

Title: Pandemic Modeling and Simulation  
Course: Modeling and Simulation  
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## 1. Introduction

Towards the end of December 2019, a cluster of cases related to pneumonia were reported in Wuhan, China. By early February 2020, China recorded 811 deaths surpassing the 2003 SARS epidemic. On March 11, 2020, the World Health Organization (WHO) declared the growing outbreak a pandemic as cases of COVID-19 are reported all over the globe. At the time of this writing the author and his fellow students and colleagues are told by the state and federal government to exhibit “social distancing”—a self imposed quarantine to reduce the spread of the disease. This pandemic will no doubt be the defining calamity of the year.

The ultimate goal of this project is to build a model valid enough to be used to simulate any epidemic or pandemic event. The model can then be used to predict growth and possibly reduce the spread of diseases in an epidemic.

## 2. Background

This pandemic model is based primarily on epidemiological science. In the model, an arbitrary disease is spread within and through neighboring populations to individuals. This is to simulate the spread of COVID-19 by contact via respiratory droplets (e.g. sneezing and coughing) or through touching a contaminated surface and then their face (however, the latter is not thought to be the primary way the virus spreads). This information is provided directly from the Center for Disease Control (CDC).

Other epidemiological factors include how contagious the virus is which directly effects the infection rate. Some viruses are more contagious than others (e.g. measles is highly contagious). It is not currently known exactly how contagious SARS-CoV-2 (the specific strain responsible for COVID-19) really is, but it is presumed that it is most contagious when the disease carrier is symptomatic (i.e. the disease is in full swing). It is hypothesized by epidemiologists that the reason what makes COVID-19 a good candidate for a pandemic is its incubation period. A longer incubation period makes it difficult to determine if a person is a carrier for the disease without a testing kit (which are very limited) and until the carrier becomes symptomatic, he/she will likely have spread the disease to others.

Another factor to consider that is mobility. Indeed, the rate at which a disease spreads to other regions in a city, state, country etc. (i.e. at a ) is influenced by the spatial and mobile patterns within the region. Had the virus been contracted in a low population, rural village with little to no transportation in and out, the disease would likely have converged (flattened-out) long ago with only a few and local casualties. Then imagine the same disease being released to a handful of people at an international airport and the difference is the latter case results in an epidemic. The mobility patterns can be estimated by looking at the population distribution grid of Wuhan and imposing this data on a map of the Wuhan Metro transit system. This can be used a rough approximation of inter-city mobility.

## 3. Model Development

The first draft of this model I tried to map data from various online sources the population density and public transportation lines onto a Google map and

then tried to construct “flow” between two latitudinal and longitudinal coordinates. While the mapping of the public transportation lines was successful, finding an accurate database of population densities by lat/long coordinates was not and the tools were complicated and difficult to use.

A different approach was to simulate a single population as a 2-dimensional grid of cells where each cell has a sub-population. The sub-populations then simulate the spread of the disease within their own cell and then next the model simulates the spread of the disease to other surrounding cells. This model was simple enough to code in Python and robust enough to produce accurate real-world results. The data needed to create the model could be extracted from interpolating points from [www.worldometer.info/coronavirus.com](http://www.worldometer.info/coronavirus.com) which tracks data about COVID-19 at a global scale. Things like infection rate and incubation period could be estimated using these numbers. Exact specifications of the model will be discussed in greater detail in following sections.

#### 4. Model Description

The model is a continuous one which makes equations and assumptions about aggregated populations and is simulated in regular time steps (days). As mentioned above, it starts with a 2D grid of cells. In each cell contains a scalar value representing the cell’s sub-population. Each sub-population is subdivided into three mutually exclusive categories: 1. Susceptible (not Infected), 2. Infected, 3. Removed (either recovered from infection or dead). Keep in mind that for any cell at any point in time must be zero-sum i.e.  $\text{Susceptible} + \text{Infected} + \text{Removed} = \text{size of sub-population}$ . Every cell in the grid starts off with only Susceptible state populations. The way the size of each sub-population was distributed for each cell was by imposing a 2D Gaussian curve over the grid. The height at each cell of the Gaussian represents the proportion of the global expected population (arbitrarily set to 1,000,000) size for that cell. Multiplying each cell by this expected population then gives the actual sub-population size for that cell.

