

Susceptible-Infected-Removed (SIR) Disease Spread Model Simulations and Variations

Yu An

Frieda Han

Yun Lin

Abstract

The project investigates a brief introduction of the Susceptible-Infected-Removed (SIR) Model and implements it through agent-based, ODE continuous and spacial simulation, indicating that this model can be quite useful in contagion studies. By reviewing a few existing literature, some extensions as well as variations under the SIR model are explored, which includes considering the effect of quarantine, estimating a time-varying parameter, and introducing a new strain.

Keywords: SIR Model, disease spread, infectious disease, contagion modeling

1. Introduction to The SIR Model And Its Variations

1.1. Basic SIR Model

The SIR (susceptible-infected-removed) model, contributed by Kermack, W. O., McKendrick, A. G, Ronald Ross and others, was formed in the early twenties and made a big breakthrough in modeling the trend of the possible disease outbreak as well as providing key information for the public and authorities regarding severeness and control. Under a fixed homogeneous population (N), three distinct subpopulations are divided ($S + I + R = N$):

1. S - those who are susceptible and have not yet caught the disease
2. I - those who are infected and may spread the disease
3. R - those who have recovered without the ability to spread and are immuned to the disease

These groups are divided in a way that during an epidemic, people are interested in the amount of infected patients that can transmit the disease, the number of agents who are immuned against the disease and the portions who are not yet being infected and are susceptible. Denote the sizes of these groups at time t by $S(t)$, $I(t)$, and $R(t)$. Moreover, introduce notations involved in this model:

1. b: a fixed number of interactions that spread the disease per day per infected individual
2. k: a fixed fraction of the infected subpopulation that recovers per day

Altogether, the SIR model consists of a system of three ordinary differential equations (ODEs):

$$\begin{aligned}\frac{ds}{dt} &= -b * s(t) * i(t) \\ \frac{di}{dt} &= b * s(t) * i(t) - k * i(t) \\ \frac{dr}{dt} &= k * i(t)\end{aligned}$$

where s, i and r here are the percentage of each group size over the total population.

1.2. SUQC Model – The Use of Quarantine

The SIR model assumes the frequency of interaction between one and the other is kept stable even when one of them is infected. According to actual situation, we can see that many countries/regions try to use quarantine to reduce the interactions between people, which is the parameter b in the SIR model. Moreover, under the case of COVID-19, there's a time delay between one being infection and being confirmed as sick. Therefore isolating infected patients is an effective way to reduce the rate of disease transmission. Based on this idea, we propose the following SUQC model to describe this situation.

In this model, the total population N is divided into 4 parts:

1. S - the individual who has not yet caught the disease
2. U - the non-quarantined infected individual who may spread the disease to susceptible individuals
3. Q - the quarantined infected individual who loss ability of infecting susceptible individuals
4. C - the individual who is confirmed sick

All parts above are functions of time t .

The number of removed individuals R is not included. This model assumes that once the infected are quarantined, we assume that the probability of infecting susceptible individuals is zero. So whether they are recovered/removed won't affect the whole dynamic system. This model exerts more focus on the direct impact of quarantine measures on disease spread.

There are 3 parameters involved in this model:

1. α : the number of individuals infected by an non-quarantined individual per day
2. β : the rate at which the cases are confirmed
3. γ : the rate at which non-quarantined infected individuals get quarantined

which gives us the following differential equations:

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\alpha * U(t) * S(t)}{N} \\ \frac{dU}{dt} &= \frac{\alpha * U(t) * S(t)}{N} - \gamma * U(t) \\ \frac{dQ}{dt} &= \gamma * U(t) - \beta * Q(t) \\ \frac{dC}{dt} &= \beta * Q(t)\end{aligned}$$

1.3. SIRDC model – The Introduction of A New State

In the SIR model, R subpopulations contains those who have recovered or have died. The real-world case can be a bit more complicated. In previous studies, researchers have found that it seemed important to separate the infectious and the recovering phase. From a biological point of view, an infectious period might average out about 4 to 5 days while resolving period average out about 14 to 20 days. Hence, it can be problematic when fitting real-world data on the SIR model. The introduction of the Susceptible-Infected-Recovery-Death(SIRD) model helps distinguish the two states. After infection, a person then comes to the 'Resolving' state with two consequences: either recovered or died. The SIRD model clarifies the transmission process and help explore the fatality in evaluating the severeness of the disease.

In this model, the total population N is divided into 5 groups:

1. $S(t)$ - the number of individuals susceptible
2. $I(t)$ - the number of individuals infected
3. $R(t)$ - the number of individuals that are being resolved
4. $D(t)$ - the number of individuals that died from the disease
5. $C(t)$ - the number of individuals that recovered from the disease

Similar to the SIR model, some of the assumptions for SIRD model requires a fixed homogeneous population with C subpopulation permanently immuned and the I subpopulation contagious at the moment of infection.

There are 4 parameters involved in this model:

1. β : a fixed number of interactions that spread the disease per day per infected individual
2. γ : a Poisson rate of infectiousness resolves
3. θ : a fixed fraction of people exiting the resolving state
4. δ : a fixed fraction of the resolved consequences being died per day

which gives us the following differential equations:

$$\begin{aligned}\frac{dS(t)}{dt} &= -\frac{\beta * S(t) * I(t)}{N} \\ \frac{dI(t)}{dt} &= \frac{\beta * S(t) * I(t)}{N} - \gamma * I(t) \\ \frac{dR(t)}{dt} &= \gamma * I(t) - \theta * R(t) \\ \frac{dD(t)}{dt} &= \delta * \theta * R(t) \\ \frac{dC(t)}{dt} &= (1 - \delta) * \theta * R(t)\end{aligned}$$

1.4. SIIRR model – The Appearance of A New Strain

It happens for diseases to reemerge due to the appearance of a new strain. An original strain may mutate through the antigenic shift or antigenic drift. The resulted newer strain does not exhibit cross-immunity with the original one, which means that people who recovered from the original strain will become susceptible to the new strain. This situation can be modelled by performing modifications of the SIR model.

