with the virus, quarantines are implemented. However, without a certainty of a unanimous amount of quarantine amongst the population, we have set two different values representing the contact rate of those who are quarantined ($\lambda^{(Q)}$) and those who are not quarantined ($\lambda^{(NQ)}$). By distinguishing these two contact rates, we may also conclude that all living individuals make up one of two subpopulations, those in quarantine ($N^{(Q)}$) and those not in quarantine ($N^{(NQ)}$):

$$N^{(Q)} = S^{(Q)} + I^{(Q)} + R^{(Q)} + C^{(Q)}$$

$$N^{(NQ)} = S^{(NQ)} + I^{(NQ)} + R^{(NQ)} + C^{(NQ)}$$

$$N = N^{(Q)} + N^{(NQ)} + D$$

Although our model includes the number of individuals who died from the virus, we are not including vital dynamics within our model. We want the purpose of our model to be the analysis of the virus on a population at a given instant based on all individuals who may be affected at the moment. Consequently, our assumption for this examination is that the vital dynamics will not affect the overall result of our analysis.

The spread of the virus itself is dependent on the number of contacts made each day (varies with quarantine or non-quarantine) and the length that the infected remain contagious. Therefore, our model can be parameterized in terms of the reproduction rate by the equation;

$$R_0 = \beta/\gamma$$

where R_0 is the reproduction rate of the virus.

We make the additional assumption that the number of infected individuals (I) is separated into two subcategories, $I^{(A)}$ and $I^{(S)}$. Here, $I^{(A)}$ represents the number of infected individuals that are asymptomatic. That is, infected individuals who do not yet show any symptoms of the virus. Whereas $I^{(S)}$ represents the number of individuals that are symptomatic and are therefore showing signs that they are indeed infected with the virus. Thus, the total number of infected individuals (I) is just the sum of these two values. Other parameters in our model include the contact rate of the virus (β), the average amount of time to become infected (θ), the death rate (δ), the rate that recovered people become susceptible (ξ), and the recovery rate from the virus (γ). Observe that the rate of increase in infectives is proportional to the contact between susceptibles and infectives. All of our variables and parameters combine to form the following system of ordinary differential equations:

$$\frac{dS}{dt} = -\frac{S(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} + \xi C$$

$$\frac{dI}{dt} = \frac{S(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} - \gamma(I^{(A)} + I^{(S)})$$

$$\frac{dR}{dt} = \gamma(I^{(A)} + I^{(S)}) - \theta R$$

$$\frac{dD}{dt} = \delta\theta R$$

$$\frac{dC}{dt} = (1 - \delta)\theta R - \xi C$$

We can simplify our model by letting I and β be the following values:

$$\beta = \lambda^{(NQ)} + \lambda^{(Q)}$$

$$I = I^{(A)} + I^{(S)}$$

Thus, we have:

$$\frac{dS}{dt} = -\frac{\beta SI}{N} + \xi C$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I$$

$$\frac{dR}{dt} = \gamma I - \theta R$$

$$\frac{dD}{dt} = \delta\theta R$$

$$\frac{dC}{dt} = (1 - \delta)\theta R - \xi C$$

where $N = S + I + R + D + C$.

This system of differential equations is more easily analyzable and shares familiarity with the original SIR(S) model that was first presented in class. Notice that the equations for $\frac{dS}{dt}$ and $\frac{dI}{dt}$ are identical to their counterparts in the original SIR model. So, we know that $\frac{dS}{dt}$ decreases when people become infected, resulting in an increase in $\frac{dI}{dt}$. The dynamic is then that the rate of contact between I and S will result in a decrease of S and increase of I dependent on our rate of contact constant $R_0 = \beta/\gamma$. Also, I will decrease by the rate γI that is proportional to the amount of people exiting the group of infectives. $\frac{dR}{dt}$ will increase only when individuals recover from the infective group such that its increase is directly proportional to the decrease in I. Our model is unique in the sense that we now add three new equations to classify the possible states of those infected with the virus. The number of resolving individuals (R) represents the infected individuals whose outcome is soon to be determined. This state can be determined into two final outcomes: individuals that die from infection (D) and individuals that recover from infection. We can clearly see that the sum of

$\frac{dD}{dt}$ and $\frac{dC}{dt}$ is θR, the proportion of resolving individuals from infected individuals. Thus, we can conclusively state that D and C are the only two disjoint outcomes for R.