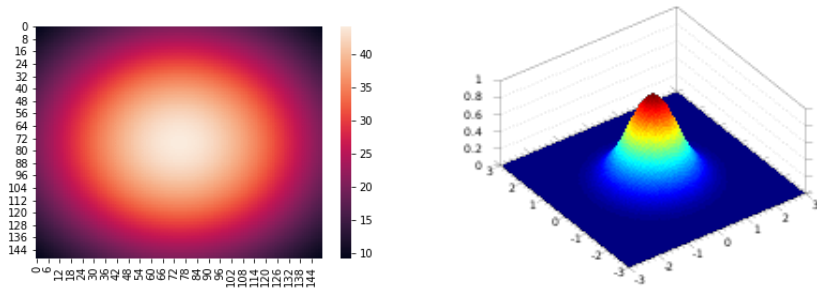


Figure 1: (a) The actual heatmap created by matplotlib in the simulation which is a Gaussian distribution over a 150 by 150 grid. The higher intensities in the center implies larger sub-population ( $> 40$  people). (b) A picture of 2D Gaussian for reference. The larger spread in (a) is done by increasing the standard deviation.

Next step was to introduce Infected cells into the population. Two cells were chosen randomly to be infected ones. The proportion of Infected was also

chosen randomly. Once chosen, the number of Susceptibles in these cells had to be appropriately deducted to maintain zero-sum rule.

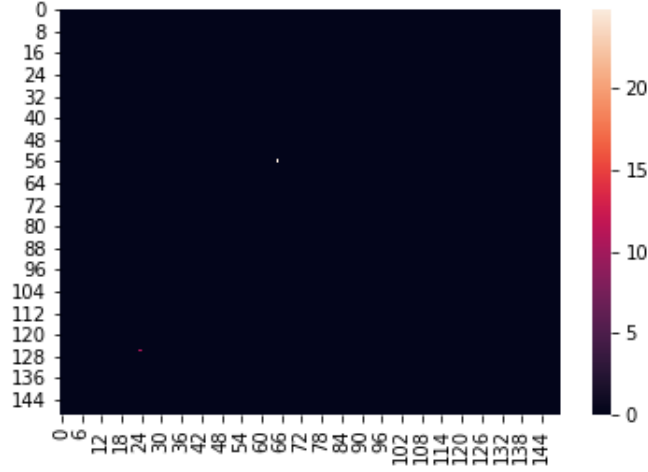


Figure 2: Two Infected cells in a separate Infected grid. Note that all other cells are black meaning there are no other Infected cells in the initial state.

After the infected cells were chosen, the model would simulate a single day by starting from an Infected cell (once a cell has more than 1 infected it counts as an infected cell) and “spreading” its infection to other random cells which were chosen by a random Gaussian process (cells closest to it were more likely to be infected than the ones further away). This was done for every cell that contained a non-zero infected population. After that, each cell was to undergo its own self-contained intra-cell (intra = within) infection process where Susceptible were converted to Infected and Infected were converted to Removed.

The procedure would be repeated until no Susceptibles remained or within a finite (10-40 days depending on the constraints, more on this later) whichever came first.

## 5. Verification and Validation

Now that a high-level overview of the model has been explained, we dive deeper into the details of the model. We discuss two important modeling choices: How intra-cell infection step is modeled and second how the inter-cell spreading step is simulated.

### Intra-Cell Spreading

Within each sub-population, Susceptibles are converted to Infected and then to Removed states. The rate at which these occur can be modeled using differential equations. Indeed, in 1927 A.G. McKendrick and W.O. Kermack developed the Kermack-McKendrick theory which gave rise to the SIR (Susceptible, Infected, Removed) model that predicts the distribution of infectious disease over time. The SIR model is used in this project for the intra-cell spreading step.