A new strain is often introduced when an equilibrium has been achieved by the original strain. It has transmission parameter b' and the recover parameter k' . Instead of three categories S , I and R in the SIR model, new categories are created:

1. S : an individual who has not yet caught the disease, but may be infected by the original strain or the new strain.
2. I_1 : an individual is infectious of the original strain.
3. I_2 : an individual is infectious of the new strain.
4. R_1 : People who have removed after being infected by the original strain. Part of them have recovered from the disease while others have died. People who recovered is susceptible of the new strain. Here we suppose the number of people died is proportional to size of infectious population of constant d .
5. R_2 : People who have removed after being infected by the newer strain.

Similar to SIR model, system of differential equations can be expressed as:

$$\begin{aligned}\frac{ds}{dt} &= -bs(t)i_1(t) - b's(t)i_2(t) \\ \frac{di_1}{dt} &= bs(t)i_1(t) - ki_1(t) \\ \frac{di_2}{dt} &= b'i_2(t)[s(t) + (1 - d)r_1(t)] - k'i_2(t) \\ \frac{dr_1}{dt} &= ki_1(t) - b'i_2(t)(1 - d)r_1(t) \\ \frac{dr_2}{dt} &= k'i_2(t)\end{aligned}$$

2. Results And Discussion

2.1. The Basic SIR Model

In this section, the population size is assumed to be 10000. The initial condition is that 0.1% of the population has been infected, while others remain susceptible. b denotes the number of interactions each day that could spread the disease (per individual) and k denotes the fraction of the infectious population which recovers each day.

2.1.1. ODE Model Simulation

From the Midterm report, the results can be summarized as:

1. If $k = b$, there will be a very small surge of infectious population (less than 10% of total population), so the susceptible population decrease a bit. Soon the infectious population will decrease to around zero since they are removed (recovered or dead).
2. If $k > b$, there will almost no surge of infectious population, the initial infectious people will soon removed. The susceptible population remains uninfected.
3. If $k < b$, there will be a significant surge of infectious population. As k is significantly larger than zero, the infectious population will eventually recovered. Moreover, when $k < b$, the larger the difference between b and k , the greater the surge will be. When b is larger than 1, it can be seen that almost the entire population will have been infected and recovered.

2.1.2. Agent-based model simulation

As checked in the Midterm report, the example plot and phase diagrams for agent-based model simulation agrees with that for ODE simulation (See Appendix).

2.2. The Spacial SIR Model

The basic SIR model developed by Kermack and McKendrick does not consider the population distribution in space. Jaewook Joo and Joel L. Lebowitz helped make up for the deficiency by considering a spatial model through the distribution of stochastic processes. In this section, an Agent-based model and a PDE model are simulated to model the spatial distribution of individuals.

2.2.1. Agent-based Model Simulation

Apart from the S , I , R state that separates the whole population, each agent also has a position within a unit square, represented by (x, y) , where $0 \leq x \leq 1$ and $0 \leq y \leq 1$. Movement of people will be modelled by making each individual take a step in a random direction of length ‘ p ’ at each time step. We set a radius q , within which all other people will be infected if the agent is in an infectious state. We chose following parameter value for agent-based model simulation: $N = 10000$, $I_0 = 10$, $T = 300$, $b = 1/2$, $q = \sqrt{b/(N * \pi)}$, $k = 1/20$, $p = [0, 0.1, \dots, 0.9]$, and set the initial break out to be the nearest I_0 people closest to the center the square $(0.5, 0.5)$.

- **Disease spreads with different step length**

By simulating with different step length p , we keep track on the time evolution of S , I and R subpopulation size.

From the plot(Figure 1), with increasing step length p , the peak of the infectious population increases first, and then decreases, plus it is faster for the disease to spread. The peak of infectious population size is maximized when p is 0.2.

From the plot(Figure 2), when p is 0, no people is being infected. The total number of people that have been infected also increases and then decreases with increasing step length p .

The simulation above reveals that with $k = 1/20$ and $b = 1/2$, the disease is unlikely to spread if people stay still. However, if people move in relatively small steps, there will be a relatively large and fast breakout of the disease. If people move in relatively large steps, the breakout of the disease will be slow and small.

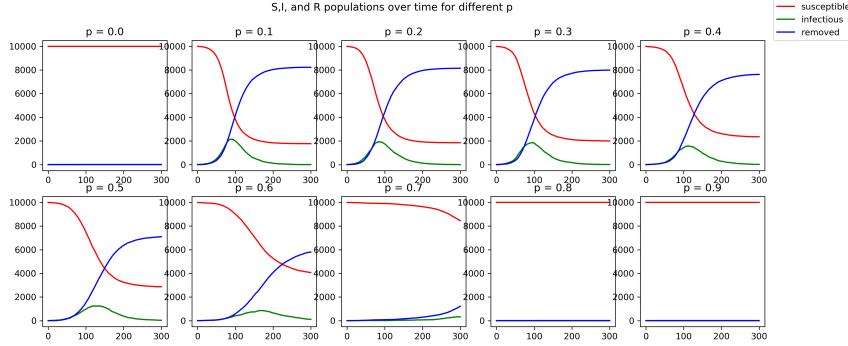


Figure 1: Plot of agent-based model with different step length p

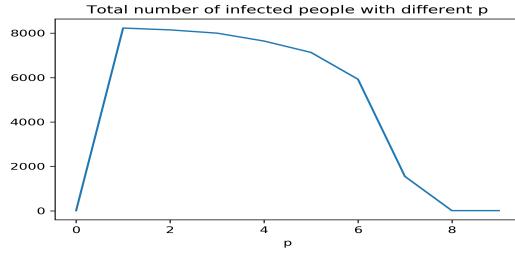


Figure 2: Plot of total number of people infected with different step length p

- **Disease spreads with different start position**

In this part, how different start position of initial infectious people affects the spread of disease is explored. We compare starting at *center*, *corner* and *random* position:

1. Center: I_0 people closest to the center of the square ($[0.5, 0.5]$)
2. Corner: I_0 people closest to the corner of the square ($[0, 0]$)
3. Random: I_0 people randomly distributed within the square