2. Theoretical Investigations of Model:

To analyze the stability of the overall system, we can take advantage of the dependency of certain equations on each other, and the general rule where $N = S + I + R + D + C$. Since $\frac{dD}{dt}$ and $\frac{dC}{dt}$ sum to θR, it is possible to recover the equilibrium values for each by writing each equation in terms of $\frac{dR}{dt}$ then analyzing the stability of $\frac{dR}{dt}$. To analyze the stability of $\frac{dR}{dt}$ it is required to find the equilibrium points of $\frac{dS}{dt}$ and $\frac{dI}{dt}$ since $\frac{dR}{dt}$ depends on I, which means that finding the equilibrium points of the first order system of $\frac{dS}{dt}$ and $\frac{dI}{dt}$ will recover the equilibrium points of $\frac{dR}{dt}$ as well as the equilibrium points of $\frac{dD}{dt}$ and $\frac{dC}{dt}$. Our goal is to analyze the following system:

$$\frac{dS}{dt} = -\frac{S(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} + \xi C$$
$$\frac{dI}{dt} = \frac{S(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} - \gamma(I^{(A)} + I^{(S)})$$

Setting ds/dt equal to 0 and solving for S, we obtain:

$$S = \xi CN/(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})$$

Setting dI/dt equal to 0 and solving for I, we obtain:

$$(I^{(A)} + I^{(S)})(S\beta/N - \gamma) = 0$$

From which two possible values occur:

$$S = \gamma N/\beta \text{ and } I^{(A)} + I^{(S)} = 0$$

Since $I^{(A)} + I^{(S)}$ equals I, we can take this as an arbitrary value. It is convenient in this model to take the value to be ξC as this will yield an equilibrium point.

This gives the following list of possible equilibrium positions:

$$((\xi CN/(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)}),\ 0)$$

$$(\gamma N/\beta,\ 0)$$

$$(\gamma N/\beta\ ,\ \xi C\ )$$

Substituting each possible equilibrium point into the first order system:

$$\frac{dS}{dt}(eq1) = -\frac{\xi CN/((\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)}))(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} + \xi C = 0$$

$$\frac{dI}{dt}(eq1) = \frac{\xi CN/((\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)}))(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} - \gamma(I^{(A)} + I^{(S)}) = \xi C$$

$$\frac{dS}{dt}(eq2) = -\frac{(\gamma N/\beta)(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} + \xi C \neq 0$$

$$\frac{dI}{dt}(eq2) = \frac{(\gamma N/\beta)(\lambda^{(NQ)}I^{(A)} + \lambda^{(NQ)}I^{(S)} + \lambda^{(Q)}I^{(A)} + \lambda^{(Q)}I^{(S)})}{N} - \gamma(I^{(A)} + I^{(S)}) = 0$$

$$\frac{dS}{dt}(eq3) = -\frac{(\gamma N/\beta)(\beta\xi C)}{N} + \xi C = 0$$

$$\frac{dI}{dt}(eq3) = \frac{(\gamma N/\beta)(\beta\xi C)}{N} - \gamma(\xi C) = 0$$

To set up the Jacobian to test the equilibrium point $(\gamma N/\beta\ ,\ \xi C\ )$, the partial necessary partial derivatives are:

$$\frac{df}{dS} = \frac{dS}{dt}I/N$$

$$\frac{df}{dI} = S\frac{dI}{dt}/N$$

$$\frac{dg}{dS} = \frac{dS}{dt}I/N$$

$$\frac{dg}{dI} = S\frac{dI}{dt}/N - \gamma\frac{dI}{dt}$$

Where df and dg represent the partial derivatives of $\frac{dS}{dt}$ and $\frac{dI}{dt}$ respectively.

Evaluating the Jacobian at the equilibrium point yields a matrix of the following column vectors:

$$(\frac{\xi C(1-\gamma)}{N}\ ,\ \frac{\xi C(1-\gamma)}{N}\ )\text{ and }(\frac{\gamma\xi C}{N}\ ,\ \frac{\gamma\xi C(1-\gamma)}{\beta}\ )$$

Which produces the following eigenvalues:

$$\lambda = \frac{\xi C(1-\gamma)}{N}(1+\gamma\xi C),\ \gamma\xi C(1-\gamma)(\frac{1}{\beta}+\frac{\xi C}{N})$$

The dependence of both eigenvalues on C and ξ along with a positive $\gamma > 1$ as in our parameters gives two negative eigenvalues. As such, we conclude the equilibrium point of ($\gamma N/\beta$, ξC) is stable. In the same vein, since the first order system between $\frac{dS}{dt}$ and $\frac{dI}{dt}$ has a stable equilibrium point, we can conclude the overall system has a stable equilibrium state as we will see in our simulations.