Let  $S(t)$ ,  $I(t)$ ,  $R(t)$  be the number of susceptibles, infected, and removed at a given time  $t$  respectively and let  $N$  be the sum of the three (always constant). Let  $\beta$  be transmission rate per person per unit time then we have our first differential equation, the Susceptible Equation:

$$\frac{dS}{dt} = -\beta I(t) \frac{S(t)}{N} \quad (1)$$

This says that the rate that susceptibles are converted to infected is related by the total number of infected times the transmission rate times the proportion of susceptibles. Assuming a purely heterogeneous population, an infected person comes into contact with a susceptible with probability  $\frac{S(t)}{N}$ , then eq (1) describes the expected number of new infected individuals at time  $t$  which is equal to the rate at which  $S(t)$  declines.

Next let  $\gamma$  be the recovery rate per person per unit time. Then the Infected Equation is as follows:

$$\frac{dI}{dt} = \beta I(t) \frac{S(t)}{N} - \gamma I(t) \quad (2)$$

The first term in this equation describes the rate at which new infected are accumulating (which is exactly the negative of eq (1)). The second term describes the rate that infected are being removed (recovered or dead) which is the rate of recovery per person,  $\gamma$  times  $I(t)$  the total number of infected. This equation is an example of inflow - outflow.

Lastly, we have the Recovery/Removed Equation:

$$\frac{dR}{dt} = \gamma I(t) \quad (3)$$

Which says the rate of recovery is equal to the rate individuals are being converted from infected to removed (the last term in eq (2)).

It is helpful to think of each of the equations with a flow diagram. Imagine three cylinders stacked on top of each other, Susceptible, Infected, and Removed respectively with water (uninfected people) contained only in the Susceptible cylinder at the start. Then a spout from S is opened and pours into I and a spout in I is opened which pours into R. The SIR equations describe the rate at which these spouts allow water to flow into each cylinder.

So for each “day” event in the simulation, these set of equations would be used to calculate the change in each of the three quantities for each sub-population by using Euler’s method of integration and letting  $\Delta t = 1$ . In the simulation the three values for S, I, and R are stored in three separate grids and for any cell  $(i, j)$ ,  $S_{i,j} + I_{i,j} + R_{i,j} = N_{i,j}$  where  $N_{i,j}$  is the total size of the sub-population.

The following will explain how this model determined the constant values of  $\beta$  and  $\gamma$ . In the study of epidemiology,  $R_0$  is the expected number of people in a susceptible population that a single infected person will spread the disease over the entire course of their infection. It is modeled formally by the following equation:

$$R_0 = \frac{\beta}{\gamma} \quad (4)$$

Where  $\beta$  and  $\gamma$  are the daily transmission rates and recovery rates respectively. Note that if  $R_0$  is less than or equal to one, the disease remains endemic (restricted at a controllable level) and for values greater than one, the disease can be become epidemic. The value of  $R_0$  was taken from various sources online which is estimated to be anywhere between 1.9 - 4.0 (taking the upper bound of 4 for this simulation but of course can be changed for different runs). The value for  $\gamma$  can be estimated by taking the reciprocal of the mean infection duration. If  $t_r$  is the mean infection period of a disease in days, then  $t_r^{-1} = \gamma$  is the average rate of recovery which is why in eq (3),  $\gamma I(t)(\Delta t := 1)$  equals the total number of people recovered on day  $t$ . The mean infection period of COVID-19 was taken from various sources and estimated to be around 9 days. Knowing these two parameters,  $\beta$  was solved for using eq (4) and estimated to be around  $\frac{4}{9}$  which can be interpreted as every 9 infected people infects 4 susceptible people every day.