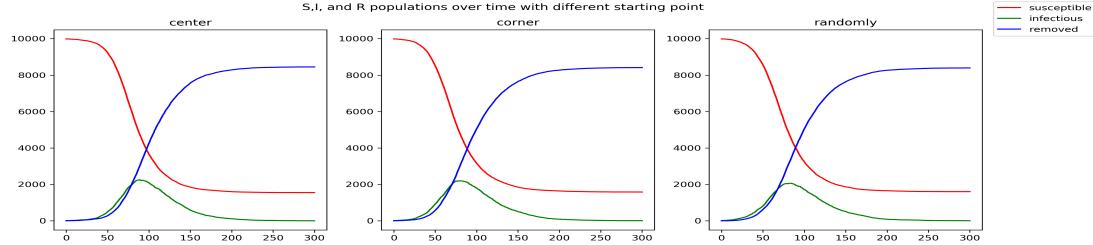


Figure 3: Plot of agent-based model with different start position

From the plot (Figure 3), the time evolution of S , I and R subpopulation size is extremely similar. This result contradicts the intuition that the disease start at the corner may spread slower, since people can only move in 1/4 of the direction and disease start randomly may spread faster. However, it can be explained by the previous part. Larger step size are equivalent to a more sparse starting position, thus a randomly initiated start will not necessarily lead to a larger breakout. On the contrary, the peak comes slightly slower and the total number of people infected is slightly lower.

2.2.2. PDE Model Simulation

The continuous model simulation total population on a unit square with $M \times M$ grid. Each time, $S(x, t)$, $I(x, t)$ and $R(x, t)$ are three $M \times M$ grids where $x = (i, j)$ is the index on the $M \times M$ grid. Similarly,

p is the step size of movement of the individuals on the grid. The parameters used in this simulation: $M = 100$, $N = 10000$, $I_0 \approx 10$, $T = 150$, $k = 0.4$, $b = 0.6$, $plist = [0.01, 0.1, 0.2, 0.3, 0.4, 0.5]$.

- **Disease spreads with different step length**

In this part, the initial infected population is gathered in 5×5 grid at the center of the grid. Then with different step size p , we keep track on the time evolution of S , I and R subpopulation size and their dynamics.

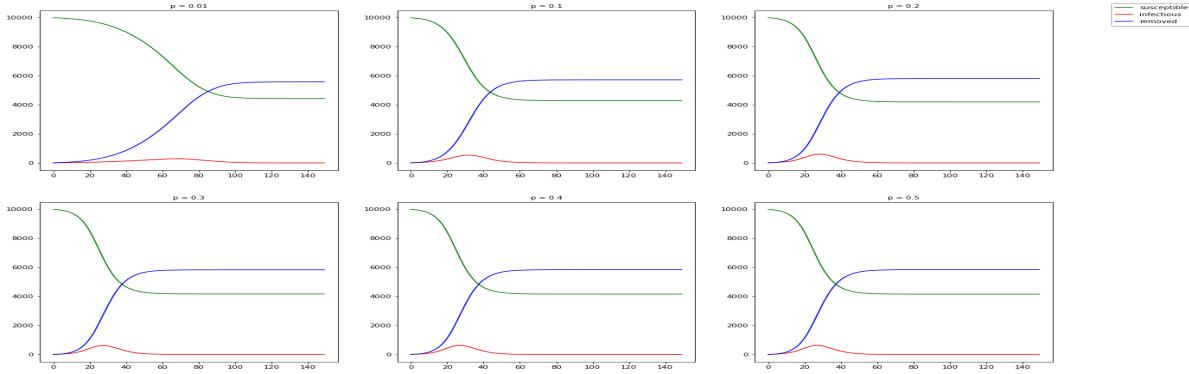


Figure 4: S, I and R subpopulations with different step size p

p	0.01	0.1	0.2	0.3	0.4	0.5
maxima of I	285.71	552.43	616.77	631.27	634.86	635.68
time of maxima	69	33	30	29	29	29
total infected ($I + R$)	5581.31	5728.33	5808.24	5830.18	5836.05	5837.82

From the plot (Figure 4) and data above, as step size p increases, the maximum value of I (population of the infected) will also increase, and the time when the maximum value of I appears will be earlier. Moreover, the total number of final infections ($I + R$) also increases slightly. However, the rate of rise and fall will gradually decrease. This means that as the distance of travel increases, infected individuals are more likely to infect other people and the spreading goes faster.

- **Disease spreads with different start position**

This part analyzes how different start position of initial infectious people affects the spread of disease is explored. The positions are similar with those in agent-based part.

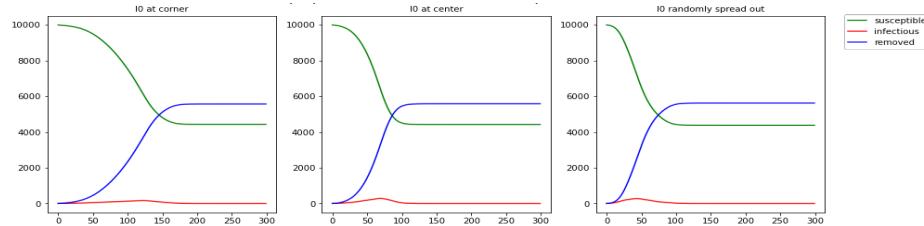


Figure 5: S, I and R subpopulations with different position I_0

position	I_0 at corner	I_0 at center	I_0 randomly spread out
maxima of I	166.35	285.61	449.62
time of maxima	121	68	32
total infected ($I + R$)	5570.42	5579.99	5696.78

The plot (Figure 5) and data above show how the starting position of the initial infected individual affects the dynamics of the system. Among the three cases, when I_0 (the initial infected individuals) is randomly spread out in the area, the number of the infected (I) reaches its top the earliest and the maxima number is the largest. Also the total number of final infections ($I + R$) is also the largest among three cases. On contrary, when starting in a single corner of the square, the maxima of I and the final number of ($I + R$) is the smallest and the time that maxima appears is the latest. This shows that scattered infected people are more potentially dangerous than clustered infected people, because scattered allows them to reach more uninfected people, which as a result makes more people infected.