3. Simulations/Data

We can test the model using the following parameters:

- γ = 0.96 (recovery rate as a percentage ~ complement to death rate)
- θ = 0.1 (10 days on average to get infected for which it may cause death before it resolves) δ = 0.04 (death rate is 4%),
- β = 3.6 (contact rate ~ scalar value based on research)
- ξ = 1 (all recovered individuals become susceptible to becoming infected again)

Most of these parameters were based off of Anzum and Islam's model and simulations from their paper [1]. The rest are based on our understanding of current interactions and reinfection rates, though we realize that they may not be completely accurate and may vary across the world.

We used the following code to simulate our SIRD Model in MATLAB:

```
%Code for simulating an SIRDC model
clear all;
close all;

totTime = 12; %total simulation time
```

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```
dt = 0.01; %time step
tvec = 0:dt:totTime;
tsteps = length(tvec); %total number of time steps

%initialize vectors for susceptibles, infected, and recovered
S = zeros(tsteps,1);
I = zeros(tsteps,1);
R = zeros(tsteps,1);
D = zeros(tsteps,1);
C = zeros(tsteps,1);

shi = 1; %re-infection probability
theta = 0.1; %incubation average
delta = 0.04; %death rate
beta = 3.6; %contact rate (you choose this!)
gamma = 0.96; %recovery rate (you choose this!)
N = 10000; %total population

%Initialize susceptibles and infected. We choose I(1) equal to some significant nonzero value for visibility.
S(1) = 9500;
I(1) = 500;
R(1) = 0;
D(1) = 0;
C(1) = 0;

for i=1:tsteps-1
    S(i+1) = S(i) + dt*((-beta*S(i)*I(i)/N) + shi*C(i));
    I(i+1) = I(i) + dt*(beta*S(i)*I(i)/N - gamma*I(i));
    R(i+1) = R(i) + dt*(gamma*I(i) - theta*R(i));
    D(i+1) = D(i) + dt*(delta*theta*R(i));
    C(i+1) = C(i) + dt*((1-delta)*theta*R(i) - shi*C(i));
end