### Inter-Cell Spreading

As discussed in section 4, before intra-cell spreading occurs, an inter-cell spreading phase happens. One of the two ways this model simulates the spreading of a disease to different locations (cells) is by using a stochastic process. It was stated earlier that each cell randomly chose neighboring cells using a 2D Gaussian distribution centered at itself and this is all there is to it. Every cell chooses a list of 8 neighbors to be its inter-cell target for the remainder of the simulation. The number 8 was chosen because a cell can technically have at most 8 neighbors (north, east, south, west, and the inbetweens). 2D Gaussian was chosen because it reflects how people in a city tend to move/travel closer distances with higher probability than further distances.

The other inter-cell spreading simulation technique used is a well-known algorithm in computer science called breadth-first search (BFS). Starting from the initial (randomly chosen) infected cells, each infected cell targets its 8 most immediate neighboring cells. The number of new infection is simulated by applying eq (1) where  $I(t)$  is taken from the center cell and  $\frac{S(t)}{N}$  are the neighboring cells. The newly infected cells are put into a queue data structure and the procedure is repeated until all cells in the grid have been visited. The effect has one that looks like the disease is gradually emanating from a source. The reason this method was chosen as an alternative was to simulate the effects of a

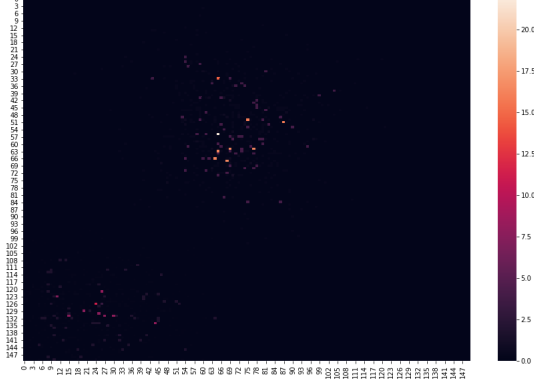


Figure 3: Gaussian inter-cell spreading. Notice the clusters spreading randomly from the initial points

stay-at-home quarantine which is imposed almost globally during the COVID-19 pandemic. We will see in greater detail in the next section how effectively a quarantine can reduce the spread of disease.

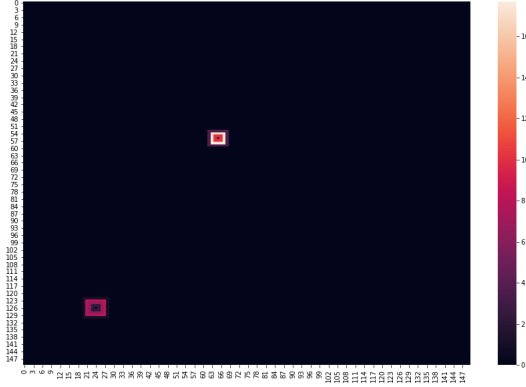


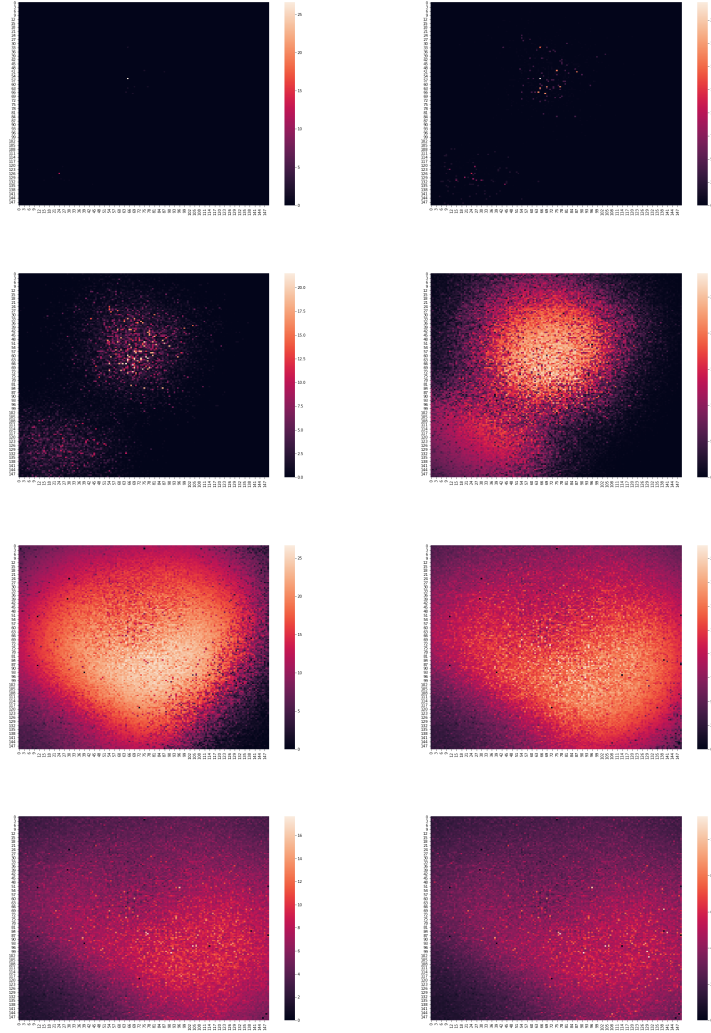
Figure 4: Breadth-First Search inter-cell spreading. Notice the more uniform spreading of the infection from the sources.