2.3. SUQC Model

The ODE simulation of SUQC Model is based on the system of differential equations mentioned in Section 1. The parameters involved are: $N = 10000$, $T = 100$, $I_0 = 10$, $a_{list} = [0.8, 1.5, 8]$, $b_{list} = [0.1, 0.5, 1]$, $c_{list} = [0.1, 0.5, 0.8]$. The balance day is approximately calculated by $\lceil \frac{dS}{dt} \rceil$, and the balance number of the susceptible are also rounds up to the nearest integer.

- Disease spreads with different number of individuals infected by an non-quarantined individual per day

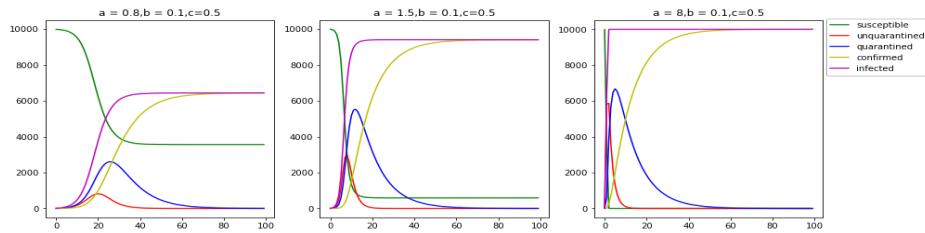


Figure 6: Compare $S U Q C I$ subpopulations with different a

a	0.8	1.5	8
balance day	51	24	3
balance number of the susceptible	3562	594	1
balance number of the infected	6438	9406	9999

According to the graph (Figure 6) and data above, with a larger a , the system reaches the balance state more quickly. Also there will be more infected individuals and less susceptible individuals after this time period.

- Disease spreads with different rate at which non-quarantined and infected individuals are quarantined

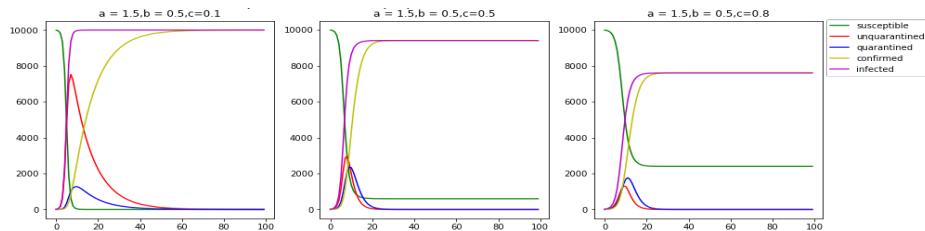


Figure 7: Compare $S U Q C I$ subpopulations with different c

c	0.1	0.5	0.8
balance day	14	24	26
balance number of the susceptible	2	595	2401
balance number of the infected	9998	9405	7599

From Figure 7, the parameter c represents the rate at which non-quarantined and infected individuals get quarantined. With the data above, it is easy to see that, promoting quarantine plays a significant role in decreasing the total number of infected individuals.

2.4. SIRDC Model

The ODE simulation of SIRDC Model is based on the system of differential equations mentioned in Section 1. The parameters involved are: $N = 10000$, $T = 100$, $I_0 = 10$, $\beta_{list} = [0.8, 1.5, 8]$, $\gamma_{list} = [0.15, 0.5, 0.6]$, $\theta_{list} = [0.1, 0.2, 0.3]$, $\delta_{list} = [0.01, 0.1, 0.5]$. Similar to the SUQC Model, consider the equilibrium to be approximately calculated by $\lceil \frac{dS}{dt} \rceil$, and assume the counts of each subpopulations at the equilibrium to be an integer.

- Disease spreads with different number of interactions that spread the disease per day per infected individual

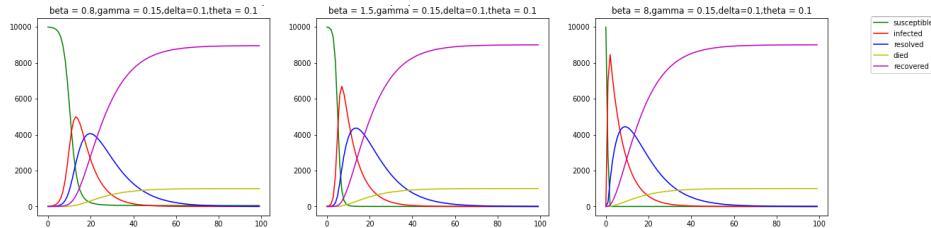


Figure 8: Compare $S I R D C$ subpopulations with different β

β	0.8	1.5	8
Day	68	35	4
number of the susceptible	49	0	0
number of the infected	2	116	6266
number of the resolved	98	1181	3166
number of the dead	984	870	56

From the plot(Figure 8) and data above, as β increases, the equilibrium date becomes earlier. At equilibrium state, with rising β values, the number of susceptible, dead individuals decreases while the number of infected and resolved individuals increases. Eventually, all counts of subpopulations seem to end up with the same value. This makes sense as β increases, the number of interactions between the infected and the susceptible increases, so that the equilibrium of the infected and the susceptible is reached at an earlier state with a higher amount of infected subpopulations. However, it does not affect the total outcome, so as time goes by, the counts end at the same value.

- Disease spreads with different Poisson rate of infectiousness resolves

γ	0.15	0.5	0.6
balance day	68	73	91
balance number of the susceptible	49	3564	5435
number of the infected	2	0	0
number of the resolved	98	48	12
number of the dead	984	638	455

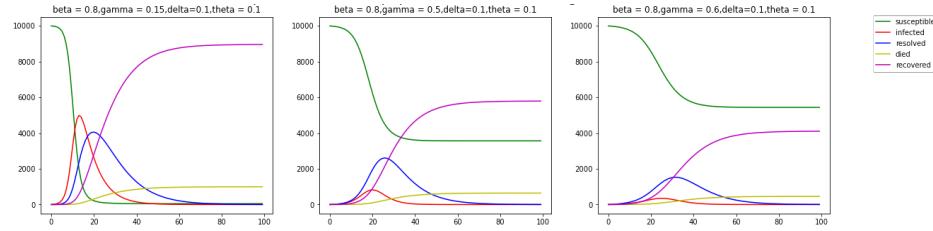


Figure 9: Compare $S I R D C$ subpopulations with different γ

From the plot (Figure 9) and data above, as γ increases, the equilibrium date becomes later. At equilibrium state, with rising γ values, the number of infected, dead individuals decreases while the number of susceptible individuals increases. Eventually, the counts of subpopulations seem to end up with the same trend as at the equilibrium state. This makes sense as γ increases, the average length of time a person is infectious decreases ($\gamma = 0.15$ means a 7-day duration), so that the equilibrium of the infected and the susceptible is reached at an later state with a lower amount of infected subpopulations.