%Plot S, I, R, D and C over time. Feel free to edit this figure to your liking.
figure(1)
plot(tvec,S,'r',tvec,I,'b',tvec,R,'g',tvec, D, 'k',tvec, C, 'c','linewidth',2)
xlabel('time')
ylabel('population')
```

We chose arbitrary numbers N = 10000, and the initial susceptible population to be 9500 and infected population to be 500 whereas the remaining groups were all 0 at time 0.

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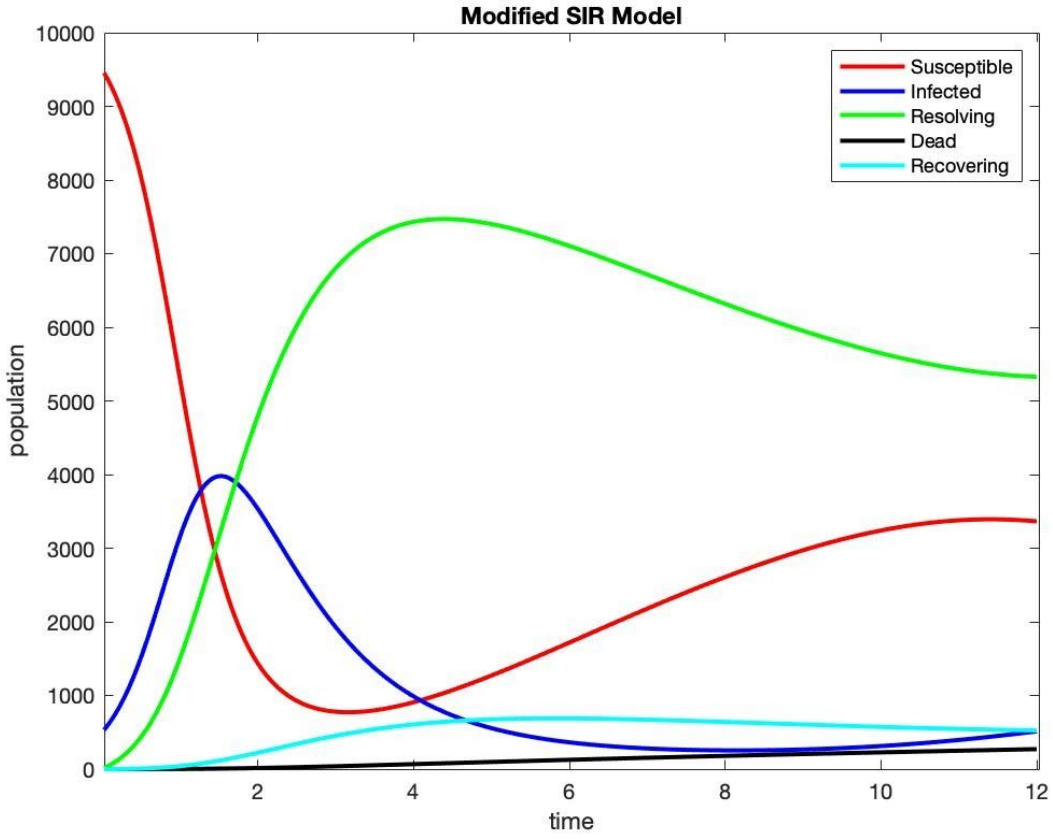

We observe that $R_0 = \beta/\gamma$ and γ equals 0.96. Further, we can assume that at time 0, S/N ≈ 1. Thus, we see that the rate of change on infectives will increase only if the contact rate $\beta > \gamma$. So, exponential spread of the virus will take form if the contact rate is greater than the recovery rate. As time goes on, it is evident that S/N is no longer 1 but at some time around 2 in our model is closer to 0. For simplicity, we can assume that it is. Then, our infectives will have exponential decay. We can assume that most recover from the disease again because of the low death rate. Thus, people go from the pool of infectives ➜ resolving ➜ susceptible such that we see increasing numbers in the cases for resolving and susceptible when S/N ≈ 0. At the same time estimate (t ≈ 2) we observe that resolving cases are growing, which is to be expected as it has a positive correlation with the number of infectives. Also very few die, and the recovery rate is very high such that increase in resolving cases is to be expected when S is low. This is also reflected by a very slow increase in the number of deaths. However, when the resolving cases are recovered, they go back into the pool of susceptibles as we expect the reinfection rate to be 1 (everyone that has covid can get it again). Hence, we see that S will with time start to increase again and reach a new peak before declination. These oscillations can reminisce how the seasonal flu operates. Even though many of us have had it at a time, we can still get reinfected. Therefore, we see the oscillation in the number of susceptibles. As infectives come from the group of susceptibles and resolving

comes from infectives, and so on these oscillations in susceptibles carry over to oscillations in infectives and resolving. Thus, we can expect that without medical intervention and governing policies (such as social distancing, face masks, hand sanitizers, etc.) that the equilibrium solution to the system may oscillate before reaching an equilibrium. From the equations we have, we can raise certain intuitive questions that are worth answering.

4. Conclusion:

In our model, we have made significant separations on different cases on our parameters such as the total population being seperated into quarantine and non-quarantine, infectives being symptomatic and asymptomatic, etc. One example we can draw from our model is that infectives in our graph is assumed to be under the entirety of the infected population. However, we have separated infectives into asymptomatic and symptomatic infections in our parametrization. Research suggests that transmission rates from the asymptomatic cases and symptomatic cases differ. This is evidently a weakness to our model as we have assumed it to be similar for both. The fact that we have seperated many of our parameters into different subcategories but still evaluated the subcategories to be equal means we have room to improve them by separating these cases into subparts with different values for a later time. Another fact that is important to consider is that governmental policies and demographics of cities can make the transmission rate change vary. In our model it is set to be constant. So, we have not accounted for variety in the transmission rate (an example is that one can expect the virus to spread faster in a city as dense as New York compared to Los Angeles, which has lower population density). Another weakness is that the re-infection rate in our graph is set to 1. In real life, we can expect antibodies to develop after infection so that one is less likely to become reinfected (meaning the probability of re-infection is assumed to be less than 1).

In the basic SIR-model from class and from one of our templates, one made the assumption that susceptibles can only decrease over time. However, the reality is that one can become reinfected. We took this into account, and it is reflected with the expression ξC which is the magnitude of recovered people that become susceptible. Thus, an important dynamic was added to our system to reflect how individuals transition from recovered into susceptibles. Also, the stability of our model is based on the complexness of our parameters that accounts for different transmission rates, differences in quarantine, etc. Thus, it gives a stronger analysis into important dynamics of the covid-spread and its equilibrium.

References

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[2] Berger, David W ,Kyle F Herkenhoff, and Simon Mongey, "An SEIR Infectious Disease Model with Testing and Conditional Quarantine," Working Paper 26901, National Bureau of Economic Research March 2020.