## 6. Model Application and Transition

Both the Gaussian and BFS models were simulated using Python. Recall, the Gaussian simulation represents a more liberal mobility pattern than the rigid BFS simulation. Therefore, the Gauss and BFS models can be thought of as a non-quarantine and quarantine simulation respectively. But to better simulate a quarantine effect, the values of  $\beta$  (transmission rate per infected

person per day) is reduced for the BFS model to  $\beta = \frac{2}{9}$  (half of the previous value) to model a slower daily person-to-person contact rate during a quarantine (i.e. “social distancing”).

Figure 5: Simulating with Gaussian spreading. Each image was saved at the end of every 5th day in a 40 day simulation. Notice in the last two images, the once bright areas become dark again, this is because the sub-populations in those cells are recovering.



## 7. Conclusions and Recommendations

From the results it is clear that mobility plays a critical factor in fueling a pandemic. By reducing the amount of travel in an epidemic region, the spread of the disease is reduced.



In January 2019, the WHO published a SEIR model specifically for COVID-19 in Wuhan.

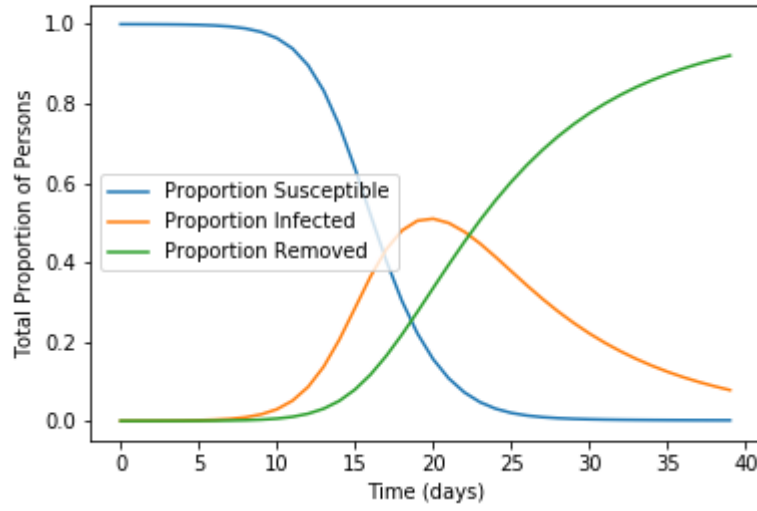


Figure 6: Gaussian spread. The plot of proportions of S, I, R over a 40 day period.