For different choices of δ and θ , the proportion of amount of the dead and the recovered subpopulations changes, but does not affect the equilibrium state of the susceptible and the infected. (See Appendix).

- **Disease spreads with time-varying number of interactions**

Suppose now β_t is a time-varying variable. If policies that can decrease the chances of interactions between people, like social distancing or lockdown are practiced, β_t would be diminishing through time until slightly fluctuates around some constant number. From the systems of differential equations in Section 1, $\beta_t = \frac{N}{S_t}(\gamma + \frac{\Delta I_t}{I_t})$. Suppose a lockdown is being practiced and β_t decays after the date of the lockdown. The initial value is chosen to be $\beta_{t0} = 0.8$ for comparison purpose.



Figure 10: Compare $S I R D C$ subpopulations with time-varying β_t

From the plot (Figure 10), the left one shows how β_t varies with time while the right one shows the trend of each subpopulations. At Day 12 when the lockdown is implemented, the value sharply drops, indicating that the interactions of the infected and the susceptible decreases drastically and around Day 20, the value stays stable at a constant level. As for the equilibrium state, on Day 47, the number of susceptible stays constant at 311, with number of infected to be 68, number of resolved to be 717, number of died to be 890.

Comparing the result with fix value $\beta = 0.8$, the equilibrium comes at an earlier state, and the number of susceptible is higher while the counts of infected is lower. The final outcome of the number of susceptible is also higher for time-varying β_t , as eventually β_t becomes smaller than $\beta = 0.8$. This indicates how a lockdown can be effective by reducing the number of susceptible being infected.

2.5. SIIRR Model

By a modified agent-based model, we studied how time when the new strain appears influences the spread of disease. It can be derived from the basic model that when $b = \frac{3}{5}$ and $k = \frac{2}{5}$, the peak of

infectious people is approximately 80 at the 25th time step and virus is eliminated at the 50th time step. Then we introduced a new strain at $T_1 = [5, 10, 15, 20, 25, 30, 35, 40]$. The new strain is normally more contagious than the original strain, thus we set $b' = 1$ and $k' = 1/5$. We then track the time evolution of infectious population size for both the original and the new strain.

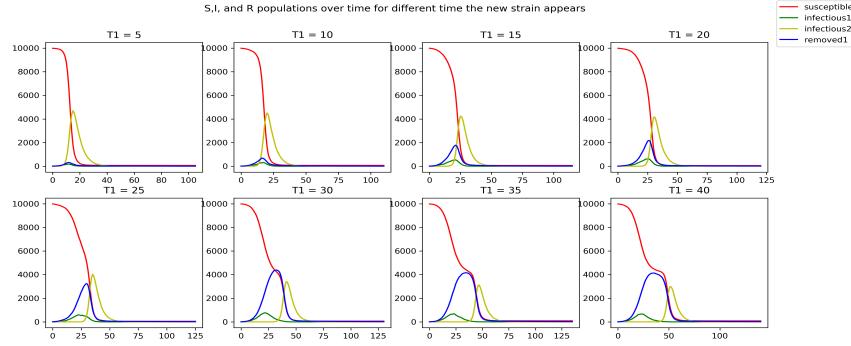


Figure 11: Plot of agent-based model with different appearance time of the new strain

From the plot(Figure 11), the surge of new strain influences the spread of the original strain if the original strain is not eliminated at that time. Moreover, different times of the surge of new strain lead to distinct spread progress. If the new strain emerge before the peak of the infectious population of original strain, the spread of the disease caused by the new strain will dominate the spread of the original strain. As the new strain emerge later, the spread of the original strain is less influences. The infectious population size of the new strain has a lower peak.

Then simulate with different b' and k' to see some patterns:

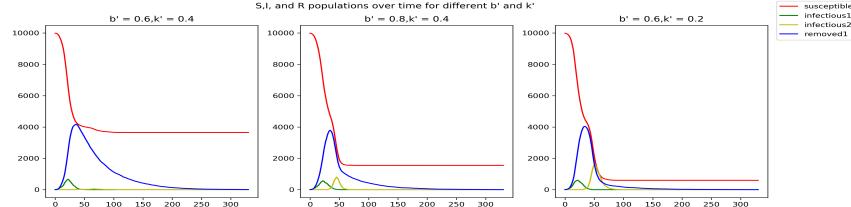


Figure 12: Plot of agent-based model with different b' and k'

If b' and k' is same as $b = 0.6$ and $k = 0.4$, the new strain almost has no surges. If b' is a bit greater, the new strain has similar peak value to the original strain. If k' is a bit smaller, the new strain has greater peak value than the original strain.

3. Conclusion

From the results above, SIR model and its variations seem to be effective in contagion modelling, which can be quite useful in real-world disease spreading predictions, prevention and control work. However, some assumptions of SIR model can bring about limitations. The closed population has no social structure and the I subpopulation has to be contagious at the moment of infection. Moreover, the model assumes a mass-action mixing, meaning the fraction of interactions between S and I is proportional to the product of their population sizes. This implies that I and S are homogeneously distributed in space.

Specifically, the spatial model helps resolve the mass-action mixing problem to some extent. To slow down the spread of disease, authorities can promote quarantine to limit people's range of activities. Moreover, gather the infected or confirmed populations and isolate them within the planned scope can also be a good strategy in controlling the spread of the disease.

From the SUQC Model, the sooner the infected are quarantined, the fewer contact they will make with uninfected people, and as a result, the fewer people will eventually be infected. The limitation of this model can be that the variables are all independent constants. However, intuitively speaking, all variables will change over time and influence each other. For example, when a large number of infections are confirmed in a certain area, people will be more likely to stay at home instead of wandering around. In addition, the model only considers the isolation of infected cases, and susceptible people can also take isolation measures to reduce potential risks.

From the SIRDC Model, the lower the average length of time a person is infectious, the smaller amount of people will be infected. Also, the time-varying parameter shows the effectiveness of a lockdown by modeling the interaction variable to be diminishing. Future studies can fit data into the model and make predictions for disease control.

From the SIIRR Model, if the new strain emerge later, the spread of the original strain is less influenced and the infectious population size of the new strain has a lower peak. Moreover, the time evolution of the two infectious population size can be used to study the infectiousness of a new strain.

4. Bibliography

The SIR model is often credited to Kermack, W. O., McKendrick, A. G, Ronald Ross (1) and early papers that contains the idea of basic SIR Model can be found (3)(8)(7). Some assumptions and limitations of the SIR Model are summarized in (9). An accessible introduction to spacial SIR model is (6). The variation of SUQC Model is based on (10), the implementation of the SIRDC Model are referenced from (4)(2) and the SIIRR model is built upon (5).

References

- [1] R. M. Anderson. Discussion: the kermack-mckendrick epidemic threshold theorem. *Bulletin of mathematical biology*, 53(1-2):1, 1991.
- [2] Y. M. Bar-On, A. Flamholz, R. Phillips, and R. Milo. Science forum: Sars-cov-2 (covid-19) by the numbers. *Elife*, 9:e57309, 2020.
- [3] I. Cooper, A. Mondal, and C. G. Antonopoulos. A sir model assumption for the spread of covid-19 in different communities. *Chaos, Solitons & Fractals*, 139:110057, 2020.
- [4] J. Fernández-Villaverde and C. I. Jones. Estimating and simulating a sird model of covid-19 for many countries, states, and cities. Technical report, National Bureau of Economic Research, 2020.
- [5] M. Fudolig and R. Howard. The local stability of a modified multi-strain sir model for emerging viral strains. *medRxiv*, 2020.
- [6] J. Joo and J. L. Lebowitz. Population dynamics in spatially heterogeneous systems with drift: The generalized contact process. *Physical Review E*, 72(3):036112, 2005.
- [7] W. MathWorld. Kermack-mckendrick model.
- [8] M. A. of America. The sir model for spread of disease - the differential equation model.
- [9] H. H. Weiss. The sir model and the foundations of public health. *Materials matemáticas*, pages 0001–17, 2013.
- [10] S. Zhao and H. Chen. Modeling the epidemic dynamics and control of covid-19 outbreak in china. *Quantitative Biology*, pages 1–9, 2020.

Appendix A. The Basic SIR Model

Appendix A.1. ODE Model Simulation

Simulated the S, I, R populations over time under different b and k . It is obvious that if k is approximate zero, the infectious population gets larger but cannot recover, hence all population will get be infectious eventually. It is also obvious that if k is approximate one, infectious people are sure to removed, hence no more susceptible people get infected. This two special but trivial cases were not simulated. We simulated for $b = 1/5, 2/5, 3/5, 4/5, 1, 2$ and $k = 1/5, 2/5, 3/5, 4/5$.

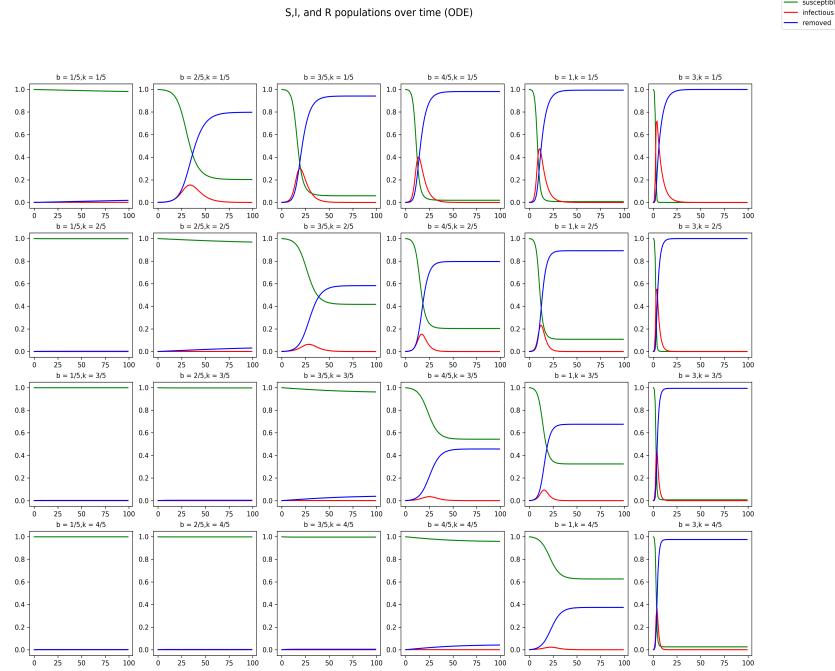


Figure A.13: Example plot for ODE simulation under different values of b and k

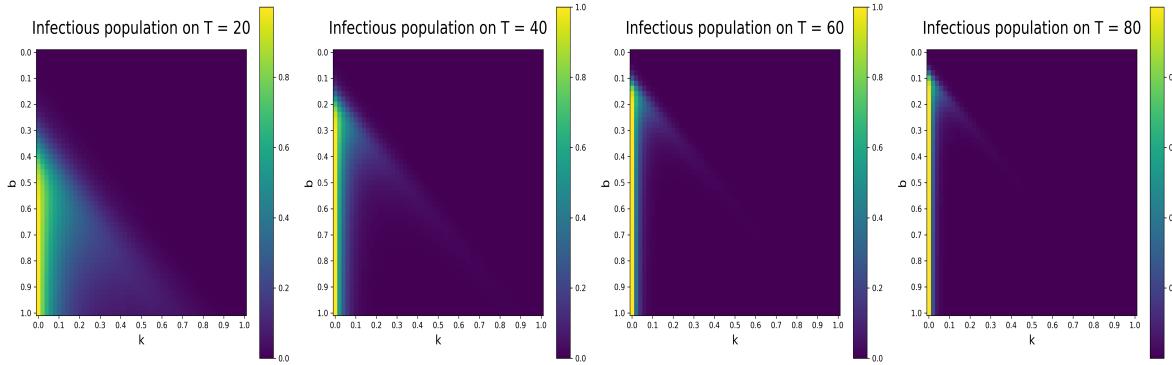


Figure A.14: Phase diagram of infectious population for ODE simulation

From the phase diagrams,

1. The dark area on top right of each diagram indicates that if $k > b$, infectious population size is close to zero all the time, which means that there is almost no surge of infectious population and the initial infectious people are soon removed. It agrees with the result of Image 1.