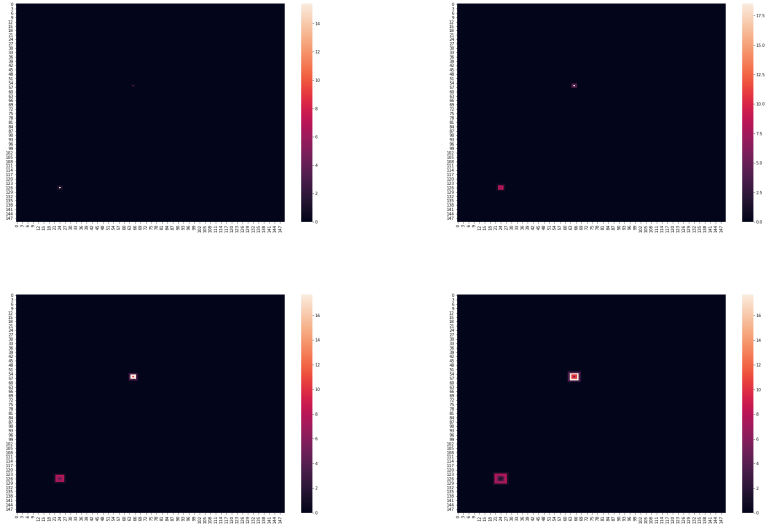


Figure 7: Simulating with BFS. Each image taken every 10 days over a 40 day period.

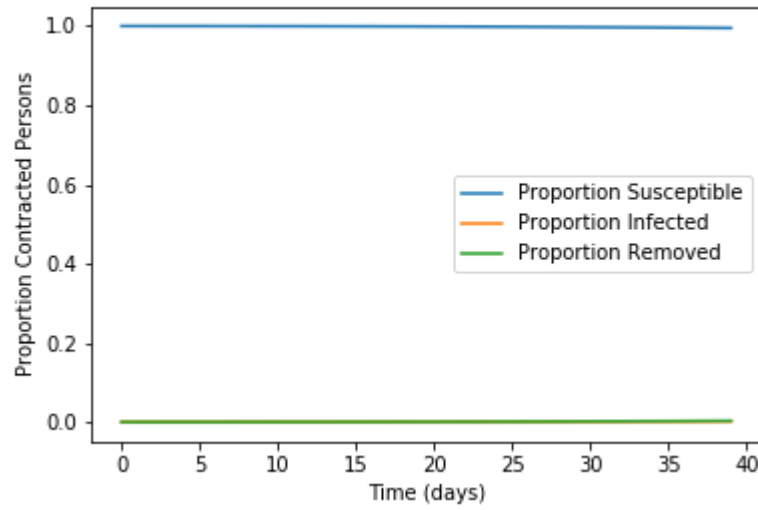


Figure 8: BFS spread. The plot of proportions of S, I, R over a 40 day period.

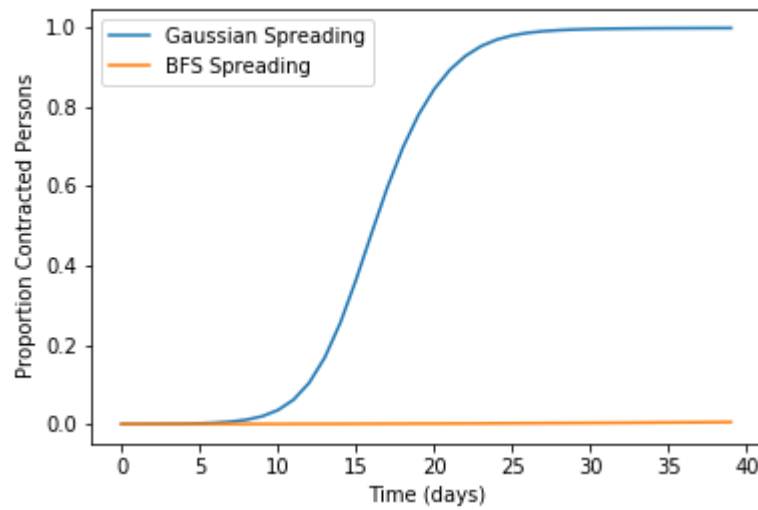


Figure 9: A comparison in the total removed over time in each model.