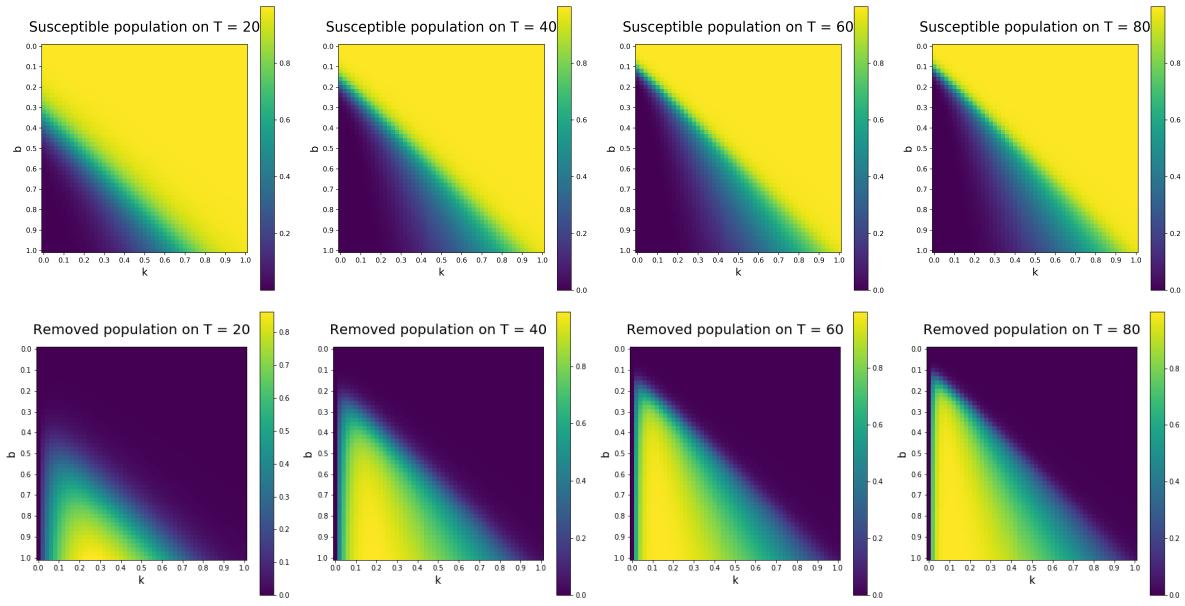


Figure A.15: Phase diagrams of susceptible and removed population for ODE simulation

2. The lighter area first appears in the lower left corner, then moves in the upper right and become darker itself at the same time. It indicates that if $k < b$, when the difference between k and b is larger, the surge of infectious population appears earlier and greater. It is also earlier and quicker for the infectious population goes to zero.
3. If $k > b$, susceptible population proportion is always close to one and removed population proportion is always close to zero, since there are almost no more infectious people.
4. IF $k < b$, After the surge and recover of infectious population, the susceptible population proportion at the end is smaller when the difference between b and k is greater. It means if b is greater enough than b , everyone is eventually infected.

Appendix A.2. Agent-based Model Simulation

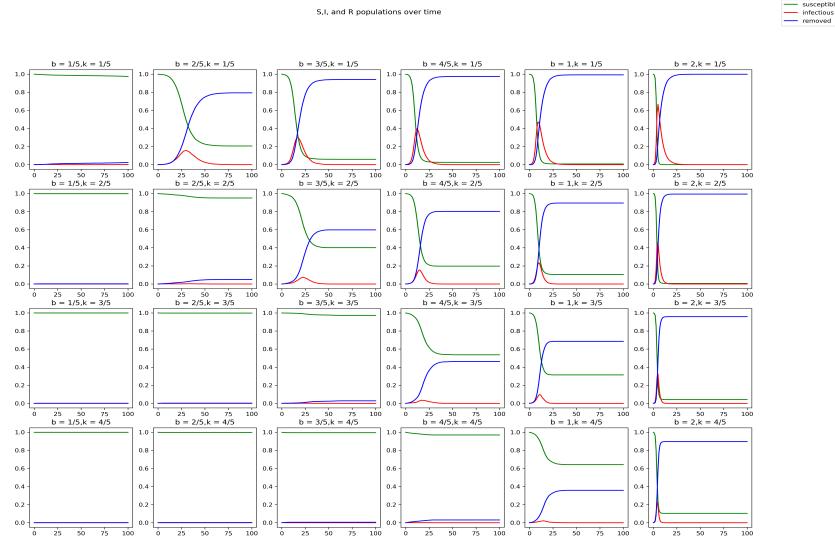


Figure A.16: Example plot for agent-based model simulation under different values of b and k

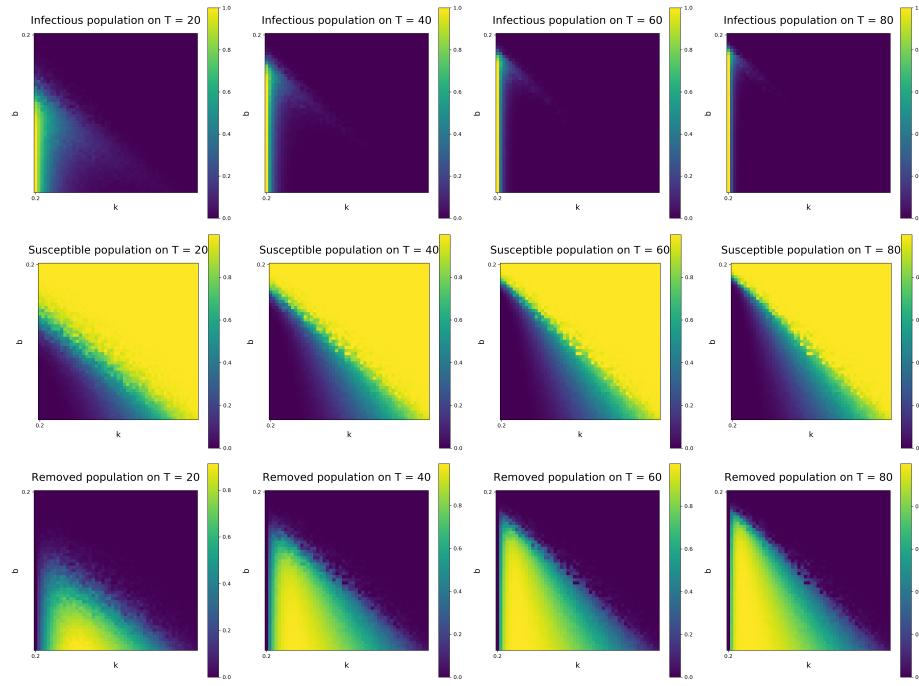


Figure A.17: Phase diagrams of susceptible and removed population for agent-based model simulation

Appendix A.3. SUQC Model

- Disease spreads with different rate at which the cases are confirmed

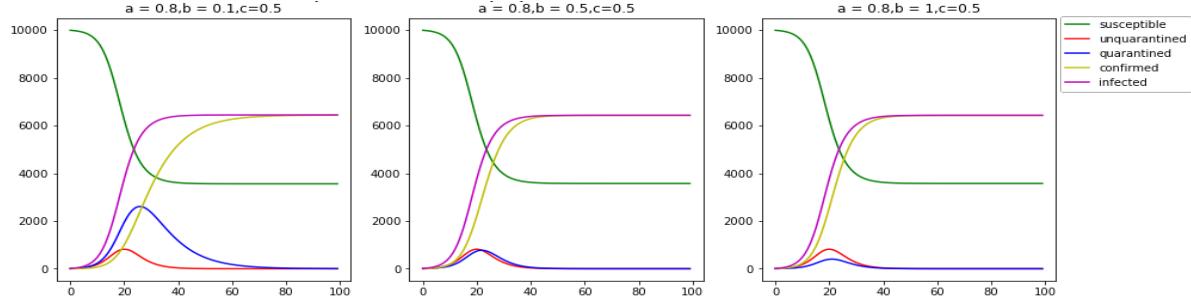


Figure A.18: Compare $S\ U\ Q\ C\ I$ subpopulations with different b

b	0.1	0.5	1
balance day	51	51	51
balance number of the susceptible	3562	3576	3577
balance number of the infected	6438	6424	6423

The parameter b represents the rate at which the cases are confirmed, which only affect the speed of Q (the infected and quarantined) transformed to C (the confirmed case). Since both Q and C lose ability to infect the susceptible, the changes of b does not exert much influence on the final amount of the infected and the susceptible.

Appendix A.4. SIRDC Model

- Disease spreads with different fraction of people exiting the resolving state

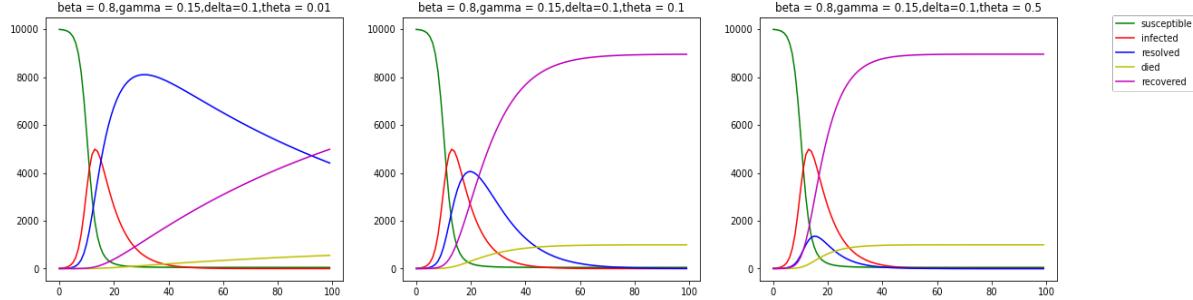


Figure A.19: Compare S I R D C subpopulations with different θ

c	0.01	0.2	0.5
balance day	69	68	68
number of the susceptible	50	49	49
number of the infected	2	2	2
number of the resolved	5953	98	1
number of the dead	399	984	994

- Disease spreads with different fraction of the resolved consequences being died per day

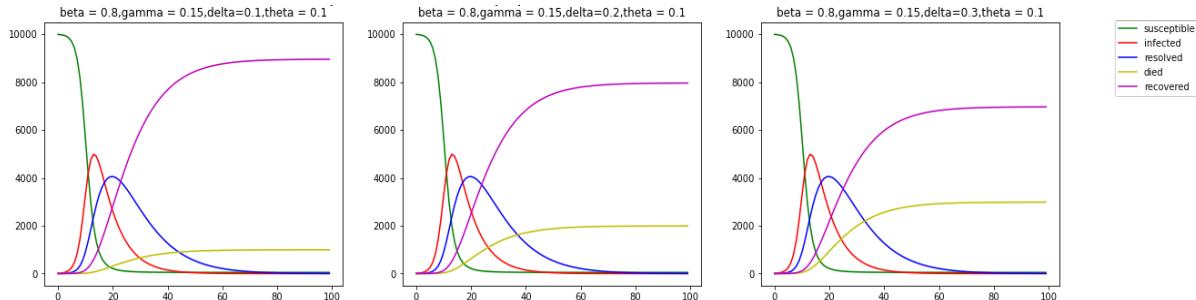


Figure A.20: Compare S I R D C subpopulations with different δ

c	0.1	0.2	0.3
balance day	68	68	68
number of the susceptible	49	49	49
number of the infected	2	2	2
number of the resolved	98	98	98
number of the dead	984	1969	2954